Quinidine hydrochloride monohydrate

Cat. No.: HY-B1302

CAS No.: 6151-40-2

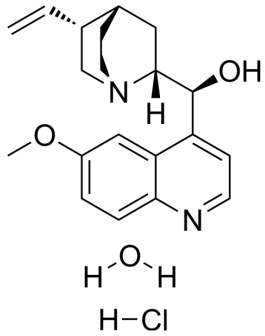
Molecular Formula: $C_{20}H_{27}ClN_2O_3$

Molecular Weight: 378.89

Target: Potassium Channel; Parasite

Pathway: Membrane Transporter/Ion Channel; Anti-infection

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

DMSO : 100 mg/mL (263.93 mM; Need ultrasonic)
H_2O : 2.5 mg/mL (6.60 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.6393 mL	13.1964 mL	26.3929 mL
5 mM	0.5279 mL	2.6393 mL	5.2786 mL
10 mM	0.2639 mL	1.3196 mL	2.6393 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 (6.60 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.60 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.60 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Quinidine hydrochloride monohydrate is an anti-arrythmic agent which is also a potent blocker of K^+ channel with an IC_{50} of 19.9 μM.

IC_{50} & Target

Plasmodium

In Vitro

Quinidine hydrochloride monohydrate blocks WT mSlo3 ($K_{Ca}5.1$) channels with an IC_{50} of 19.9±1.41 μM and Hill slope of 1.15±0.15 (n=7). Again, the potency of inhibition by Quinidine hydrochloride monohydrate is higher for F304Y mSlo3 (IC_{50} of 2.42±0.60 μM, n=9, P<0.005; Hill slope of 0.98±0.12), but lower with R196Q mSlo3 (IC_{50} of 38.4±6.77 μM, n=5, P<0.001; Hill

	slope of 1.05±0.16). The inhibition of F304Y mSlo3 by Quinidine hydrochloride monohydrate is observed to have some time dependence[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Direct application of Quinidine hydrochloride monohydrate on the sciatic nerve produces a dose-related decrease in amplitude at ascending somato-sensory evoked potential (SSEP) and descending compound muscle action potentials (CMAP) when comparing baseline with other time points, or when comparing the experimental left limb to the right contra-lateral glucose-treated limb. The latencies of SSEPs and CMAP potentials after Quinidine hydrochloride monohydrate applications are increased compare to baseline and the contralateral side[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Mouse (m) Slo3 (K_{Ca}5.1) channels or mutant forms are expressed in Xenopus oocytes and currents recorded with 2-electrode voltage-clamp. Gain-of-function mSlo3 mutations are used to explore the state-dependence of the inhibition. The interaction between Quinidine hydrochloride monohydrate and mSlo3 channels is modelled by in silico docking[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	24 rats are randomly divided into three groups with eight rats in each group. Groups Q_1, Q_3, and Q_5 receive Quinidine hydrochloride monohydrate 1, 3, and 5 μmol, respectively, in 5 % glucose 0.1 mL. The sciatic nerve is exposed by making an incision from the left sciatic notch to the distal thigh. The subcutaneous tissue is bluntly dissected to expose the biceps femoris. The sciatic nerve is freed from its investing fascia. The procedure is then repeated on the right side. The somato-sensory evoked potential (SSEP) and compound muscle action potentials (CMAP) are recorded at baseline, immediately after Quinidine hydrochloride monohydrate treatment, then every 15 min thereafter for 1 h, then every 30 min thereafter for 3 h. The animals are allowed to recover and then kept separately for 2 weeks. After performing behavioral examinations, electrophysiological examinations are performed with the animals under intra-peritoneal anesthesia[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

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

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REFERENCES

[1]. Wrighton DC, et al. Mechanism of inhibition of mouse Slo3 (KCa 5.1) potassium channels by quinine, quinidine and barium. Br J Pharmacol. 2015 Sep;172(17):4355-63.

[2]. Cheng KI, et al. Application of quinidine on rat sciatic nerve decreases the amplitude and increases the latency of evoked responses. J Anesth. 2014 Aug;28(4):559-68.

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

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