Pramipexole dihydrochloride

Cat. No.: HY-17355

CAS No.: 104632-25-9

Molecular Formula: $C_{10}H_{19}Cl_2N_3S$

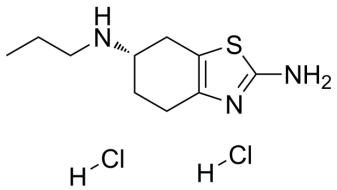
Molecular Weight: 284.25

Target: Dopamine Receptor

Pathway: GPCR/G Protein; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 50 mg/mL (175.90 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5180 mL	17.5902 mL	35.1803 mL
5 mM	0.7036 mL	3.5180 mL	7.0361 mL
10 mM	0.3518 mL	1.7590 mL	3.5180 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (351.80 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Pramipexole dihydrochloride is a selective and blood-brain barrier (BBB) penetrant dopamine D2-type receptor agonist, with K_is of 2.2 nM, 3.9 nM, 0.5 nM and 1.3 nM for D2-type receptor, D_2, D_3 and D_4 receptors, respectively. Pramipexole dihydrochloride can be used for the research of Parkinson's disease (PD) and restless legs syndrome (RLS)[1][2][3].

IC_{50} & Target	D_2 Receptor	D_3 Receptor	D_4 Receptor

In Vitro

Pramipexole shows a low binding affinity for D1-type receptor, with an IC_{50} of >50,000 nM[1].

?Pramipexole dihydrochloride (0.01-10?μM; 72 hours) produces dose-dependent increases of dendritic arborization and soma size[3].

?Pramipexole dihydrochloride attenuates levodopa-induced toxicity in mesencephalic cultures, suggests that pramipexole may be cytoprotective to dopamine neurons in tissue culture[4].

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

In Vivo

Pramipexole dihydrochloride (0.25-1 mg/kg; i.p.) significantly reduces the infarction volume in animals[5].
?Pramipexole dihydrochloride improves neurological recovery[5].
?Pramipexole dihydrochloride prevents ischemic cell death via mitochondrial pathways in ischemic stroke[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats weighing 250-300 g (16-18 weeks old)[5]
Dosage:	0.25 mg/kg, 1 mg/kg
Administration:	Intraperitoneal injection, at 1 hour, 6 hours, 12 hours, 18 hours post-occlusion
Result:	Decreased infarction volume as compared to tMCAO (transient middle cerebral artery occlusion)-only animals.

CUSTOMER VALIDATION

- Prog Neurobiol. 2023 Oct 5:102536.
- Neurochem Int. 2021 Jan 22;104972.
- PeerJ. 2023 Sep 11.
- J Stroke Cerebrovasc Dis. 2023 Apr 25;32(7):107142.

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REFERENCES

[1]. Kvernmo, T., et al. A review of the receptor-binding and pharmacokinetic properties of dopamine agonists. Clin Ther, 2006. 28(8): p. 1065-78.

[2]. Takashi Okura, et al. Blood-brain barrier transport of pramipexole, a dopamine D2 agonist. Life Sci. 2007 Apr 3;80(17):1564-71.

[3]. Ginetta Collo, et al. Ropinirole and Pramipexole Promote Structural Plasticity in Human iPSC-Derived Dopaminergic Neurons via BDNF and mTOR Signaling. Neural Plast. 2018; 2018: 4196961.

[4]. P M Carvey, et al. Attenuation of levodopa-induced toxicity in mesencephalic cultures by pramipexole. J Neural Transm (Vienna). 1997;104(2-3):209-28.

[5]. Syed Suhail Andrabi, et al. Pramipexole prevents ischemic cell death via mitochondrial pathways in ischemic stroke. Dis Model Mech. 2019 Aug 1; 12(8): dmm033860.

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