

# PNU-120596

**Catalog No: tcsc0415** 

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:

501925-31-1

Formula:

 $\mathsf{C}_{13}\mathsf{H}_{14}\mathsf{CIN}_3\mathsf{O}_4$ 

Pathway: Neuronal Signaling;Membrane Transporter/Ion Channel

**Target:** 

nAChR;nAChR

Purity / Grade:

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

### **Alternative Names:**

NSC 216666

## **Observed Molecular Weight:**

311.72

# **Product Description**

Copyright 2021 Taiclone Biotech Corp.



PNU-120596 (NSC 216666 ) is a potent and selective positive allosteric  $\alpha$ 7 nAChR modulator with an EC50 of 0.2  $\mu$ M.

#### IC50 value: 0.2 uM (EC50) [1]

#### Target: α7

in vitro: PNU-120596 increases agonist (Ach)-evoked calcium flux mediated by an engineered variant of the human  $\alpha$ 7 nAChR. PNU-120596 increases agonists (choline and ACh)-evoked currents mediated by wild-type receptors and also demonstrates a pronounced prolongation of the evoked response in the continued presence of agonist in Xenopus oocytes. PNU-120596 increases the channel mean open time of  $\alpha$ 7 nAChRs but has no effect on ion selectivity and relatively little, if any, effect on unitary conductance. When applied to acute hippocampal slices, PNU-120596 increases the frequency of ACh-evoked GABAergic postsynaptic currents measured in pyramidal neurons; this effect is suppressed by TTX, suggesting that PNU-120596 modulates the function of  $\alpha$ 7 nAChRs located on the somatodendritic membrane of hippocampal interneurons [1]. Besides the positive modulation to  $\alpha$ 7 nAChR, PNU-120596 induces a profound retardation of the kinetics of desensitization, raising the potential of Ca2+-induced toxicity through excessive stimulation of  $\alpha$ 7 nAChR [2]. PNU-120596 causes changes in cysteine accessibility at the inner beta sheet, transition zone and agonist binding site while binding to  $\alpha$ 7 nAChR. Binding sites for PNU-120596 are not in the agonist-binding sites and PNU-120596 enhances agonistevoked gating of nicotinic receptors by eliciting conformational effects that are similar but nonidentical to the gating conformations promoted by Ach [3].

in vivo: Systemic administration of PNU-120596 (1 mg/kg) to rats improves the auditory gating deficit caused by amphetamine, a model proposed to reflect a circuit level disturbance associated with schizophrenia. [1] When administered prior to Carrageenan, 30 mg/kg PNU-1230596 significantly blunts mechanical hyperalgesia and weight bearing deficits for up to 4 hours. PNU-120596 attenuates the carrageenan-induced increase in levels of TNF- $\alpha$  and IL-6 within the hindpaw oedema, diclofenac only attenuated IL-6 levels. Established mechanical hyperalgesia induced by Carrageenan or CFA is also partially reversed by PNU-120596 [4].





All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!

Copyright 2021 Taiclone Biotech Corp.