

Product Data Sheet

Product Name: MitoPQ
Cat. No.: GC44204

Chemical Properties

Cas. No. 1821370-28-8

Chemical Name 1-methyl-1'-(10-(triphenylphosphonio)decyl)-[4,4'-bipyridine]-1,1'-dium iodide

SMILES C[N+](C=C1)=CC=C1C(C=C2)=CC=[N+]2CCCCCCCCCCC[P+](C3=CC=CC=C3)(C4=CC=CC=C4)C5=CC=CC=C5.[I-].[I-].[I-]

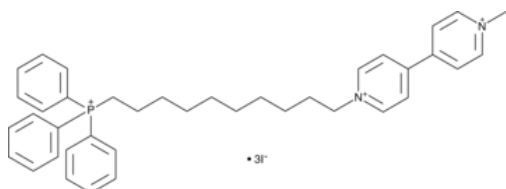
Formula $C_{39}H_{46}N_2P \cdot 3I$ M.Wt 954.5

Solubility 10mM in ethanol or 100mM in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines	C2C12 cells (mouse myoblasts cell line)
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Preparation Method	In C2C12 myoblasts, mitochondrial superoxide and hydrogen peroxide production were monitored by live cell imaging using the fluorescent dye MitoSOX. Individual cells were tracked through time, and fluorescence intensity was analyzed using Fiji imaging processing and analysis software. C2C12 cells were treated with 5μM MitoPQ or PQ, and changes in MitoSOX fluorescence intensity were observed over 20min.
Reaction Conditions	5μM; 20min
Applications	MitoPQ increased MitoSOX fluorescence, and this oxidation increased over time. In contrast, PQ was unable to increase MitoSOX oxidation over this timescale, even at a thousand times greater concentration than used for MitoPQ.
Animal experiment [2]:	
Animal models	C57BL/6J

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Preparation Method	Mice were fasted for 16h and then received 0.16mg/kg MitoPQ by i.p. injection. 1.5h later, mice were anaesthetized with 100mg/kg ketamine and 10mg/kg xylazine and then injected with 0.75U/kg insulin into the portal vein. Livers were collected 3min later and immediately frozen in liquid N ₂ . Epididymal adipose tissue and gastrocnemius were next collected and frozen until protein extraction.
Dosage form	0.16mg/kg; i.p.
Applications	MitoPQ decreased hepatic insulin signalling in vivo, as indicated by the reduced levels of phosphorylated insulin receptor (IR), AKT and GSK3 α in the liver.
References: [1] Robb EL, Gawel JM, Aksentijević D, Cochemé HM, Stewart TS, Shchepinova MM, Qiang H, Prime TA, Bright TP, James AM, Shattock MJ. Selective superoxide generation within mitochondria by the targeted redox cyler MitoParaquat. Free Radical Biology and Medicine. 2015 Dec 1;89:883-94. [2] Goncalves RL, Wang ZB, Riveros JK, Parlakgöl G, Inouye KE, Lee GY, Fu X, Saksi J, Rosique C, Hui ST, Coll M. CoQ imbalance drives reverse electron transport to disrupt liver metabolism. Nature. 2025 May 28:1-9.	

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Background

MitoPQ (MitoParaquat) is a mitochondria-targeted redox cycler that selectively increases superoxide production within the mitochondrial matrix in vivo and in cells^[1]. MitoPQ accumulates in the mitochondrial matrix and generates $O_2^{\cdot -}$ through redox cycling at the flavin site of complex I (Fig. 1)^[1,2]. MitoPQ is commonly used to study the role of mitochondrial superoxide production in health and disease in both cells and in vivo^[3,4].

