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

Catalogue Number	CS-O-47860
Product Name	Indinavir Monohydrate
CAS No.	180683-37-8
Category	API
Synonyms	(S)-1-((2S,4R)-4-benzyl-2-hydroxy-5-(((1S,2R)-2-hydroxy-2,3-dihydro-1H-inden-1-yl)amino)-5-oxopentyl)-N-(tert-butyl)-4-(pyridin-3-ylmethyl)piperazine-2-carboxamide hydrate
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
H315	Causes skin irritation.

Precautionary Statement(s)

Code	Statement
P264	Wash hands thoroughly after handling.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P321	Specific treatment (see … on this label).
P332+P317	If skin irritation occurs: Get medical help.
P362+P364	Take off contaminated clothing and wash it before reuse.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Indinavir Monohydrate
CAS Number : 180683-37-8
Molecular Formula : C36H49N5O5
Molecular Weight : 631.80
Parent Chemical : Indinavir
Synonyms : (S)-1-((2S,4R)-4-benzyl-2-hydroxy-5-(((1S,2R)-2-hydroxy-2,3-dihydro-1H-inden-1-yl)amino)-5-oxopentyl)-N-(tert-butyl)-4-(pyridin-3-ylmethyl)piperazine-2-carboxamide hydrate
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: A 2001 review of Indinavir overdoses (n=79) took into account all reports of indinavir overdose. It defined an acute overdose as a single dose of 2400 mg and a chronic overdose as recurring doses less than or equal to 2400 mg. The study found that 66% of patients (52/79) experienced an adverse event. It is important to note that the most common reactions for chronic and acute overdoses were gastrointestinal (nausea, vomiting, abdominal pain) and renal-related (nephrolithiasis/flank pain/renal colic pain). Common treatment methods for symptomatic Indinavir overdose include hydration (oral or intravenous), diuresis, and activated charcoal to decrease absorption. 87% of patients recovered based on follow-up information provided (n=45). Additionally, an overdose report suggested that 50 mg of activated charcoal was given orally in combination with IV fluids, and the patient was asymptomatic within 4 hours of treatment. Some degree of serum aminotransferase elevations occur in a high proportion of patients taking indinavir containing antiretroviral regimens. Moderate-to severe elevations in serum aminotransferase levels (>5 times the upper limit of normal) are found in 3% to 10% of patients, although rates may be higher in patients with HIV-HCV coinfection. These elevations are usually asymptomatic and self-limited and can resolve even with continuation of the medication. Indinavir therapy also causes increases in unconjugated (indirect) and total serum bilirubin that can manifest as jaundice in up to 10% of patients. These elevations are due to the inhibition of UDP glucuronyl transferase, the hepatic enzyme responsible for conjugation of bilirubin that is deficient in Gilbert syndrome. The hyperbilirubinemia is usually mild, the increases averaging 0.3-0.5 mg/dL, but can be more marked in patients with Gilbert syndrome with increases of 1.5 mg/dL or more and clinical jaundice. The jaundice, however, is not indicative of hepatic injury. Clinically apparent acute liver injury due to indinavir is rare. The few cases that have been reported have arisen after 1 to 8 weeks of starting indinavir, and the pattern of serum enzyme elevations has varied from hepatocellular to cholestatic. Signs of hypersensitivity (fever, rash, eosinophilia) are rare as is autoantibody formation. The acute liver injury due to indinavir is usually self-limited, but it can be severe, and isolated cases of acute liver failure have been reported. In addition, initiation of indinavir based highly active antiretroviral therapy can lead to exacerbation of an underlying chronic hepatitis B or C in coinfected individuals, typically arising 2 to 12 months after starting therapy and associated with a hepatocellular pattern of serum enzyme elevations and increases in serum levels of hepatitis B virus (HBV) DNA or hepatitis C virus (HCV) RNA. Indinavir therapy has not been clearly linked to lactic acidosis and acute fatty liver that is reported in association with several nucleoside analogue reverse transcriptase inhibitors. Likelihood score: C (frequent cause of serum bilirubin elevations and probable cause of rare instances of clinically apparent liver injury).
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- 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: A 2001 review of Indinavir overdoses (n=79) took into account all reports of indinavir overdose. It defined an acute overdose as a single dose of 2400 mg and a chronic overdose as recurring doses less than or equal to 2400 mg. The study found that 66% of patients (52/79) experienced an adverse event. It is important to note that the most common reactions for chronic and acute overdoses were gastrointestinal (nausea, vomiting, abdominal pain) and renal-related (nephrolithiasis/flank pain/renal colic pain). Common treatment methods for symptomatic Indinavir overdose include hydration (oral or intravenous), diuresis, and activated charcoal to decrease absorption. 87% of patients recovered based on follow-up information provided (n=45). Additionally, an

overdose report suggested that 50 mg of activated charcoal was given orally in combination with IV fluids, and the patient was asymptomatic within 4 hours of treatment. Some degree of serum aminotransferase elevations occur in a high proportion of patients taking indinavir containing antiretroviral regimens. Moderate-to severe elevations in serum aminotransferase levels (>5 times the upper limit of normal) are found in 3% to 10% of patients, although rates may be higher in patients with HIV-HCV coinfection. These elevations are usually asymptomatic and self-limited and can resolve even with continuation of the medication. Indinavir therapy also causes increases in unconjugated (indirect) and total serum bilirubin that can manifest as jaundice in up to 10% of patients. These elevations are due to the inhibition of UDP glucuronyl transferase, the hepatic enzyme responsible for conjugation of bilirubin that is deficient in Gilbert syndrome. The hyperbilirubinemia is usually mild, the increases averaging 0.3-0.5 mg/dL, but can be more marked in patients with Gilbert syndrome with increases of 1.5 mg/dL or more and clinical jaundice. The jaundice, however, is not indicative of hepatic injury. Clinically apparent acute liver injury due to indinavir is rare. The few cases that have been reported have arisen after 1 to 8 weeks of starting indinavir, and the pattern of serum enzyme elevations has varied from hepatocellular to cholestatic. Signs of hypersensitivity (fever, rash, eosinophilia) are rare as is autoantibody formation. The acute liver injury due to indinavir is usually self-limited, but it can be severe, and isolated cases of acute liver failure have been reported. In addition, initiation of indinavir based highly active antiretroviral therapy can lead to exacerbation of an underlying chronic hepatitis B or C in coinfected individuals, typically arising 2 to 12 months after starting therapy and associated with a hepatocellular pattern of serum enzyme elevations and increases in serum levels of hepatitis B virus (HBV) DNA or hepatitis C virus (HCV) RNA. Indinavir therapy has not been clearly linked to lactic acidosis and acute fatty liver that is reported in association with several nucleoside analogue reverse transcriptase inhibitors. Likelihood score: C (frequent cause of serum bilirubin elevations and probable cause of rare instances of clinically apparent liver injury).
- Aspiration hazard: No data available.

Likely routes of exposure
- No data available.

Symptoms related to the physical, chemical and toxicological characteristics
- A 2001 review of Indinavir overdoses (n=79) took into account all reports of indinavir overdose. It defined an acute overdose as a single dose of 2400 mg and a chronic overdose as recurring doses less than or equal to 2400 mg. The study found that 66% of patients (52/79) experienced an adverse event. It is important to note that the most common reactions for chronic and acute overdoses were gastrointestinal (nausea, vomiting, abdominal pain) and renal-related (nephrolithiasis/flank pain/renal colic pain). Common treatment methods for symptomatic Indinavir overdose include hydration (oral or intravenous), diuresis, and activated charcoal to decrease absorption. 87% of patients recovered based on follow-up information provided (n=45). Additionally, an overdose report suggested that 50 mg of activated charcoal was given orally in combination with IV fluids, and the patient was asymptomatic within 4 hours of treatment.

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