Taurodeoxycholic acid

Cat. No.: HY-B1899

CAS No.: 516-50-7

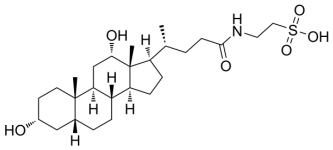
Molecular Formula: $C_{26}H_{45}NO_6S$

Molecular Weight: 499.7

Target: Endogenous Metabolite; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: -20°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (200.12 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0012 mL	10.0060 mL	20.0120 mL
	5 mM	0.4002 mL	2.0012 mL	4.0024 mL
	10 mM	0.2001 mL	1.0006 mL	2.0012 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 (5.00 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.00 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.00 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Taurodeoxycholic acid, a bile acid, stabilizes the mitochondrial membrane, decreases free radical formation. Taurodeoxycholic acid inhibits apoptosis by blocking a calcium-mediated apoptotic pathway as well as caspase-12 activation. Taurodeoxycholic acid exhibits neuroprotective effect in 3-nitropropionic acid induced mouse model or genetic mouse model of Huntington's disease (HD)[1][2][3][4].

IC_{50} & Target

Microbial Metabolite

In Vitro

Taurodeoxycholic acid (50 μM, 100 μM; 4 h) increases oligonucleosomal DNA cleavage and apoptotic nuclei in primary human hepatocytes[1].

	Taurodeoxycholic acid (400 μM; 18-24 h) increases DNA fragmentation and PARP cleavage in human liver-derived cell line Huh7 cells, thus induces apoptosis[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Taurodeoxycholic acid (50 mg/kg; i.p.; once daliy for 34 d) prevents neuropathology and associated behavioral deficits in the 3-nitropropionic acid rat model of Huntington's disease (HD)[3]. Taurodeoxycholic acid (500 mg/kg; s.c.; once every 3 d for 7 weeks) leads to a significant reduction in striatal neuropathology of the R6/2 transgenic HD mouse[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Huntington's disease model in mouse[3]
Dosage:	50 mg/kg
Administration:	Intraperitoneal injection; once daliy for 34 d, injected 3-NP at 6 hr after Taurodeoxycholic acid treatment
Result:	Reduced striatal atrophy, decreased striatal apoptosis, as well as fewer and smaller size ubiquitinated neuronal intranuclear huntingtin inclusions. Significantly improved locomotor and sensorimotor deficits.

REFERENCES

[1]. Benz C, et al. Effect of tauroursodeoxycholic acid on bile acid-induced apoptosis in primary human hepatocytes. Eur J Clin Invest. 2000 Mar;30(3):203-9.

[2]. Xie Q, et al. Effect of tauroursodeoxycholic acid on endoplasmic reticulum stress-induced caspase-12 activation. Hepatology. 2002 Sep;36(3):592-601.

[3]. Keene CD, et al. A bile acid protects against motor and cognitive deficits and reduces striatal degeneration in the 3-nitropropionic acid model of Huntington's disease. Exp Neurol. 2001 Oct;171(2):351-60.

[4]. Keene CD, et al. Tauroursodeoxycholic acid, a bile acid, is neuroprotective in a transgenic animal model of Huntington's disease. Proc Natl Acad Sci U S A. 2002 Aug 6;99(16):10671-6.

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