CGS 21680 Hydrochloride

Cat. No.: HY-13201A

CAS No.: 124431-80-7

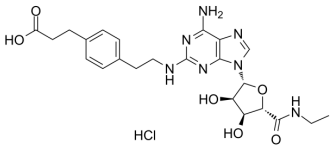
Molecular Formula: $C_{23}H_{30}ClN_7O_6$

Molecular Weight: 535.98

Target: Adenosine Receptor

Pathway: GPCR/G Protein

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 : ≥ 20 mg/mL (37.31 mM)
H_2O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.8657 mL	9.3287 mL	18.6574 mL
5 mM	0.3731 mL	1.8657 mL	3.7315 mL
10 mM	0.1866 mL	0.9329 mL	1.8657 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.66 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.66 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.66 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CGS 21680 Hydrochloride is a selective adenosine A2A receptor agonist with a K_i of 27 nM.

IC_{50} & Target

Ki: 27 nM (Adenosine A2A receptor)[5]

In Vitro

CGS21680 significantly upregulates CD39 and CD73 expression. CGS21680 accelerates the adenosine triphosphate (ATP) hydrolysis and adenosine generation[1]. CGS21680 (10 nM) alone shows only small survival activity, but the activity is significantly enhanced by the addition of a phosphodiesterase inhibitor, IBMX. The survival activity of CGS21680 on cultured

	motoneurons is exerted by mixed effects of the adenylate cyclase-cAMP-PKA pathway and the transactivation of neurotrophin receptors[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	CGS21680 (1 mg/kg/i.p.) intervention promotes the development of EAN. CGS21680 exacerbates experimental autoimmune neuritis in Lewis rats induced with bovine peripheral myelin. The exacerbation is accompanied with reduced $CD4^+$ $Foxp3^+$ T cells, increased $CD4^+$ $CXCR5^+$ T cells, B cells, dendritic cells and antigen-specific autoantibodies, which is possibly due to the inhibition of IL-2 induced by CGS21680[2]. CGS21680 (0.1 mg/kg, i.p.) transiently increases heart frequency but does not modify blood pressure of rat, and does not modify either heart frequency or blood pressure at 0.01 mg/kg. Following transient MCAo, CGS21680 at both doses protects from neurological deficit from the first day up to 7 days thereafter. At this time, it has reduced microgliosis, astrogliosis and improved myelin organization in the striatum and cytoarchitecture of the ischemic cortex and striatum. Two days after transient MCAo, CGS21680 has reduced the number of infiltrated granulocytes into the ischemic tissue[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	10×10^6 MNCs from each group are re-suspended in 2 mL RPMI 1640. Cell suspensions are added with carboxy-fluorescein diacetate, succinimidyl ester (CFSE, final concentration 2.5 μM) and thoroughly mixed. After incubation in the dark for 15 min at 37°C, the staining process is quenched by adding 10 mL ice-cold complete RPMI 1640 (containing 10% FBS) and incubated on ice for 5 min. Then cells are washed twice with RPMI 1640. Cell pellets are re-suspended in complete RPMI 1640 (containing 10% FBS). The stained MNCs (1×10^6 cells/mL, 1 mL/well) are cultured in triplicates in 24-well culture plates in the dark at 37°C. Each well is supplied with 50 μL of Concanavalin A (ConA, final concentration 5 μg/mL) or 50 μL of P0 peptide (final concentration 10 μg/mL). 72 h later, cells are collected and stained with PE-labeled anti-rat CD4 antibody for 30 min at 4°C. Finally, cells are analyzed with a flow cytometer. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Female Lewis rats aged 6-8 weeks (body weight, 140-160 g) are housed under specific pathogen-free conditions in the local animal facility with free access to water and food. Administration of CGS21680 (at a dose of 1 mg/kg in PBS) starts on day 5 p.i. Rats in experimental group are injected with CGS21680 intraperitoneally (i.p.) every two days until the end of the experiments. Rats in control group are given equal volume of PBS in the same way. The doses (1 mg/kg/i.p.) and the treatment regimen (every two days, start on day 5 p.i.) are determined. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Dis. 2020 Mar 23;11(3):202.
- J Neuroinflammation. 2022 Feb 18;19(1):52.
- Neurochem Int. 2021 Feb 9;145:104983.
- Iran J Basic Med Sci. 10, Oct 2021.
- Research Square Preprint. 2021 Aug.

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REFERENCES

[1]. Bao R, et al. Adenosine and the adenosine A2A receptor agonist, CGS21680, upregulate CD39 and CD73 expression through E2F-1 and CREB in regulatory T cells

isolated from septic mice. Int J Mol Med. 2016 Sep;38(3):969-75.

[2]. Zhang M, et al. Activation of the adenosine A2A receptor exacerbates experimental autoimmune neuritis in Lewis rats in association with enhanced humoral immunity. J Neuroimmunol. 2016 Apr 15;293:129-36.

[3]. Melani A, et al. Low doses of the selective adenosine A2A receptor agonist CGS21680 are protective in a rat model of transient cerebral ischemia. Brain Res. 2014 Mar 10;1551:59-72.

[4]. Komaki S, et al. Trk and cAMP-dependent survival activity of adenosine A(2A) agonist CGS21680 on rat motoneurons in culture. Neurosci Lett. 2012 Jul 26;522(1):21-4.

[5]. Rosaria Volpini, et al. Adenosine receptor agonists: synthesis and binding affinity of 2-(aryl)alkylthioadenosine derivatives.

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