Mitochonic acid 5

Cat. No.: HY-111536

CAS No.: 1354707-41-7

Molecular Formula: $C_{18}H_{13}F_2NO_3$

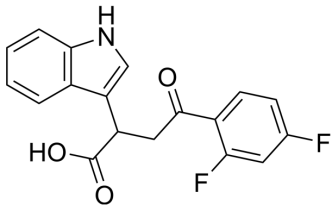
Molecular Weight: 329.3

Target: Mitochondrial Metabolism; Oxidative Phosphorylation

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (303.67 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0367 mL	15.1837 mL	30.3674 mL
5 mM	0.6073 mL	3.0367 mL	6.0735 mL
10 mM	0.3037 mL	1.5184 mL	3.0367 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.59 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.59 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.59 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mitochonic acid 5 binds mitochondria and ameliorates renal tubular and cardiac myocyte damage. Mitochonic acid 5 modulates mitochondrial ATP synthesis.

IC_{50} & Target

Mitochondrial Metabolism[1]

In Vitro	Mitochonic acid 5 (MA-5) modulates mitochondrial ATP synthesis independently of oxidative phosphorylation and the electron transport chain. Mitochondrial dysfunction causes increased oxidative stress and depletion of ATP, which are involved in the etiology of a variety of renal diseases[1]. Mitochonic acid 5 (MA-5), which is derived from the plant growth hormone indole-3-acetic acid, can protect mitochondrial function by regulating energy metabolism and reducing mitochondrial oxidative stress. To observe the protective role of Mitochonic acid 5 in microglia under inflammatory conditions, TNFα is applied. Subsequently, the MTT assay is used to evaluate cell viability. In response to the TNFα treatment, cell viability significantly decreases. However, this effect is dose-dependently inhibited by Mitochonic acid 5 treatment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Administration of Mitochonic acid 5 (MA-5) to an ischemia-reperfusion injury model and a cisplatin-induced nephropathy model improved renal function. To examine the tissue-protective effect of Mitochonic acid 5, the oral bioavailability is examined. Oral administration of Mitochonic acid 5 increases the plasma concentration in a dose-response manner at the peak time of 1 hour[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	The mouse BV-2 cells used in this study are cultured in L-DMEM supplemented with 10% fetal bovine serum (FBS) at 37°C in an atmosphere with 5% CO_2. To induce inflammatory injury, cells are treated with 10 ng/mL TNFα for about 12 h. Mitochonic acid 5 (0-10 μM) is incubated with BV-2 cells for about 12 h with TNFα treatment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice[1] For evaluation of the blood concentrations of Mitochonic acid 5 (MA-5), Mitochonic acid 5 is orally administered at doses of 25, 50, or 150 mg/kg to C57/BL 6 mice, and blood samples are collected at the designated times. After 1 hour, the mice are euthanized. The blood concentration of Mitochonic acid 5 is determined by LC/MS/MS[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Small. 2023 Jan 12;e2207194.
- Cell Death Dis. 2022 Jun 20;13(6):557.
- Life Sci. 2023 Apr 1;121653.

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REFERENCES

[1]. Suzuki T, et al. Mitochonic Acid 5 Binds Mitochondria and Ameliorates Renal Tubular and Cardiac Myocyte Damage. J Am Soc Nephrol. 2016 Jul;27(7):1925-32.

[2]. Lei Q, et al. Mitochonic acid 5 activates the MAPK-ERK-yap signaling pathways to protect mouse microglial BV-2 cells against TNFα-induced apoptosis via increased Bnip3-related mitophagy. Cell Mol Biol Lett. 2018 Apr 5;23:14.

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

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