SECTION-1: Identification of the substance / mixture and the company / undertaking

Catalogue Number	CS-T-87453
Product Name	Triglycol
CAS No.	112-27-6
Category	Reagents
Synonyms	-
Brand	Clearsynth Labs Ltd.
Identified uses	Laboratory Chemicals
Uses advised against	Not available
Company	Clearsynth Labs Ltd. Mumbai, India
Emergency Phone #	+91-22-245045900
REACH No.	Not available

SECTION 2: Hazards identification

Disclaimer: This is sample MSDS. Please email **sales@clearsynth.com** for more details.

2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Not available

2.2 Label Elements

Signal Word: Not available

Not available

Hazard Statement(s)

Code	Statement
Not available	Not available

Precautionary Statement(s)

Code	Statement
Not available	Not available

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Triglycol
CAS Number : 112-27-6
Molecular Formula : C■H■■O■
Molecular Weight : 150.17
Parent Chemical : -
Synonyms : -
Concentration : Not available

SECTION 4: First aid measures

SECTION 4: First-aid measures

4.1 Description of first aid measures
- General advice: Remove contaminated clothing and shoes. Seek medical attention if symptoms persist.
- Inhalation: Move person to fresh air. If breathing is difficult, seek medical attention.
- Skin contact: Wash with plenty of soap and water. Get medical attention if irritation develops or persists.
- Eye contact: Rinse cautiously with water for several minutes. Remove contact lenses if present and easy to do. Continue rinsing. Seek medical attention if irritation persists.
- Ingestion: Rinse mouth. Do not induce vomiting unless directed by medical personnel. Seek medical attention if you feel unwell.

4.2 Most important symptoms and effects, both acute and delayed
- Not available.

4.3 Indication of any immediate medical attention and special treatment needed
- Treat symptomatically. No data available.

SECTION 5: Firefighting measures

SECTION 5: Fire-fighting measures

5.1 Extinguishing media
- Suitable extinguishing media: Use extinguishing measures appropriate to surrounding fire (e.g., water spray, alcohol-resistant foam, dry chemical, carbon dioxide).
- Unsuitable extinguishing media: Not available.

5.2 Special hazards arising from the substance or mixture
- Specific hazards: Not available.
- Hazardous combustion products: Not available.

5.3 Advice for firefighters
- Protective equipment: Wear self-contained breathing apparatus and full protective gear.
- Additional information: Cool containers exposed to fire with water spray. Prevent fire-fighting water from entering drains or waterways.

SECTION 6: Accidental release measures

SECTION 6: Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures

- Avoid contact with skin and eyes. Avoid breathing vapors/mist.
- Use appropriate personal protective equipment (see Section 8).
- Ensure adequate ventilation.

6.2 Environmental precautions
- Avoid release to the environment. Prevent entry into drains, surface waters, and soil.

6.3 Methods and material for containment and cleaning up
- Contain spill. Absorb with inert material (e.g., sand, earth, vermiculite).
- Collect into suitable, labeled containers for disposal.
- Wash spill area after material pickup is complete.

6.4 Reference to other sections
- See Section 8 for personal protective equipment and Section 13 for disposal considerations.

SECTION-7: Handling and storage

SECTION 7: Handling and storage

7.1 Precautions for safe handling
- Handle in accordance with good industrial hygiene and safety practice.
- Avoid contact with skin, eyes, and clothing.
- Avoid breathing vapors/mist. Use with adequate ventilation.
- Keep container tightly closed when not in use.

7.2 Conditions for safe storage, including any incompatibilities
- Store in a cool, dry, well-ventilated place.
- Keep away from incompatible materials.
- Incompatible materials: Not available.

7.3 Specific end use(s)
- Reagent. No data available for specific risk management measures.

SECTION 8: Exposure controls / personal protection

SECTION 8: Exposure controls/personal protection

8.1 Control parameters
- Occupational exposure limits: Not available.
- Biological limit values: Not available.

8.2 Exposure controls
- Engineering controls: Provide adequate general and/or local exhaust ventilation to control airborne levels.
- Personal protective equipment (PPE):
- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Protective gloves. Wear appropriate protective clothing.
- Respiratory protection: If ventilation is inadequate, use appropriate respiratory protection.
- Hygiene measures: Wash hands thoroughly after handling. Do not eat, drink, or smoke when using this product.

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	Clear viscous liquid
IR spectrum	No data available
pH	No data available
Solubility	Complies

Property	Value
a) Physical State	No data available
b) Color	No data available
c) Odor	No data available
d) pH	No data available
e) Vapour Pressure	No data available
f) Viscosity	No data available
g) Initial Boiling Point and boiling range	No data available
h) Melting Point / Freezing Point	No data available
i) Auto Ignition Temperature	No data available
j) Flash Point	No data available
k) Explosion Limit, Lower	No data available
l) Explosion Limit, Upper	No data available
m) Decomposition Temperature	No data available
n) Loss on Drying	No data available
o) Relative Density	No data available
p) Solubility (in DMSO)	No data available
q) Oxidizing Properties	No data available

SECTION 10: Stability and reactivity

SECTION 10: Stability and reactivity

10.1 Reactivity

- No data available.

10.2 Chemical stability
- Stable under recommended storage conditions.

10.3 Possibility of hazardous reactions
- No data available.

10.4 Conditions to avoid
- Not available.

10.5 Incompatible materials
- Not available.

10.6 Hazardous decomposition products
- Not available.

SECTION 11: Toxicological information

11.1 Information on toxicological effects
- Acute toxicity: Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin iritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is normally by skin and eye contact. Local and systemic adverse health effects by cutaneous exposure are likely not to occur, and eye contact will produce transient irritation without corneal injury. The very low vapor pressure of TEG makes it unlikely that significant vapor exposure will occur. Aerosol exposure is not a usual exposure mode, and

acute aerosol exposures are unlikely to be harmful, although a peripheral sensory irritant effect may develop. However, repeated exposures to a TEG aerosol may result in respiratory tract irritation, with cough, shortness of breath and tightness of the chest. Recommended protective and precautionary measures include protective gloves, goggles or safety glasses and mechanical room ventilation. LC(50) data to various fish, aquatic invertebrates and algae, indicate that TEG is essentially nontoxic to aquatic organisms. Also, sustained exposure studies have demonstrated that TEG is of a low order of chronic aquatic toxicity. The bioconcentration potential, environmental hydrolysis, and photolysis rates are low, and soil mobility high. In the atmosphere TEG is degraded by reacting with photochemically produced hydroxyl radicals. These considerations indicate that the potential for ecotoxicological effects with TEG is low. /SIGNS AND SYMPTOMS/ Splash contamination in man ... causes acute smarting, & this may be followed by transitory disturbance of corneal epithelium with gradually diminishing sensation & signs of irritation, but no persistent injury is to be expected.
- Skin corrosion/irritation: /LABORATORY ANIMALS: Acute Exposure/ Primary Dermal Irritation Study in Rabbits. Methods: In one study, 2 sq cm gauze pads wetted with triethylene glycol were applied to shaven abdomen of rabbits and held in place ... for 24 hr. The 24 hr evaluation indicated no irritation ... . In another study, the chemical was used without dilution. Neodermotest occlusive patches were held with absorbent gauze and adhesive bandage to the left and right flank of the rabbits... . Irritation was scored: non-irritant, < 0.5 and slightly irritant, 0.5 - 2.0. Results: The above studies indicated that the primary dermal irritation potential of triethylene glycol was negligible. The mean dermal irritation score from /the second/ study was 0.08...
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin iritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is

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- Germ cell mutagenicity: Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin iritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is normally by skin and eye contact. Local and systemic adverse health effects by cutaneous exposure are likely not to occur, and eye contact will produce transient irritation without corneal injury. The very low vapor pressure of TEG makes it unlikely that significant vapor exposure will occur. Aerosol exposure is not a usual exposure mode, and acute aerosol exposures are unlikely to be harmful, although a peripheral sensory irritant effect may develop. However, repeated exposures to a TEG aerosol may result in respiratory tract irritation, with cough, shortness of breath and tightness of the chest. Recommended protective and precautionary measures include protective gloves,

