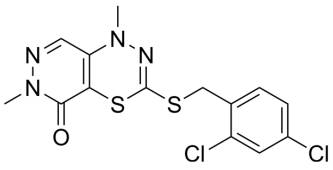
BTB09089

Cat. No.:	HY-149164
CAS No.:	245728-44-3
Molecular Formula:	$C_{14}H_{12}Cl_2N_4OS_2$
Molecular Weight:	387.31
Target:	Others
Pathway:	Others

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (258.19 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5819 mL	12.9096 mL	25.8191 mL
	5 mM	0.5164 mL	2.5819 mL	5.1638 mL
	10 mM	0.2582 mL	1.2910 mL	2.5819 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: 5 mg/mL (12.91 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BTB09089 is a T cell death-associated gene 8 (TDAG8/GPR65) specific agonist. BTB09089 increases TDAG8 expression and regulates the cytokine production of T cells and macrophages[1].

IC_{50} & Target

TDAG8/GPR65[1]

In Vitro

BTB09089 (0-18 μM; 30 min) significantly increases cAMP accumulation in hTDAG8 and mTDAG8 transiently expressing HEK293 cells, but not in control HEK 293 cells. BTB09089 does not increase cAMP accumulation in hGPR4 expressing HEK293 cells nor does it increase inositol phosphate accumulation in hOGR1 expressing HEK293 cells[1].

BTB09089 (0-18 μM; 30 min) significantly enhances the cAMP accumulation in a dose-dependent manner at pH 7.0-7.9 but not at pH 6.5[1].

BTB09089 (1-5 μM; 20 h) suppresses IL-2 production in splenocytes from WT mice in a dose-dependent manner but not from TDAG8 KO mice without affecting the cell viability[1].

	BTB09089 (1-5 μM; 18 h) suppresses LPS (HY-D1056)-stimulated TNF-α and IL-6 production, and enhances LPS-stimulated IL-10 production in thioglycollate induced peritoneal exude cells (TG-PEC)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BTB09089 (5-20 μM, 8 μL; i.c.v.; 6 hours prior to MCAO) reduces cerebral ischaemia-reperfusion injury in rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male SD rats (270-280 g), middle cerebral artery occlusion (MCAO) model[2]
Dosage:	5, 10 and 20 μM, 8 μL
Administration:	Intracerebroventricular injection, six hours prior to MCAO
Result:	Up-regulated TDAG8 and Bcl-2 expression and down-regulated cleaved caspase-3 expression, while the infarction volume was reduced, and neurological deficits were ameliorated 24 and 72 h after MCAO.

REFERENCES

[1]. Onozawa Y, et al. Activation of T cell death-associated gene 8 regulates the cytokine production of T cells and macrophages in vitro. Eur J Pharmacol. 2012 May 15;683(1-3):325-31.

[2]. Ma XD, et al. TDAG8 activation attenuates cerebral ischaemia-reperfusion injury via Akt signalling in rats. Exp Neurol. 2017 Jul;293:115-123.

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