Alda-1

Cat. No.: HY-18936
CAS No.: 349438-38-6
Molecular Formula: C₁₅H₁₁Cl₂NO₃
Molecular Weight: 324.16
Target: Aldehyde Dehydrogenase (ALDH)
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: ≥ 51 mg/mL (157.33 mM)
* "≥" means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Solvent Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparing Stock Solutions</td>
<td>1 mM</td>
<td>3.0849 mL</td>
<td>15.4245 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.6170 mL</td>
<td>3.0849 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3085 mL</td>
<td>1.5424 mL</td>
</tr>
</tbody>
</table>

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

In Vivo
1. Alda-1 is dissolved in solution (5% DMSO+45% PEG400+water)[5].

BIOLOGICAL ACTIVITY

Description
Alda-1 is a potent ALDH2 agonist, which significantly improves ALDH2 activity.

In Vivo
Alda-1 treatment results in a significant decrease of 4-HNE-protein content in the plasma of apoE−/− mice. Alda-1 administration leads to a slight increase in gene expression related to neurogenesis (Nog), mitochondrial biogenesis (CYTB, ND1), and apoptosis (Bax, Gsk3b) in the Hp of apoE−/− mice. Alda-1 administration leads to 2 and 10 differentially expressed proteins in the FCx and Hp of apoE−/− mice, respectively[2]. Alda-1 (1.5 mg/kg, b.w., i.p.) administration significantly increases the climbing time, tends to reduce the immobility time and increases the swimming time of the prenatally stressed rats in the forced swim test. Moreover, treatment of prenatally stressed rats with Alda-1 significantly increases number of entries into the open arms of the maze and the time spent therein, as assessed by elevated plus-maze test[2]. Alda-1 (8.5 mg/kg, i.p.) with glucose significantly lowers 4-HNE and FJB-positive cells in the cerebral cortex of Alda-1-treated rats than in DMSO-treated rats 24 h after glucose administration.
Alda-1 (10 mg/kg per day) treatment prevents aldehydic overload, mitochondrial dysfunction and improves ventricular function in post-MI cardiomyopathy rats.

**PROTOCOL**

**Cell Assay**

Spleen cells (4×10⁶ cells/mL) are stimulated by optimal concentrations of concanavalin A (Con A; 2.5 μg/mL and 0.6 μg/mL) and lipopolysaccharide (LPS, 5 μg/mL) and are incubated in 96-well plates at final volume of 0.2 mL for 72 h. Cell proliferation is determined by adding 0.5 μCi of [³H]-thymidine per well at 16 h before the end of the incubation. The cultures are harvested with an automatic cell harvester, and [³H] thymidine incorporation is assessed using a liquid scintillation counter.

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

**Animal Administration**

After behavioral verification at three months of age, the animals are divided into the following four groups: control, control + Alda-1, prenatally stressed and prenatally stressed + Alda-1 (6 animals per group). Alda-1 injections are given intraperitoneally (i.p.) once daily at a dose of 1.5 mg/kg b.w. (dissolved in 1 mL/kg b.w. DMSO/water 50/50) for 14 days. At the same time, the control and prenatally stressed rats receive 1 mL/kg b.w. DMSO/water 50/50. The injections of Alda-1 and vehicle are given between 10 a.m and 11 a.m. In the last five days of Alda-1 treatment the behavioral parameters in the elevated plus maze test and then in the forced swim test are measured.

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**CUSTOMER VALIDATION**

- J Immunol Res. 7 August 2018.

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**REFERENCES**


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

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