

# Polynomial epidemics and clustering in contact networks

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**It is widely known that the spread of the human immunodeficiency virus was slower than exponential in several populations, even at the very beginning of the epidemic. We show that this implies a significant reduction in the effective reproductive rate of the epidemic, and describe a general mechanism, related to the clustering properties of the disease transmission network, that is capable of explaining this reduction. Our considerations provide what is, to our knowledge, a new angle on polynomial epidemic processes, and may have implications for the choice of strategy against such epidemics.**

**Keywords:** epidemic; polynomial spreading; contact network; clustering

Our purpose in this paper is to discuss polynomial epidemic growth in general terms, drawing attention to a particular aspect which, in our view, has hitherto been neglected. As a guiding example, consider figure 1, which plots the growth of the human immunodeficiency virus (HIV) epidemic in different populations. In several cases one observes polynomial growth (May & Anderson 1987; Colgate *et al.* 1989): over a long time period, the total number of infected individuals at time  $t$ ,  $I(t)$ , is well described by a polynomial function  $\text{const} \times t^n$  for some integer  $n$ . Such a relationship is valid even at early stages of the epidemic, when global saturation effects could not yet play a role and when there was no effective intervention, as in US cities in the early 1980s or in Kenya in the late 1980s.

There are two fundamental parameters governing disease dynamics, which are central to our discussion (Anderson & May 1991; Hethcote 2000). The first is the basic reproductive rate  $R_0$ , the number of new infections that a host would produce in a totally susceptible population. The second is the effective reproductive rate  $R$  (also called the infectee number or replacement number), the average number of actual new infections produced by a host among his contacts during the epidemic process. These quantities satisfy the inequality  $R \leq R_0$ .

The polynomial growth of the HIV/AIDS epidemic cannot be explained by saturation effects (Yorke *et al.* 1978), because it is observed at the earliest stages. The first explanation was proposed by May & Anderson (1988), arguing that heterogeneity in the distribution of contacts signifi-

cantly changes the dynamics of a contact epidemic. Models incorporating extreme heterogeneous mixing lead to sub-exponential epidemic curves, because the highly active classes begin to saturate and new infections come from the slower dissemination of infection to less active individuals. Colgate and colleagues (1989) demonstrated that by making strenuous assumptions about the kind and distribution of the number of contacts, and postulating relatively small and independent subgroups within the population, a polynomial growth rate can indeed be recovered.

Here, we propose an alternative point of view. A simple argument shows that, for an epidemic that is observed to spread polynomially over a long period of time, the value of the effective reproductive rate  $R$  is strongly constrained. Indeed, if  $R > 1 + c$ , where  $c$  is a positive constant, the number of infections after time  $t$  will exceed  $(1 + c)^t$  and the disease spreads exponentially. If  $R < 1 - c$ , then of course the epidemic dies out. Consequently, if the epidemic is observed to spread more slowly than exponentially but without disappearing altogether,  $R$  must approach unity.

We conclude in particular that in all of the polynomial epidemic processes of figure 1, the average number of new HIV infections must approach one per infected host. This surprising and counter-intuitive result is in stark contrast to the estimates of May & Anderson (1987), putting the basic reproductive rate  $R_0$  of HIV well above unity. This raises the problem of finding the mechanisms responsible for the reduction from a high value of  $R_0$  to the effective  $R = 1$  required for polynomial growth. How is it possible that in several different populations, which presumably have different sexual contact structures, every HIV-positive individual on average infects only one new person?

