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Commentary

Evaluation of Basic process of HIV Infection During Anti-Retroviral Therapy

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DESCRIPTION

A mathematical host model for Human Immunodeficiency Virus (HIV) transmission that takes therapy into account is developed. The effectiveness of combination antiretroviral therapy on viral proliferation and T cell populations in human blood is taken into account by the model. It is established that there is a healthy, infection-free endemic balance. The next generation matrix approach is used to obtain the fundamental reproduction number. We analyze the equilibria points' local and global stability and demonstrate that if the infection-free equilibrium is globally asymptotically stable, then the virus is theoretically eliminated and the disease disappears, and if the endemic equilibrium is globally asymptotically stable, then the virus is likely still present in the host. The impact of treatment on the dynamics of the infection within the host is investigated using numerical simulations.

The Human Immunodeficiency Virus (HIV) has posed a serious threat to human life. Acquired Immunodeficiency Virus (AIDS), a disease that has decimated the human population worldwide, is brought on by HIV infection in humans. There has been a lot of research done on the disease's containment or eradication since its discovery. Viral diseases including HIV, Hepatitis B, Hepatitis C, and Dengue Fever can now be predicted and controlled for spreading thanks to mathematical modelling of viral infections, which has improved understanding of virus dynamics. The basic model, one of the earliest models of HIV infection, was utilized, and it was successful in statistically reproducing the dynamics of HIV's early phases and its target CD4+ cells following an infection event.

Recent research has concentrated on the dynamics of intracellular delays, latent infection, viral mutation, and geographic heterogeneity in HIV viral and cellular infections. As an illustration, researchers looked at the global stability of within-host virus models with cell-to-cell viral transmission and came up with a comprehensive analytic description of equilibria. It was suggested to use a four-dimensional system of delayed differential equations, where the virus and cell production and removal rates are determined by common nonlinear functions. Incorporating humoral immune response, their model looked at the dynamic behaviour of virus target and cell target incidences.

Infection-free equilibrium, chronic free equilibrium with inactive humoral immune response, and chronic infection equilibrium with active humoral immune response were established as the three main equilibrium results. The authors demonstrated the equilibria's overall stability utilizing Lyapunov functionals and Lasalle's invariance principle along with dynamics controlled by the two bifurcation parameters basic reproduction numbers and the humoral immunity numbers.

CONCLUSION

In recent years, there has been a lot of interest in include therapy in mathematical modelling at the within-host and between-host levels. For instance, epidemiological models with saturated treatment functions have been developed at between-host levels. Early models highlighted the effects of azidothymidine on viral replication. Research on within host models that incorporate therapy has been conducted over the years. One of the main conclusions is that viral decrease is drug-dependent.

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