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Antigenic Distance, Glassy Dynamics, and Localization in the Immune System

The immune system normally protects the human host against death by infection. I will introduce a hierarchical spin glass model of the evolutionary dynamics that occurs in the antibody-mediated and T cell-mediated immune responses. The theory will be used to provide a mechanism for original antigenic sin, wherein an initial exposure to antigen degrades the response of the immune system upon subsequent exposure to related, but different, antigens. A new order parameter to characterize antigenic distance will be introduced from the theory. This order parameter predicts effectiveness of the influenza vaccine more reliably than do results from animal model studies currently used by world health authorities. This order parameter would seem to be a valuable new tool for making vaccine-related public health policy decisions. Next, I will note that while the immune system normally protects the human host against death by infection, the method used by the immune system to search sequence space is rather slow --- interestingly there exist biological mechanisms that can find antibodies with higher affinity and also find them more quickly. Thus, one would think that these more powerful evolutionary mechanisms would give an immune system that responds faster and more effectively against disease. So, why didn't we evolve that kind of adaptive response? I will show that the slow glassy dynamics of the immune system serves a functional role of inhibiting the autoimmune diseases that these more powerful searching mechanisms would induce. I will suggest that the controversy related to the correlation between chronic infection and autoimmune disease might be addressed by searching for the broad distribution of onset times for autoimmune disease predicted from the theory.