Amodiaquine

Cat. No.: HY-B1322A

CAS No.: 86-42-0

Molecular Formula: $C_{20}H_{22}ClN_3O$

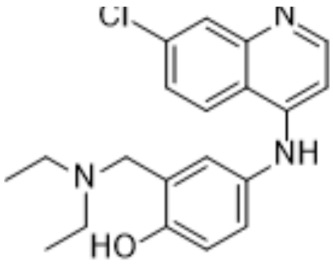
Molecular Weight: 355.86

Target: Histone Methyltransferase; Parasite; Nuclear Hormone Receptor 4A/NR4A

Pathway: Epigenetics; Anti-infection; Vitamin D Related/Nuclear Receptor

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 66.67 mg/mL (187.35 mM; ultrasonic and adjust pH to 3 with 1M HCl)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8101 mL	14.0505 mL	28.1009 mL
	5 mM	0.5620 mL	2.8101 mL	5.6202 mL
	10 mM	0.2810 mL	1.4050 mL	2.8101 mL

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

BIOLOGICAL ACTIVITY

Description

Amodiaquine (Amodiaquin), a 4-aminoquinoline class of antimalarial agent, is a potent and orally active histamine N-methyltransferase inhibitor. Amodiaquine is also a Nurr1 agonist and specifically binds to Nurr1-LBD (ligand binding domain) with an EC_{50} of ~20 μM. Anti-inflammatory effect[1][2][3][4].

IC_{50} & Target		
	Plasmodium	Nurr1/NR4A2

In Vitro

Amodiaquine (10-20 μM; 4 hours) treatment suppresses LPS-induced expression of proinflammatory cytokines (IL-1β, interleukin-6, TNF-α and iNOS) in a dose-dependent manner[1].

Amodiaquine (5 μM; 24 hours) significantly inhibits neurotoxin (6-OHDA-induced cell death in primary dopamine cells as examined by the number of TH^+ neurons and dopamine uptake. The neuroprotective effect of Amodiaquine is also observed in rat PC12 cells[1].

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

RT-PCR[1]

Cell Line:	Primary microglia

Concentration:	10 μM, 15 μM, 20 μM
Incubation Time:	4 hours
Result:	Suppressed LPS-induced expression of proinflammatory cytokines (IL-1β, interleukin-6, TNF-α and iNOS) in a dose-dependent manner.

In Vivo

Amodiaquine (40 mg/kg; intraperitoneal injection; daily; for 3 days; male ICR mice) treatment diminishes perihematomal activation of microglia/macrophages and astrocytes. Amodiaquine also suppresses ICH-induced mRNA expression of IL-1β, CCL2 and CXCL2, and ameliorated motor dysfunction of mice[2].

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

Animal Model:	Male ICR mice (8-10 weeks of age) induced ntracerebral hemorrhage (ICH)[2]
Dosage:	40 mg/kg
Administration:	Intraperitoneal injection; daily; for 3 days
Result:	Diminished perihematomal activation of microglia/macrophages and astrocytes.

CUSTOMER VALIDATION

- Pharmacol Res. 2023 Mar 20;106717.
- Cell Rep. 2021 Apr 6;35(1):108959.
- J Virol. 2024 Jan 18:e0121623.
- Metab Brain Dis. 2021 Jan 28.
- Biochem Biophys Res Commun. 2020 Feb 19;522(4):862-868.

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REFERENCES

[1]. Chun-Hyung Kim, et al. Nuclear receptor Nurr1 agonists enhance its dual functions and improve behavioral deficits in an animal model of Parkinson's disease. Proc Natl Acad Sci U S A. 2015 Jul 14;112(28):8756-61.

[2]. Keita Kinoshita, et al. A Nurr1 agonist amodiaquine attenuates inflammatory events and neurological deficits in a mouse model of intracerebral hemorrhage. J Neuroimmunol. 2019 May 15;330:48-54.

[3]. Akira Yokoyama, et al. Effect of amodiaquine, a histamine N-methyltransferase inhibitor, on, Propionibacterium acnes and lipopolysaccharide-induced hepatitis in mice. Eur J Pharmacol. 2007 Mar 8;558(1-3):179-84.

[4]. M T HOEKENGA. The treatment of acute malaria with single oral doses of amodiaquin, chloroquine, hydroxychloroquine and pyrimethamine. Am J Trop Med Hyg. 1954 Sep;3(5):833-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA