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

Catalogue Number	CS-BX-00566
Product Name	Phosphine
CAS No.	7803-51-2
Category	Pesticide Standards
Synonyms	Hydrogen phosphide; Phosphine (PH3); Phosphorus hydride; Phosphorus hydride (PH3); Phosphorus trihydride; TK-Gas; Vaporphos
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: Warning



Hazard Statement(s)

Code	Statement
H220	Not available
H314	Not available
H330	Not available

H400	Not available
H280	Not available
H370	Not available
H318	Causes serious eye damage.

Precautionary Statement(s)

Code	Statement
P203	Not available
P210	Not available
P222	Not available
P260	Not available
P264	Wash hands thoroughly after handling.
P271	Use only outdoors or in a well-ventilated area.
P273	Not available
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P284	Not available
P301+P330+P331	Not available
P302+P361+P354	Not available
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P354+P338	Not available
P316	Not available
P320	Not available
P321	Specific treatment (see … on this label).
P363	Not available
P377	Not available
P381	Not available
P391	Not available
P403	Not available
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.

P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P410+P403	Not available
P270	Not available
P308+P316	Not available
P264+P265	Not available
P317	Not available

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Phosphine
CAS Number : 7803-51-2
Molecular Formula : H3P
Molecular Weight : 34.00
Parent Chemical : -
Synonyms : Hydrogen phosphide; Phosphine (PH3); Phosphorus hydride; Phosphorus hydride (PH3); Phosphorus trihydride; TK-Gas; Vaporphos
Concentration : Not available

SECTION 4: First aid measures

Not available

SECTION 5: Firefighting measures

Not available

SECTION 6: Accidental release measures

Not available

SECTION-7: Handling and storage

Not available

SECTION 8: Exposure controls / personal protection

Not available

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
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

Not available

SECTION 11: Toxicological information

11.1 Information on toxicological effects

- Acute toxicity: IDENTIFICATION AND USE: Phosphine is a colorless gas with a garlic-like odor. Phosphine is a fumigant, used indoors to control a broad spectrum of insects for non-food/non-feed commodities in sealed containers or structures. It is also used as a doping agent for electronic components, in chemical synthesis, and as an intermediate for the preparation of several flame retardants. HUMAN STUDIES: Direct contact with phosphine liquid may cause frostbite. Toxic exposures to phosphine have been documented as a result of grain fumigation, attempted suicide, and ferrosilicon decomposition. Potential symptoms of overexposure are nausea, vomiting, abdominal pain, diarrhea, thirst, chest tightness, dyspnea, muscle pain, chills, stupor or syncope, and pulmonary edema. A productive cough with fluorescent green sputum, acute dyspnea, and pulmonary edema may develop. Death may be preceded by tonic convulsions, which may ensue after apparent recovery. Death may occur after 1/2 to 1 hr of exposure at concentrations of 400 to 600 ppm. Serious effects may be produced by exposure to 5 to 10 ppm for several hours. Main histopathologic findings of fatal phosphine poisoning in the liver are fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Fumigant applicators had significantly increased stable chromosome rearrangements, primarily translocations in G-banded lymphocytes. Less stable aberrations included chromatid deletions and gaps but these were significantly increased only during the application season, and not at later time points. Exposure of human lymphocytes to phosphine (1.4 to 4.5 ug/L) for 20 mins yielded increased chromosome aberrations after 96 hr of lymphocyte culture, indicating that the expression of genotoxicity of phosphine is delayed. ANIMAL STUDIES: In rats, signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea. Rats exposed to phosphine repeatedly at 4 ppm for 4 hours daily on 9 of 12 days exhibited a slightly reduced weight gain, which returned to normal during the 14-day recovery period. Signs of mild respiratory irritation were observed in these animals. One study of animal teratogenicity with exposure as high as 4.9 ppm during days 6 to 15 of gestation in rats showed neither maternal toxicity nor developmental toxicity. In an in vitro cytogenetic assay with Chinese hamster ovary (CHO) cells, phosphine was positive at 2500 and 5000 ppm without metabolic activation. This resulted in a significant, but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with metabolic activation, but not at the highest dose tested (5000 ppm). Increased micronucleus (MN) induction in bone marrow polychromatic erythrocytes (PCE) was seen following subchronic exposure to phosphine in male mice at 5.0 ppm, and female mice at 2.5 and 5.0 ppm. In rats, MN elevation in bone marrow PCE and pulmonary alveolar macrophages was seen at 1.0 and 4.0 ppm, respectively, following subchronic exposure. ECOTOXICITY STUDIES: Three turkeys were exposed to phosphine at a concentration of 211 mg/cu m and 6 hens at 224 mg/cu m in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. Hens exhibited tonic-clonic convulsions and died after an average of 59 min. Their organs were also congested with oxygenated blood. In rice seeds, phosphine has inhibitory effects on seed germination. In addition, phosphine exposure caused oxidative stress in the seeds. Inhalation of phosphine may cause severe pulmonary irritation leading to acute pulmonary oedema, cardiovascular dysfunction, CNS excitation, coma and death. Gastrointestinal disorders, renal damage and leukopenia may also occur. Chronic exposure to phosphine can result in anemia, bronchitis, gastrointestinal effects, and visual, speech and motor problems. (L980, L982)
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: - Phosphine gas produces no known adverse effects on the eyes. - Ocular exposure to phosphine gas has not been known to cause systemic toxicity. - Direct eye contact with liquefied or compressed phosphine gas, which is unlikely, may cause frostbite.
- Respiratory or skin sensitization: No data available.

