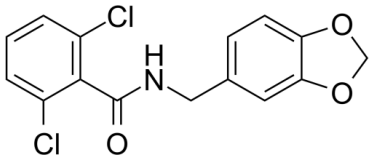
Data Sheet

Product Name:	Alda-1
Cat. No.:	CS-4995
CAS No.:	349438-38-6
Molecular Formula:	C15H11Cl2NO3
Molecular Weight:	324.16
Target:	Aldehyde Dehydrogenase (ALDH); Apoptosis
Pathway:	Apoptosis; Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 51 mg/mL (157.33 mM)



BIOLOGICAL ACTIVITY:

Alda-1 is a potent **ALDH2** agonist, which activates wild-type ALDH2 and restores near wild-type activity to ALDH2*2. IC50 & Target: ALDH2[1] **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., 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[3]. Alda-1 (10 mg/kg per day) treatment prevents aldehydic overload, mitochondrial dysfunction and improves ventricular function in post-MI cardiomyopathy rats[4].

PROTOCOL (Extracted from published papers and Only for reference)

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. **Animal Administration:** Alda-1 is dissolved in 1 mL/kg b.w. DMSO/water 50/50.[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.

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.

CAIndexNames:

Benzamide, N-(1,3-benzodioxol-5-ylmethyl)-2,6-dichloro-

SMILES:

O=C(NCC1=CC=C(OCO2)C2=C1)C3=C(Cl)C=CC=C3Cl

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