H3R antagonist 1 hydrochloride

Cat. No.: HY-112219A

CAS No.: 2319790-07-1

Molecular Formula: $C_{19}H_{24}ClN_3O_3$

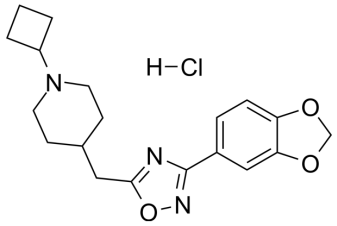
Molecular Weight: 377.87

Target: Histamine Receptor

Pathway: GPCR/G Protein; Immunology/Inflammation; 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

DMSO : 25 mg/mL (66.16 mM; Need warming)

H_2O : 25 mg/mL (66.16 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6464 mL	13.2321 mL	26.4641 mL
5 mM	0.5293 mL	2.6464 mL	5.2928 mL
10 mM	0.2646 mL	1.3232 mL	2.6464 mL

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

BIOLOGICAL ACTIVITY

Description

H3R antagonist 1 hydrochloride is a histamine receptor 3 (H3R) inverse agonist extracted from patent WO2013107336A1, compound example 2.

IC_{50} & Target

H_3 receptor

In Vitro

Treatment with H3R antagonist 1 hydrochloride, which is a H3R inverse agonist, promotes oligodendrocyte precursor cell (OPC) differentiation in a dose-dependent manner, at EC_{50}=25 nM. Western blot reveals a significant increase in expression levels of two markers of mature oligodendrocytes, myelin-associated glycoprotein (MAG) and myeline basic protein (MBP) in differentiating oligodendrocytes after treatment with H3R antagonist 1 hydrochloride, which suggests that treatment with H3R antagonist 1 hydrochloride drives more OPCs to differentiate. H3R antagonist 1 hydrochloride increases the Forskolin-stimulated cAMP level in the primary oligodendrocyte precursor cells in a dose-dependent manner[1].

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

In Vivo

The ability of H3R antagonist 1 hydrochloride-1 to enhance in vivo remyelination is determined with the Cuprizone/Rapamycin-induced demyelination model. Mice are treated with Cuprizone diet combined with intraperitoneal injections of Rapamycin for 5 weeks followed by 9 days of compound administration. Cuprizone diet plus intraperitoneal

injections of Rapamycin induced severe demyelination in both corpus callosum and cortex and treatment with H3R antagonist 1 hydrochloride (30 mg/kg, 9 days) significantly increases density of myelin specific Black-gold II staining in the lesion of corpus callosum and cortex in forebrain, compared to vehicle control group[1].

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

PROTOCOL

Animal Administration [1]

Mice[1]

The C57BL/6 mice at age of 8 weeks are fed with powder mouse food mixed freshly with 0.2% Cuprizone (w/w) and receive daily intraperitoneal injection of Rapamycin (10 mg/kg body weight) for 5 weeks to induce demyelination, then animals are allowed to recover (removal of Cuprizone from the diet and Rapamycin injection) and administrated with H3R-IN-1, at 30 mg/kg body weight orally, b.i.d. for an additional 9 days prior to sacrifice. The brain samples are collected for pathologic analysis[1].

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

REFERENCES

[1]. WANG, Rong, et al. THERAPEUTIC USES. WO2013107336A1.

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