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Threshold dynamics of an age-structured HIV model with virus-to-cell, cell-to-cell transmissions, and CTL immune response

Both virus-to-cell and cell-to-cell transmission modes play a crucial role in the long-term dynamics of HIV infection. Additionally, the immune response — particularly the activity of cytotoxic T lymphocytes (CTLs) — can significantly influence the threshold conditions for viral persistence. Considering that the delay between cells being infected and becoming infectious may vary, we established an infection age structure model with a general nonlinear incidence rate to explore the intricacies of HIV transmission and progression. This model allows key parameters, including the viral particle production rate, the infection rate of healthy cells, the mortality rate of infected cells, and the proliferation and clearance rates of CTLs, to vary with the age of infection.

We derive the immune-inactivated reproduction number $\mathcal{R}_0$ and the immune-activated reproduction number $\mathcal{R}_1$ to identify conditions for the persistence of the virus within the host.

When $\mathcal{R}_0 < 1$, the virus-free steady state E_0 is globally asymptotically stable, indicating that the virus is eventually cleared.

When $\left(1 - \frac{1}{\mathcal{R}_0}\right) \frac{s\omega_2}{d_3} < 1 < \mathcal{R}_0$, the CTL-inactivated infection steady state E_1^* is locally stable. Under these circumstances, we establish criteria for the global stability of E_1^*, which implies the virus and infected cells persist, resulting in a chronic infection without activating a CTL immune response.

When $\mathcal{R}_1 > 1$, the CTL-activated infection steady state E_2^* becomes locally stable, and condition for the global stability of E_2^* is provided. In this case, the infection remains chronic, sustained by an active CTL immune response.