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Effects of density of infected population to the spreading of HIV epidemic in communities

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ABSTRACT

We present a dynamical model for the spread of HIV in a finite discrete population of size n represented by the set V. The model takes into account the structure of the sexual network, the density of infected population in each individual's sexual partners, and a reasonable amount of the influence of noninfectious HIV positive individuals on HIV infectious partners which may occur in communities. In our analytic results, we give a precise epidemic threshold which, together with certain network properties, is then used to analyse and investigate the existence of epidemic and stability in the HIV spreading dynamics. Our results reproduce common observed patterns in the dynamics of HIV spreading in communities.

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1. Introduction

The most urgent public-health problem in Sub-Saharan Africa and other parts of the world today, in the absence of a cure, is to devise effective strategies to minimise the spreading of the human immunodeficiency virus (HIV). In general mathematical models have been used in such complex problems to make predictions which help in the understanding of the infectious disease dynamics and thereby assisting public-health researchers and policy makers to prepare for, detect, and respond to these infectious disease threats. To date, most mathematical models [1–5] put forward to explain HIV spreading among humans are standard compartmental models based on ordinary or partial differential equations. Although most of these traditional models have succeeded in explaining important aspects involved in the HIV transmission, they have some limitations. Firstly, they assume a very large density of interacting agents [6], yet in some communities the number of interacting actors maybe small; or even if it is large, the local number of interacting actors can be very low. These standard epidemiological models assume that interaction of agents or actors is random. For sexually transmitted diseases, however, random mixing is clearly a poor approximation of the underlying social reality [7]. Sex is simply not random (see, Ref. [8]).

Secondly, they assume that every actor has equal contact with every other actor in the population. Thus, these models provide a good approximation of virus spreading in networks where the contact among individuals is sufficiently homogeneous. However, most infectious diseases are transmitted through populations via links formed by contacts among individuals. The patterns of these contacts tend to be highly heterogeneous. For instance empirical studies (see, for example Refs. [8,9]) demonstrate that variations in the number of sexual partners is very large and while most people report 1–4 sexual partners during their lifetimes, some report several hundred or more. In recent applications (see, for example Ref. [10]) of the standard models to Severe Acute Respiratory Syndrome (SARS), the estimates of the basic reproduction number, R_0, predicted by the model were different from the observed epidemiology patterns. This inconsistency was attributed to two factors, namely, the basic premise of standard models that all individuals in a community are equally

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likely to become infected (or infect others) and the fact that in standard models $R_0 > 1$ predicts, with certainty, an epidemic, whereas in practise $R_0 > 1$ does not guarantee that an outbreak will ignite an epidemic [10].

Thirdly, these models often assume a universal infection rate, based on the fact that rate of infection is largely determined by the density of infected population [11]. Traditional epidemiological theory assumes that each infected actor infects R_0 other actors. In practise it is conceivable that certain individuals only infect one or zero other actors whereas other individuals infect dozens of other actors—the latter individuals being the superspreaders [10].

As such various discrete models [12,7,13,14,10] have also been proposed to explain the spread of diseases in general. The starting point for most discrete models is the fact that the human population can be described as a network (graph) whose vertices are the single individuals and edges representing interactions among them. Analytic tools are then sought to conceptualise interaction structure at the level of persons and as such a cross-disciplinary field known as Social Network Analysis (SNA) is providing these tools [8]. These models have considerable success in capturing local behaviour of actors. Some of the limitations and applicability of these earlier discrete models are reported in Refs. [8,11,15]. Apart from assuming homogeneity and assuming particular propagation topology, e.g., models that are tailored to fit special-case graphs (BA power-law, Erdos–Renyi), most of these models have been prized as powerful tools to conceptualise static networks, but because sexual interaction is not static [8], they are inefficient to make predictions. Another tool used in these discrete models is computer simulation. Although computer simulations allow for all sorts of heterogeneity of persons and networks to be easily built into the model, it is difficult to ignore the fact that this results in the model containing a very large parameter space. Consequently the model becomes much harder to thoroughly analyse than in an analytical mathematical model [8]. Further, some of the network models (for example, Ref. [11,10]) offer simple and accurate predictions, but are just not applicable to HIV studies.

