



Published in final edited form as:

Am J Public Health. 2013 February ; 103(2): 322–329. doi:10.2105/AJPH.2012.300908.

Sex, Drugs and Race: How Behaviors Differentially Contribute to STI-Risk Network Structure*

Jimi Adams,
Arizona State University

James Moody, and
Duke University

Martina Morris
University of Washington

Abstract

Objectives—To examine how risk behaviors differentially connect a population at “high risk” for sexually transmitted infections.

Methods—Starting from observed networks representing the “full” risk network and the risk network among respondents only, we construct a series of edge-deleted counterfactual networks that selectively remove sex ties, drug ties and ties involving both sex and drugs and a comparison random set. With these edge-deleted networks we demonstrate how each tie-type differentially contributes to the connectivity of the observed networks on a series of standard network connectivity measures (component and bicomponent size, distance and transitivity ratio), and the observed network racial segregation.

Results—We find that sex ties are unique from the other tie-types in the network, providing wider reach in the network, in relatively non-redundant ways. Additionally, in this population, sex ties are more likely to bridge across races than are other tie types.

Conclusions—Interventions based only on one mode of transmission at a time (e.g., condom promotion *or* needle exchange) would have different potential for curtailing STI spread through the population than would attempts that simultaneously address risk-relevant behaviors.

Introduction

While relationships involving sex or needle-sharing contacts are both salient for the transmission of a sexually transmitted infection (STI), from one person to another (1–4), research has demonstrated that these different types of risky contacts provide different probabilities for STI transmission (5–8). New modeling techniques have demonstrated how contact type and partnership networks combine to determine the dynamics of infection transmission through networks (9). Framed in a classical susceptible-exposed-infected-recovered (SEIR) framework, the conclusion that needle-sharing ties tend to provide greater risk than sexual ties (5–8, 10) has stemmed from research largely addressing one of two questions. First, given a population of uninfected individuals, how do differences in risky

*This work was supported in part by the National Institutes of Health (grants DA 12831, HD 41877, and HD68317-2). We thank Steven Q. Muth and John Potterat for their help in interpreting these data, especially the racial dynamics of the population. Richard Rothenberg, David Schaefer, the Networks and Health Working Group at Columbia University, and the Structural Dynamics Working Group at Arizona State University provided helpful comments on previous versions of this article. Note. The views in this article reflect those of the authors and do not necessarily represent those of the National Institutes of Health.

Please direct correspondence to jimmi adams (jadams@american.edu).

behaviors lead to differences in subsequent STIs (i.e., focus on the transition between S-I)? Second, given sexual or needle-sharing contact between sero-discordant individuals, what is the differential likelihood of infection depending on type of contact (i.e., focus on the transition between E-I)? We know comparably less about whether and how sex ties and needle-sharing ties may differentially contribute to the observed connectivity across a full risk network (i.e., focus on the potential transition between S-E).

Networks and STIs

While it is well known that both unprotected sexual contact and needle sharing contact can lead to infection spread, we know little about the role each type of tie plays in connecting a wider population. The potential breadth of an STI epidemic rests on two network-related issues, which can either promote or constrain transmission across a population. First, given ties between infected and susceptible individuals, the probability of infection varies by type of risk contact. For example, the probability of an individual being infected with HIV in a single contact varies according to type of sexual contact and is considerably higher for needle-sharing or other “sharps” contact than for any single sex act (5, 6, 10, 11). This network-related aspect directly aligns with the existing research regarding the E-I transition described above (6, 8, 11–14). Such work provides important explanations of observed transmission dynamics and is at the core of discussions regarding varying epidemic trajectories in different parts of the world (11–13).

Second, the levels of connection between infected individuals and the wider population alters the course of an epidemic, whether via direct ties (those linking partners) or indirect ties (those linking individuals to their partners’ partners and their partners’ partners’ partners, etc.). Network reach identifies how many people are linked together through direct and indirect paths – and thus how wide an infection could potentially spread through a population. Network redundancy identifies the number and pattern of links within that population, which influences the likelihood that infections will actually spread through the population. Previous research demonstrates how reach, redundancy and other network characteristics shape the potential spread of an infection through a population (15) and how common such patterns are in observed networks (16) or epidemics (4). In practice, research taking this approach focuses on how readily such measures account for population-level transitions between S-I.

Here we focus on a third question that has not been readily addressed previously – the link between S-E. We examine whether and how sex and drug ties serve to differentially provide STI-exposure potential for uninfected individuals. While combining each of the above approaches in a single study would allow partial estimation of this effect, we demonstrate that our direct attention to this specific question provides new insights about population STI-risk not available through any of the previous approaches. We discuss the implications of these differences for future STI research and intervention efforts.

Race, Networks and STIs

In the United States, research consistently observes that African Americans have substantially higher rates of STIs than whites (17–25). Potential explanations of the sources of these differences have ranged over empirical observations of African Americans having more partners (19), higher rates of partnership concurrency (i.e., sex with more than one partner within a given time period) (26), and different mixing patterns – that include bridging risk groups (e.g., linking network cores to non-core individuals) (27), or bridging population groupings that are not directly risk-relevant (e.g., race, geography, etc.) (24). While each of these differences provides a partial explanation for racial disparities in STI prevalence, the differences remain robust despite controls for these factors (19, 21, 22).

