Gnetol

Cat. No.: HY-126052

CAS No.: 86361-55-9

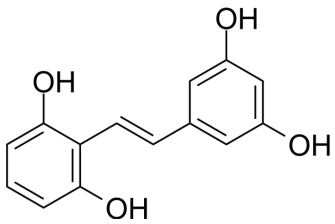
Molecular Formula: $C_{14}H_{12}O_4$

Molecular Weight: 244.24

Target: Tyrosinase; COX; HDAC

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; Cell Cycle/DNA Damage; Epigenetics

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 (409.43 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0943 mL	20.4717 mL	40.9433 mL
	5 mM	0.8189 mL	4.0943 mL	8.1887 mL
	10 mM	0.4094 mL	2.0472 mL	4.0943 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 (10.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.24 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Gnetol is a phenolic compound isolated from the root of Gnetum montanum . Gnetol potently inhibits COX-1 (IC_{50} of 0.78 μM) and HDAC. Gnetol is a potent tyrosinase inhibitor with an IC_{50} of 4.5 μM for murine tyrosinase and suppresses melanin biosynthesis. Gnetol has antioxidant, antiproliferative, anticancer and hepatoprotective activity. Gnetol also possesses concentration-dependent α-Amylase, α-glucosidase, and adipogenesis activities[1][2][3].

IC_{50} & Target			
	COX-1 0.78 μM (IC_{50})	Tyrosinase 4.5 μM (IC_{50})	HDAC

In Vitro	The antiproliferative activities of Gnetol are tested in HCT-116, Hep-G2, MDA-MB-231, and PC-3 cell lines by measuring cell viability after treatment with 4.1 μM, 40.9 μM, 204.7 μM, 409.4 μM, and 1023.6 μM. Gnetol shows concentration-dependent reductions in cell viability in cancer cell lines with greatest activity in colorectal cancer[1]. Gnetol at 200 μg/mL significantly offers the highest protection of 54.3% against the toxicant. A lower dose of Gnetol (50 μg/mL) also shields the cell line from the toxic effects of CCl_4[3]. The ligand molecule TGF-β and PPARα protein show that Gnetol has the binding affinity of 7.0 and 8.4, respectively[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Male Sprague-Dawley rats were cannulated and dosed either intravenously with Gnetol (10 μg/kg) or orally (100 mg/kg). After oral and intravenous administration, Gnetol is detected in both serum and urine as the parent compound and as a glucuronidated metabolite. The bioavailability of Gnetol is determined to be 6%. Gnetol is rapidly glucuronidated and is excreted in urine and via nonrenal routes[1]. Pretreatment of Male NIH Swiss mice (20-35 g) with Gnetol (50mg/kg, SC) is able to increase the latency period to response in analgesia models[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Remsberg CM, et al. Preclinical Pharmacokinetics and Pharmacodynamics and Content Analysis of Gnetol in Foodstuffs. Phytother Res. 2015 Aug;29(8):1168-79.

[2]. Ohguchi K, et al. Gnetol as a potent tyrosinase inhibitor from genus Gnetum. Biosci Biotechnol Biochem. 2003 Mar;67(3):663-5.

[3]. Jinadatta P, et al. In silico, in vitro: antioxidant and antihepatotoxic activity of gnetol from Gnetum ula Brongn. Bioimpacts. 2019;9(4):239-249.

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