

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

<b>Catalogue Number</b>	CS-O-15097
<b>Product Name</b>	Taurocholic Acid
<b>CAS No.</b>	81-24-3
<b>Category</b>	API
<b>Synonyms</b>	Not available
<b>Brand</b>	Clearsynth Labs Ltd.
<b>Identified uses</b>	Laboratory Chemicals
<b>Uses advised against</b>	Not available
<b>Company</b>	Clearsynth Labs Ltd. Mumbai, India
<b>Emergency Phone #</b>	+91-22-245045900
<b>REACH No.</b>	Not available

### SECTION 2: Hazards identification

**Disclaimer:** This is sample MSDS. Please email [sales@clearsynth.com](mailto:sales@clearsynth.com) for more details.

#### 2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Acute toxicity (Category 4)

#### 2.2 Label Elements

**Signal Word:** Warning



#### Hazard Statement(s)

Code	Statement
H302	Harmful if swallowed.

#### Precautionary Statement(s)

Code	Statement
------	-----------

P264	Wash hands thoroughly after handling.
P270	Not available
P301+P317	Not available
P330	Not available
P501	Dispose of contents/container in accordance with local/regional/national/international regulation

### SECTION 3: Composition / information on ingredients

#### 3.1 Substance

Component : Taurocholic Acid

CAS Number : 81-24-3

Molecular Formula : Not available

Molecular Weight : Not available

Parent Chemical : Not available

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	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

Not available

## SECTION 11: Toxicological information

11.1 Information on toxicological effects

- Acute toxicity: /LABORATORY ANIMALS: Acute Exposure/ Membrane-associated cytotoxicity induced by hydrophobic bile salts is a major contributing factor leading to liver diseases. Administration of ursodeoxycholate reduces serum liver enzymes in chronic liver diseases but the nature of this effect is still unclear. Using alcohol metabolising enzymes as cellular markers, the hepatotoxic properties of hydrophobic bile salts and the putative hepatoprotective effect of ursodeoxycholate was examined. Two animal models of biliary retention, bile duct obstruction and choledochocaval fistula was used to investigate the effect of taurocholate on the hepatic alcohol metabolizing enzymes: cytosolic alcohol dehydrogenase, microsomal ethanol oxidizing system, catalase and aldehyde dehydrogenase before and after the infusion of taurocholic acid or tauroursodeoxycholic acid for two days period. Bile duct obstruction was found to be similar to or slightly exceeds choledochocaval fistula in the degree of retention. Following the taurocholic acid infusion, the serum alcohol dehydrogenase activity as well as microsomal ethanol oxidizing system and aldehyde dehydrogenase were greatly increased but the level of cytosolic alcohol dehydrogenase and catalase activities was found to be lower in either or both models in comparison with the control animals. However, the tauroursodeoxycholic acid infusion did not induce any significant changes in the levels of all the alcohol metabolizing enzyme activities in either or both models. These findings suggest that hydrophobic taurocholic acid (7 $\alpha$ ) affects the plasmalemma to allow leakage of cytosolic alcohol dehydrogenase into the blood circulation, stimulates the biosynthesis of microsomal ethanol oxidizing system and aldehyde dehydrogenase, and suppresses the biosynthesis of alcohol dehydrogenase and catalase. But in contrast, the hydrophilic tauroursodeoxycholic acid (7 $\beta$ ) provided hepatoprotective effect. /LABORATORY ANIMALS: Acute Exposure/ It is claimed that apoA-I expression is repressed in mice by cholic acid (CA) and its taurine conjugate, taurocholic acid (TCA) via farnesoid X receptor (FXR) activation. We measured apoA-I expression in mice, hamsters, and rats treated with highly potent and selective synthetic FXR agonists or with TCA. All of the synthetic agonists bound to FXR with high affinity in a scintillation proximity assay. However, TCA did not compete with the radio ligand up to the highest concentration used (100  $\mu$ M). The C-site regulatory region of apoA-I, through which FXR has been reported to regulate its expression, is completely conserved across the species investigated. In both male and female human apoA-I-transgenic mice, we reproduced the previously reported strong inhibition of human apoA-I expression upon treatment with the typical supraphysiological dose of TCA used in such studies. However, in contrast to some previous reports, TCA did not repress murine apoA-I expression in the same mice. Also, more-potent and -selective FXR agonists did not affect human or murine apoA-I expression in this model. In LDL receptor-deficient mice and Golden Syrian hamsters, selective FXR agonists did not affect apoA-I expression, whereas in Wistar rats, some even increased apoA-I expression. In conclusion, selective FXR agonists do not repress apoA-I expression in rodents. Repression of human apoA-I expression by TCA in transgenic mice is probably mediated through FXR-independent mechanisms.

- 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: /LABORATORY ANIMALS: Acute Exposure/ Membrane-associated cytotoxicity induced by hydrophobic bile salts is a major contributing factor leading to liver diseases. Administration of ursodeoxycholate reduces serum liver enzymes in chronic liver diseases but the nature of this effect is still unclear. Using alcohol metabolising enzymes as cellular markers, the hepatotoxic properties of hydrophobic bile salts and the putative hepatoprotective effect of ursodeoxycholate was examined. Two animal models of biliary retention, bile duct obstruction and choledochocaval fistula was used to investigate the effect of taurocholate on the hepatic alcohol metabolizing enzymes: cytosolic alcohol dehydrogenase, microsomal ethanol oxidizing system, catalase and

aldehyde dehydrogenase before and after the infusion of taurocholic acid or tauroursodeoxycholic acid for two days period. Bile duct obstruction was found to be similar to or slightly exceeds choledochocaval fistula in the degree of retention. Following the taurocholic acid infusion, the serum alcohol dehydrogenase activity as well as microsomal ethanol oxidizing system and aldehyde dehydrogenase were greatly increased but the level of cytosolic alcohol dehydrogenase and catalase activities was found to be lower in either or both models in comparison with the control animals. However, the tauroursodeoxycholic acid infusion did not induce any significant changes in the levels of all the alcohol metabolizing enzyme activities in either or both models. These findings suggest that hydrophobic taurocholic acid (7alpha) affects the plasmalemma to allow leakage of cytosolic alcohol dehydrogenase into the blood circulation, stimulates the biosynthesis of microsomal ethanol oxidizing system and aldehyde dehydrogenase, and suppresses the biosynthesis of alcohol dehydrogenase and catalase. But in contrast, the hydrophilic tauroursodeoxycholic acid (7beta) provided hepatoprotective effect.

- Aspiration hazard: No data available.

Likely routes of exposure

- No data available.

Symptoms related to the physical, chemical and toxicological characteristics

- Not available.

### 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

### DISCLAIMER

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