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Modelling latency using a distribution of life spans among infected cells

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Understanding the mechanisms of HIV latency is important in the development of strategies for managing infection. Time from infection until production of virus has been shown to vary among infected cells, hence challenging the dichotomous assumption that cells are either latent or productively infected at time of infection. In this paper, we will explore the implications of an alternative hypothesis that rates of activation follow a probability distribution, of which latency is just an extreme of this spectrum. We show the emerging dynamics from this mathematical model and test its ability to explain features or reservoir formation and decay observed in SIV data. Analysis of SIV DNA levels in macaques that start treatment at different times shows that the decay rates of cells are statistically different in early and late initiation of treatment. Modelling suggests that data can be explained by a spectrum of reactivation rates of infected cells.

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