DL-AP5

Cat. No.: HY-100714

CAS No.: 76326-31-3

Molecular Formula: $C_5H_{12}NO_5P$

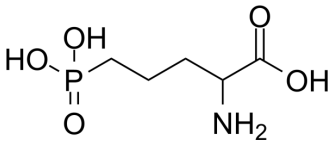
Molecular Weight: 197.13

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 : 33.33 mg/mL (169.08 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / 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: 50 mg/mL (253.64 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

DL-AP5 (2-APV) is a competitive NMDA (N-methyl-D-aspartate) receptor antagonist. DL-AP5 shows significantly antinociceptive activity. DL-AP5 specifically blocks on channels in the rabbit retina[1][2][3].

IC_{50} & Target

NMDA Receptor

In Vitro

DL-AP5 (100 μM) partially prevents glutamate-induced increase in Arc/Arg3.1 protein levels[5].
DL-AP5 decreases the NMDA-induced Arc/Arg3.1 upregulation[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

DL-AP5 (0-10 μg/rat, Intra-CA1) significantly decreases the effect of NMDA[3].
DL-AP5 (0-10 nmol, Intracerebroventricular injection) causes a dose-dependent increase in food consumption[4].
DL-AP5 (5 nmol, Intracerebroventricular injection) attenuates the decreased food consumption induced by the intracerebroventricular injection of ghrelin[4].

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

Animal Model:	Male Wistar rats (180-230 g)[3]
Dosage:	1, 3.2 and 10 μg/rat
Administration:	Injected into the intra-dorsal hippocampal (intra-CA1) immediately after shock administration, once
Result:	Significantly decreased the effect of NMDA (10^{-2} μg/rat, intra-CA1) with significant interaction.

Animal Model:	Broilers cockerels (3-h fooddeprived (FD3), n=8 for each group)[4]
Dosage:	0, 2.5, 5, and 10 nmol; in a volume of 10 μL
Administration:	Intracerebroventricular injection
Result:	Caused a dose-dependent increase in food consumption which was significant for 5 and 10 nmol doses.

Animal Model:	Broilers cockerels (3-h fooddeprived (FD3), n=8 for each group)[4]
Dosage:	5 nmol
Administration:	Intracerebroventricular injection, followed by ghrelin (0.6 nmol)
Result:	Attenuated the decreased food consumption induced by the intracerebroventricular injection of ghrelin.

REFERENCES

[1]. Murray CW, et al. Neurokinin and NMDA antagonists (but not a kainic acid antagonist) are antinociceptive in the mouse formalin model. Pain. 1991;44(2):179-185.

[2]. Massey SC, et al. N-methyl-D-aspartate receptors of ganglion cells in rabbit retina. J Neurophysiol. 1990;63(1):16-30.

[3]. Jafari-Sabet M. NMDA receptor blockers prevents the facilitatory effects of post-training intra-dorsal hippocampal NMDA and physostigmine on memory retention of passive avoidance learning in rats. Behav Brain Res. 2006 Apr 25;169(1):120-7.

[4]. Taati M, et al. The effects of DL-AP5 and glutamate on ghrelin-induced feeding behavior in 3-h food-deprived broiler cockerels. J Physiol Biochem. 2011 Jun;67(2):217-23.

[5]. Chen T, et al. Glutamate-induced rapid induction of Arc/Arg3.1 requires NMDA receptor-mediated phosphorylation of ERK and CREB. Neurosci Lett. 2017 Nov 20;661:23-28.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA