Product Data Sheet

Product Name: PSB 1115
Cat. No.: GC17756

Chemical Properties

Cas. No.	152529-79-8		
Chemical Name	4-(2,6-dioxo-1-propyl-2,3,6,7-tetrahydro-1H-purin-8-yl)benzenesulfonic acid		
SMILES	O=C(C(NC(C(C=C1)=CC=C1S(O)(=O)=O)=N2)=C2NC3=O)N3CCC		
Formula	$C_{14}H_{14}N_4O_5S$	M.Wt	350.35
Solubility	<38.84mg/ml in DMSO; <1mg/ml in Water	Storage	Store at -20°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	PSB 1115		

Protocol

Cell experiment [1]:

Cell lines	MDA-MB-231 cells

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Preparation Method	MDA-MB-231 cells were cultured under standard conditions (37°C, 5% CO_2) in MEM medium supplemented with 10% fetal bovine serum and 1% penicillin-streptomycin. MDA-MB-231 cells were seeded in 6-well plates (5×10^4/ml) and cultured at 37°C, 5% CO_2 for 24 hours. Then, 1μM of Cucumarioside A_0-1 (Cuc A_0-1), 2μM of Djakonovioside A and 1μM PSB 1115 were added to the cells and cultured for 6 hours. Subsequently, RIPA lysis buffer was added for cell lysis (10,000×g, 15 minutes, 4°C). The cell lysate supernatant was collected and the cAMP level was analyzed.
Reaction Conditions	1μM; 6h
Applications	PSB 1115 treatment significantly reduced cAMP levels in MDA-MB-231 cells.

Animal experiment [2]:

Animal models	Female Athymic Nude-Foxn1^{nu} mice
Preparation Method	Female Athymic Nude-Foxn1^{nu} mice (6-8 weeks old) were housed in an animal room of specific pathogen-free (SPF) grade. 2×10^5 B16-F10 cells were subcutaneously injected into the right abdomen of anesthetized mice. Ten days after tumor cell implantation, when the tumors were palpable, Bay 60-6583 (0.2mg/kg/day) or PSB 1115 (1mg/kg/day) was administered by peritumoral injection to the mice for 4 consecutive days, and the mouse tumor tissues were collected for analysis.
Dosage form	1mg/kg/day for 4 days; peritumoral injection

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Applications	PSB 1115 treatment significantly reduced the levels of tumor-infiltrating $CD11b^+$ $Gr1^+$ cells in the tumor tissues of B16-F10 cell-xenograft mice.

References:
[1] Zelepuga E A, Chingizova E A, Menchinskaya E S, et al. Anticancer Activity of Triterpene Glycosides Cucumarioside A0-1 and Djakonovioside A Against MDA-MB-231 as A2B Adenosine Receptor Antagonists[J]. International Journal of Molecular Sciences, 2025, 26(21): 10327.
[2] Iannone R, Miele L, Maiolino P, et al. Blockade of A2b adenosine receptor reduces tumor growth and immune suppression mediated by myeloid-derived suppressor cells in a mouse model of melanoma[J]. Neoplasia, 2013, 15(12): 1400-IN10.

Background

PSB 1115 is a selective A2B Adenosine Receptor antagonist[1]. PSB 1115 can inhibit the expression of the TRPV1 gene, reduce calcium ion influx, and thereby regulate the sensory hypersensitivity and pain-related behaviors in animals[2]. PSB 1115 has been

widely used in mouse models of middle cerebral artery occlusion to reduce the infarct area[3].

In vitro, PSB 1115 treatment for 15 minutes significantly inhibited the increase in cAMP induced by adenosine (100μM) in T84 cells, with an IC_{50} value of 84nM[4]. Treatment with 1μM PSB 1115 for 24 hours resulted in a significant increase in the expression levels of p-JNK1/2 and JNK1/2 in MDA-MB-231 cells[5].

In vivo, PSB 1115 treatment at a dose of 1mg/kg/day via peritumoral injection for 4 consecutive days significantly inhibited tumor growth in the melanoma mouse model and led to a significant reduction in the number of tumor-infiltrating $CD11b^+$ $Gr1^+$ cells in the in the tumors of mice[6]. A single intravenous injection of 10mg/kg dose of PSB 1115 for 10min significantly inhibited the tachycardia and the dilation of the kidneys and mesenteric blood vessels caused by A1-receptor bitopic ligand VCP746 in rats[7]. For a consecutive week, 1mg/kg dose of PSB 1115 was intraperitoneally injected into mice carrying B16.F10 tumors every day, resulting in a significant decrease in VEGF expression and microvessel density in the tumor tissues[8].

References:
[1] Rüsing D, Müller C E, Verspohl E J. The impact of adenosine and A2B receptors on glucose homoeostasis[J]. Journal of Pharmacy and Pharmacology, 2006, 58(12): 1639-1645.
[2] Hu X, Adebiyi M G, Luo J, et al. Sustained elevated adenosine via ADORA2B promotes chronic pain through neuro-immune interaction[J]. Cell Reports, 2016, 16(1): 106-119.
[3] Weitzel L B, Grewal H, Herson P S, et al. Abstract T P82: The Role of the A2B Receptor in a Mouse Model Of Stroke[J]. Stroke, 2015, 46(suppl_1): ATP82-ATP82.
[4] Asano T, Noda Y, Tanaka K I, et al. A2B adenosine receptor inhibition by the dihydropyridine calcium channel blocker nifedipine involves colonic fluid secretion[J]. Scientific Reports, 2020, 10(1): 3555.
[5] Zelepuga E A, Chingizova E A, Menchinskaya E S, et al. Anticancer Activity of Triterpene Glycosides Cucumarioside A0-1 and Djakonovioside A Against MDA-MB-231 as A2B Adenosine Receptor Antagonists[J]. International Journal of Molecular Sciences, 2025, 26(21): 10327.
[6] Iannone R, Miele L, Maiolino P, et al. Blockade of A2b adenosine receptor reduces

tumor growth and immune suppression mediated by myeloid-derived suppressor cells in a mouse model of melanoma[J]. Neoplasia, 2013, 15(12): 1400-IN10.
[7] Cooper S L, Wragg E, March J, et al. Effects of an Adenosine Receptor Bitopic Ligand on The Cardiovascular System[J]. The FASEB Journal, 2020, 34(S1): 1-1.
[8] Sorrentino C, Miele L, Porta A, et al. Myeloid-derived suppressor cells contribute to A2B adenosine receptor-induced VEGF production and angiogenesis in a mouse melanoma model[J]. Oncotarget, 2015, 6(29): 27478.