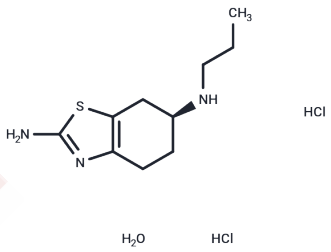
Data Sheet (Cat.No.T6951)

TargetMol

Pramipexole dihydrochloride hydrate

Chemical Properties



CAS No. : 191217-81-9

Formula: C10H17N3S·2HCl·H2O

Molecular Weight: 302.26

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Pramipexole dihydrochloride hydrate (Mirapex) is the hydrochloride salt of pramipexole, a benzothiazole derivative. As a nonergot dopamine agonist, pramipexole binds to D2 and D3 dopamine receptors in the striatum and substantia nigra of the brain.
Targets(IC50)	Dopamine Receptor
In vitro	Pramipexole (4-100 mM) significantly attenuated DA- or L-DOPA-induced cytotoxicity and apoptosis, an effect that was not inhibited by the D3 receptor antagonist, U-99194 A, or the D2 receptor antagonist, raclopride.Pramipexole also protected MES23.5 cells from hydrogen peroxide-induced cytotoxicity in a dose-dependent manner. cytotoxicity. Pramipexole effectively inhibited melanin formation.Pramipexole was neuroprotective against nigrostriatal dopamine neurons in hypoxic-ischemic and methamphetamine models. When incubated with SH-SY5Y cells and perfused into rat striatum, Pramipexole reduced the level of oxygen radicals produced by methylpyridinium ion (MPP+). Pramipexole also concentration-dependently inhibited the opening of mitochondrial transporter pore induced by calcium, phosphate, or MPP+. Pramipexole dose Pramipexole dose-dependently decreased dopamine metabolite levels, while striatal dopamine levels remained unchanged.
In vivo	Pramipexole (4-100 mM) significantly attenuated DA- or L-DOPA-induced cytotoxicity and apoptosis, an effect that was not inhibited by the D3 receptor antagonist, U-99194 A, or the D2 receptor antagonist, raclopride.Pramipexole also protected MES23.5 cells from hydrogen peroxide-induced cytotoxicity in a dose-dependent manner. cytotoxicity. Pramipexole effectively inhibited melanin formation.Pramipexole was neuroprotective against nigrostriatal dopamine neurons in hypoxic-ischemic and methamphetamine models. When incubated with SH-SY5Y cells and perfused into rat striatum, Pramipexole reduced the level of oxygen radicals produced by methylpyridinium ion (MPP+). Pramipexole also concentration-dependently inhibited the opening of mitochondrial transporter pore induced by calcium, phosphate, or MPP+. Pramipexole dose Pramipexole dose-dependently decreased dopamine metabolite levels, while striatal dopamine levels remained unchanged.

Solubility Information

Solubility	DMSO: 3.46 mg/mL (11.45 mM),Sonication is recommended. H2O: 55 mg/mL (181.96 mM),Sonication is recommended. Ethanol: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 1 mg/mL (3.31 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.3084 mL	16.542 mL	33.0841 mL
5 mM	0.6617 mL	3.3084 mL	6.6168 mL
10 mM	0.3308 mL	1.6542 mL	3.3084 mL
50 mM	0.0662 mL	0.3308 mL	0.6617 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Cassarino DS, et al. J Neurochem,1998, 71(1), 295-301.
Xu C, Zhou Y, Liu Y, et al.Specific pharmacological and Gi/o protein responses of some native GPCRs in neurons. Nature Communications.2024, 15(1): 1990.
Mierau J, et al. Eur J Pharmacol,1992, 215(2-3), 161-170.
Hall ED, et al. Brain Res,1996, 742(1-2), 80-88.
Zou L, et al. Life Sci, 1999, 64(15), 1275-1285.