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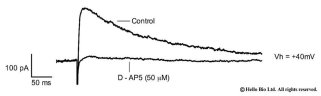
DATASHEET

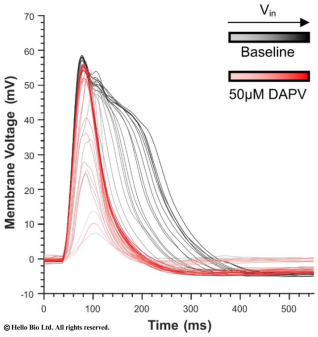
D-AP5

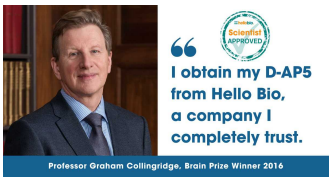
Product overview

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** *The D-AP5 works as expected, great price.* **Verified customer, UCSF**
Description	Selective, competitive NMDA receptor antagonist. Inhibits NMDAR-synaptic plasticity.

Images







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.
Application notes	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 µM. D-AP5 from Hello Bio completely abolishes evoked NMDAR mediated currents at 50 µM and reduces NMDA currents at lower concentrations of 1 and 10 µM (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 Mg^{2+} 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 50µM (see Fig 2 above).

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

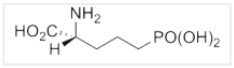
- Pyramidal neurones from adult Wistar rats were patched in CA1 using a KMeSO4 internal solution with addition of 1mM QX-314 (HB1030) to prevent action potentials.
- Cells were first held in V_{clamp} at -70mV for 10 minutes to wash out LTP before being transferred to I_{clamp} (again at -70mV) where 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 $GABA_B$ antagonist GCP55845 (1µM, HB0960) and 50µM 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 ± 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	D-(-)-2-Amino-5-phosphonopentanoic acid

Molecular Weight	197.13
Chemical structure	
Molecular Formula	$C_5H_{12}NO_5P$
CAS Number	79055-68-8
PubChem identifier	135342
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