

Product Data Sheet

Product Name: Valinomycin

Cat. No.: GC16948

Chemical Properties

Cas. No. 2001-95-8

Chemical Name	(3S,6R,9R,12S,15S,18S,21R,24R,27R,30R,33R,36S)-3,6,9,15,18,21,27,30,33-nonaisopropyl-12,24,36-trimethyl-1,7,13,19,25,31-hexaoxa-4,10,16,22,28,34-hexaazacyclohexatriacontane-2,5,8,11,14,17,20,23,26,29,32,35-dodecaone
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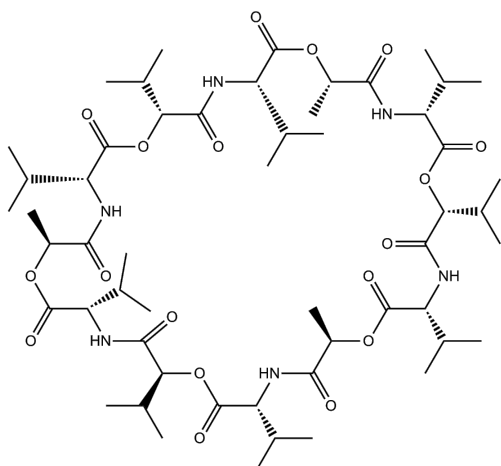
Formula	C ₅₄ H ₉₀ N ₆ O ₁₈	M.Wt	1111.32
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Solubility	DMF: 30 mg/ml,DMSO: 5 mg/ml,Ethanol: 10 mg/ml	Storage	Store at -20°C,protect from light
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General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.
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Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.
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Structure



Protocol

Caution: Product has not been fully validated for medical applications. For research use only.

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Cell experiment [1]:

Cell lines	CHO cells
Preparation method	Valinomycin is added into the culture medium.
Reaction Conditions	1-100μM;12-24 h
Applications	100 μM valinomycin caused 77% and 86% cell death at 12 h and 24 h, respectively.

References:

[1]. Abdalah R, Wei L, et al. Valinomycin-induced apoptosis in Chinese hamster ovary cells. *Neurosci Lett*. 2006 Sep 11;405(1-2):68-73. doi: 10.1016/j.neulet.2006.06.055. Epub 2006 Jul 20. PMID: 16857314.

Background

Valinomycin is a dodecadepsipeptide antibiotic, which is obtained from several *Streptomyces* strains. Valinomycin functions as a potassium-specific transporter and promotes the movement of K⁺ through lipid membranes. Excessive K⁺ efflux is an ionic mechanism underlying apoptosis. It can be used as uncoupling agent to induce depolarization[1-3].

Valinomycin(1-100μM;12-24 h) treated CHO cells underwent an apoptotic event due to potassium efflux[4]. Valinomycin (2 nM) induced energy-dependent mitochondrial swelling, cytochrome c release, cytosolic NADH/cytochrome c oxidation and apoptosis[5]. Neurite formation and membrane changes of mouse neuroblastoma cells induced by valinomycin(100 μM) [6].

Incorporation of valinomycin into multilamellar liposomes composed of dimyristoyl phosphatidyl choline:cholesterol:phosphatidyl serine (10:4:1 M ratio) results in a profound reduction in toxicity with

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maintenance of antitumor efficacy in mice, the LD50 for liposome incorporated valinomycin (MVL-VM) is in excess of 50 mg/kg[7].

References:

- [1]. Varma S, Sabo D, ,et.al. K⁺/Na⁺ selectivity in K channels and valinomycin: over-coordination versus cavity-size constraints. J Mol Biol. 2008 Feb 8;376(1):13-22. doi: 10.1016/j.jmb.2007.11.059. Epub 2007 Nov 28. PMID: 18155244; PMCID: PMC2390915.
- [2]. Daniele RP, Holian SK. A potassium ionophore (valinomycin) inhibits lymphocyte proliferation by its effects on the cell membrane. Proc Natl Acad Sci U S A. 1976 Oct;73(10):3599-602. doi: 10.1073/pnas.73.10.3599. PMID: 1068473; PMCID: PMC431165.
- [3]. Berezin SK. Valinomycin as a Classical Anionophore: Mechanism and Ion Selectivity. J Membr Biol. 2015 Aug;248(4):713-26. doi: 10.1007/s00232-015-9784-y. Epub 2015 Mar 4. PMID: 25736817.
- [4]. Abdalah R, Wei L, ,et.al. Valinomycin-induced apoptosis in Chinese hamster ovary cells. Neurosci Lett. 2006 Sep 11;405(1-2):68-73. doi: 10.1016/j.neulet.2006.06.055. Epub 2006 Jul 20. PMID: 16857314.
- [5]. Lofrumento DD, La Piana G, ,et.al. Valinomycin induced energy-dependent mitochondrial swelling, cytochrome c release, cytosolic NADH/cytochrome c oxidation and apoptosis. Apoptosis. 2011 Oct;16(10):1004-13. doi: 10.1007/s10495-011-0628-7. PMID: 21739274.
- [6]. Koike T. Neurite formation and membrane changes of mouse neuroblastoma cells induced by valinomycin. Biochim Biophys Acta. 1978 Jun 2;509(3):429-39. doi: 10.1016/0005-2736(78)90237-7. PMID: 656419.
- [7]. Daoud SS, Juliano RL. Reduced toxicity and enhanced antitumor effects in mice of the ionophoric drug valinomycin when incorporated in liposomes. Cancer Res. 1986 Nov;46(11):5518-23. PMID: 3756900.

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