In this paper, we aim to develop a general simple non-static model for HIV spreading in communities. Our dynamical model assumes neither homogeneity in connectivity of the network nor a universal infection rate and it makes no *a priori* assumptions about the community network structure. In the model individuals and sexual relationships are represented as a general graph and the spread of the virus via contacts is monitored over time. Our results are derived from rigorous mathematical proof, and not from computer simulations. The article is organised as follows: In the next section we present the model. Results are presented in Sections 3 and 4 is reserved for discussion.

2. The model

Unlike in standard *SIR* models for epidemics (see, for example Ref. [2]) where the population is divided into three compartments, susceptibles, infectives and recovered, here for HIV we adopt (see, for example Ref. [4]) three basic states which an actor in the community may have at a given time: *susceptible* (S), *infectious* (I) and *noninfectious* (P). The compartment of susceptibles consists of those actors not infected by the virus, i.e., the HIV negative individuals. The infectious class consists of HIV positive individuals who do not take due care and can transmit the virus, and the noninfectious class consists of AIDS patients and HIV positive individuals with a deontologist view on the risks involved in sexual partnering and therefore cannot transmit the virus. In the dynamics, infectious actors are the only actors who can transmit the virus to the susceptibles. Further, we propose that the spread of the HIV from one actor to the other in each discrete time step (which may range from days to years) is governed by the following basic transitional rules:

R1: *A susceptible individual becomes infected in the next time step if the density of his/her HIV infectious sexual partners in his/her sexual partners exceeds a fraction α^*; otherwise the individual stays susceptible.*
   This rule is an actor-localised version of the fact that the rate of infection is largely determined by the density of the infected population [2,4,11]. Further, this is also how we incorporate each uninfected individual's number of sexual partners and the states of these sexual partners. We will call α^* the parameter of infectivity.

R2: *An infectious actor v becomes noninfectious if at least half of its partners are noninfectious, otherwise it stays infectious.*
   This is how we model the fact that opinion change on HIV infectious class follows a majority process in which at each time step an infectious individual changes his/her opinion regarding taking due care, i.e., joins the noninfectious class, if at least half of his/her contacts have a deontological view on HIV spreading consequences. In general, majority processes do not seem to be relevant to the spread of disease, but they are relevant for opinion change [16,17]. Hence here we assume the majority-rule model, which states that individuals preferentially follow the crowd in their opinion update.

R3: *Noninfectious actors remain noninfectious.*

In this paper we investigate, using graph-theoretic methods, how changes in α^*, in conjunction with the graph structure, determine the spreading dynamics of HIV.

2.1. Graphs

A *graph* $G = (V, E)$ consists of a finite set $V = V(G)$ of *vertices* together with a set $E = E(G)$ of *edges* joining certain pairs of vertices of G. The *distance* $d(u, v)$ between vertices u and v in G is defined as the length of a shortest path joining u and v in G. For a subset $S \subseteq V$ and a vertex v of G the distance $d(v, S)$ between v and S is defined as the minimum value of $d(v, x), x \in S$. The set of neighbours of v is denoted by $N(v) = \{x \in V : d(v, x) = 1\}$ and $|N(v)|$ is the *degree* of v denoted $\deg(v)$. An *end-vertex* is a vertex of degree 1. We will denote the largest degree of vertices in G by Δ.
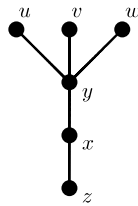
Fig. 1. G: $\alpha^* = 50\%$; initial configuration: $c_0(y) = $ red, $c_0(\{u, v, w, x\}) = $ white, $c_0(z) = $ black.

