Reticuline

Cat. No.: HY-N1356

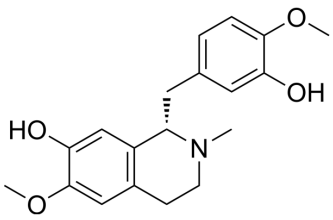
CAS No.: 485-19-8

Molecular Formula: $C_{19}H_{23}NO_4$

Molecular Weight: 329.39

Target: JAK; STAT; NF-κB; Endogenous Metabolite

Pathway: Epigenetics; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Stem Cell/Wnt; NF-κB; Metabolic Enzyme/Protease



Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (303.59 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0359 mL	15.1796 mL	30.3591 mL
	5 mM	0.6072 mL	3.0359 mL	6.0718 mL
	10 mM	0.3036 mL	1.5180 mL	3.0359 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 (7.59 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.59 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Reticuline shows anti-inflammatory effects through JAK2/STAT3 and NF-κB signaling pathways. Reticuline inhibits mRNA expressions of TNF-α, and IL-6 and reduces the phosphorylation levels of JAK2 and STAT3[1]. Reticuline exhibits cardiovascular effects[2].

IC_{50} & Target				
	JAK2	STAT3	NF-κB	Human Endogenous Metabolite

In Vitro

Reticuline (3 μM, 30 μM, 300 μM, 900 μM and 1.5 mM) inhibits in a concentration-dependent manner the contractions induced by Phenylephrine (1 μM), KCl (80 mM) and KCl (30 mM), (IC_{50}=40±10, 240±40 and 300±40 μM, respectively) in isolated

	rat aortic rings with intact endothelium[2]. Reticuline (3 μM, 30 μM, 300 μM, 900 μM and 1.5 mM) antagonizes $CaCl_2$-induced contractions, and also inhibits the intracellular calcium dependent transient contractions induced by Norepinephrine (1μM), but not those induced by Caffeine (20 mM)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Reticuline (5, 10 and 20 mg/kg, i. v., randomly) injections produced an intense hypotension in normotensive rats. The hypotensive effect of Reticuline is probably due to a peripheral vasodilation in consequence of: 1) muscarinic stimulation and NOS activation in the vascular endothelium, 2) voltage-dependent Ca^{2+} channel blockade and/or 3) inhibition of Ca^{2+} release from norepinephrine-sensitive intracellular stores[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Yang X, et al. Anti-Inflammatory Effects of Boldine and Reticuline Isolated from Litsea cubeba through JAK2/STAT3 and NF-κB Signaling Pathways. Planta Med. 2018 Jan;84(1):20-25.

[2]. Katy Lísias Dias, et al. Cardiovascular effects induced by reticuline in normotensive rats. Planta Med. 2004 Apr;70(4):328-33.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA