Prototypical Galectins and the Innate Immunity Against Molecular Mimicry

McDonald, Marissa (School: Union Grove High School)

The objective of this project was to characterize gal-7 as an antibiotic. The purpose of this research was to identify a model for glycan-specific antibiotics capable of reducing the risk of leaky gut syndrome and auto-immune disease. Thus, the research question was, "What are the effects of human prototypical galectin-7 on the viability of E. coli O86, K. pneumoniae O1, K. pneumoniae O4, P. alcalifaciens O5, and P. alcalifaciens O19?" The hypothesis was, "If human prototypical galectin-7 is combined with E. coli O86, K. pneumoniae O1, K. pneumoniae O4, P. alcalifaciens O5, and P. alcalifaciens O19, then gal-7 will kill the bacteria that it respectively binds to due to its inherent binding structures, enabling them to disrupt the bacterial membrane." Binding assays were made by combining each strain with gal-7. Assays were analyzed using flow cytometry/microscopy. Bacteria with positive binding results were included in galectin killing assays; dosage response assays were conducted using varying concentrations of gal-7. Killing assays were later plated on agar plates and incubated overnight. Data was collected by counting colony forming units (CFUs) and standardizing numbers against the negative control to achieve a percentage of CFUs remaining after incubation with gal-7. Gal-7 was found to bind to the membranes of O86, KPO1, and PAO5, killing all three strains. Binding became increasingly polarized overtime, suggesting a disruption of membrane potential as a mechanism for killing. More research is needed to include a deeper analysis on how galectins kill bacteria. Early engineering/design should also be done to begin modeling antibiotics after these highly effective and specific proteins capable of answering some of the biggest problems in the biomedical industry.

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