Plicamycin

Cat. No.: HY-A0122
CAS No.: 18378-89-7
Molecular Formula: C₅₂H₇₆O₂₄
Molecular Weight: 1085.15
Target: DNA/RNA Synthesis
Pathway: Cell Cycle/DNA Damage
Storage: Powder -20°C 3 years
In solvent -80°C 6 months
-20°C 1 month

BIOLOGICAL ACTIVITY

Description
Plicamycin is a selective specificity protein 1 (Sp1) inhibitor. Plicamycin inhibits the growth of various cancers by decreasing Sp1 protein.

IC₅₀ & Target
Sp1 transcription factor[1]

In Vitro
Sp1 is a zinc-finger transcription factor that regulates multiple cellular functions and promotes tumor progression by controlling expression of genes involved in cell cycle, apoptosis and DNA damage. Sp1 binds to GC-rich motifs of promoters and interacts with components of the general transcriptional machinery and co-activator complexes of multiple signaling pathways. Plicamycin (Mith) decreases Sp1 protein by inducing proteasome-dependent degradation, thereby suppressing cervical cancer growth through a DR5/caspase-8/Bid signaling pathway. To assess the antiproliferative effects of Plicamycin on cervical cancer cells, two cervical cancer cell lines with different genetic backgrounds are grown with or without treatment with Plicamycin at different concentrations. Plicamycin inhibits HEp-2 and KB cell growth in a concentration-dependent manner after 48 h. Apoptotic cell death is qualitatively estimated by DAPI staining for nuclear condensation and fragmentation. Plicamycin leads to significant DNA fragmentation compared to untreated controls[1].

In Vivo
The antitumorogenic activity of Plicamycin (0.2 mg/kg/day) is determined in a xenograft model and observed reduction in tumor volume and weight. No significant mouse body weight loss is observed in Plicamycin-treatment groups, indicating that Plicamycin-associated toxicity is minimal. Plicamycin also increases TUNEL-positive cells in tumor xenografts. No notable intergroup differences are observed among organs, indicating no marked signs of systemic toxicity at the Plicamycin dose used in this study[1].

PROTOCOL

Cell Assay[1]
HEp-2 cells and KB cells are cultured in DMEM 100 U/mL each of Penicillin and Streptomycin and 10% FBS for HEp-2 cells and 5% FBS for KB in a humidified atmosphere containing 5% CO₂ at 37°C. Equal numbers of cells are seeded and allowed to attach. At 50-60% confluence, cells are treated with DMSO or indicated concentrations of Plicamycin (50, 100, and 200 nM for HEp-2 cells; 20, 40, and 80 nM for KB cells). Cell viability is determined using CellTiter 96 Aqueous One Solution Cell Proliferation Assay Kits. In brief, cells are seeded in 96-well plates and incubated with...
Plicamycin. After treatment, 30 µL MTS solution is added to each well and cells are incubated for 2 h at 37°C. MTS solution is analyzed using a microplate reader at 490 nm and 690 nm[1].

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

**Animal Administration [1]**

Female nude mice are used. KB cells are suspended in sterile PBS and injected subcutaneously into the right flank of mice. Mice are randomized into two groups containing five mice each and treated with 0.2 mg/kg/day of Plicamycin (i.p.) three times per week for 29 days. Control mice receive an equal volume of vehicle. After 29 days, bodies, organs and tumors are weighed and tumor volumes determined. Tumors are measured[1].

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**REFERENCES**