Product Data Sheet

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.

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

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

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