

Product Data Sheet

Product Name: Mitochonic acid 5 (MA-5)

Cat. No.: GC31123

Chemical Properties

Cas. No. 1354707-41-7

SMILES O=C(O)C(CC(C1=CC=C(F)C=C1F)=O)C2=CNC3=C2C=CC=C3

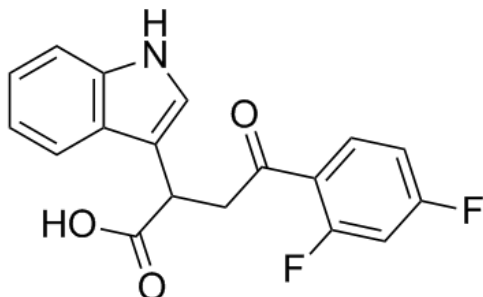
Formula $C_{18}H_{13}F_2NO_3$ M.Wt 329.3

Solubility DMSO : ≥ 106.66 mg/mL (323.90 mM) 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

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

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Cell experiment: 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% CO₂. To induce inflammatory injury, cells are treated with 10 ng/mL TNF α for about 12 h. Mitochonic acid 5 (0-10 μ M) is incubated with BV-2 cells for about 12 h with TNF α treatment[2].

Animal experiment: 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].

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 TNF α -induced apoptosis via increased Bnip3-related mitophagy. Cell Mol Biol Lett. 2018 Apr 5;23:14.

Background

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

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, TNF α is applied. Subsequently, the MTT assay is used to evaluate cell

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viability. In response to the TNF α treatment, cell viability significantly decreases. However, this effect is dose-dependently inhibited by Mitochondic acid 5 treatment[2].

Administration of Mitochondic 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 Mitochondic acid 5, the oral bioavailability is examined. Oral administration of Mitochondic acid 5 increases the plasma concentration in a dose-response manner at the peak time of 1 hour[1].

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

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