DL-AP5 sodium

Cat. No.: HY-100714C

CAS No.: 1303993-72-7

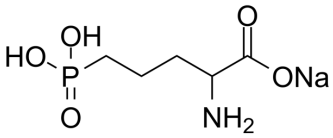
Molecular Formula: $C_5H_{11}NNaO_5P$

Molecular Weight: 219.11

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	DL-AP5 (2-APV) sodium is a competitive NMDA (N-methyl-D-aspartate) receptor antagonist. DL-AP5 sodium shows significantly antinociceptive activity. DL-AP5 sodium 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.

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