Mitochondric acid 5

Cat. No.: HY-111536
CAS No.: 1354707-41-7
Molecular Formula: C₁₈H₁₃F₂NO₃
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 : ≥ 106.66 mg/mL (323.90 mM)
H₂O : < 0.1 mg/mL (insoluble)

* “≥” means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>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>3.0367 mL</td>
<td>15.1837 mL</td>
<td>30.3674 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.6073 mL</td>
<td>3.0367 mL</td>
<td>6.0735 mL</td>
</tr>
<tr>
<td>10 mM</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.

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
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
Mitochondrial 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. Mitochondrial 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 Mitochondrial acid 5 in microglia under inflammatory conditions, TNF-α is applied. Subsequently, the MTT assay is used to evaluate cell viability. In response to the TNF-α treatment, cell viability significantly decreases. However, this effect is dose-dependently inhibited by Mitochondrial acid 5 treatment.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### In Vivo
Administration of Mitochondrial 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 Mitochondrial acid 5, the oral bioavailability is examined. Oral administration of Mitochondrial acid 5 increases the plasma concentration in a dose-response manner at the peak time of 1 hour.

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### PROTOCOL

#### Cell Assay

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. Mitochondrial acid 5 (0-10 μM) is incubated with BV-2 cells for about 12 h with TNF-α treatment.

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#### Animal Administration

For evaluation of the blood concentrations of Mitochondrial acid 5 (MA-5), Mitochondrial acid 5 is orally administered at doses of 25, 50, or 150 mg/kg to C57/BL6 mice, and blood samples are collected at the designated times. After 1 hour, the mice are euthanized. The blood concentration of Mitochondrial acid 5 is determined by LC/MS/MS.

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### CUSTOMER VALIDATION

- Small. 2023 Jan 12;e2207194.
- Life Sci. 2023 Apr 1;121653.

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### REFERENCES


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

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