Taurocholic acid

Cat. No.: HY-B1788

CAS No.: 81-24-3

Molecular Formula: $C_{26}H_{45}NO_7S$

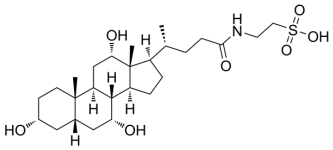
Molecular Weight: 515.7

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (193.91 mM; Need ultrasonic)

H_2O : 100 mg/mL (193.91 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9391 mL	9.6956 mL	19.3911 mL
5 mM	0.3878 mL	1.9391 mL	3.8782 mL
10 mM	0.1939 mL	0.9696 mL	1.9391 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.85 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.85 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.85 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Taurocholic acid (N-Choloyltaurine) has marked bioactive effects such as an inhibitory potential against hepatic artery ligation induced biliary damage by upregulation of VEGF-A expression. Taurocholic acid has immunoregulation effect[1].

IC_{50} & Target	Microbial Metabolite	Human Endogenous Metabolite

In Vitro

Taurocholic acid (100 μM, 24 h) decreases the proportion of CD3+CD8+ T and NK cells in isolated PBMCs from HBeAg-positive

CHB patients[2].
Taurocholic acid (100 μM, 24 h) decreases IFN-α stimulated cytokine and cytotoxic granule levels (IFN-γ, TNF-α, granzyme B) in CD3+CD8+ T and NK cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Taurocholic acid (oral gavage, 100 mg/kg, 2 weeks) promotes HBV replication by reducing the percentage of NK and CD3+CD8+ T cells in C57BL/6 mice with tail vein injection with rAAV8-1.3HBV[2].
Taurocholic acid (1% in diet, 1 week) prevents hepatic artery ligation (HAL)-induced cholangiocyte damage in rats by upregulation of VEGF-A expression[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice[2]
Dosage:	100-mg/kg
Administration:	oral gavage, for 2 weeks after tail vein injection with rAAV8-1.3HBV for 6 weeks
Result:	Reduced the percentage of NK and CD3+CD8+ T cells. Increases serum HBsAg, HBeAg, and HBV DNA levels.

CUSTOMER VALIDATION

- Science. 2024 Mar 22;383(6689):eadj4591.
- Research (Wash D C). 2022 Nov 2;2022:9784081.
- Antiviral Res. 2019 Jun 27;169:104544.
- Food Funct. 2024 Apr 11.
- Biomolecules. 2022, 12(8), 1063.

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REFERENCES

[1]. Xun Z,et al. Taurocholic acid inhibits the response to interferon-α therapy in patients with HBeAg-positive chronic hepatitis B by impairing CD8+ T and NK cell function. Cell Mol Immunol. 2021 Feb;18(2):461-471.

[2]. Glaser S, et al. Taurocholic acid prevents biliary damage induced by hepatic artery ligation in cholestatic rats. Dig Liver Dis. 2010 Oct;42(10):709-17.

[3]. Mooranian A, et al. The effect of a tertiary bile acid, taurocholic acid, on the morphology and physical characteristics of microencapsulated probucol: potential applications in diabetes: a characterization study. Drug Deliv Transl Res. 2015 Oct;5(5):511-22.

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