- Germ cell mutagenicity: IDENTIFICATION AND USE: Phosphine is a colorless gas with a garlic-like odor. Phosphine is a fumigant, used indoors to control a broad spectrum of insects for non-food/non-feed commodities in sealed containers or structures. It is also used as a doping agent for electronic components, in chemical synthesis, and as an intermediate for the preparation of several flame retardants. HUMAN STUDIES: Direct contact with phosphine liquid may cause frostbite. Toxic exposures to phosphine have been documented as a result of grain fumigation, attempted suicide, and ferrosilicon decomposition. Potential symptoms of overexposure are nausea, vomiting, abdominal pain, diarrhea, thirst, chest tightness, dyspnea, muscle pain, chills, stupor or syncope, and pulmonary edema. A productive cough with fluorescent green sputum, acute dyspnea, and pulmonary edema may develop. Death may be preceded by tonic convulsions, which may ensue after apparent recovery. Death may occur after 1/2 to 1 hr of exposure at concentrations of 400 to 600 ppm. Serious effects may be produced by exposure to 5 to 10 ppm for several hours. Main histopathologic findings of fatal phosphine poisoning in the liver are fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Fumigant applicators had significantly increased stable chromosome rearrangements, primarily translocations in G-banded lymphocytes. Less stable aberrations included chromatid deletions and gaps but these were significantly increased only during the application season, and not at later time points. Exposure of human lymphocytes to phosphine (1.4 to 4.5 ug/L) for 20 mins yielded increased chromosome aberrations after 96 hr of lymphocyte culture, indicating that the expression of genotoxicity of phosphine is delayed. ANIMAL STUDIES: In rats, signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea. Rats exposed to phosphine repeatedly at 4 ppm for 4 hours daily on 9 of 12 days exhibited a slightly reduced weight gain, which returned to normal during the 14-day recovery period. Signs of mild respiratory irritation were observed in these animals. One study of animal teratogenicity with exposure as high as 4.9 ppm during days 6 to 15 of gestation in rats showed neither maternal toxicity nor developmental toxicity. In an in vitro cytogenetic assay with Chinese hamster ovary (CHO) cells, phosphine was positive at 2500 and 5000 ppm without metabolic activation. This resulted in a significant, but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with metabolic activation, but not at the highest dose tested (5000 ppm). Increased micronucleus (MN) induction in bone marrow polychromatic erythrocytes (PCE) was seen following subchronic exposure to phosphine in male mice at 5.0 ppm, and female mice at 2.5 and 5.0 ppm. In rats, MN elevation in bone marrow PCE and pulmonary alveolar macrophages was seen at 1.0 and 4.0 ppm, respectively, following subchronic exposure. ECOTOXICITY STUDIES: Three turkeys were exposed to phosphine at a concentration of 211 mg/cu m and 6 hens at 224 mg/cu m in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. Hens exhibited tonic-clonic convulsions and died after an average of 59 min. Their organs were also congested with oxygenated blood. In rice seeds, phosphine has inhibitory effects on seed germination. In addition, phosphine exposure caused oxidative stress in the seeds. CLASSIFICATION: D; not classifiable as to human carcinogenicity. BASIS FOR CLASSIFICATION: Based on inadequate data in animals and no tumor data in humans. While phospine has not been associated with cancer in humans, there is some evidence of chromosomal damage (transient chromatid deletions, gaps and breaks, persistent chromosomal translocations). A relationship between these genetic effects and the development of cancer in humans is sometimes postulated. HUMAN CARCINOGENICITY DATA: None. ANIMAL CARCINOGENICITY DATA: Inadequate.
- Carcinogenicity: CLASSIFICATION: D; not classifiable as to human carcinogenicity. BASIS FOR CLASSIFICATION: Based on inadequate data in animals and no tumor data in humans. While phospine has not been associated with cancer in humans, there is some evidence of chromosomal damage (transient chromatid deletions, gaps and breaks, persistent chromosomal translocations). A relationship between these genetic effects and the development of cancer in humans is sometimes postulated. HUMAN CARCINOGENICITY DATA: None. ANIMAL CARCINOGENICITY DATA: Inadequate.

