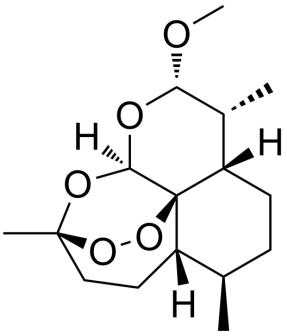
Artemether

Cat. No.:	HY-N0402
CAS No.:	71963-77-4
Molecular Formula:	$C_{16}H_{26}O_5$
Molecular Weight:	298.37
Target:	Parasite
Pathway:	Anti-infection
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (335.15 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3515 mL	16.7577 mL	33.5154 mL
5 mM	0.6703 mL	3.3515 mL	6.7031 mL
10 mM	0.3352 mL	1.6758 mL	3.3515 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 (8.38 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.38 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	Artemether is an anti-malarial compound that targets drug-resistant strains of falciparum malaria[1].
IC_{50} & Target	Plasmodium
In Vitro	Artemether (0-200 μg/mL, 24-72 h) inhibits rat C6 glioma cell growth in a dose- and time-dependent manner[2]. Artemether (0-10 μM, 72 h) inhibits RANKL-induced osteoclast (osteoclast precursor cells (BMMs)) formation and related

gene expression (TRAP, NFATc1, V-ATPase-d2, CTSK, DC-STAMP, MMP-9)[2].
Artemether (48 or 96 h) inhibits ConA- or alloantigen-induced BALB/c splenocyte proliferation (IC_{50}: 6.3 and 3.5 μM)[4].
Artemether (0-50 μM, 16-36 h) inhibits production of the IL-2 and IFN-γ in BALB/c splenocyte[4].
Artemether (0-50 μM, 72 h) inhibits ConA-induced splenocyte, CD4+T- and CD8+ T-cell divisions, and inhibits cell cycle progression through G1/S transition[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	RAW264.7 cells (treated with 100 ng/mL RANKL)
Concentration:	10 μM
Incubation Time:	6 h
Result:	Inhibited the activation of MAPK subfamilies, including ERK, JNK, and p38.

Cell Cycle Analysis[4]

Cell Line:	ConA-stimulated T lymphocytes
Concentration:	1, 10 and 50 μM
Incubation Time:	72 h
Result:	Arrested 47, 56 and 91% (at 1, 10 and 50 μM) of the cells at G0/G1 phases, respectively.

In Vivo

Artemether (0-66 mg/kg, p.o.) inhibits tumor growth and angiogenesis in SD rats bearing C6 glioma cells[2].
Artemether (10 mg/kg, i.p., 8 days) protects mice against LPS-induced osteolytic bone loss[3].
Artemether (50 and 100 mg/kg, p.o.) inhibits T-cell-mediated immune responses (ear swelling) in DNFB-induced DTH model in BALB/c mice[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	LPS (5 mg/kg) treated mice[3]
Dosage:	10 mg/kg
Administration:	i.p., 8 days
Result:	Prevented LPS induced osteolytic bone loss and the reduction in bone volume. Increased bone volume/total volume (BV/TV), decreased osteoclast surface/bone surface (Oc.S/BS) and number of TRAP-positive cells.

CUSTOMER VALIDATION

- J Nutr Biochem. 2024 Jun 10:109687.

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REFERENCES

[1]. Wu H, et al. Artemether attenuates LPS-induced inflammatory bone loss by inhibiting osteoclastogenesis and bone resorption via suppression of MAPK signaling pathway. Cell Death Dis. 2018 May 1;9(5):498.

[2]. Wang JX, et al. Investigation of the immunosuppressive activity of artemether on T-cell activation and proliferation. Br J Pharmacol. 2007 Mar;150(5):652-61.

[3]. Xiao, S., et al., Recent investigations of artemether, a novel agent for the prevention of schistosomiasis japonica, mansoni and haematobia. Acta Trop, 2002. 82(2): p. 175-81.

[4]. Wu, Z.P., et al., Inhibitive effect of artemether on tumor growth and angiogenesis in the rat C6 orthotopic brain gliomas model. Integr Cancer Ther, 2009. 8(1): p. 88-92.

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