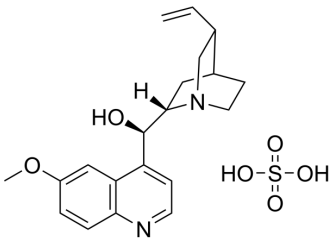
Quinine sulfate

Cat. No.:	HY-B1751B
CAS No.:	549-56-4
Molecular Formula:	$C_{20}H_{26}N_2O_6S$
Molecular Weight:	422.5
Target:	Potassium Channel; Parasite; Cytochrome P450
Pathway:	Membrane Transporter/Ion Channel; Anti-infection; Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Quinidine sulfate is an antiarrhythmic agent. Quinidine sulfate is a potent, orally active, selective cytochrome P450db inhibitor. Quinidine sulfate is also a K^+ channel blocker with an IC_{50} of 19.9 μM. Quinidine sulfate can be used for malaria research[1][2][3].
IC_{50} & Target	Plasmodium
In Vitro	Quinidine sulfate is an anti-arrythmic drug which affects ionic currents in heart muscle and which has also been shown to be a potent blocker of several classes of K^+ channel in a variety of cell types[1]. Bath application of quinidine sulfate causes a dose-dependent reduction of the peak amplitude of I_k. The K_d for blockade of I_k at 0 mV is estimated to be 41 μM[1]. Quinidine sulfate elicits a dose-dependent increase of the rate of the decay of I_k and this effect is enhanced by membrane depolarization. Quinidine sulfate also causes a 5 mV hyperpolarizing shift of the steady-state inactivation curve and increases the half-time for recovery from inactivation. Quinidine sulfate does not affect the onset of inactivation measured at -30 mV[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Quinidine sulfate is rapidly absorbed, with peak plasma concentrations 60-90 min after an oral dose. Other salts (gluconate, polygalacturonate) are more slowly absorbed, with lower peak concentrations[2]. Quinidine sulfate is approximately 70-90 % bound to plasma proteins. It undergoes hepatic oxidative metabolism to form an N-oxide, a 3-hydroxy form, an O-demethyl form and 2'-quinidinone[2]. Quinidine sulfate inhibits metabolism of amphetamine in rats. Quinidine sulfate pretreatment results in a significant decrease in the excretion of p-hydroxyamphetamine at 24 and 48 h to 7.2 and 24.1% of the vehicle-control levels, respectively, accompanied by a significant increase in amphetamine excretion between 24 and 48 h to 542% of the control[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Mol Med Rep. 2021 Mar 2.
- Norwegian University of Science and Technology, Faculty of Medicine and Health sciences. 2019 Sep.

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REFERENCES

[1]. 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.

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

[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.

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