Lithocholic acid

Cat. No.: HY-B0172

CAS No.: 434-13-9

Molecular Formula: $C_{24}H_{40}O_3$

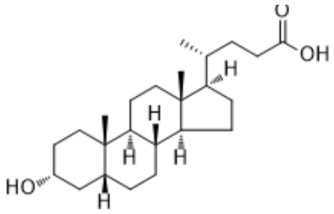
Molecular Weight: 376.57

Target: Autophagy; Endogenous Metabolite; Apoptosis; FXR

Pathway: Autophagy; Metabolic Enzyme/Protease; Apoptosis

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 : 250 mg/mL (663.89 mM; Need ultrasonic)

Ethanol : 10 mg/mL (26.56 mM; ultrasonic and warming and heat to 60°C)

H_2O : 0.99 mg/mL (2.63 mM; ultrasonic and warming and adjust pH to 11 with NaOH and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6555 mL	13.2777 mL	26.5555 mL
5 mM	0.5311 mL	2.6555 mL	5.3111 mL
10 mM	0.2656 mL	1.3278 mL	2.6555 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.52 mM); Clear solution
3. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (2.66 mM); Clear solution
4. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (2.66 mM); Clear solution
5. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 1 mg/mL (2.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Lithocholic acid is a toxic secondary bile acid that can promote intrahepatic cholestasis and promote tumorigenesis.

	Lithocholic acid is also a FXR antagonist and a PXR/SXR agonist[1][2][3][4][5].
IC_{50} & Target	Microbial Metabolite / Human Endogenous Metabolite
In Vitro	Lithocholic acid inhibits CDCA- and GW4064-induced FXR activation with an $IC_{50}$of 0.7 and 1.4 μM, respectively[5]. Lithocholic acid (10-30 μM, 24 h) inhibits the 100 nM GW4064 induced BSEP expression in HepG2 cells[5]. Lithocholic acid (0-500 μM) dose-dependently inhibits the proliferation of neuroblastoma cells (BE(2)-m17, SK-n-SH, SK-n-MCIXC and Lan-1)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Lithocholic acid (0.6% in supplement diet, 7 days) increases TGFB1, TGFBR1, and TGFBR2 mRNA levels in the liver of male mice (C57BL/6), and activates SMAD3, and induces biliary injury[4]. Lithocholic acid (125 mg/kg, i.p., twice a day for four days) induces liver damage, and increased AST, ALT and ALP level in male C57BL/6 mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male mice (C57BL/6)[4].
Dosage:	0.6% LCA-supplement diet, with the AIN93G diet as a control
Administration:	in diet, for 6 days
Result:	Induced liver injury. Activated TGFβ-SMAD3 signaling. Increased serum ALP activities.

Animal Model:	Male mice (C57BL/6)[2].
Dosage:	125 mg/kg, dissolved in corn oil
Administration:	i.p., twice a day for four days
Result:	Induced liver injury, generated necrosis and neutrophilic-granulocytic infiltrate (H&E staining). Increased AST, ALT and ALP level.

CUSTOMER VALIDATION

- Cell Res. 2019 Mar;29(3):193-205.
- Cell Host Microbe. 2024 Jan 11:S1931-3128(23)00510-3.
- Pharmacol Res. 2023 Aug 30;106902.
- Cell Prolif. 2023 Apr 26.
- J Transl Med. 2023 Aug 30;21(1):581.

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REFERENCES

[1]. Yu J, et al. Lithocholic acid decreases expression of bile salt export pump through farnesoid X receptor antagonist activity. J Biol Chem. 2002 Aug 30;277(35):31441-7.

[2]. Jenkins, D.J., et al., Effect on blood lipids of very high intakes of fiber in diets low in saturated fat and cholesterol. N Engl J Med, 1993. 329(1): p. 21-6.

[3]. Goldberg, A.A., et al., Lithocholic bile acid selectively kills neuroblastoma cells, while sparing normal neuronal cells. Oncotarget, 2011. 2(10): p. 761-82.

[4]. Matsubara, T., et al., TGF-beta-SMAD3 signaling mediates hepatic bile acid and phospholipid metabolism following lithocholic acid-induced liver injury. J Lipid Res, 2012. 53(12): p. 2698-707.

[5]. Yang R, et al. Metabolomic analysis of cholestatic liver damage in mice. Food Chem Toxicol. 2018 Jul 14;120:253-260.

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