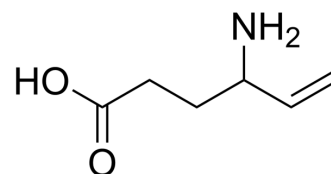


Vigabatrin

Cat. No.:	HY-15399		
CAS No.:	68506-86-5		
Molecular Formula:	C ₆ H ₁₁ NO ₂		
Molecular Weight:	129.16		
Target:	GABA Receptor		
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	H ₂ O : 50 mg/mL (387.12 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	7.7423 mL	38.7117 mL	77.4233 mL
		5 mM	1.5485 mL	7.7423 mL	15.4847 mL
10 mM		0.7742 mL	3.8712 mL	7.7423 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (774.23 mM); Clear solution; Need ultrasonic				

BIOLOGICAL ACTIVITY

Description	Vigabatrin (γ-Vinyl-GABA), an inhibitory neurotransmitter GABA vinyl-derivative, is an orally active and irreversible GABA transaminase inhibitor. Vigabatrin is an antiepileptic agent, which acts by increasing GABA levels in the brain by inhibiting the catabolism of GABA by GABA transaminase ^{[1][2][3]} .
In Vitro	Vigabatrin inhibits the uptake of taurine in Caco-2 and MDCK cells to 34% and 53%, respectively, at a concentration of 30 mM. In Caco-2 cells the uptake of Vigabatrin under neutral pH conditions is concentration-dependent and saturable with a K _m -value of 27 mM. Vigabatrin is able to inhibit the uptake of taurine in intestinal and renal cell culture models ^[2] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	A significant increase in seizure threshold is observed following systemic (i.p.) administration of high (600 or 1200 mg/kg) doses of Vigabatrin. Bilateral microinjection of Vigabatrin (10 μg) into either the anterior or posterior substantia nigra pars reticulata (SNr) also increased seizure threshold, but less markedly than systemic treatment. Focal delivery into the

subthalamic nucleus (STN) increased seizure threshold more markedly than either intranigral or systemic administration of Vigabatrin^[1].

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

CUSTOMER VALIDATION

- Redox Biol. 2021 Jul 26;46:102082.
- Cell Rep. 2022 Dec 6;41(10):111770.
- J Exp Bot. 2020 Feb 19;71(4):1459-1474.
- Plants. 2020 Apr 3;9(4):449.
- Animals. 2020 Mar 27;10(4):567.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Broeer, et al. Vigabatrin for focal drug delivery in epilepsy: Bilateral microinfusion into the subthalamic nucleus is more effective than intranigral or systemic administration in a rat seizure model. *Neurobiology of Disease* (2012), 46(2), 362-376.

[2]. Gaily, Eija Vigabatrin monotherapy for infantile spasms. *Expert Review of Neurotherapeutics* (2012), 12(3), 275-286.

[3]. Jakob Plum, et al. The anti-epileptic drug substance vigabatrin inhibits taurine transport in intestinal and renal cell culture models. *Int J Pharm.* 2014 Oct 1;473(1-2):395-7.

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