goggles or safety glasses and mechanical room ventilation. LC(50) data to various fish, aquatic invertebrates and algae, indicate that TEG is essentially nontoxic to aquatic organisms. Also, sustained exposure studies have demonstrated that TEG is of a low order of chronic aquatic toxicity. The bioconcentration potential, environmental hydrolysis, and photolysis rates are low, and soil mobility high. In the atmosphere TEG is degraded by reacting with photochemically produced hydroxyl radicals. These considerations indicate that the potential for ecotoxicological effects with TEG is low.
- Carcinogenicity: Inadequate information to assess carcinogenic potential
- Reproductive toxicity: Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin iritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is normally by skin and eye contact. Local and systemic adverse health effects by cutaneous exposure are likely not to occur, and eye contact will produce transient irritation without corneal injury. The very low vapor pressure of TEG makes it unlikely that significant vapor exposure will occur. Aerosol exposure is not a usual exposure mode, and acute aerosol exposures are unlikely to be harmful, although a peripheral sensory irritant effect may develop. However, repeated exposures to a TEG aerosol may result in respiratory tract irritation, with cough, shortness of breath and tightness of the chest. Recommended protective and precautionary measures include protective gloves, goggles or safety glasses and mechanical room ventilation. LC(50) data to various fish, aquatic invertebrates and algae, indicate that TEG is essentially nontoxic to aquatic organisms. Also, sustained exposure studies have demonstrated that TEG is of a low order of chronic aquatic toxicity. The bioconcentration potential, environmental hydrolysis, and photolysis rates are low, and soil mobility high. In the atmosphere TEG is degraded by reacting with photochemically produced hydroxyl radicals. These considerations indicate that the potential for ecotoxicological effects with TEG is low.

- STOT-single exposure: No data available.
- STOT-repeated exposure: Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin irritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is normally by skin and eye contact. Local and systemic adverse health effects by cutaneous exposure are likely not to occur, and eye contact will produce transient irritation without corneal injury. The very low vapor pressure of TEG makes it unlikely that significant vapor exposure will occur. Aerosol exposure is not a usual exposure mode, and acute aerosol exposures are unlikely to be harmful, although a peripheral sensory irritant effect may develop. However, repeated exposures to a TEG aerosol may result in respiratory tract irritation, with cough, shortness of breath and tightness of the chest. Recommended protective and precautionary measures include protective gloves, goggles or safety glasses and mechanical room ventilation. LC(50) data to various fish, aquatic invertebrates and algae, indicate that TEG is essentially nontoxic to aquatic organisms. Also, sustained exposure studies have demonstrated that TEG is of a low order of chronic aquatic toxicity. The bioconcentration potential, environmental hydrolysis, and photolysis rates are low, and soil mobility high. In the atmosphere TEG is degraded by reacting with photochemically produced hydroxyl radicals. These considerations indicate that the potential for ecotoxicological effects with TEG is low.
- Aspiration hazard: No data available.

Likely routes of exposure
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human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably due to hemodilution following absorption of TEG. The NOAEL was 20,000 ppm TEG in diet. Short-term repeated aerosol exposure studies in the rat demonstrated that, by nose-only exposure, the threshold for effects by respiratory tract exposure was 1036 mg/cu m. Neither high dosage acute nor repeated exposures to TEG produce hepatorenal injury characteristic of that caused by the lower glycol homologues. Elimination studies with acute peroral doses of TEG given to rats and rabbits showed high recoveries (91-98% over 5 days), with the major fraction appearing in urine (84-94%) and only 1% as carbon dioxide. TEG in urine is present in unchanged and oxidized forms, but only negligible amounts as oxalic acid. Developmental toxicity studies with undiluted TEG given by gavage produced maternal toxicity in rats (body weight, food consumption, water consumption, and relative kidney weight) with a NOEL of 1126 mg/kg/day, and mice (relative kidney weight) with a NOEL of 5630 mg/kg/day. Developmental toxicity, expressed as fetotoxicity, had a NOEL of 5630 mg/kg/day with the rat and 563 mg/kg/day with mice. Neither species showed any evidence of embryotoxicity or teratogenicity. There was no evidence for reproductive toxicity with mice given up to 3% TEG in drinking water in a continuous breeding study. TEG did not produce mutagenic or clastogenic effects in the following in vitro genetic toxicology studies: Salmonella typhimurium reverse mutation test, SOS-chromotest in E. coli, CHO forward gene mutation test (HGPRT locus), CHO sister chromatid exchange test, and a chromosome aberration test with CHO cells. The use patterns suggest that exposure to TEG is mainly occupational, with limited exposures by consumers. Exposure is normally by skin and eye contact. Local and systemic adverse health effects by cutaneous exposure are likely not to occur, and eye contact will produce transient irritation without corneal injury. The very low vapor pressure of TEG makes it unlikely that significant vapor exposure will occur. Aerosol exposure is not a usual exposure mode, and acute aerosol exposures are unlikely to be harmful, although a peripheral sensory irritant effect may develop. However, repeated exposures to a TEG aerosol may result in respiratory tract irritation, with cough, shortness of breath and tightness of the chest. Recommended protective and precautionary measures include protective gloves, goggles or safety glasses and mechanical room ventilation. LC(50) data to various fish, aquatic invertebrates and algae, indicate that TEG is essentially nontoxic to aquatic organisms. Also, sustained exposure studies have demonstrated that TEG is of a low order of chronic aquatic toxicity. The bioconcentration potential, environmental hydrolysis, and photolysis rates are low, and soil mobility high. In the atmosphere TEG is degraded by reacting with photochemically produced hydroxyl radicals. These considerations indicate that the potential for ecotoxicological effects with TEG is low.

