ABT-107

Cat. No.: HY-108038

CAS No.: 855291-54-2

Molecular Formula: $C_{19}H_{20}N_4O$

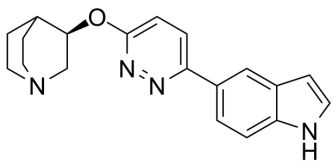
Molecular Weight: 320.39

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; 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

DMSO : 100 mg/mL (312.12 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1212 mL	15.6060 mL	31.2120 mL
5 mM	0.6242 mL	3.1212 mL	6.2424 mL
10 mM	0.3121 mL	1.5606 mL	3.1212 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 (7.80 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.80 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.80 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ABT-107 is a selective α7 neuronal nicotinic receptor agonist. ABT-107 protects against nigrostriatal damage in rats with unilateral 6-hydroxydopamine lesions[1][2].

In Vivo

ABT-107 exhibits good bioavailability in mouse (orally, 51.1%; intraperitoneally,100%), rat (orally, 81.2%; intraperitoneally, 100.0%), and monkey (orally, 40.6%; intramuscularly, 100%), and good CNS penetration in rodents with a brain/plasma ratio of 1[1].

ABT-107 (0.01-1 μmol/kg i.p., 15 min before sacrifice) produces a dose-dependent increase in ERK1/2 and CREB[1].

ABT-107 (0.01, 0.1, and 1.0 mg/kg i.p.) increases S^9-GSK3 and decreases p-tau in mouse cortex and hippocampus in mice[1].
ABT-107 (5 mg/kg/day i.p.) infusion attenuates tau hyperphosphorylation in AD transgenic APP-tau mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Rats (male Sprague-Dawley; 350-380 g b.wt.)[1].
Dosage:	1, 3 μmol/kg.
Administration:	I.P. daily for 3 consecutive days.
Result:	Induced a significant, dose-dependent increase in ACh release by day 3 of repeated administration. Higher doses may be required to evoke ACh release in naive rats not engaged in stimulated, i.e., cognitive-related behavior.

Animal Model:	Female TAPP (and wild-type littermates) mice[1].
Dosage:	1 mg/kg.
Administration:	Continuous subcutaneous infusion for 2 weeks.
Result:	Produced a dose-dependent increase in Ser9 phosphorylation in the cingulate cortex 15 min after acute administration in mice.

REFERENCES

[1]. R Scott Bitner, et al. In vivo pharmacological characterization of a novel selective alpha7 neuronal nicotinic acetylcholine receptor agonist ABT-107: preclinical considerations in Alzheimer's disease. J Pharmacol Exp Ther. 2010 Sep 1;334(3):875-86.

[2]. Tanuja Bordia, et al. The α7 nicotinic receptor agonist ABT-107 protects against nigrostriatal damage in rats with unilateral 6-hydroxydopamine lesions. Exp Neurol. 2015 Jan;263:277-84.

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