ORIGINAL ARTICLE

Network structural dynamics and infectious disease propagation

J J Potterat BA¹, R B Rothenberg MD MPH² and S Q Muth BA¹

¹El Paso County Department of Health and Environment, Colorado Springs and
²Department of Family and Preventive Medicine, Emory University School of Medicine, Atlanta, USA

Summary: We aimed to relate dynamic changes in risk-network (sex and/or injecting drug) structure to observe STD/HIV transmission. We analysed macro- and micro-structural elements in 2 heterosexual networks, augmented by ethnographic observations. In a Colorado cohort of injecting drug users (n=595), measures of subgroup formation and of density of activity show decrease of network cohesion over time; only one HIV transmission was observed in 3 years. In a group of adolescent heterosexuals in Georgia (n=99), the reverse process (increase in structural cohesion) was associated with efficient syphilis transmission: 10 cases were observed. Changes in personal risk behaviours over time were modest.

STD/HIV transmission patterns were associated with intensification or diminution of network cohesion. Network and ethnographic data suggest that enhanced connectivity facilitates transmission while segmentation impedes it, suggesting opportunities for interventions. These data also emphasize the need to re-evaluate purely behavioural explanations of STD/HIV transmission.

Keywords: STD/HIV transmission, social networks, infectious disease transmission

INTRODUCTION

The social organization of risk behaviours influences patterns of infectious disease propagation¹,². One framework for understanding social organization is network analysis, the study of how people connect in social structures and of its implications. Such analyses rely on applications of graph theory³. In a network, individuals (or groups) are represented as points on a graph, and their relations by lines between these points. The analytic aim is to describe network structure and to explain the effect of structure on variables. The basic proposition is that 'the structure of a network has consequences for its individual members and for the network as a whole over and above effects of characteristics and behaviour of the individuals involved'².

Since the mid-1980s, several investigators have applied social network concepts to elucidate patterns of sexually transmissible disease (STD) and human immunodeficiency virus (HIV) transmission. Auerbach and colleagues published a network-based analysis of 44 homosexual men who were afflicted with the newly emerging syndrome of immunodeficiency; depiction of these patients’ connections provided a symbolic merging of network analysis and STD/HIV epidemiology⁴. Grimson and Darrow, in a more mathematical approach of the same data, demonstrated that the probability of such connections in the absence of an infectious agent was vanishingly small⁵. Klovdahl provided a more formal network analysis of the same group, demonstrating the connection in time and space among distinct, yet connected, groups². Thus, social network analysis provided persuasive support for the hypothesis that AIDS was caused by an infectious, transmissible agent. A few years later, Haraldsdottir and her colleagues analysed sexual network patterns of HIV-infected homosexual men in a relatively isolated community to develop a sense for the future of the epidemic in Iceland⁶. Starting in the late 1980s, several researchers sought empiric network data from populations at elevated risk for HIV (heterosexual injecting drug users) in Colorado⁷–¹⁰, New York¹¹, and Arizona¹².

These studies not only clearly suggested that network structural elements influence HIV epidemic patterns, but also served to attract mathematical modellers. By the early 1990s, a few researchers began to model network structural properties and to evaluate their implications for HIV transmission. Such models have provided important insights concerning the influence of network structure itself²; of predictors of disease

Correspondence to: John J Potterat, 301 South Union Blvd, Colorado Springs, CO 80910, USA. E-mail: smuth@rmi.net
establishment in a community; of determinants of disease prevalence; of the crucial role of concurrent sexual partnerships and of the critical issues surrounding network sampling and power calculations.

To date, empiric investigators have focused primarily on static rather than dynamic network analyses. Were dynamic structural changes to be empirically associated with facilitating or inhibiting STD/HIV transmission, the case for structural influence on patterns of infectious disease propagation would be strengthened.

In this communication, we summarize our exploration of structural changes over time in 2 networks of heterosexuals at elevated risk for STD/HIV. Specifically, we show that disease transmission is associated with intensification, and lack of transmission with diminution, of network cohesion. We focus on the conceptual; technical details of the empirical data are described elsewhere.

METHODS

Study populations and network structural analysis

Population I was studied prospectively. In brief, between 1988 and 1992 in Colorado Springs, Colorado, we continuously recruited heterosexuals (n=595) at risk for HIV to study their social networks and to track HIV transmission. Each respondent was asked to provide a sample of blood and to specifically nominate their social, sexual, illicit drug, and injecting drug partners (for the 6 months preceding interview) and to divulge these partners’ relationships to each other. Interviews and blood samples were to be repeated at yearly intervals. A total of 17 (3%) respondents tested positive for antibody to HIV at baseline; only one new case of HIV was observed prospectively.

In Population II we retrospectively analysed, starting in the Spring of 1996, a focal syphilis outbreak (n=99) in a suburban adolescent population near Atlanta, Georgia. Connections between actors were reconstructed using contact interviewing/tracing data, and studied with the tools of social network analysis. Ten cases of infectious syphilis were recorded during this outbreak. Connections between people can be viewed graph-theoretically at the macro- or micro-structural level. At the macro level are components—regions of social networks where every person is ultimately (directly or indirectly) connected to everyone else. At the micro level are measures of subgroup formation. These latter have simple visual analogues (Figure 1) and can be lower- or higher-order microstructures, depending on the complexity of their connectivity.

Densely connected structures are both intuitively and stochastically associated with increased probability of STD/HIV transmission. Thus, for example, a preponderance of higher-order microstructures in a given network is postulated to be predictive of transmission.

