Glycocholic acid sodium

Cat. No.: HY-N1423A

CAS No.: 863-57-0

Molecular Formula: $C_{26}H_{42}NNaO_6$

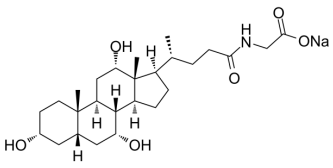
Molecular Weight: 487.6

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (205.09 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0509 mL	10.2543 mL	20.5086 mL
5 mM	0.4102 mL	2.0509 mL	4.1017 mL
10 mM	0.2051 mL	1.0254 mL	2.0509 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 (5.13 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.13 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.13 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Glycocholic acid sodium is an orally active bile acid with anticancer activity, targeting against pump resistance-related and non-pump resistance-related pathways[1].

IC_{50} & Target

Microbial Metabolite | Human Endogenous Metabolite

In Vitro

Glycocholic acid (GC) increases the cytotoxicity of epirubicin, significantly increases the intracellular accumulation of epirubicin in Caco-2 cells and the absorption of epirubicin in rat small intestine, and intensified epirubicin-induced apoptosis. Glycocholic acid and epirubicin significantly reduce mRNA expression levels of human intestinal MDR1, MDR-

associated protein (MRP)1, and MRP2; downregulate the MDR1 promoter region; suppress the mRNA expression of Bcl-2; induce the mRNA expression of Bax; and significantly increase the Bax-to-Bcl-2 ratio and the mRNA levels of p53, caspase-9 and -3. A combination of anticancer drugs with Glycocholic acid can control MDR via a mechanism that involves modulating P-gp and MRPs as well as regulating apoptosis-related pathways[1].

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

REFERENCES

[1]. Lo YL, et al. Inhibit multidrug resistance and induce apoptosis by using glycocholic acid and epirubicin. Eur J Pharm Sci. 2008 Sep 2;35(1-2):52-67.

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