

Abstract Submitted
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Stochastic extinction dynamics of HIV-1¹ IRA SCHWARTZ², US Naval Research Laboratory, ERIC FORGOSTON³, Montclair State University, LEOR WEINBERGER⁴, Gladstone Institute of Virology and Immunology and University of California, San Francisco — We consider an HIV-1 within host model in which T cells are infected by the virus. Due to small numbers of molecules, stochastic effects play an important role in the dynamical outcomes in that two states are observed experimentally: a replication state in which the virus is active, or a dormant state leading to latency in which the virus becomes active after a delay. The two states are conjectured to be governed by the Tat gene protein transcription process, which does not possess two stable attractors. Rather, the active state is stable, while the dormant state is unstable. Therefore the dormant state can only be achieved through the dynamics of stochastic fluctuations in which noise organizes a path to dormancy. Here we use optimal path theory applied to a Tat gene stochastic model to show how random fluctuations generate the dormant state by deriving a path which optimizes the probability of achieving the dormant state. We explicitly show how the probability of achieving dormancy scales with the transition rate parameters.

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