PNU-120596

Cat. No.: HY-12152

CAS No.: 501925-31-1

Molecular Formula: $C_{13}H_{14}ClN_3O_4$

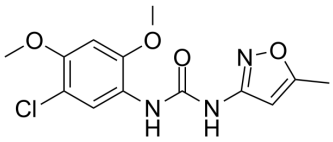
Molecular Weight: 311.72

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (160.40 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2080 mL	16.0400 mL	32.0801 mL
5 mM	0.6416 mL	3.2080 mL	6.4160 mL
10 mM	0.3208 mL	1.6040 mL	3.2080 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 (8.02 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.02 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PNU-120596 (NSC 216666) is a potent and selective α7 nAChR positive allosteric modulator (PMA) with an EC_{50} of 216 nM. PNU-120596 is inactive against α4β2, α3β4, and α9α10 nAChRs. PNU-120596 has the potential for psychiatric and neurological disorders research[1].

IC_{50} & Target

EC50: 216 nM (α7 nAChR)[1]

In Vitro

PNU-120596 increases agonist-evoked calcium flux mediated by an engineered variant of the human α7 nAChR. Electrophysiology studies confirme that PNU-120596 increases peak agonist-evoked currents mediated by wild-type receptors and also demonstrates a pronounced prolongation of the evoked response in the continued presence of agonist. PNU-120596 increases the channel mean open time of α7 nAChRs[1].

	When applied to acute hippocampal slices, PNU-120596 increases the frequency of ACh-evoked GABAergic postsynaptic currents measured in pyramidal neurons[1]. PNU-120596 enhances agonist-evoked gating of nicotinic receptors by eliciting conformational effects that are similar but nonidentical to the gating conformations promoted by ACh[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PNU-120596 (1 mg/kg; intravenous injection; once) treatment improves the auditory gating deficit caused by Amphetamine in rats, a model proposed to reflect a circuit level disturbance associated with schizophrenia[1]. When administered before carrageenan, NU-120596 (30 mg/kg; i.p.) significantly reduces mechanical hyperalgesia and weight-bearing deficits for up to 4 h in Sprague-Dawley rats. PNU-120596 attenuates the carrageenan-induced increase in levels of TNF-α and IL-6 within the hind paw oedema[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague Dawley rats (250-300 g) treated with Amphetamine[1]
Dosage:	1 mg/kg
Administration:	Intravenous injection; once
Result:	Improved the auditory gating deficit caused by Amphetamine.

REFERENCES

[1]. Hurst RS, et al. A novel positive allosteric modulator of the alpha7 neuronal nicotinic acetylcholine receptor: in vitro and in vivo characterization. J Neurosci, 2005, 25(17), 4396-4405.

[2]. Barron SC, et al. An allosteric modulator of alpha7 nicotinic receptors, N-(5-Chloro-2,4-dimethoxyphenyl)-N'-(5-methyl-3-isoxazolyl)-urea (PNU-120596), causes conformational changes in the extracellular ligand binding domain similar to those caused by ace

[3]. Munro G, et al. The α7 nicotinic ACh receptor agonist compound B and positive allosteric modulator PNU-120596 both alleviate inflammatory hyperalgesia and cytokine release in the rat. Br J Pharmacol, 2012, doi: 10.1111/j.1476-5381.2012.02003.x

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