

Product Data Sheet

Product Name: Levistilide A

Cat. No.: GN10044

Chemical Properties

Cas. No. 88182-33-6

Chemical Name (1Z,5S,5aS,8Z,10bS,10cS)-1,8-dibutylidene-5,5a,6,7-tetrahydro-1H-5,10c-ethanonaphtho[1,2-c:7,8-c']difuran-3,10(8H,10bH)-dione

SMILES O=C(O/C1=C\CCC)C2=C1CC[C@]3([H])[C@@]2([H])[C@@]45C(C(O/C4=C\CCC)=O)=C[C@]3(CC5)[H]

Formula C₂₄H₂₈O₄

M.Wt 380

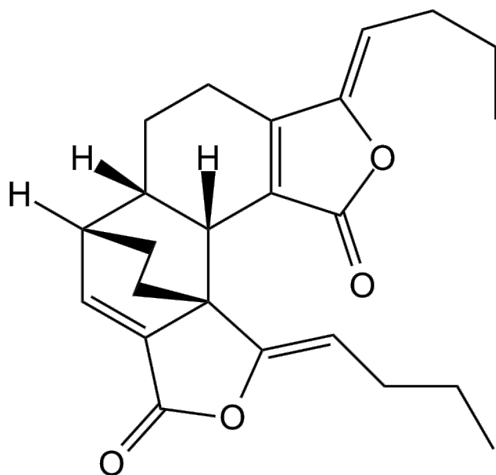
Solubility ≥ 38mg/mL in DMSO

Storage Store at 2-8°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines	BV-2 cells (mouse microglial cell line) and primary microglial cells
Preparation Method	Cells were stimulated with lipopolysaccharide (LPS) at 200ng/ml to 1µg/ml and treated with Levistilide A.
Reaction Conditions	1 to 10µM; 24 hours
Applications	Levistilide A significantly inhibited the production of pro-inflammatory cytokines (NO, IL-6, TNF-α) and the expression of iNOS, IL-6, TNF-α, and IL-1β mRNA, reduced ROS production, suppressed IKB-α phosphorylation and NF-κB p65 nuclear translocation, reversed glucose metabolism reprogramming (decreased glycolysis and increased oxidative phosphorylation), inhibited glucose uptake and downregulated glycolysis enzymes (GLUT-1, PKM2, HK-1, HK-2), modulated the AMPK/mTOR pathway (increased AMPK phosphorylation and decreased mTOR phosphorylation).
Animal experiment [2]:	
Animal models	Wistar rats
Preparation Method	Rats were subcutaneously injected with CCl ₄ twice a week for six weeks to duplicate the liver fibrosis model and then treated with Levistilide A from the fourth week to the end of the experiment.
Dosage form	3mg/kg and 6mg/kg; intraperitoneal injection; daily for three weeks

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Applications

Levistilide A ameliorated serum liver function (reversed the increase of ALT and AST and the decrease of Alb), alleviated liver fibrosis (reduced collagen deposition, decreased hepatic hydroxyproline content, and downregulated the expression of collagen I and α -SMA), and inhibited angiogenesis (reduced the number of new microvessels and vWF expression, partly recovered sinusoidal fenestration, and downregulated the expression of CD31, VEGF, and VEGF-R2).

References:

- [1] Zhang M, Duan C, Lin W, et al. Levistilide A Exerts a Neuroprotective Effect by Suppressing Glucose Metabolism Reprogramming and Preventing Microglia Polarization Shift: Implications for Parkinson's Disease. *Molecules*. 2024 Feb 19;29(4):912.
- [2] Zhao ZM, Liu HL, Sun X, et al. Levistilide A inhibits angiogenesis in liver fibrosis via vascular endothelial growth factor signaling pathway. *Exp Biol Med (Maywood)*. 2017 May;242(9):974-985.