The transmission of a contact disease like HIV/AIDS is constrained by the network of connections along which transmission is possible (Klovdahl 1985; Potterat *et al.* 1999; Lloyd & May 2001). This opens the way for the use of network models to simulate the spread of epidemics (Lloyd & May 2001; Pastor-Satorras & Vespignani 2001). The epidemic curves of figure 1 indicate the existence of networks that give rise to an effective  $R = 1$  and consequently subexponential spreading in epidemic processes without recovery. It is easily shown that regular lattices, as well as random spatially constrained networks with homogeneous spatial distribution, give rise to an effective  $R = 1$  and polynomial spreading curves. However, such models clearly do not constitute reasonable models of human sexual interactions. We know of no random network construction to date that constitutes a reasonable model of human sexual contacts and that gives polynomial epidemic curves. Note especially that no small-world network, such as the preferential attachment scale-free network model of Barabási & Albert (1999), is suitable: such a network necessarily produces exponential spreading in epidemic processes without recovery.

There is an important network characteristic, not emphasized in earlier work, that plays a part in contact epidemic dynamics: the presence of clustering in the transmission network. We propose that network clustering can have a sufficient local effect on epidemic dynamics to quench the reproduction rate from a high  $R_0$  value to a local effective  $R = 1$  throughout the population. The models and arguments of Yorke *et al.* (1978) May &

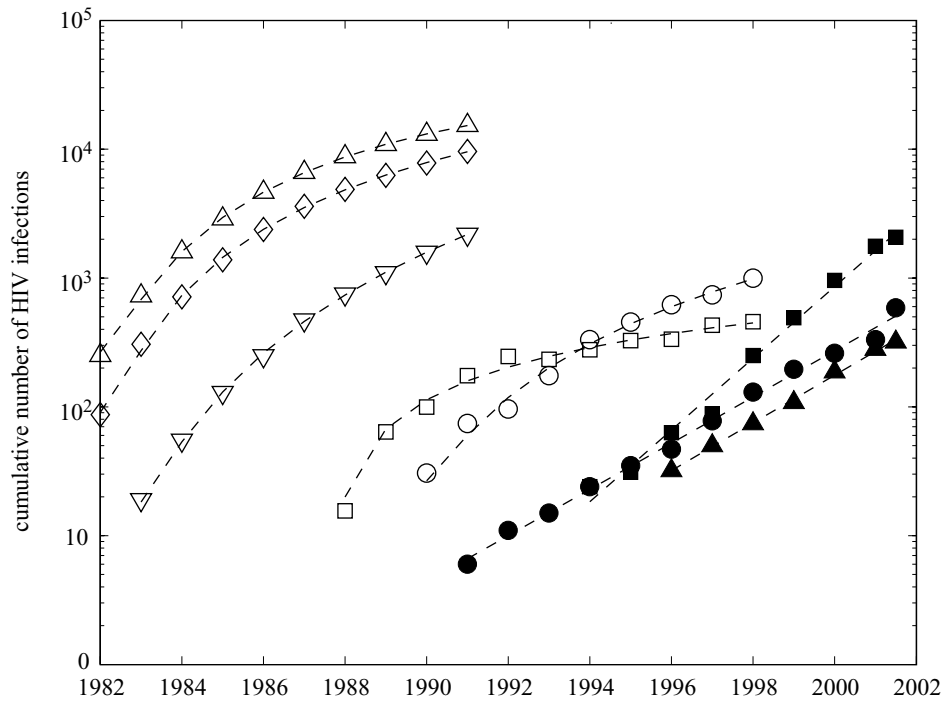


Figure 1. The time development of the HIV epidemic on a semi-logarithmic plot in several populations. In US cities (cumulative total of reported HIV infections among homosexuals (HOM) and heterosexuals (HET)) and sub-Saharan African countries (number of HIV-infected individuals, estimated from the prevalence among women attending antenatal clinics) we observe a sub-exponential growth, shown by the strong curvature in the plot, well fitted with a polynomial curve (open symbols (degree of polynomial in parentheses): upright triangles, New York HOM (3); inverted triangles, New York HET (3); diamonds, San Francisco HOM (3); circles, South Africa  $\times 10^{-4}$  (2); squares, Kenya  $\times 10^{-4}$  (2)). In Eastern European countries (cumulative total of reported HIV infections), the epidemic is exponential, as shown by the straight lines of the plot (filled symbols: triangles, Georgia; squares, Latvia; circles, Lithuania). US data are from Centers for Disease Control and Prevention, South African data are from the Department of Health of the Republic of South Africa and all other data are from UNAIDS/EUROHIV (<http://www.unaids.org>; <http://www.eurohiv.org>) databases.

