Glycochenodeoxycholic acid

Cat. No.: HY-N2334

CAS No.: 640-79-9

Molecular Formula: $C_{26}H_{43}NO_5$

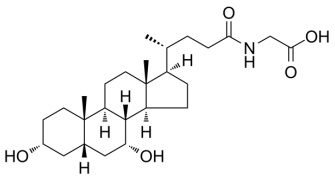
Molecular Weight: 449.62

Target: Endogenous Metabolite; Apoptosis; STAT; BCL6; Interleukin Related; Caspase

Pathway: Metabolic Enzyme/Protease; Apoptosis; JAK/STAT Signaling; Stem Cell/Wnt; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (222.41 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2241 mL	11.1205 mL	22.2410 mL
	5 mM	0.4448 mL	2.2241 mL	4.4482 mL
	10 mM	0.2224 mL	1.1121 mL	2.2241 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.56 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.56 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Glycochenodeoxycholic acid (Chenodeoxycholylglycine) is a relatively toxic bile salt generated in the liver from chenodeoxycholic acid and glycine. Glycochenodeoxycholic acid inhibits Autophagosome formation and impairs lysosomal function by inhibiting lysosomal proteolysis and increasing lysosomal pH in human normal liver cells, leading to the Apoptosis of human hepatocyte cells. Glycochenodeoxycholic acid induces stemness and chemoresistance via activating STAT3 signaling pathway in hepatocellular carcinoma cells (HCC). Glycochenodeoxycholic acid is promising for research in the field of cholestasis desease, hepatocellular carcinoma and primary sclerosing cholangitis (PSC)[1][2][3][4].

IC_{50} & Target	Microbial Metabolite	Human Endogenous Metabolite

In Vitro

Glycochenodeoxycholic acid (0-100 μM, 6 h) significantly increases the amount of dead cells and decreases in LC3, ATG5 and BECN1 expression in human normal liver cells, leading to Autophagosome formation inhibition[1].

Glycochenodeoxycholic acid (5–500 μM, 24 h) exerts no induction or reduction of TGF-β mRNA expression in KMBC cells and LX-2 cells[2].

Glycochenodeoxycholic acid (200 μM, 24 h and 48 h) enhances stemness and chemoresistance of hepatocellular carcinoma cells (HCC) by activating the STAT3 signaling pathway, suppressing the expression of apoptotic genes (Bcl10, Caspase 3, Caspase 4, Tp53, BAD) and increasing the expression of anti-apoptotic genes (Bcl2, Bcl-xl and IL10)[4].

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

RT-PCR[1]

Cell Line:	Human normal liver cells
Concentration:	0-100 μM
Incubation Time:	6 h
Result:	Decreased the TFE3 levels in a dose-dependent manner in human normal liver cells.

Apoptosis Analysis[4]

Cell Line:	Huh7 and LM3 cell lines
Concentration:	200 μM
Incubation Time:	24 h and 48 h
Result:	Suppressed the expression of apoptotic genes (Bcl10, Caspase 3, Caspase 4, Tp53, BAD) and increased the expression of anti-apoptotic genes (Bcl2, Bcl-xl and Il10) in Huh7 and LM3 cell lines.Increased the cell viability and promoted chemoresistance treated with 5-FU (HY-90006) (120 μg/mL) and cisplatinin (10 μg/mL) in Huh7 and LM3 cell lines.

Cell Autophagy Assay[1]

Cell Line:	L02 cells
Concentration:	100 μM
Incubation Time:	6 h
Result:	Significantly reduced GFP-LC3 puncta and decreased microtubule-associated protein 1 light chain 3 (LC3), autophagy related 5 (ATG5) and beclin 1 (BECN1) expression in L02 cells.

RT-PCR[2]

Cell Line:	KMBC cells
Concentration:	5–500 μM
Incubation Time:	24 h
Result:	Unaltered TGF-β mRNA expression in KMBC cells and LX-2 cells.

CUSTOMER VALIDATION

- Hepatol Int. 2024 Jan 3.
- Discov Oncol. 2023 Jan 11;14(1):4.
- SSRN. 2022 Jan 26.

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REFERENCES

[1]. Lan W, et al. Glycochenodeoxycholic acid impairs transcription factor E3 -dependent autophagy-lysosome machinery by disrupting reactive oxygen species homeostasis in L02 cells[J]. Toxicol Lett. 2020 Oct 1;331:11-21.

[2]. Wang A, et al. Glycochenodeoxycholic Acid Does Not Increase Transforming Growth Factor-Beta Expression in Bile Duct Epithelial Cells or Collagen Synthesis in Myofibroblasts[J]. J Clin Exp Hepatol. 2017 Dec;7(4):316-320.

[3]. Shi C, et al. Glycochenodeoxycholic acid induces stemness and chemoresistance via the STAT3 signaling pathway in hepatocellular carcinoma cells[J]. Aging (Albany NY). 2020 Aug 3;12(15):15546-15555.

[4]. Gonzalez B, et al. Glycochenodeoxycholic acid (GCDC) induced hepatocyte apoptosis is associated with early modulation of intracellular PKC activity. Mol Cell Biochem. 2000 Apr;207(1-2):19-27.

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