Adiphenine hydrochloride

Cat. No.: HY-B0379A

CAS No.: 50-42-0

Molecular Formula: $C_{20}H_{26}ClNO_2$

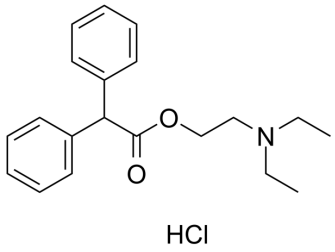
Molecular Weight: 347.88

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (287.46 mM)

H_2O : ≥ 50 mg/mL (143.73 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8746 mL	14.3728 mL	28.7455 mL
	5 mM	0.5749 mL	2.8746 mL	5.7491 mL
	10 mM	0.2875 mL	1.4373 mL	2.8746 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: ≥ 3.25 mg/mL (9.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3.25 mg/mL (9.34 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3.25 mg/mL (9.34 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Adiphenine hydrochloride is a non-competitive inhibitor of nicotinic acetylcholine receptor (nAChR), with an IC_{50}s of 1.9, 1.8, 3.7, and 6.3 μM for α1, α3β4, α4β2, and α4β4, respectively. Adiphenine hydrochloride has anticonvulsant effects[1][2][3].

IC_{50} & Target

IC50: 1.9 μM (α1 nAChR), 1.8 μM (α3β4 nAChR), 3.7 μM (α4β2 nAChR), 6.3 μM (α4β4 nAChR)[1]

In Vitro

Adiphenine (10 nM-1 mM; 3 min) blocks the function of α1*-nAChR in a dose-dependent manner in TE671/RD cells, with an IC_{50} of 1.9 μM[1].

	Adiphenine (10 nM-1 mM; 3 min) blocks the function of α3α4*-nAChR in a dose-dependent manner in SH-SY5Y cells, with an IC_{50} of 1.8 μM[1]. Adiphenine (10 nM-1 mM; 3 min) blocks the function of α4β2- and α4β4-nAChR in a dose-dependent manner in SH-EP1 cells, with IC_{50}s of 3.7 and 6.3 μM, respectively[1]. Adiphenine (50-200 μM; 30-60 s) decreases the frequency of ACh-induced single-channel currents in HEK 293 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Adiphenine (i.p.) prevents the hindleg tonic-extensor component of maximal electroshock seizures (MES), with an ED_{50} of 62 mg/kg in mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Spitzmaul, G., et al., The local anaesthetics proadifen and adiphenine inhibit nicotinic receptors by different molecular mechanisms. Br J Pharmacol, 2009. 157(5): p. 804-17.

[2]. Michelot, J., et al., Adiphenine plasma levels and blood-brain barrier crossing in the rat. Eur J Drug Metab Pharmacokinet, 1985. 10(4): p. 273-8.

[3]. Gentry CL, et, al. Local anesthetics noncompetitively inhibit function of four distinct nicotinic acetylcholine receptor subtypes. J Pharmacol Exp Ther. 2001 Dec;299(3):1038-48.

[4]. TANAKA K, et, al. Anticonvulsant properties of procaine, cocaine, adiphenine and related structures. Proc Soc Exp Biol Med. 1955 Oct;90(1):192-5.

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