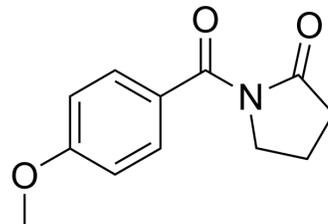


Aniracetam

Cat. No.:	HY-10932		
CAS No.:	72432-10-1		
Molecular Formula:	C ₁₂ H ₁₃ NO ₃		
Molecular Weight:	219.24		
Target:	nAChR; 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 (456.12 mM)
 H₂O : 0.33 mg/mL (1.51 mM; Need ultrasonic)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	4.5612 mL	22.8061 mL	45.6121 mL
	5 mM	0.9122 mL	4.5612 mL	9.1224 mL
	10 mM	0.4561 mL	2.2806 mL	4.5612 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (11.40 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (11.40 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (11.40 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Aniracetam (Ro 13-5057) is an orally active neuroprotective agent, possessing nootropics effects. Aniracetam potentiates the ionotropic quisqualate (iQA) responses in the CA1 region of rat hippocampal slices. Aniracetam also potentiates the excitatory post synaptic potentials (EPSPs) in Schaffer collateral-commissural synapses. Aniracetam can prevents the CO₂-induced impairment of acquisition in hypercapnia model rats. Aniracetam can be used to research cerebral dysfunctional disorders^{[1][2][3][4]}.

In Vitro	<p>Aniracetam concentration-dependently counteracts glutamate-, kainate-, or α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid-induced cell death and greatly facilitated neuroprotective response achieved by different concentrations of both quisqualate and trans-1-aminocyclopentane-1, 3-dicarboxylate in primary cultures of cerebellar granule cells^[4].</p> <p>Aniracetam potentiates the mGluR-coupled stimulation of phospholipase C in primary cultures of cerebellar granule cells^[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>																								
In Vivo	<p>Aniracetam (1 mM; 30-75 min) potentiates the iQA receptors and produces remarkable facilitation of the native synaptic transmission in rats^[1].</p> <p>Aniracetam (10-100 mg/kg; p.o.; single dosage) prevents the CO₂-induced impairment of acquisition in rats^[2].</p> <p>Aniracetam (30-300 mg/kg; p.o.; single dosage) increases the percentage of rats showing passive avoidance^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" data-bbox="347 554 1515 825"> <tbody> <tr> <td>Animal Model:</td> <td>Pyramidal neurons from male Wistar rats^[1]</td> </tr> <tr> <td>Dosage:</td> <td>1 mM</td> </tr> <tr> <td>Administration:</td> <td>30-75 min</td> </tr> <tr> <td>Result:</td> <td>Potentiated the iQA receptors existing in the brain and produced remarkable facilitation of the native synaptic transmission.</td> </tr> </tbody> </table> <table border="1" data-bbox="347 863 1515 1098"> <tbody> <tr> <td>Animal Model:</td> <td>Male rats (100-120 g; hypercapnia induced by pure CO₂)^[2]</td> </tr> <tr> <td>Dosage:</td> <td>10, 30, 50 and 100 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>p.o.; single dosage (60 min before hypercapnia)</td> </tr> <tr> <td>Result:</td> <td>Significantly prevented the CO₂-induced impairment of acquisition at 30 and 50 mg/kg.</td> </tr> </tbody> </table> <table border="1" data-bbox="347 1136 1515 1444"> <tbody> <tr> <td>Animal Model:</td> <td>Male rats and male mice (100-120 g and 21-25 g; 0.5 mg/kg Scopolamine-induced transient disruption of the memory of a passive avoidance procedure)^[2]</td> </tr> <tr> <td>Dosage:</td> <td>30, 50, 100 and 300 mg/kg,</td> </tr> <tr> <td>Administration:</td> <td>p.o.; single dosage</td> </tr> <tr> <td>Result:</td> <td>Significantly increased the percentage of rats showing passive avoidance 2 h after Scopolamine (HY-N0296) at 50 and 100 mg/kg.</td> </tr> </tbody> </table>	Animal Model:	Pyramidal neurons from male Wistar rats ^[1]	Dosage:	1 mM	Administration:	30-75 min	Result:	Potentiated the iQA receptors existing in the brain and produced remarkable facilitation of the native synaptic transmission.	Animal Model:	Male rats (100-120 g; hypercapnia induced by pure CO ₂) ^[2]	Dosage:	10, 30, 50 and 100 mg/kg	Administration:	p.o.; single dosage (60 min before hypercapnia)	Result:	Significantly prevented the CO ₂ -induced impairment of acquisition at 30 and 50 mg/kg.	Animal Model:	Male rats and male mice (100-120 g and 21-25 g; 0.5 mg/kg Scopolamine -induced transient disruption of the memory of a passive avoidance procedure) ^[2]	Dosage:	30, 50, 100 and 300 mg/kg,	Administration:	p.o.; single dosage	Result:	Significantly increased the percentage of rats showing passive avoidance 2 h after Scopolamine (HY-N0296) at 50 and 100 mg/kg.
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REFERENCES

- [1]. Ito I, et al. Allosteric potentiation of quisqualate receptors by a nootropic drug aniracetam. *J Physiol.* 1990 May;424:533-43.
- [2]. Cumin R, et al. Effects of the novel compound aniracetam (Ro 13-5057) upon impaired learning and memory in rodents. *Psychopharmacology (Berl).* 1982;78(2):104-11.
- [3]. Nakamura K. Aniracetam: its novel therapeutic potential in cerebral dysfunctional disorders based on recent pharmacological discoveries. *CNS Drug Rev.* 2002 Spring;8(1):70-89.
- [4]. Pizzi M, et al. Attenuation of excitatory amino acid toxicity by metabotropic glutamate receptor agonists and aniracetam in primary cultures of cerebellar granule cells. *J Neurochem.* 1993 Aug;61(2):683-9.
- [5]. Nakamura K, et al. Anxiolytic effects of aniracetam in three different mouse models of anxiety and the underlying mechanism. *Eur J Pharmacol.* 2001 May 18;420(1):33-43.

[6]. Vaglenova J, et al. Aniracetam reversed learning and memory deficits following prenatal ethanol exposure by modulating functions of synaptic AMPA receptors. *Neuropsychopharmacology*. 2008 Apr;33(5):1071-83. Epub 2007 Jul 4.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA