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Epidemic potential by sexual activity distributions

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Abstract

For sexually transmitted infections like HIV to propagate through a population, there must be a path linking susceptible cases to currently infectious cases. The existence of such paths depends in part on the *degree distribution*. Here, we use simulation methods to examine how two features of the degree distribution affect network connectivity: Mean degree captures a volume dimension, while the skewness of the upper tail captures a shape dimension. We find a clear interaction between shape and volume: When mean degree is low, connectivity is greater for long-tailed distributions, but at higher mean degree, connectivity is greater in short-tailed distributions. The phase transition to a giant component and giant bicomponent emerges as a positive function of volume, but it rises more sharply and ultimately reaches more people in short-tail distributions than in long-tail distributions. These findings suggest that any interventions should be attuned to how practices affect both the volume and shape of the degree distribution, noting potential unanticipated effects. For example, policies that primarily affect high-volume nodes may not be effective if they simply redistribute volume among lower degree actors, which appears to exacerbate underlying network connectivity.

Keywords: *sexually transmitted infections, degree distributions, simulation, cohesion, connectivity, dynamic network diffusion*

1 Introduction

For sexually transmitted infections (STIs), such as HIV, to propagate through a population, there must be an unbroken chain of susceptible cases exposed to those who are currently infectious. The transmission potential in any setting thus depends on the contact timing (Moody, 2002; Morris et al., 2009) and structure (Dombrowski et al., 2013; Ferrari et al., 2006) of the underlying sexual network. This structure is determined in part by each person's number of partners—the *degree distribution* (Hamilton et al., 2008; Newman et al., 2001). Unfortunately, we rarely observe the

network structure itself. Despite continued interest in the association between networks and STI transmission, our understanding of the links between network connectivity and the degree distribution has focused mainly on the effect of average degree. For example, prevention strategies that target infection transmission points—such as condom use, needle cleaning, and circumcision—or individual exposure risk—such as reducing numbers of partners—rarely ask how these changes affect the population network that ultimately sustains the epidemic. However, since network connectivity is an emergent feature unobservable to individuals, it is possible that changes that lower risk for one group might increase overall population risk. Without knowing how changes in the shape of the partnership distribution affect the resulting network connectivity, we risk missing the structural forest by focusing on the largest trees.

Here, we use simulation methods to identify how network connectivity varies by degree distribution *shape* and *volume* in static networks,[1] using measures of these properties that can be obtained from sample survey data. Simple network connectivity is an imperfect indicator of epidemic potential; while it captures the upper bound for reachability, it ignores transmission limits imposed by partnership dynamics and likely overstates risk. As such, we also identify the extent of redundant connectivity, those substructures of the contact network that are most likely capable of sustaining transmission because they are more robust to transmission disruptions. Our results show that when the average number of sexual partners (among those who have partners) in a population is less than about 1.75, epidemic potential is higher in long-tailed distributions, but that for populations with larger average degree, short-tailed distributions create more robustly connected networks.[2] In this region, the networks are characterized by multiply-connected cores that could be capable of sustaining transmission as well as very wide total connectivity covering the vast majority of the population. As a function of volume, these large multiple connected sets exhibit a sharp phase transition, moving from nearly disconnected to widely connected over a narrow volume range. These findings suggest that policy choices should focus on how induced behavior might affect both the volume and shape of the sexual degree distribution. In particular, if policies (such as enhanced law enforcement) remove high-degree actors but fail to lower overall volume, they might exacerbate risk.

2 Sexual behavior and network connectivity

A striking feature of sexual networks measured over long time periods (Lauman et al., 1994; Liljeros et al., 2001), is that a small number of people have a disproportionately large number of partners. The visibility of these people has made them a prominent target for both policy and theory (Barabasi & Albert, 1999; Dezso & Barabasi, 2002; Hethcote, 2000), based on the assumption that people with many partners play a key role either in global connectivity or as transmission hubs. The argument

[1] We do not specify temporal dynamics on the networks because we do not want to conflate reachability due to timing with reachability due to structure. Unless the network is fully concurrent, timing effects lower connectivity (Moody, 2002). Assuming concurrency is evenly distributed across the edges in our networks, any given level of concurrency should produce patterns similar to what we observe here, but at lower absolute levels.

[2] The volume of ties we explore here are consistent with relations observed over a moderate time window, such as within the last year (Morris et al., 2010).

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for the importance of long-tailed distributions has been made most clearly in the "scale-free" network literature. In a scale-free degree distribution, most of a network's connectivity is created by a small number of high-degree nodes, which makes the network robust to random interventions but highly sensitive to disruption if one successfully targets the connecting hubs (Dezso & Barabasi, 2002). The implication for STI networks is that any attempts at generalized interventions need either extremely general adoption or carefully targeted success to be effective. This is similar to the epidemiological focus on network "hubs" that channel the flow across different parts of the network, so attempts at disrupting flow will be most successful when focused on hubs (Dezso & Barabasi, 2002).

While this model provides a clear role for hubs in transmission, focusing solely on high-degree nodes fails to address the more general question of when disease-sustaining network structures might emerge. Empirical support for long-tail sexual networks only emerges when behavior is viewed over long time frames, and most studies showing scale-free distributions are based the number of partners over the last year or lifetime (Liljeros et al., 2001). However, the infectious duration of many STIs can be far shorter (e.g., gonorrhea and chancroid) and even lifelong infections have infectivity that peaks in the early months (e.g., HIV and syphilis; Holmes et al., 1999). This suggests that the disease-relevant time frame is much shorter. Over such short temporal spans, most degree distributions are characterized by a surprisingly short tail (Armbruster et al., 2016; Brewer et al., 2000; Helleringer & Kohler, 2007; Todd et al., 2009; Young et al., 2014) This raises the important question of how the underlying network structure varies jointly by the mean and the skewness of the degree distribution.

We explore the effect of two basic features of the degree distribution on network connectivity.[3] First, the per capita volume of sexual activity, or *average degree,* captures the volume of contacts and we know that network connectivity generally increases with mean degree, though not linearly. Second, the *shape* of the degree distribution ranges from homogeneous distributions (everyone having the same number of partners) to highly unequal distributions (where a few people have many more partners than anyone else). We are thus interested in knowing: *How do mean degree and degree skewness jointly determine network connectivity?* The answer to this question can inform policy and help policymakers understand the multiple ways interventions that affect sexual activity distributions might affect risk. Moreover, since these two moments can be estimated with sample data, public health officials can monitor the potential STI risk in a setting using only local anonymous data.[4]

3 Network connectivity and epidemic potential

The sexual contact network forms the foundation over which a disease is transmitted; the actual transmission network—the set of links that carry the disease through

[3] Other features, particularly assortative mixing by degree or demographic and behavioral activity, also shape the underlying networks. We ignore these features here for simplicity, but see Morris et al. (2009) for examinations of the unique effects of behavioral mixing.

[4] There are closed-form solutions for estimating the simple (single-path) connectivity of a network for any degree distribution (Newman et al., 2001), though non for bi-connectivity. There have been no explorations of the general association between degree skewness and volume across a range of relevant parameter values. Thus, our work provides general insights into this association, which will help anticipate the effects of policies that impact average degree or the skewness of the degree distribution.

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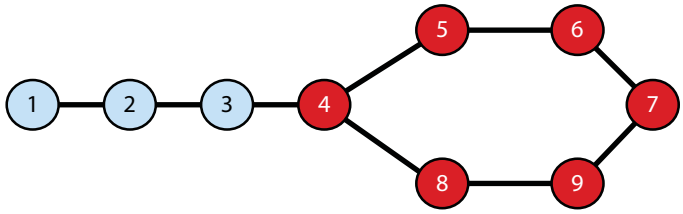

Fig. 1. Example network illustrating multiple connectivity. (Color online)

a population—is a disease-specific stochastic function over the observed contact network. This transmission network depends jointly on the network structure, tie duration/turnover, and the level and duration of infectivity (Hethcote, 2000). For analytic clarity, we focus on the connectivity properties that emerge solely as a function of degree features, ignoring temporal aspects of relational turnover, disease-specific transmission features (such as gender differentials in transmissibility), or other network structuring features (such as degree assortativity or clustering imposed by other demographic characteristics). We focus on moderate degree levels—with average degree ranging from just over 1 to just under 3—as this is the region most reflected in empirical data, that sets the bounds for transmission over moderate time frames, and represents the space with variability in connectivity.[5] Following standard graph theory (Harary, 1969), we say that person i can reach person j if there is a chain of relations (a *path*) connecting them; a *component* of a network consists of all nodes that are connected by at least one path, and the largest component represents the maximum possible range for epidemic spread within any observed network. A network is said to have a *giant component* if the largest connected component is greater than 50% of the population. We use the maximum *component size* as one summary measure of epidemic potential, recognizing that this is generally a significant over-estimate of the actual population proportion at risk. Since transmission is stochastic, diffusion potential is higher when there are multiple routes around actors that might otherwise block transmission due to activity (such as condom use) or relationship timing (Moody, 2002). This feature of network structure is illustrated in Figure 1.

Networks characterized by long single-linked chains are less likely to carry infection, since transmission stops if even one person along the chain fails to pass the infection. Multiple connectivity increases robustness by providing ways around such breaks, providing alternative routes to augment transmissibility. For example, if an STI were to enter this network at node 1 and had a 0.25 chance of being transferred across the edges, then the likelihood of making it to node 4 (a three-step distance) would be approximately 0.016 (0.25^3); the same infectivity that

[5] Our simulations show how much connectivity will be observed at each point in the degree distribution space, ignoring temporal transmission aspects. Thus, if the data were collected for "relations in the last 6 months," then the results show what the cumulative contact network would look like over that time span. If the questions were for the last week, then the resulting contact connectivity is similarly bounded. All else equal, as time spans increase (last year, last 5 years, lifetime), the relevance of the cumulative connectivity to disease transmission decreases, as relational timing and sequencing reduces the number of reachable paths in long-term contact network. See Moody (2002) for examples contrasting long-term cumulative contact networks with temporal reach or Hellerginer & Kohler's (2007) work finding connectivity similar to the volume discussed here.

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enters at node 7 would have about a 0.031 chance ($2*0.25^3$) of making it to node 4 (also a three-step path). The probability is doubled since there are two independent transmission routes.

We operationalize structural transmission robustness by identifying the portions of the network that are connected by multiple paths (Moody & White, 2003). Two network paths are *node-independent* if they only have their end nodes in common but otherwise do not overlap. Generally, a *k-connected component* can only be separated by removing at least k nodes, and every pair in a *k-component* is linked by at least k node-independent paths (Harary, 1969). Since no single person can block transmission in a network with k-connectivity greater than 1, epidemic potential becomes a collective property and we consider two-connected components (called *bicomponents*) a minimum graph-theoretic operationalization of a robust potential STD core (Rothenberg et al., 1998).[6] In Figure 1 (and below), red nodes are members of the biconnected core while blue nodes are members of the largest connected component.

