Web: www.taiclone.com
Tel: +886-2-2735-9682
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Chenodeoxycholic Acid

Catalog No: tcsc0834

Available Sizes

Size: 100mg

Size: 500mg

Specifications

CAS No:
474-25-9

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

Pathway:
Metabolic Enzyme/Protease;Metabolic Enzyme/Protease

Target:
FXR;Endogenous Metabolite

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 50 mg/mL (127.37 mM)

Alternative Names:
CDCA

Observed Molecular Weight:
392.57

Product Description

Chenodeoxycholic Acid is a hydrophobic primary bile acid that activates nuclear receptors (**FXR**) involved in cholesterol metabolism.

In Vitro:

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Chenodeoxycholic acid (CDCA) and Deoxycholic acid (DCA) both inhibit 11 beta HSD2 with IC_{50} values of 22 mM and 38 mM, respectively and causes cortisol-dependent nuclear translocation and increases transcriptionalactivity of mineralocorticoid receptor (MR)[1]. Chenodeoxycholic acid is able to stimulate Ishikawa cell growth by inducing a significant increase in Cyclin D1 protein and mRNA expression through the activation of the membrane G protein-coupled receptor (TGR5)-dependent pathway[2]. Chenodeoxycholic acid (CDCA) induces LDL receptor mRNA levels approximately 4 fold and mRNA levels for HMG-CoA reductase and HMG-CoA synthase two fold in a cultured human hepatoblastoma cell line, Hep G2[3]. Chenodeoxycholic acid-induced Isc is inhibited (≥67%) by Bumetanide, $BaCl_2$, and the cystic fibrosis transmembrane conductance regulator (CFTR) inhibitor CFTRinh-172. Chenodeoxycholic acid-stimulated Isc is decreased 43% by the adenylate cyclase inhibitor MDL12330A and Chenodeoxycholic acid increases intracellular cAMP concentration[4]. Chenodeoxycholic acid treatment activates C/EBPβ, as shown by increases in its phosphorylation, nuclear accumulation, and expression in HepG2 cells. Chenodeoxycholic acid enhances luciferase gene transcription from the construct containing -1.65-kb GSTA2 promoter, which contains C/EBP response element (pGL-1651). Chenodeoxycholic acid treatment activates AMP-activated protein kinase (AMPK), which leads to extracellular signal-regulated kinase 1/2 (ERK1/2) activation, as evidenced by the results of experiments using a dominant-negative mutant of AMPKα and chemical inhibitor[5].