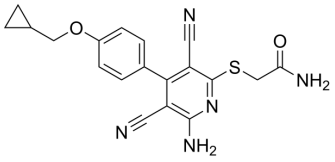
BAY 60-6583

Cat. No.:	HY-103171
CAS No.:	910487-58-0
Molecular Formula:	$C_{19}H_{17}N_5O_2S$
Molecular Weight:	379.44
Target:	Adenosine Receptor
Pathway:	GPCR/G Protein
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (263.55 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6355 mL	13.1773 mL	26.3546 mL
	5 mM	0.5271 mL	2.6355 mL	5.2709 mL
	10 mM	0.2635 mL	1.3177 mL	2.6355 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: ≥ 4.17 mg/mL (10.99 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 4.17 mg/mL (10.99 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BAY 60-6583 is a potent and high-affinity agonist of adenosine A_{2B} receptor (EC_{50} = 3 nM) over A1, A2A, and A3 receptors. BAY 60-6583 binds to mouse, rabbit, and dog A2BAR with K_i values of 750 nM, 340 nM and 330 nM, respectively. BAY 60-6583 has a cardioprotective effect in a myocardial ischemia model[1][5].

In Vitro

BAY 60-6583 exhibits? EC_{50} values for receptor activation >10,000 nM for both A1 and A2A AR and 3 nM for A2B AR subtype in CHO cells expressing recombinant human A1, A2A or A2B ARs[1].

?BAY 60-6583(0-10 μM) exhibits the maximum agonist effect of BAY in the absence of siRNA is 68 %, which is significantly different from that in the presence of 5, 50 and 500 nM siRNA (54%, 48% and 36%, respectively). It exhibits EC_{50} ?values of BAY in the absence and presence siRNA with 98±22, 102±17, 127±31 and 93±19 nM, respectively, in T24 cells[3].

?BAY 60-6583 (5 μM; 24 hours) increases the accumulation of cells at the G1 phase with a decrease in G2/M phase in

RAW264.7 preosteoclasts[4].
?BAY 60-6583 (5 μM; 24 hours) specifically inhibits the activation of Akt by M-CSF, whereas M-CSF-induced ERK1/2 activation is not affected by BAY 60-6583 treatment in RAW264.7 preosteoclasts[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[4]

Cell Line:	RAW264.7 preosteoclasts
Concentration:	5 μM
Incubation Time:	48 hours
Result:	Caused an arrest of cells at the G1 phase.

Western Blot Analysis[4]

Cell Line:	RAW264.7 preosteoclasts
Concentration:	5 μM
Incubation Time:	48 hours
Result:	Exhibited an inhibition of M-CSF-mediated Akt activation and resulted in the decrease of osteoclast proliferation.

In Vivo

BAY 60-6583 (intravenous?injection; 100 mcg/kg) reduces the infarction area just prior to reperfusion in ischaemic rabbit hearts[1].
?BAY 60-6583 (intraperitoneal?injection; 2 mg/kg) attenuates LPS-induced lung injury, pre-treatment with this compound can significantly decrease LPS-increased? IL-6 levels in WT-mice, In contrast, BAY 60-6583 treatment is ineffective in abrogating these inflammatory parameters in ?A2BAR$^{?/?}$?mice[2].
?BAY 60-6583 (intratumoral administration) causes a significant increase in tumor-infiltrating MDSCs, it does not affect neither their ability to suppress T-cell proliferation nor their degree of maturation, it also stimulates the production of IL-10 and CCL2 in the tumor tissue[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	A2BAR$^{-/-}$ mice on a C57BL/6J mice[1]
Dosage:	2 mg/kg
Administration:	Intraperitoneal injection; 2 mg/kg
Result:	Demonstrated attenuation of lung inflammation and pulmonary edema in wild-type but not in gene-targeted mice for the A2BAR.

CUSTOMER VALIDATION

- Sci Adv. 2022 Dec 23;8(51):eadd3709.
- PeerJ. 2023 Aug 30.
- Tissue Cell. 2022 May 20;77:101828.
- Br J Pharmacol. 2023 Apr 28.

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REFERENCES

[1]. Aherne CM, et al. Epithelial-specific A2B adenosine receptor signaling protects the colonic epithelial barrier during acute colitis.Mucosal Immunol. 2015 Nov;8(6):1324-38.

[2]. Schingnitz U, et al. Signaling through the A2B adenosine receptor dampens endotoxin-induced acute lung injury.J Immunol. 2010 May 1;184(9):5271-9.

[3]. Gao ZG, et al. Probing biased/partial agonism at the G protein-coupled A(2B) adenosine receptor.Biochem Pharmacol. 2014 Aug 1;90(3):297-306.

[4]. Yoon Taek Oh, et al. A2B Adenosine Receptor Stimulation Down-regulates M-CSF-mediated Osteoclast Proliferation. Biomed Sci Letters 2017;23:194-200

[5]. John A. Auchampach, et al. Characterization of the A2B Adenosine Receptor from Mouse, Rabbit, and Dog. J Pharmacol Exp Ther. 2009 Apr;329(1):2-13.

[6]. Morello S1, et al. Targeting the adenosine A2b receptor in the tumor microenvironment overcomes local immunosuppression by myeloid-derived suppressor cells.Oncoimmunology. 2014 Feb 14;3:e27989. eCollection 2014.

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