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Viral dynamics with immune responses: effects of distributed delays and Filippov antiretroviral therapy

In this talk, we propose a general viral infection model that incorporates two distinct infection pathways—virus-to-cell and cell-to-cell transmission—as well as the cytotoxic T lymphocyte (CTL) immune response and distributed intracellular delays associated with viral infection, viral production, and CTL recruitment processes. We investigate the existence, the uniqueness, and global stability of three equilibria: the infection-free equilibrium E_0, the immune-inactivated equilibrium E_1 and the immune-activated equilibrium E_2. Our analysis shows that the viral dynamics are governed by two threshold parameters: the basic reproduction number for infection, R_0, and the basic reproduction number for immune response R_{IM}.

In addition to the theoretical results, we numerically explore the viral dynamics beyond equilibrium stability. Our simulations reveal that delays in CTL recruitment can induce transitions between stable equilibria and sustained oscillatory behavior in the viral load, and vice versa. Furthermore, we compare the relative contributions of virus-to-cell and cell-to-cell infection modes to the overall infection level and identify key parameters that influence this balance.

Finally, we demonstrate how Filippov control methods can be employed in antiretroviral therapy to achieve desirable treatment outcomes.