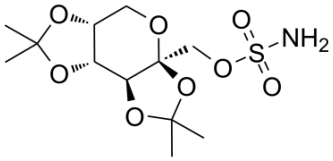
Topiramate

Cat. No.: HY-B0122

CAS No.: 97240-79-4

Molecular Formula: $C_{12}H_{21}NO_8S$

Molecular Weight: 339.36



Target: iGluR; GABA Receptor; Sodium Channel; Calcium Channel; Potassium Channel; Carbonic Anhydrase

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (294.67 mM)

H_2O : 4 mg/mL (11.79 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9467 mL	14.7336 mL	29.4672 mL
5 mM	0.5893 mL	2.9467 mL	5.8934 mL
10 mM	0.2947 mL	1.4734 mL	2.9467 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 (7.37 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Topiramate (McN 4853) is a broad-spectrum antiepileptic agent. Topiramate is a GluR5 receptor antagonist. Topiramate produces its antiepileptic effects through enhancement of GABAergic activity, inhibition of kainate/AMPA receptors, inhibition of voltage-sensitive sodium and calcium channels, increases in potassium conductance, and inhibition of carbonic anhydrase[1][2][3].

IC_{50} & Target	GluR5 receptor[1]; GABAergic[2]; Kainate/AMPA[2]; Sodium channel[2]; Calcium channel[2]; Potassium channel[2]; Carbonic anhydrase[2]
In Vitro	Topiramate has been believed to be a type of antiepileptic drug that blocks spread of seizures. Thus far, the mechanisms of its actions have been proven to include use-dependent inhibition of voltage-dependent Na+ channels in neurons, potentiation of GABA (γ-amino-butyric acid)-induced Cl- influx, and inhibitory effects on inward currents by antagonizing kainate/alpha-amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) receptors[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Anal Chem. 2020 Nov 21.
- ETH Zurich. 2020 Dec.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Lyseng-Williamson KA, et al. Topiramate: a review of its use in the treatment of epilepsy. Drugs. 2007;67(15):2231-56.

[2]. Nakamura J, et al. Target pharmacology of topiramate, a new antiepileptic drug. Nihon Yakurigaku Zasshi. 2000 Jan;115(1):53-7.

[3]. Kaminski RM, et al. Topiramate selectively protects against seizures induced by ATPA, a GluR5 kainate receptor agonist. Neuropharmacology. 2004 Jun;46(8):1097-104.

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