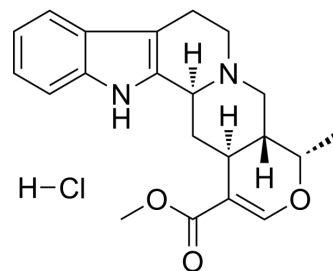


## Ajmalicine hydrochloride

<b>Cat. No.:</b>	HY-N1919A
<b>CAS No.:</b>	4373-34-6
<b>Molecular Formula:</b>	C <sub>21</sub> H <sub>25</sub> ClN <sub>2</sub> O <sub>3</sub>
<b>Molecular Weight:</b>	388.89
<b>Target:</b>	Adrenergic Receptor; Cholinesterase (ChE)
<b>Pathway:</b>	GPCR/G Protein; Neuronal Signaling
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Ajmalicine (Raubasine) hydrochloride is a potent adrenergic agent which preferentially blocks $\alpha_1$ -adrenoceptor. Ajmalicine hydrochloride is a reversible but non-competitive nicotine receptor full inhibitor, with an $IC_{50}$ of 72.3 $\mu$ M. Ajmalicine hydrochloride also can be used as anti-hypertensive, and serpentine, with sedative activity <sup>[1][2]</sup> .									
<b>IC<sub>50</sub> &amp; Target</b>	$\alpha_1$ -adrenergic receptor	$\alpha_2$ -adrenergic receptor								
<b>In Vitro</b>	<p>Ajmalicine hydrochloride preferentially blocks <math>\alpha_1</math>-adrenoceptor than <math>\alpha_2</math>-adrenoceptor<sup>[1]</sup>.</p> <p>Ajmalicine hydrochloride inhibits contractions in a concentration-dependent manner (<math>IC_{50}=72.3 \pm 22.5 \mu</math>M)<sup>[2]</sup>.</p> <p>Ajmalicine hydrochloride 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 a pA2 value of 6.2<sup>[3]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>									
<b>In Vivo</b>	<p>Ajmalicine hydrochloride blocking the pressor action of electrical stimulation and is active against sympathetic stimulation<sup>[1]</sup>.</p> <p>Ajmalicine hydrochloride (0.5-4 mg/kg) induces a marked dose-dependent inhibition against the pressor response to noradrenaline<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 30%;">Animal Model:</td> <td>Male Wistar rats (300-350 g)<sup>[1]</sup></td> </tr> <tr> <td>Dosage:</td> <td>0.5, 1, 2, and 4 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>IV, once</td> </tr> <tr> <td>Result:</td> <td>Induced a marked dose-dependent inhibition against the pressor response to noradrenaline.</td> </tr> </table>		Animal Model:	Male Wistar rats (300-350 g) <sup>[1]</sup>	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.
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Result:	Induced a marked dose-dependent inhibition against the pressor response to noradrenaline.									

### 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.

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

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