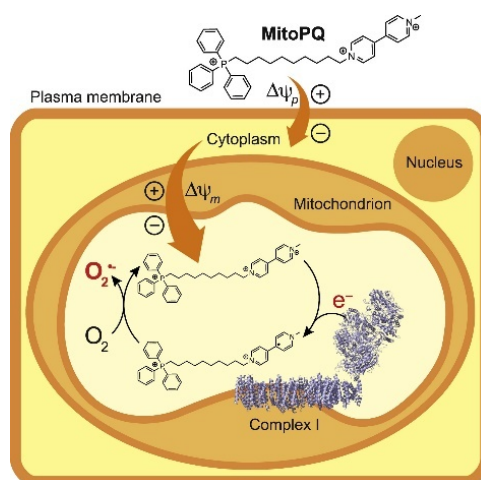


Fig. 1. Rationale for the development of MitoParaquat^[1,2]. MitoParaquat (MitoPQ) is composed of a redox cycling paraquat moiety, and a hydrophobic carbon chain linking it to a mitochondria-targeting triphenylphosphonium cation. MitoPQ is accumulated by mitochondria driven by the plasma ($\Delta\psi_p$) and mitochondrial ($\Delta\psi_m$) membrane potentials. Within the matrix, the dicationic viologen component of MitoPQ is reduced to a radical monocation by one-electron reduction at the flavin site of complex I. The radical monocation then reacts very rapidly with O_2 to generate superoxide. This localized redox cycling leads to the selective production of superoxide within the mitochondrial matrix.

In vitro, treatment of C2C12 myoblasts with MitoPQ (5 μ M) for 20min time-dependently and significantly increased MitoSOX fluorescence intensity, whereas paraquat (PQ) at equivalent conditions failed to increase MitoSOX oxidation within this timeframe. Treatment of HCT116 cells with MitoPQ (1-10 μ M) for 24h induced cell death in a dose-dependent manner, with significantly higher toxicity compared to PQ^[1]. Treatment of Raw264.7 cells with MitoPQ (0.5 μ M) for 16h

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significantly disrupted the mitochondrial membrane potential, an effect that was attenuated by the addition of 10mM N-acetylcysteine (NAC)^[5]. Treatment of 3T3-L1 adipocytes with MitoPQ (10μM) for 2h specifically increased mitochondrial superoxide and hydrogen peroxide levels without affecting global cellular respiration^[6].

In vivo, acute intraperitoneal injection of MitoPQ (0.16mg/kg) into wild-type mice fasted for 16h significantly impaired glucose tolerance 2h post-injection. Intraperitoneal injection of MitoPQ (0.16mg/kg) in wild-type mice for 1.5h impaired hepatic insulin signaling in vivo, as evidenced by reduced levels of phosphorylated insulin receptor (IR), AKT, and GSK3α^[7]. Daily intraperitoneal injection of MitoPQ (0.1mg/kg/day) for 7 days in cardiomyocyte-specific Nrf3 knockout mice significantly reduced survival rates and attenuated the beneficial effects of Nrf3 gene deletion on cardiac function and remodeling after myocardial infarction^[8].

References:

- [1] ROBB E L, GAWEL J M, AKSENTIJEVIĆ D, et al. Selective superoxide generation within mitochondria by the targeted redox cyler MitoParaquat[J]. Free Radical Biology and Medicine, 2015, 89: 883-894.
- [2] COCHENÉ H M, MURPHY M P. Complex I is the major site of mitochondrial superoxide production by paraquat[J]. Journal of Biological Chemistry, 2008, 283(4): 1786-1798.
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- [4] GOLEVA T N, LYAMZAEV K G, ROGOV A G, et al. Mitochondria-targeted 1, 4-naphthoquinone (SkQN) is a powerful prooxidant and cytotoxic agent[J]. Biochimica et Biophysica Acta (BBA)-Bioenergetics, 2020, 1861(8): 148210.
- [5] CHOWDHURY A R, ZIELONKA J, KALYANARAMAN B, et al. Mitochondria-targeted paraquat and metformin mediate ROS production to induce multiple pathways of retrograde signaling: A dose-dependent phenomenon[J]. Redox Biology, 2020, 36: 101606.
- [6] FAZAKERLEY D J, MINARD A Y, KRYCER J R, et al. Mitochondrial oxidative stress causes insulin resistance without disrupting oxidative phosphorylation[J]. Journal of Biological Chemistry,

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[8] CHEN Q, ZHENG A, XU X, et al. Nrf3-Mediated mitochondrial superoxide promotes cardiomyocyte apoptosis and impairs cardiac functions by suppressing Pitx2[J]. Circulation, 2025, 151(14): 1024-1046.

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