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Diagnostik & molekulare Diagnostik



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Lieferung & Zahlungsart

siehe unsere [Liefer- und Versandbedingungen](#)

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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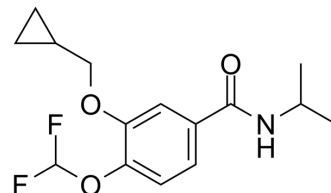
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FCPR03

Cat. No.:	HY-117977		
CAS No.:	1917347-65-9		
Molecular Formula:	C ₁₅ H ₁₉ F ₂ NO 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)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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 IC₅₀ 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]}.

IC₅₀ & Target

PDE4 catalytic domain 60 nM (IC ₅₀)	PDE4B1 31 nM (IC ₅₀)	PDE4D7 47 nM (IC ₅₀)
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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 μ M, 10 μ M, 20 μ M
Incubation Time:	30 hours
Result:	Increased cell viability in a dose-dependent manner.

Apoptosis Analysis^[1]

Cell Line:	HT-22 cells
Concentration:	20 μ M
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 μ M
Incubation Time:	30 hours
Result:	Increased the levels of phosphorylated AKT, glycogen synthase kinase-3 β (GSK3 β), 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, GSK3 β 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/GSK3 β / 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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