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Lieferung & Zahlungsart

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Zuschläge

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AZ10606120 dihydrochloride

Cat. No.: HY-108669

CAS No.: 607378-18-7

Molecular Formula: $C_{25}H_{36}Cl_2N_4O_2$

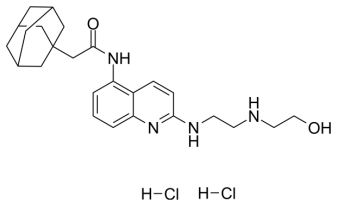
Molecular Weight: 495.48

Target: P2X Receptor

Pathway: Membrane Transporter/Ion Channel

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 : 8.33 mg/mL (16.81 mM; Need ultrasonic)

H_2O : 2 mg/mL (4.04 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0182 mL	10.0912 mL	20.1824 mL
5 mM	0.4036 mL	2.0182 mL	4.0365 mL
10 mM	0.2018 mL	1.0091 mL	2.0182 mL

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

BIOLOGICAL ACTIVITY

Description

AZ10606120 dihydrochloride is a selective, high affinity antagonist for P2X7 receptor (P2X7R) at human and rat with an IC_{50} of about 10 nM. AZ10606120 dihydrochloride is little or no effect at other P2XR subtypes. AZ10606120 dihydrochloride has anti-depressant effects and reduces tumour growth[1].

IC_{50} & Target

P2X7 Receptor

In Vitro

AZ10606120 (1-100 μM, 72 h) dihydrochloride depletes tumour cells in patient-derived primary glioblastoma samples[2].

AZ10606120 (1-100 μM, 72 h) dihydrochloride increases Lactate dehydrogenase (LDH) levels in human primary glioblastoma cultures[2].

AZ10606120 (10 μM) dihydrochloride reduces proliferation (60 h), cell migration (1 h) and invasion (24 h) in PDAC cell lines[3].

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

In Vivo

AZ10606120 (100 μg/kg, i.p., every 2 days for additional 15 days) dihydrochloride reverses Streptozotocin (HY-13753)-induced VEGF and IL-6 expression in the retinae of rats[4].

AZ10606120 (2 mg/kg i.p.) dihydrochloride shows an antidepressant phenotype in LPS-induced anhedonia mice[5].

AZ10606120 (5 mg/kg, i.m.) dihydrochloride and DNR (0.75 mg/kg, i.m.) combined administration is more effective in

reducing HL-60 tumor growth in nude mice in comparison to their single administration[6].

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

Animal Model:	LPS-induced anhedonia mice[5]
Dosage:	2 mg/kg
Administration:	i.p., pretreated at 30 min before LPS injection
Result:	Restored the decline in sucrose consumption, indicated by an sucrose preference test (SPT).

CUSTOMER VALIDATION

- Int J Med Microbiol. 18 October 2022, 151571.

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REFERENCES

[1]. Kan LK, et al. P2X7 receptor antagonism by AZ10606120 significantly reduced in vitro tumour growth in human glioblastoma. Sci Rep. 2023 May 24;13(1):8435.

[2]. Giannuzzo A, et al. The P2X7 receptor regulates cell survival, migration and invasion of pancreatic ductal adenocarcinoma cells. Mol Cancer. 2015 Nov 25;14:203.

[3]. Clapp C, et al. Pharmacological blockade of the P2X7 receptor reverses retinal damage in a rat model of type 1 diabetes. Acta Diabetol. 2019 Sep;56(9):1031-1036.

[4]. Csölle C, et al. Neurochemical Changes in the Mouse Hippocampus Underlying the Antidepressant Effect of Genetic Deletion of P2X7 Receptors. PLoS One. 2013 Jun 21;8(6):e66547.

[5]. Pegoraro A, et al. Differential sensitivity of acute myeloid leukemia cells to daunorubicin depends on P2X7A versus P2X7B receptor expression. Cell Death Dis. 2020 Oct 18;11(10):876.

[6]. Allsopp RC, et al. Unique residues in the ATP gated human P2X7 receptor define a novel allosteric binding pocket for the selective antagonist AZ10606120. Sci Rep. 2017 Apr 7;7(1):725.

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