Mitochondric acid 5

Cat. No.: HY-111536
CAS No.: 1354707-41-7
Molecular Formula: C₁₈H₁₃F₂NO₃
Molecular Weight: 329.3
Target: Mitochondrial Metabolism
Pathway: Metabolic Enzyme/Protease
Storage: Powder -20°C 3 years
4°C 2 years
In solvent -80°C 6 months
-20°C 1 month

Solvent & Solubility

In Vitro DMSO: ≥ 106.66 mg/mL (323.90 mM)
*“≥” means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td></td>
<td>3.0367 mL</td>
<td>15.1837 mL</td>
<td>30.3674 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td></td>
<td>0.6073 mL</td>
<td>3.0367 mL</td>
<td>6.0735 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td></td>
<td>0.3037 mL</td>
<td>1.5184 mL</td>
<td>3.0367 mL</td>
</tr>
</tbody>
</table>

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

BIOLOGICAL ACTIVITY

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

IC₅₀ & Target Mitochondrial Metabolism[1]

In Vitro Mitochondric 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]. Mitochondric 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 Mitochondric 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 Mitochondric acid 5 treatment[2].
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\(^1\).

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 TNF\(\alpha\) for about 12 h. *Mitochonic acid 5* (0-10 \(\mu\)M) is incubated with BV-2 cells for about 12 h with TNF\(\alpha\) 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.

REFERENCES


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

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