- Reproductive toxicity: IDENTIFICATION AND USE: Phosphine is a colorless gas with a garlic-like odor. Phosphine is a fumigant, used indoors to control a broad spectrum of insects for non-food/non-feed commodities in sealed containers or structures. It is also used as a doping agent for electronic components, in chemical synthesis, and as an intermediate for the preparation of several flame retardants. HUMAN STUDIES: Direct contact with phosphine liquid may cause frostbite. Toxic exposures to phosphine have been documented as a result of grain fumigation, attempted suicide, and ferrosilicon decomposition. Potential symptoms of overexposure are nausea, vomiting, abdominal pain, diarrhea, thirst, chest tightness, dyspnea, muscle pain, chills, stupor or syncope, and pulmonary edema. A productive cough with fluorescent green sputum, acute dyspnea, and pulmonary edema may develop. Death may be preceded by tonic convulsions, which may ensue after apparent recovery. Death may occur after 1/2 to 1 hr of exposure at concentrations of 400 to 600 ppm. Serious effects may be produced by exposure to 5 to 10 ppm for several hours. Main histopathologic findings of fatal phosphine poisoning in the liver are fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Fumigant applicators had significantly increased stable chromosome rearrangements, primarily translocations in G-banded lymphocytes. Less stable aberrations included chromatid deletions and gaps but these were significantly increased only during the application season, and not at later time points. Exposure of human lymphocytes to phosphine (1.4 to 4.5 ug/L) for 20 mins yielded increased chromosome aberrations after 96 hr of lymphocyte culture, indicating that the expression of genotoxicity of phosphine is delayed. ANIMAL STUDIES: In rats, signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea. Rats exposed to phosphine repeatedly at 4 ppm for 4 hours daily on 9 of 12 days exhibited a slightly reduced weight gain, which returned to normal during the 14-day recovery period. Signs of mild respiratory irritation were observed in these animals. One study of animal teratogenicity with exposure as high as 4.9 ppm during days 6 to 15 of gestation in rats showed neither maternal toxicity nor developmental toxicity. In an in vitro cytogenetic assay with Chinese hamster ovary (CHO) cells, phosphine was positive at 2500 and 5000 ppm without metabolic activation. This resulted in a significant, but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with metabolic activation, but not at the highest dose tested (5000 ppm). Increased micronucleus (MN) induction in bone marrow polychromatic erythrocytes (PCE) was seen following subchronic exposure to phosphine in male mice at 5.0 ppm, and female mice at 2.5 and 5.0 ppm. In rats, MN elevation in bone marrow PCE and pulmonary alveolar macrophages was seen at 1.0 and 4.0 ppm, respectively, following subchronic exposure. ECOTOXICITY STUDIES: Three turkeys were exposed to phosphine at a concentration of 211 mg/cu m and 6 hens at 224 mg/cu m in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. Hens exhibited tonic-clonic convulsions and died after an average of 59 min. Their organs were also congested with oxygenated blood. In rice seeds, phosphine has inhibitory effects on seed germination. In addition, phosphine exposure caused oxidative stress in the seeds.
- STOT-single exposure: No data available.
- STOT-repeated exposure: IDENTIFICATION AND USE: Phosphine is a colorless gas with a garlic-like odor. Phosphine is a fumigant, used indoors to control a broad spectrum of insects for non-food/non-feed commodities in sealed containers or structures. It is also used as a doping agent for electronic components, in chemical synthesis, and as an intermediate for the preparation of several flame retardants. HUMAN STUDIES: Direct contact with phosphine liquid may cause frostbite. Toxic exposures to phosphine have been documented as a result of grain fumigation, attempted suicide, and ferrosilicon decomposition. Potential symptoms of overexposure are nausea, vomiting, abdominal pain, diarrhea, thirst, chest tightness, dyspnea, muscle pain, chills, stupor or syncope, and pulmonary edema. A productive cough with fluorescent green sputum, acute dyspnea, and pulmonary edema may develop. Death may be preceded by tonic convulsions, which may ensue after apparent recovery. Death may occur after 1/2 to 1 hr of exposure at concentrations of 400 to 600 ppm. Serious effects may be produced by exposure to 5 to 10 ppm for several hours. Main histopathologic findings of fatal phosphine poisoning in the liver are fine

cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Fumigant applicators had significantly increased stable chromosome rearrangements, primarily translocations in G-banded lymphocytes. Less stable aberrations included chromatid deletions and gaps but these were significantly increased only during the application season, and not at later time points. Exposure of human lymphocytes to phosphine (1.4 to 4.5 ug/L) for 20 mins yielded increased chromosome aberrations after 96 hr of lymphocyte culture, indicating that the expression of genotoxicity of phosphine is delayed. ANIMAL STUDIES: In rats, signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea. Rats exposed to phosphine repeatedly at 4 ppm for 4 hours daily on 9 of 12 days exhibited a slightly reduced weight gain, which returned to normal during the 14-day recovery period. Signs of mild respiratory irritation were observed in these animals. One study of animal teratogenicity with exposure as high as 4.9 ppm during days 6 to 15 of gestation in rats showed neither maternal toxicity nor developmental toxicity. In an in vitro cytogenetic assay with Chinese hamster ovary (CHO) cells, phosphine was positive at 2500 and 5000 ppm without metabolic activation. This resulted in a significant, but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with metabolic activation, but not at the highest dose tested (5000 ppm). Increased micronucleus (MN) induction in bone marrow polychromatic erythrocytes (PCE) was seen following subchronic exposure to phosphine in male mice at 5.0 ppm, and female mice at 2.5 and 5.0 ppm. In rats, MN elevation in bone marrow PCE and pulmonary alveolar macrophages was seen at 1.0 and 4.0 ppm, respectively, following subchronic exposure. ECOTOXICITY STUDIES: Three turkeys were exposed to phosphine at a concentration of 211 mg/cu m and 6 hens at 224 mg/cu m in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. Hens exhibited tonic-clonic convulsions and died after an average of 59 min. Their organs were also congested with oxygenated blood. In rice seeds, phosphine has inhibitory effects on seed germination. In addition, phosphine exposure caused oxidative stress in the seeds. Inhalation of phosphine may cause severe pulmonary irritation leading to acute pulmonary oedema, cardiovascular dysfunction, CNS excitation, coma and death. Gastrointestinal disorders, renal damage and leukopenia may also occur. Chronic exposure to phosphine can result in anemia, bronchitis, gastrointestinal effects, and visual, speech and motor problems. (L980, L982)
- Aspiration hazard: No data available.

