TG4-155

Cat. No.: HY-18971

CAS No.: 1164462-05-8

Molecular Formula: $C_{23}H_{26}N_2O_4$

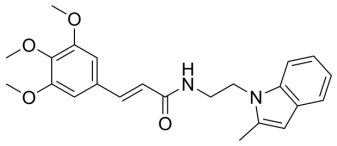
Molecular Weight: 394.46

Target: Prostaglandin 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 : 125 mg/mL (316.89 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5351 mL	12.6756 mL	25.3511 mL
	5 mM	0.5070 mL	2.5351 mL	5.0702 mL
	10 mM	0.2535 mL	1.2676 mL	2.5351 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: ≥ 2.08 mg/mL (5.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TG4-155 is a potent, brain-permeant and selective EP2 receptor antagonist with a K_i of 9.9 nM[1][2]. TG4-155 shows low nanomolar antagonist activity against only EP2 and DP1[1]. TG4-155 has an EP2 Schild K_B of 2.4 nM and displays 550-4750-fold selectivity for EP2 over EP1, EP3, EP4 and IP, but only 14-fold selectivity against the DP1 receptor[2].

IC_{50} & Target

EP2
9.9 nM (Ki)

In Vitro

TG4-155 inhibits the serotonin 5-HT2B receptor with IC_{50}=2.6 μM and hERG (human Ether-à-go-go-Related Gene) with IC_{50} =12 μM[1].
PGE_2 (0.1-10 μM) stimulation significantly enhances human prostate cancer cell line PC3 cell growth in a concentration-

dependent manner with a maximal response being obtained at approximately 1 μM. This PGE_2-induced cancer cell proliferation is significantly suppressed by TG4-155 (0.01-1μM ; 48 hours) in a concentration-dependent manner[1].

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

Cell Viability Assay[1]

Cell Line:	PC3 cells
Concentration:	48 hours
Incubation Time:	0.01, 0.1, and 1 μM
Result:	Significantly suppressed PGE_2-induced cancer cell proliferation in a concentration-dependent manner.

In Vivo

Administration of TG4-155 (5 mg/kg, i.p.; at 1 and 12 h) significantly reduces status epilepticus (SE)-induced neurodegeneration scores in C57BL/6 mice[3].

TG4-155 (3 mg/kg; i.p.) displays a bioavailability of 61% (i.p. route compared with i.v.), a plasma half-life ($t_{1/2}$) of 0.6 h, and a brain/plasma ratio of 0.3 in C57BL/6 mice[3].

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

Animal Model:	C57BL/6 mice (8-12 wk old)[3]
Dosage:	5 mg/kg
Administration:	I.p.; at 1 and 12 h
Result:	Administration significantly reduced SE-induced neurodegeneration scores by 91% in hippocampal subregions CA1, by 80% in CA3, and by 63% in hilus.

Animal Model:	C57BL/6 mice[3]
Dosage:	3 mg/kg
Administration:	I.p.
Result:	Displayed a bioavailability of 61% (i.p. route compared with i.v.), $t_{1/2}$ of 0.6 h, and a brain/plasma ratio of 0.3.

CUSTOMER VALIDATION

- J Cell Mol Med. 2021 Oct 5.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Jiang J, et al. Role of prostaglandin receptor EP2 in the regulations of cancer cell proliferation, invasion, and inflammation. J Pharmacol Exp Ther. 2013 Feb;344(2):360-7.

[2]. Ganesh T. Prostanoid receptor EP2 as a therapeutic target. J Med Chem. 2014 Jun 12;57(11):4454-65.

[3]. Jiang J, et al. Small molecule antagonist reveals seizure-induced mediation of neuronal injury by prostaglandin E2 receptor subtype EP2. Proc Natl Acad Sci U S A. 2012 Feb 21;109(8):3149-54.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA