Network-related Mechanisms May Help Explain Long-term HIV-1 Seroprevalence Levels That Remain High but Do Not Approach Population-Group Saturation

Samuel R. Friedman,1 Benny J. Kottiri,1 Alan Neaigus,1 Richard Curtis,1,2 Sten H. Vermund,3 and Don C. Des Jarlais1,4

In many cities, human immunodeficiency virus (HIV)-1 seroprevalence among drug injectors stabilizes at 30–70% for many years without secondary outbreaks that increase seroprevalence by 15% or more. The authors considered how HIV-1 incidence can remain moderate at seroprevalence levels that would give maximum incidence. Previously suggested answers include behavioral risk reduction and network saturation within high-risk subgroups. Among 767 drug injectors studied in 1991–1993, during a period of stable high seroprevalence in New York City, risk behaviors remained common, and networks were far from saturated. The authors suggest a different network-based mechanism: in stable high-prevalence situations, the relatively small sizes of subnetworks of linked seronegatives (within larger networks containing both infected and uninfected persons) may limit infectious outbreaks. Any primary infection outbreak would probably be limited to members of connected subcomponents of seronegatives, and the largest such subcomponent in the study contained only 18 members (of 415 seronegatives). Research and mathematical modeling should study conditions that may affect the size and stability of subcomponents of seronegatives. Finally, if the existence of small, connected components of seronegatives prevents secondary outbreaks, this protection may weaken, and vulnerability to new outbreaks increase, if HIV-1 seroprevalence falls. Thus, in situations of declining prevalence, prevention programs should be maintained or strengthened. Am J Epidemiol 2000;152:913–22.

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Rapid increases in human immunodeficiency virus (HIV)-1 prevalence have occurred within many populations of injecting drug users (IDUs). These increases have typically been followed by periods of stable seroprevalence. Thus, prevalence may increase from 5 to 30–60 percent within a few years and then remain stable for 10 years or longer. Examples of rapid increases in HIV-1 seroprevalence followed by stability for at least 4 years among drug injectors include Bangkok, Thailand; New York, New York; Amsterdam, the Netherlands; and Madrid and Valencia, Spain (1–3). The period of stable seroprevalence is characterized by moderate rates of HIV-1 incidence, typically 4–6 per 100 person-years at risk among seronegatives (4, 5), that balance the loss of seropositives, usually due to death and disease.

Of particular interest, we have not observed any cases of a second period of increasing seroprevalence that followed a period of stable seroprevalence. Two primary hypotheses have been offered for the stabilization of seroprevalence without later increases: 1) risk reduction among drug injectors, especially a reduction in the numbers of “sharing partners” (5), and 2) saturation of high-risk subgroups with only modest levels of HIV-1 infection among low-risk subgroups (6, 7). While these two hypotheses are not necessarily mutually exclusive, their implications for public health policy are quite different. The behavior-change hypothesis implies that HIV prevention programs can be of critical importance even in the middle of an “outbreak” of HIV transmission among drug injectors, while the saturation hypothesis implies that the outbreak will end even without prevention efforts.

In this study, we used risk network analysis to examine whether network saturation (which is not necessarily what modelers mean by “subgroup saturation”) could explain a continuing stable seroprevalence among IDUs in New York City. Risk networks are patterns of relations involving risk behaviors between and among people. Sociometric risk networks, which describe both direct and indirect linkages
among persons at risk, through which HIV or other similarly transmitted agents can be spread among them, provide a structural model for analyzing the kinds of linkages used to trace contacts (8–21).

We also studied the possibility that risk network patterns, rather than saturation, may serve to contain highly infectious primary HIV infections within a high-seroprevalence population. In primary infection, which can occur an estimated 2 weeks to 4 months after initial infection, a person with HIV-1 may be 50–100 times more infectious than during succeeding years. Primary-infection-driven transmission may be key to epidemic outbreaks of HIV-1, although this possibility has been questioned (22–26).

Specifically, a mechanism that may prevent the new infections that do occur in stable high-prevalence epidemics from creating secondary outbreaks is based on patterns of sociometric linkages between seronegatives and seropositives. In these situations, the subnetworks of linked seronegatives (within larger networks that contain both infected and uninfected persons) may be quite small and thus limit infectious outbreaks. This argument is based in part on the dynamics of primary infection. Persons who have passed the primary infection stage and still have functioning antibody responses generally do not have very high infectivity. Thus, most seropositives in a long-term epidemic should be less-efficient transmitters of HIV-1 to others than newly infected people are during primary infection. It seems reasonable to suggest that the epidemiologic/social space for rapid spread of primary infection is among the uninfected (and the newly infected as transmitters) and, in this context, that long-term seropositives may, unless they engage in extremely high rates of very high risk behavior with uninfected persons, be able to serve as buffers or “firewalls” between a person with primary infection and others who are as yet uninfected (figure 1).

In this study, we used cross-sectional data on New York City drug injectors in 1991–1993, a time when HIV-1 seroprevalence had been stable at around 50 percent for at least 8 years, to examine the distribution of HIV-1 seroprevalence in different connected components (the way in which social network analysts conceive of networks), with particular attention to whether such components are “saturated.” We also developed hypotheses about how sociometric risk network configurations might help explain the lack of large, primary-infection-driven outbreaks in stable high-prevalence epidemics.

MATERIALS AND METHODS

Subjects and data

Drug injectors (n = 767) were recruited through street outreach in areas with heavy drug use and through chain referral by other subjects, as described elsewhere (9, 21, 27). Eligible subjects had to have injected drugs within the prior year. They were interviewed with informed consent from July 1991 to January 1993 in the Bushwick community of Brooklyn, New York. Most (687, or 90 percent) were tested for HIV-1 antibody (replicate enzyme-linked immunosorbent assay with Western blot confirmation) after standard pretest counseling; they were then offered risk-reduction advice, supplies (including bleach and condoms), and referrals to drug abuse treatment and other services. Post-test counseling also offered advice, referrals, and supplies.

Network studies raise complex human subjects issues, which have been discussed at some length (28, 29). All of our procedures were approved by the institutional review boards for the National Development and Research Institutes and the New York State Department of Health. Confidentiality is critical in network studies; it was protected in our study by training and supervising all staff carefully, limiting written records about contacts to first names or street names, locking all records with names in containers separate from other data, and obtaining a federal certificate of confidentiality. (Contacts whose first names or street names were given by subjects as part of their networks did not sign informed consent forms unless they themselves became study participants.)

A face-to-face, structured interview was conducted. The interview gathered data on subjects’ sociodemographic and biographic backgrounds, drug and sexual risk behaviors during the prior 30 days and the prior 2 years, medical history, health beliefs, social roles in the drug scene, and networks.

FIGURE 1. Diagram of the second largest connected component (“network”) in the data gathered in 1991–1993 on human immunodeficiency virus (HIV-1)-seroprevalence levels in New York City. As shown, if person A becomes infected with primary infection, the uninfected members of this component will probably be protected from the high infectivity of A because B, a longer-term infected person whose immune system probably still contains viral infectiousness, will likely buffer (“preempt”) A’s infection from spreading.
Network data

Subjects provided the first names or street names and additional information about as many as 10 persons with whom they had injected drugs or had sex during the prior 30 days. These data included how long they had known each network member, the nature of their relationship, their perceptions of network members’ risk behavior, and their risk behaviors together.

Questionnaire and other data collected during the project were used to define sociometric risk network linkages among research participants. Such linkages were operationalized as follows. First, one or both subjects were required to name the other as someone with whom they had, in the 30 days prior to the interview, injected drugs or had sex. Second, confirmation was required that the named other person was in fact the one we interviewed. A link was considered “confirmed” if two participants 1) engaged in face-to-face contact with research staff at the same time or 2) were observed together in public settings by project ethnographic staff. Links could also be confirmed (9, 27) by matching selected characteristics reported by index subject A (first name and/or street name, age within 5 years, race/ethnicity, and gender) with descriptors of subject A provided by another subject who had nominated him or her.

Analyses and constructed variables

Defining sociometric risk network components. UCINET software (30) was used to detect “connected components” (which are sometimes called “networks”). A connected component is a set of subjects linked to each other directly or indirectly in a chain linkage. As previously presented (21), the component structure was as follows:

1. A large connected component of 230 members.
2. Ninety-one smaller components with a total of 228 members. Sixty-eight of these components were dyads, fourteen had 3 members each, four had 4 members, three had 5 members, one had 7 members, and one (figure 1) had 12 members.
3. Three hundred nine persons who were not linked to other members of the data set, including 183 who nominated other IDUs as persons with whom they had injected drugs or had sex within the previous 30 days but who were not linked to other participants in this project and 126 unlinked IDUs who did not make such nominations.

Additional network analyses. In assessing the degree of saturation in components (networks), we analyzed HIV-1 prevalence within components. In these particular analyses, we treated participants for whom HIV-1 data were missing as if they were not component members; thus, a three-member component with one member who lacked HIV-1 data was analyzed as a two-member component. Our focus here was on whether the distribution of seroprevalences across connected components fit the U-shape (concentrated near 0 percent or 100 percent infected) that a “saturated network” hypothesis implies. It is important to understand that members of these components often were not linked to each other directly but only through chains of one or more other drug injectors in between (figure 1). Thus, a seropositive might have been in the same component as seronegatives but might, for example, only have injected or had sex with another seropositive who, in turn, might have injected or had sex with another seropositive, a seronegative, or perhaps no one else.

We also used UCINET software (30) to detect the component structure of two subsets of our total data set. We examined the subset comprised only of HIV-1-seronegative participants to study sociometrically linked groups who would become at risk for primary-infection-driven outbreaks if one HIV-1-negative member of the component were to become infected. We also examined the (larger) subset comprised of the HIV-1-seronegative participants plus the HIV-1-undetermined participants. (“Undetermined” included 44 subjects who were not tested, 17 whose tests were indeterminate, and 19 with other forms of missing HIV-1-test data). Studying this subset enabled us to examine the possible range of a primary-infection-driven outbreak if all “undetermined” participants were actually uninfected.

Further analyses. The risk behavior of seronegatives during the 30 days prior to the interview was assessed to determine the extent to which uninfected drug injectors remained at behavioral risk for HIV-1. (For this study, shooting gallery use was not considered as a measure of particularly high-risk injecting or an indicator of a type of network-related risk too anonymous and random to be assessed by using network techniques (10, 31); when these data were collected, shooting galleries in the area were not anonymous, large-scale public situations of semirandom mixing. Instead, they were environments in which smaller numbers of people, most of whom knew each other, injected in more socially structured ways.) The duration of network ties was assessed by examining how much time had elapsed since subjects had first injected with current injection partners.

RESULTS

Risk behaviors

Since about 1980, as discussed previously (5, 32–34), a considerable change in risk-reducing behavior has occurred among New York City drug injectors. Nonetheless, according to the data on seronegatives analyzed here, in the last 30 days 36 percent of these subjects had injected with someone else’s syringe, 36 percent had passed a used syringe to another IDU, 14 percent had engaged in receptive back loading (in which drug solution is transferred from somebody else’s syringe to the subject’s), and 36 percent had had unprotected sex with another drug injector.

Link duration

The mean time since linked members first injected together was 42 months; the median time was 18 months. Approximately 75 percent of the linkages had lasted 6 months or more.
Dyads

One way in which networks can be conceptualized is in terms of the linked pairs (dyads) from which they are constructed. Of all dyads in our data set, 21 percent were between seropositives, 24 percent were between seronegatives, 30 percent were serodiscordant, and 25 percent included at least one person whose HIV-1 serostatus was not determined. One hundred thirty-one seronegative drug injectors were directly linked to no seropositives, 63 were directly linked to one seropositive, 37 were directly linked to two seropositives, and 11 were directly linked to three to nine (inclusive) seropositives.

Networks

Components. There was a large component of 230 members; HIV-1 prevalence in this component was 45 percent, which raises the question of why it was so far from network saturation. The next largest component had 12 members.

Were components (networks) saturated? In analyses of components to determine their seroprevalence levels, we found that 31 of 80 components contained no members who were infected, 5 components were 14–34 percent infected, 28 components (including the largest one) were 45–50 percent infected, 6 components were 67–75 percent infected, and 10 components were 100 percent infected. Figures 2–4, respectively, present three frequency diagrams: an “ideal” of what the frequency distribution should look like in a “network saturation” situation in which most components either have no virus present or have had it spread throughout the component, the observed distribution showing the proportion of components at each seroprevalence level, and the observed distribution showing the proportion of subjects in components of a given seroprevalence level. Note that the observed distributions clearly do not even approximate the U-shape that should be anticipated if the network were saturated.

Twenty-eight (57 percent) of the 49 networks that HIV-1 had entered (and which therefore had nonzero seroprevalence) were 45–50 percent infected. Ten of the 49 (20 percent) were fully saturated (that is, 100 percent seropositive), and 6 (12 percent) were 67–75 percent infected. All 10 of the components in which all members were HIV-1 seropositive were dyads with only two members.

Thus, in a city in which HIV-1 prevalence had been stable at about 50 percent for 8 years, only 20 (2.9 percent) of the 687 subjects whose HIV-1 serostatus was determined were in components that were 100 percent saturated, and another 19 (2.8 percent) were in components that were 67–75 percent infected. In total, the 16 components that even approached saturation (that is, in which 66 percent or more of the members were seropositive) included only 33 (12.4 percent) of the 267 seropositive subjects.

Sociometric risk network configurations may help explain the lack of primary-infection-driven outbreaks. In analyses to detect components among the seronegative subjects considered as a subset, we found 59 components; the largest component had 18 members, the next largest had 7 members, 2 had 6 members each, 1 had 5 members, 1 had 4 members, 5 had 3 members each, and 48 had 2 members each. Even if all untested subjects were HIV-1 negative, the largest seronegative subcomponent would have had only 19 members.

Figure 5 shows the largest (19-member) connected component of seronegatives and drug injectors of unknown serostatus together with the 5 HIV-1 seropositives to whom they are linked. These 5 seropositives were in turn linked to 17 other seropositives, 9 seronegatives, and 2 IDUs of unknown serostatus (not shown).

Limitations

Several limitations of this study should be mentioned. As in all studies of “hidden populations,” it was impossible to select a random sample of drug injectors (21, 35). Missing

![Figure 2](https://academic.oup.com/aje/article-abstract/152/10/913/55512)
HIV-1 data created some uncertainty about the true distribution of HIV-1 seroprevalence across components and minor uncertainty about the true size of the largest connected subcomponent of seronegatives. Similarly, uncertainty was created by possible underdetection of subject linkages if subjects underreported the number of persons with whom they had injected, had sex, or otherwise interacted in the last 30 days or if the 30-day period was too short to include some important linkages. Such underreporting of contacts, and our inability to recruit some of the named drug-injecting contacts, might also have affected the representativeness of study participants.

One serious limitation is that data were not available on how long seropositives had been infected or on their viral loads. Such knowledge might have increased our understanding of the extent to which small-scale primary infection outbreaks had recently occurred among the seropositives. Future studies should attempt to collect these data.

Furthermore, in future studies, full analysis would benefit from data on which seropositives are using medications that might have reduced infectivity to near zero (which was not a relevant possibility when our data were collected), on which seropositives are end stage and have increased infectivity, and on possible behavioral differences among these groups (9, 22).

Our data reflect the situation in only one section of one city, which limits their generality even though the New York City HIV-1 epidemic has been the largest among IDUs to date and has served as an epicenter for viral spread (1, 5, 9, 32, 36). Clearly, the network dynamics of “stable high seroprevalence” involve issues of influx due to new people beginning high-risk behaviors and efflux due to people stopping them, perhaps because of death; these dynamics may vary by risk category and by geographic location.

Furthermore, in some localities—such as Ruili, China; Bhamo, Myanmar; and Manipur, India—HIV-1 seropreva-


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The lower infectivity of postprimary infections that maintain low viral loads, have relatively indolent clinical courses, and have presumed lower infectivity. The absence of primary infection outbreaks, even to increased seroprevalence, since they were balanced by uninfected persons beginning to inject drugs and by infected ones dying). Our findings on the lack of network saturation indicate that the absence of a new explosive epidemic was not the result of network saturation.

Sociometric network characteristics, unlike network saturation, may well have helped prevent further epidemic outbreaks in cities with a high prevalence. In particular, the small size of connected subcomponents of seronegatives in this study (only 18 or 19 in the largest instance, as compared with a likely 200 or more early in the epidemic) could limit any primary-infection-driven outbreak. (Here, although the network data were collected by asking whom subjects had injected with or had sex with during the last 30 days, three-quarters of the risk relations had lasted for at least 6 months. Thus, although some changes in networks will occur during any potential primary infection outbreak, we would not expect them to be of sufficient magnitude to change the effective sizes of these connected components of seronegatives to any meaningful degree).

If network sizes did not decrease, a single new infection in a large network early in the epidemic could have precipitated rapid spread of HIV-1 to many of the network’s other 229 members (and their unmeasured contacts). Seronegative IDUs in New York City clearly engaged in enough risky injection behavior to provide starting points for primary-infection-driven epidemics, as indicated by the high seroconversion rate during this period as well as by the risk behavior data presented above. Network saturation is unlikely to prevent further epidemic outbreaks, since most networks in which virus was present were clearly unsaturated. Independent of behavioral risk reduction, “firewalls” of seropositives (not susceptible to primary infection) may limit later HIV spread within large networks to far smaller “islands of seronegatives.”

Consideration should be given to how these ideas about network effects on the spread of infection may be affected by issues related to viral or host-related phenomena. First, infection by variants of different infectiousness might result in higher saturation of some networks and lower saturation of others. There are well-documented examples of HIV-1 infections that maintain low viral loads, have relatively indolent clinical courses, and have presumed lower infectiousness. The nef-deleted mutants transmitted to six
Australian blood transfusion recipients from a single donor illustrate how variable viral genetics can diminish pathogenicity and infectiousness (41). If these less-infectious organisms were to predominate in certain networks of drug users, then failure to saturate the high-risk pool of persons is more understandable.

Although lack of data on primary viral infectivity limited our ability to analyze infectivity directly, the viruses extant in New York City drug injectors early in the epidemic were sufficiently infectious for rapid spread to occur (13 seroconversions per 100 person-years at risk). In addition, when our data were collected, viruses in both this sample and others in the New York City area were sufficiently infectious for approximately 20–25 percent of new injectors to be infected within the first 6 years of their injection experience (36, 42).

Host immune parameters might also influence network saturation with HIV-1; some persons may be inhospitable hosts who are very difficult to infect with HIV-1. This situation is most dramatically noted by the homozygous CCR-5 mutation associated with a much lower than normal risk of HIV infection (43, 44). However, the frequency of this mutation in measured populations to date and, in particular, its rarity among minority populations argue strongly against this phenomenon as a mitigating factor in our study (with 67 percent of 767 subjects of African-American or Hispanic race/ethnicity). An important consideration is that host immunogenetic profiles may diminish the risk of HIV infection and/or progression to acquired immunodeficiency syndrome among persons with certain tissue antigen profiles, including human leukocyte antigens (HLA), transporters associated with antigen processing (TAP), and chemokine receptors. This is the topic of intense investigation and will result in a complex matrix of interacting immunogenetic variables, some of which will differ among various racial/ethnic groups (45–48).

We acknowledge this important area but do not find that partial community-level protection due to diminished immunogenetically determined host risk is incompatible with our principal thesis that the small size of connected subcomponents of the uninfected can prevent large-scale secondary outbreaks of the epidemic. If some uninfected persons are more heavily represented by immunogenetically privileged persons, it adds merely one more protective variable, a biologic one, to our quantitative socioepidemiologic argument. (Diminished immunogenetically determined host risk might, however, be another factor, along with decreased infectivity of postprimary infection and behavioral risk reduction, that helps explain why so many networks were unsaturated. The importance of this factor would be a function of the prevalence of such lowered susceptibility.)

Similarly, even though it appears that a person’s viral load is quite stable during long periods of time after primary infection has subsided (49, 50), there may be fluctuations that do result in higher infectiousness (51) for periods of several days. However, the viral load is much higher (on the order of $10^6/µl$ of plasma) during primary infection than during steady-state primary infection (approximately $10^2/µl$ of plasma) or even during these transient peaks (when it is about $10^2$ to $10^4/µl$ of plasma) over short periods of time (52).

We do not propose that these viral or host-related factors are incompatible with our thesis. Quite the contrary; it may be that all of these factors help protect uninfected persons: network dynamics as outlined in this paper (figure 5), diminished infectiousness from lower-virulence strains or among persons with favorable immunogenetic profiles, immunogenetic protective factors among some uninfected persons, and blunted viral load peaks in already infected persons.

The protective effect of small-sized “islands of seronegatives” (the sociometric risk network structures described in this paper) may be conceptualized as a sociostructural form of herd immunity. The mechanism behind this herd immunity is the small size of subnetworks of uninfected persons who can become infected consequent to a new HIV-1 infection. (At the end of such a minioutbreak within a subnetwork of previously uninfected persons, prevention (53) occurs wherein the persons to whom infected persons transmit the virus are already infected.) Similar to other forms of herd immunity, deaths of seropositives and recruitment of uninfected persons into injection drug use may erode its protective effect. The conditions under which, over time, larger islands of seronegatives would come into existence should be investigated. Similarly, research is needed into the conditions under which this process might create an equilibrium in which a moderately high rate of seroconversions would occur over the long term (perhaps 6 percent per year) rather than a renewed “epidemic outbreak” that lifts seroprevalence levels by 10–13 percent or more per year or some form of long-term cyclic variation in seroprevalence (54).

