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SZABO-SCANDIC Handels GmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

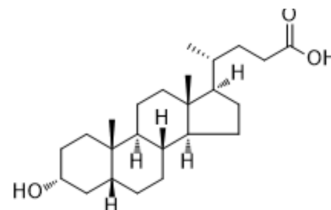
mail@szabo-scandic.com

www.szabo-scandic.com

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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)
 H₂O : 0.99 mg/mL (2.63 mM; ultrasonic and warming and adjust pH to 11 with NaOH and heat to 60°C)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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

- 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
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.08 mg/mL (5.52 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 1 mg/mL (2.66 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1 mg/mL (2.66 mM); Clear solution
- 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 IC₅₀ 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. <table><tr><td>Animal Model:</td><td>Male mice (C57BL/6)^[4].</td></tr><tr><td>Dosage:</td><td>0.6% LCA-supplement diet, with the AIN93G diet as a control</td></tr><tr><td>Administration:</td><td>in diet, for 6 days</td></tr><tr><td>Result:</td><td>Induced liver injury. Activated TGFβ-SMAD3 signaling. Increased serum ALP activities.</td></tr></table> <table><tr><td>Animal Model:</td><td>Male mice (C57BL/6)^[2].</td></tr><tr><td>Dosage:</td><td>125 mg/kg, dissolved in corn oil</td></tr><tr><td>Administration:</td><td>i.p., twice a day for four days</td></tr><tr><td>Result:</td><td>Induced liver injury, generated necrosis and neutrophilic-granulocytic infiltrate (H&E staining). Increased AST, ALT and ALP level.</td></tr></table>		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.
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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.

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- [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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Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA