Alda-1

Cat. No.: HY-18936

CAS No.: 349438-38-6

Molecular Formula: $C_{15}H_{11}Cl_2NO_3$

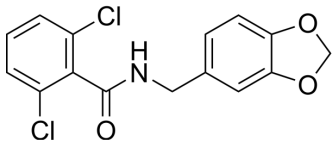
Molecular Weight: 324.16

Target: Aldehyde Dehydrogenase (ALDH); Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

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.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0849 mL	15.4245 mL	30.8490 mL
	5 mM	0.6170 mL	3.0849 mL	6.1698 mL
	10 mM	0.3085 mL	1.5424 mL	3.0849 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 24 mg/mL (74.04 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Alda-1 is a potent and selective ALDH2 agonist, which activates wild-type ALDH2 and restores near wild-type activity to ALDH2*2.

IC_{50} & Target

ALDH2

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[1].

Alda-1 (1.5 mg/kg, b.w., IP) 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; IP) 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[3]. Alda-1 (10 mg/kg per day) treatment prevents aldehydic overload, mitochondrial dysfunction and improves ventricular function in post-MI cardiomyopathy rats[4].

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

PROTOCOL

Cell Assay [2]

Spleen cells (4×10^6 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 [^{3}H]-thymidine per well at 16 h before the end of the incubation. The cultures are harvested with an automatic cell harvester, and [^{3}H] thymidine incorporation is assessed using a liquid scintillation counter.

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Animal Administration [2]

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

- Carbohydr Polym. 1 July 2022, 119326.
- Cell Death Dis. 2023 Jan 20;14(1):45.
- Arterioscler Thromb Vasc Biol. 2019 Nov;39(11):2303-2319.
- Free Radic Biol Med. 2023 Dec 15:212:34-48.
- Anal Chem. 2021 Dec 28.

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REFERENCES

[1]. Stachowicz A, et al. Proteomic Analysis of Mitochondria-Enriched Fraction Isolated from the Frontal Cortex and Hippocampus of Apolipoprotein E Knockout Mice Treated with Alda-1, an Activator of Mitochondrial Aldehyde Dehydrogenase (ALDH2). Int J Mol Sci.

[2]. Stachowicz A, et al. The impact of mitochondrial aldehyde dehydrogenase (ALDH2) activation by Alda-1 on the behavioral and biochemical disturbances in animal model of depression. Brain Behav Immun. 2016 Jan;51:144-53.

[3]. Ikeda T, et al. Effects of Alda-1, an Aldehyde Dehydrogenase-2 Agonist, on Hypoglycemic Neuronal Death. PLoS One. 2015 Jun 17;10(6):e0128844.

[4]. Gomes KM, et al. Aldehydic load and aldehyde dehydrogenase 2 profile during the progression of post-myocardial infarction cardiomyopathy: benefits of Alda-1. Int J Cardiol. 2015 Jan 20;179:129-138.

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

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