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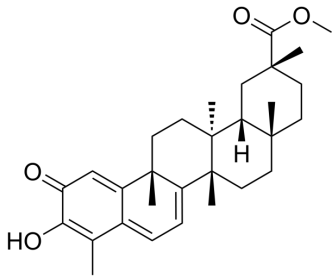
Pristimerin

Cat. No.:	HY-N1937
CAS No.:	1258-84-0
Molecular Formula:	$C_{30}H_{40}O_4$
Molecular Weight:	464.64
Target:	Bacterial
Pathway:	Anti-infection
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMF : 25 mg/mL (53.81 mM; Need ultrasonic)
DMSO : 20 mg/mL (43.04 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1522 mL	10.7610 mL	21.5220 mL
	5 mM	0.4304 mL	2.1522 mL	4.3044 mL
	10 mM	0.2152 mL	1.0761 mL	2.1522 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2 mg/mL (4.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pristimerin is a potent and reversible monoacylglycerol lipase (MGL) inhibitor with an IC_{50} of 93 nM.

IC_{50} & Target

IC50: 93 nM (MGL)[1]

In Vitro

Pristimerin inhibits the activity of purified MGL with an IC_{50} of 93±8 nM and that of non-purified MGL (cell lysates of MGL-transfected HeLa cells) with an IC_{50} of 398±68 nM. Pristimerin inhibits MGL through a mechanism that is rapid, reversible and non-competitive. The binding of pristimerin to MGL might be strengthened by formation of a polar interaction with a regulatory cysteine, possibly Cys^{208}[1]. Pristimerin inhibits HFLS-RA and HUVEC cell viability in a dose- and time-dependent manner. Pristimerin decreases VEGF-induced autophosphorylation of VEGFR2 and attenuates the activation of the VEGF-induced VEGFR2-mediated signaling pathway [2].

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

In Vivo	Pristimerin inhibits inflammation and tumor angiogenesis. Pristimerin significantly reduces vessel density in synovial membrane tissues of inflamed joints and reduces the expression of pro-angiogenic factors in sera, including TNF-α, Ang-1, and MMP-9[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	HFLS-RA (5×10^3 cells/mL) or HUVECs (1×10^4 cells/well) are seeded in 96-well plates and cultured in normal growth medium for 24 h. The cells are then incubated with different Pristimerin concentrations (0, 0.125, 0.25, 0.5 μM). The effects of Pristimerin on HUVECs viability are determined under VEGF-induced conditions. Cell viability is quantified by MTT assay. At 4 h before the end of the culture period, 30 μL of MTT solution (5.0 mg/mL) is added to each well. Cells without Pristimerin or VEGF served as a vehicle control[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration	Rat: Pristimerin is dissolved in DMSO (0.4%) and intraperitoneally injected daily into Male Sprague-Dawley rats in the experimental group (low-dose group, 0.40 mg/kg of body weight; high-dose group, 0.80 mg/kg of body weight) from day 11 to day 24 of immunization. The model group received vehicle (DMSO, 0.4%), and the normal control group received normal saline (NS). Methotrexate (positive control) is suspended in NS and orally administered in the autoimmune phase at an interval of 5 days[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Phytomedicine. 2021 Jan;80:153399.
- Int Immunopharmacol. 2021 Mar 23;94:107491.
- Drug Des Dev Ther. 2020 Oct 9;14:4189-4203.

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REFERENCES

[1]. King AR, et al. Discovery of potent and reversible monoacylglycerol lipase inhibitors. Chem Biol. 2009 Oct 30;16(10):1045-52.

[2]. Deng Q, et al. Pristimerin inhibits angiogenesis in adjuvant-induced arthritic rats by suppressing VEGFR2 signaling pathways. Int Immunopharmacol. 2015 Dec;29(2):302-13.

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