Dynamics of an HIV Model with Variable CD4⁺ T-Cell Source and Saturated Incidence

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Abstract:
A nonlinear within-host HIV infection model is analyzed. The model incorporates a variable CD4⁺ T-cell source term and Holling type II saturated incidence. This accounts for immune exhaustion, finite receptor availability, and physiological limitations in T-cell proliferation, thereby extending classical mass-action formulations. The basic reproduction number () is derived using the next-generation matrix approach and shown to govern the threshold dynamics of infection. When , the disease-free equilibrium is globally asymptotically stable; when , the system exhibits a locally stable endemic equilibrium representing chronic infection. Local stability of the disease-free equilibrium is investigated via the Routh–Hurwitz criterion, while Lyapunov functional techniques are employed to establish global stability results. The existence and local stability of the endemic equilibrium are also demonstrated. Numerical simulations produced clinically observed HIV dynamics. This shows that adding biologically inspired mechanisms improves the within-host HIV models' clinical interpretability and mathematical stability.

Keywords:
HIV dynamics, Holling type II incidence, immune exhaustion, CD4⁺ T cells, stability analysis.