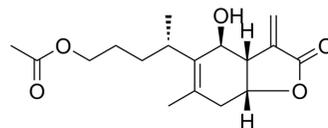


Inulicin

Cat. No.:	HY-N0896
CAS No.:	33627-41-7
Molecular Formula:	C ₁₇ H ₂₄ O ₅
Molecular Weight:	308.37
Target:	NF-κB; COX
Pathway:	NF-κB; Immunology/Inflammation
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 (324.29 mM; Need ultrasonic)
Ethanol : 50 mg/mL (162.14 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	3.2429 mL	16.2143 mL	32.4286 mL
	5 mM	0.6486 mL	3.2429 mL	6.4857 mL
	10 mM	0.3243 mL	1.6214 mL	3.2429 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution
- Add each solvent one by one: 10% EtOH >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (8.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Inulicin (1-O-Acetylbritannilactone) is an active compound that inhibits VEGF-mediated activation of Src and FAK. Inulicin (1-

	O-Acetylbritannilactone) inhibits LPS-induced PGE ₂ production and COX-2 expression, and NF-κB activation and translocation.	
IC₅₀ & Target	NF-κB	COX-2
In Vitro	<p>Inulicin (1-O-Acetylbritannilactone) inhibits angiogenesis and lung cancer cell growth through regulating VEGF-Src-FAK signaling. Inulicin dose-dependently inhibits vascular endothelial growth factor (VEGF)-induced proliferation, migration, and capillary structure formation of cultured human umbilical vascular endothelial cells (HUVECs). Treatment of A549 NSCLC cells with Inulicin results in cell growth inhibition and Src-FAK in-activation. The potential effect of Inulicin is tested on Src and FAK phosphorylation in A549 lung cancer cells. Significant high levels of Src and FAK phosphorylations are noticed in A549 cells, which are both inhibited by treatment of Inulicin (5 μM and 10 μM). Src and FAK are both important for cancer cell proliferation. Thus, A549 cell growth, tested by MTT assay and clonogenicity assay, is remarkably inhibited by corresponding Inulicin treatment. The anti-A549 cell growth activity of Inulicin is again dose-dependent^[1]. Inulicin (1-O-Acetylbritannilactone) isolated from <i>Inula britannica</i>-F., inhibits inflammatory responses in vascular smooth muscle cells (VSMCs). Inulicin (5, 10, 20 μM) has several concentration dependent effects, including inhibition of lipopolysaccharide (LPS)-induced PGE₂ production and COX-2 expression, and blockade of NF-κB activation and translocation. In addition, Inulicin directly inhibits the binding of active NF-κB to specific DNA cis-element^[2].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>	
In Vivo	<p>Administration of a single dose of Inulicin (1-O-Acetylbritannilactone; 12 mg/kg/day) remarkably suppresses growth of A549 xenografts in nude mice. In vivo microvessels formation and Src activation are also significantly inhibited in Inulicin-treated xenograft tumors. To investigate the potential activity of Inulicin in vivo, a nude mice xenograft model is applied. A single dose of Inulicin (12 mg/kg/day, i.p.) dramatically inhibits the growth of A549 xenografts in nude mice. Further, the weights of Inulicin-treated tumors are remarkably lighter than that of vehicle-treated tumors. Notably, Inulicin administration does not affect mice body weights^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>	

PROTOCOL

Cell Assay ^[1]	<p>HUVECs or A549 cells are plated in 60 mm plates (300 cells/plate). After overnight incubation, cells are treated with applied agents (e.g., Inulicin; 5 μM and 10 μM) for 24 h. Cells are then washed, and fresh media are added. After 10 days of incubation, surviving colonies are fixed, stained, and manually counted^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[1]	<p>Mice^[1]</p> <p>Male nude mice (4-6weeks old, BALB/c) are used. A549 cells (five million cells in 0.1 mL of culture medium) are subcutaneously injected at the right thigh of nude mice, and treatment is started when the tumors reach an average volume about 100 mm³. Animals are randomized into two groups with 10 mice per group: (a) Vehicle; (b) 12 mg/kg of Inulicin. Inulicin is injected intraperitoneally (i.p.) daily. The mice are examined daily for toxicity/mortality relevant to treatment, and the tumor is measured with a caliper every two days. The tumor volume (in mm³) is calculated, and the tumor growth curve is presented. At the end of experiments, xenograft tumors are isolated through surgery and weighted^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

CUSTOMER VALIDATION

- Phytomedicine. 2019 May;58:152754.

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REFERENCES

- [1]. Zhengfu H, et al. 1-o-acetylbritannilactone (ABL) inhibits angiogenesis and lung cancer cell growth through regulating VEGF-Src-FAK signaling. *Biochem Biophys Res Commun*. 2015 Aug 21;464(2):422-7.
- [2]. Liu YP, et al. Acetylbritannilactone suppresses lipopolysaccharide-induced vascular smooth muscle cell inflammatory response. *Eur J Pharmacol*. 2007 Dec 22;577(1-3):28-34.
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Caution: Product has not been fully validated for medical applications. For research use only.

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