Quinidine (15% dihydroquinidine)

Cat. No.: HY-B1751

CAS No.: 56-54-2

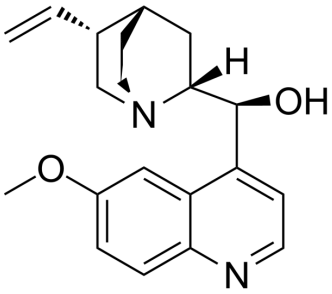
Molecular Formula: $C_{20}H_{24}N_2O_2$

Molecular Weight: 324.42

Target: Potassium Channel; Cytochrome P450; Apoptosis; Parasite

Pathway: Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease; Apoptosis; Anti-infection

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 1 year; -20°C, 6 months (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (154.12 mM)
Ethanol : 14.29 mg/mL (44.05 mM; ultrasonic and warming and heat to 60°C)
H_2O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.0824 mL	15.4121 mL	30.8242 mL
	5 mM	0.6165 mL	3.0824 mL	6.1648 mL
	10 mM	0.3082 mL	1.5412 mL	3.0824 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 (7.71 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Quinidine (15% dihydroquinidine) is an antiarrhythmic agent. Quinidine is a potent, orally active, selective cytochrome P450db inhibitor. Quinidine is also a K^+ channel blocker with an IC_{50} of 19.9 μM, and can induce apoptosis. Quinidine can be used for malaria research[1][2][3][4].

IC_{50} & Target	Plasmodium

In Vitro

Quinidine shows cytotoxicity against MES-SA cells, and induces apoptosis[4].

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

Cell Cytotoxicity Assay[4]

Cell Line:	MES-SA and MESSA/DX5 cells
Concentration:	10 μM
Incubation Time:	24 hours
Result:	Showed cytotoxicity against MES-SA cells in a concentration-dependent manner.

Apoptosis Analysis[4]

Cell Line:	MES-SA and MESSA/DX5 cells
Concentration:	10 μM
Incubation Time:	24 hours
Result:	Increased the apoptotic portion sub-G1 DNA contents induced by paclitaxel, while paclitaxel had no effect on sub-G1 DNA contents undergoing apoptosis.

In Vivo

Quinidine shows effects on the PTZ-induced seizure threshold[5].

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

Animal Model:	Male mice of the NMRI strain (age 5-6 weeks and weight 25-30 g)[5
Dosage:	10, 20, and 30 mg/kg
Administration:	Intraperitoneal injection; 10, 20, and 30 mg/kg; once
Result:	Increased the threshold dose for the onset to tonic hind limb extension at a dose of 30 mg/kg, compared to the saline-treated control group ($p<0.05$).

CUSTOMER VALIDATION

- J Hazard Mater. 2021 Aug 15;416:125764.
- Environ Int. 2019 Jun;127:694-703.
- Chemosphere. 2021, 131347.
- J Med Chem. 2021 Mar 11;64(5):2725-2738.
- J Med Chem. 2020 Oct 8;63(19):11085-11099.

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REFERENCES

[1]. Sang-Yun Lee, et al. Hydrocinchonine, cinchonine, and quinidine potentiate paclitaxel-induced cytotoxicity and apoptosis via multidrug resistance reversal in MES-SA/DX5 uterine sarcoma cells. Environ Toxicol. 2011 Aug;26(4):424-31.

[2]. Hassan Jamali, et al. Effect of dextromethorphan/quinidine on pentylenetetrazole- induced clonic and tonic seizure thresholds in mice. Neurosci Lett. 2020 Jun 11;729:134988.

[3]. Moody DE, et al. Quinidine inhibits in vivo metabolism of amphetamine in rats: impact upon correlation between GC/MS and immunoassay findings in rat urine. J Anal Toxicol. 1990 Sep-Oct;14(5):311-7.

[4]. Kehl SJ, et al. Quinidine-induced inhibition of the fast transient outward K+ current in rat melanotrophs. Br J Pharmacol. 1991 Jul;103(3):1807-13.

[5]. Roden DM, et al. Class I antiarrhythmic agents: quinidine, procainamide and N-acetylprocainamide, disopyramide.

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