Quinacrine dihydrochloride

Cat. No.: HY-13735A

CAS No.: 69-05-6

Molecular Formula: $C_{23}H_{32}Cl_3N_3O$

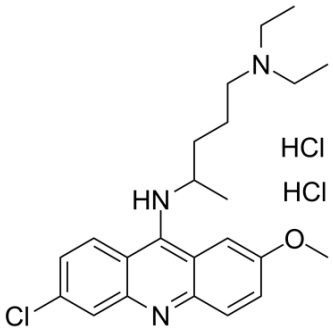
Molecular Weight: 472.88

Target: Parasite; Apoptosis; Autophagy; Mitophagy

Pathway: Anti-infection; Apoptosis; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 44 mg/mL (93.05 mM)

H_2O : 13.89 mg/mL (29.37 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	2.1147 mL	10.5735 mL	21.1470 mL
5 mM	0.4229 mL	2.1147 mL	4.2294 mL
10 mM	0.2115 mL	1.0574 mL	2.1147 mL

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

BIOLOGICAL ACTIVITY

Description

Quinacrine (Mepacrine) dihydrochloride is an orally bioavailable antimalarial agent, which possess anticancer effect both in vitro and vivo. Quinacrine dihydrochloride suppresses NF-κB and activate p53 signaling, which results in the induction of the apoptosis[1].

In Vitro

Quinacrine (5-20 μM; 24 hours) inhibits the growth of SGC-7901 cells[1].

Quinacrine (7.5 and 15 μM; 24 hours) induces apoptosis in SGC-7901 cells, which is associated with mitochondria-dependent signal pathway and involves p53 upregulation and caspase-3 activation pathway[1].

Quinacrine (15 μM; 24 hours) treatment significantly increased the levels of proapoptotic proteins, including cytochrome c, Bax, and p53, and decreased the levels of antiapoptotic protein Bcl-2, thus shifting the ratio of Bax/Bcl-2 in favor of apoptosis [1].

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

Cell Viability Assay[1]

Cell Line:	SGC-7901 cells
Concentration:	0, 5, 10, 15, and 20 μM
Incubation Time:	24 hours
Result:	Cell viability was inhibited in a dose-dependent manner, and the mean IC_{50} value is 16.18 μM.

Apoptosis Analysis[1]

Cell Line:	SGC-7901 cells
Concentration:	7.5 and 15 μM
Incubation Time:	24 hours
Result:	The percentage of apoptotic cells, including the early phase and late phase apoptosis, increased to 26.30%, compared with control group of 3.37%.

Western Blot Analysis[1]

Cell Line:	SGC-7901 cells
Concentration:	15 μM
Incubation Time:	24 hours
Result:	The relative quantity of cytochrome c protein was upregulated, increased from 0.10 to 0.24. The relative quantity of p53 protein was dramatically increased, from 0.06 to 0.19. The Bax/Bcl-2 ratio was dramatically elevated from 1.21 to 2.59.

In Vivo

Quinacrine (100 mg/kg three times per week for two consecutive weeks) significantly suppresses circulating blast cells at days 30/31 and increases the median survival time (MST). Quinacrine does not decrease the body weight of treated animals at the tested dose[2].

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Animal Model:	Female SCID mice with acute myeloid leukemia (AML)-PS model[2]
Dosage:	100 mg/kg
Administration:	Administered by oral gavage (po); three times a week for two consecutive weeks
Result:	In the first AML mouse in vivo study, evaluation of circulating leukemic cells detected in blood samples (in percent of white blood cells (WBC)) at day 30/31 showed 72% human tumor cells in the control mice, whereas in mice treated with Quinacrine, this was only 2.2%. The MST of control mice was 34 days whereas it was 46 days in Quinacrine-treated mice.

CUSTOMER VALIDATION

- ACS Nano. 2020 Jun 23;14(6):7639-7650.

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REFERENCES

[1]. Xiaoyang Wu, et al. Quinacrine Inhibits Cell Growth and Induces Apoptosis in Human Gastric Cancer Cell Line SGC-7901. Curr Ther Res Clin Exp. 2012 Feb;73(1-2):52-64.

[2]. Anna Eriksson, et al. Towards repositioning of quinacrine for treatment of acute myeloid leukemia - Promising synergies and in vivo effects. Leuk Res. 2017 Dec;63:41-46.

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

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