Fosphenytoin disodium

Cat. No.: HY-B1657A

CAS No.: 92134-98-0

Molecular Formula: $C_{16}H_{13}N_2Na_2O_6P$

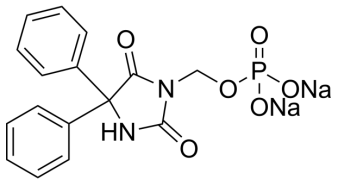
Molecular Weight: 406.24

Target: Sodium Channel

Pathway: Membrane Transporter/Ion Channel

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (246.16 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4616 mL	12.3080 mL	24.6160 mL
5 mM	0.4923 mL	2.4616 mL	4.9232 mL
10 mM	0.2462 mL	1.2308 mL	2.4616 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 25 mg/mL (61.54 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Fosphenytoin sodium is a phenytoin proagent with similar anticonvulsant properties. Its main mechanism is to block frequency-dependent, use-dependent and voltage-dependent neuronal sodium channels, and therefore limit repetitive firing of action potentials.

In Vivo

Fosphenytoin is an effective neuroprotectant against ischemia-induced damage. In fosphenytoin (30 mg/kg, i.m.)-treated rat 5 min after ischemia episode, hippocampal CA1 pyramidal neurons remain at near control level (13.90 +/- 0.92), however, GFAP staining iss not significantly changed[1]. In fosphenytoin (84 mg/kg)-treated rat, the relative bioavailability of fosphenytoin is 83%. In fully kindled female Wistar rats, fosphenytoin dose-dependently increases the focal seizure (afterdischarge) threshold. Seizure severity and duration at threshold are reduced only after the highest does of fosphenytoin tested (84 mg/kg)[2].

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

PROTOCOL

Animal Administration [1]

A total of four groups of rats, including normal (n=2), sham operated (n=2), ischemia with saline-treated (n=2), and ischemia with fosphenytoin-treated (n=2), are studied. Postischemic rats in saline-treated and fosphenytoin-treated groups receive a single i.m. injection of saline or fosphenytoin (30 mg/kg), respectively, in the right hind limb 5 minutes after resuscitation. Sham-operated animals are treated similarly except for chest compression. All rats are killed on the 7th postischemic day by decapitation. Brains are immediately removed, bisected longitudinally, and immersed in 4% buffered neutral formaldehyde containing 0.25% glutaraldehyde for a minimum of 2 days at 4°C. Portions of the brain containing the dorsal hippocampus are coronally sectioned with a vibratome at 40 μm. With the aid of a dissecting microscope, rectangular blocks of about 1 mm^2 in size encompassing the mid-CA1 region from sections that approximate Bregma -3.6 are dissected, postfixed in 2% osmium tetroxide, and dehydrated in ascending concentrations of ethanol before being embedded in Araldite 502. Sections of polymerized blocks 1 μm thick are cut and toluidine blue stained for light microscopic examination.

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

REFERENCES

[1]. Chan SA, et al. Fosphenytoin reduces hippocampal neuronal damage in rat following transient global ischemia. Acta Neurochir (Wien). 1998;140(2):175-80.

[2]. Loscher W, et al. Anticonvulsant effect of fosphenytoin in amygdala-kindled rats: comparison with phenytoin. Epilepsy Res. 1998 Mar;30(1):69-76.

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