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

Catalogue Number	CS-O-36265
Product Name	Vinyl Chloride Solution 2000 µg/mL in methanol
CAS No.	75-01-4
Category	Fine Chemicals
Synonyms	Not available
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:

Skin irritation (Category 2)

2.2 Label Elements

Signal Word: Warning

Hazard Statement(s)

Code	Statement
H220	Not available
H350	Not available
H280	Not available
H341	Not available

H412	Not available
H336	Not available
H361	Not available
H370	Not available
H372	Not available
H315	Causes skin irritation.
H320	Not available

Precautionary Statement(s)

Code	Statement
P203	Not available
P210	Not available
P222	Not available
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P318	Not available
P377	Not available
P381	Not available
P403	Not available
P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P273	Not available
P410+P403	Not available
P260	Not available
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P270	Not available
P271	Use only outdoors or in a well-ventilated area.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P308+P316	Not available
P319	Get medical help if you feel unwell.

P321	Specific treatment (see … on this label).
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P332+P317	If skin irritation occurs: Get medical help.
P362+P364	Take off contaminated clothing and wash it before reuse.
P264+P265	Not available
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if presen
P337+P317	If eye irritation persists: Get medical help.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Vinyl Chloride Solution 2000 µg/mL in methanol
CAS Number : 75-01-4
Molecular Formula : C2H3Cl
Molecular Weight : 62.50
Parent Chemical : Not available
Synonyms : Not available
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 or are severe.
Inhalation: Move person to fresh air. Keep at rest. If breathing is difficult, seek medical attention.
Skin contact: Wash with plenty of soap and water. Seek 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.
Ingestion: Rinse mouth. Do NOT induce vomiting. Seek medical attention.

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 local circumstances and the surrounding environment (e.g., dry chemical, carbon dioxide, alcohol-resistant foam, water spray).
Unsuitable extinguishing media: Not available.

5.2 Special hazards arising from the substance or mixture
Flammable liquid and vapor (methanol component). Vapors may form explosive mixtures with air. Thermal decomposition may produce hazardous gases/fumes. Specific decomposition products: Not available.

5.3 Advice for firefighters
Wear self-contained breathing apparatus (SCBA) and full protective gear. Cool containers with water spray. Fight fire from a safe distance. 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
Evacuate unnecessary personnel. Eliminate all ignition sources. Ensure adequate ventilation. Avoid breathing vapors/mist. Avoid contact with skin and eyes. Wear appropriate personal protective equipment.

6.2 Environmental precautions
Prevent further leakage or spillage if safe to do so. Prevent entry into waterways, sewers, basements or confined areas.

6.3 Methods and material for containment and cleaning up
Contain spill. Absorb with inert material (e.g., sand, silica gel, universal binder). Collect in suitable, closed containers for disposal. Clean contaminated area with appropriate methods while avoiding ignition sources.

6.4 Reference to other sections
For personal protective equipment see Section 8. For disposal considerations see Section 13.

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. Use only with adequate ventilation (preferably in a fume hood). Avoid breathing vapors/mist. Avoid contact with skin, eyes, and clothing. Keep away from heat, sparks, open flames, and hot surfaces. Use non-sparking tools and explosion-proof equipment where applicable. Ground/bond container and receiving equipment.

7.2 Conditions for safe storage, including any incompatibilities
Store tightly closed in a cool, dry, well-ventilated place. Keep away from ignition sources. Protect from heat and direct sunlight. Store in appropriate flammable-liquid storage area/cabinet. Incompatible materials: Not available.

7.3 Specific end use(s)
Laboratory/research use. No data available.

SECTION 8: Exposure controls / personal protection

SECTION 8: Exposure controls/personal protection

8.1 Control parameters
Occupational exposure limits:
- Vinyl chloride (CAS 75-01-4): Not available.
- Methanol (CAS 67-56-1): Not available.
Biological limit values: Not available.

8.2 Exposure controls
Appropriate engineering controls: Use local exhaust ventilation or general dilution ventilation to maintain exposure below applicable limits. Use explosion-proof ventilation where required.
Personal protective equipment (PPE):
- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Wear protective gloves (material selection dependent on use conditions; no data available). Wear protective clothing.
- Respiratory protection: If ventilation is inadequate, use appropriate respiratory protection. Specific respirator type: Not available.
- Hygiene measures: Wash hands after handling. Do not eat, drink, or smoke when using this product. Remove contaminated clothing and wash before reuse.
Environmental exposure controls: Avoid release to the environment.

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	No data available
IR spectrum	No data available
pH	No data available
Solubility	No data available

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

Property	Value
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
Heat, flames, sparks, and other ignition sources. Exposure to elevated temperatures. Static discharge.

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: IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with

reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found in 36 workers with vinyl chloride-induced hepatic injury following long-time industrial exposure. Pathologic porphyrinuria, especially secondary coproporphyrinuria with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions. The major immunological abnormalities reported in vinyl chloride disease patients include hyperimmunoglobulinemia with a polyclonal increase in IgG, cryoglobulinemia, cryofibrinogenemia, and in vivo activation of complement. Vinyl chloride is an occupational carcinogen which caused micronuclei in human cells. There was significant increase in chromosomal abnormalities in cultured peripheral lymphocytes from 57 male workers when compared with controls. Sister chromatid exchange was the more sensitive endpoint for indicating a biological response. ANIMAL STUDIES: Brief (30 minutes) exposures to concentrations of vinyl chloride ranging from 100,000 to 400,000 ppm have been shown to be fatal in rats, guinea pigs and mice. Symptoms of intoxication in rats and mice include muscular incoordination and twitching, CNS depression and respiratory failure. Intense salivation and lacrimation have been noted in rats, guinea pigs and rabbits exposed acutely to high concentrations (375-700 mg/L) of vinyl chloride gas. When placed on skin or in eyes, liquid vinyl chloride may freeze tissue and produce a chemical burn as it evaporates, causing damage to the underlying tissue. Profound CNS depression was reported in guinea-pigs exposed to vinyl chloride at 65,000 mg/cu m for 90 min. Ataxia was observed at this dose level after 5 min of exposure. The anesthetic action of vinyl chloride was also observed in dogs and mice. Investigators reported deep CNS depression in rats and mice exposed to 260,000 mg/cu m for 30 min. The CNS depressant effect was preceded by increased motor activity after 5 min of exposure, twitching of extremities (after 10 min), ataxia (after 15 min) and tremor (after 15 min). Rats exposed to 130,000 mg/cu m for 60 min showed ataxia preceded by hyperactivity but no /CNS depressant/ effect. Forty rabbits were exposed for 4 hours/day on 5 days/week for 12 months to air containing (10,000 ppm) vinyl chloride. Between 9 and 15 months exposure, 12 skin acanthomas and 6 lung adenocarcinomas were seen. No similar tumors occurred in 20 controls after 15 months observation. Rats were exposed to 10,000 ppm vinyl chloride in air for 4 hours/day on 5 days/week for 5 weeks, starting at the age of 13 weeks (120 rats per group) or 1 day (43 and 46 rats). Animals were observed for 135 weeks. One hepatoma was reported in the older rats in newborn rats, 10 angiosarcomas and 15 hepatomas were found. No liver tumors were reported in 249 controls. Vinyl chloride was administered for 7 hr/day on days 6-18 of gestation in mice, rats, and rabbits. It was concluded that although maternal toxicity observed, vinyl chloride alone did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at concentrations tested. Vinyl chloride produced a significant increase in the frequency of recessive lethal mutations in male Drosophila melangaster. Mutagenic activity of vinyl chloride was reported in yeast (S. pombe and S. cerevisiae) in the presence of metabolic activation. Vinyl chloride was mutagenic to S. pombe in the "host mediated" assay when mice were treated with an oral dose of 700 mg/kg of vinyl chloride. Using Salmonella tester strains, direct mutagenicity of vinyl chloride was reported at 20% (v/v) in air (200,000 ppm) in the absence of metabolic activation. Mutagenic response was increased by metabolic activation. However, 20% vinyl chloride (v/v in air) was inactive in systems employing S. typhimurium strains TA1536, TA1537 and TA1538. ECOTOXICITY STUDIES: In Daphnia magna exposure, results indicated impacts of vinyl chloride on the regulation of genes related to glutathione-S-transferase (GST), juvenile hormone esterase (JHE), and the vitelline outer layer membrane protein (VMO1). Vinyl chloride poisoning exhibits many of the characteristics of autoimmune diseases. This is believed to be the result of a reactive vinyl chloride intermediate metabolite binding to an immunoglobulin, altering the protein and initiating an immune response. The metabolites of vinyl chloride, especially choloroethylene

oxide, are mutagenic and act by covalently binding to DNA. This produces cyclic etheno-adducts, which cause base-pair transitions during transcription and DNA crosslinks. Metabolites also may cause oxidative stress and affecting tumor supressor genes, as vinyl chloride has been known to produce specific mutations in the p53 and Ki-ras genes. Vinyl chloride metabolites are also believed to exert toxic effects in the liver by covalently binding to liver proteins, resulting in cellular toxicity. (L3, A65)
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found in 36 workers with vinyl chloride-induced hepatic injury following long-time industrial exposure. Pathologic porphyrinuria, especially secondary coproporphyrinuria with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions. The major immunological abnormalities reported in vinyl chloride disease patients include hyperimmunoglobulinemia with a polyclonal increase in IgG, cryoglobulinemia, cryofibrinogenemia, and in vivo activation of complement. Vinyl chloride is an occupational carcinogen which caused micronuclei in human cells. There was significant increase in chromosomal abnormalities in cultured peripheral lymphocytes from 57 male workers when compared with controls. Sister chromatid exchange was the more sensitive endpoint for indicating a biological response. ANIMAL STUDIES: Brief (30 minutes) exposures to concentrations of vinyl chloride ranging from 100,000 to 400,000 ppm have been shown to be fatal in rats, guinea pigs and mice. Symptoms of intoxication in rats and mice include muscular incoordination and twitching, CNS depression and respiratory failure. Intense salivation and lacrimation have been noted in rats, guinea pigs and rabbits exposed acutely to high concentrations (375-700 mg/L) of vinyl chloride gas. When placed on skin or in eyes, liquid vinyl chloride may freeze tissue and produce a chemical burn as it evaporates, causing damage to the underlying tissue. Profound CNS depression was reported in guinea-pigs exposed to vinyl chloride at 65,000 mg/cu m for 90 min. Ataxia was observed at this dose level after 5 min of exposure. The anesthetic action of vinyl chloride was also observed in dogs and mice. Investigators reported deep CNS depression in rats and mice exposed to 260,000 mg/cu m for 30 min. The CNS depressant effect was preceded by increased motor activity after 5 min of exposure, twitching of extremities (after 10 min), ataxia (after 15 min) and tremor (after 15 min). Rats exposed to 130,000 mg/cu m for 60 min showed ataxia preceded by hyperactivity but no /CNS depressant/ effect. Forty rabbits were exposed for 4 hours/day on 5 days/week for 12 months to air containing (10,000 ppm) vinyl chloride. Between 9 and 15 months exposure, 12 skin acanthomas and 6 lung adenocarcinomas were seen. No similar tumors occurred in 20 controls after 15 months observation. Rats were exposed to 10,000 ppm vinyl chloride in air for 4 hours/day on 5 days/week for 5 weeks, starting at the age of 13 weeks (120 rats per group) or 1 day (43 and 46 rats). Animals were observed for 135 weeks. One hepatoma was reported in the older rats in newborn rats,

10 angiosarcomas and 15 hepatomas were found. No liver tumors were reported in 249 controls. Vinyl chloride was administered for 7 hr/day on days 6-18 of gestation in mice, rats, and rabbits. It was concluded that although maternal toxicity observed, vinyl chloride alone did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at concentrations tested. Vinyl chloride produced a significant increase in the frequency of recessive lethal mutations in male Drosophila melangaster. Mutagenic activity of vinyl chloride was reported in yeast (S. pombe and S. cerevisiae) in the presence of metabolic activation. Vinyl chloride was mutagenic to S. pombe in the "host mediated" assay when mice were treated with an oral dose of 700 mg/kg of vinyl chloride. Using Salmonella tester strains, direct mutagenicity of vinyl chloride was reported at 20% (v/v) in air (200,000 ppm) in the absence of metabolic activation. Mutagenic response was increased by metabolic activation. However, 20% vinyl chloride (v/v in air) was inactive in systems employing S. typhimurium strains TA1536, TA1537 and TA1538. ECOTOXICITY STUDIES: In Daphnia magna exposure, results indicated impacts of vinyl chloride on the regulation of genes related to glutathione-S-transferase (GST), juvenile hormone esterase (JHE), and the vitelline outer layer membrane protein (VMO1). Vinyl chloride poisoning exhibits many of the characteristics of autoimmune diseases. This is believed to be the result of a reactive vinyl chloride intermediate metabolite binding to an immunoglobulin, altering the protein and initiating an immune response. The metabolites of vinyl chloride, especially choloroethylene oxide, are mutagenic and act by covalently binding to DNA. This produces cyclic etheno-adducts, which cause base-pair transitions during transcription and DNA crosslinks. Metabolites also may cause oxidative stress and affecting tumor supressor genes, as vinyl chloride has been known to produce specific mutations in the p53 and Ki-ras genes. Vinyl chloride metabolites are also believed to exert toxic effects in the liver by covalently binding to liver proteins, resulting in cellular toxicity. (L3, A65)
- Carcinogenicity: IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found in 36 workers with vinyl chloride-induced hepatic injury following long-time industrial exposure. Pathologic porphyrinuria, especially secondary coproporphyrinuria with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions. The major immunological abnormalities reported in vinyl chloride disease patients include hyperimmunoglobulinemia with a polyclonal increase in IgG, cryoglobulinemia, cryofibrinogenemia, and in vivo activation of complement. Vinyl chloride is an occupational carcinogen which caused micronuclei in human cells. There was significant increase in chromosomal abnormalities in cultured peripheral lymphocytes from 57 male workers when compared with controls. Sister chromatid exchange was the more sensitive endpoint for indicating a biological response. ANIMAL STUDIES: Brief (30 minutes) exposures to concentrations of vinyl chloride ranging from 100,000 to 400,000 ppm have been shown to be fatal in rats, guinea pigs and mice. Symptoms of intoxication in rats and mice include muscular incoordination and twitching, CNS depression and respiratory failure. Intense salivation and lacrimation have been noted in rats, guinea pigs and rabbits exposed acutely to high concentrations (375-700 mg/L) of vinyl chloride gas. When placed

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- Reproductive toxicity: IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found

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- STOT-single exposure: No data available.
- STOT-repeated exposure: IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of

acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found in 36 workers with vinyl chloride-induced hepatic injury following long-time industrial exposure. Pathologic porphyrinuria, especially secondary coproporphyrinuria with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions. The major immunological abnormalities reported in vinyl chloride disease patients include hyperimmunoglobulinemia with a polyclonal increase in IgG, cryoglobulinemia, cryofibrinogenemia, and in vivo activation of complement. Vinyl chloride is an occupational carcinogen which caused micronuclei in human cells. There was significant increase in chromosomal abnormalities in cultured peripheral lymphocytes from 57 male workers when compared with controls. Sister chromatid exchange was the more sensitive endpoint for indicating a biological response. ANIMAL STUDIES: Brief (30 minutes) exposures to concentrations of vinyl chloride ranging from 100,000 to 400,000 ppm have been shown to be fatal in rats, guinea pigs and mice. Symptoms of intoxication in rats and mice include muscular incoordination and twitching, CNS depression and respiratory failure. Intense salivation and lacrimation have been noted in rats, guinea pigs and rabbits exposed acutely to high concentrations (375-700 mg/L) of vinyl chloride gas. When placed on skin or in eyes, liquid vinyl chloride may freeze tissue and produce a chemical burn as it evaporates, causing damage to the underlying tissue. Profound CNS depression was reported in guinea-pigs exposed to vinyl chloride at 65,000 mg/cu m for 90 min. Ataxia was observed at this dose level after 5 min of exposure. The anesthetic action of vinyl chloride was also observed in dogs and mice. Investigators reported deep CNS depression in rats and mice exposed to 260,000 mg/cu m for 30 min. The CNS depressant effect was preceded by increased motor activity after 5 min of exposure, twitching of extremities (after 10 min), ataxia (after 15 min) and tremor (after 15 min). Rats exposed to 130,000 mg/cu m for 60 min showed ataxia preceded by hyperactivity but no /CNS depressant/ effect. Forty rabbits were exposed for 4 hours/day on 5 days/week for 12 months to air containing (10,000 ppm) vinyl chloride. Between 9 and 15 months exposure, 12 skin acanthomas and 6 lung adenocarcinomas were seen. No similar tumors occurred in 20 controls after 15 months observation. Rats were exposed to 10,000 ppm vinyl chloride in air for 4 hours/day on 5 days/week for 5 weeks, starting at the age of 13 weeks (120 rats per group) or 1 day (43 and 46 rats). Animals were observed for 135 weeks. One hepatoma was reported in the older rats in newborn rats, 10 angiosarcomas and 15 hepatomas were found. No liver tumors were reported in 249 controls. Vinyl chloride was administered for 7 hr/day on days 6-18 of gestation in mice, rats, and rabbits. It was concluded that although maternal toxicity observed, vinyl chloride alone did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at concentrations tested. Vinyl chloride produced a significant increase in the frequency of recessive lethal mutations in male Drosophila melangaster. Mutagenic activity of vinyl chloride was reported in yeast (S. pombe and S. cerevisiae) in the presence of metabolic activation. Vinyl chloride was mutagenic to S. pombe in the "host mediated" assay when mice were treated with an oral dose of 700 mg/kg of vinyl chloride. Using Salmonella tester strains, direct mutagenicity of vinyl chloride was reported at 20% (v/v) in air (200,000 ppm) in the absence of metabolic activation. Mutagenic response was increased by metabolic activation. However, 20% vinyl chloride (v/v in air) was inactive in systems employing S. typhimurium strains TA1536, TA1537 and TA1538. ECOTOXICITY STUDIES: In Daphnia magna exposure, results indicated impacts of vinyl chloride on the regulation of genes related to glutathione-S-transferase (GST), juvenile hormone esterase (JHE), and the vitelline outer layer membrane protein (VMO1). Symptoms of acute vinyl chloride exposure include headache, nausea, dizziness, and drowsiness, possibly resulting in loss of conciousness, coma or cardiac arrhythmias at higher levels. Chronic exposure can lead to lung and kidney irritation, inhibition of bloodclotting, numbness and pain in the fingers, memory loss, and sleep disurbances. (L3)

