

Product Data Sheet

Product Name: L- α -Aminoadipic Acid

Cat. No.: GC13740

Chemical Properties

Cas. No. 1118-90-7

Chemical Name 2S-amino-hexanedioic acid

SMILES OC(CCC[C@H](N)C(=O)O)=O

Formula $C_6H_{11}NO_4$ M.Wt 161.2

Solubility $\leq 10\text{mg/ml}$ in 1N HCl; 0.5mg/ml in PBS Storage Store at -20°C , protect from light

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

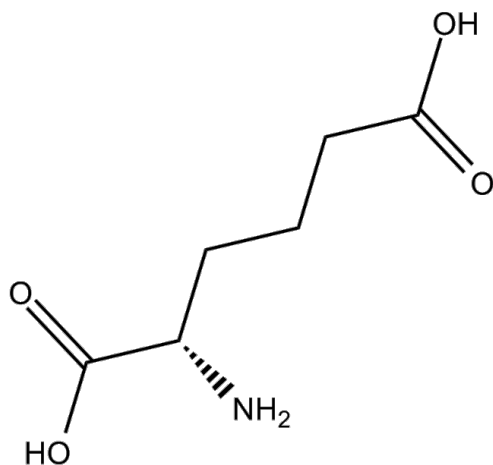
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Protocol

Cell experiment [1]:

Cell lines Postnatal mouse cerebellum

Preparation Method Cells dissociated from early postnatal mouse cerebellum were maintained in vitro for 4 days and then exposed to L-α-Aminoadipic Acid (0.34mM; 40h), then cultures grown on poly-D-lysine coated glass coverslips were subjected to immunohistochemistry.

Reaction
Conditions 0.34mM; 40h

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Applications After a 2h exposure to 0.34mM L- α -Aminoadipic Acid, approximately 80% of the astrocytes with characteristic nuclear and cytoplasmic swelling recovered, whereas after a 40h exposure, the same concentration killed 97% of the astrocytes.

Animal experiment [2]:

Animal models Adult male Wistar rats

Preparation Method Experimental group (L- α -Aminoadipic Acid), with stereotaxic surgery, received L- α -Aminoadipic Acid at dose of 25 μ g/ μ L once daily (1 μ L/rat (0.5 μ L on each side) of L- α -Aminoadipic Acid were injected into the CA1 hippocampal area by a Hamilton micro-syringe over one minute) for three consecutive days and evaluated for behavioral test. The number of GFAP-ir astrocytes was counted in a 30000 μ m² area at three regions (CA1, CA3, and DG regions) of the hippocampus by the cellSens standard 1.14 software.

Dosage form 25 μ g/ μ L/day; 1 μ L/rat (0.5 μ L on each side); 3 days; intrahippocampal injection

Applications Microinjection of L- α -Aminoadipic Acid into the CA1 subfield of the hippocampus significantly decreased the step-through latency time in the passive avoidance test, decreased time spent in the open arm, and increased time spent in the closed arm in the elevated plus maze test. Also, the administration of L- α -Aminoadipic Acid significantly declined the density of GFAP-ir astrocytes in the hippocampus.

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References:

- [1] HUCK S, GRASS F, HATTEN M E. Gliotoxic effects of α -amino adipic acid on monolayer cultures of dissociated postnatal mouse cerebellum[J]. Neuroscience, 1984, 12(3): 783-791.
- [2] JAHANSHAH M, ELYASI L, NIKMAHZAR E. L- α -amino adipic Acid-Induced Astrocytes Inhibition in the Hippocampal CA1 Region Leads to Anxiety-like Behavior and Memory Impairment[J]. Basic and Clinical Neuroscience, 2025, 16(1).

Background

L- α -Amino adipic Acid is a lysine metabolite with neuroexcitatory properties, which competitively inhibits glutamate transporters with a K_i value of $192\mu\text{M}$ ^[1,2]. L- α -Amino adipic Acid is one of the isomeric forms of 2-Amino adipic acid and is crucial for neurotransmitter regulation and glutamate transport inhibition^[3]. L- α -Amino adipic Acid is commonly used in research to selectively inhibit astrocyte function for investigating their physiological and pathological roles, and also serves as a precursor for β -lactam antibiotics in the development of novel antibiotics^[4,5].

In vitro, treatment of mouse cerebellar cells with L- α -Amino adipic Acid (0.34mM) for 2h induced reversible swelling in approximately 80% of astrocytes; prolonged treatment for 40h resulted in the death of 97% of astrocytes^[6]. In rat amygdala slices, treatment with L- α -Amino adipic Acid (1mM) for $\geq 90\text{min}$ increased the nuclear area of glial fibrillary acidic protein (GFAP)-positive astrocytes and significantly reduced the N-methyl-D-aspartate receptor (NMDAR)-mediated excitatory postsynaptic currents (EPSCs) evoked by strong stimulation in the thalamo-LA pathway^[7].

In vivo, intravitreal injection of L- α -Amino adipic Acid ($8\mu\text{M}/\text{eye}$; $50\mu\text{L}$) in carp led to a reduction in S-potentials and a relatively lower resting potential in retinal horizontal cells within 2-4h; the voltage characteristics of horizontal cells returned to normal within 1-2 days^[8]. In adult male Wistar rats, bilateral microinjection of L- α -Amino adipic Acid ($25\mu\text{g}/\mu\text{L}/\text{day}$; $1\mu\text{L}/\text{rat}$ ($0.5\mu\text{L}$ per side)) into the hippocampal CA1 region for 3 days significantly reduced the number of GFAP-immunoreactive (GFAP-ir) astrocytes in the hippocampus, resulting in impaired passive avoidance memory and increased anxiety-like behavior^[9].

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- [1] WU H Q, UNGERSTEDT U, SCHWARCZ R. I- α -Aminoadipic acid as a regulator of kynurenic acid production in the hippocampus: a microdialysis study in freely moving rats[J]. European journal of pharmacology, 1995, 281(1): 55-61.
- [2] MCBEAN G J. Inhibition of the glutamate transporter and glial enzymes in rat striatum by the gliotoxin, alpha aminoadipate[J]. British journal of pharmacology, 1994, 113(2): 536.
- [3] SHI W, YANG Z, FU P, et al. Metabolite 2-aminoadipic acid: implications for metabolic disorders and therapeutic opportunities[J]. Frontiers in Pharmacology, 2025, 16: 1569020.
- [4] ISOBE K, TOKUTA K, NARITA Y, et al. Production of N α -benzyloxycarbonyl-L-aminoadipic acid and N α -benzyloxycarbonyl-D-aminoadipic acid with Rhodococcus sp. AIU Z-35-1[J]. Journal of Molecular Catalysis B: Enzymatic, 2004, 32(1/2): 27-32.
- [5] GUIDETTI P, SCHWARCZ R. Determination of α -aminoadipic acid in brain, peripheral tissues, and body fluids using GC/MS with negative chemical ionization[J]. Molecular brain research, 2003, 118(1/2): 132-139.
- [6] HUCK S, GRASS F, HATTEN M E. Gliotoxic effects of α -aminoadipic acid on monolayer cultures of dissociated postnatal mouse cerebellum[J]. Neuroscience, 1984, 12(3): 783-791.
- [7] LI Y, SACCHI S, POLLEGIONI L, et al. Identity of endogenous NMDAR glycine site agonist in amygdala is determined by synaptic activity level[J]. Nature communications, 2013, 4(1): 1760.
- [8] SUGAWARA K, TORIGOE K, OKOYAMA S, et al. Neurotoxic effects of I- α -aminoadipic acid on the carp retina: a long term observation[J]. Neuroscience, 1990, 36(1): 155-163.
- [9] JAHANSHAH M, ELYASI L, NIKMAHZAR E. L- α -aminoadipic Acid-Induced Astrocytes Inhibition in the Hippocampal CA1 Region Leads to Anxiety-like Behavior and Memory Impairment[J]. Basic and Clinical Neuroscience, 2025, 16(1).

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