INDIAN INSTITUTE OF TECHNOLOGY GUWAHATI
SHORT ABSTRACT OF THESIS

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In this thesis, a mathematical model for HCV dynamics incorporating various aspects such as virus-to-cell as well as cell-to-cell transmission and non-cytolytic cure rate with the role of B cells in the activation of the humoral immune response is presented and analyzed. Next, the model is modified by involving delay in the generation of B cells (delay differential equations model). Further, the model is modified by considering the spatial mobility of the virions as well as the immune B cells with general nonlinear incidence functions for both the modes of infection spread (reaction-diffusion model). The feasibility of all these three models is justified by establishing the uniqueness, non-negativity and boundedness of the solutions to the corresponding system. The local as well as global stability of the three equilibria, namely, infection-free equilibrium, immune-free infected equilibrium and immune-activation infected equilibrium for these three models are investigated theoretically as well as numerically in terms of conditions on the basic reproduction number and humoral immune reproduction number. The global stability analysis for these models is examined by constructing suitable Lyapunov functionals. The conditions for the existence of Hopf bifurcation and consequent occurrence of bifurcating periodic orbits around the interior equilibrium for the delay differential equations model is determined by taking time delay as the bifurcation parameter. Numerical simulations are performed to support the theoretical results obtained from these three models. A numerical comparison of the dynamics for HCV models under various consideration is illustrated. The effect of some sensitive parameters such as cell-to-cell transmission rate, cure rate and development rate of B cells are numerically presented and discussed. The numerical findings indicate that the inclusion of cell-to-cell transmission increases the concentration of infected hepatocytes, while the cure rate increases the level of uninfected hepatocytes. The effect of the humoral immune response is crucial in neutralizing the virions and is less impactful on the reduction of infection. The results obtained from the delay model also show that the system becomes unstable from stable and regains stability from instability depending on the development rate of B cells for a fixed delay value. In addition, a numerical comparison of the dynamics resulting from the reaction-diffusion model with bilinear as well as Holling type-II incidence functions for both modes of transmission is studied. Finally, stochastic models for HCV dynamics are constructed and analyzed using the property of linear transformation for multivariate normal distribution, based on both budding and bursting processes with fixed as well as variable burst size for the

release of virions from the infected hepatocytes. The branching process approximation technique is used to determine the probability of virus extinction. The stochastic means with standard deviations for the model populations are numerically calculated and are compared with the results from the deterministic model. Moreover, the forward Kolmogorov and moment equations associated to the stochastic models for both budding and bursting processes are derived and numerically illustrated with a particular case.