



SZABO SCANDIC

Part of Europa Biosite

Produktinformation



Forschungsprodukte & Biochemikalien



Zellkultur & Verbrauchsmaterial



Diagnostik & molekulare Diagnostik



Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!



Lieferung & Zahlungsart

siehe unsere [Liefer- und Versandbedingungen](#)

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

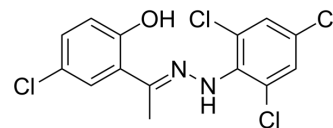
www.szabo-scandic.com

[linkedin.com/company/szaboscandic](https://www.linkedin.com/company/szaboscandic)



Mitochondrial fusion promoter M1

Cat. No.:	HY-111475
CAS No.:	219315-22-7
Molecular Formula:	C ₁₄ H ₁₀ Cl ₄ N ₂ O
Molecular Weight:	364.05
Target:	Mitochondrial Metabolism
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro	DMSO : 41.67 mg/mL (114.46 mM; ultrasonic and warming and heat to 60°C)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		2.7469 mL	13.7344 mL	27.4688 mL
		5 mM		0.5494 mL	2.7469 mL	5.4938 mL
		10 mM		0.2747 mL	1.3734 mL	2.7469 mL
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (5.71 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: 0.5 mg/mL (1.37 mM); Clear solution; Need ultrasonic					
	3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 0.5 mg/mL (1.37 mM); Suspended solution; Need ultrasonic					

BIOLOGICAL ACTIVITY

Description	Mitochondrial fusion promoter M1 is a mitochondrial dynamic modulator. Mitochondrial fusion promoter M1 preserves the mitochondrial function and promotes cellular respiration. Mitochondrial fusion promoter M1 alleviates cardiac and brain damage in rats with cardiac ischemia/reperfusion injury ^{[1][2][3]} .
In Vitro	Mitochondrial fusion promoter M1 (5-25 μM; 24 h) promotes mitochondrial elongation in both Mitofusin-1 and Mitofusin-2 knock-out fibroblasts ^[2] . Mitochondrial fusion promoter M1 (20 μM; 12 h) decreases mito ROS to 1.0±0.44 fold, enhances mitochondrial membrane potential from 0.29±0.05 fold to 0.5±0.07 fold and restores mitochondrial architecture in BRIN-BD11 pancreatic beta cells ^[3] .

	Mitochondrial fusion promoter M1 (20 μM; 12 h) prevents the impairment of oxygen consumption rate in pancreatic beta cells due to cholesterol exposure^[3]. Mitochondrial fusion promoter M1 (20 μM; 12 h) prevents the impairment of non-mitochondrial respiration and extracellular acidification rate (ECAR) in cholesterol enriched pancreatic beta cells^[3]. Mitochondrial fusion promoter M1 (20 μM; 12 h) restores Glucose Stimulated Insulin Secretion (GSIS) in cholesterol treated pancreatic beta cells^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.								
In Vivo	Mitochondrial fusion promoter M1 (2 mg/kg; i.v.) significantly protects rats with cardiac I/R injury against brain damage^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. <table> <tr> <td>Animal Model:</td><td>Male Wistar rats (250-300g) receiving cardiac ischemia/reperfusion (I/R)^[1]</td></tr> <tr> <td>Dosage:</td><td>2 mg/kg</td></tr> <tr> <td>Administration:</td><td>I.v. 15 minutes before cardiac I/R injury</td></tr> <tr> <td>Result:</td><td> Increased brain mitochondrial fusion. Increased blood-brain barrier (BBB) tight junction protein, and reduced macrophage infiltration in the brain. Reduced brain mitochondrial dysfunction and apoptosis, but it did not reduce mitochondrial oxidative stress. Reduced the expression of Alzheimer's disease (AD)-related proteins. </td></tr> </table>	Animal Model:	Male Wistar rats (250-300g) receiving cardiac ischemia/reperfusion (I/R) ^[1]	Dosage:	2 mg/kg	Administration:	I.v. 15 minutes before cardiac I/R injury	Result:	Increased brain mitochondrial fusion. Increased blood-brain barrier (BBB) tight junction protein, and reduced macrophage infiltration in the brain. Reduced brain mitochondrial dysfunction and apoptosis, but it did not reduce mitochondrial oxidative stress. Reduced the expression of Alzheimer's disease (AD)-related proteins.
Animal Model:	Male Wistar rats (250-300g) receiving cardiac ischemia/reperfusion (I/R) ^[1]								
Dosage:	2 mg/kg								
Administration:	I.v. 15 minutes before cardiac I/R injury								
Result:	Increased brain mitochondrial fusion. Increased blood-brain barrier (BBB) tight junction protein, and reduced macrophage infiltration in the brain. Reduced brain mitochondrial dysfunction and apoptosis, but it did not reduce mitochondrial oxidative stress. Reduced the expression of Alzheimer's disease (AD)-related proteins.								

CUSTOMER VALIDATION

- Ecotoxicol Environ Saf. 2023 Apr 5;256:114876.

See more customer validations on www.MedChemExpress.com

REFERENCES

- [1]. Surinkaew P, et, al. Mitochondrial Fusion Promoter Alleviates Brain Damage in Rats with Cardiac Ischemia/Reperfusion Injury. J Alzheimers Dis. 2020;77(3):993-1003.
- [2]. Wang D, et, al. A small molecule promotes mitochondrial fusion in mammalian cells. Angew Chem Int Ed Engl. 2012 Sep 10;51(37):9302-5.
- [3]. Asalla S, et, al. Restoring Mitochondrial Function: A Small Molecule-mediated Approach to Enhance Glucose Stimulated Insulin Secretion in Cholesterol Accumulated Pancreatic beta cells. Sci Rep. 2016 Jun 10;6:27513.

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

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