Blarcamesine hydrochloride

Cat. No.: HY-101864

CAS No.: 195615-84-0

Molecular Formula: $C_{19}H_{24}ClNO$

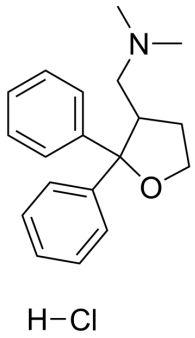
Molecular Weight: 317.85

Target: Sigma Receptor

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

H_2O : 100 mg/mL (314.61 mM; Need ultrasonic)
DMSO : 25 mg/mL (78.65 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1461 mL	15.7307 mL	31.4614 mL
5 mM	0.6292 mL	3.1461 mL	6.2923 mL
10 mM	0.3146 mL	1.5731 mL	3.1461 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: ≥ 2.5 mg/mL (7.87 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.87 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.87 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Blarcamesine hydrochloride is a Sigma-1 Receptor agonist with an IC_{50} of 860 nM.

IC_{50} & Target

IC50: 860 nM (Sigma-1 Receptor)[1]

In Vivo

The pre-administration of Blarcamesine leads to a dose-dependent attenuation of the scopolamine induced alternation deficit, significant at 1 and 3 mg/kg. The pre-treatment with Blarcamesine hydrochloride attenuates the impairments of

step-through latency, dose dependently and significantly at doses higher than 0.3 mg/kg[1]. The Blarcamesine hydrochloride treatment dose-dependently blocks the recognition memory deficit, with a significant effect measured at 1 mg/kg. One day after injections, the significant $A\beta_{25-35}$-induced decrease in Akt phosphorylation is significantly attenuated by Blarcamesine hydrochloride at 0.1 and 1 mg/kg dose. Seven days after injections, Blarcamesine hydrochloride attenuates the decrease in Ser^9 phosphorylation induced by the peptide at 0.3 and 1 mg/kg. The Blarcamesine hydrochloride treatment dose-dependently prevents the $A\beta_{25-35}$-induced increase in $A\beta_{1-42}$ content, with a significant effect at the highest dose tested[2].

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

PROTOCOL

Animal Administration [2]

Male mice aged 7-9 weeks and weighing 32±2 g are used. Drugs (including Blarcamesine hydrochloride) are brought up to each dose by dilution and injected in a volume of 100 μL/20 g body weight. Animals are used between days 1 and 9 after i.c.v. injections for behavioral testing or killed before biochemical measures[2].

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

REFERENCES

[1]. Villard V, et al. Anti-amnesic and neuroprotective potentials of the mixed muscarinic receptor/sigma 1 (σ1) ligand ANAVEX2-73, a novel aminotetrahydrofuran derivative. J Psychopharmacol. 2011 Aug;25(8):1101-17.

[2]. Valentine Lahmy, et al. Blockade of Tau Hyperphosphorylation and Aβ1-42 Generation by the Aminotetrahydrofuran Derivative ANAVEX2-73, a Mixed Muscarinic andσ1 Receptor Agonist, in a Nontransgenic Mouse Model of Alzheimer's Disease. Neuropsychopharmacology. 2013 Aug; 38(9): 1706-1723.

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