- Aspiration hazard: No data available.

Likely routes of exposure
- /HUMAN EXPOSURE STUDIES/ Inhalation at levels less than 8000 ppm for 5 minutes may be tolerated without developing symptoms of toxicity. Inhalation of concentrations ranging from 12000 to 20,000 ppm for 5 minutes may produce slight anesthetic effects including dizziness, headache and/or nausea. Deaths due to /CNS depression/ have been reported at undocumented concentrations.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Vinyl chloride is a colorless gas or liquid (below 77 degrees F). It is used in the plastics industry to manufacture polyvinyl chloride, and in organic syntheses. It has been used as refrigerant and spray can propellant. HUMAN STUDIES: Vinyl chloride causes angiosarcoma of the liver, and hepatocellular carcinoma. Past occupational exposure to several hundred ppm of vinyl chloride for periods ranging from one month to 3 years has been associated with development of "vinyl chloride disease". Vinyl chloride disease is characterized by acroosteolysis, a condition characterized by lytic lesions of bones (primarily of fingers), scleroderma of the connective tissue in the fingers with dermal thickening, and a Raynaud-like condition with reversible arteriole constriction causing numbness, pallor and cyanosis of the fingers. The attribution of acroosteolysis to vinyl chloride exposure is based almost entirely on case reports and has been estimated to affect <3% of workers involved in the polymerization of vinyl chloride. In patients with chronic occupational exposure, neurological disturbances include sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances included neurasthenic or depressive syndromes. Sleeplessness and loss of sexual functions were frequently encountered. Pathological EEG alterations were found in a high proportion of patients. A chronic hepatic disorder of porphyrin metabolism was found in 36 workers with vinyl chloride-induced hepatic injury following long-time industrial exposure. Pathologic porphyrinuria, especially secondary coproporphyrinuria with transition to subclinical chronic hepatic porphyria, is a consistent pathobiochemical parameter for the recognition of vinyl chloride hepatic lesions. The major immunological abnormalities reported in vinyl chloride disease patients include hyperimmunoglobulinemia with a polyclonal increase in IgG, cryoglobulinemia, cryofibrinogenemia, and in vivo activation of complement. Vinyl chloride is an occupational carcinogen which caused micronuclei in human cells. There was significant increase in chromosomal abnormalities in cultured peripheral lymphocytes from 57 male workers when compared with controls. Sister chromatid exchange was the more sensitive endpoint for indicating a biological response. ANIMAL STUDIES: Brief (30 minutes) exposures to concentrations of vinyl chloride ranging from 100,000 to 400,000 ppm have been shown to be fatal in rats, guinea pigs and mice. Symptoms of intoxication in rats and mice include muscular incoordination and twitching, CNS depression and respiratory failure. Intense salivation and lacrimation have been noted in rats, guinea pigs and rabbits exposed acutely to high concentrations (375-700 mg/L) of vinyl chloride gas. When placed on skin or in eyes, liquid vinyl chloride may freeze tissue and produce a chemical burn as it evaporates, causing damage to the underlying tissue. Profound CNS depression was reported in guinea-pigs exposed to vinyl chloride at 65,000 mg/cu m for 90 min. Ataxia was observed at this dose level after 5 min of exposure. The anesthetic action of vinyl chloride was also observed in dogs and mice. Investigators reported deep CNS depression in rats and mice exposed to 260,000 mg/cu m for 30 min. The CNS depressant effect was preceded by increased motor activity after 5 min of exposure, twitching of extremities (after 10 min), ataxia (after 15 min) and tremor (after 15 min). Rats exposed to 130,000 mg/cu m for 60 min showed ataxia preceded by hyperactivity but no /CNS depressant/ effect. Forty rabbits were exposed for 4 hours/day on 5 days/week for 12 months to air containing (10,000 ppm) vinyl chloride. Between 9 and 15 months exposure, 12 skin acanthomas and 6 lung adenocarcinomas were seen. No similar tumors occurred in 20 controls after 15 months observation. Rats were exposed to 10,000 ppm vinyl chloride in air for 4 hours/day on 5 days/week for 5 weeks, starting at the age of 13 weeks (120 rats per group) or 1 day (43 and 46 rats). Animals were observed for 135 weeks. One hepatoma was reported in the older rats in newborn rats,

