Alvameline

Cat. No.: HY-101586

CAS No.: 120241-31-8

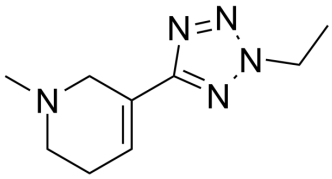
Molecular Formula: $C_9H_{15}N_5$

Molecular Weight: 193.25

Target: mAChR

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Alvameline (Lu25-109) is a partial M1 agonist and M2/M3 antagonist.
In Vitro	Alvameline is metabolized by human liver microsomes to Lu 31-126 mainly by CYP2D6; to Lu 29-297 and Lu 25-077 mainly by CYP1A2, CYP2A6, CYP2C19, and CYP3A4; and to Lu 32-181 by CYP1A2 and possibly by CYP2C19. One metabolite, Lu 32-181, could be reduced back to alvameline, a reaction not inhibited by the applied cytochrome P-450 inhibitors[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Alvameline competitively and effectively antagonizes carbachol-induced contractions and contractions induced by electrical field stimulation in human detrusor muscle. Alvameline produces a concentration-dependent rightward shift of the concentration-response curves for carbachol in both human and pig detrusor, the pK_b values being 6.2 and 5.8. Contractions induced by electrical field stimulation in human detrusor are almost completely inhibited by 100 μM alvameline. In contrast, electrical field stimulation-induced contractions in pig detrusor are less sensitive to alvameline, resulting in a final inhibition of 32% with the highest concentration used (100 μM)[2]. Alvameline has been shown to improve cognitivefunction following traumatic brain injury in rats. Alvameline treated rats causes a 13% and 5% decrease in the medial septal nucleus, a 48 and 23% decrease in the vertical limb nucleus of the diagonal band, and a 51 and 28% decrease in the nucleus basalis magnocellularis, respectively[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [2]	Rats: Treatment with alvameline is initiated 24 h following TBI and rats are injected (sc) once daily for the first 15 days after injury or sham injury. Injured rats are injected daily with either saline or 15 μmol/kg of alvameline. Sham-injured rats are injected (sc) daily with either saline or 15 μmol/kg of alvameline-T[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Jensen KG, et al. In vitro metabolism of the M1-muscarinic agonist 5-(2-ethyl-2H-tetrazol-5-yl)-1-methyl-1,2,3,6-tetrahydropyridine by human hepatic cytochromes P-450 determined at pH 7.4 and 8.5. Drug Metab Dispos. 1999 Jan;27(1):125-32.

[2]. Waldeck K, et al. Actions of the new antimuscarinic compound Alvameline on isolated human and pig detrusor. Neurourol Urodyn. 2002;21(1):92-8.

[3]. Pike BR, et al. Chronic administration of a partial muscarinic M1 receptor agonist attenuates decreases in forebrain choline acetyltransferase immunoreactivity following experimental brain trauma. Exp Neurol. 1997 Sep;147(1):55-65.

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