(20S)-Protopanaxatriol

Cat. No.:	HY-N0835				
CAS No.:	34080-08-5				
Molecular Formula:	$C_{30}H_{52}O_4$				
Molecular Weight:	476.73				
Target:	Glucocorticoid Receptor; Estrogen Receptor/ERR; LXR; Apoptosis				
Pathway:	Immunology/Inflammation; Vitamin D Related/Nuclear Receptor; Metabolic HO Enzyme/Protease; Apoptosis				
Storage:	Powder	-20°C 4°C	3 years 2 years		
	In solvent	-80°C	6 months		
		-20 C	THIOHUI		



Inhibitors • Screening Libraries • Proteins

SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (209.76 mM; Need ultrasonic)						
		Solvent Mass Concentration	1 mg	5 mg	10 mg		
	Preparing Stock Solutions	1 mM	2.0976 mL	10.4881 mL	20.9762 mL		
		5 mM	0.4195 mL	2.0976 mL	4.1952 mL		
		10 mM	0.2098 mL	1.0488 mL	2.0976 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 (4.36 mM); Clear solution						
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.08 mg/mL (4.36 mM); Clear solution						
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (4.36 mM); Clear solution						

BIOLOGICAL ACTIV	
DIOLOGICALACITY	
Description	(20S)-Protopanaxatriol is a metabolite of ginsenoside. (20S)-Protopanaxatriol works through the glucocorticoid receptor (GR) and estrogen receptor (ER), and is also a LXRα inhibitor. (20S)-Protopanaxatriol shows a broad spectrum of antitumor effects ^{[1][2][3]} .
IC₅₀ & Target	Glucocorticoid receptor, Oestrogen receptor ^[1] ; $LXR\alpha^{[2]}$

In Vitro	(20S)-Protopanaxatriol works through the glucocorticoid receptor (GR) and oestrogen receptor (ER) in human umbilical vein endothelial cells (HUVECs). (20S)-Protopanaxatriol (PPT) increases [Ca ²⁺] _i with an EC ₅₀ of 482 nM in HUVECs. ((20S)- Protopanaxatriol (1 μM) elevates NO production via ERβ ^[1] . (20S)-Protopanaxatriol inhibits the autonomous transactivation of Gal4-LXRα LBD, the T0901317-dependent transcription of SREBP-1c and its promoter. (20S)-Protopanaxatriol (10 μg/mL) blocks the recruitment of RNA polymerase II to the LXRE region of SREBP-1c. (20S)-Protopanaxatriol also inhibits T0901317- dependent transcription of LXRα target genes related to lipogenesis, and reduces T0901317-induced cellular triglyceride (TG) accumulation in primary hepatocytes, but does not alter transcription of ABCA1, also an LXRα target gene ^[2] . Both In HCC827GR and H1975 cell lines, g-PPT (100?nM, 1?μM, 10?μM, 20?μM; 48 hours) results in SCD1 expression decreased [3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.				
In Vivo	(20S)-Protopanaxatriol (10?mg/kg; i.p.; daily for four weeks) synergizes with Gefitinib to inhibit xenograft growth ^[3] . (20S)-Protopanaxatriol (50-100 mg/kg; p.o.; 25 days; female BALB/c nude mice bearing breast cancer MCF-7 cell) inhibits the growth of MCF-7 breast cancer cells in a nude mice xenograft assay ^[4] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.				
	Animal Model:	H1975 murine xenograft tumor model			
	Dosage:	10 mg/kg			
	Administration:	I.p.; daily for four weeks			
	Result:	The combined g-PPT and Gefitinib (50 mg/kg/day) treatment clearly reduced p-EGFR and KI67 expression and increased c-Caspase3 expression compared to Gefitinib or g-PPT treatment alone.			

REFERENCES

[1]. Leung KW, et al. Protopanaxadiol and protopanaxatriol bind to glucocorticoid and oestrogen receptors in endothelial cells. Br J Pharmacol. 2009 Feb;156(4):626-37.

[2]. Oh GS, et al. 20(S)-protopanaxatriol inhibits liver X receptor α-mediated expression of lipogenic genes in hepatocytes. J Pharmacol Sci. 2015 Jun;128(2):71-7.

[3]. Huang Q, et al. Co-administration of 20(S)-protopanaxatriol (g-PPT) and EGFR-TKI overcomes EGFR-TKI resistance by decreasing SCD1 induced lipid accumulation in non-small cell lung cancer. J Exp Clin Cancer Res. 2019;38(1):129. Published 2019 Mar 15.

[4]. Zhang H, et al. 20(S)-Protopanaxadiol-Induced Apoptosis in MCF-7 Breast Cancer Cell Line through the Inhibition of PI3K/AKT/mTOR Signaling Pathway. Int J Mol Sci. 2018;19(4):1053. Published 2018 Apr 2.

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

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