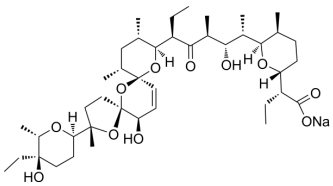
Salinomycin sodium salt

Cat. No.:	HY-17439
CAS No.:	55721-31-8
Molecular Formula:	$C_{42}H_{69}NaO_{11}$
Molecular Weight:	772.98
Target:	Bacterial; Wnt; β-catenin; Autophagy; Apoptosis; Antibiotic; Parasite
Pathway:	Anti-infection; Stem Cell/Wnt; Autophagy; Apoptosis
Storage:	4°C, stored under nitrogen * In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (129.37 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.2937 mL	6.4685 mL	12.9369 mL
	5 mM	0.2587 mL	1.2937 mL	2.5874 mL
	10 mM	0.1294 mL	0.6468 mL	1.2937 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 (3.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Salinomycin sodium salt (Salinomycin sodium), an antibiotic potassium ionophore, is a potent inhibitor of Wnt/β-catenin signaling. Salinomycin sodium salt (Salinomycin sodium) acts on the Wnt/Fzd/LRP complex, blocks Wnt-induced LRP6 phosphorylation, and causes degradation of the LRP6 protein. Salinomycin sodium salt (Salinomycin sodium) shows selective activity against human cancer stem cells[1][2][3].

IC_{50} & Target

Wnt/β-catenin[1]

In Vitro

Salinomycin (0.1-8 μM; 48 h) inhibits the growth of HUVECs in a dose-dependent manner, accounting for 32.1 and 59.2% inhibition at 4 and 8 μM, respectively. HUVECs exposed to 2, 4 and 8 μM of Salinomycin for 48 h show a dose-dependent reduction in cell number and a change in cell morphology. Salinomycin (4 μM) treatment effectively inhibits HUVEC

	migration and invasion, and significantly disrupt the capillary-like tube formation of HUVECs. Salinomycin significantly suppresses the expression levels of phosphorylated (p)-FAK in a time- and dose-dependent manner in HUVECs. Salinomycin inhibits HUVEC angiogenesis by disturbing the VEGF-VEGFR2-AKT signaling axis[1]. Combination of RSVL and Salinomycin synergistically inhibits the proliferation of TNBC (MDA-MB-231) cells. RSVL and Salinomycin effectively reduce wound healing, colony and tumorosphere forming capability in TNBC cells. Synergistic combination of RSVL and Salinomycin induces apoptosis in both culture conditions by significant upregulation of Bax with decreased Bcl-2 expression as comparison to untreated and alone drug treatments[2]. Salinomycin (0, 2, 4, 8 and 16 μM) significantly inhibits the proliferation of A2780 and SK-OV-3 cell lines in a dose- and time-dependent manner, ($IC_{50\ 24h}$: 13.8 μM, $IC_{50\ 48h}$: 6.888 μM and $IC_{50\ 72h}$: 4.382 μM for A2780 cell lines), ($IC_{50\ 24h}$: 12.7 μM, $IC_{50\ 48h}$: 9.869 μM and $IC_{50\ 72h}$: 5.022 μM for SK-OV-3 cell lines). Salinomycin blocks the Wnt/β-catenin pathway in EOC cells[3]. Salinomycin (2 μM) reduces cancer cell proliferation, inhibits STAT3 phosphorylation and P38 and β-catenin expressions, and suppresses epithelial-mesenchymal transition in colorectal cancer cells. Salinomycin (1-5 μM) inhibits cancer cell proliferation and STAT3 signaling in colorectal cancer cells. Furthermore, Salinomycin activates Akt (Ser 473) and down-regulates Hsp27 (Ser 82) phosphorylation in HT-29 and SW480. Salinomycin down-regulates hTERT and reduces telomerase activity when combined with telomerase inhibitor [4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Salinomycin (5 and 10 mg/kg) significantly supresses the average tumor volume and tumor weight. Salinomycin hinders the U251 human glioma cell growth in vivo via inhibition of angiogenesis with involvement of AKT and FAK dephosphorylation[1]. Salinomycin (0.5 mg/kg b.wt.) enhances the mean survival time of the tumor bearing Swiss albino mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	The effect of Salinomycin on HUVEC growth is determined by MTT assay. Briefly, HUVECs (6,000 cells/well) are seeded in 96-well culture plates for 24 h and incubated with different concentrations of Salinomycin. In the preliminary experiments, Salinomycin treatment for 12, 24, 48 and 72 h shows time-dependent effects on cell growth inhibition. However, treatment for 48 h is the optimal time and is selected for further mechanism evaluation. After Salinomycin treatment for 48 h, 20 μL/well of MTT solution (5 mg/mL) is added and incubated for 5 h. The medium is aspirated and replaced with 200 μL/well of DMSO to dissolve the formazan Salinomycint formed. The color intensity of the formazan solution is measured at 570 nm by a microplate spectrophotometer. The cell viability is expressed as % of the control (as 100%). MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Human glioma U251 cells (1×10^7) suspended in 100 μL PBS are injected into the right lower hind flank of each 6-week-old male nude mouse. The mice are then randomly assigned into three groups of 10 mice in each group. After one week, Salinomycin (5 and 10 mg/kg) is administered into the caudal vein every other day for 16 days. Control mice receive an equal volume of vehicle (Salinomycinine) only. Body weight and tumor volume are monitored every two days. At the end of the experiments, tumors are excised, photographed, and weighed. Tumors from each group are used for western blotting and immunohistochemical (IHC) assay. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- EMBO Mol Med. 2019 Oct;11(10):e9930.
- J Control Release. 2020 Oct 10;326:387-395.
- Acta Biomater. 2022 Aug 23;S1742-7061(22)00501-3.
- Anal Chem. 2022 Sep 19.
- Cell Death Dis. 2023 Mar 11;14(3):193.

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REFERENCES

[1]. Lu D, et al. Salinomycin inhibits Wnt signaling and selectively induces apoptosis in chronic lymphocytic leukemia cells. Proc Natl Acad Sci U S A. 2011 Aug 9;108(32):13253-7.

[2]. Zhou J, et al. Salinomycin induces apoptosis in cisplatin-resistant colorectal cancer cells by accumulation of reactiveoxygen species. Toxicol Lett. 2013 Oct 24;222(2):139-45.

[3]. Klose J, et al. Salinomycin: Anti-tumor activity in a pre-clinical colorectal cancer model. PLoS One. 2019 Feb 14;14(2):e0211916.

[4]. Wang F, et al. Salinomycin Inhibits Proliferation and Induces Apoptosis of Human Hepatocellular Carcinoma Cells In Vitro and In Vivo. PLoS One. 2012; 7(12): e50638.

[5]. Qu H, et al. Effect of salinomycin on metastasis and invasion of bladder cancer cell line T24. Asian Pac J Trop Med. 2015 Jul;8(7):578-82.

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