Methoctramine tetrahydrochloride

Cat. No.: HY-116294A

CAS No.: 104807-46-7

Molecular Formula: $C_{36}H_{66}Cl_4N_4O_2$

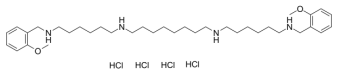
Molecular Weight: 728.75

Target: mAChR

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: -20°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



BIOLOGICAL ACTIVITY

Description	Methoctramine tetrahydrochloride is a potent and cardioselectivity antagonist of M2 muscarinic receptor. Methoctramine tetrahydrochloride can inhibit Muscarine-induced bradycardia in vivo[1][2][3].
IC_{50} & Target	M2 muscarinic receptor[1]
In Vitro	Methoctramine tetrahydrochloride attenuates Acetylcholine (ACh)- and Arecaidine propargyl ester (APE)-induced increases in PG synthesis in a concentration-dependent manner[1]. Methoctramine (0.01-1 μM) tetrahydrochloride causes facilitation of contractions induced by both pre- and postganglionic nerve stimulation in the guinea-pig isolated, innervated tracheal tube preparation[2]. Methoctramine (≥10 μM) tetrahydrochloride reduces responses to both nerve stimulation and exogenous ACh[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Methoctramine (300 μg/kg; i.v.) tetrahydrochloride strongly inhibits the Methacholine- and Muscarine-induced bradycardia in the anaesthetized rat, respectively[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Jaiswal N, et, al. Methoctramine, a cardioselective antagonist: muscarinic receptor mediating prostaglandin synthesis in isolated rabbit heart. Eur J Pharmacol. 1991 Jan 3;192(1):63-70.

[2]. Watson N, et, al. Actions of methoctramine, a muscarinic M2 receptor antagonist, on muscarinic and nicotinic cholinoceptors in guinea-pig airways in vivo and in vitro. Br J Pharmacol. 1992 Jan;105(1):107-12.

[3]. Wess J, et, al. Methoctramine selectively blocks cardiac muscarinic M2 receptors in vivo. Naunyn Schmiedebergs Arch Pharmacol. 1988 Sep;338(3):246-9.

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