The Efficacy of Ribosomal-Targeting Antibiotics on Breast Cancer Cell Proliferation: A Comparative Study of Chloramphenicol and Gentamicin Impact on Eukaryotic Ribosomal Subunits

Chen, Michelle (School: Joel E. Ferris High School)

The use of antibiotics to inhibit breast cancer cell proliferation is a novel approach to cancer treatment. Antibiotics, such as gentamicin and chloramphenicol, primarily known for their antibacterial properties, have been shown to interfere with protein synthesis by targeting ribosomal components. Gentamicin targets the 30S ribosomal subunit, while chloramphenicol targets the 50S subunit in prokaryotic ribosomes, which have similarities in protein synthesis machinery to mitochondrial ribosomes in eukaryotic cells. The cells were treated with various concentrations of the antibiotics gentamicin and chloramphenicol to determine the optimal doses, which were found to be 0.25 mg/mL and 0.1 mg/mL, respectively. The cell lines were analyzed for cell proliferation through a WST-1 cell proliferation assay and protein expression through western blotting and immunofluorescence staining. Data for the cell proliferation assay was quantified through a Tukey HSD test. ImageJ software was used to quantify data for western blotting and immunofluorescence staining. After data analysis, it was determined that chloramphenicol selectively inhibits the proliferation of MDA-MB-231 breast cancer cells while sparing non-cancerous MCF-10A cells, suggesting its potential as a targeted cancer therapy. The observed changes in protein expression, including decreased eIF4A1, increased Calnexin, and relatively stable Syntaxin levels, highlight the complex cellular responses to antibiotic treatment. Overall, this research supports the repurposing of antibiotics like chloramphenicol as innovative strategies in cancer treatment, warranting further investigation into their therapeutic potential.

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