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Antibody responses during Hepatitis B viral infection

Infection with hepatitis B virus results in the synthesis of a large excess of subviral particles, which are empty particles with viral proteins on their surface but without viral nucleic acids. The reasons for their overproduction and the contribution they play in HBV pathogenesis is not understood. Here, we investigate whether subviral particles can serve as a decoy by adsorbing neutralizing antibodies and therefore delaying the clearance of infection. We develop a mathematical model of HBV-antibody interaction and determine the quantitative contributions of virus-antibody and subviral particles-antibody formation to the control of infection. We extend the results to account for the presence of multiple Hepatitis B surface proteins, each of which can potentially facilitate infection. Using this extended model we investigate the necessity for the antibody to bind all available surface proteins to offer protection.