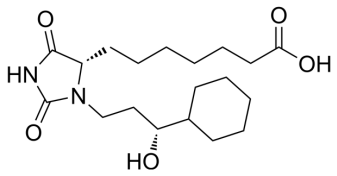
BW 245C

Cat. No.:	HY-101987
CAS No.:	72814-32-5
Molecular Formula:	$C_{19}H_{32}N_2O_5$
Molecular Weight:	368
Target:	Prostaglandin Receptor
Pathway:	GPCR/G Protein
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (135.87 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7174 mL	13.5870 mL	27.1739 mL
	5 mM	0.5435 mL	2.7174 mL	5.4348 mL
	10 mM	0.2717 mL	1.3587 mL	2.7174 mL

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

BIOLOGICAL ACTIVITY

Description

BW 245C is a prostanoid DP-receptor (DP1) agonist, used to treat stroke.

IC_{50} & Target

DP

In Vitro

BW245C (0.01-1 μM) suppresses TGF-β-induced collagen secretion in a dose-dependent manner in Th2 cells. BW245C (0.01-1 μM) also increases intracellular cAMP in lung fibroblasts[3]. BW245C (0.1-3 μmol/L) dose-dependently increases transendothelial electrical resistance and decreases the FITC-dextran permeability of human umbilical vein endothelial cells. BW245C (0.3 μmol/L) increases the intracellular cAMP level and subsequent protein kinase A (PKA) activity[4].

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

In Vivo

BW245C (0.02, 0.2, and 2.0 mg/kg) in WT mice results in a significant increase in CBF, but this effect of this treatment is absent in $DP1^{-/-}$ mice. BW245C attenuates functional deficits after stroke. BW245C significantly reverses these conditions that neurologic deficit is significantly augmented, whereas locomotor activity is significantly reduced after stroke in WT mice. BW245C (0.2 mg/kg) injection 1 h after stroke results in a significant decrease in brain infarction in WT mice, whereas the effect of this treatment is not observed in $DP1^{-/-}$ mice. BW245C improves CBF during and after stroke. BW245C results in significantly prolonged bleeding compared with the vehicle-treated group[1]. BW 245C (100 nM) does not significantly

	increase MBP-positive eosinophils in esophageal epithelium in OVA-sensitized guinea pigs[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	The mice are untreated or given an i.p. injection of the vehicle (1% DMSO) or 0.2-mg/kg BW245C. Thirty minutes after the injection, the mice are anesthetized and placed on a thermoregulated pad to maintain body temperature, and 3 mm of the tail tip is excised. The tail is immediately dipped in warm PBS (37.0±0.5°C) and time to visible cessation of bleeding is recorded. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Ahmad AS, et al. PGD_2 DP1 receptor stimulation following stroke ameliorates cerebral blood flow and outcomes. Neuroscience. 2014 Oct 24;279:260-8.

[2]. Zhang S, et al. Prostaglandin D_2 receptor D-type prostanoid receptor 2 mediates eosinophil trafficking into the esophagus. Dis Esophagus. 2014 Aug;27(6):601-6.

[3]. Ayabe S, et al. Prostaglandin D_2 inhibits collagen secretion from lung fibroblasts by activating the DP receptor. J Pharmacol Sci. 2013;121(4):312-7. Epub 2013 Mar 29.

[4]. Kobayashi K, et al. Prostaglandin D_2-DP signaling promotes endothelial barrier function via the cAMP/PKA/Tiam1/Rac1 pathway. Arterioscler Thromb Vasc Biol. 2013 Mar;33(3):565-71.

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