The Effect of Naringin on Oct-1 Deficient Cells: The Fabulous Flavonoid, Year Two

Clark, Kendall

The goal of this project was to determine the possible application of naringin as a cancer treatment. This particular study used Octamer Transcription Factor 1 Knockout Mouse Embryo Fibroblasts and Wild Type Mouse Embryo Fibroblasts to determine how naringin would effect healthy cells in comparison to those deficient in OCT-1, which is a deficiency seen in several cancers. The researcher hypothesized that the naringin would have little to no effect upon the Wild Type MEFs at any concentration, and that OCT-1 deficient cells would go through apoptosis at rates proportional to the concentration of naringin used as a treatment, but would stop this trend after the 500 micromolar dose and would flatten out at the 1 millimolar dose. To conduct the trial the researcher used naringin in a serial dilution, starting at 1 millimolar and then decreasing by half (500 micromolar, 250 micromolar, etc.) to the 11th well in the row. The twelfth well was left without any naringin to provide a negative control. This process was completed for 8 rows in both the trail with Wild Type cells and the trial with the Knockout cells. Then both cell types were incubated and stained using Crystal Violet Stain. The trial using the Wild Type cells was then analyzed using ImageJ and the trial with the OCT-1 Knockouts used a cell plate reader. It was found through this experiment that naringin in a dose greater than or equal to 500 micromoles caused at least some wild type cells (healthy cells) to die; a dose of 1 millimole however, caused a great rate of cellular death. In the knockout cells it was seen in a common trend that the rate of apoptosis was proportional to the amount of naringin applied, with the greatest amount seen at the 1 millimolar dose.

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