Actinomycin D

Dactinomycin, also known as actinomycin D, is a chemotherapy medication used to treat a number of types of cancer.

This includes Wilms tumor, rhabdomyosarcoma, Ewing's sarcoma, trophoblastic neoplasm, testicular cancer, and certain types of ovarian cancer. It is given by injection into a vein.

Most people develop side effects.

Common side effects include bone marrow suppression, vomiting, mouth ulcers, hair loss, liver problems, infections, and muscle pains.

Other serious side effects include future cancers, allergic reactions, and tissue death at the site of injection.

Use in pregnancy may harm the baby.

Dactinomycin is in the cytotoxic antibiotic family of medications.

It is believed to work by blocking the creation of RNA.

Dactinomycin was approved for medical use in the United States in 1964. It is on the World Health Organization's List of Essential Medicines, the most effective and safe medicines needed in a health system.

The wholesale cost in the developing world is about 24.73 USD per 500 mcg vial.

It is produced from bacteria Streptomyces parvullus.

Taylor et al. validated Actinomycin D as a potential repurposed therapeutic for glioblastoma in three-dimensional glioma stem-like cell lines (GSCs) cultures and patient-derived xenograft models of Glioblastoma recurrence.

Twelve patient-derived GSCs were screened at 10μM, as multicellular spheroids, in a 384-well serum-free assay with 133 FDA-approved compounds. GSCs were then treated in vitro with Actinomycin D at established IC50 concentrations. Downregulation of Sox2, a stem-cell transcription factor, was investigated via western blot and through immunohistological assessment of murine brain tissue.

Treatment with Actinomycin D was shown to significantly reduce tumor growth in two recurrent Glioblastoma (rGlioblastoma) patient-derived models and significantly increased survival. Actinomycin D is also shown to specifically downregulate the expression of Sox2 both in vitro and in vivo.

These findings indicate that, as predicted by the high throughput screening (HTS, Actinomycin D could deplete the cancer stem cell population within the tumor mass, ultimately leading to a delay in tumor progression [1].

1)

Taylor JT, Ellison S, Pandele A, Wood S, Nathan E, Forte G, Parker H, Zindy E, Elvin M, Dickson A,

Williams KJ, Karabatsou K, McCabe M, McBain C, Bigger BW. Actinomycin D Downregulates Sox2 and Improves Survival in Preclinical Models of Glioblastoma recurrence. Neuro Oncol. 2020 Mar 30. pii: noaa051. doi: 10.1093/neuonc/noaa051. [Epub ahead of print] PubMed PMID: 32227096.