From a prevention perspective, if the small size of islands of seronegatives helps to restrain the spread of HIV, research is needed to determine what social conditions, police practices, or policy conditions tend to maintain such small sizes and which conditions tend to threaten them. Relatedly, we should determine the conditions under which seronegatives tend to have relatively stable network patterns and those under which they tend to engage in frequent changes in those persons with whom they take risks. It may also be possible to develop ways to detect and work with drug injectors in these “firewalls of seropositives” and “islands of seronegatives” to both further behavioral risk reduction and minimize linkage changes that could pose the risk of reducing the level of overall (or localized) herd immunity.

Research on the issues raised in this paper should involve a combination of empirical studies and mathematical modeling. Data similar to those for New York City should be collected in other stable high-prevalence cities (among drug injectors but also among other transmission categories). If possible, it would be useful to collect sociometric risk network data during a period of initial epidemic breakout and (hopefully) stabilization. Had a network study been in place, this collection might have been feasible in Vancouver, British Columbia, for example, over the period 1994–1997.

Studies of age-mixing patterns in egocentric networks (55, 56) can be used to model infection patterns, and cohort studies of sociometric risk networks—such as those currently under way in Flagstaff, Arizona; Atlanta, Georgia; Washington, DC; and Houston, Texas (57, 58)—can provide
important data about changes in linkages and in sociometric network component structures over time.

Hepatitis B and C viruses are spread in ways somewhat similar to HIV. Only preliminary thoughts about social networks and hepatitis B and C virus transmission seem feasible at this time. Hepatitis C virus leads to persistent infection, usually accompanied by levels of infectiousness that are considerably higher than for HIV during its "latent" stage, in approximately 80 percent of cases. Hepatitis C virus reaches very high (65–95 percent) prevalence levels approaching “saturation” in many IDU populations. Hepatitis B virus is also highly infectious, leading to high (50 percent or more) exposure rates in many IDU communities, but it usually leads to immunity, with only 5–10 percent becoming carriers (59–61). We speculate that hepatitis C virus prevalence levels of components would exhibit the U-shape (or J-shape, since most will be nearly 100 percent infected) typical of saturation. The meaning of this theory for the spread of hepatitis C virus is unclear, since reinfec-
tion, perhaps with different strains, remains possible. For hepatitis B virus, we speculate that such a U-shape would not occur. It is difficult to anticipate whether there might be a “small subnetworks of uninfecteds” effect for the hepatitis B virus. These comments are only preliminary, however. Further empirical studies of social networks and the transmission of blood-borne viruses are clearly needed.

Mathematical modeling can also make a valuable contribution and be used to address alternative hypotheses that we could not study. We anticipate that different forms of models would have different capacities to model these processes. Compartmental models might be expected to have difficulty incorporating explicit models of sociometric structure. This difficulty is certainly reflected by the evident awkwardness in trying to interpret our findings about network saturation as terms that can be incorporated in compartmental models. Nevertheless, this technique might be used to test whether the network distributions of seronegatives in this study differ from what would be derived if the modeled numbers of infections were probabilistically assigned to compartment members in our data set. Discrete-events stochastic simulation models (62–64) can be analyzed post hoc to assess the sociometric network structures created in the simulation process. In such exercises, it will be important to use data from empirical network studies to parameterize the simulation process and also as a post-hoc validation criterion. Such models might also provide guidance on whether other infectious agents, such as hepatitis viruses, are subject to similar network-related mechanisms.

Finally, one public health implication of this analysis should be underlined. Prevention of epidemic outbreaks by the small size of connected components of seronegatives is a form of herd immunity. While seroprevalence is high, it helps to limit the number of new infections that occur. This protection weakens greatly as HIV seroprevalence falls, however. In New York City, seroprevalence among drug injectors fell from about 50 percent in the 1980s to approximately 30 percent in the late 1990s, and it appears that it may continue to fall (36). Among White drug injectors, sero-
prevalence fell to approximately 15 percent. In such situa-
tions, the protective effects discussed in this paper are weak-
ened. We expect the size of connected components of seronegatives to begin to increase. Ultimately, these compo-
ments may again become very large and thus capable of transmitting primary-infection-driven outbreaks to large numbers of drug injectors in a very short time. In public health terms, then, situations of declining prevalence are also likely to be situations in which vulnerability to new outbreaks is increasing. Thus, it is imperative that prevention programs be maintained, and perhaps even strengthened, as the proportion of those infected declines.

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