Tunicamycin

Cat. No.: HY-A0098

CAS No.: 11089-65-9

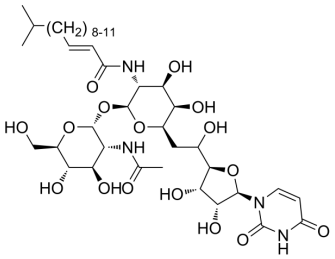
Molecular Formula: $C_{39}H_{64}N_4O_{16}$

Target: Bacterial; Fungal; Influenza Virus; Antibiotic

Pathway: Anti-infection

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

In Vivo

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

BIOLOGICAL ACTIVITY

Description

Tunicamycin is a mixture of homologous nucleoside antibiotic that inhibits N-linked glycosylation and blocks GlcNAc phosphotransferase (GPT). Tunicamycin causes accumulation of unfolded proteins in cell endoplasmic reticulum (ER) and induces ER stress, and causes blocking of DNA synthesis and cell cycle arrest in G1 phase. Tunicamycin inhibits gram-positive bacteria, yeasts, fungi, and viruses and has anti-cancer activity[1][2][3].Tunicamycin increases exosome release in cervical cancer cells[4].

In Vitro

Tunicamycin (2 μg/mL; 24 hours; CD44+/CD24- and original MCF7 cells) treatment increases the spliced XBP-1, ATF6 nuclear translocation level and CHOP protein expression in CD44+/CD24- and original MCF7 cells[1].
Tunicamycin-induced ER stress suppresses CD44+/CD24- phenotype cell subpopulation and in vitro invasion and accelerates tumorosphore formation. Under effect of Tunicamycin, the results show that inhibited invasion, increased cell death, suppressed proliferation and reduced migration in the CD44+/CD24- and CD44+/CD24- rich MCF7 cell culture[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis

Cell Line:	CD44+/CD24- and original MCF7 cells[1]
Concentration:	2 μg/mL

Incubation Time:	24 hours
Result:	Increased level of spliced XBP-1, ATF6 nuclear translocation and CHOP protein expression are detected in CD44+/CD24- and original MCF7 cells.

In Vivo

Tunicamycin (0.1 mg/kg or 0.5 mg/kg) treatment dramatically suppresses tumor growth in the $CD133^{+/-}$ MHCC97L cells xenograft model (BALB/c (nu/nu) mice)[2].

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

CUSTOMER VALIDATION

- Nature. 2020 Mar;579(7799):433-437.
- Cell. 2023 Feb 16;186(4):803-820.e25.
- Nat Commun. 2023 May 19;14(1):2859.
- Nat Commun. 2023 Feb 23;14(1):1020.
- Nat Commun. 2022 Apr 6;13(1):1853.

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REFERENCES

[1]. Hsu JL, et al. Tunicamycin induces resistance to camptothecin and etoposide in human hepatocellular carcinoma cells: role of cell-cycle arrest and GRP78. Naunyn Schmiedebergs Arch Pharmacol. 2009 Nov;380(5):373-82.

[2]. Han C, et al. Endoplasmic reticulum stress inhibits cell cycle progression via induction of p27 in melanoma cells. Cell Signal. 2013 Jan;25(1):144-9.

[3]. Hou H, et al. DPAGT1/Akt/ABCG2 pathway in mouse Xenograft models of human hepatocellular carcinoma. Mol Cancer Ther. 2013 Dec;12(12):2874-84.

[4]. Kathleen M McAndrews, et al. Mechanisms associated with biogenesis of exosomes in cancer. Mol Cancer. 2019 Mar 30;18(1):52.

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