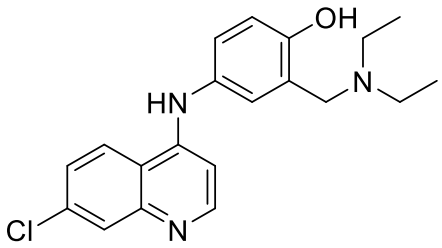
Product data sheet

MedKoo Cat#: 317185 Name: Amodiaquine free base CAS#: 86-42-0 (free base) Chemical Formula: $C_{20}H_{22}ClN_3O$ Exact Mass: 355.14514 Molecular Weight: 355.86	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Amodiaquine (trade names Camoquin, Flavoquine), a 4-aminoquinoline compound related to chloroquine, is used as an antimalarial and anti-inflammatory agent. Amodiaquine has been shown to be more effective than chloroquine in treating chloroquine-resistant Plasmodium falciparum malaria infections and may give more protection than chloroquine when used as weekly prophylaxis. Amodiaquine, like chloroquine, is generally well tolerated. Amodiaquine is a histamine N-methyltransferase inhibitor. It is on the World Health Organization's List of Essential Medicines, the most important medications needed in a basic health system.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	38.0	106.78
Ethanol	12.0	33.72

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.81 mL	14.05 mL	28.10 mL
5 mM	0.56 mL	2.81 mL	5.62 mL
10 mM	0.28 mL	1.41 mL	2.81 mL
50 mM	0.06 mL	0.28 mL	0.56 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Won HY, Shin JH, Oh S, Jeong H, Hwang ES. Enhanced CD25+Foxp3+ regulatory T cell development by amodiaquine through activation of nuclear receptor 4A. Sci Rep. 2017 Dec 5;7(1):16946. doi: 10.1038/s41598-017-17073-y. PMID: 29208963; PMCID: PMC5717225.
2. Espinoza JA, Zisi A, Kanellis DC, Carreras-Puigvert J, Henriksson M, Hühn D, Watanabe K, Helleday T, Lindström MS, Bartek J. The antimalarial drug amodiaquine stabilizes p53 through ribosome biogenesis stress, independently of its autophagy-inhibitory activity. Cell Death Differ. 2020 Feb;27(2):773-789. doi: 10.1038/s41418-019-0387-5. Epub 2019 Jul 8. PMID: 31285544; PMCID: PMC7205879.

In vivo study

1. Won HY, Shin JH, Oh S, Jeong H, Hwang ES. Enhanced CD25+Foxp3+ regulatory T cell development by amodiaquine through activation of nuclear receptor 4A. Sci Rep. 2017 Dec 5;7(1):16946. doi: 10.1038/s41598-017-17073-y. PMID: 29208963; PMCID: PMC5717225.

2. DeWald LE, Johnson JC, Gerhardt DM, Torzewski LM, Postnikova E, Honko AN, Janosko K, Huzella L, Dowling WE, Eakin AE, Osborn BL, Gahagen J, Tang L, Green CE, Mirsalis JC, Holbrook MR, Jahrling PB, Dyall J, Hensley LE. In Vivo Activity of Amodiaquine against Ebola Virus Infection. Sci Rep. 2019 Dec 27;9(1):20199. doi: 10.1038/s41598-019-56481-0. PMID: 31882748; PMCID: PMC6934550.

7. Bioactivity

Biological target:

Amodiaquine (Amodiaquin), a 4-aminoquinoline class of antimalarial agent, is a potent histamine N-methyltransferase inhibitor and Nurr1 agonist that specifically binds to Nurr1-LBD (ligand binding domain) with an EC50 of ~20 μM.

In vitro activity

Cells treated with 1–10 μM AQ (Amodiaquine) for 6 h accumulated lysosomes (LAMP1-positive puncta) (Fig. 3a, b) while concentrations between 10 and 20 μM further enhanced autophagosome accumulation (Fig. 3c, d; LC3B puncta). Notably, 10–20 μM AQ also induced generation of nucleolar caps (Fig. 3c), showing that both autophagy inhibition and nucleolar stress take place simultaneously. Strikingly, even though AQ and CQ showed similar levels of lipidated LC3 (LC3-II) (Fig. 2a) and accumulation of cytoplasmic vesicles (Fig. 3e), only AQ caused remarkable condensation of nucleolar chromatin (Fig. 3e). Among the 4-aminoquinoline family members, CQ and H-CQ inhibit autophagy with efficiency similar to AQ, but RPA194 degradation and p53 stabilization was only induced by AQ (Fig. 3f). Furthermore, only AQ induced a strong reduction of 47S rRNA synthesis among all autophagy inhibitors tested (Fig. 3g). Overall, these findings indicate that ribosome biogenesis stress is not a general consequence of autophagy inhibition and that AQ stands out among the 4-aminoquinoline family as a compound operating through two independent mechanisms: autophagy inhibition in the cytoplasm and ribosome biogenesis stress in the nucleolus. Taken together, the data reveals unsuspected activity of a drug approved and used in the clinics for over 30 years, and provide rationale for repurposing amodiaquine in cancer therapy.

Reference: Cell Death Differ. 2020 Feb; 27(2): 773–789. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7205879/

In vivo activity

To confirm the immune suppression by AQ in vivo, mice were fed DSS and intraperitoneally injected with either vehicle or AQ. Body weight loss and colon shortening were induced in the DSS-treated group, but these changes were attenuated in the AQ-treated group (Fig. 7A,B). Histological examination demonstrated that AQ reduced the immune cell infiltration and inhibited epithelial cell destruction with goblet cell depletion in the DSS-induced colitis model (Fig. 7C). Furthermore, T cell-induced colitis in recombinase-activating gene (RAG) knockout (KO) mice was diminished by administration with AQ, as demonstrated by the decreases in disease activity index and histopathology (Fig. 7D,E). Foxp3+ Treg cells were more frequently found in AQ-treated colon tissues (Fig. 7E). Consistently, AQ administration increased the expression of Foxp3, but decreased the expression of inflammatory markers RORγt and T-bet (Fig. 7F). Moreover, the inflammatory cytokines IL-17 and IFN-γ produced by inflammatory T cells were significantly diminished in the AQ-treated group (Fig. 7G). The ability of anti-malarial AQ to potentiate iTreg cell development makes it a promising drug for preventing and treating inflammatory and autoimmune diseases.

Reference: Sci Rep. 2017 Dec 5;7(1):16946. https://pubmed.ncbi.nlm.nih.gov/29208963/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.