4 Methods

We define a space of possible networks by the volume of sexual activity (mean number of partners) and the length of the high-degree tail, which varies between a very "short-tailed" network, where nobody has significantly large numbers of partners, to the characteristically long-tailed degree defined by the scale-free distribution.[7] Operationally, we approach the question in two phases, we first examine the extreme ends of the space to identify the basic contours of the results and help build intuition. We generate short-tailed degree distributions by assigning a minimum degree of 1 and adding two random draws from a bernulli distribution with p ranging from 0.25 to 0.75. For the boundary case of short-tailed networks, degree ranges from 1 to a maximum of 3, while the scale-free networks have the same number of edges but no limit on highest degree. We generate low-volume scale-free networks by manipulating the exponent (see footnote 8) to arrive at matching average degree values, conditional on degree $\geqslant 1$. In the second phase, we interpolate across the range of distributions possible between these two boundary cases by examining all possible degree distributions with a minimum of 1 and maximum of 6. Networks are generated by looping over each degree value and assigning a proportion from 0 to 1 in 0.025 increments. We then retaine only those distributions that summed to 1.0, generating about 98,000 valid degree distributions. We use the degree distributions

[6] Higher order k-components are necessarily nested within lower order k-components, and should track the same general pattern by volume and distribution found here. Since higher order k-components are computationally expensive to identify (Moody & White, 2003), bicomponents allow us to identify the connectivity profile by volume and shape of the degree distribution in a computationally efficient manner.

[7] We use the term scale-free in keeping with the literature, where the $p(k)\ k^{-\lambda}$; that the probability of having degree k is distributed as that degree to a negative exponent. Technically, the scale-free nature of this distribution only holds in infinite graphs with higher volume than explored here, qualitatively the issue is having a long tail to the distribution means that the hubs responsible for connectivity are few and thus hard to identify through random intervention.

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generated above and the method in Newman et al. (2001) to simulate 10,000 node networks for each of the degree distributions.[8]

We measure relational volume with mean degree. To measure the shape of the degree distribution, we focus on the *dispersion* of people across the degree distribution and the *symmetry* of that distribution. We combine these into a *variability* measure, which we define as the standard deviation multiplied by skewness, with a constant added to skewness to keep everything positive. Skewness captures shape but is scale invariant, while the standard deviation gives a better sense of the amount of variation it averages across both sides of the mean, by combining the two moments we get a simple composite measure of the length of the high-degree tail. While one could define the variability in other ways, particularly making use of inequality measures such as the Gini or Theil indices, this simple metric is easy to implement and thus we hope more useful for practitioners. High values capture wide dispersion on a long-tailed distribution. After generating each network, we calculate the size of the largest component and bicomponent.[9] The maximum extent of an epidemic is bounded by the largest component, and of a robust epidemic potential by the largest bicomponent.

5 Results

Figure 2, building on prior work (Morris et al., 2007), shows an example of this process on two short-tail degree distributions. In the top panel, average degree is about 1.7, with 40% of the nodes having a single partner, 50% having two partners, and 10% having three partners. This degree distribution generates a network without a giant component; the largest connected chains are only about 1% to 1.7% of the total population, and none of these connected sets contain a biconnected core. In such a setting, it would be extremely difficult to sustain disease transmission. In the bottom panel, tie volume increases to 1.9, by moving about half of those with one partner in the top panel to two partners. This change results in a dramatic shift in the overall network connectivity, as now more than half of the population is connected in the giant component (57%) and about 16% are members of the biconnected core.

Figure 3 expands this across two archetypal network shapes: one long-tailed (scale-free) distribution (red tones) and one short-tailed (max degree = 3) distribution (blue tones) (Morris et al., 2007). We find a sharp phase transition for the short-tailed networks at 1.73. The largest component comprises less than 3% of the population before the threshold, about 38% at the threshold, and over 90% of the population when the mean degree reaches 2. The size of the largest bicomponent also increases

[8] We compared our results to smaller networks and find a very similar pattern. While there is some evidence that phase transitions to connectivity vary in small village-scale networks (Carnegie & Morris, 2012), our results seem very robust to overall size. Thanks to a reviewer for suggesting these comparisons; results are available from the first author on request.

[9] While analytic solutions exist for the size of a component with arbitrary degree distributions (Molloy & Reed, 1998; Newman et al., 2001), no solution exists for the size of the largest bicomponent, requiring us to generate the network and measure it directly. Since finding components in networks is computationally cheap, we calculate component size directly as well. We compared the analytic solutions for component size to the simulated results finding a nearly perfect fit, which suggests our networks are being simulated accurately.

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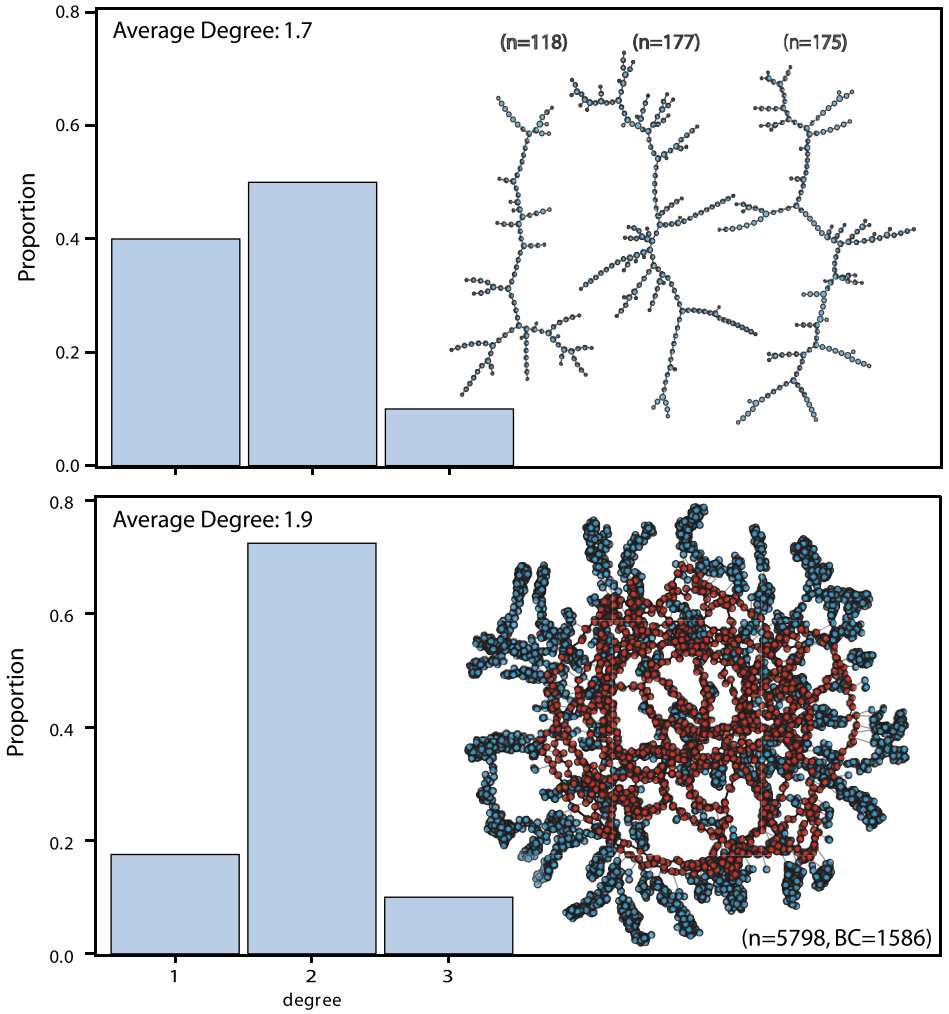

Fig. 2. Connectivity in two short-tailed, low-volume networks.

rapidly as a function of volume, though less steeply: From 25% of the population, when the mean degree is 1.9, to about 56% when the mean rises to 2.0 and ultimately reaching about 90% of the population. The amount of connectivity that emerges as populations pass through the phase transition is striking and represents a qualitative shift in the structure. Just below the threshold, the network is composed mainly of small trees. As volume increases, these disconnected sets join together forming larger components, and re-join to form the biconnected core.

