

Product Data Sheet

Product Name: Sodium Tauroursodeoxycholate (TUDC)

Cat. No.: GC17425

Chemical Properties

Cas. No. 35807-85-3

Chemical Name sodium (R,Z)-4-((3R,5S,7S,8R,9S,10S,13R,14S,17R)-3,7-dihydroxy-10,13-dimethylhexadecahydro-1H-cyclopenta[a]phenanthren-17-yl)-N-(2-sulfoethyl)pentanimidate

SMILES C[C@]([C@@]1([H])CC[C@@]2([H])[C@@]([C@](O)([H])C[C@]3([H])C[C@@](O)([H])CC[C@@]34C)([H])[C@]4([H])CC[C@]12C)([H])CC/C([O-])=N/CCS(=O)(=O)[O-].[Na+]

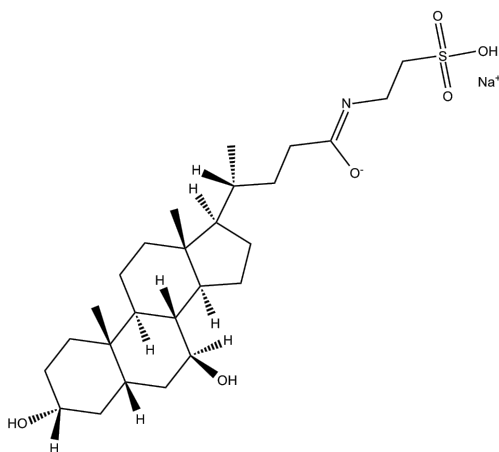
Formula $C_{26}H_{44}NNaO_6S$ M.Wt 521.69

Solubility ≥ 26.1 mg/mL in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell experiment: Cell viability and proliferation are measured using Ez-Cytox. VSMCs (5×10³ 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 µM) 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 µM) and 7-hydroxystaurosporine (H7, 10 µM) 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 µM) is added to the hVSMCs, with or without PDGF-BB (50 ng/mL) and cultured. After addition of 10 µL of Ez-Cytox into each well, cell viability is evaluated by measuring the optical density at 450 nm[1].

Animal experiment: 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 µm) 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].

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References:

- [1]. Kim SY, et al. Tauroursodeoxycholate (TUDCA) inhibits neointimal hyperplasia by suppression of ERK via PKC 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.

Background

Sodium tauroursodeoxycholate (TUDC) shows therapeutic effect on cholestasis [1, 2]. In human erythrocytes, it inhibited 2',7'-bis-(carboxypropyl)-5(6)-carboxyfluorescein (BCPCF) efflux induced by bile salts with an IC₅₀ value of 560 μ M [3].

Cholestasis is the syndrome resulted from the impairment of the formation of bile, a vital function [4].

cVA-of-CLF means the canalicular vacuolar accumulation of cholyllysylfluorescein [1]. cVA of CLF is a parameter to indicate overall biliary secretion [5]. Incubation with 17 β EG dose-dependently decreased the cVA-of-CLF in cells. 17 β EG at a concentration of 50 μ M decreased cVA-of-CLF by 40%. The simultaneous incubation with TUDC and 17 β EG improved the decreased cVA by 24%. The simultaneous incubation with SAME and 17 β EG improved the decreased cVA by 18%. The simultaneous incubation with TUDC, SAME and 17 β EG improved the decreased cVA by 28%. But the effect of TUDC + SAME was not significantly greater than the effect of either protectant alone [1].

In rats, intrahepatic cholestasis was induced by the administration of phalloidin at an i.p. dose of 500 μ g/kg for 7 days. In these treated rats, bile flow was decreased, and activities of glutamic pyruvic transaminase, leucine aminopeptidase, serum alkaline phosphatase, and concentrations of bile acid, phospholipid and cholesterol were increased. But these effects were significantly suppressed by tauroursodeoxycholate. In these rats, excretion rates of biliary cholesterol and phospholipid were significantly improved by tauroursodeoxycholate [2].

References:

- [1]. Milkiewicz P, Roma MG, Cardenas R, et al. Effect of tauroursodeoxycholate and S-adenosyl-L-methionine on 17 β -estradiol glucuronide-induced cholestasis. *Journal of hepatology*, 2001, 34(2): 184-191.

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- [2]. Ishizaki K, Kinbara S, Hirabayashi N, et al. Effect of sodium tauroursodeoxycholate on phalloidin-induced cholestasis in rats. *European journal of pharmacology*, 2001, 421(1): 55-60.
- [3]. Mrowczynska L, Bobrowska-Hgerstrand M, Wrobel A, et al. Inhibition of MRP1-mediated efflux in human erythrocytes by mono-anionic bile salts. *Anticancer research*, 2005, 25(5): 3173-3178.
- [4]. Trauner M, Meier PJ, Boyer JL. Molecular pathogenesis of cholestasis. *New England Journal of Medicine*, 1998, 339(17): 1217-1227.
- [5]. Milkiewicz P, Roma MG, Elias E, et al. Hepatoprotection with tauroursodeoxycholate and β muricholate against tauroolithocholate induced cholestasis: involvement of signal transduction pathways. *Gut*, 2002, 51(1): 113-119.

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