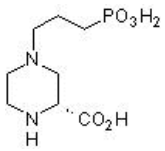
abcam

Product datasheet

(R)-CPP, NMDA antagonist ab120159

21 References 2 图像

概述

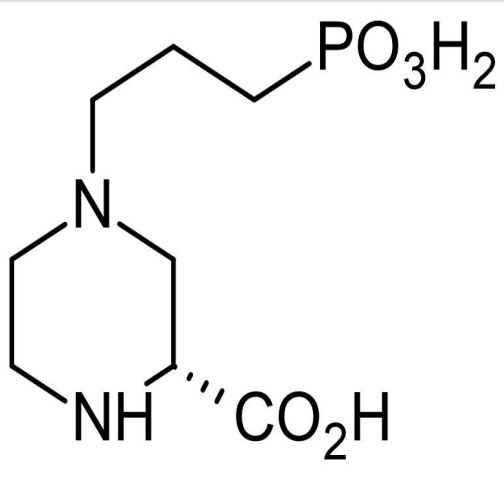
产品名称	(R)-CPP，NMDA拮抗剂
描述	Potent NMDA拮抗剂
生物学描述	Highly potent, competitive NMDA antagonist; more active enantiomer of (*RS*)-CPP (**ab120160**). (K_i values are 0.04 (NR1/NR2A), 0.3 (NR1/NR2B), 0.6 (NR1/NR2C) and 2.0 µM (NR1/NR2D)). Also available in simple stock solutions (**ab144495**) - add 1 ml of water to get an exact, ready-to-use concentration.
CAS编号	126453-07-4
化学结构	

性能

化学名称	(*R*)-3-(2-Carboxypiperazin-4-yl)propyl-1-phosphonic acid
分子量	252.20
分子式	$C_8H_{17}N_2O_5P$
PubChem识别号	6603754
存放说明	Store at Room Temperature. Store under desiccating conditions. The product can be stored for up to 12 months.
溶解度概述	Soluble in water to 100 mM
处理	Wherever possible, you should prepare and use solutions on the same day. However, if you need to make up stock solutions in advance, we recommend that you store the solution as aliquots in tightly sealed vials at -20°C. Generally, these will be useable for up to one month. Before use, and prior to opening the vial we recommend that you allow your product to equilibrate to room temperature for at least 1 hour. Need more advice on solubility, usage and handling? Please visit our **frequently asked questions (FAQ) page** for more details.
SMILES	O=C(O)[C@H]1CN(CCCP(=O)(O)O)CCN1

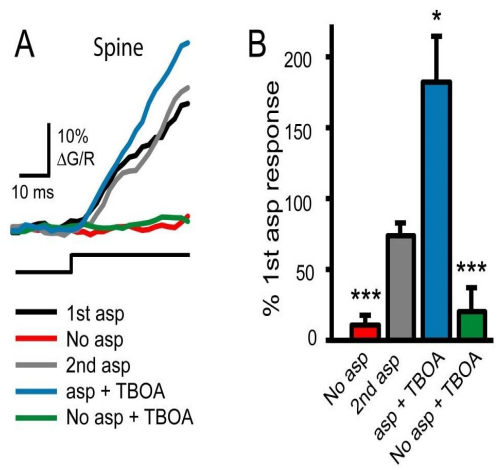
来源 Synthetic

图片



Chemical Structure - (R)-CPP, NMDA antagonist (ab120159)

2D chemical structure image of ab120159, (R)-CPP, NMDA antagonist



Functional Studies - (R)-CPP, NMDA antagonist (ab120159)

Transporter blockade does not reveal an ambient glutamate concentration gradient between extracellular compartments.

A. Average Ca^{2+} increase in a spine during a 40 ms voltage step, with iontophoresis of L-aspartate (black), without iontophoresis (red), a second L-aspartate application (gray), L-aspartate in the presence of 100 µM TBOA (blue), and TBOA alone (green).

B. Comparison of spine Ca^{2+} transients in each condition, normalized to the first response to L-aspartate iontophoresis (n=5). Error bars indicate SEM. Significance determined by Friedman ANOVA with Conover posthoc test: *p<0.05; **p<0.01; ***p<0.001.

If the extrasynaptic glutamate concentration is higher than that in the cleft because transporters prevent diffusion of glutamate into the synapse, blocking transporters should result in a large Ca^{2+} increase in the spine as extrasynaptic glutamate rushes into the cleft and activates synaptic NMDARs. Spines exhibited a Ca^{2+} increase during a 40 ms depolarization with iontophoresis of the glutamate transporter substrate and NMDAR agonist, L-aspartate (A; black and gray traces), confirming the presence of NMDARs. However, TBOA (100 µM) did not increase the Ca^{2+} transient in the same spines during the 40 ms depolarization when compared to the control voltage step without L-aspartate iontophoresis (See image compare green and red traces; 20.6±13.62%; p>0.5; n=5;). TBOA was effective in blocking transporters, however, as the NMDAR-mediated Ca^{2+} signal evoked by iontophoresis of L-

aspartate was increased in the presence of TBOA (See image). This result indicates that glutamate transporters do not normally generate a concentration gradient of ambient glutamate between extrasynaptic and synaptic extracellular compartments.

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