Hindawi Publishing Corporation
Discrete Dynamics in Nature and Society
Volume 2013, Article ID 720818, 16 pages
http://dx.doi.org/10.1155/2013/720818

Research Article

Network Firewall Dynamics and the Subsaturation Stabilization of HIV

Bilal Khan,[1] Kirk Dombrowski,[2] Mohamed Saad,[3] Katherine McLean,[4] and Samuel Friedman[5,6]

[1] *Department of Math and Computer Science, John Jay College (CUNY), New York, NY 10019, USA*
[2] *Department of Anthropology, John Jay College (CUNY), New York, NY 10019, USA*
[3] *Social Networks Research Group, John Jay College (CUNY), New York, NY 10019, USA*
[4] *Department of Sociology, CUNY Graduate Center, New York, NY 10016, USA*
[5] *Institute for AIDS Research at National Development and Research Institutes, Inc., New York, NY 10010, USA*
[6] *Center for Drug Use and HIV Research, New York, NY 10003, USA*

Correspondence should be addressed to Bilal Khan; bkhan@jjay.cuny.edu

Received 29 December 2012; Revised 20 March 2013; Accepted 25 March 2013

Academic Editor: Carlo Piccardi

In 2001, Friedman et al. conjectured the existence of a "firewall effect" in which individuals who are infected with HIV, but remain in a state of low infectiousness, serve to prevent the virus from spreading. To evaluate this historical conjecture, we develop a new graph-theoretic measure that quantifies the extent to which Friedman's firewall hypothesis (FH) holds in a risk network. We compute this new measure across simulated trajectories of a stochastic discrete dynamical system that models a social network of 25,000 individuals engaging in risk acts over a period of 15 years. The model's parameters are based on analyses of data collected in prior studies of the real-world risk networks of people who inject drugs (PWID) in New York City. Analysis of system trajectories reveals the structural mechanisms by which individuals with mature HIV infections tend to partition the network into homogeneous clusters (with respect to infection status) and how uninfected clusters remain relatively stable (with respect to infection status) over long stretches of time. We confirm the spontaneous emergence of network firewalls in the system and reveal their structural role in the nonspreading of HIV.

1. Introduction

Social network research among people who inject drugs (PWID) has produced considerable data on HIV-1 infection profiles and equally detailed data on the broad demographic and behavioral profiles of injecting communities and their risk behaviors. However, prior research has not—and for reasons of cost often cannot—produce long-term, dynamic data on these same populations. Risk networks—graphs whose vertices are individuals and edges are social connections bearing disease transmission risk—are now widely recognized as a critical construct in understanding infection patterns [1, 2], as they represent the natural environment in which risk behaviors take place and through which infection propagates. Such a representation shifts our view of risk away from individual behaviors to collective, social bodies as the carriers and transmitters of infections [3, 4]. Modeling risk networks as (stochastic) discrete dynamical systems provides an opportunity to understand (through both analysis and simulation) the long-term behavior of PWID risk networks themselves—well beyond what can be seen by considering their constituent individuals in isolation.

HIV has been investigated extensively in a number of PWID communities, including New York City [5], where there was a rapid initial spread of the virus among PWID in the early 1980s, but where HIV prevalence stabilized to between 40 and 50% (i.e., at much lower than 100% or "saturation" levels), despite the fact that risk behaviors could result in infection remained common [6]. One interesting aspect of HIV's natural history is the fact that its viral burden has a tendency to transition from an acute, highly infectious phase to a chronic phase where overall infectiousness is much