Thus, we examine whether the remaining unaccounted racial difference in STIs partially stems from the differential network connectivity different types of risk relationships provide.

We address these questions using Colorado Springs, Project 90 data (28–31), to examine how sex ties, needle-sharing ties, and ties involving both sex and needles differentially connect a high-risk population. By selectively removing types of ties from the contact network, we can evaluate the relative importance of each tie type for various measures of network connectivity. In the discussion we elaborate how these would alter the potential spread of an STI through a population.

Data and Methods

Data

Our data come from the Colorado Springs “Project 90” study, which was a CDC-funded project focused on HIV transmission in heterosexual and injecting drug user (IDU) populations. Data were collected from 595 respondents using face-to-face interviews between 1988–92, using an open cohort design. The data collection focused on eliciting characteristics of risk-partnership networks that allowed the researchers to identify and interview as many people in the target population as possible (IDU, prostitutes, and their sex partners), and to assess the size, structure and epidemic potential of the high-risk partnership network. Detailed overviews of the study and sample design have been published previously (28–30, 32) and the data have been used to examine the impact of network structure on disease transmission (3, 4, 30, 33).

Analytic Strategy

We construct two observed networks: each represents ties involving sexual contact, shared drugs, and ties involving *both* sexual and drug-sharing contact. The “Respondent-Only” network consists of the 595 respondents and the 1296 reported connections among them (see Figure 1, Panel 2). The respondent-only network is extracted from the larger “Full” network, which additionally includes all ties respondents reported having with other individuals (“alters”) who were not study participants.¹ The full network consists of 6,019 individuals (595 respondents and 5,424 non-respondents) and the 13,901 reported ties among them (see Figure 1, Panel 1). Table 1 presents descriptive statistics for these respondents and their named alters.

We examine how sex ties, drug ties, and ties involving both differentially contribute to the observed connectivity of the network separately for the respondent-only and full networks. First, we compute the connectivity measures described below. We develop a strategy for assessing how each tie type contributes to these network connectivity measures by selectively removing them from the observed networks then quantifying their changes in the edge-removed networks. This “edge removal” process starts with the observed networks, then randomly selects n ties of type k , 500 times for each combination of settings. The results described below show n in increments of two percent, ranging from two to twelve percent of all ties in the full network and up to 14 percent of all ties in the respondent-only network.² To compare the effects of different tie types on connectivity, k includes 4 different types of ties. The first three are those of analytical interest - sex ties, drug ties and ties that include both sex and drugs. A baseline for comparison involves edge-removals

¹In addition to reporting their own ties to these alters, respondents could additionally report on the ties among their alters (called “matrix ties”), and ties those alters had with up to one other associate who was not among the respondent’s contacts (called “associate ties”). The analyses conducted here include *all* of these additional (matrix and associate) tie nominations (for discussion see Adams & Moody 2007).

where ties are selected completely at random (i.e., indifferent as to whether it involves sex, drugs or both sex and drugs ties) following the same process described above.

Measures

Network Connectivity—To capture the extent of network connectivity, we compute a series of measures on the observed and edge-removed networks. A “component” is a set of persons connected by a path of any length (i.e., a direct *or* indirect path containing any number of intermediaries). Components can be thought of as capturing the widest potential diffusion of a single STI epidemic. We compute the *size of the largest component* as the count of the number of persons linked together within the largest component. Most large networks contain a “giant component” (35) comprising over half of all network members connected through a chain of relations, and this is true of both observed respondent and full networks here. Table 2 presents the base-level statistics for each of the network-based measures from the observed networks separately for the “Respondent-only” (Column 1) and the “Full” networks (Column 2).

Network components are considered fragile if single nodes or edges are responsible for connecting different portions of the network. Measures of network robustness capture portions of the networks that have greater than minimal connectivity (i.e., comprise a component). A bicomponent identifies subgroups within the largest component where every person is connected by at least *two* completely independent paths (15, 36). In terms of risk-contact networks, bi-components are sub-sections of the network where the likelihood of transmission is elevated by the potential for pathogens to follow distinct routes between pairs of nodes. Because the bicomponent is a subset of the largest component, to avoid documenting the same contributions twice as we could if we simply computed the size of the largest bicomponent, we measure the *relative size of the largest bicomponent*, which represents the proportion of nodes within the largest component that is also bi-connected.

Many observed connected networks display relatively short distances between any two randomly selected nodes (37). The geodesic distance between nodes refers to the number of ties on the shortest path between them. In terms of epidemic potential, diseases spread much more efficiently across shorter distances. Thus, we measure the *relative distance* as a ratio of the average geodesic distance between all observed pairs of nodes in the connected component to the maximum potential length of that path.³

The *transitivity ratio* identifies the proportion of *i-k* pairs for which a tie exists given the presence of ties between *i-j* and *j-k* (38). A higher transitivity ratio indicates greater clustering of a network, which can be thought of as the amount of “recursion” in the networks. This would indicate networks that are more locally robust and have a smaller span than networks with lower transitivity and similar density. In terms of epidemic-potential this can be thought of as indicating the greater likelihood of successful transmission over short distances, at the expense of efficient transmission over longer distances.

Racial/Ethnic Segregation—The final characteristic of the networks that we examine is the level of observed *racial/ethnic segregation*. For this, we use Freeman’s (39) segregation index, which captures how much an observed network differs from random mixing. A value

²We stop at twelve percent for the full network because when removing that proportion of all ties as ties including both sex and drugs, all “both” ties have been removed. Similarly, for the respondent-only network, beyond 14 percent, all sex ties have been removed. We extended comparisons of other remaining ties beyond these cutoffs, but patterns do not change appreciably. We therefore present only the range for which we can compare the contributions of all three tie types. The first author can provide versions of figures with complete comparisons on request.

³This measure is necessarily limited to those who are connected (i.e., are part of a single component). The longest a path could be between a pair of nodes in the connected component is the size of the connected component minus 1.

of 0 would mean that ties within and between categories are distributed at random; a value of +1 would indicate perfectly segregated networks (all ties within category), while negative values indicate greater-than-random cross-group ties – as would be seen in heterogamous features. Here, the index identifies the extent of cross race ties compared to what would be expected if ties were formed at random with respect to race, accounting for the racial distribution of the population.⁴

Results

We present the analyses for the observed and edge-deleted networks first for the full network. Then, for the respondent-only network, we limit our discussion to how those findings differ from findings for the full network.

Full Network

Connectivity Measures—Panel A of Figure 2 presents line-graphs for how the edge-deletions of the various tie types differentially influenced network connectivity for the full network.⁵ The observed network has a giant component that connects approximately 70 percent of the population who have any ties involving sex and/or drugs. At each level of edge-removal, sex ties reduces the size of the connected component more severely than any other tie type, with drug tie removals diminishing the giant component size at a lesser rate than does removing ties at random. Approximately 40 percent of the individuals in the observed connected component are connected via more than one pathway (i.e., are part of the largest observed bicomponent). Removing sex ties *increases* the proportion of ties within the connected component that are in the biconnected core, while drug tie removals *decrease* the proportion who are robustly connected in this way. Conceptually, this means that, drug ties are more likely to provide additional redundant indirect pathways among those in the connected component, while sex ties do not provide this same network robustness. With respect to the average distance, we find that removing sex ties *increases* the distance between nodes. Drug tie removals also *increase* the relative average distance observed in the graph, but at a rate lower than removing random ties. With respect to transitivity in the network, among all instances where two people share a common alter, approximately 25 percent of those pairs are also directly tied to one another. As ties are removed from the network, sex tie removals *increase* the levels of transitivity in the network. Drug-tie and both-tie removals each *decrease* the observed transitivity ratio, but at rates greater and less than random tie removals, respectively.

While each of these individual patterns describe important aspects of sex and drug tie contributions to network connectivity in this population, the full contribution of these analyses only come through their combination. In general, the combined findings suggest that the most dramatic effects on connectivity are due to sex ties. Specifically, sex ties spread the network to the widest population, but they do so with connectivity that is somewhat fragile: they do not involve the same level of recursion that drug ties do – whether at the local level (via transitivity)⁶ or at the level of longer indirect pathways (i.e.,

⁴Respondents were able to separately report their race and ethnicity. In this population however, virtually all respondents who identified as Hispanic also identified as White. As such, for this population, race/ethnicity were not identifiably independent dimensions (hence the four categories reported in Table 1). More important for the current examination, while the segregation index used is computed using all four race/ethnicity categories identified, race (and not ethnicity) is the dominant characteristic on which segregation of ties is observed. As such, while both are used in the construction of the measure, we focus the discussion on race, which was the salient driver of relational segregation we observe and attempt to explain. We thank Steve Muth and John Potterat for their assistance in clarifying this interpretation.

⁵Please note, baseline values for each measure (i.e., the value observed with 0% of ties removed) are found in Table 2. While we do not discuss the significance of the differences described – all differences that can be seen in the presented graphs are significantly different ($p < 0.01$). Versions of these graphs with confidence intervals are available from the first author on request.

bicomponent connectivity). This can be interpreted as thinking of sex ties as producing “tendrils” that reach out into the wider population, but provide (comparatively) fewer re-connections to the strongest core(s) of the network.

Racial/Ethnic Segregation—Figure 3 presents the network racial/ethnic segregation for respective levels of edge-deletion. Sex ties also uniquely contribute to observed racial/ethnic segregation in the network. Sex-tie edge-removals *increase* the level of segregation, while drug tie removals *decrease* the level of racial segregation in the network.⁷ This suggests that in the full network, sex ties more frequently serve as a bridge across races in this population, while drug ties appear to serve to robustly connect populations of the same race.⁸ Overall the level of racial segregation in the observed network is moderate (~0.41).

Respondent-Only Network

Connectivity Measures—More of the respondent-only network is contained within the giant connected component, has more of that component also part of the bicomponent, has nodes that are separated by comparatively shorter distances and are more likely to exhibit transitivity, compared to the full network (see Table 2). Panel B of Figure 2 shows the edge-removal effects on each of the network connectivity measures for the respondent-only network.

Within the respondent-only network, edge-removals largely affect connectivity measures in the same pattern as was observed for the full network. Sex ties occupy unique positions in the network, and appear to add “tendrils” like additional, sparse connectivity to the network. In the full network (panel A) drug ties played the primary counter-role to sex ties (e.g., their removal led to declines in measured transitivity whereas sex tie removals increased transitivity), while ties involving both sex and drugs had little appreciably different effect compared to removing ties at random. For the respondent-only network (panel B), this counter role is more consistently filled by ties that include both sex and drugs (e.g., they serve the role of providing the redundant ties within the network), while it is drug-only ties that do not appreciably differ from removing ties at random.

Racial/Ethnic Segregation—Edge-removals in the respondent-only network reveal the same pattern described above for the full network – sex ties are substantially more likely to form bridges across racial groups, and drug ties are more likely to be contained within race. Overall, the level of racial/ethnic segregation in the respondent-only network is substantially lower (~0.23) than in the full network.

Discussion

In this study, we found that sex ties were key to network expansiveness, but that this expansiveness is fragile: sex ties bring in more of the population, but the people reached through sex-only ties tend not to be multiply linked to the core network. Moreover, sex ties are key to bridging across race. This pattern is true both for the directly-observed

⁶It is important to point out that in a network comprised exclusively of heterosexual sex ties, transitivity is impossible. From a “disease-eye” view however, ties can combine in any pattern, and thus once combined with drug ties among members of the network, sex ties are no longer precluded from providing this form of local robustness in the network. Our analyses examine how each tie type differentially contributes to the overall connectivity patterns in the *combined* network.

⁷Edge removals of ties involving both sex and drugs do not differ substantially from removing random ties, each having virtually no effect on racial segregation in counterfactual networks.

⁸There are several potential ways that this observed pattern could arise, in particular potential racial differences in participation in commercial sex work among the population. It is also possible that geographic constraints contribute to observed patterns of racial mixing (40).

respondent-only network and the larger, more racially segregated full network they report on.

These findings have potentially important implications for how we understand STI-spread through a population. They suggest that interventions focused only on one mode of transmission at a time (e.g., condom promotion *or* needle exchange programs) would have different potential for curtailing STI-spread. For example, interventions based on condom promotion alone might reduce the breadth of a potential epidemic in the full population, while needle-exchange programs might reduce the likelihood of STI growth within the core of the network. Since epidemic potential turns on transmissibility, these results suggest that interventions aimed at a highly-infectious STI would do best to focus on the broad-but-weak reaching sex ties; while interventions focusing on hard-to-transmit STIs might best be targeted at the redundancies built into drug exchange networks. Ultimately, of course, intervention efforts focused on both would be necessary, because it is clear that neither of these risk behaviors is sufficiently uniquely positioned in the network that they alone explain network epidemic potential.

Our findings also have important implications for interpreting STI risk in the Project 90 context. For this population, we learned that - compared to other ties involving risky behavior - sex ties provide unique connectivity patterns. In particular, their “bridging” characteristics seem much more akin to the reach provided by “weak” social ties (41), an effect that has been observed also among high school romantic relationships (42). This pattern is likely the result of the unique social configurations of drug and sex behavior. I.e., the drug ties have more “strong tie” characteristics, with higher density among partners’ partners (41) – perhaps through trust developed from co-participating in illegal behavior, or through shared relationships that provide robust access to drug supply in the event of a single node’s removal (e.g., through arrest). What is less clear is how what we learned from this specific sample can be translated to other contexts. First, for the risk-population at large in Colorado Springs, we learned an exclusive focus on only the “at-risk” population would have led to different conclusions than when including their named alters. In particular, the racial composition of ties to the wider sample was more diverse than that within the high-risk set alone, leading to greater cross-race epidemic potential than might be assumed from the respondents only. Most generally, this work suggests that prediction of effects from targeted intervention attempts rooted on existing network-based approaches (e.g., reducing bridging ties (17)) may be misestimated if generated from a core sample that might be unrepresentative of the wider at-risk population. As with all case studies, we need to further examine how these differential risk contributions may be different outside the Colorado Springs context.

In general, the network foundations of public health research will likely benefit by taking seriously the multiplex nature of disease-carrying ties. The effect of risk behaviors on corresponding network patterns, and ultimately on epidemic potential, could not have been captured if the study focused on any of these ties to the exclusion of others. While sex ties play a particularly unique role in connecting members of this population, they do so in a way that is fundamentally intertwined with the unique, and in some ways counter-balancing - patterns contributed by drug ties (in the full network), and ties involving both sex and drugs (in the respondent-only network). The biggest-picture implication of this work, then, is that future public health network research should fully explore the multiple ways people are connected. Rather than simply a connection of “pipes” that carry disease, network ties likely unfold and evolve in characteristic ways. Here, we see the trace of that character in the structural location of types of ties, but we might similarly find differential behavior based on the life-course of a relation. We know that long-term sex partners are less likely to use condoms, for example, but does this “life-course” effect differ if the tie was first embedded

in a drug exchange? As we move on to the next generation of public health relevant network science, integration of these sorts of questions with our "networks as pipes" models will be crucial.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Morris M. Sexual Networks and HIV. *AIDS*. 1997; 11(Suppl A):S209–S216. [PubMed: 9451987]
2. Morris M, Kretzschmar M. Concurrent Partnerships and Transmission Dynamics in Networks. *Social Networks*. 1995; 17:299–318.
3. Potterat JJ, Rothenberg RB, Muth SQ. Network Structural Dynamics and Infectious Disease Propagation. *International Journal of STD & AIDS*. 1999; 10:182–185. [PubMed: 10340199]
4. Rothenberg RB, Potterat JJ, Woodhouse DE, Muth SQ, Darrow WW, Klovdahl A. Social Network dynamics and HIV transmission. *AIDS*. 1998; 12:1529–1536. [PubMed: 9727575]
5. Gray RH, Wawer MJ, Brookmeyer R, Sewankambo NK, Serwadda D, Wabwire-Mangen F, et al. Probability of HIV-1 transmission per coital act in monogamous, heterosexual, HIV-1 discordant couples in Rakai, Uganda. *Lancet*. 2001; 357:1149–1153. [PubMed: 11323041]
6. Baggaley RF, Boily M-C, White RG, Alary M. Risk of HIV-1 Transmission for Parenteral Exposure and Blood Transfusion: A Systematic Review and Meta-Analysis. *AIDS*. 2006; 20:805–812. [PubMed: 16549963]
7. Gisselquist D. Estimating HIV-I Transmission Efficiency through Unsafe Medical Injections. *International Journal of STD & AIDS*. 2002; 13:152–159. [PubMed: 11860690]
8. Varghese B, Maher JE, Peterman TA, Branson BM, Steketee RW. Reducing the risk of sexual HIV transmission: quantifying the per-act risk for HIV on the basis of choice of partner, sex act, and condom use. *Sexually Transmitted Diseases*. 2002; 29(1):38–43. [PubMed: 11773877]
9. Morris, M. NIH Director's Wednesday Afternoon Lecture Series. Washington, D.C.: 2007. Local Acts, Global Consequences: Networks and the Spread of HIV.
10. Wilson DP, Law MG, Grulich AE, Cooper DA, Kaldor JM. Relation between HIV Viral Load and Infectiousness: A Model-Based Analysis. *Lancet*. 2008; 372(9635):9314–9320.
11. Boily M-C, Baggaley RF, Wang L, Masse B, White RG, Hayes RJ, et al. Heterosexual risk of HIV-1 infection per sex act: systematic review and meta-analysis of observational studies. *Lancet Infectious Diseases*. 2009; 9:118–129. [PubMed: 19179227]
12. Gisselquist D, Upham G, Potterat John J. Efficiency of Human Immunodeficiency Virus Transmission Through Injections and Other Medical Procedures: Evidence, Estimates, and Unfinished Business. *Infection Control and Hospital Epidemiology*. 2006; 27(9):944–952.
13. Rothenberg R, Gisselquist D, Potterat John J. A Simulation to assess the conditions required for high level heterosexual transmission of HIV in Africa. *International Journal of STD & AIDS*. 2004; 15:529–532. [PubMed: 15307963]
14. Wawer MJ, Gray RH, Sewankambo NK, Serwadda D, Li X, Laeyendecker O, et al. Rates of HIV-1 Transmission per Coital Act, by Stage of HIV-1 Infection, in Rakai, Uganda. *The Journal of Infectious Diseases*. 2005; 191:1403–1409. [PubMed: 15809897]
15. Moody J, White DR. Structural cohesion and embeddedness: A hierarchical concept of social groups. *American Sociological Review*. 2003; 68(1):103–127.
16. HELLERINGER S, KOHLER H-P. Sexual Network Structure and the Spread of HIV in Africa: Evidence from Likoma Island, Malawi. *AIDS*. 2007; 21:2323–2332. [PubMed: 18090281]
17. Aral SO. Understanding Racial-Ethnic and Societal Differences in STI. *Sexually Transmitted Infections*. 2002; 78:2–4. [PubMed: 11872846]
18. Chaisson RE, Keruly JC, Moore RD. Race, Sex, Drug Use and Progression of Human Immunodeficiency Virus Disease. *New England Journal of Medicine*. 1995; 333(12):751–756. [PubMed: 7643881]

19. Tanfer K, Cubbins LA, Billy JOG. Gender, Race, Class and Self-Reported Sexually Transmitted Disease Incidence. *Family Planning Perspectives*. 1995; 27(5):196–202. [PubMed: 9104606]
20. CDC. HIV/AIDS Surveillance Report, 2006. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 2008.
21. Adimora AA, Schoenbach VJ. Social Context, Sexual Networks, and Racial Disparities in Rates of Sexually Transmitted Infections. *Journal of Infectious Diseases*. 2005; 191(Suppl 1):S115–S122. [PubMed: 15627221]
22. Hallfors DD, Iritani BJ, Miller WC, Bauer DJ. Sexual and Drug Behavior Patterns and HIV and STD Racial Disparities: The Need for New Directions. *American Journal of Public Health*. 2007; 97(1):125–132. [PubMed: 17138921]
23. Kottiri BJ, Friedman SR, Neaigus A, Curtis R, Des Jarlais DC. Risk Networks and Racial/Ethnic Differences in the Prevalence of HIV Infection Among Injection Drug Users. *Journal of Acquired Immune Deficiency Syndromes*. 2002; 30:95–104. [PubMed: 12048369]
24. Laumann EO, Youm Y. Racial/Ethnic Differences in the Prevalence of Sexually Transmitted Diseases in the United States: A Network Explanation. *Sexually Transmitted Diseases*. 1999; 26:250–261. [PubMed: 10333277]
25. CDC CfDCaP. Update to Racial/Ethnic Disparities in Diagnoses of HIV/AIDS - 33 States, 2001–2005. *Morbidity and Mortality Weekly Report*. 2007; 56(9):189–193.
26. Morris M, Kurth AE, Hamilton DT, Moody J, Wakefield S. Concurrent Partnerships and HIV Prevalence Disparities by Race: Linking Science and Public Health Practice. *American Journal of Public Health*. 2009; 99:1023–1031. [PubMed: 19372508]
27. Potterat JJ, Rothenberg RB, Zimmerman-Rogers H, Green DL, Taylor JE, Bonney MS, et al. Sexual Network structure as an indicator of epidemic phase. *Sexually Transmitted Infections*. 2002; 78:i152–i158. [PubMed: 12083436]
28. Potterat, JJ.; Woodhouse, DE.; Muth, SQ.; Rothenberg, RB.; Darrow, WW.; Klovdahl, AS., et al. Network dynamism: history and lessons of the Colorado Springs study. In: Morris, M., editor. *Network Epidemiology: A Handbook for Survey Design and Data Collection*. Oxford: Oxford University Press; 2004.
29. Woodhouse DE, Rothenberg RB, Potterat John J, Darrow WW, Muth SQ, Klovdahl AS, et al. Mapping a social network of heterosexuals at high risk of human immunodeficiency virus infection. *AIDS*. 1994; 8:1331–1336. [PubMed: 7802989]
30. Darrow WW, Potterat JJ, Rothenberg RB, Woodhouse DE, Muth SQ, Klovdahl AS. Using knowledge of social networks to prevent human immunodeficiency virus infections: the Colorado Springs study. *Sociological Focus*. 1999; 32:143–158.
31. Klovdahl AS, Potterat JJ, Woodhouse DE, Muth JB, Muth SQ, Darrow SQ. Social Networks and Infectious Disease: The Colorado Springs Study. *Social Science & Medicine*. 1994; 38(1):79–88. [PubMed: 8146718]
32. Klovdahl AS, Potterat JJ, Woodhouse DE, Muth J, Muth SQ, Darrow WW. HIV infection in an urban social network: a progress report. *Bulletin De Methodologie Sociologique*. 1992; 36:2433.
33. Rothenberg RB, Woodhouse DE, Potterat JJ, Muth SQ, Darrow WW, Klovdahl AS. Social Networks in disease transmission: The Colorado Springs Study. *National Institute on Drug Abuse, Research Monograph Series*. 1995; 151:3–19. [PubMed: 8742758]
34. Adams, J.; Moody, J. *Code Book for Colorado Springs Sexual and Drug User Networks*. Manuscript: Ohio State University. 2002.
35. Palmer, EN. *Graphical Evolution: An introduction to the Theory of Random Graphs*. New York: John Wiley and Sons; 1985.
36. Harary, F. *Graph Theory*. Reading, Massachusetts: Addison-Wesley; 1969.
37. Watts, DJ. *Small Worlds: The dynamics of Networks between Order and Randomness*. Princeton: Princeton University Press; 1999.
38. Holland PW, Leinhardt S. Some Evidence on the Transitivity of Positive interpersonal sentiment. *American Journal of Sociology*. 1972; 72:1205.
39. Freeman LC. Segregation in Social Networks. *Sociological Methods and Research*. 1972; 6:411–430.

40. Rothenberg R, Muth SQ, Malone S, Potterat John J, Woodhouse D. Social and Geographic Distance in HIV Risk. *Sex Transm Dis*. 2005; 32(8):506–512. [PubMed: 16041254]
41. Granovetter M. The Strength of Weak Ties. *American Journal of Sociology*. 1973; 81:1287–1303.
42. Bearman PS, Moody J, Stovel K. Chains of affection: The structure of adolescent romantic and sexual networks. *American Journal of Sociology*. 2004; 110(1):44–91.