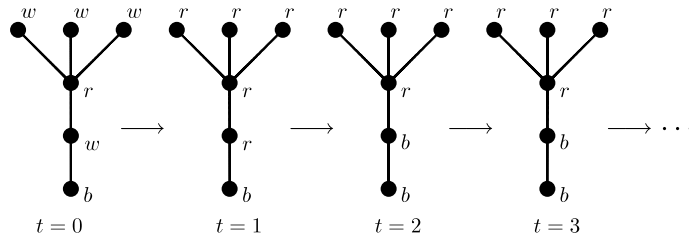**Fig. 2.** Applying the rules for G above: $w = $ white, $r = $ red, and $b = $ black.

We model the above situation, i.e., the community and its sexual partnering by a graph G as follows. The vertex set of G is the set of people in which two vertices are joined by an edge if and only if the corresponding individuals have a sexual relationship. Thus, for example, the number of neighbours of a vertex is simply the number of sexual partners the corresponding individual has. At each time step we will, according to rules equivalent to $R1$, $R2$ and $R3$ above, colour the vertices of G using colours, *white*, *red* and *black*. The colours signify a susceptible individual, infectious individual, and a noninfectious individual, respectively. We will use the following notation. Let v be a vertex of G. Then $c_t(v)$ denotes the colour of v at time t and $\eta_t(v, C)$ denotes the number of neighbours of v with colour C at time t. If $B \subseteq V$ is such that all its vertices have the same colour C, say, at time t, we write $c_t(B) = C$.

Now we play the following game on G. A move consists of an update of the colours of the vertices in a synchronised parallel way, according to the following rules: Initially at time 0 some vertices of G are coloured white and a few are coloured red and black.

Let $v \in V$.

G1: If $c_t(v) = $ white, then

$$c_{t+1}(v) = \begin{cases} \text{red} & \text{if } \dfrac{\eta_t(v, \text{red})}{\deg(v)} \geq \alpha^* \\ \text{white} & \text{otherwise.} \end{cases}$$

G2: If $c_t(v) = $ red, then

$$c_{t+1}(v) = \begin{cases} \text{black} & \text{if } \dfrac{\eta_t(v, \text{black})}{\deg(v)} \geq \dfrac{1}{2} \\ \text{red} & \text{otherwise.} \end{cases}$$

G3: If $c_t(v) = $ black, then $c_{t+1}(v) = $ black.

For an example, see Figs. 1 and 2.

Here and in the sequel I and P are subsets of V consisting of all vertices of G coloured red and black, respectively, in the initial configuration. Note that in practise, I and P represent the set of infectious and noninfectious actors, respectively, in the initial infection. We also use the following notation. The integer $e = e(I \cup P)$ is the maximum value of $d(x, I \cup P)$, $x \in V$. For each integer $i = 0, 1, 2, \ldots, e$, let N_i denote the set $N_i = \{x \in V : d(x, I \cup P) = i\}$. So for $t > e$, $N_t = \emptyset$. It is elementary to show that for $i \neq j$, $N_i \cap N_j = \emptyset$ and that no vertex in N_i has a neighbour in N_{i+2}, $i = 0, 1, \ldots, e - 2$. Further, we will also assume that $e > 1$. For our purposes, this assumption is reasonable since in a community it is natural to expect that not all actors are partners of initially infectious or noninfectious actors. We will also assume that the graph G is connected since the virus can only be transmitted between connected actors.

3. Results

We will make an investigation on the conditions guaranteeing an epidemic and stability. Unlike in continuous compartmental methods where a threshold, R_0, widely known as the reproduction number, depends on the transmissivity rate, the recovery rate and the initial number of susceptibles, we will show here that a critical value for our model depends on the infectivity parameter, α^*, and the largest of the degrees of the vertices in the network. Recall that we denote the

maximum degree of G by Δ. A vertex with the biggest degree represents an actor with the largest number of sexual partners. In most considered homogeneous models all actors are assumed to have the same degree, i.e., Δ. For brevity we denote the quantity $\frac{\Delta-1}{\alpha^*\Delta}$ by R_0. We will prove that R_0 is a threshold for this model though it possesses no obvious direct relationship with the reproduction number for standard models.

3.1. Existence of infection free subpopulation: $R_0 < 1$

We will show in the next theorem that if $R_0 < 1$, then there exists a subpopulation of a reasonable size which, despite being connected to the network, will never contract the disease.

