Phenytoin

Cat. No.: HY-B0448

CAS No.: 57-41-0

Molecular Formula: $C_{15}H_{12}N_2O_2$

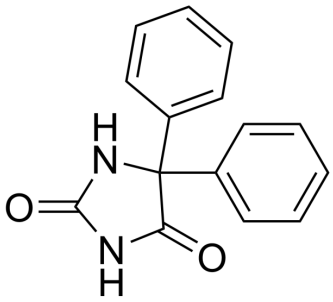
Molecular Weight: 252.27

Target: Sodium Channel; Virus Protease

Pathway: Membrane Transporter/Ion Channel; Anti-infection

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 : 50 mg/mL (198.20 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.9640 mL	19.8200 mL	39.6401 mL
	5 mM	0.7928 mL	3.9640 mL	7.9280 mL
	10 mM	0.3964 mL	1.9820 mL	3.9640 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 (9.91 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.91 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.91 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Phenytoin (5,5-Diphenylhydantoin) is a potent Voltage-gated Na^+ channels (VGSCs) blocker. Phenytoin has antiepileptic activity and reduces breast tumour growth and metastasis in mice[1][2].

In Vitro

Phenytoin is an antiepileptic drug. It is useful to partial seizures and generalized tonic-clonic seizures but not primary generalized seizures such as absence seizures or myoclonic seizures. Phenytoin is believed to protect against seizures by causing voltage-dependent block of voltage-gated sodium channels[2].
Phenytoin has low affinity for resting sodium channels at hyperpolarized membrane potentials[3].

	When neurons are depolarized and the channels transition into the open and inactivated states, greater binding and block occur. The inhibitory potency is strongly use dependent, so that block accumulates with prolonged or repetitive activation, such as occurs during a seizure discharge. The blocking of sodium channels by phenytoin is of slow onset. The time course of fast sodium currents is therefore not altered in the presence of the drug and action potentials evoked by synaptic depolarizations of ordinary duration are not blocked. Thus phenytoin is able to selectively inhibit pathological hyperexcitability in epilepsy without unduly impairing ongoing activity. Phenytoin also blocks persistent sodium current and this may be of particular importance in seizure control. Phenytoin is a class 1b antiarrhythmic[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Phenytoin (5,5-Diphenylhydantoin; 60 mg/kg; daily; 28 days) reduces tumour growth in six week-old female $Rag2^{-/-}$ $Il2rg^{-/-}$ mice with MDA-MB-231 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Int Immunopharmacol. 2023 Jan 11;115:109706.
- Regen Ther. 2023 Dec, 24, 201-210.
- University of Glasgow. School of Cancer Sciences.
- Oxid Med Cell Longev. 2022 Mar 28;2022:8538296.

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REFERENCES

[1]. Rogawski, M.A. and W. Loscher, The neurobiology of antiepileptic drugs. Nat Rev Neurosci, 2004. 5(7): p. 553-64.

[2]. Porter, R.J., et al., Mechanisms of action of antiseizure drugs. Handb Clin Neurol, 2012. 108: p. 663-81.

[3]. Balaji, S., Medical therapy for sudden death. Pediatr Clin North Am, 2004. 51(5): p. 1379-87.

[4]. Michaela Nelson, et al. The sodium channel-blocking antiepileptic drug phenytoin inhibits breast tumour growth and metastasis. Mol Cancer. 2015 Jan 27;14(1):13.

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

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