Hyodeoxycholic acid

Cat. No.: HY-N0169

CAS No.: 83-49-8

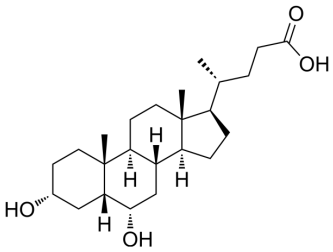
Molecular Formula: $C_{24}H_{40}O_4$

Molecular Weight: 392.57

Target: Endogenous Metabolite; G protein-coupled Bile Acid Receptor 1

Pathway: Metabolic Enzyme/Protease; GPCR/G Protein

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (254.73 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5473 mL	12.7366 mL	25.4732 mL
5 mM	0.5095 mL	2.5473 mL	5.0946 mL
10 mM	0.2547 mL	1.2737 mL	2.5473 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 25 mg/mL (63.68 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (6.37 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.37 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.37 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hyodeoxycholic acid is a secondary bile acid formed in the small intestine by the gut flora, and acts as a TGR5 (GPCR19) agonist, with an EC_{50} of 31.6 μM in CHO cells.

IC_{50} & Target	Microbial Metabolite	Human Endogenous Metabolite

In Vitro

Hyodeoxycholic acid is a secondary hydrophilic bile acid formed in the small intestine by the gut flora, and acts as an agonist

	of TGR5, with an EC_{50} of 31.6 μM in CHO cells[1]. Hyodeoxycholic acid (50, 100 μM) increases the expression of genes (Abca1, Abcg1, and Apoe) involved in cholesterol efflux in RAW 264.7 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Hyodeoxycholic acid (HDCA; 1.25% (wt/wt)) obviously decreases fat mass and increases lean mass but does not raise the serum levels of any organ toxicity markers in LDLRKO mice. Hyodeoxycholic acid inhibits atherosclerotic lesion formation in LDLRKO at multiple sites, improves plasma lipoprotein profiles, decreases plasma glucose level and intestinal cholesterol absorption efficiency and increases daily cholesterol excretion through fecal output. Hyodeoxycholic acid also improves HDL function as measured by a cholesterol efflux assay[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Mice[2] For atherosclerosis studies, 8-wk-old female LDLRKO mice are fed a Western diet (21% fat, 0.15% cholesterol; TD.88137) for 8 wk. One group of mice (baseline group) is euthanized at this time point for lesion measurement in the aortic root region and in the innominate artery. Atherosclerotic lesion in the whole aorta is not examined in the baseline group. The remaining mice are then divided into 2 groups and fed the following diets for another 15 wk before euthanasia: group 1, chow diet (5% fat, AIN-76A Rodent Diet); and group 2, chow diet + 1.25% (wt/wt) Hyodeoxycholic acid. For other studies, 8-wk-old female LDLRKO mice are fed a chow diet or chow diet + 1.25% Hyodeoxycholic acid for 3 wk before phenotype measurements. Food consumption and body weight are recorded weekly. Animals are measured for total body fat mass and lean mass by magnetic resonance imaging (MRI) using Bruker Minispec with software from Eco Medical Systems[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Sato H, et al. Novel potent and selective bile acid derivatives as TGR5 agonists: biological screening, structure-activity relationships, and molecular modeling studies. J Med Chem. 2008 Mar 27;51(6):1831-41.

[2]. Shih DM, et al. Hyodeoxycholic acid improves HDL function and inhibits atherosclerotic lesion formation in LDLR-knockout mice. FASEB J. 2013 Sep;27(9):3805-17.

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