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DATASHEET

D-AP5

Product overview

 Name
 D-AP5

 Cat No
 HB0225

Alternative names APV, 2-APV, D-APV, dAPV, dAP5

Biological actionAntagonistMode of actionAntagonistPurity>99%

Customer commentsI made the discovery that the NMDA receptor is the trigger for the induction of LTP using D-AP5 synthesized by Jeff Watkins, the discoverer of the NMDA receptor... I now obtain my D-AP5 from

Hello Bio. I love their products and ethos and that is why I accepted a position on their Scientific

Advisory Board.

Professor Graham Collingridge, winner of The Brain Prize, 2016

My lab used D-AP5 from Hello Bio and were very happy with it. It behaved exactly as expected! Professor Kei Cho, Chair of Neuroscience, University of Bristol, UK (Hello Bio Scientific Advisory Board Member)

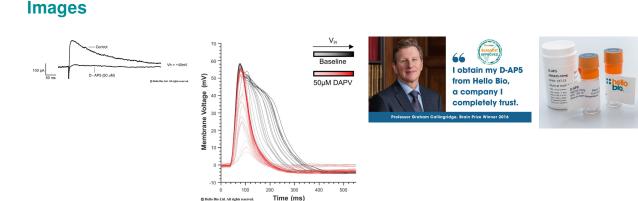
My lab is very satisfied with your D-AP5 quality and price. Verified customer, European Brain Research Institute (EBRI)

I used to buy D-AP5 from another company, but Hello Bio is far more cost-effective and works great in our experiments. Verified customer, University of South Carolina

The D-AP5 works as expected, great price. Verified customer, UCSF

Selective, competitive NMDA receptor antagonist. Inhibits NMDAR-synaptic plasticity.

Description



Biological Data

Biological description

Widely used, selective and competitive NMDA receptor antagonist which binds at the glutamate site. It is the more active form of DL-AP5.

D-AP5 blocks induction of LTP (long term potentiation) in a reversible manner and is frequently used to inhibit NMDAR-mediated synaptic plasticity. Also impairs spatial learning.

Application notes

#Figure 1: D-AP5 inhibition of evoked NMDAR mediated EPSCs in mouse cortical neuron

D-AP5 is commonly used to inhibit NMDA mediated synaptic plasticity. It is often used at concentrations of 50 μ M. D-AP5 from Hello Bio completely abolishes evoked NMDAR mediated currents at 50 μ M and reduces NMDA currents at lower concentrations of 1 and 10 μ M (see Fig 1 above).

#Protocol 1: Evoked NMDA receptor currents

- Whole cell voltage clamp recordings were obtained from layer V neurons of the mouse prelimbic cortex brain slice.
- NMDA currents were evoked via a stimulating electrode placed in layers II/III and evoked by a single square (150 μs) pulse every 10 sec at a stimulus intensity that gave a reliable NMDA current
- Neurons were held a +40 mV to relieve NMDA currents from their voltage-dependent Mg²⁺ block.
- NMDA currents were continually stimulated and recorded in response to continual bath applications of D-AP5 until NMDA currents were completely abolished.
- All NMDAR recordings were made in the presence of GABA_A-R and AMPAR antagonists.

#Figure 2: D-AP5 inhibition of NMDAR mediated dendritic plateau potentials in rat CA1 pyramidal neurones.

D-AP5 is commonly used to inhibit NMDA mediated synaptic events such as dendritic plateau potentials. Figure 2 shows that D-AP5 from Hello Bio completely abolishes plateau potential formation at $50\mu M$ (see Fig 2 above).

#Protocol 2: Inhibition of NMDAR mediated dendritic plateau potentials in rat CA1 Pyramidal neurones

- Pyramidal neurones from adult Wistar rats were patched in CA1 using a KMeSO4 internal solution with addition of 1mM QX-314 (HB1030) to prevent action potentials.
- Cells were first held in V_{clamp} at -70mV for 10 minutes to wash out LTP before being transferred to I_{clamp} (again at -70mV) where they were stimulated sequentially every 15 seconds in the Schaffer collateral pathway.
- Stimulation consisted of one single stimulation followed 400ms later by 5 stimulations at 100Hz.
- Experiments took place in the presence of the GABAB antagonist GCP55845 (1 μ M, HB0960) and 50 μ M PTX.
- Stimulation intensity was initially adjusted to evoke responses of approximately 1mV before stimulation was successively increased until robust plateau potentials were observed in all pathways.
- Stimulation was then turned off and 50µM DAPV was washed on to the slice for 10 minutes before another stimulation response was conducted in the same cell.
- \bullet Throughout the experiment input current was adjusted to maintain the cell at -70mV \pm 0.5mV.

Solubility & Handling

Solubility overview Storage instructions Storage of solutions Soluble in water (100mM) Room temperature

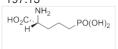
Shipping Conditions Important Prepare and use solutions on the same day if possible. Store solutions at -20 °C for up to one month if storage is required. Equilibrate to RT and ensure the solution is precipitate free before use.

Stable for ambient temperature shipping. Follow storage instructions on receipt.

This product is for RESEARCH USE ONLY and is not intended for therapeutic or diagnostic use. Not for human or veterinary use

Chemical Data

Chemical name Molecular Weight Chemical structure D-(-)-2-Amino-5-phosphonopentanoic acid



Molecular Formula $C_5H_{12}NO_5P$ CAS Number79055-68-8PubChem identifier135342

SMILES N[C@H](CCCP(=O)(O)O)C(=O)O

Source Synthetic

InChi InChi=1S/C5H12NO5P/c6-4(5(7)8)2-1-3-12(9,10)11/h4H,1-3,6H2,(H,7,8)(H2,9,10,11)/t4-/m1/s1

InChiKey VOROEQBFPPIACJ-SCSAIBSYSA-N

MDL number MFCD00078839
Appearance White solid

References

NMDA receptors, learning and memory: chronic intraventricular infusion of the NMDA receptor antagonist d-AP5 interacts directly with the neural mechanisms of spatial learning.

Morris RG *et al* (2013) Eur J Neurosci 37(5) **PubMedID**23311352

Actions of D and L forms of 2-amino-5-phosphonovalerate and 2-amino-4-phosphonobutyrate in the cat spinal cord.

Davies J *et al* (1982) Brain Res 235(2) **PubMedID**6145492

Effects of pre or posttraining dorsal hippocampus D-AP5 injection on fear conditioning to tone, background, and foreground context

Schenberg EE *et al* (2008) Hippocampus 18(11) **PubMedID** 18727044

Age-dependent hippocampal network dysfunction in a mouse model of alpha-synucleinopathy

Tweedy et al (2018) Thessis University of Newcastle