

Product Data Sheet

Product Name: Dehydrolithocholic Acid

Cat. No.: GC47182

Chemical Properties

Cas. No. 1553-56-6

SMILES C[C@H](CCC(O)=O)[C@@]1([H])CC[C@@]2([H])[C@]3([H])CC[C@]4([H])CC(CC[C@]4(C)[C@@]3([H])CC[C@@]21C)=O

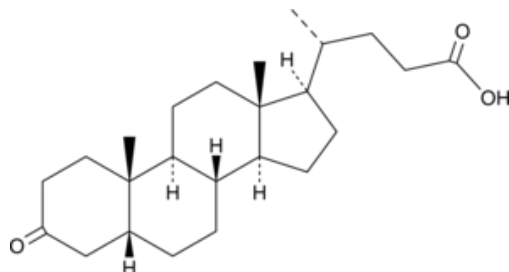
Formula C₂₄H₃₈O₃ M.Wt 374.6

Solubility DMF: 30 mg/ml, DMF:PBS (pH 7.2) Storage Store at -20°C
(1:4): 0.20 mg/ml, DMSO: 15 mg/ml, Ethanol: 10 mg/ml

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

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

Structure



Protocol

Cell experiment [1]:

Cell lines Primary CD4⁺T cells from wild-type B6 mice

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

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Preparation Method A low concentration of TGF- β (0.01ng/ml) was used for Treg culture. DMSO, Dehydrolithocholic acid (20 μ M) or isoalloLCA (20 μ M) was added on day 0 and CD4⁺ T cells were gated for analyses on day 3 for Th17 and Treg, day 5 for Th1 and Th2.

Reaction Conditions 20 μ M ; 48h

Applications Dehydrolithocholic acid can inhibit Th 17 differentiation of cells and reduce IL-17 α expression levels, but does not affect T cell differentiation into Th 1 or Th 2 cells

Animal experiment [1]:

Animal models C57BL/6J mice from Jackson Laboratories (Jax)

Preparation Method Jax-B6 mice were fed with fecal slurry containing SFB (segmented filamentous bacteria from C57 BL/6 NTac mice) and fed with a control diet or a diet containing 0.3% (w/w) Dehydrolithocholic acid for one week. The average concentration of Dehydrolithocholic acid in the cecal contents is 24picomol/mg wet weight (approximately equivalent to μ M)

Dosage form 0.3% (w/w) dietary administration

Applications Dehydrolithocholic Acid treatment significantly reduced the percentage of Th17 cells in the ileum

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References:

[1] Saiyu Hang, Donggi Paik, et al. Bile acid metabolites control Th17 and Treg cell differentiation. Nature. 2019 Dec;576(7785):143-148.

Background

Dehydrolithocholic acid (3-Oxo-5 β -cholanoic acid; 3-oxoLCA; DLA) is a bile acid metabolite that inhibits T helper cell 17 (Th17) differentiation by interacting with retinoic acid receptor associated orphan receptor (ROR γ t) [1]. Bile acids are natural surfactants derived from cholesterol, which are crucial for lipid digestion and glucose metabolism, protecting the intestinal epithelium and endowing the mammalian gut with resistance to pathogens[2]. Dehydrolithocholic acid can be a potential candidate drug for the treatment of inflammatory bowel disease[1].

In vitro, use Dehydrolithocholic acid (5 μ M; 24 hours) can activate the TGR 5-ERK 1/2 pathway on the surface of bronchial epithelial cells (HBEpC) and increase the expression of cathelicidin in the cells[3]. Use Dehydrolithocholic Acid (20 μ M; 48 hours) treatment with primary CD4⁺T cells can inhibit Th 17 differentiation and reduce IL-17 α expression levels, but does not affect T cell differentiation into Th 1 or Th 2 cells[4]. MKN 45 was cultured in serum-free medium and treated with 10ng/mL LIF alone or in combination with Dehydrolithocholic acid (1, 3, 10, and 20 μ M) to reverse LIF induced cell growth at the lowest concentration of 1 μ M[5].

In vivo, Jax-B6 mice were fed with fecal slurry containing SFB (segmented filamentous bacteria from C57 BL/6 NTac mice) and fed with a control diet or a diet containing 0.3% (w/w) Dehydrolithocholic acid for one week. Treatment with Dehydrolithocholic acid significantly reduced the percentage of Th17 cells in the mouse gut[4]. The rat MCAO model was treated with Dehydrolithocholic acid (pre ischemic administration; 10mg/kg; ip.), which significantly promoted neuronal death in primary culture after OGD injury by downregulating PLOD 2 levels and increasing PTEN expression[6].

References:

- [1] Hang S, Paik D, Yao L, et al. Bile acid metabolites control T(H)17 and T(reg) cell differentiation[J]. Nature, 2019, 576(7785): 143-148.
- [2] Cheng W, Zhou X, Jin C, et al. Acid-base transformative HADLA micelles alleviate colitis by

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restoring adaptive immunity and gut microbiome[J]. J Control Release, 2023, 364: 283-296.

[3] Iwona T Myszor, Kornelia Lapka, et al. Bile acid metabolites enhance expression of cathelicidin antimicrobial peptide in airway epithelium through activation of the TGR5-ERK1/2 pathway. Sci Rep. 2024 Mar 21;14(1):6750.

[4] Saiyu Hang, Donggi Paik, et al. Bile acid metabolites control Th17 and Treg cell differentiation. Nature. 2019 Dec;576(7785):143-148.

[5] Cristina Di Giorgio, Elva Morretta, et al. Bile acids serve as endogenous antagonists of the Leukemia inhibitory factor (LIF) receptor in oncogenesis. Biochem Pharmacol. 2024 May;223:116134.

[6] Xiangyi Kong, Xujin Yao, et al. tDCS Regulates ASBT-3-OxoLCA-PLOD2-PTEN Signaling Pathway to Confer Neuroprotection Following Rat Cerebral Ischemia-Reperfusion Injury. Mol Neurobiol. 2023 Nov;60(11):6715-6730.

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