### abcam

### Product datasheet

### (R)-CPP, NMDA antagonist ab120159

**21 References** 画像数 2

#### 製品の概要

製品名 (R)-CPP, NMDA antagonist

製品の詳細 Potent NMDA antagonist

生理活性の詳細 Highly potent, competitive NMDA antagonist; more active enantiomer of (RS)-CPP (ab120160).

(K<sub>i</sub> 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 番号** 126453-07-4

**構造式** PO<sub>3</sub>H<sub>2</sub>

N CO<sub>2</sub>H

#### 製品の特性

体系名 (R)-3-(2-Carboxypiperazin-4-yl)propyl-1-phosphonic acid

分子量 252.20

**分子式** C<sub>8</sub>H<sub>17</sub>N<sub>2</sub>O<sub>5</sub>P

**PubChem 登録番号** 6603754

保存方法 Store at Room Temperature. Store under desiccating conditions. The product can be stored for

up to 12 months.

溶解性 Soluble in water to 100 mM

使用に関する注意 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.

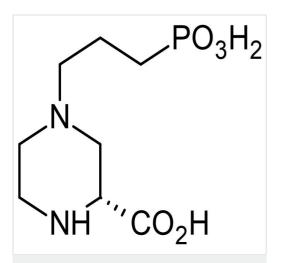
Need more advice on solubility, usage and handling? Please visit our frequently asked

questions (FAQ) page for more details.

SMILES 線形表記 O=C(O)[C@H]1CN(CCCP(=O)(O)O)CCN1

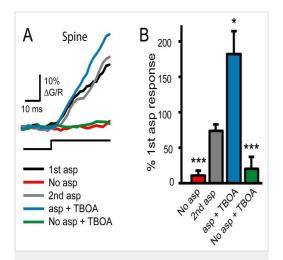
#### 画像

(ab120159)



Chemical Structure - (R)-CPP, NMDA antagonist

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



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

Herman et al PLoS One. 2011;6(11):e26501. doi: 10.1371/journal.pone.0026501. Epub 2011 Nov 1. Fig 3. Reproduced under the Creative Commons license http://creativecommons.org/licenses/by/4.0/

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

A. Average Ca<sup>2+</sup> 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<sup>2+</sup> 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  $\mu$ M) 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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