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

Catalogue Number	CS-IP-00056
Product Name	Fenbendazole impurity A
CAS No.	10605-21-7
Category	Pesticide Standards
Synonyms	methyl 1H-benzo[d]imidazol-2-ylcarbamate
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:

Serious eye damage/eye irritation (Category 2)

2.2 Label Elements

Signal Word: Warning



Hazard Statement(s)

Code	Statement
H317	May cause an allergic skin reaction.
H340	Not available
H400	Not available
H410	Not available

H360	Not available
H371	Not available
H373	Not available
H319	Causes serious eye irritation.
H341	Not available

Precautionary Statement(s)

Code	Statement
P203	Not available
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P272	Not available
P273	Not available
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P318	Not available
P321	Specific treatment (see … on this label).
P333+P317	Not available
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Not available
P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P260	Not available
P264	Wash hands thoroughly after handling.
P270	Not available
P308+P316	Not available
P319	Get medical help if you feel unwell.
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 : Fenbendazole impurity A
CAS Number : 10605-21-7
Molecular Formula : C9H9N3O2
Molecular Weight : 191.19 g/mol
Parent Chemical : Fenbendazole
Synonyms : methyl 1H-benzo[d]imidazol-2-ylcarbamate
Concentration : Not available

SECTION 4: First aid measures

SECTION 4: First-aid measures

4.1 Description of first aid measures
- General advice: Seek medical attention if symptoms occur or persist. Show this SDS to medical personnel.
- Inhalation: Move person to fresh air. If breathing is difficult, seek medical attention.
- Skin contact: Wash with soap and water. Remove contaminated clothing and wash before reuse.
- 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.

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

4.3 Indication of 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, dry chemical, foam, carbon dioxide).
- Unsuitable extinguishing media: Not available.

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

5.3 Advice for firefighters
- Wear self-contained breathing apparatus (SCBA) and full protective gear.
- Avoid inhalation of combustion products.
- Use water spray to cool unopened containers exposed to heat.

SECTION 6: Accidental release measures

SECTION 6: Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures

- Avoid dust formation and inhalation.
- 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 water, or soil.

6.3 Methods and material for containment and cleaning up
- Collect spilled material using methods that minimize dust generation.
- Place in a suitable, labeled container for disposal.
- Clean contaminated area after material pickup.

6.4 Reference to other sections
- See Section 8 for exposure controls/personal protection 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 and eyes.
- Avoid breathing dust.
- Use with adequate ventilation.

7.2 Conditions for safe storage, including any incompatibilities
- Store in a tightly closed container.
- Store in a cool, dry, well-ventilated place.
- Protect from moisture.
- Incompatible materials: Not available.

7.3 Specific end use(s)
- Laboratory/research standard. Not for food, drug, or household use.

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: Use local exhaust ventilation or general ventilation to minimize dust exposure.
- Personal protective equipment (PPE):
- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Protective gloves. Protective clothing as appropriate.
- Respiratory protection: If dust or aerosols are generated and ventilation is inadequate, use an appropriate particulate respirator.
- Hygiene measures: Wash hands 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	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	2.7
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
- Avoid excessive heat. Avoid dust generation.

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: Carbendazim is a white powder. it is a systemic leaf and soil fungicide, absorbed through the roots and green tissues. HUMAN EXPOSURE AND TOXICITY: Six human chromosomes were investigated in pairs (1 and 8, 11 and 18, and X and 17). Abnormalities were classified as chromosome loss (including centromeric positive micronuclei), chromosome gain, non-disjunction, or polyploidy. ANIMAL STUDIES: Previous studies indicate that carbendazim may interfere with mitosis and thus may disrupt or inhibit microtubule function resulting in apoptosis. Carbendazim even at low dose exhibited toxicity, affected the liver and also caused specific changes in hematological and biochemical parameters in the rat. Male rats (6 per dose level) were gavaged with 200, 3400 and 5000 mg/kg 5 days/wk for 2 wk. Two out of the 6 rats died at the dose level of 3400 mg/kg per day. At all dose levels, gross and microscopic evidence of adverse effects on the testes and reduction or absence of sperm in the epididymides was seen. The testes were small and discolored, with tubular degeneration and evidence of aspermatogenesis. At the dose level of 3400 mg/kg per day, there were also morphological changes in the duodenum (edema and focal necrosis), bone marrow (reduction in the blood forming elements) and liver (decrease in the large globular shaped vacuoles). Groups of 1 yr old beagles (4 males, 4 females) were admin carbendazim in the diet for 3 months at dietary levels of 0, 100, 500 and 2500 mg/kg. Females at the mid dose level showed a trend toward incr cholesterol levels at 1, 2 and 3 months compared with the pre-test and control values. High dose females also had elevated cholesterol levels. Organ to body weight changes were observed in the case of the thymus of low and mid dose males and the prostate of mid dose males. Groups of pregnant rats were administered carbendazim by gavage on days 6-15 of gestation at dose levels up to 80 mg/kg/day. Groups of pregnant rabbits were similarly administered up to 160 mg/kg/day on days 6-18 of gestation. In the rats, dead and resorbed fetuses accounted for 29% of conceptions in controls, 48% at 20 mg/kg, 64% at 40 mg/kg, and 73% at 80 mg/kg. In rabbits, no dead or resorbed fetuses were seen in controls whereas 15-33% were found in carbendazim-treated rabbits. There were no differences among rats or rabbits in mean weight of live fetuses, and there were no malformations. Carbendazim induced chromosome aberrations in spermatids with a high incidence of aneuploidy. Carbendazim induced micronuclei in mouse bone marrow cells. 2,3-diaminophenazine (DAP) and 2-amino-3-hydroxyphenazine (AHP) were detected in mutagenic carbendazim samples. Carbendazim samples containing DAP or AHP at levels as low as 5 or 10 ppm, respectively, were positive in the Salmonella/Ames test with activation when tested at 5000 ug/plate. Purified carbendazim was not mutagenic. ECOTOXICITY STUDIES: Amazonian fish appeared to be slightly less sensitive for carbendazim than temperate fish with LC50 values ranging between 1648 and 4238 ug/L,

and Amazonian invertebrates were found to be significantly more resistant than their temperate counterparts, with LC50 values higher than 16000 ug/L. In plants, carbendazim causes methylation or demethylation of certain genes and changes the expression of these genes. Acute oral LD50 for rats is >15000 mg/kg and >2500 mg/kg for dogs
- Skin corrosion/irritation: Skin redness and skin irritation. Fetuses exposed to high levels may exhibit microphthalmia (small eyes) or anaphthalmia (no eyes).
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: IDENTIFICATION AND USE: Carbendazim is a white powder. it is a systemic leaf and soil fungicide, absorbed through the roots and green tissues. HUMAN EXPOSURE AND TOXICITY: Six human chromosomes were investigated in pairs (1 and 8, 11 and 18, and X and 17). Abnormalities were classified as chromosome loss (including centromeric positive micronuclei), chromosome gain, non-disjunction, or polyploidy. ANIMAL STUDIES: Previous studies indicate that carbendazim may interfere with mitosis and thus may disrupt or inhibit microtubule function resulting in apoptosis. Carbendazim even at low dose exhibited toxicity, affected the liver and also caused specific changes in hematological and biochemical parameters in the rat. Male rats (6 per dose level) were gavaged with 200, 3400 and 5000 mg/kg 5 days/wk for 2 wk. Two out of the 6 rats died at the dose level of 3400 mg/kg per day. At all dose levels, gross and microscopic evidence of adverse effects on the testes and reduction or absence of sperm in the epididymides was seen. The testes were small and discolored, with tubular degeneration and evidence of aspermatogenesis. At the dose level of 3400 mg/kg per day, there were also morphological changes in the duodenum (edema and focal necrosis), bone marrow (reduction in the blood forming elements) and liver (decrease in the large globular shaped vacuoles). Groups of 1 yr old beagles (4 males, 4 females) were admin carbendazim in the diet for 3 months at dietary levels of 0, 100, 500 and 2500 mg/kg. Females at the mid dose level showed a trend toward incr cholesterol levels at 1, 2 and 3 months compared with the pre-test and control values. High dose females also had elevated cholesterol levels. Organ to body weight changes were observed in the case of the thymus of low and mid dose males and the prostate of mid dose males. Groups of pregnant rats were administered carbendazim by gavage on days 6-15 of gestation at dose levels up to 80 mg/kg/day. Groups of pregnant rabbits were similarly administered up to 160 mg/kg/day on days 6-18 of gestation. In the rats, dead and resorbed fetuses accounted for 29% of conceptions in controls, 48% at 20 mg/kg, 64% at 40 mg/kg, and 73% at 80 mg/kg. In rabbits, no dead or resorbed fetuses were seen in controls whereas 15-33% were found in carbendazim-treated rabbits. There were no differences among rats or rabbits in mean weight of live fetuses, and there were no malformations. Carbendazim induced chromosome aberrations in spermatids with a high incidence of aneuploidy. Carbendazim induced micronuclei in mouse bone marrow cells. 2,3-diaminophenazine (DAP) and 2-amino-3-hydroxyphenazine (AHP) were detected in mutagenic carbendazim samples. Carbendazim samples containing DAP or AHP at levels as low as 5 or 10 ppm, respectively, were positive in the Salmonella/Ames test with activation when tested at 5000 ug/plate. Purified carbendazim was not mutagenic. ECOTOXICITY STUDIES: Amazonian fish appeared to be slightly less sensitive for carbendazim than temperate fish with LC50 values ranging between 1648 and 4238 ug/L, and Amazonian invertebrates were found to be significantly more resistant than their temperate counterparts, with LC50 values higher than 16000 ug/L. In plants, carbendazim causes methylation or demethylation of certain genes and changes the expression of these genes. /GENOTOXICITY/ ... Human lymphocytes were exposed in vitro to ... five concentrations of carbendazim representing the threshold concentrations for chromosome non-disjunction and chromosome loss, two concentrations below the lowest threshold and one concentration between the two threshold values. After 48 hr exposure to the aneugen, induction of apoptosis was analyzed by the annexin-V test. The frequencies of chromosome non-disjunction and chromosome loss were estimated in cytokinesis-blocked human lymphocytes in combination with fluorescence in situ hybridization (FISH); this methodology was applied to whole cell cultures as well as to apoptotic and viable cell fractions obtained using magnetic annexin microbead cell sorting. Our results suggest that elimination of aneuploid cells does occur. However, the efficiency of disappearance of micronucleated

