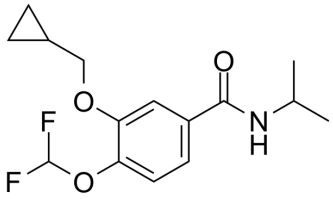
FCPR03

Cat. No.:	HY-117977
CAS No.:	1917347-65-9
Molecular Formula:	$C_{15}H_{19}F_2NO_3$
Molecular Weight:	299.31
Target:	Phosphodiesterase (PDE)
Pathway:	Metabolic Enzyme/Protease
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 (334.10 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3410 mL	16.7051 mL	33.4102 mL
	5 mM	0.6682 mL	3.3410 mL	6.6820 mL
	10 mM	0.3341 mL	1.6705 mL	3.3410 mL

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

BIOLOGICAL ACTIVITY

Description

FCPR03 is a potent and selective phosphodiesterase 4 (PDE4) inhibitor with IC_{50} values of 60 nM, 31 nM and 47 nM for PDE4 catalytic domain, PDE4B1 and PDE4D7, respectively. FCPR03 displays at least 2100-fold selectivity over other PDEs (PDE1-3 and PDE5-11). FCPR03 has anti-inflammatory, neuroprotective and antidepressant-like effects[1][2].

IC_{50} & Target			
	PDE4 catalytic domain 60 nM (IC_{50})	PDE4B1 31 nM (IC_{50})	PDE4D7 47 nM (IC_{50})

In Vitro

FCPR03 (5-20 μM; 30 hours; HT-22 cells) treatment increases cell viability (oxygen-glucose deprivation (OGD)-induced) in a dose-dependent manner, and 10 μM FCPR03 shows significant protective effects[1].

FCPR03 (20 μM; 30 hours; HT-22 cells) treatment protects against OGD-induced cellular apoptosis in both HT-22 cells and cortical neurons. The levels of mitochondrial membrane potential (MMP) and ROS are also restored by FCPR03[1].

FCPR03 (20 μM; 30 hours; HT-22 cells) treatment increases the levels of phosphorylated AKT, glycogen synthase kinase-3β (GSK3β), and β-catenin[1].

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

Cell Viability Assay[1]

Cell Line:	HT-22 cells
Concentration:	5 μM, 10 μM, 20 μM
Incubation Time:	30 hours
Result:	Increased cell viability in a dose-dependent manner.

Apoptosis Analysis[1]

Cell Line:	HT-22 cells
Concentration:	20 μM
Incubation Time:	30 hours
Result:	Reversed the effects of OGD and decreased the ratio of apoptotic cells.

Western Blot Analysis[1]

Cell Line:	HT-22 cells
Concentration:	20 μM
Incubation Time:	30 hours
Result:	Increased the levels of phosphorylated AKT, glycogen synthase kinase-3β (GSK3β), and β-catenin.

In Vivo

FCPR03 (1.25-5 mg/kg; intraperitoneal injection; once; adult male Sprague-Dawley rats) treatment reduces the infarct volume and improves neurobehavioral outcomes in rats following MCAO. FCPR03 increases the levels of phosphorylated AKT, GSK3β and β-catenin within the ischemic penumbra of rats following cerebral ischemia-reperfusion[1].

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

Animal Model:	Adult male Sprague-Dawley rats (250-280 g) induced middle cerebral artery occlusion (MCAO)[1]
Dosage:	1.25 mg/kg, 2.5 mg/kg, 5 mg/kg
Administration:	Intraperitoneal injection; once
Result:	Reduced the infarct volume and improved neurobehavioral outcomes in rats following MCAO.

REFERENCES

[1]. Bingtian Xu, et al. FCPR03, a Novel Phosphodiesterase 4 Inhibitor, Alleviates Cerebral ischemia/reperfusion Injury Through Activation of the AKT/GSK3β/ β-catenin Signaling Pathway. Biochem Pharmacol. 2019 May;163:234-249.

[2]. Zheng-Qiang Zou, et al. Novel Phosphodiesterase 4 Inhibitor FCPR03 Alleviates Lipopolysaccharide-Induced Neuroinflammation by Regulation of the cAMP/PKA/CREB Signaling Pathway and NF- κ B Inhibition. J Pharmacol Exp Ther. 2017 Jul;362(1):67-77.

Caution: Product has not been fully validated for medical applications. For research use only.

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