Topiramate

®

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Cat. No.:	HY-B0122					
CAS No.:	97240-79-4					
Molecular Formula:	C ₁₂ H ₂₁ NO ₈	5				
Molecular Weight:	339.36					
Target:	iGluR; GABA Receptor; Sodium Channel; Calcium Channel; Potassium Channel; O					
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Metabolic Enzyme/Protease					
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 (294.67 mM) H ₂ O : 4 mg/mL (11.79 mM; Need ultrasonic) * "≥" means soluble, but saturation unknown.							
		Solvent Mass Concentration	1 mg	5 mg	10 mg			
	Preparing Stock Solutions	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	 Add each solvent one by one: PBS Solubility: 16.67 mg/mL (49.12 mM); Clear solution; Need ultrasonic 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 3. 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							
	 4. 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,

Product Data Sheet

	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

- Acta Pharmacol Sin. 2022 Jul 27.
- Anal Chem. 2020 Dec 15;92(24):15745-15756.
- ETH Zurich. 2020 Dec.
- Personalized Medicine Universe. 2019 May.

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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.

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

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