Eventually, these networks take on a core-periphery structure, with a robustly connected core and multiple chains emanating from the center. In contrast, the scale-free distribution reaches the threshold for component size at lower average degree (1.3) but grows more slowly as we add relational volume, and at the maximum volume settings reaches only about 80% of the population. The disparity between the models is most striking for the size of the robust biconnected core, which never reaches more than 20% of the population in the scale-free networks but continues to grow rapidly in the short-tailed networks. Qualitatively, this smoother transition

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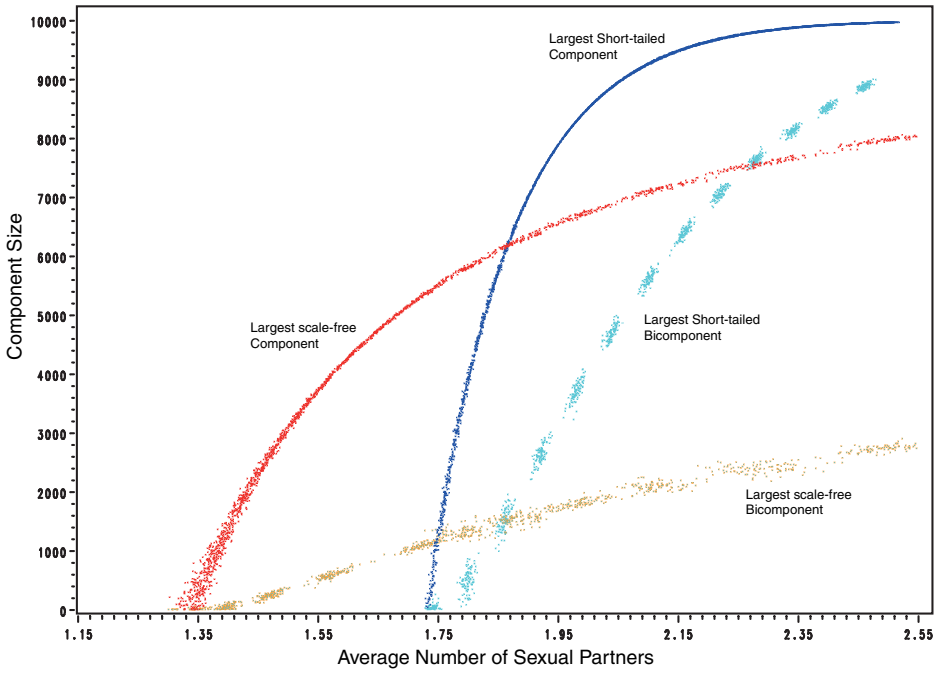

Fig. 3. Size of Largest component and bicomponent by average number of sexual partners for short-tailed and scale-free distributions. The curves plot the growth of the largest component and bicomponent as a function of the average degree, based on 100 simulations of a 10,000-node network at each degree setting. The red curve plots the analytic solution for the size of the giant component for the simulated networks with scale-free distributions, and the orange curve plots the largest bicomponent. The dark blue curve plots the analytic solution for the size of the largest component for the simulated low-degree networks, and the light blue curve plots the size of the largest bicomponent. The bicomponent curves are not continuous due to sampling.

among the scale-free networks results from adding disconnected dyads to the star hubs and slowly growing connectivity in the periphery of the network.