10 angiosarcomas and 15 hepatomas were found. No liver tumors were reported in 249 controls. Vinyl chloride was administered for 7 hr/day on days 6-18 of gestation in mice, rats, and rabbits. It was concluded that although maternal toxicity observed, vinyl chloride alone did not cause significant embryonal or fetal toxicity and was not teratogenic in any of the species at concentrations tested. Vinyl chloride produced a significant increase in the frequency of recessive lethal mutations in male Drosophila melangaster. Mutagenic activity of vinyl chloride was reported in yeast (S. pombe and S. cerevisiae) in the presence of metabolic activation. Vinyl chloride was mutagenic to S. pombe in the "host mediated" assay when mice were treated with an oral dose of 700 mg/kg of vinyl chloride. Using Salmonella tester strains, direct mutagenicity of vinyl chloride was reported at 20% (v/v) in air (200,000 ppm) in the absence of metabolic activation. Mutagenic response was increased by metabolic activation. However, 20% vinyl chloride (v/v in air) was inactive in systems employing S. typhimurium strains TA1536, TA1537 and TA1538. ECOTOXICITY STUDIES: In Daphnia magna exposure, results indicated impacts of vinyl chloride on the regulation of genes related to glutathione-S-transferase (GST), juvenile hormone esterase (JHE), and the vitelline outer layer membrane protein (VMO1).

SECTION 12: Ecological information

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12.1 Toxicity
Not available.

12.2 Persistence and degradability
Not available.

12.3 Bioaccumulative potential
Not available.

12.4 Mobility in soil
Not available.

12.5 Results of PBT and vPvB assessment
Not available.

12.6 Endocrine disrupting properties
Not available.

12.7 Other adverse effects
Not available.

SECTION 13: Disposal considerations

SECTION 13: Disposal considerations

13.1 Waste treatment methods
Dispose of contents/container in accordance with local/regional/national/international regulations. Treat as hazardous waste. Do not discharge to drains.
Contaminated packaging: Dispose of as unused product or according to local regulations.
Waste codes: Not available.

SECTION 14: Transport information

SECTION 14: Transport information

14.1 UN number
Not available.

14.2 UN proper shipping name
Not available.

14.3 Transport hazard class(es)
Not available.

14.4 Packing group
Not available.

14.5 Environmental hazards
Not available.

14.6 Special precautions for user
Not available.

14.7 Maritime transport in bulk according to IMO instruments
Not available.

SECTION 15: Regulatory information

SECTION 15: Regulatory information

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: Vinyl Chloride Solution 2000 µg/mL in methanol
Catalog No.: CS-O-36265
CAS No. (vinyl chloride): 75-01-4
Supplier: Clearsynth Labs Ltd., Mumbai, India
Emergency phone: +91-22-245045900

Revision date: Not available
SDS version: Not available

Disclaimer: The information provided is based on data believed to be accurate; it is provided for guidance for safe handling, use, processing, storage, transportation, disposal and release and is not considered a warranty or quality specification. Not all data may be available for this mixture.

DISCLAIMER

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