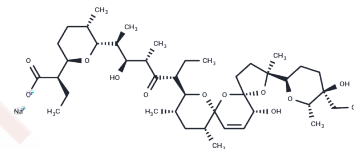


## Salinomycin sodium salt

## Chemical Properties

CAS No. :	55721-31-8
Formula:	C42H69NaO11
Molecular Weight:	772.98
Storage:	Store at low temperature Powder: -20°C for 3 years   In solvent: -80°C for 1 year <small>Actual storage temperature shall be subject to the COA.</small>



## Biological Description

Description	Salinomycin sodium salt (Sodium salinomycin), an antibiotic potassium ionophore, is an effective inhibitor of Wnt/ $\beta$ -catenin signaling.
Targets(IC50)	Apoptosis, Antibacterial, Antibiotic, Parasite, Autophagy, Wnt/beta-catenin
In vitro	Salinomycin (0.1-8 $\mu$ M) inhibits the growth of HUVECs in a dose-dependent manner, accounting for 32.1 and 59.2% inhibition at 4 and 8 $\mu$ M, respectively. HUVECs exposed to 2, 4 and 8 $\mu$ M of Salinomycin for 48 h show a dose-dependent reduction in cell number and a change in cell morphology. Salinomycin (4 $\mu$ M) 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]. A combination of RSVL and Salinomycin synergistically inhibits the proliferation of TNBC (MDA-MB-231) cells. RSVL and Salinomycin effectively reduce wound healing, colony and tumorsphere forming capability in TNBC cells [2]. Salinomycin (0, 2, 4, 8 and 16 $\mu$ M) significantly inhibits the proliferation of A2780 and SK-OV-3 cell lines in a dose- and time-dependent manner, (IC50 24h: 13.8 $\mu$ M, IC50 48h: 6.888 $\mu$ M and IC50 72h: 4.382 $\mu$ M for A2780 cell lines), (IC50 24h: 12.7 $\mu$ M, IC50 48h: 9.869 $\mu$ M and IC50 72h: 5.022 $\mu$ M for SK-OV-3 cell lines). Salinomycin blocks the Wnt/ $\beta$ -catenin pathway in EOC cells [3]. Salinomycin (2 $\mu$ M) reduces cancer cell proliferation, inhibits STAT3 phosphorylation and P38 and $\beta$ -catenin expressions, and suppresses epithelial-mesenchymal transition in colorectal cancer cells. Salinomycin (1-5 $\mu$ M) 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].
In vivo	Salinomycin (5 and 10 mg/kg) effectively reduces both the average tumor volume and weight, demonstrating its ability to inhibit U251 human glioma cell proliferation in vivo, chiefly through obstructing angiogenesis and the dephosphorylation of AKT and FAK [1]. Additionally, Salinomycin (0.5 mg/kg b.wt.) significantly prolongs the mean survival time of tumor-bearing Swiss albino mice [2].

Cell Research	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 $\mu$ L/well of MTT solution (5 mg/mL) is added and incubated for 5 h. The medium is aspirated and replaced with 200 $\mu$ L/well of DMSO to dissolve the formazan Salinomycin 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%) [1].
Animal Research	Human glioma U251 cells ( $1 \times 10^7$ ) suspended in 100 $\mu$ L 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. Bodyweight 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 assay [1].

### Solubility Information

Solubility	DMSO: 135 mg/mL (174.65 mM),Sonication is recommended. ( $< 1$ mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (2.59 mM),Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>

### Preparing Stock Solutions

	1mg	5mg	10mg
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
50 mM	0.0259 mL	0.1294 mL	0.2587 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

### Reference

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

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