nAChR-IN-1

Cat. No.: HY-151129

CAS No.: 849461-90-1

Molecular Formula: $C_{16}H_{31}NO_2$

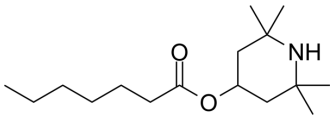
Molecular Weight: 269.42

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (371.17 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7117 mL	18.5584 mL	37.1168 mL
	5 mM	0.7423 mL	3.7117 mL	7.4234 mL
	10 mM	0.3712 mL	1.8558 mL	3.7117 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 (9.28 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

nAChR-IN-1 (2,2,6,6-Tetramethylpiperidin-4-yl heptanoate) is a tetramethylpiperidine heptanoate, a selective nicotinic acetylcholine receptor (nAChR) inhibitor that inhibits nAChRs lacking α5, α6, or β3 subunits. nAChR-IN-1 has the effect of preventing nerve disorder, can be used for nicotinic acetylcholine receptor dysfunction or neurological disorders research[1].

In Vitro

nAChR-IN-1 (100 nM) inhibits nAChR with inhibition rate of 90% in Xenovus oocytes, and suppresses AChR subtypes (mouse muscle-type (α1β1εδ) nAChR, three different pairwise combinations of rat neuronal alpha and beta subunits (α3β4, α4β2, and α3β2), and α7 homomeric neuronal nAChR) with IC_{50}s (area) of 390, 1.2, 110, 75, 440, 460, 430, 2.7, 1.0, 1.4, and 18.3 nM, respectively, based on the decrease in the net charge of the ACh response[1].

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

In Vivo

nAChR-IN-1 (20 mg/kg; s.c.; single dose) improves nicotine-induced hypomotility and hypothermia in a time-dependent manner in mice[1].

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

Animal Model:	Nicotine-induced mice model[1]
Dosage:	20 mg/kg
Administration:	Subcutaneous injection; single dose; 15 minutes prior to the injection of nicotine (1.5 mg/kg)
Result:	Increased locomotor activity in mice, even if nicotine-induced decrease. Showed no effect on body temperature.

Animal Model:	Tail-flick and hot-platemodel in mice[1]
Dosage:	0, 0.1, 1, 5 mg/kg
Administration:	Subcutaneous injection; single dose
Result:	Blocked the antinociceptive effect of nicotine in the hot-plate. Time-dependently blocked nicotine-induced antinociception.

REFERENCES

[1]. Papke RL, et, al. Compositions et methodes d'inhibition selective des recepteurs nicotiniques de l'acetylcholine. WO2005032479A2

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