cells is higher than for cells presenting chromosome non-disjunction. The correlation found between early apoptotic events and micronucleus formation could account, at least in part, for the specific elimination of aneuploid cells.
- Carcinogenicity: Cancer Classification: Group C Possible Human Carcinogen
- Reproductive toxicity: Carbendazim is a suspected endocrine disruptor. It is also a developmental toxin. Animals exposed to carbendazim in the womb to have serious deformities such as lack of eyes and hydrocephalus (water on the brain). Carbendazim can disrupt the development of sperm and damage testicular development in adult rats. /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ This study was designed to investigate the endocrine-disrupting activity of carbendazim-induced reproductive and developmental toxicity in Sprague-Dawley rats treated orally with the fungicide. Cotreatment of male rats with 675 mg/kg carbendazim and 50 or 100 mg/kg flutamide, an androgen receptor antagonist, once daily for 28 d blocked decrease of testis weight induced by treatment with carbendazim alone. The cotreatment prevented losses of spermatozoa and cell morphology and decrease of sperm concentration induced by carbendazim. Premating treatment of male and female rats with 200 mg/kg carbendazim for 28 d produced androgenic effects including incomplete development of uterine horn, enlargement of uretha, absence of vagina, and induction of seminal vesicles in female offspring, without marked effects in male offspring. Premating treatment with 100mg/kg benomyl, the parent compound of carbendazim, resulted in incomplete development of uterine horn and absence of vagina in female offspring and produced testis and epidydimis atrophy in male offspring. Treatment of male rats with 25, 50, 100, 200, 400, and 800 mg/kg carbendazim for 56 d produced dose-dependent increases of androgen receptor concentrations in testis and epididymis. Additions of 5, 50, and 500 uM carbendazim to testis extract from untreated rats replaced binding of [3H]-5 alpha-dihydrotestosterone to androgen receptor in a concentration-dependent manner. The present study demonstrates that reproductive toxicity induced by carbendazim is blocked by an androgen receptor antagonist in male rats and developmental toxicity of the fungicide shows androgenic properties in female offspring. These results suggest that androgen- and androgen receptor-dependent mechanisms are possibly involved in carbendazim-induced toxicity.
- STOT-single exposure: /AQUATIC SPECIES/ ... In this study, the two main conceptual models for mixture evaluation based on the effect of individual compounds, concentration addition (CA) and independent action (IA) and deviations to synergism/antagonism, "dose ratio" and "dose level" dependency were used. The single and combined effects of cadmium, carbendazim and low dissolved oxygen levels were assayed for life-cycle parameters (survival and feeding) of the water flea Daphnia magna Straus. The results of single exposures revealed an increase of acute and chronic toxicity as concentrations of cadmium and carbendazim increases. At low dissolved oxygen levels both survival and feeding parameters were significantly affected (p</=0.05). In the acute mixture exposure of cadmium and carbendazim a "dose ratio" dependency was observed with a higher toxicity when cadmium was dominant whereas at high concentrations ofcarbendazim a lower effect on survival was observed. At chronic exposures an antagonistic deviation from IA model was observed for this mixture. The IA model showed to be adequate for toxicity prediction on acute exposure combinations with low DO levels where a synergistic behavior was observed. However, at sublethal exposures IA and CA models failed by underestimation. ...
- STOT-repeated exposure: /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ This study was designed to investigate the endocrine-disrupting activity of carbendazim-induced reproductive and developmental toxicity in Sprague-Dawley rats treated orally with the fungicide. Cotreatment of male rats with 675 mg/kg carbendazim and 50 or 100 mg/kg flutamide, an androgen receptor antagonist, once daily for 28 d blocked decrease of testis weight induced by treatment with carbendazim alone. The cotreatment prevented losses of spermatozoa and cell morphology and decrease of sperm concentration induced by carbendazim. Premating treatment of male and female rats with 200 mg/kg carbendazim for 28 d produced androgenic effects including incomplete development of uterine horn, enlargement of uretha, absence of vagina, and induction of seminal vesicles in female offspring, without marked effects in male offspring. Premating treatment with 100mg/kg benomyl, the parent compound of carbendazim, resulted in incomplete development of uterine horn and absence of vagina in female

offspring and produced testis and epididymis atrophy in male offspring. Treatment of male rats with 25, 50, 100, 200, 400, and 800 mg/kg carbendazim for 56 d produced dose-dependent increases of androgen receptor concentrations in testis and epididymis. Additions of 5, 50, and 500 uM carbendazim to testis extract from untreated rats replaced binding of [3H]-5 alpha-dihydrotestosterone to androgen receptor in a concentration-dependent manner. The present study demonstrates that reproductive toxicity induced by carbendazim is blocked by an androgen receptor antagonist in male rats and developmental toxicity of the fungicide shows androgenic properties in female offspring. These results suggest that androgen- and androgen receptor-dependent mechanisms are possibly involved in carbendazim-induced toxicity. /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ In this study, the effects of carbendazim on the thymus in male rats were evaluated. Carbendazim was administered at 0, 150, 300 and 600 mg/kg/day doses by gavage to male rats for 15 weeks. Body weights of rats in all groups were recorded weekly during treatment. At the end of the experiment, the effects of carbendazim on the thymus were investigated histopathologically and morphologically. Also, based on these effects, change in immunolocalization of fibronectin (FN), which is a component of the extracellular matrix, was investigated immunohistochemically. Fibrosis and oedema were observed in the thymus of rats treated with 300 and 600 mg/kg/day doses of carbendazim. Also in this region, an increase in FN density was noted at the end of the immunohistochemical investigation. A decrease was observed in absolute and relative thymus weights of rats treated with carbendazim compared with the control group.
...
- Aspiration hazard: No data available.

