# Nigericin sodium salt

**Cat. No.:** HY-100381

**CAS No.:** 28643-80-3

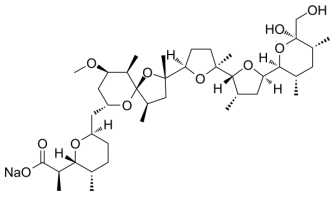
**Molecular Formula:** $C_{40}H_{67}NaO_{11}$

**Molecular Weight:** 746.94

**Target:** Potassium Channel; NOD-like Receptor (NLR); Bacterial; Antibiotic

**Pathway:** Membrane Transporter/Ion Channel; Immunology/Inflammation; Anti-infection

**Storage:** 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



## SOLVENT & SOLUBILITY

**In Vitro**

Ethanol : ≥ 50 mg/mL (66.94 mM)
* "≥" means soluble, but saturation unknown.

**Preparing Stock Solutions**

| Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|
| 1 mM | 1.3388 mL | 6.6940 mL | 13.3880 mL |
| 5 mM | 0.2678 mL | 1.3388 mL | 2.6776 mL |
| 10 mM | 0.1339 mL | 0.6694 mL | 1.3388 mL |

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

**In Vivo**

1. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.35 mM); Clear solution
2. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (3.35 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.35 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Nigericin sodium salt is an antibiotic from Streptomyces hygroscopicus that works by acting as an $H^+$, $K^+$, and $Pb^{2+}$ ionophore, a NLRP3 activator[1].

**$IC_{50}$ & Target**

NLRP3

**In Vitro**

Nigericin (0.1 μM) decreases inhibits proliferation and clonogenicity of H460 lung cancer cells in a dose dependent manner. Nigericin inhibits migration and invasion of H460 lung cancer cells[1]. Nigericin (0.1-10 nM) has apparently a dual effect on cell volume, that is a shrinking effect at lower Nigericin concentrations and a swelling effect at higher concentrations.

| | |
|---|---|
| | Nigericin (0.1-1 nM) significantly decreases cytosolic pH (pHi), and slightly increases the pHi at 5 and 10 nM[2]. Nigericin exhibits higher toxicity on S18 cells than S26 cells, with $IC_{50}$ of 2.03±0.55 μM and 4.77±2.35 μM, respectively. Nigericin can selectively kill cancer stem cells in NPC in vitro. Nigericin dramatically reduces the migration ability of S18 and HONE-1 cells [3]. Nigericin exhibits gteat toxicity for the HT29 and SW116 cell line with $IC_{50}$ of 12.92±0.25 μmol and 15.86±0.18 μmol. Nigericin also shows a decreased ability to form colonies under anchorage-independent conditions in a standard soft agar assay[4].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **In Vivo** | Ngericin (4 mg/kg, i.p.) significantly reduces tumor growth and acts synergistically with the chemotherapeutic agent DDP, as shown by the tumor volumes. Nigericin markedly decreases Bmi-1 in vivo. Overexpression of Bmi-1 partially restores CSC content and metastatic ability of NPC cells under Nigericin treatment. The downregulation of Bmi-1 may be involved in the inhibitory effect of Nigericin on CSCs in NPC[3].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

| | |
|---|---|
| **Cell Assay** [1] | For RCCs, cells (appr 2000 cells/well) are plated in 96-well cell-culture microplates and incubated over nigericinht in complete media (CM)-RPMI 1640 supplemented with 5% FBS and 2 mM l-glutamine. Cells are then exposed to the appropriate concentration of drug or vehicle for 72 h. For PPSS, cells (appr 500 cells/well) are plated in 96-well cell-culture microplates incubated over Nigericinht in CM to allow them to adhere and then maintained in serum-free media for 7-8 days and then treated with the appropriate concentration of drug or vehicle for 72 h in SFM. Cell viability for cells growing under RCCs and PPSS are evaluated by the MTT assay. The absorbance of solubilized formazan is read at 570 nm using ELISA (enzyme-linked immunosorbent assay) reader. In all cases, the highest concentration of DMSO is used in the control and this concentration is maintained below 0.001% (v/v). This DMSO concentration does not show any significant antiproliferative effect on the cell line in a short-term assay.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration** [3] | Mice[3]<br>The S18 cells are injected near the scapula of the nude mice. Nine days after injection, the mice are randomly divided into four groups with six animals each (control, DDP, Nigericin and DDP combined with Nigericin). DDP (2.5 mg/kg) is injected intraperitoneally for five continuous days and nigericin (4 mg/kg) is administrated intraperitoneally every two days. Tumor length and width are measured with a vernier caliper every other day. Tumor volume is calculated using the formula $V=0.5\times(length\times width^2)$. The body weights of the mice are recorded every two days. Mice are humanely euthanized when the tumor volume reach 2000 $mm^3$.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Cell Res. 2023 Sep 19.
- Cell Host Microbe. 2023 Nov 8;31(11):1820-1836.e10.
- Cell Metab. 2020 May 5;31(5):892-908.e11.
- Bioact Mater. 20 July 2022.
- J Extracell Vesicles. 2023 Feb;12(2):e12310.

See more customer validations on www.MedChemExpress.com

## REFERENCES

[1]. Yakisich JS, et al. Nigericin decreases the viability of multidrug-resistant cancer cells and lung tumorspheres and potentiates the effects of cardiac glycosides. Tumour Biol. 2017 Mar;39(3):1010428317694310

[2]. Bissinger R, et al. Triggering of Suicidal Erythrocyte Death by the Antibiotic Ionophore Nigericin. Basic Clin Pharmacol Toxicol. 2016 May;118(5):381-9

[3]. Deng CC, et al. Nigericin selectively targets cancer stem cells in nasopharyngeal carcinoma. Int J Biochem Cell Biol. 2013 Sep;45(9):1997-2006

[4]. Zhou HM, et al. Suppression of colorectal cancer metastasis by nigericin through inhibition of epithelial-mesenchymal transition. World J Gastroenterol. 2012 Jun 7;18(21):2640-8



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