Vigabatrin hydrochloride

Cat. No.: HY-B0033

CAS No.: 1391054-02-6

Molecular Formula: $C_6H_{12}ClNO_2$

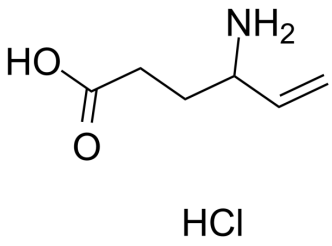
Molecular Weight: 165.62

Target: GABA Receptor

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 33.33 mg/mL (201.24 mM; Need ultrasonic)

DMSO : 27.5 mg/mL (166.04 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.0379 mL	30.1896 mL	60.3792 mL
5 mM	1.2076 mL	6.0379 mL	12.0758 mL
10 mM	0.6038 mL	3.0190 mL	6.0379 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 10 mg/mL (60.38 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (15.09 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.09 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.09 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vigabatrin hydrochloride (γ-Vinyl-GABA hydrochloride), a inhibitory neurotransmitter GABA vinyl-derivative, is an orally active and irreversible GABA transaminase inhibitor. Vigabatrin hydrochloride 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

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.
In Vivo	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 Km-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.

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.
- Horticulturae. 2023, 9(2), 268.

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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.

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