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

Catalogue Number	CS-O-13694
Product Name	Digoxin EP Impurity A
CAS No.	71-63-6
Category	Impurity
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:

Not available

2.2 Label Elements

Signal Word: Warning



Hazard Statement(s)

Code	Statement
H301	Not available
H331	Not available
H373	Not available
H300	Not available

Precautionary Statement(s)

Code	Statement
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.
P301+P316	Not available
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P316	Not available
P319	Get medical help if you feel unwell.
P321	Specific treatment (see … on this label).
P330	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

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Digoxin EP Impurity A
CAS Number : 71-63-6
Molecular Formula : .
Molecular Weight : .
Parent Chemical : Digoxin
Synonyms : Not available
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

Property	Value
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: Digitoxin is a cardiac glycoside, which was used is in the treatment of low output congestive heart failure. HUMAN STUDIES: Digitoxin applied in eyedrops or ointment in sufficient concentration to reduce intraocular pressure, tends to cause corneal edema and clouding. One neonatal death has been reported, allegedly due to digitoxin overdosage in utero. The widespread use of cardiac glycosides and the very narrow margin between effective therapeutic and toxic dosages contributed to the high incidence of toxicity and the relatively high associated mortality rate. Overdosage of cardiac glycosides is manifested by a wide variety of signs and symptoms that are difficult to distinguish from effects associated with cardiac disease. The extracardiac manifestations of cardiac glycoside intoxication are similar in both acute and chronic intoxication. However, GI effects and, to a lesser extent, CNS and visual disturbances may be more pronounced following acute overdosage. Acute toxicity may cause hyperkalemia, whereas patients with chronic toxicity may be hypokalemic or normokalemic. Anorexia, nausea, and vomiting are common early signs of toxicity and may precede or follow evidence of cardiotoxicity. Headache, fatigue, malaise, drowsiness, and generalized muscle weakness are common nervous system signs of cardiac glycoside toxicity. Dizziness, vertigo, syncope, apathy, lethargy, excitement, euphoria, insomnia, irritability, agitation, hiccups, restlessness, nervousness, seizures, opisthotonos, stupor, and coma have also occurred. Visual disturbances induced by toxic doses of cardiac glycosides probably result from a direct effect on the retina (cones are affected more than rods). Transient retrobulbar neuritis has been reported to cause visual changes in cardiac glycoside intoxication. Cardiac glycosides have caused almost every kind of cardiac arrhythmia, and various combinations of arrhythmias may occur in the same patient. In addition, arrhythmias associated with cardiac glycoside intoxication may result in worsening of congestive heart failure. Otherwise healthy individuals with acute toxicity frequently present with atrioventricular conduction disturbances and supraventricular arrhythmias, such as sinus bradycardia. Ventricular arrhythmias are uncommon in these individuals; however, when present, they are associated with severe toxicity and high mortality. Pediatric patients with healthy hearts often present with sinus bradycardia and conduction disturbances; ventricular arrhythmias also occur but are less common than in adults. In neonates, premonitory signs of toxicity may include sinus bradycardia, sinoatrial arrest, or prolongation of the PR interval. Paroxysmal and nonparoxysmal atrioventricular junctional rhythms, especially nonparoxysmal atrioventricular junctional tachycardia, atrioventricular dissociation (with or without some degree of atrioventricular block), and paroxysmal atrial tachycardia with variable atrioventricular block, are common in both

adults and children. Cardiac glycoside toxicity may also cause various atrial and sinoatrial nodal arrhythmias and conduction disorders including atrial tachycardia, atrial fibrillation, atria flutter, atrial premature complexes, wandering atrial pacemaker, sinus bradycardia, sinoatrial arrest, sinoatrial exit block, and sinus tachycardia. Hypersensitivity reactions to cardiac glycosides are rare but may occur, usually within 6-10 days after initiating therapy. Skin reactions may be erythematous, scarlatiniform. papular, vesicular, or bullous. Rashes are usually accompanied by eosinophilia; eosinophilia also may occur without skin reactions. Urticaria; fever; pruritus; facial, angioneurotic, or laryngeal edema; alopecia of the scalp; shedding of finger and toe nails; and desquamation have been reported. Rarely, thrombocytopenic purpura has been reported to occur during administration of cardiac glycosides, particularly digitoxin. ANIMAL STUDIES: ECG monitoring of adult and 1 week old rats during severe acute digitoxin toxicity showed lack of cardiotoxicity despite marked neurotoxicity in both age groups. High adrenal concentration noted in all animals. /SIGNS AND SYMPTOMS/ The toxic effects of cardiac glycosides that are excreted relatively rapidly (eg, digoxin) usually dissipate more rapidly than those of glycosides that are excreted slowly (eg, digitoxin). The toxicities of cardiac glycosides are additive and when toxicity is caused by one cardiac glycoside, administration of all others is contraindicated. Most cases of cardiac glycoside toxicity occur following multiple doses and result, at least in part, from the cumulative effects of the drug. ... /Cardiac glycosides/

- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: IDENTIFICATION AND USE: Digitoxin is a cardiac glycoside, which was used is in the treatment of low output congestive heart failure. HUMAN STUDIES: Digitoxin applied in eyedrops or ointment in sufficient concentration to reduce intraocular pressure, tends to cause corneal edema and clouding. One neonatal death has been reported, allegedly due to digitoxin overdosage in utero. The widespread use of cardiac glycosides and the very narrow margin between effective therapeutic and toxic dosages contributed to the high incidence of toxicity and the relatively high associated mortality rate. Overdosage of cardiac glycosides is manifested by a wide variety of signs and symptoms that are difficult to distinguish from effects associated with cardiac disease. The extracardiac manifestations of cardiac glycoside intoxication are similar in both acute and chronic intoxication. However, GI effects and, to a lesser extent, CNS and visual disturbances may be more pronounced following acute overdosage. Acute toxicity may cause hyperkalemia, whereas patients with chronic toxicity may be hypokalemic or normokalemic. Anorexia, nausea, and vomiting are common early signs of toxicity and may precede or follow evidence of cardiotoxicity. Headache, fatigue, malaise, drowsiness, and generalized muscle weakness are common nervous system signs of cardiac glycoside toxicity. Dizziness, vertigo, syncope, apathy, lethargy, excitement, euphoria, insomnia, irritability, agitation, hiccups, restlessness, nervousness, seizures, opisthotonos, stupor, and coma have also occurred. Visual disturbances induced by toxic doses of cardiac glycosides probably result from a direct effect on the retina (cones are affected more than rods). Transient retrobulbar neuritis has been reported to cause visual changes in cardiac glycoside intoxication. Cardiac glycosides have caused almost every kind of cardiac arrhythmia, and various combinations of arrhythmias may occur in the same patient. In addition, arrhythmias associated with cardiac glycoside intoxication may result in worsening of congestive heart failure. Otherwise healthy individuals with acute toxicity frequently present with atrioventricular conduction disturbances and supraventricular arrhythmias, such as sinus bradycardia. Ventricular arrhythmias are uncommon in these individuals; however, when present, they are associated with severe toxicity and high mortality. Pediatric patients with healthy hearts often present with sinus bradycardia and conduction disturbances; ventricular arrhythmias also occur but are less common than in adults. In neonates, premonitory signs of toxicity may include sinus bradycardia, sinoatrial arrest, or prolongation of the PR interval. Paroxysmal and nonparoxysmal atrioventricular junctional rhythms, especially nonparoxysmal atrioventricular junctional tachycardia, atrioventricular dissociation (with or without some degree of atrioventricular block), and paroxysmal atrial tachycardia with variable atrioventricular block, are common in both adults and children. Cardiac glycoside toxicity may also cause various atrial and sinoatrial nodal arrhythmias and conduction disorders including atrial tachycardia, atrial fibrillation, atria flutter, atrial premature complexes, wandering atrial pacemaker, sinus bradycardia, sinoatrial arrest, sinoatrial exit block, and sinus tachycardia.