Likely routes of exposure
- /HUMAN EXPOSURE STUDIES/ Moderately toxic by skin contact. Mildly toxic by ingestion.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Carbendazim is a white powder. it is a systemic leaf and soil fungicide, absorbed through the roots and green tissues. HUMAN EXPOSURE AND TOXICITY: Six human chromosomes were investigated in pairs (1 and 8, 11 and 18, and X and 17). Abnormalities were classified as chromosome loss (including centromeric positive micronuclei), chromosome gain, non-disjunction, or polyploidy. ANIMAL STUDIES: Previous studies indicate that carbendazim may interfere with mitosis and thus may disrupt or inhibit microtubule function resulting in apoptosis. Carbendazim even at low dose exhibited toxicity, affected the liver and also caused specific changes in hematological and biochemical parameters in the rat. Male rats (6 per dose level) were gavaged with 200, 3400 and 5000 mg/kg 5 days/wk for 2 wk. Two out of the 6 rats died at the dose level of 3400 mg/kg per day. At all dose levels, gross and microscopic evidence of adverse effects on the testes and reduction or absence of sperm in the epididymides was seen. The testes were small and discolored, with tubular degeneration and evidence of aspermatogenesis. At the dose level of 3400 mg/kg per day, there were also morphological changes in the duodenum (edema and focal necrosis), bone marrow (reduction in the blood forming elements) and liver (decrease in the large globular shaped vacuoles). Groups of 1 yr old beagles (4 males, 4 females) were admin carbendazim in the diet for 3 months at dietary levels of 0, 100, 500 and 2500 mg/kg. Females at the mid dose level showed a trend toward incr cholesterol levels at 1, 2 and 3 months compared with the pre-test and control values. High dose females also had elevated cholesterol levels. Organ to body weight changes were observed in the case of the thymus of low and mid dose males and the prostate of mid dose males. Groups of pregnant rats were administered carbendazim by gavage on days 6-15 of gestation at dose levels up to 80 mg/kg/day. Groups of pregnant rabbits were similarly administered up to 160 mg/kg/day on days 6-18 of gestation. In the rats, dead and resorbed fetuses accounted for 29% of conceptions in controls, 48% at 20 mg/kg, 64% at 40 mg/kg, and 73% at 80 mg/kg. In rabbits, no dead or resorbed fetuses were seen in controls whereas 15-33% were found in carbendazim-treated rabbits. There were no differences among rats or rabbits in mean weight of live fetuses, and there were no malformations. Carbendazim induced chromosome aberrations in spermatids with a high incidence of aneuploidy. Carbendazim induced micronuclei in mouse bone marrow cells. 2,3-diaminophenazine (DAP) and 2-amino-3-hydroxyphenazine

(AHP) were detected in mutagenic carbendazim samples. Carbendazim samples containing DAP or AHP at levels as low as 5 or 10 ppm, respectively, were positive in the Salmonella/Ames test with activation when tested at 5000 ug/plate. Purified carbendazim was not mutagenic. ECOTOXICITY STUDIES: Amazonian fish appeared to be slightly less sensitive for carbendazim than temperate fish with LC50 values ranging between 1648 and 4238 ug/L, and Amazonian invertebrates were found to be significantly more resistant than their temperate counterparts, with LC50 values higher than 16000 ug/L. In plants, carbendazim causes methylation or demethylation of certain genes and changes the expression of these genes.

SECTION 12: Ecological information

SECTION 12: Ecological information

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.
- Do not discharge to drains or the environment.
- Recommended disposal method: Not available.

SECTION 14: Transport information

SECTION 14: Transport information

- 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

SECTION 15: Regulatory information

15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture
- Regulatory status: Not available.
- Chemical inventories (e.g., TSCA/DSL/EINECS/ENCS/AICS/IECSC): Not available.

15.2 Chemical safety assessment
- Not available.

SECTION 16: Other information

SECTION 16: Other information

- Product name: Fenbendazole impurity A
- CAS No.: 10605-21-7
- Synonyms: methyl 1H-benzo[d]imidazol-2-ylcarbamate
- Catalog No.: CS-IP-00056
- Supplier: Clearsynth Labs Ltd., Mumbai, India
- Emergency phone: +91-22-245045900

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