Ajmalicine

Cat. No.: HY-N1919

CAS No.: 483-04-5

Molecular Formula: $C_{21}H_{24}N_2O_3$

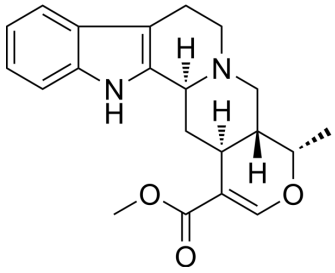
Molecular Weight: 352.43

Target: Adrenergic Receptor; Cholinesterase (ChE)

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5.56 mg/mL (15.78 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8374 mL	14.1872 mL	28.3744 mL
	5 mM	0.5675 mL	2.8374 mL	5.6749 mL
	10 mM	0.2837 mL	1.4187 mL	2.8374 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: ≥ 0.56 mg/mL (1.59 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.56 mg/mL (1.59 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ajmalicine (Raubasine) is a potent adrenolytic agent which preferentially blocks α_1-adrenoceptor. Ajmalicine is an reversible but non-competitive nicotine receptor full inhibitor, with an IC_{50} of 72.3 μM. Ajmalicine also can be used as anti-hypertensive, and serpentine, with sedative activity[1][2].

IC_{50} & Target

α1-adrenergic receptor	α2-adrenergic receptor

In Vitro

Ajmalicine preferentially blocks α_1-adrenoceptor than α_2-adrenoceptor[1].
Ajmalicine inhibits contractions in a concentration-dependent manner (IC_{50}=72.3 ± 22.5 μM)[2].
Ajmalicine acts preferentially at postsynaptic sites, competitively antagonizes the effect of noradrenaline on postsynaptic alpha-adrenoceptor with a pA2 value of 6.57, blocks the inhibitory effect of clonidine with an pA2 value of 6.2[3].

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

In Vivo

Ajmalicine blocking the pressor action of electrical stimulation and is active against sympathetic stimulation[1].
Ajmalicine (0.5-4 mg/kg) induces a marked dose-dependent inhibition against the pressor response to noradrenaline[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats (300-350 g)[1]
Dosage:	0.5, 1, 2, and 4 mg/kg
Administration:	IV, once
Result:	Induced a marked dose-dependent inhibition against the pressor response to noradrenaline.

CUSTOMER VALIDATION

- J Biochem Mol Toxicol. 2023 Dec 8:e23614.

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REFERENCES

[1]. Roquebert J, et al. Inhibition of the alpha 1 and alpha 2-adrenoceptor-mediated pressor response in pithed rats by raubasine, tetrahydroalstonine and akuammigine. Eur J Pharmacol. 1984 Oct 30;106(1):203-5.

[2]. Pereira DM, et al. Pharmacological effects of Catharanthus roseus root alkaloids in acetylcholinesterase inhibition and cholinergic neurotransmission. Phytomedicine. 2010 Jul;17(8-9):646-52.

[3]. Demichel P, et al. Effects of raubasine stereoisomers on pre- and postsynaptic alpha-adrenoceptors in the rat vas deferens. Br J Pharmacol. 1984 Oct;83(2):505-10

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