E4CPG

Cat. No.: HY-100372

CAS No.: 170846-89-6

Molecular Formula: $C_{11}H_{13}NO_4$

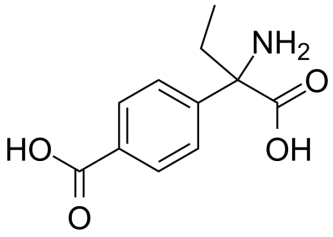
Molecular Weight: 223.23

Target: mGluR

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (44.80 mM; ultrasonic and adjust pH to 12 with NaOH)
Ethanol : < 1 mg/mL (insoluble)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.4797 mL	22.3984 mL	44.7968 mL
5 mM	0.8959 mL	4.4797 mL	8.9594 mL
10 mM	0.4480 mL	2.2398 mL	4.4797 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

E4CPG ((RS)-ECPG) is a Group I/Group II metabotropic glutamate receptor (mGluR) antagonist. E4CPG can inhibit the paired-pulse ratio of monosynaptic inhibitory postsynaptic currents (IPSC) potentiation[1][2].

IC_{50} & Target

mGluR

In Vitro

E4CPG acts at rat cortical mGluR with the K_B value of 0.367 mM[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

E4CPG (3-30 nmol/site (i.t.), 1-10 μmol/paw (i.pl.), and 1-10 nmol/site (i.c.v.)) significantly inhibits the nociception induced by the Glutamate-injection (i.pl.; 30 μmol/paw), and the maximal inhibition values for the antinociceptive action of E4CPG in Glutamate-induced nociception are 48% (i.pl.), 49% (i.t.) and 40% (i.c.v.) [4].

E4CPG (35 nM/3.5 μL, i.c.v.) completely blocks long-term depression (LTD) induced by the group I mGluR agonist Dihydroxyphenylglycine (DHPG, 100 nM/5 μL, i.c.v.) in male Sprague-Dawley rats[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Swiss mice (25-35 g)[4]
Dosage:	3-30 nmol/site (i.t.), 1-10 μmol/paw (i.pl.) and 1-10 nmol/ site (i.c.v.)
Administration:	Single injection
Result:	The maximal inhibition values for the antinociceptive action of E4CPG in glutamate-induced nociception were 48% (i.pl.), 49% (i.t.) and 40% (i.c.v.).

Animal Model:	Male Sprague-Dawley rats[5]
Dosage:	35 nM/3.5 μL
Administration:	Single injection, i.c.v.
Result:	Completely blocked LTD induced by the group I mGluR agonist dihydroxyphenylglycine (DHPG).

REFERENCES

[1]. N Sekiyama, et al. Structure-activity relationships of new agonists and antagonists of different metabotropic glutamate receptorsubtypes. Br J Pharmacol, 1996 Apr, 117(7):1493-503.

[2]. J S Bedingfield, et al. Structure-activity relationships for a series of phenylglycine derivatives acting at metabotropic glutamate receptors (mGluRs). Br J Pharmacol. 1995 Dec;116(8):3323-9.

[3]. Christian Patenaude, et al. GABAB receptor- and metabotropic glutamate receptor-dependent cooperative long-term potentiation of rat hippocampal GABAA synaptic transmission. J Physiol. 2003 Nov 15;553(Pt 1):155-67.

[4]. Alessandra Beirith, et al. Mechanisms underlying the nociception and paw oedema caused by injection of glutamate into the mouse paw. Brain Res. 2002 Jan 11;924(2):219-28.

[5]. Jing Han, et al. Acute cannabinoids impair working memory through astroglial CB1 receptor modulation of hippocampal LTD. Cell. 2012 Mar 2;148(5):1039-50.

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