JP1302 dihydrochloride

Cat. No.: HY-103213

CAS No.: 1259314-65-2

Molecular Formula: $C_{24}H_{26}Cl_2N_4$

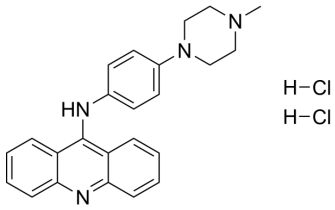
Molecular Weight: 441.4

Target: Adrenergic 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 : 12.5 mg/mL (28.32 mM; Need ultrasonic)

DMSO : 5 mg/mL (11.33 mM; ultrasonic and adjust pH to 5 with NaOH)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2655 mL	11.3276 mL	22.6552 mL
5 mM	0.4531 mL	2.2655 mL	4.5310 mL
10 mM	0.2266 mL	1.1328 mL	2.2655 mL

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

In Vivo

1. Add each solvent one by one: PBS

Solubility: 5 mg/mL (11.33 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

JP1302 dihydrochloride is a potent, selective, high affinity antagonist of the α_{2C}-adrenoceptor, with a K_b of 16 nM and a K_i of 28 nM for the human α_{2C}-receptor. JP1302 dihydrochloride shows antidepressant and antipsychotic-like effects. JP1302 dihydrochloride can be used for neuropsychiatric disorders and renal dysfunction research[1][2][3].

IC_{50} & Target

human α2C-adrenoceptor	human α2B-adrenoceptor	human α2A-adrenoceptor	rodent α2D-adrenoceptor
28±2 nM (Ki)	1470±130 nM (Ki)	3150±50 nM (Ki)	1700±200 nM (Ki)

In Vitro

JP1302 shows about 100-fold higher affinity than for α_{2A} or α_{2B}[1].

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

In Vivo

JP1302 (1-10 μmol/kg) decreases immobility time in the FST to a level similar to that seen with 10-30 μmol/kg of the antidepressant Desipramine (HY-B1272A)[1].

JP1302 (5 μmol/kg, once) is capable of complete reversal of the impairment in PPI induced in Sprague-Dawley rats by the

psychotomimetic NMDA receptor antagonist, phencyclidine and similar results are found in Wistar rats[1].
JP1302 (3 mg/kg, IV, once) significantly ameliorates renal dysfunction[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague Dawley rats (8 weeks old)[3]
Dosage:	3 mg/kg
Administration:	IV, pre-treatment: administered 5 min before the induction of ischemia, post-treatment: injected 45 min after the initiation of reperfusion
Result:	Significantly ameliorated renal dysfunction in the rats at 24 h after reperfusion. post-ischemic administration of JP-1302 significantly ameliorated renal dysfunction, histological damage and reduced apoptotic cells and pro-inflammatory cytokine mRNA expression.

REFERENCES

[1]. Shimokawa T, et al. Post-treatment with JP-1302 protects against renal ischemia/reperfusion-induced acute kidney injury in rats. J Pharmacol Sci. 2019 Mar;139(3):137-142.

[2]. Tricklebank MD, et al. JP-1302: a new tool to shed light on the roles of alpha2C-adrenoceptors in brain. Br J Pharmacol. 2007 Feb;150(4):381-2.

[3]. Sallinen J, et al. Pharmacological characterization and CNS effects of a novel highly selective alpha2C-adrenoceptor antagonist JP-1302. Br J Pharmacol. 2007 Feb;150(4):391-402.

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