Monensin sodium

Cat. No.: HY-N0150

CAS No.: 22373-78-0

Molecular Formula: $C_{36}H_{61}NaO_{11}$

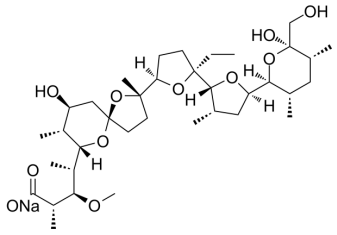
Molecular Weight: 693

Target: Bacterial; Antibiotic; Na+/H+ Exchanger (NHE); Parasite; Apoptosis; Fungal; Wnt

Pathway: Anti-infection; Membrane Transporter/Ion Channel; Apoptosis; Stem Cell/Wnt

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 : 20 mg/mL (28.86 mM; Need ultrasonic)

DMSO : 2 mg/mL (2.89 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.4430 mL	7.2150 mL	14.4300 mL
5 mM	0.2886 mL	1.4430 mL	2.8860 mL
10 mM	0.1443 mL	0.7215 mL	1.4430 mL

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

In Vivo

1. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 2 mg/mL (2.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Monensin (Monensin A) sodium, an orally active antibiotic, is an ionophore that mediates Na^+/H^+ exchange. Monensin sodium is a potent Wnt signaling inhibitor. Monensin sodium causes a marked enlargement of the multivesicular bodies (MVBs) and regulates exosome secretion. Monensin sodium can be used for bacterial, fungal, and parasitic infections research, and shows anticancer effects[1][2][3][4].

IC_{50} & Target

bacterial[1]

In Vitro

Monensin (1-5 μM; 48 h) sodium results in a marked decrease in viability in a dose-dependent manner[1].

Monensin (1-5 μM; 24 h) sodium shows a statistically significant induction of apoptosis[1].

Monensin (0.1-1 μM; 24 h) sodium inhibits pEGFR and its downstream targets pAKT and pERK[1].

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

Cell Viability Assay[1]

Cell Line:	SCC9, SCC25, and GM-38 cell lines
Concentration:	1-5 μM
Incubation Time:	48 h
Result:	Resulted in a marked decrease in viability in a dose-dependent manner.

Apoptosis Analysis[1]

Cell Line:	SCC25 cells
Concentration:	1 μM, or 5 μM
Incubation Time:	24 hours
Result:	Induced a potent apoptotic response.

Western Blot Analysis[1]

Cell Line:	SCC9 and SCC25 cells
Concentration:	0.1 μM, or 1 μM
Incubation Time:	24 hours
Result:	Induced approximately a 50% inhibition of EGF-treated SCC9 cells with respect to pEGFR and its downstream targets pAKT and pERK.

In Vivo

Monensin (10 mg/kg; po; daily; for 6 weeks) sodium suppresses progression of the intestinal tumors without any sign of toxicity on normal mucosa in multiple intestinal neoplasia (Min) mice[2].

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

Animal Model:	Multiple intestinal neoplasia (Min) mice (four-week-old) [2]
Dosage:	10 mg/kg
Administration:	Orally application; daily; for 6 weeks
Result:	Suppressed progression of the intestinal tumors without any sign of toxicity on normal mucosa.

CUSTOMER VALIDATION

- Nat Methods. 2023 Dec 4.
- Signal Transduct Target Ther. 2023 Jul 17;8(1):273.
- Nat Commun. 2022 Jul 22;13(1):4255.
- J Exp Med. 2023 Mar 6;220(3):e20221316.
- Small. 2021 Nov 1;e2103984.

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REFERENCES

[1]. Dayekh K, et al. Monensin inhibits epidermal growth factor receptor trafficking and activation: synergistic cytotoxicity in combination with EGFR inhibitors. Mol Cancer Ther. 2014 Nov;13(11):2559-71.

[2]. Tumova L, et al. Monensin inhibits canonical Wnt signaling in human colorectal cancer cells and suppresses tumor growth in multiple intestinal neoplasia mice. Mol Cancer Ther. 2014 Apr;13(4):812-22.

[3]. Youhua Huang, et al. Autophagy Participates in Lysosomal Vacuolation-Mediated Cell Death in RGNNV-Infected Cells. Front Microbiol. 2020 Apr 30:11:790.

[4]. Ariel Savina, et al. Rab11 promotes docking and fusion of multivesicular bodies in a calcium-dependent manner. Traffic. 2005 Feb;6(2):131-43.

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