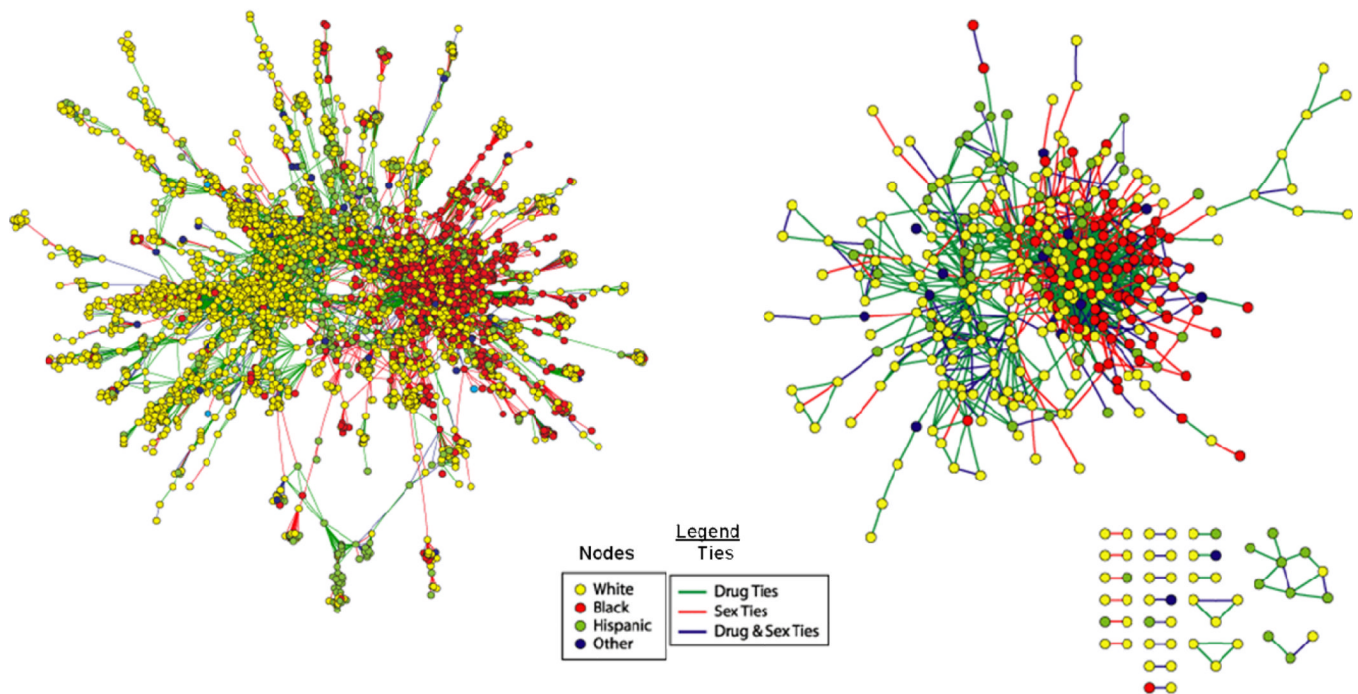


Figure 1. Observed Networks

Panel A presents the giant connected component for the Full Network, which includes 4319 people (of 6019 total named) and the 13,901 ties between them. Panel B presents the observed Respondent Only network. This panel consists of 595 respondents and the 1296 reported connections between them.

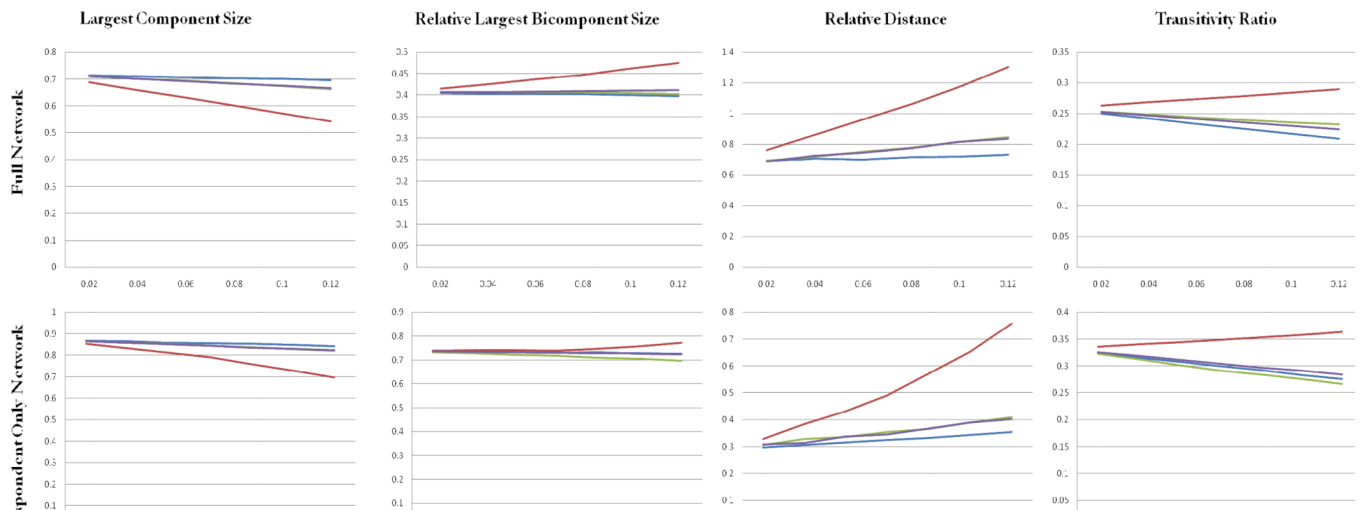


Figure 2. Edge Deletion Results – Connectivity Measures

These line graphs represent the changes in described measures for the simulated counterfactual networks with the indicated percentage of all edges removed as sex ties (red), drug ties (blue), ties involving sex and drugs (green) or random ties (purple). The top panel presents the edge-deletion results for the complete network (left side in Figure 1), while the bottom panel restricts analyses to the network only including respondents (right side in Figure 1).

