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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Dehydroabietic acid

Cat. No.: HY-N6869

CAS No.: 1740-19-8

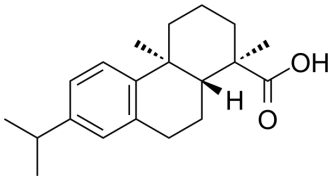
Molecular Formula: $C_{20}H_{28}O_2$

Molecular Weight: 300.44

Target: Antibiotic; PPAR; Bacterial; Fungal

Pathway: Anti-infection; Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor

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 (332.85 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3285 mL	16.6423 mL	33.2845 mL
	5 mM	0.6657 mL	3.3285 mL	6.6569 mL
	10 mM	0.3328 mL	1.6642 mL	3.3285 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 (8.32 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 (8.32 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dehydroabietic acid is a diterpene resin acid that can be isolated from Pinus and Picea. Dehydroabietic acid has anti-bacterial, anti-fungal, anti-inflammatory, and anticancer activities. Dehydroabietic acid is a dual PPAR-α/γ agonist and PPAR-γ partial agonist, which can attenuate insulin resistance (IR) and hepatic steatosis induced by HFD-consumption in mice[1][2].

In Vitro

Dehydroabietic acid (0-100 μM, 30 min) decreases NO production in RAW264.7 cells[1].
Dehydroabietic acid (0-100 μM, 6 h) reduces the mRNA expression levels of inflammatory mediators including inducible nitric oxide (iNOS) and TNF-α in RAW264.7 cells[1].

Dehydroabietic acid (0-100 μM, 24 h) reduces the MyD88-induced NF-κB and AP-1 transcriptional activities in HEK293T cells [1].

Dehydroabietic acid (100 μM, 24 h) inactivates both Src and Syk kinases in Src- or Syk-overexpressing HEK293T cells[1].

Dehydroabietic acid (2.5-15 μM, 4 days) promotes 3T3-L1 adipocyte differentiation in a dose-dependent manner[2].

Dehydroabietic acid (10 μM, 0-6 days) increases mRNA expression of PPAR-γ target genes (Glut-4 and Cyp4a10) in 3T3-L1 cells[2].

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

Western Blot Analysis[1]

Cell Line:	AW264.7 cells
Concentration:	100 μM
Incubation Time:	5-60 min, 120-240 min
Result:	Blocked the phosphorylation of IκBα at 5 and 15 min. Reduced c-Jun N-terminal kinase (JNK) phosphorylation. Reduced phosphorylated levels of mitogen-activated protein kinase kinase 4 (MKK4) and MKK7 at 60 mim. Decreased the phosphorylation of MKK4 and MKK7 and their downstream protein JNK at 120 and 240 min.

In Vivo

Dehydroabietic acid (10-20 mg/kg, i.g., daily, 9 weeks) alleviates HFD-induced hepatic steatosis and inflammation in HFD mice[2].

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

Animal Model:	HFD mice[2]
Dosage:	10-20 mg/kg
Administration:	i.g., daily, 9 weeks
Result:	Decreased the levels of liver injury markers, ALT and AST. Decreased blood TG, TC and LDL-c levels and increased the HDL-c levels. Activated PPAR-α and its target gene which were reduced by HFD, including acyl-Coenzyme A dehydrogenase, C-4 to C-12 straight chain and CPT1α. Decreased mRNA expression of inflammatory factors commonly involved in liver diseases and injury (IL-1β, IL-6, TNF-α, COX-1 and COX-2). Upregulated mRNA expression of PPAR-γ, Glut-4, Adipor, FSP27, ACOX-1, FABP4, Adiponectin.

REFERENCES

[1]. Kim E, et al. Dehydroabietic Acid Suppresses Inflammatory Response Via Suppression of Src-, Syk-, and TAK1-Mediated Pathways. Int J Mol Sci. 2019 Mar 29;20(7):1593.

[2]. Xie Z, et al. Dehydroabietic acid alleviates high fat diet-induced insulin resistance and hepatic steatosis through dual activation of PPAR-γ and PPAR-α. Biomed Pharmacother. 2020 Jul;127:110155.

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

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