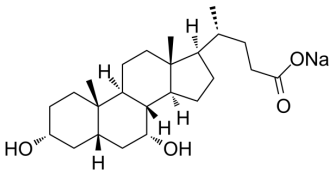
Chenodeoxycholic acid sodium

Cat. No.:	HY-76847A
CAS No.:	2646-38-0
Molecular Formula:	$C_{24}H_{39}NaO_4$
Molecular Weight:	414.55
Target:	FXR; Autophagy; Endogenous Metabolite
Pathway:	Metabolic Enzyme/Protease; Autophagy
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

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

In Vitro

Chenodeoxycholic acid sodium (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 sodium 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 sodium (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 sodium-induced Isc is inhibited (≥67%) by Bumetanide, $BaCl_2$, and the cystic fibrosis transmembrane conductance regulator (CFTR) inhibitor CFTRinh-172. Chenodeoxycholic acid sodium-stimulated Isc is decreased 43% by the adenylate cyclase inhibitor MDL12330A and Chenodeoxycholic acid sodium increases intracellular cAMP concentration[4]. Chenodeoxycholic acid sodium treatment activates C/EBPβ, as shown by increases in its phosphorylation, nuclear accumulation, and expression in HepG2 cells. Chenodeoxycholic acid sodium enhances luciferase gene transcription from the construct containing -1.65-kb GSTA2 promoter, which contains C/EBP response element (pGL-1651). Chenodeoxycholic acid sodium 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].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [1]

Briefly, transfected HEK-293 cells, incubated in charcoal-treated Dulbecco's modified Eagle's medium for 24 h, are washed once with Hanks' solution and resuspended in a buffer containing 100 mM NaCl, 1 mM $MgCl_2$, 1 mM EDTA, 1 mM EGTA, 250 mMsucrose, 20 mM Tris-HCl, pH 7.4. Cells are lysed by freezing in liquid nitrogen. Dehydrogenase activity is measured in a final volume of 20 µL containing the appropriate concentration of bile acid, 30 nCi of [^{3}H]cortisol, and unlabeled cortisol to a final concentrations of 50 nM. The reaction is started by mixing cell lysate with the reaction mixture. Alternatively, endoplasmic reticulum microsomes are prepared from transfected HEK-293 cells and incubated with reaction mixture containing various concentrations of cortisol and CDCA. Incubation proceeded for 20 min, and the conversion of cortisol to cortisone is determined by thin layer chromatography (TLC). Because of the inaccuracy of the TLC method at low conversion rates and the end-product inhibition of 11βHSD2 at conversion rates higher than 60-70%, only conversion rates between 10

	and 60% are considered for calculation. The inhibitory constant IC_{50} is evaluated using the curve-fitting program. Results are expressed as means±S.E. and consist of at least four independent measurements. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [1]	The cell viability is analyzed by incubating transfected HEK-293 cells and CHO cells for 1 h with the corresponding concentration of bile acid and staining with trypan blue. The toxicity of bile acids is analyzed using the tetrazolium salt MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) according to the cell proliferation kit I. No significant differences between control and bile acid-treated cells are obtained in both tests. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2019 Mar;29(3):193-205.
- Cell Host Microbe. 2024 Jan 11:S1931-3128(23)00510-3.
- Research (Wash D C). 2022 Nov 2;2022:9784081.
- Sci Total Environ. 2023 Apr 15.
- Phytother Res. 2021 Mar;35(3):1495-1507.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Stauffer AT, et al. Chenodeoxycholic acid and deoxycholic acid inhibit 11 beta-hydroxysteroid dehydrogenase type 2 and cause cortisol-induced transcriptional activation of the mineralocorticoid receptor. J Biol Chem. 2002 Jul 19;277(29):26286-92

[2]. Noh K, et al. Farnesoid X receptor activation by chenodeoxycholic acid induces detoxifying enzymes through AMP-activated protein kinase and extracellular signal-regulated kinase 1/2-mediated phosphorylation of CCAAT/enhancer binding protein β. Drug Metab

[3]. Casaburi I, et al. Chenodeoxycholic acid through a TGR5-dependent CREB signaling activation enhances cyclin D1 expression and promotes human endometrial cancer cell proliferation. Cell Cycle. 2012 Jul 15;11(14):2699-710

[4]. Ao M, et al. Chenodeoxycholic acid stimulates Cl(-) secretion via cAMP signaling and increases cystic fibrosis transmembrane conductance regulator phosphorylation in T84 cells. Am J Physiol Cell Physiol. 2013 Aug 15;305(4):C447-56

[5]. Kawabe Y, et al. The molecular mechanism of the induction of the low density lipoprotein receptor by chenodeoxycholic acid in cultured human cells. Biochem Biophys Res Commun. 1995 Mar 8;208(1):405-11.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA