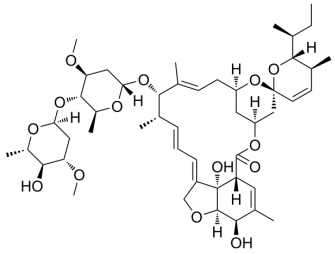
Avermectin B1a

Cat. No.:	HY-15308
CAS No.:	65195-55-3
Molecular Formula:	$C_{48}H_{72}O_{14}$
Molecular Weight:	873.08
Target:	Parasite; Antibiotic
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 : 25 mg/mL (28.63 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.1454 mL	5.7269 mL	11.4537 mL
	5 mM	0.2291 mL	1.1454 mL	2.2907 mL
	10 mM	0.1145 mL	0.5727 mL	1.1454 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 (2.86 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.86 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Avermectin B1a is an antiparasitic agent that paralyzes nematodes without causing hypercontraction or flaccid paralysis.

In Vitro

[^{3}H]AVM B1a preferentially binds to synaptic membranes from several regions of rat brain. [^{3}H]AVM B1a specific binding to intact monolayers of granule cells increases rapidly with time of incubation and reaches equilibrium after approximately 20 min at 24°C. Higher concentrations of [^{3}H]AVM B1a leads to markedly greater nonspecific binding, 60% at 25 nM. Various AVM analogs also produce concentration-dependent inhibition of [^{3}H]AVM B1a binding in intact cerebellar neurons. AVM B1a and moxidectin are similar in potency (IC_{50} values, 120 and 126 nM, respectively)[3]. AVMB1a-stimulated chloride efflux from mouse brain synaptic vesicles results from the activation of GABA-insensitive chloride channels and that this action is distinct from their previously documented effects on GABA-gated chloride channels in mouse brain preparations[4].

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

In Vivo	Bacteria are significantly inhibited when the AVM B1a concentration is higher than 83.3 mg/kg, while fungi are less impaired in soil. Soil respiration is also inhibited by high concentration AVM B1a, which differs with soil types. The half lethal dosage (LD_{50}) of AVM B1a to soil earthworm is estimated as 4.63 mg × cm^2 in filter paper contact test, and as 24.13 and 17.06 mg/kg, respectively after treated 7 and 14 days in artificial soil[1]. Iin artificial soil, the LC50 of AVM B1a on earthworms are 24.1 mg/kg and 17.1 mg/kg, respectively, for 7 and 14 days. About 80.0% and 94.8% of the accumulated AVM B1a are eliminated respectively in two groups within 1 day after they are exposed to AVM B1a-free soil, but a trace amount of AVM B1a is found for a relative long time in earthworms[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Sun Y, et al. [Effects of avermectin B1a on soil microorganism and earthworm (Eisenia fetida)]. Ying Yong Sheng Tai Xue Bao. 2005 Nov;16(11):2140-3.

[2]. Sun Y, et al. Bioaccumulation and elimination of avermectin B1a in the earthworms (Eisenia fetida). Chemosphere. 2005 Jul;60(5):699-704

[3]. Huang J, et al. Avermectin B1a binds to high- and low-affinity sites with dual effects on the gamma-aminobutyric acid-gated chloride channel of cultured cerebellar granule neurons. J Pharmacol Exp Ther. 1997 Apr;281(1):261-6.

[4]. Payne GT, et al. Activation of gamma-aminobutyric acid insensitive chloride channels in mouse brain synaptic vesicles by avermectin B1a. J Biochem Toxicol. 1991 Winter;6(4):283-92.

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