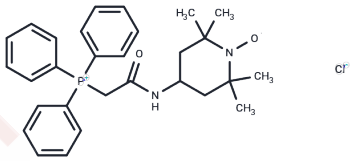
Data Sheet (Cat.No.T19428)

TargetMol

Mito-TEMPO

Chemical Properties



CAS No. : 1334850-99-5

Formula: C29H35N2O2P.Cl

Molecular Weight: 510.03

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	Mito-TEMPO, a mitochondria-targeted superoxide dismutase mimetic, scavenges superoxide and alkyl radicals, preventing mitochondrial oxidation, necrosis, and apoptosis.
Targets(IC50)	Reactive Oxygen Species,Mitochondrial Metabolism,ROS
In vitro	**METHODS**: Human neuroblastoma cells SH-SY5Y were treated with Mito-TEMPO (25-100 μM) for 24 h. Cell viability was detected using MTT assay. **RESULTS**: No cytotoxic effect was shown on the cells in the Mito-TEMPO-treated group, and a significant increase in cell viability was detected after Mito-TEMPO treatment. [1] **METHODS**: Normal rat proximal renal tubular epithelial cell line NRK-52E was pretreated with Mito-TEMPO (10 μM) for 1 h, then stimulated with oxalate (700 μM) for 1 h. The mitochondrial membrane potential was detected by using MMP assay kit (JC-1). **RESULTS**: The control cells showed bright red fluorescence. Compared with the control, oxalate treatment attenuated the red fluorescence, and these changes were reversed by pretreatment with Mito-TEMPO. The **RESULTS** suggest that oxalate induces mitochondrial dysfunction, and Mito-TEMPO can inhibit this effect. [2]
In vivo	**METHODS**: To investigate the protective effect against hepatotoxicity, APAP (300 mg/kg) was intraperitoneally injected into C57BL/6J mice, and Mito-TEMPO (20 mg/kg in saline) was injected intraperitoneally 1.5-3 h later. **RESULTS**: Mito-TEMPO had a protective effect on the late hepatotoxicity of APAP. [3] **METHODS**: To investigate the effects on coronary vasodilatation and endothelial SK channel activity, Mito-TEMPO (1 mg/kg in saline) was intraperitoneally injected into C57BL/6J mice with or without diabetes once daily for four weeks. **RESULTS**: After 4 weeks of treatment with Mito-TEMPO, diabetic mice showed significantly improved endothelium-dependent diastolic responses of coronary arteries to ADP or NS309 and endothelial SK channel currents compared to untreated diabetic mice. [4]

Solubility Information

Solubility	H2O: 60 mg/mL (117.64 mM),Sonication is recommended. DMSO: 255 mg/mL (499.97 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 5 mg/mL (9.8 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.9607 mL	9.8033 mL	19.6067 mL
5 mM	0.3921 mL	1.9607 mL	3.9213 mL
10 mM	0.1961 mL	0.9803 mL	1.9607 mL
50 mM	0.0392 mL	0.1961 mL	0.3921 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

Mukem S, et al. Mito-Tempo suppresses autophagic flux via the PI3K/Akt/mTOR signaling pathway in neuroblastoma SH-SY5Y cells. Heliyon. 2021 Jun 15;7(6):e07310.
Zhao X, Miao G, Zhang L, et al. Chlamydia pneumoniae Infection Induces Vascular Smooth Muscle Cell Migration and Atherosclerosis Through Mitochondrial Reactive Oxygen Species-Mediated JunB-Fra-1 Activation. Frontiers in Cell and Developmental Biology. 2022, 10: 879023-879023
Tian C, Han X, He L, et al. Transient receptor potential ankyrin 1 contributes to the ATP-elicited oxidative stress and inflammation in THP-1-derived macrophage. Molecular and Cellular Biochemistry. 2020: 1-14
Zhang J, et al. MitoTEMPO Prevents Oxalate Induced Injury in NRK-52E Cells via Inhibiting Mitochondrial Dysfunction and Modulating Oxidative Stress. Oxid Med Cell Longev. 2017;2017:7528090.
Du K, et al. Mito-tempo protects against acute liver injury but induces limited secondary apoptosis during the late phase of acetaminophen hepatotoxicity. Arch Toxicol. 2019 Jan;93(1):163-178.
Wang Y, He X, Xue M, et al.Germacrone protects renal tubular cells against ferroptotic death and ROS release by re-activating mitophagy in diabetic nephropathy.Free Radical Research.2023 (just-accepted): 1-36.
Xing H, et al. Chronic Inhibition of mROS Protects Against Coronary Endothelial Dysfunction in Mice With Diabetes. Front Cell Dev Biol. 2021 Feb 18;9:643810.
Yan Z, Niu L, Wang S, et al.Intestinal Piezo1 aggravates intestinal barrier dysfunction during sepsis by mediating Ca2+ influx.Journal of Translational Medicine.2024, 22(1): 1-12.
Tang X, Zheng N, Lin Q, et al.Hypoxia-preconditioned bone marrow-derived mesenchymal stem cells protect neurons from cardiac arrest-induced pyroptosis.Neural Regeneration Research.2025: 10.4103.
Xu Y, Li G, Ge D, et al.Mitochondrial dysfunction in kidney stones and relief of kidney stones after reducing mtROS. Urolithiasis.2024, 52(1): 117.
Liu B, Chen L, Gao M, et al.A Comparative Study of the Efficiency of Mitochondria-Targeted Antioxidants MitoTEMPO and SKQ1 under Oxidative Stress.Free Radical Biology and Medicine.2024
XIA L, YUAN H, GAO Z, et al.The role of mitochondrial reactive oxygen species in initiating mitochondrial damage and inflammation in wasp-venom-induced acute kidney injury.Journal of Toxicologic Pathology.2024
Xu H, Chen X, Luo S, et al.Cardiomyocyte-specific Piezo1 deficiency mitigates ischemia-reperfusion injury by preserving mitochondrial homeostasis.Redox Biology.2024: 103471.

Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481