Abstract

Numerous models of mathematics have existed to pronounce the immunological response to contagion by human immunodeficiency virus (HIV-1). The models have been used to envisage the regression of HIV-1 in vitro and in vivo dynamics. Ordinarily the studies have been on the interface of HIV virions, CD4+T-cells and Antiretroviral (ARV). In this study, time delay, chemotherapy and role of CD8+T-cells is considered in the HIV-1 in-vivo dynamics. The delay is used to account for the latent time that elapses between exposure of a host cell to HIV-1 and the production of contagious virus from the host cell. This is the period needed to cause HIV-1 to replicate within the host cell in adequate number to become transmittable. Chemotherapy is by use of combination of Reverse transcriptase inhibitor and Protease inhibitor. CD8+T-cells is innate immune response. The model has six variables: Healthy CD4+T-cells, Sick CD4+T-cells, Infectious virus, Non-infectious virus, used CD8+T-cells and unused CD8+T-cells. Positivity and boundedness of the solutions to the model equations is proved. In addition, Reproduction number (R_0) is derived from Next Generation Matrix approach. The stability of disease free equilibrium is checked by use of linearization of the model equation. We show that the Disease Free Equilibrium is locally stable if and only if $R_0<1$ and unstable otherwise. Of significance is the effect of CD8+ T- cells, time delay and drug efficacy on stability of Disease Free Equilibrium (DFE). From analytical results it is evident that for all $\tau > 0$, Disease Free Equilibrium is stable when $\tau = 0.67$. This stability is only achieved if drug efficacy is administered. The results show that when drug efficacy of $\alpha_1=0.723$ and $\alpha_2=0.723$ the DFE is achieved.