Glycodeoxycholic Acid

Cat. No.: HY-125731

CAS No.: 360-65-6

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

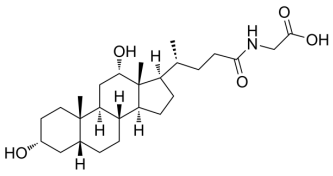
Molecular Weight: 449.62

Target: Endogenous Metabolite; STAT; Autophagy

Pathway: Metabolic Enzyme/Protease; JAK/STAT Signaling; Stem Cell/Wnt; Autophagy

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 : 125 mg/mL (278.01 mM; ultrasonic and warming and heat to 60°C)

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: ≥ 2.08 mg/mL (4.63 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.63 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.63 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Glycodeoxycholic Acid is a natural product found in Streptomyces nigricans, Trypanosoma brucei and C. elegans. Glycodeoxycholic Acid induces hepatocyte necrosis and autophagy in patients with obstructive cholestasis[1][2][3].

In Vitro

Glycodeoxycholic Acid (200 μM, 24-48 h) induces stemness and chemotherapy resistance of hepatocellular carcinoma cells through STAT3 signaling pathway[1].

Glycodeoxycholic Acid (50 μM, pretreatment for 1 h) can eliminate UCB-induced cytochrome c oxidase inhibition, and significantly prevent oxidative stress, metabolic changes and cell death[2].

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

Cell Viability Assay[1]

Cell Line:	Huh7, LM3
Concentration:	200 μM
Incubation Time:	24, 48 h
Result:	Increased cell viability treated with 5-FU and cisplatin.

Western Blot Analysis[1]

Cell Line:	Huh7, LM3
Concentration:	200 μM
Incubation Time:	24, 48 h
Result:	Suppressed the expression of apoptotic genes and increased anti-apoptotic genes. Promoted the expression of Sox2, Sox9, Nanog and CD133. Down-regulated the level of E-cadherin and up-regulated vimentin. Decreased the levels of SOCS2, SOCS5, PTPN1 and PTPN11.

In Vivo

Glycodeoxycholic Acid (11.20 mg/kg, biliary and pancreatic duct injection) can induce acute pancreatitis in rhesus monkeys [3].

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

Animal Model:	Experimental macaque model[3]
Dosage:	11.20 mg/kg
Administration:	injected along the biliopancreatic duct
Result:	Increased the levels of Serum amylase and lipase. Elevated Blood pressure and heart rate.

REFERENCES

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

[2]. Vaz AR, et al. Bilirubin selectively inhibits cytochrome c oxidase activity and induces apoptosis in immature cortical neurons: assessment of the protective effects of glycoursodeoxycholic acid. J Neurochem. 2010 Jan;112(1):56-65.

[3]. Fauzi A, et al. Role of glycodeoxycholic acid to induce acute pancreatitis in Macaca nemestrina. J Med Primatol. 2022 Jun;51(3):134-142. doi: 10.1111/jmp.12577. Epub 2022 Mar 20. PMID: 35306662; PMCID: PMC9310849.

Caution: Product has not been fully validated for medical applications. For research use only.

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