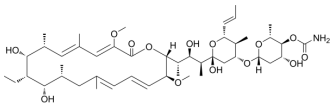
Concanamycin A

Cat. No.:	HY-N1724
CAS No.:	80890-47-7
Molecular Formula:	$C_{46}H_{75}NO_{14}$
Molecular Weight:	866.09
Target:	Proton Pump; Bacterial; Antibiotic
Pathway:	Membrane Transporter/Ion Channel; Anti-infection
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

Methanol : 10 mg/mL (11.55 mM; Need ultrasonic and warming)

DMSO : ≥ 10 mg/mL (11.55 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.1546 mL	5.7731 mL	11.5461 mL
5 mM	0.2309 mL	1.1546 mL	2.3092 mL
10 mM	0.1155 mL	0.5773 mL	1.1546 mL

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

BIOLOGICAL ACTIVITY

Description

Concanamycin A (Folimycin; Antibiotic X 4357B) is a macrolide antibiotic, a vacuolar type H^+-ATPase (V-ATPase) inhibitor. Concanamycin A is also an inhibitor of lysosomal acidification, can be used to T cell-mediated inflammation research[1]-[5].

IC_{50} & Target

Macrolide

In Vitro

Concanamycin A can come from S. diastatochromogenes, as a microbial metabolite with immunomodulatory activity[1].

?Concanamycin A (100 nM; 0-20 h) results DNA fragmentation in $CD4^+$ and selectively induces $CD8^+$ T cells rapid cell death between normal and the immunized mice source, while $CD8^+$ population in mice immunized is more sensitive[2].

?Concanamycin A (3-50 nM; 16 h) inhibits LPS-induced NO production in elicited peritoneal macrophages, but (25 nM; 7 h) doesn't inhibit LPS-induced TNF-α production[3].

?Concanamycin A (0.01 nM-1 nM) inhibits the acidification of rat liver lysosomes (IC_{50} =0.061 nM), and inhibits oleate incorporation into cholesteryl ester (IC_{50} =14 nM)[4].

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

Cell Viability Assay[2]

Cell Line:	$CD8^+$ cytotoxic T lymphocytes (CTLs)
Concentration:	100 nM
Incubation Time:	0, 4, 8, 12, 16, 20 hours
Result:	Induced rapid cell death instead of apoptosis at 20 h, without observed condensed nuclei.

In Vivo

Concanamycin A (15 mg/kg; i.v.; 0, 10 or 24 h prior to sacrifice) induces T cell-mediated hepatitis in mice[5].

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

Animal Model:	Wild type mice[5]
Dosage:	15 mg/kg
Administration:	Intravenous injection 0, 10 or 24 h prior to sacrifice
Result:	Resulted significant liver injury as demonstrated by serum transaminase levels, inflammatory cell infiltrate, hepatocyte apoptosis, and expression of several cytokines including interleukin 4 (IL4) and interferon gamma (IFNγ) at 10 h and 24 h following administration.

CUSTOMER VALIDATION

- Sci Adv. 2023 Oct 13;9(41):eadh1134.
- Autophagy. 2022 Nov 30.
- Cell Death Dis. 2023 Aug 29;14(8):571.
- Biomed Pharmacother. 2022 Jul 1;153:113328.
- Plant Physiol. 2023 Mar 22;kiad188.

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REFERENCES

[1]. Hines IN, et al. Impaired T cell-mediated hepatitis in peroxisome proliferator activated receptor alpha (PPARα)-deficient mice. Biol Res. 2018 Feb 15;51(1):5.

[2]. Woo JT, et al. Isolation, characterization and biological activities of concanamycins as inhibitors of lysosomal acidification. J Antibiot (Tokyo). 1992 Jul;45(7):1108-16.

[3]. Vaněk, Z., et al. Immunomodulators isolated from microorganisms. Folia Microbiol. 1991. 36:99–111.

[4]. Togashi K, et al. Concanamycin A, a vacuolar type H(+)-ATPase inhibitor, induces cell death in activated CD8(+) CTL. Cytotechnology. 1997 Nov;25(1-3):127-35.

[5]. Eswarappa SM, et al. Folimycin (concanamycin A) inhibits LPS-induced nitric oxide production and reduces surface localization of TLR4 in murine macrophages. Innate Immun. 2008 Feb;14(1):13-24.

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