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Mathematical Models of the Role of Immune Exhaustion in Hepatitis B and Delta Coinfection

Hepatitis Delta Virus (HDV) is a dependent satellite virus of the more common Hepatitis B Virus. HDV encodes only one protein of its own, relying on HBV to supply the additional proteins needed for its replication cycle. Although it HDV is noncytotoxic and present few targets for immune reaction, the symptoms of patients with HBV-HDV coinfection are much worse than those infected with HBV alone. The cause of this negative outcome is not clear. This work presents o.d.e. models for the interaction of HBV, HDV and the specific immune responses to each, and analyzes the implication of these models for understanding patient outcomes. In particular, the role of T cell exhaustion in chronic HBV is explored, and how superinfection with HDV may actually strengthen the HBV specific immune response, indirectly leading to the observed symptoms. Additionally, the role of nonspecific immune responses is explored.