Taurochenodeoxycholic acid sodium

Cat. No.: HY-N1429

CAS No.: 6009-98-9

Molecular Formula: $C_{26}H_{44}NNaO_6S$

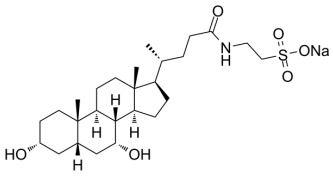
Molecular Weight: 521.69

Target: Apoptosis; Endogenous Metabolite

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (191.68 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9168 mL	9.5842 mL	19.1685 mL
	5 mM	0.3834 mL	1.9168 mL	3.8337 mL
	10 mM	0.1917 mL	0.9584 mL	1.9168 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: ≥ 2.08 mg/mL (3.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.99 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Taurochenodeoxycholic acid (12-Deoxycholyltaurine) sodium is one of the main bioactive substances of animals' bile acid. Taurochenodeoxycholic acid sodium induces apoptosis and shows obvious anti-inflammatory and immune regulation properties[1][2].

IC_{50} & Target	Microbial Metabolite	Human Endogenous Metabolite

In Vitro

Taurochenodeoxycholic acid (12-Deoxycholyltaurine) sodium dramatically improves the apoptosis rate of NR8383 cells in a concentration-dependent manner. In the meantime, Taurochenodeoxycholic acid sodium significantly augments PKC mRNA levels, activities and increases JNK, caspase-3 and caspase-8 mRNA expression levels, activities[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Taurochenodeoxycholic acid (12-Deoxycholyltaurine; TCDCA; 0.05, 0.1g/kg) sodium decreases the pulmonary coefficient in the model mice and reduces the pathological damages on their lungs; it can decrease the expression levels of TNF-α and TIMP-2 in pulmonary tissues in the pulmonary fibrosis mice and has no significant effects on MMP2[2]. Taurochenodeoxycholic acid sodium significantly normalizes the clinical inflammatory parameters, prevented indomethacin-induced increases in the biliary contents of secondary bile acids and hydrophobicity index, and tended to attenuate the intestinal inflammation[3]. Taurochenodeoxycholic acid sodium significantly suppresses paw swelling and polyarthritis index, increases the loss body weight and index of thymus and spleen, and amends radiologic changes in AA rats. The overproduction and mRNA expression of TNF-α, IL-1β and IL-6 are remarkably suppressed in serum and synovium tissue of all TCDCA-treated rats[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Wang X, et al. Taurochenodeoxycholic acid induces NR8383 cells apoptosis via PKC/JNK-dependent pathway. Eur J Pharmacol. 2016 Sep 5;786:109-15.

[2]. Zhou C, et al. The effects of taurochenodeoxycholic acid in preventing pulmonary fibrosis in mice. Pak J Pharm Sci. 2013 Jul;26(4):761-5.

[3]. Uchida A, et al. Taurochenodeoxycholic acid ameliorates and ursodeoxycholic acid exacerbates small intestinal inflammation. Am J Physiol. 1997 May;272(5 Pt 1):G1249-57.

[4]. Liu M, et al. Effects of taurochenodeoxycholic acid on adjuvant arthritis in rats. Int Immunopharmacol. 2011 Dec;11(12):2150-8.

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