Omberacetam

Cat. No.: HY-17456

CAS No.: 157115-85-0

Molecular Formula: $C_{17}H_{22}N_2O_4$

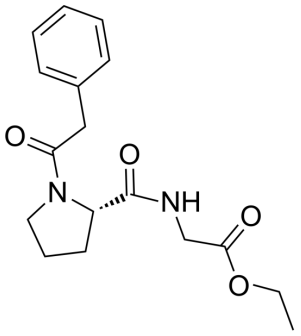
Molecular Weight: 318.37

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; 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 : ≥ 100 mg/mL (314.10 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1410 mL	15.7050 mL	31.4100 mL
5 mM	0.6282 mL	3.1410 mL	6.2820 mL
10 mM	0.3141 mL	1.5705 mL	3.1410 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.85 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.85 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.85 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Omberacetam (GVS-111) is a medication promoted and prescribed in Russia and neighbouring countries as a nootropic.

In Vitro

Nooglutil exhibits pharmacologically significant competition with a selective agonist of AMPA receptors ([G-3H]Ro 48-8587) for the receptor binding sites (with IC50 = 6.4 +/- 0.2 microM), while the competition of noopept for these receptor binding sites was lower by an order of magnitude (IC50 = 80 +/- 5.6 microM) [1]. GVS-111 significantly increased neuronal survival after H(2)O(2)-treatment displaying a dose-dependent neuroprotective activity from 10 nM to 100 microM, and an IC(50) value of 1.21+/-0.07 microM. GVS-111 inhibited the accumulation of intracellular free radicals and lipid peroxidation damage

	in neurons treated with H(2)O(2) or FeSO(4), suggesting an antioxidant mechanism of action [2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	N-Phenylacetyl-L-prolylglycine ethyl ester (GVS-111) administered intravenously at a dose of 0.5 mg/kg/day, for the first time 1 h after ischaemic lesion and then for 9 post-operative days, with the last administration 15 min before testing, attenuated the deficit [3]. GVS-111 itself was not found in rat brain 1 h after 5 mg/kg i.p. administration up to limit of detection (LOD) under high performance liquid chromatography (HPLC) conditions [4]. The most pronounced antiinflammatory effect of dipeptide was observed on the model of adjuvant arthritis in rats, where the drug administered over 25 days in a daily dose of 0.5 mg/kg (i.m.) or 5 mg/kg (p.o.) significantly reduced the chronic immune inflammation (on the 12th day, by 94.0 and 74.1%, respectively) [5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Firstova Iulu, et al. Studying specific effects of nootropic drugs on glutamate receptors in the rat brain. Eksp Klin Farmakol. 2011;74(1):6-10.

[2]. Pelsman A, et al. GVS-111 prevents oxidative damage and apoptosis in normal and Down's syndrome human cortical neurons. Int J Dev Neurosci. 2003 May;21(3):117-24.

[3]. Ostrovskaya RU, et al. Memory restoring and neuroprotective effects of the proline-containing dipeptide, GVS-111, in a photochemical stroke model. Behav Pharmacol. 1999 Sep;10(5):549-53.

[4]. Gudasheva TA, et al. The major metabolite of dipeptide piracetam analogue GVS-111 in rat brain and its similarity to endogenous neuropeptide cyclo-L-prolylglycine. Eur J Drug Metab Pharmacokinet. 1997 Jul-Sep;22(3):245-52.

[5]. Kovalenko LP, et al. Anti-inflammatory properties of noopept (dipeptide nootropic agent GVS-111). Eksp Klin Farmakol. 2002 Mar-Apr;65(2):53-5.

[6]. Kovalenko LP, et al. Preclinical study of noopept toxicity. Eksp Klin Farmakol. 2002 Jan-Feb;65(1):62-4.

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