Background

Levistilide A is a natural phthalide compound extracted from the traditional Chinese medicine *Ligusticum chuanxiong*, possessing multiple biological activities. Levistilide A can induce cancer cell apoptosis by activating PPAR γ and inhibiting the GSK3 α/β pathway, as well as promoting ROS generation. Levistilide A can be used in research related to Alzheimer's disease, tumors, and kidney diseases^[1-4].

In vitro, Levistilide A (0–80 μ M) was used to treat MDA-MB-231 and MCF-7 breast cancer cells for 24–48 hours. Levistilide A significantly reduced cell viability and increased lactate dehydrogenase release in a dose-dependent manner, promoting cell damage and death. Levistilide A treatment significantly inhibited GPX4 protein expression while upregulating the mRNA expression of SLC7A11, SLC3A2, GCLC, GCLM, GSS, TFRC, SLC11A2, and NCOA4. Levistilide A treatment markedly increased the protein and mRNA expression levels of Nrf2 and HO-1^[5]. Levistilide A (1–10 μ M) was co-treated with LPS (200ng/ml–1 μ g/ml) in BV-2 microglial cells and mouse primary microglial cells for 24 hours. Levistilide A significantly inhibited the expression of pro-inflammatory factors NO, IL-6, and TNF- α , reducing the inflammatory response^[6].

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In vivo, Levistilide A (3mg/kg and 6mg/kg; once daily) was intraperitoneally injected into CCl₄-induced liver fibrosis Wistar rats for three weeks. Levistilide A improved the rats' serum liver function, alleviated liver fibrosis, and inhibited angiogenesis^[7]. Levistilide A (20mg/kg; once daily) was administered orally to lauric acid-induced thromboangiitis obliterans Wistar rats, starting two days before surgery and continuing for 16 days. Levistilide A improved blood flow, reduced muscle inflammatory necrosis, and decreased serum levels of IL-1 β , IL-6, and CRP, as well as NLRP3 expression^[8].

References:

- [1] Yang Y, Zhang Y, Wang L, et al. Levistolide A Induces Apoptosis via ROS-Mediated ER Stress Pathway in Colon Cancer Cells. *Cell Physiol Biochem*. 2017;42(3):929-938.
- [2] Qu X, Guan P, Han L, et al. Levistolide A Attenuates Alzheimer's Pathology Through Activation of the PPAR γ Pathway. *Neurotherapeutics*. 2021 Jan;18(1):326-339.
- [3] Zeng W, Ren J, Li Z, et al. Levistolide A Inhibits PEDV Replication via Inducing ROS Generation. *Viruses*. 2022 Jan 27;14(2):258.
- [4] Li S, Zhao W, Zhao Z, et al. Levistilide A reverses rat hepatic fibrosis by suppressing angiotensin II-induced hepatic stellate cells activation. *Mol Med Rep*. 2020 Sep;22(3):2191-2198.
- [5] Jing S, Lu Y, Zhang J, Ren Y, et al. Levistilide a Induces Ferroptosis by Activating the Nrf2/HO-1 Signaling Pathway in Breast Cancer Cells. *Drug Des Devel Ther*. 2022 Sep 7;16:2981-2993.
- [6] Zhang M, Duan C, Lin W, et al. Levistilide A Exerts a Neuroprotective Effect by Suppressing Glucose Metabolism Reprogramming and Preventing Microglia Polarization Shift: Implications for Parkinson's Disease. *Molecules*. 2024 Feb 19;29(4):912.
- [7] Zhao ZM, Liu HL, Sun X, et al. Levistilide A inhibits angiogenesis in liver fibrosis via vascular endothelial growth factor signaling pathway. *Exp Biol Med (Maywood)*. 2017 May;242(9):974-985.
- [8] Guo H, Sun L, Ling S, et al. Levistilide A Ameliorates NLRP3 Expression Involving the Syk-p38/JNK Pathway and Peripheral Obliterans in Rats. *Mediators Inflamm*. 2018 Aug 12;2018:7304096.

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