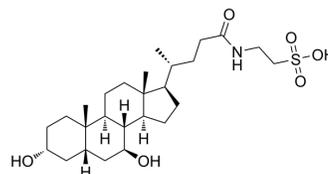


Tauroursodeoxycholate

Cat. No.:	HY-19696		
CAS No.:	14605-22-2		
Molecular Formula:	C ₂₆ H ₄₅ NO ₆ S		
Molecular Weight:	499.7		
Target:	ERK; Caspase; Endogenous Metabolite; Apoptosis		
Pathway:	MAPK/ERK Pathway; Stem Cell/Wnt; Apoptosis; 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 : 50 mg/mL (100.06 mM; Need ultrasonic)
 H₂O : 25 mg/mL (50.03 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent Concentration	Mass		
		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: PBS
 Solubility: 100 mg/mL (200.12 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Tauroursodeoxycholate (Tauroursodeoxycholic acid) is an endoplasmic reticulum (ER) stress inhibitor. Tauroursodeoxycholate significantly reduces expression of apoptosis molecules, such as caspase-3 and caspase-12. Tauroursodeoxycholate also inhibits ERK.

IC₅₀ & Target

ERK	Caspase-3	Caspase-12	Human Endogenous Metabolite
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In Vitro

Tauroursodeoxycholate (TUDCA) suppresses both viability and migration of vascular smooth muscle cells (VSMCs) through inhibition of ERK phosphorylation, by induction of mitogen-activated protein kinase phosphatase-1 (MKP-1) via PKCα. Tauroursodeoxycholate inhibits both the proliferation and migration of VSMCs via inhibition of ERK, through Ca²⁺-dependent PKCα translocation. Tauroursodeoxycholate prevents platelet-derived growth factor (PDGF) and vascular injury-

induced MMP-9 expression. The knock-down of MKP-1 using specific si-RNA restores the reduced VSMC viability by Tauroursodeoxycholate (200 μ M), which suggests that anti-proliferative effect of Tauroursodeoxycholate depended on the MKP-1 expression^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

The effects of Tauroursodeoxycholate (TUDCA) on proliferation and apoptosis of VSMCs in vivo are examined using immunohistochemistry for proliferating cell nuclear antigen (PCNA) and the transferase dUTP nick-end labeling (TUNEL) assay. Tauroursodeoxycholate (10, 50, and 100 mg/kg) increases the caspase 3 activity of injured tissues in a dose-dependent manner, indicating that Tauroursodeoxycholate induces apoptosis of VSMCs in the neointima. Using the injured tissues, further examination and comparison of the phosphorylation level of ERK and MMP-9 expression is performed at 1 week after injury, compared with normal controls. Balloon injury increased both the phosphorylation of ERK and the expression of MMP-9 in the tissues. Tauroursodeoxycholate (10, 50, and 100 mg/kg) inhibits phosphorylation of ERK and MMP-9 expression in a dose-dependent manner^[1]. Tauroursodeoxycholate (TUDCA) is a hydrophilic bile acid. Tauroursodeoxycholate as a cytoprotective agent improves liver function and can prevent hepatocellular carcinoma by reducing ER stress and apoptosis. Tauroursodeoxycholate significantly reduces the expression of apoptosis molecules, such as caspase-3, caspase-12, C/EBP homologous protein, c-Jun N-terminal kinase (JNK), activating transcription factor 4 (ATF4), X-box binding protein (XBP), and eukaryotic initiation factor 2 α (eIF2 α) in Ang II-induced ApoE^{-/-} mice (p<0.05). Tauroursodeoxycholate reduces Angiotensin (Ang) II induced abdominal aortic aneurysm (AAA) formation in ApoE^{-/-} mice. Tauroursodeoxycholate is used at a dose of 0.5 g/kg/day in treating Ang II-induced ApoE^{-/-} mice (ER stress inhibitor group). Systolic blood pressure (141.3 \pm 5.6 mmHg vs 145.9 \pm 8.9 mmHg; p>0.05) and total cholesterol levels (663.6 \pm 88.7 mg/dL vs 655.7 \pm 65.4 mg/dL; p>0.05) do not differ between the AAA model group and Tauroursodeoxycholate group. In addition, the maximum aortic diameter is significantly smaller in those in the Tauroursodeoxycholate group compared with those in the AAA model group (0.95 \pm 0.03 mm vs 1.79 \pm 0.04 mm; p<0.05). AAA lesion areas are also smaller in those in Tauroursodeoxycholate group than in those in the AAA model group (0.37 \pm 0.03 mm² vs 1.51 \pm 0.06 mm²; p<0.05)^[2].

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PROTOCOL

Cell Assay ^[1]

Cell viability and proliferation are measured using Ez-Cytox. VSMCs (5 \times 10³ cells) are seeded onto 96-well plates in Smooth Muscle Cell Growth Medium 2 (SMCGM2) and cultured. After serum starvation, Tauroursodeoxycholate (0, 50, 100, and 200 μ M) is added to the hVSMCs, with or without 1,2-bis(o-aminophenoxy) ethane-N,N,N',N'-tetraacetic acid tetra(acetoxymethyl) ester (BAPTA, 10 μ M) and 7-hydroxystaurosporine (H7, 10 μ M) and cultured for 24 h. To assess the effect of Tauroursodeoxycholate on the PDGF-stimulated hVSMC proliferation, hVSMCs are seeded onto 96-well plates and cultured. After serum starvation, Tauroursodeoxycholate (0, 50, 100, and 200 μ M) is added to the hVSMCs, with or without PDGF-BB (50 ng/mL) and cultured. After addition of 10 μ L of Ez-Cytox into each well, cell viability is evaluated by measuring the optical density at 450 nm^[1].

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Animal Administration ^{[1][2]}

Rats^[1]

Sprague-Dawley rats are anaesthetized with a combined anaesthetic (Ketamine, 70 mg/kg; Xylazine, 7 mg/kg ip). Tauroursodeoxycholate is administered orally once a day, in different concentrations (i.e. vehicle, 10, 50, and 100 mg/kg) for 2 weeks. The carotid arteries are fixed by perfusion with 4% formaldehyde, then the tissues are embedded in paraffin, and sections (8 μ m) are stained with H&E^[1].

Mice^[2]

Thirty ApoE^{-/-} C57BL/6 male mice aged 8 weeks are randomly divided into three groups (n=10 in each group): (i) sham operated and injected with physiologic (0.9%) saline as vehicle (normal: group); (ii) mini-osmotic pumps are implanted subcutaneously into the right flank of ApoE^{-/-} mice to release Ang II (1000 ng/kg/min) over the course of 28 days (AAA model group); (iii) AAA model mice treated with Tauroursodeoxycholate daily for 4 weeks at a dosage of 0.5 g/kg/day in drinking water (Tauroursodeoxycholate group). Mice are sacrificed after 28 days of Ang II infusion^[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Cell Biol. 2023 May;25(5):726-739.
- Adv Sci (Weinh). 2021 Nov;8(21):e2101936.
- Mol Ther. 2022 Dec 24;S1525-0016(22)00718-3.
- Redox Biol. 2023 Aug 25, 102861.
- Cell Death Dis. 2023 Aug 15;14(8):524.

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REFERENCES

- [1]. Kim SY, et al. Tauroursodeoxycholate (TUDCA) inhibits neointimal hyperplasia by suppression of ERK viaPKC α -mediated MKP-1 induction. Cardiovasc Res. 2011 Nov 1;92(2):307-16.
- [2]. Qin Y, et al. Tauroursodeoxycholic Acid Attenuates Angiotensin II Induced Abdominal Aortic Aneurysm Formation in Apolipoprotein E-deficient Mice by Inhibiting Endoplasmic Reticulum Stress. Eur J Vasc Endovasc Surg. 2017 Mar;53(3):337-345.
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