

Product Data Sheet

Product Name: 12-Ketodeoxycholic acid

Cat. No.: GC60443

Chemical Properties

Cas. No. 5130-29-0

SMILES C[C@H](CCC(O)=O)[C@H]([C@]12C)CC[C@@]1([H])[C@]3([H])CC[C@]4([H])C[C@H](O)CC[C@]4(C)[C@@]3([H])CC2=O

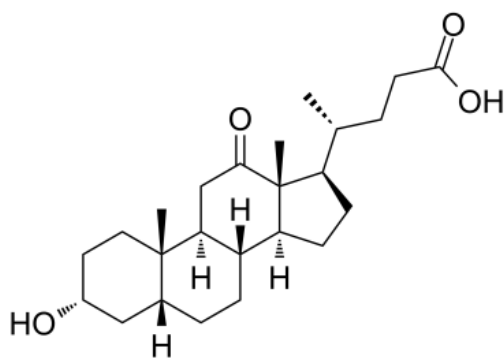
Formula $C_{24}H_{38}O_4$ M.Wt 390.56

Solubility DMF: 30 mg/ml, DMSO: 30 mg/ml, Ethanol: 30 mg/ml, Ethanol: PBS (pH 7.2) (1:3): 0.25 mg/ml
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

Cell experiment [1]:

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Cell lines HEK293T cells

HEK293T cells were cultured in Dulbecco's modified eagle medium (DMEM) supplemented with 10% fetal bovine serum at 37°C in 5% CO₂. Cells were maintained in a humidified incubator under standard cell culture conditions. To assess the anti-inflammatory effects of 12-Ketodeoxycholic acid, cells were stimulated with lipopolysaccharide (LPS) at a concentration of 5µg/mL for 24h. After 24h, the culture medium was replaced with LPS-free medium, and the cells were treated with different concentrations (50, 75, and 100µM) of 12-Ketodeoxycholic acid for 24h. Then cytokine levels in cell culture supernatants were determined by ELISA kits.

Reaction
Conditions 50, 75, and 100µM; 24h

Applications Treatment of LPS-stimulated 293T cells with 12-Ketodeoxycholic acid significantly reduced the levels of pro-inflammatory cytokines IL-6, IL-1β, and TNF-α and increased anti-inflammatory IL-10 levels.

Animal experiment [1]:

Animal models Seven-week-old female SPF C57BL/6J mice

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Preparation Method	Seven-week-old female SPF C57BL/6J mice were individually housed in an independent ventilation cage for 1 week to acclimatize to the laboratory environment. During this acclimatization period, mice had ad libitum access to standard rodent chow and water. To induce chemical colitis in mice, a 2.5% (wt/vol) solution of DSS with a molecular weight range of 36 000-50 000 was prepared and provided ad libitum in the drinking water. At the onset of apparent diarrhea and significant weight loss compared to the control group, the DSS treatment was discontinued and the following experimental procedures were initiated. To evaluate the therapeutic effects of 12-Ketodeoxycholic acid in DSS-induced colitis, mice were randomly allocated into three groups: a normal control group receiving regular drinking water, a DSS+PBS group receiving 3% DSS in the drinking water for 7 days followed by PBS administration every other day, and a DSS+12-Ketodeoxycholic acid group receiving 50mg/kg/d of 12-Ketodeoxycholic acid. Body weights were recorded every 2 days, and diarrhea occurrence was monitored daily. At the conclusion of the experiment, mice were euthanized, and spleen weights were recorded. Serum, colonic contents, and colonic tissues were collected for further analyses, with serum stored at -20°C and colonic contents/tissues preserved in liquid nitrogen for subsequent analyses.
Dosage form	50mg/kg/day; 7 days; oral administration
Applications	12-Ketodeoxycholic acid alleviated colonic inflammation, reduced the expression of pro-inflammatory cytokines IL-6, IL-1 β , and TNF- α , increased anti-inflammatory IL-10 levels, and inhibited the mTORC1 signaling pathway in the colon of DSS-induced colitis mice.

References:

[1] Zhang K, Xu Y, Zheng Y, et al. Bifidobacterium pseudolongum-Derived Bile Acid from Dietary Carvacrol and Thymol Supplementation Attenuates Colitis via cGMP-PKG-mTORC1 Pathway. *Adv Sci (Weinh)*. 2024;11(43):e2406917.

Background

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12-Ketodeoxycholic acid is a metabolite of the secondary bile acid deoxycholic acid [1]. Bile acids are primarily synthesized from cholesterol in the liver and then secreted into the intestine through bile. In the intestine, some bile acids are metabolized by gut microbiota and reabsorbed into the liver through the enterohepatic circulation. Additionally, some bile acids and their metabolites are filtered by the kidneys and excreted in urine [2]. 12-Ketodeoxycholic acid can be used as potential metabolite biomarkers or therapeutic approaches for metabolic diseases such as atherosclerosis and non-alcoholic fatty liver disease (NAFLD) [3][4]. The level changes of 12-Ketodeoxycholic acid can also serve as a biomarker for kidney injury [5]. 12-Ketodeoxycholic acid is usually used in the research of intestinal inflammation [6].

In vitro, treatment of LPS-stimulated 293T cells with 12-Ketodeoxycholic acid (50, 75, and 100 μ M; 24h) significantly reduced the levels of pro-inflammatory cytokines IL-6, IL-1 β , and TNF- α and increased anti-inflammatory IL-10 levels [7].

In vivo, 12-Ketodeoxycholic acid (50mg/kg/day; 7 days; oral administration) alleviated colonic inflammation, reduced the expression of pro-inflammatory cytokines IL-6, IL-1 β , and TNF- α , increased anti-inflammatory IL-10 levels, and inhibited the mTORC1 signaling pathway in the colon of DSS-induced colitis mice [7].

References:

- [1] Franco P, Porru E, Fiori J, et al. Identification and quantification of oxo-bile acids in human faeces with liquid chromatography-mass spectrometry: A potent tool for human gut acidic sterolbiome studies. *J Chromatogr A*. 2019;1585:70-81.
- [2] Cai J, Rimal B, Jiang C, Chiang JYL, Patterson AD. Bile acid metabolism and signaling, the microbiota, and metabolic disease. *Pharmacol Ther*. 2022;237:108238.
- [3] Xue J, Allaband C, Zuffa S, et al. Gut Microbiota and Derived Metabolites Mediate Obstructive Sleep Apnea Induced Atherosclerosis. Preprint. *bioRxiv*. 2024;2024.11.18.624205.
- [4] Ahmad MI, Umair Ijaz M, Hussain M, et al. High fat diet incorporated with meat proteins changes biomarkers of lipid metabolism, antioxidant activities, and the serum metabolomic profile in Glrx1^{-/-} mice. *Food Funct*. 2020;11(1):236-252.
- [5] Chen H, Cao G, Chen DQ, et al. Metabolomics insights into activated redox signaling and lipid metabolism dysfunction in chronic kidney disease progression. *Redox Biol*. 2016;10:168-178.

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- [6] Feng P, Li Q, Liu L, et al. Crocetin Prolongs Recovery Period of DSS-Induced Colitis via Altering Intestinal Microbiome and Increasing Intestinal Permeability. *Int J Mol Sci.* 2022;23(7):3832.
- [7] Zhang K, Xu Y, Zheng Y, et al. Bifidobacterium pseudolongum-Derived Bile Acid from Dietary Carvacrol and Thymol Supplementation Attenuates Colitis via cGMP-PKG-mTORC1 Pathway. *Adv Sci (Weinh).* 2024;11(43):e2406917.

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