D-AP5

Cat. No.: HY-100714A

CAS No.: 79055-68-8

Molecular Formula: $C_5H_{12}NO_5P$

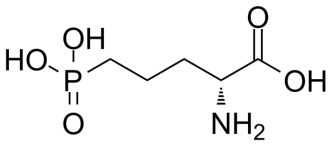
Molecular Weight: 197.13

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 2 mg/mL (10.15 mM; Need ultrasonic)
DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)
Ethanol : < 1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.0728 mL	25.3640 mL	50.7279 mL
5 mM	1.0146 mL	5.0728 mL	10.1456 mL
10 mM	0.5073 mL	2.5364 mL	5.0728 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (507.28 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

D-AP5 (D-APV) is a selective and competitive NMDA receptor antagonist with a K_d of 1.4 μM. D-AP5 (D-APV) inhibits the glutamate binding site of NMDA receptors[1][2].

IC_{50} & Target

NMDA[1]

In Vivo

D-AP5 is a NMDA receptor antagonist. Chronic intraventricular infusion of D-AP5 causes a parallel dose-dependent impairment of spatial learning and long-term potentiation (LTP) in vivo. Intracerebral concentrations of D-AP5 fail to induce any measurable sensorimotor disturbance when spatial learning is prevented[2].
D-AP5 infusion is associated with a progressive reduction in swim speed over trials. D-AP5 causes sensorimotor disturbances in the spatial task, but these gradually worsened as the animals fail to learn. Rats treated with D-AP5 show a delay-dependent deficit in spatial memory in the delayed matching-to-place protocol for the water maze[3].

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

PROTOCOL

Animal Administration [1]

Male Lister hooded rats are used in this study. They are housed individually and have free access to food and water at all times except for 12 hr prior to surgery. Animals are implanted with 30 mM D-AP5 (N=6) and tested in the water maze[1].

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

CUSTOMER VALIDATION

- Brain Behav Immun. 2023 Jun 5;S0889-1591(23)00141-1.
- Theranostics. 2023 May 11;13(9):2946-2961.
- Theranostics. 2021 Mar 4;11(10):4616-4636.
- Environ Sci Technol. 2023 Aug 9.
- Sens Actuators B Chem. 2020, 129190.

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REFERENCES

[1]. Davis S, et al. The NMDA receptor antagonist D-2-amino-5-phosphonopentanoate (D-AP5) impairs spatial learning and LTP in vivo at intracerebral concentrations comparable to those that block LTP in vitro. J Neurosci. 1992 Jan;12(1):21-34.

[2]. Morris RG, et al. N-methyl-d-aspartate receptors, learning and memory: chronic intraventricular infusion of the NMDA receptor antagonist d-AP5 interacts directly with the neural mechanisms of spatial learning. Eur J Neurosci. 2013 Mar;37(5):700-17.

[3]. R H Evans, et al. The effects of a series of omega-phosphonic alpha-carboxylic amino acids on electrically evoked and excitant amino acid-induced responses in isolated spinal cord preparations. Br J Pharmacol. 1982 Jan;75(1):65-75.

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