Symptoms related to the physical, chemical and toxicological characteristics
- Triethylene glycol (TEG) is a liquid higher glycol of very low vapor pressure with uses that are primarily industrial. It has a very low order of acute toxicity by iv, ip, peroral, percutaneous and inhalation (vapor and aerosol) routes of exposure. It does not produce primary skin iritation. Acute eye contact with the liquid causes mild local transient irritation (conjunctival hyperemia and slight chemosis) but does not induce corneal injury. Animal maximization and human volunteer repeated insult patch tests studies have shown that TEG does not cause skin sensitization. A study with Swiss-Webster mice demonstrated that TEG aerosol has properties of a peripheral chemosensory irritant material and caused a depression of breathing rate with an RD(50) of 5140 mg/ cu m. Continuous subchronic peroral dosing of TEG in the diet of rats did not produce any systemic cumulative or long-term toxicity. The effects seen were dose-related increased relative kidney weight, increased urine volume and decreased urine pH, probably a result of the renal excretion of TEG and metabolites following the absorption of large doses of TEG. There was also decreased hemoglobin concentration, decreased hematocrit and increased mean corpuscular volume, probably

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SECTION 12: Ecological information

SECTION 12: Ecological information

12.1 Toxicity
- No data available.

12.2 Persistence and degradability
- No data available.

12.3 Bioaccumulative potential
- No data available.

12.4 Mobility in soil
- No data available.

12.5 Results of PBT and vPvB assessment
- Not available.

12.6 Endocrine disrupting properties
- No data available.

12.7 Other adverse effects
- No data available.

SECTION 13: Disposal considerations

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13.1 Waste treatment methods
- Dispose of contents/container in accordance with local/regional/national/international regulations.
- Do not discharge to drains or the environment.
- Recommended disposal method: Not available (follow site-specific waste evaluation).
- Contaminated packaging: Dispose of as unused product unless cleaned and permitted by regulations.

SECTION 14: Transport information

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- UN number: Not available.
- UN proper shipping name: Not available.
- Transport hazard class(es): Not available.
- Packing group: Not available.
- Environmental hazards: Not available.
- Special precautions for user: Not available.
- Transport in bulk according to IMO instruments: Not available.

SECTION 15: Regulatory information

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15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture
- Not available.

15.2 Chemical safety assessment
- No data available.

SECTION 16: Other information

SECTION 16: Other information

- Product name: Triglycol
- Catalog No.: CS-T-87453
- CAS No.: 112-27-6
- Category: Reagents
- Molecular weight: 150.17
- Supplier: Clearsynth Labs Ltd., Mumbai, India
- Emergency phone: +91-22-245045900

Disclaimer: The information provided is based on data available at the time of preparation and is intended for guidance for safe handling, use, processing, storage, transportation, disposal, and release. It does not constitute a

warranty of any kind. Not all data may be available for this product.

DISCLAIMER

This MSDS is system-generated. Please verify and confirm all data, statements, and values with the Support Team before use or distribution.