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Title of Poster Presentation: Activation Level-Dependent Mutation Rates in the Affinity Maturation of B-cells

Abstract:
During a specific immune response, classes of B-cells are activated and then migrate to the germinal centers of lymph nodes. Within these germinal centers, the activated B-cells undergo affinity maturation, a process in which the correspondence between the recognition sites of B-cells and the invading pathogens dramatically increases. For the optimal response to an infection, affinity maturation, part of the germinal center reaction (GCR), must occur quickly. Current hypotheses indicate that the RNA-editor Activation-induced deaminase (AID) initiates the processes that alter the recognition sites of B-cells. It is known that AID facilitates both somatic hypermutation (SHM), the method primarily responsible for modifications of the recognition sites of B-Cells during the GCR, and class-switching recombination (CSR), another method responsible for alterations in the recognition sites. AID is thought to invoke a double-stand break in a DNA molecule within a break-repair pathway that is essential to SHM and CSR. This project used a model to compare affinity maturation with and without activation, where the double strand-break initiated by AID was considered activation. The purpose was to determine whether AID influenced affinity maturation through its activation. In accordance with the biological necessity of AID, the AID-activated simulations revealed a shorter, biologically-preferable time for B-cells to reach the optimal level of affinity for the pathogen.