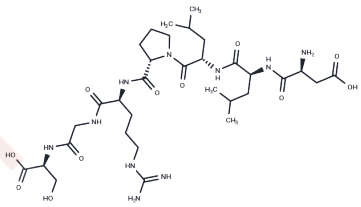
Data Sheet (Cat.No.TP1947)

TargetMol

P110

Chemical Properties



CAS No. : 1411976-18-5

Formula: C100H179N45O25

Molecular Weight: 2411.80

Storage: Keep away from moisture
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	P110 heptapeptide is a specific peptide inhibitor that interacts with Drp1/Fis1 (IC_{50}=1.8 μM). By competitively blocking the binding of Drp1 to Fis1, P110 heptapeptide inhibits pathological mitochondrial fission while preserving the physiological function of Drp1, thereby reducing pathological features in numerous models of neurodegeneration, ischemia, and sepsis. P110 heptapeptide exhibits neuroprotective, anti-inflammatory, and anti-sepsis activity and can be used in research on neurodegenerative diseases and conditions associated with mitochondrial dysfunction.
Targets(IC50)	Others
In vitro	Methods: RAW264.7 cells were co-treated with P110 (1 μM) and LPS (10 ng/mL) for 16 h, and mitochondrial membrane potential was measured using JC-1 fluorescence. Results: P110 reduced Drp1 mitochondrial localization and restored the LPS-induced increase in membrane potential and decline in respiratory function. [1] Methods: H9c2 cells were treated with P110 (2 μM) and LPS (2 μg/mL) for 24 h. Anti-Drp1 and anti-Fis1 immunostaining was performed, followed by confocal z-stack imaging to determine the ratio of Drp1 foci to mitochondrial area. Results: LPS increased Drp1 mitochondrial localization, while P110 reduced Drp1 mitochondrial localization to levels below those of the control.[2]
In vivo	Methods: BALB/c mice were administered LPS (1 mg/kg) and P110 (0.5 mg/kg/day) intraperitoneally. After 18 hours of tolerance induction, blood samples were collected 90 minutes following treatment with a high dose of LPS (10 mg/kg). Results: P110 restored the pro-inflammatory cytokine response to LPS or CLP in endotoxin-tolerant mice; it improved extracellular mitochondrial membrane potential in plasma cells without affecting their number. [1] Methods: Rats underwent transient middle cerebral artery occlusion (tMCAO) surgery. After 2 h of ischemia, PKH26-labeled EXO-P110 was administered via the tail vein, with treatment lasting 24 hours. Results: P110 significantly attenuated cerebral ischemia-reperfusion injury in male rats with transient middle cerebral artery occlusion, reduced infarct size, and improved neurological function.[3]

Solubility Information

Solubility	DMSO: 40.00 mg/mL (16.59 mM),Sonication is recommended. H2O: 1.00 mg/mL (0.41 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	0.4146 mL	2.0731 mL	4.1463 mL
5 mM	0.0829 mL	0.4146 mL	0.8293 mL
10 mM	0.0415 mL	0.2073 mL	0.4146 mL
50 mM	0.0083 mL	0.0415 mL	0.0829 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

Mukherjee R, et al. Drp1/Fis1-Dependent Pathologic Fission and Associated Damaged Extracellular Mitochondria Contribute to Macrophage Dysfunction in Endotoxin Tolerance. Crit Care Med. 2022 Jun 1;50(6):e504-e515.
Rios L, et al. Targeting an allosteric site in dynamin-related protein 1 to inhibit Fis1-mediated mitochondrial dysfunction. Nat Commun. 2023;14(1):4356. Published 2023 Jul 19.
Liu W, et al. Brain-targeted heptapeptide-loaded exosomes attenuated ischemia-reperfusion injury by promoting the transfer of healthy mitochondria from astrocytes to neurons. J Nanobiotechnology. 2022 May 23;20(1):242.

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