Theorem 1. *Assume the above notation. If $R_0 < 1$, then there exists a set $W \subset V$ such that*

$$c_t(W) = white \text{ for all } t.$$

Moreover $|W| \geq e(I \cup P)$.

Proof. $R_0 < 1$ iff $\frac{\Delta-1}{\Delta} < \alpha^*$. Let $A = \{x \in N_1 : N(x) \subseteq I \cup P\}$ and set $W = V - (I \cup P \cup A)$. We show that

$$\text{if } v \in W, \quad \text{then } c_t(v) = white \text{ for all } t \tag{1}$$

from which it follows that $c_t(W) = $ white for all t. Clearly $c_0(W) = $ white. Therefore, to establish (1), it is sufficient to show that there is no vertex v of W for which $c_t(v) = $ red for some t. Suppose to the contrary that $v \in W$ is such that $c_t(v) = $ red. We choose v so that t is the smallest integer with this property. By $G1$, we must have

$$\frac{\eta_{t-1}(v, \text{red})}{\deg(v)} \geq \alpha^* > \frac{\Delta-1}{\Delta}. \tag{2}$$

Denote $\eta_{t-1}(v, \text{red})$ by p, $\eta_{t-1}(v, \text{black})$ by k and $\eta_{t-1}(v, \text{white})$ by q. Then $\deg(v) = p + k + q$ and in conjunction with (2), we obtain

$$\frac{p}{p+k+q} > \frac{\Delta-1}{\Delta}.$$

Hence $\Delta(k + q) < p + k + q = \deg(v) \leq \Delta$. Therefore, $k = q = 0$. Thus $\eta_{t-1}(v, \text{black}) = 0$ and $\eta_{t-1}(v, \text{white}) = 0$, i.e., at time $t - 1$, all the neighbours of v are red. By construction of W, v has at least one neighbour, x say, in W. Then $c_{t-1}(x) = $ red, contradicting our choice of v and the minimality of t. This proves (1).

We now prove that $|W| \geq e(I \cup P)$. Since $e > 1$, $|N_1 - A| \geq 1$. Note that $W = (N_1 - A) \cup N_2 \cup N_3 \cup \cdots \cup N_e$, where $e = e(I \cup P)$. Since $|N_i| \geq 1$ for all $i = 0, \ldots, e$, we have $|W| = |N_1 - A| + \sum_{i=2}^{e} |N_i| \geq e(I \cup P)$, as desired. □

3.2. Existence of an epidemic: $R_0 > 1$

In this subsection we will show that the presence of certain substructures enhance the spread of HIV. Each substructure is rooted on an individual whom we will refer to here as a "superspreader". We formally define this idea below, but first we require some notation.

For a vertex v of G we will denote the set $\{x \in N(v) \mid \deg(x) = 1\}$, i.e., the set of all neighbours of v which are end-vertices, by $S(v)$ and $S[v] = S(v) \cup \{v\}$.

Definition 1. Let v be a vertex of G for which $|S(v)| > \frac{\deg(v)}{2} \geq 1$. We say that v is an i-superspreader if $c_i(v) = $ red and $c_i(S(v)) = $ white.

As an example consider Figs. 1 and 2. Note that $S(y) = \{u, v, w\}$ and vertex y is a 0-superspreader.

Now let $s(t)$ be the number of i-superspreaders of G, $i \leq t$. Clearly s is a non-decreasing function and since n is finite, there exists a unique integer t^* satisfying $s(t) \leq s(t^*)$ for all t.

Theorem 2. *Assume the above notation. If $R_0 > 1$, then there exists a set $D \subset V$ such that*

$$c_t(D) = red \text{ for all } t > t^*.$$

Moreover $|D| \geq 2s(t^*)$.

Proof. Let W be the set of all i-superspreaders, $i \leq t^*$ and let $D = \cup_{v \in W} S[v]$. We first show that $c_t(D) = $ red for all $t > t^*$. Let $v \in W$. Then $c_i(v) = $ red and $c_i(S(v)) = $ white for some $i \leq t^*$. Now

$$\frac{\eta_i(v, \text{black})}{\deg(v)} \leq \frac{\deg(v) - |S(v)|}{\deg(v)} < \frac{\deg(v) - \deg(v)/2}{\deg(v)} = \frac{1}{2}.$$
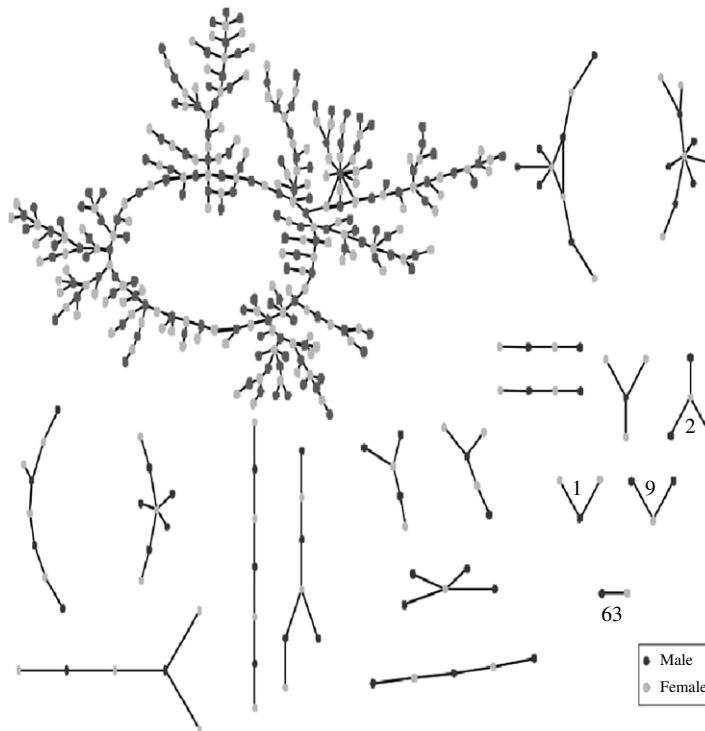
Fig. 3. Sexual Network at Jefferson High [7]; circles denote individual students and links between circles indicate romantic or sexual relationship between the corresponding students.

Therefore, by $G2$, $c_{i+1}(v) = $ red. Since $R_0 > 1$, we have that $\alpha^* < \frac{\Delta - 1}{\Delta}$. If $x \in S(v)$, then

$$\frac{\eta_i(x, \text{red})}{\deg(x)} = |\{v\}| > \frac{\Delta - 1}{\Delta} > \alpha^*.$$

Hence, by $G1$, $c_{i+1}(x) = $ red and so $c_{i+1}(S(v)) = $ red. From the fact that $c_{i+1}(S(v)) = $ red we deduce, as above, that $\frac{\eta_{i+1}(v, \text{black})}{\deg(v)} < \frac{1}{2}$. Therefore, by $G2$, $c_{i+2}(v) = $ red. If $x \in S(v)$, then from the fact that $c_{i+1}(v) = $ red, we have $\eta_{i+1}(x, \text{black}) = 0$. Hence by $G2$, $c_{i+2}(x) = $ red. It follows that $c_{i+2}(S(v)) = $ red. Inductively, $c_j(S[v]) = $ red for all $j \geq i + 1$. This, the fact that $i \leq t^*$, in conjunction with the equality $D = \cup_{v \in W} S[v]$ proves that $c_t(D) = $ red for all $t > t^*$, as claimed.

We now prove the second part of the theorem. First note that if u and v are distinct vertices in W, then $S(u) \cap S(v) = \emptyset$. Thus

$$\begin{aligned}
|D| &= |\cup_{v \in W} S[v]| \\
&= \sum_{v \in W} |S[v]| \\
&> \sum_{v \in W} \left[1 + \frac{\deg(v)}{2} \right] \\
&\geq \sum_{v \in W} 2 = 2s(t^*),
\end{aligned}$$

as desired. □

4. Conclusion

Our contribution has been to provide, for the first time, an analytical discrete dynamic model, for the study of HIV propagation in communities, which is more general and which accurately incorporates local density of HIV positive agents in an individual's sexual partners, network structure, and the influence on opinion change of HIV positive individuals. Moreover, our model has a high ability, through sexual relations (edges), to capture realistic patterns of mixing behaviour, which undoubtedly has a huge impact on the transmission patterns. Our results proved in Theorem 1 show that for a community, if $R_0 < 1$, then the epidemic does not ensue. Thus we have managed to find a single property of the network, Δ, and a threshold value depending on it which informs us when epidemic is prevented.
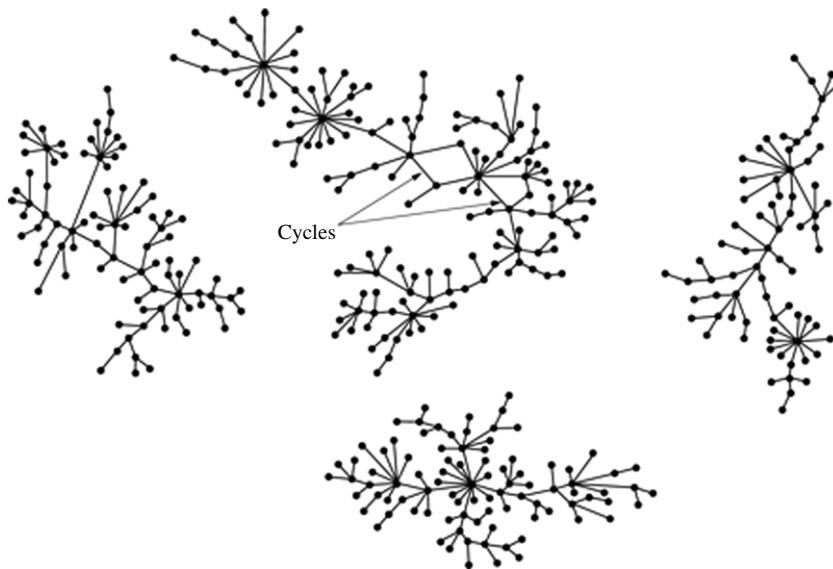
Fig. 4. Graph of the four largest chlamydia components, Colorado Springs [18].

We have identified critical substructures $S[v]$, where v is a "superspreader", in the network and showed in Theorem 2 that the existence of these special substructures ignites an epidemic. Empirical evidence (see, for example Refs. [7,18,19,8]) reveal a high presence of such substructures in sexual networks of communities. Examples of graphs visualising these substructures are presented in Figs. 3 and 4. On one hand Fig. 3 shows the observed romantic and sexual network at Jefferson High linking 573 students. On the other hand Fig. 4 illustrates the results of the study in Ref. [18] where sexual network structure in a community was used as an indicator of a sexually transmitted disease (chlamydia) epidemic phase and graphs were drawn from historical contact tracing data set recorded. In Ref. [19], the first sociocentric study of sexual networks among a general population of Sub-Saharan Africa, a giant sexual network of 1803 young adults in the sample villages was constructed. The network constructed has a high prevalence of these substructures.

Medical anthropologists are of the view that in many communities individuals classify their sexual partners into two categories, namely, the "besties" and the "spares". The observed tendency (see for example Ref. [20]) is that no HIV preventive method, such as condoms, is used during intercourse with besties; preventive methods are only reserved for spares. In our substructure, $S[v]$, where v is a superspreader, the interpretation is simple. Every individual x in $S(v)$ has only one partner v who, by default, is the individual's bestie. It follows that x will not take due care on interaction with v. It is therefore conceivable that the high prevalence of these substructures in empirical networks explain why there have been reports (see, for example Ref. [9]), in some communities, of a low condom use during intercourse. Our results on substructures encourage policy-makers and public healthy campaigners to direct their prevention efforts on described superspreader individuals rather than pursuing any policy based on large-scale random distribution of the available resources. Thus while it is obvious that in the present form our abstract model is still a toy model, it offers an excellent starting point for relevant research in the subject.

Acknowledgments

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