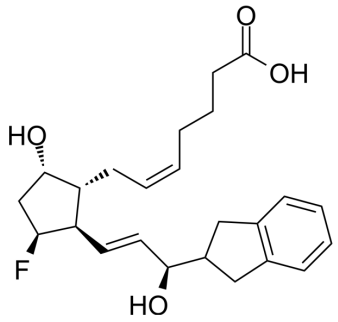
AL-8810

Cat. No.:	HY-100449
CAS No.:	246246-19-5
Molecular Formula:	$C_{24}H_{31}FO_4$
Molecular Weight:	402.5
Target:	Prostaglandin Receptor; p38 MAPK; ERK
Pathway:	GPCR/G Protein; MAPK/ERK Pathway; Stem Cell/Wnt
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



BIOLOGICAL ACTIVITY

Description

AL-8810 is a potent and selective antagonist of the $PGF_{2\alpha}$ receptor (FP receptor). AL-8810 is an activator of MAPK and ERK1/2 . The K_i of the FP receptor of mouse 3T3 cells and rat A7r5 cells are 0.2±0.06 μM and 0.4±0.1 μM, respectively. AL-8810 can be used in the study of elevated intraocular pressure (OHT) and primary open-angle glaucoma (POAG)[1][2][3][4][5].

In Vitro

AL-8810 (0.1, 1, 10 μM, 24 h) can significantly reduce the neuronal cell death of WT mice after OGD-induced injury[2].
AL-8810 (1, 10 μM, 24 h) promotes the activation of ERK1/2 in HEK 293 and MG-63 cells through epidermal growth factor receptor deactivation mechanism[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	HEK 293, MG-63
Concentration:	1, 10 μM
Incubation Time:	24 h
Result:	Activated ERK1/2 and MAPK. Activated MAPK through EGFR transactivation.

In Vivo

AL-8810 (1 or 10 mg/kg, intravenously) reduces ischemic brain injury and neurotoxicity in mice[2].
AL-8810 (10 mg/kg, intraperitoneal injection) can improve the prognosis after experimental traumatic brain injury in mice[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Stroke mouse model[2]
Dosage:	1 or 10 mg/kg
Administration:	i.v.
Result:	Reduced the cortical infarct volume and tape-removal times.

Animal Model:	Traumatic brain injury (TBI) model[5]

Dosage:	10 mg/kg
Administration:	i.p.
Result:	Improved neurological deficit scores (NDS) at 24 and 48 hours after controlled cortical impact (CCI).

REFERENCES

[1]. Kim YT, et al. Prostaglandin FP receptor inhibitor reduces ischemic brain damage and neurotoxicity. Neurobiol Dis. 2012 Oct;48(1):58-65. doi: 10.1016/j.nbd.2012.06.003. Epub 2012 Jun 16.

[2]. Goupil E, et al. Biasing the prostaglandin F2α receptor responses toward EGFR-dependent transactivation of MAPK. Mol Endocrinol. 2012 Jul;26(7):1189-202.

[3]. Sharif NA, et al. Prostaglandin FP receptor antagonists: discovery, pharmacological characterization and therapeutic utility. Br J Pharmacol. 2019 Apr;176(8):1059-1078.

[4]. Glushakov AV, et al. Prostaglandin F2α FP receptor antagonist improves outcomes after experimental traumatic brain injury. J Neuroinflammation. 2013 Oct 30;10:132.

[5]. Griffin BW, et al. AL-8810: a novel prostaglandin F2 alpha analog with selective antagonist effects at the prostaglandin F2 alpha (FP) receptor. J Pharmacol Exp Ther. 1999 Sep;290(3):1278-84.

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