Racial/Ethnic Segregation

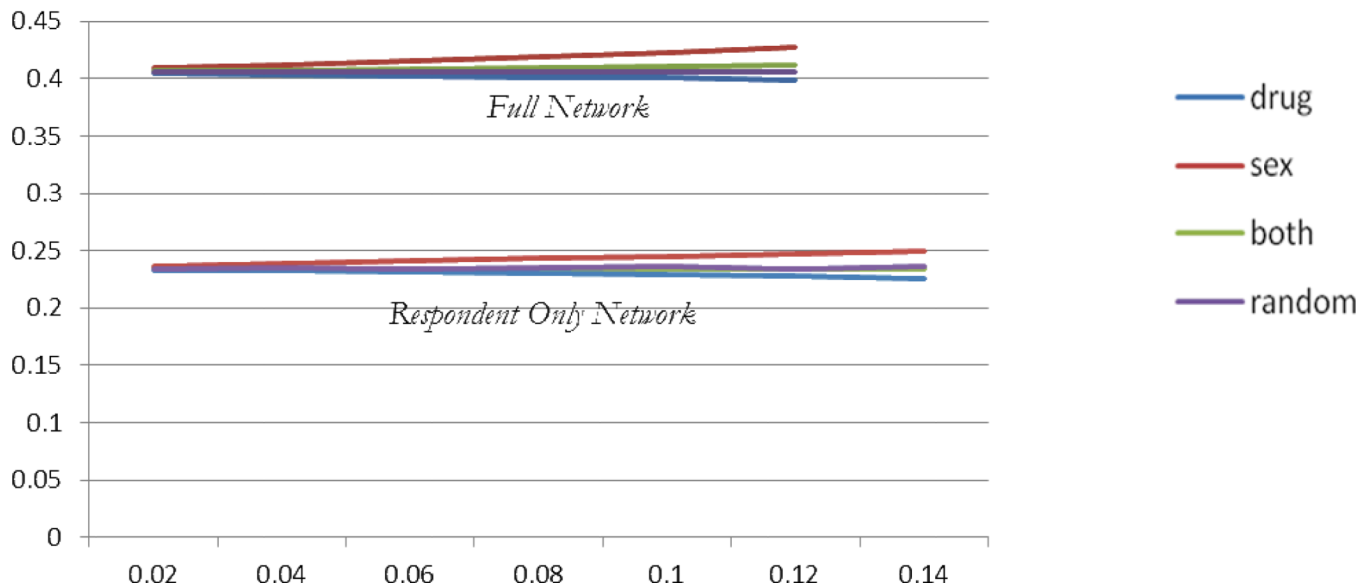


Figure 3. Edge Deletion Results – Racial/Ethnic Segregation
 This Figure presents how the edge-deletions alter the level of racial/ethnic segregation (38) in the complete network (top lines) and the network consisting only of P90 respondents.

Table 1

Observed Respondent Characteristics

Attribute	Description	Percent
Respondent Characteristics		
Sex	Female	45.9
Race / Ethnicity	White	60.5
	Black	19.3
	Hispanic	15.0
	Other	5.2
Age *	Years	29.1
Risk Group:	IDU	51.1
	Prostitute	22.2 [44.6]
	Pimp	7.9 [15.8]
Sex/Drug ties:	At least one	97.6
	Two or more	89.8
	Average # of named Partners *	13.0

NOTES:

* All numbers presented are the percent of each category who have the described characteristic, except age, which is the mean (in years) and average number of named partners.

For “pimps” and “prostitutes” we also report [in brackets] the gender-specific percentage, i.e., the percent of in-category males who are pimps and of females who are prostitutes.

Table 2

Observed Dyad and Network Characteristics

Dyadic Characteristics	Respondents + Named Alters	Respondents Only
<i>Number of Ties</i>		
Sex	2400	200
Drug	9686	853
Both Sex & Drug	1815	243
Total	13901	1296
<i>Racial/Ethnic Segregation Index</i>		
Sex	0.02	-0.07
Drug	0.38	0.03
Sex <i>and</i> Drug	0.18	0.03
All	0.29	0.02
<u>Full Network Characteristics</u>		
# Nodes	6019	595
Component Membership	0.72	0.87
Bicomponent Membership	0.41	0.74
Relative Reach	0.0003	0.009
Transitivity Ratio	0.26	0.33
Racial/Ethnic Segregation Index	0.41	0.23