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- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: No data available.
- Carcinogenicity: No data available.
- Reproductive toxicity: No data available.
- STOT-single exposure: No data available.
- STOT-repeated exposure: IDENTIFICATION AND USE: Digitoxin is a cardiac glycoside, which was used is in the treatment of low output congestive heart failure. HUMAN STUDIES: Digitoxin applied in eyedrops or ointment in sufficient concentration to reduce intraocular pressure, tends to cause corneal edema and clouding. One neonatal death has been reported, allegedly due to digitoxin overdosage in utero. The widespread use of cardiac glycosides and the very narrow margin between effective therapeutic and toxic dosages contributed to the high incidence of toxicity and the relatively high associated mortality rate. Overdosage of cardiac glycosides is manifested by a wide variety of signs and symptoms that are difficult to distinguish from effects associated with cardiac disease. The extracardiac manifestations of cardiac glycoside intoxication are similar in both acute and chronic intoxication. However, GI effects and, to a lesser extent, CNS and visual disturbances may be more pronounced following acute overdosage. Acute toxicity may cause hyperkalemia, whereas patients with chronic toxicity may be hypokalemic or normokalemic. Anorexia, nausea, and vomiting are common early signs of toxicity and may precede or follow evidence of cardiotoxicity. Headache, fatigue, malaise, drowsiness, and generalized muscle weakness are common nervous system signs of cardiac glycoside toxicity. Dizziness, vertigo, syncope, apathy, lethargy, excitement, euphoria, insomnia, irritability, agitation, hiccups, restlessness, nervousness, seizures, opisthotonos, stupor, and coma have also occurred. Visual disturbances induced by toxic doses of cardiac glycosides probably result from a direct effect on the retina (cones are affected more than rods). Transient retrobulbar neuritis has been reported to cause visual changes in cardiac glycoside intoxication. Cardiac glycosides have caused almost every kind of cardiac arrhythmia, and various combinations of arrhythmias may occur in the same patient. In addition, arrhythmias associated with cardiac glycoside intoxication may result in worsening of congestive heart failure. Otherwise healthy individuals with acute toxicity frequently present with atrioventricular conduction disturbances and supraventricular arrhythmias, such as sinus bradycardia. Ventricular arrhythmias are uncommon in these individuals; however, when present, they are associated with severe toxicity and high mortality. Pediatric patients with healthy hearts often present with sinus bradycardia and conduction disturbances; ventricular arrhythmias also occur but are less common than in adults. In neonates, premonitory signs of toxicity may include sinus bradycardia, sinoatrial arrest, or prolongation of the PR interval. Paroxysmal and nonparoxysmal atrioventricular junctional rhythms, especially nonparoxysmal atrioventricular junctional tachycardia, atrioventricular dissociation (with or without some degree of atrioventricular block), and paroxysmal atrial tachycardia with variable atrioventricular block, are common in both adults and children. Cardiac glycoside toxicity may also cause various atrial and sinoatrial nodal arrhythmias and conduction disorders including atrial tachycardia, atrial fibrillation, atria flutter, atrial premature complexes, wandering atrial pacemaker, sinus bradycardia, sinoatrial arrest, sinoatrial exit block, and sinus tachycardia. Hypersensitivity reactions to cardiac glycosides are rare but may occur, usually within 6-10 days after initiating therapy. Skin reactions may be erythematous, scarlatiniform. papular, vesicular, or bullous. Rashes are usually accompanied by eosinophilia; eosinophilia also may occur without skin reactions. Urticaria; fever; pruritus; facial,

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- Aspiration hazard: No data available.

Likely routes of exposure
- No data available.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Digitoxin is a cardiac glycoside, which was used is in the treatment of low output congestive heart failure. HUMAN STUDIES: Digitoxin applied in eyedrops or ointment in sufficient concentration to reduce intraocular pressure, tends to cause corneal edema and clouding. One neonatal death has been reported, allegedly due to digitoxin overdosage in utero. The widespread use of cardiac glycosides and the very narrow margin between effective therapeutic and toxic dosages contributed to the high incidence of toxicity and the relatively high associated mortality rate. Overdosage of cardiac glycosides is manifested by a wide variety of signs and symptoms that are difficult to distinguish from effects associated with cardiac disease. The extracardiac manifestations of cardiac glycoside intoxication are similar in both acute and chronic intoxication. However, GI effects and, to a lesser extent, CNS and visual disturbances may be more pronounced following acute overdosage. Acute toxicity may cause hyperkalemia, whereas patients with chronic toxicity may be hypokalemic or normokalemic. Anorexia, nausea, and vomiting are common early signs of toxicity and may precede or follow evidence of cardiotoxicity. Headache, fatigue, malaise, drowsiness, and generalized muscle weakness are common nervous system signs of cardiac glycoside toxicity. Dizziness, vertigo, syncope, apathy, lethargy, excitement, euphoria, insomnia, irritability, agitation, hiccups, restlessness, nervousness, seizures, opisthotonos, stupor, and coma have also occurred. Visual disturbances induced by toxic doses of cardiac glycosides probably result from a direct effect on the retina (cones are affected more than rods). Transient retrobulbar neuritis has been reported to cause visual changes in cardiac glycoside intoxication. Cardiac glycosides have caused almost every kind of cardiac arrhythmia, and various combinations of arrhythmias may occur in the same patient. In addition, arrhythmias associated with cardiac glycoside intoxication may result in worsening of congestive heart failure. Otherwise healthy individuals with acute toxicity frequently present with atrioventricular conduction disturbances and supraventricular arrhythmias, such as sinus bradycardia. Ventricular arrhythmias are uncommon in these individuals; however, when present, they are associated with severe toxicity and high mortality. Pediatric patients with healthy hearts often present with sinus bradycardia and conduction disturbances; ventricular arrhythmias also occur but are less common than in adults. In neonates, premonitory signs of toxicity may include sinus bradycardia, sinoatrial arrest, or prolongation of the PR interval. Paroxysmal and nonparoxysmal atrioventricular junctional rhythms, especially nonparoxysmal atrioventricular junctional tachycardia, atrioventricular dissociation (with or without some degree of atrioventricular block), and paroxysmal atrial tachycardia with variable atrioventricular block, are common in both adults and children. Cardiac glycoside toxicity may also cause various atrial and sinoatrial nodal arrhythmias and conduction disorders including atrial tachycardia, atrial fibrillation, atria flutter, atrial premature complexes,

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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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