Alisol B

Cat. No.: HY-N0805A

CAS No.: 18649-93-9

Molecular Formula: $C_{30}H_{48}O_4$

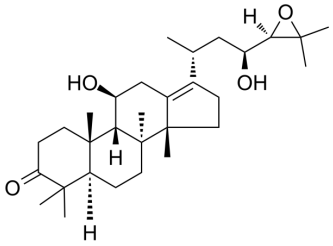
Molecular Weight: 472.7

Target: Others

Pathway: Others

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (211.55 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1155 mL	10.5775 mL	21.1551 mL
	5 mM	0.4231 mL	2.1155 mL	4.2310 mL
	10 mM	0.2116 mL	1.0578 mL	2.1155 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 (5.29 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.29 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.29 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Alisol B is a potentially novel therapeutic compound for bone disorders by targeting the differentiation of osteoclasts as well as their functions.IC50 Value:Target:In vitro: The in vitro cultured human renal tubular epithelial HK-2 cells were intervened with 5 ng/mL transforming growth factor-beta (TGF-beta), 0.1 micromol C3a, and 0.1 micromol C3a + 10 micromol alisol B, respectively. Exogenous C3a could induce renal tubular EMT. Alisol B was capable of suppressing C3a induced EMT [1]. Alisol-B strongly inhibited RANKL-induced osteoclast formation when added during the early stage of cultures, suggesting that alisol-B acts on osteoclast precursors to inhibit RANKL/RANK signaling. Among the RANK signaling pathways, alisol-B inhibited the phosphorylation of JNK, which are upregulated in response to RANKL in bone marrow macrophages, alisol-B also inhibited RANKL-induced expression of NFATc1 and c-Fos, which are key transcription factors for osteoclastogenesis. In addition, alisol-B suppressed the pit-forming activity and disrupted the actin ring formation of mature osteoclasts [2]. Alisol

B induced calcium mobilization from internal stores, leading to autophagy through the activation of the CaMKK-AMPK-mammalian target of rapamycin pathway. Moreover, the disruption of calcium homeostasis induces endoplasmic reticulum stress and unfolded protein responses in alisol B-treated cells, leading to apoptotic cell death. Finally, by computational virtual docking analysis and biochemical assays, it was showed that the molecular target of alisol B is the sarcoplasmic/endoplasmic reticulum Ca(2+) ATPase [3].In vivo:

REFERENCES

[1]. Zhang RF, et al.[Alisol B inhibited complement 3a-induced human renal tubular epithelial to mesenchymal transition]. Zhongguo Zhong Xi Yi Jie He Za Zhi. 2012 Oct;32(10):1407-12.

[2]. Lee JW, et al. Alisol-B, a novel phyto-steroid, suppresses the RANKL-induced osteoclast formation and prevents bone loss in mice. Biochem Pharmacol. 2010 Aug 1;80(3):352-61.

[3]. Law BY, et al. Alisol B, a novel inhibitor of the sarcoplasmic/endoplasmic reticulum Ca(2+) ATPase pump, induces autophagy, endoplasmic reticulum stress, and apoptosis. Mol Cancer Ther. 2010 Mar;9(3):718-30.

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