RESULTS

Structural properties and STD/HIV transmission

In Population I, the number of connected components (groups of persons in which there is a path of some length between all members) increased during the period of observation, indicating increasing segmentation of the overall network. In addition, average component size decreased, thereby reducing opportunities for sustaining transmission. The most striking macro-structural finding concerned the location of HIV-infected persons within the network. The static picture—obtained by collapsing all the network connections over time—revealed one very large component followed by many small components. HIV cases tended to reside in small components with no demonstrable links to the large one; the few HIV positives in the large component tended to occupy peripheral positions (i.e. had low network prominence scores).

The most important micro-structural finding was that there were virtually no higher order microstructures at inception of the study (meaning that there were no small groups of, say, 4 people within which each person was related by needle-sharing or sexual activity to 2 of the others). This structurally anaemic transmission environment was rendered even more so over time. Virtually all measures of small subgroup formation, and of the density of risk activity within such small subgroups, indicated diminution of network cohesion. For example, the number of so-called 2-cliques (groups of at least 3 people who are connected to each other by 2 or fewer steps) diminished appreciably during the study period.

During the same period of observation, there were some declines in risk taking—particularly in the sharing of needles—but sexual risks and the use of personal prophylaxis (e.g. condom use) did not change dramatically. These modest declines would not, by themselves, predict the failure to
generate endemic or epidemic propagation of disease that we observed.

In contrast, the macrostructure of Population II—the group of adolescents involved in the transmission of syphilis—exhibited increasing cohesion over time. For example, the number of components decreased during a period of 18 months, from about a dozen small ones to one large connected component of 95 people and one small component containing 4 people. Importantly, micro-structural analysis revealed a close relationship between the temporal distribution of diagnosed syphilis cases (n=10) and the increasing complexity of the network’s microstructures. Ethnographic observations revealed little change in basic risk behaviour patterns. Abatement of this syphilis outbreak was seemingly related to network fragmentation—typified by a reduction in group sexual activity—that may have been a consequence of more mature relationship formation or a realization that vulnerability to syphilis implies vulnerability to HIV, rather than to basic behaviour change. The latter hypothesis is supported by the observation that nearly three-quarters of young women at greatest risk became pregnant, most of them after syphilis was controlled within the group.

DISCUSSION

Current thinking about HIV transmission is usually based on observations about personal risk behaviours and on the insights of deterministic models using ‘compartment’ approaches to group interaction. Starting in the late 1980s, workers began to model network structure and to seek network data empirically to study HIV transmission dynamics. The work presented in this essay is a product of this endeavour.

Our data from 2 distinct networks of persons at risk for STD/HIV clearly support theoretical considerations that postulate network structure as a determinant of disease propagation. In our prospective study of injecting drug users and their partners in Colorado, network structure was not only shown to be weakly cohesive at baseline but to exhibit decrease in cohesiveness during the 3 years of observation. Prospectively, only one case of HIV transmission (via injecting drug use) was observed. In contrast, in our retrospective study of a focal syphilis outbreak in Georgia, increase in network structural cohesiveness was associated with relatively efficient syphilis transmission: all 10 cases were diagnosed during the period of increasing network cohesion.

Both of the transmission outcomes we observed were, in effect, predicted by the newer generation of stochastic models that take network structure into account. These models emphasize the crucial role played by degree of network interconnectedness as a predictor of disease establishment and prevalence. Importantly, documentation that neither lack of HIV transmission (Colorado) nor abatement of syphilis transmission (Georgia) were associated with substantial changes in reported risk behaviours supports the need to re-evaluate purely behavioural explanations of STD/HIV transmission.

In a recent study of selected network structural features of injecting drug users in New York City, Friedman and colleagues conclude that an important structural feature (network location) is an independent predictor of HIV status; our studies lend support to their tentative interpretation.

Although evidence to support this new paradigm in infectious disease epidemiology is steadily accumulating, caution in data interpretation is warranted. Empiric network studies suffer from 2 important shortcomings: uncertainties about sampling validity and error estimation and from lacunae in data collection. Until these issues are resolved our results must be viewed with appropriate circumspection; importantly, they should stimulate research in this promising line of inquiry. In the meanwhile, network analysis may be viewed as complementary to other infectious disease propagation paradigms.

Thus far, the network approach has been useful mainly as a tool for epidemiological assessment (‘Is this where the action really is?’) and evaluation (‘Is what we are doing effective?’). Individual-based interventions derived from the insights of network-informed studies currently await implementation. Theoretically, one can imagine practical ways to counsel at-risk clients (showing diagrams, for example, of a potentially nefarious network context using the spider’s web analogy). Morris and Kretzschmar suggest that, to discourage the structural cohesiveness that is a consequence of simultaneous sexual partnerships (‘concurrency’), ‘messages promoting one partner at a time may be as important as messages promoting fewer partners’. Indeed, such messages may be more behaviourally acceptable for at-risk persons than messages promoting reduction in number of partners or employing condom use. Friedman and colleagues speculate that ‘determining how components . . . can become centres for promulgating and enforcing norms and values that support risk . . . reduction’ may be a workable intervention. Elsewhere we suggest that ‘segmentation of social networks may be an important method of interrupting transmission. In practical terms, closing of bathhouses and shooting galleries may represent such segmentation’. Finally, micro-computers and appropriate software provide opportunities for analysing even partial network data to presumably identify persons of positional importance and to thus apply intervention energies focally.

In our samples, STD/HIV transmission or its impedance was associated with structural properties of risk networks. This observation suggests that just as language can be conceptualized as a flow of words structured by rules of grammar, so may
epidemics be viewed as a flow of microbes structured by the ‘grammar’ of social network structures.

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