Likely routes of exposure
- IDENTIFICATION AND USE: Phosphine is a colorless gas with a garlic-like odor. Phosphine is a fumigant, used indoors to control a broad spectrum of insects for non-food/non-feed commodities in sealed containers or structures. It is also used as a doping agent for electronic components, in chemical synthesis, and as an intermediate for the preparation of several flame retardants. HUMAN STUDIES: Direct contact with phosphine liquid may cause frostbite. Toxic exposures to phosphine have been documented as a result of grain fumigation, attempted suicide, and ferrosilicon decomposition. Potential symptoms of overexposure are nausea, vomiting, abdominal pain, diarrhea, thirst, chest tightness, dyspnea, muscle pain, chills, stupor or syncope, and pulmonary edema. A productive cough with fluorescent green sputum, acute dyspnea, and pulmonary edema may develop. Death may be preceded by tonic convulsions, which may ensue after apparent recovery. Death may occur after 1/2 to 1 hr of exposure at concentrations of 400 to 600 ppm. Serious effects may be produced by exposure to 5 to 10 ppm for several hours. Main histopathologic findings of fatal phosphine poisoning in the liver are fine cytoplasmic vacuolization of hepatocytes and sinusoidal congestion. Fumigant applicators had significantly increased stable chromosome rearrangements, primarily translocations in G-banded lymphocytes. Less stable aberrations included chromatid deletions and gaps but these were significantly increased only during the application season, and not at later time points. Exposure of human lymphocytes to phosphine (1.4 to 4.5 ug/L) for 20 mins yielded increased chromosome aberrations after 96 hr of lymphocyte culture, indicating that the expression of genotoxicity of phosphine is delayed. ANIMAL STUDIES: In rats, signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea. Rats exposed to phosphine repeatedly at 4 ppm for 4 hours

daily on 9 of 12 days exhibited a slightly reduced weight gain, which returned to normal during the 14-day recovery period. Signs of mild respiratory irritation were observed in these animals. One study of animal teratogenicity with exposure as high as 4.9 ppm during days 6 to 15 of gestation in rats showed neither maternal toxicity nor developmental toxicity. In an in vitro cytogenetic assay with Chinese hamster ovary (CHO) cells, phosphine was positive at 2500 and 5000 ppm without metabolic activation. This resulted in a significant, but not dose-related increase in the frequency of cells with structural chromosome aberrations. Significant clastogenic effects were also noted at 2500 ppm with metabolic activation, but not at the highest dose tested (5000 ppm). Increased micronucleus (MN) induction in bone marrow polychromatic erythrocytes (PCE) was seen following subchronic exposure to phosphine in male mice at 5.0 ppm, and female mice at 2.5 and 5.0 ppm. In rats, MN elevation in bone marrow PCE and pulmonary alveolar macrophages was seen at 1.0 and 4.0 ppm, respectively, following subchronic exposure. ECOTOXICITY STUDIES: Three turkeys were exposed to phosphine at a concentration of 211 mg/cu m and 6 hens at 224 mg/cu m in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. Hens exhibited tonic-clonic convulsions and died after an average of 59 min. Their organs were also congested with oxygenated blood. In rice seeds, phosphine has inhibitory effects on seed germination. In addition, phosphine exposure caused oxidative stress in the seeds.

Symptoms related to the physical, chemical and toxicological characteristics
- Signs of exposure were typical of respiratory irritation and included hyperemia of the ears, salivation, lacrimation, face-pawing, and dyspnea.

SECTION 12: Ecological information

Not available

SECTION 13: Disposal considerations

Not available

SECTION 14: Transport information

Not available

SECTION 15: Regulatory information

Not available

SECTION 16: Other information

Not available

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