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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 action	Antagonist
Mode of action	Antagonist
Purity	>99%
Customer comments	I 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
Description	The D-AP5 works as expected, great price. Verified customer, UCSF Selective, competitive NMDA receptor antagonist. Inhibits NMDAR-synaptic plasticity.

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### **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. **#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  $\mu$ M. D-AP5 from Hello Bio completely abolishes evoked NMDAR mediated currents at 50  $\mu$ M and reduces NMDA currents at lower concentrations of 1 and 10  $\mu$ 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 voltagedependent Mg<sup>2+</sup> 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<sub>A</sub>-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<sub>clamp</sub> at -70mV for 10 minutes to wash out LTP before being transferred to I<sub>clamp</sub> (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 GABA<sub>B</sub> antagonist GCP55845 (1 $\mu$ M, HB0960) and 50 $\mu$ 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.
- Throughout the experiment input current was adjusted to maintain the cell at -70mV  $\pm$  0.5mV.

### Solubility & Handling

 Storage instructions
 Room temperature

 Solubility overview
 Soluble in water (100mM)

 Important
 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	197.13
Chemical structure	HO <sub>2</sub> C·····PO(OH) <sub>2</sub>
Molecular Formula	C <sub>5</sub> H <sub>12</sub> NO <sub>5</sub> P
CAS Number	79055-68-8
PubChem identifier	135342
SMILES	N[C@H](CCCP(=O)(O)O)C(=O)O
Source	Synthetic
InChi	InChl=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**

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

Davies J <i>et al</i> (1982)	Brain Res 235(2)
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 Schenberg EE et al (2008) Hippocampus 18(11)

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

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