Anderson (1988), Colgate *et al.* (1989), Lloyd & May (2001) and Pastor-Satorras & Vespignani (2001) all disregard network clustering.

A network is said to be clustered if it contains many more triangles than its other characteristics would imply. In the case of heterosexual networks, which contain no triangles, cycles of length four play the same role. The presence of many triangles can equivalently be formulated as the statement that two random contacts of a node are also connected to each other with high probability. One obtains a quantitative measure of clustering in a network by calculating the local clustering coefficient of a node as the ratio of the number of connections between neighbours of the node and the total possible number of neighbour pairs; the clustering coefficient  $C$  of the whole network is then obtained as the average of the local clustering coefficients over all of the nodes. This measure easily generalizes to cycles of length four in the case of heterosexual networks. Many social and biological networks are known to be clustered, with a value of  $C$  several orders of magnitude higher than random networks of equal size and edge density (Watts & Strogatz 1998), and there are strong indications that human social and sexual networks also share this property (Rothenberg *et al.* 1988).

The important point to note is that, in a clustered network, the contacts of an infected individual do not form a random sample of the population, in contrast to the usual assumption in epidemic modelling. The proportion of infected persons in this sample is much higher than in the

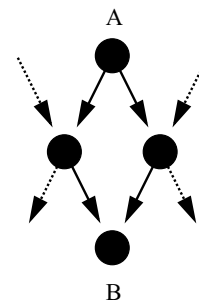


Figure 2. An example of the connection between clustering and multiple infections. Arrows indicate the passage of the infection. In this example, A passed on the infection to two individuals, but most people are multiply infected; B gets the infection from two sources. This local phenomenon reduces a global  $R_0$  value near two to an effective reproductive rate  $R$  that is close to one. Note that, as a result of clustering, the contacts of an infected person do not form a random sample of the population, but are much more likely to be infected than the general population at a given time.

population at large, even early in an epidemic. In other words, the number of non-infected individuals in the immediate neighbourhood of infecting agents is strongly constrained (Keeling *et al.* 1997; Read & Keeling 2003). This leads to a significant reduction in the reproductive rate that is possibly sufficient to turn an  $R_0$  that is well

over unity into an effective rate of  $R = 1$ , which is required by the observed polynomial growth (see figure 2).

The proposal that network clustering plays a role in polynomial epidemics is perhaps testable given sufficient data in a well-documented disease population. Using the links of the known interaction network and transmission routes, it would be possible to compare the competing effects of contact heterogeneity and clustering directly. It would be of particular interest to study risk networks and epidemic spread in sub-Saharan Africa, and we hope that relevant data will become available soon.

If our proposal is correct, the observed time development of a contact disease correlates with the clustering properties of its transmission network. The exponential spread (figure 1) of the HIV epidemic in Eastern Europe is compatible with the fact that its principal transmission mechanism is known to be needle sharing among intravenous drug users (UNAIDS 2000). Indeed, there are indications that needle sharing leads to a network that is significantly less clustered than social or sexual networks (Rothenberg *et al.* 1988). By contrast, the sub-exponential spread of HIV in sub-Saharan Africa requires an infection mechanism that satisfies the constraint that the average number of new infections per host is one. The only mechanism compatible with this is likely to be sexual contact (Walker *et al.* 2003), with a highly clustered transmission network. We predict a high abundance of multiple infections in these populations; therefore, although drug treatment improves the life of HIV-infected individuals, investing in the encouragement of safe-sex practices is likely to be a far better strategy against the epidemic.

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