Figure 4 extends these results for the full enumeration of all possible degree distributions spanning the space between these two archetypical networks distributions. The x-axis in Figure 4 is the same as that in Figure 3 (average degree), the y-axis is our variability measure, capturing the continuum between short-tailed networks (low values) and long-tailed networks (high values). The z-axis (color and contour) captures the size of the largest component (left panel) and bicomponent (right panel). The contours for component and bicomponent size in Figure 4 help identify equivalent epidemic risk across the space. The region characterized by "left-leaning" contour lines (around average degree < 1.75) is where long-tailed distributions will create larger connected components at any given volume level. Where the contours are roughly perpendicular to the x-axis, the epidemic risk is equivalent for long- and short-tailed distributions, whereas the portions of the space characterized by "right leaning" contours, risk at any given volume is higher for short-tailed distributions than for long tailed. The speed of this transition by volume is given by how quickly component size increases for any level of variability—when variability is low, changes in volume generate rapid changes in connectivity (contour lines close together), while

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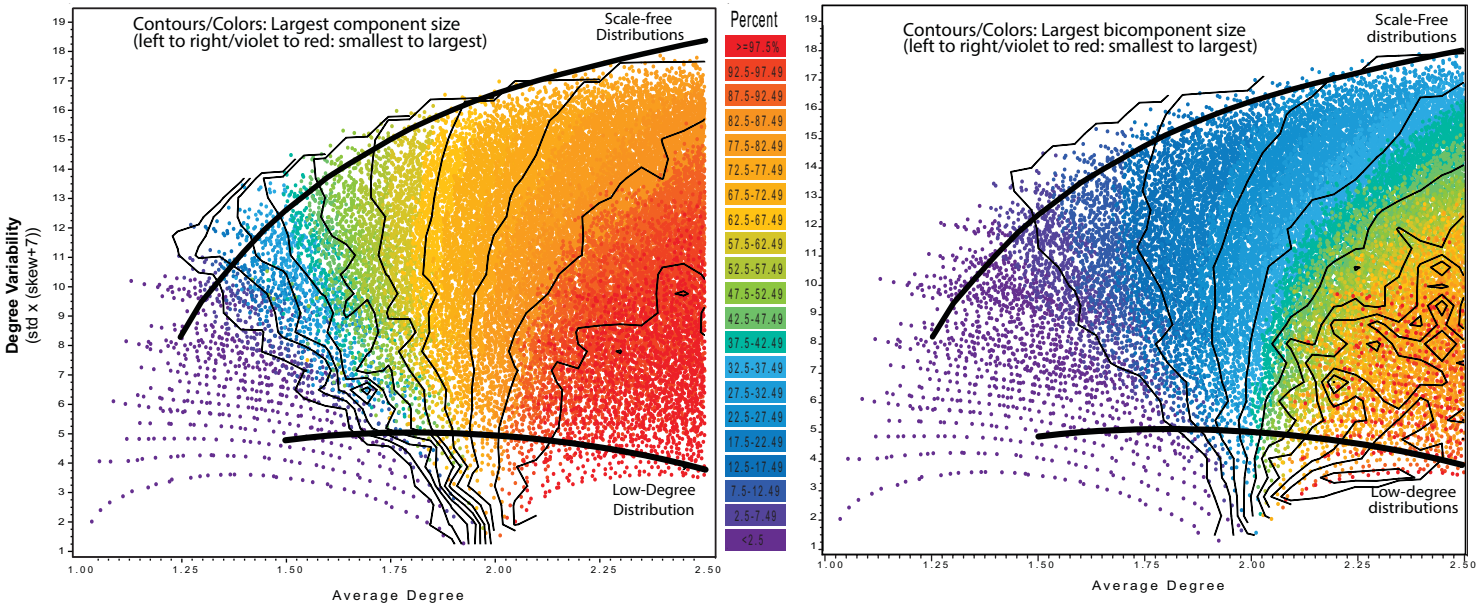

Fig. 4. Size of largest component and bicomponent over the degree distribution state space. Point color and contour lines corresponds to the size of the largest component (left panel) and bicomponent (right panel) respectively. Contour lines mark approximate decile levels of the distributions. The scale-free and low-degree distributions from Figure 2 are plotted as lines across this space for reference. To avoid point stacking, a small amount of randomness has been added to the mean degree score for each plot.

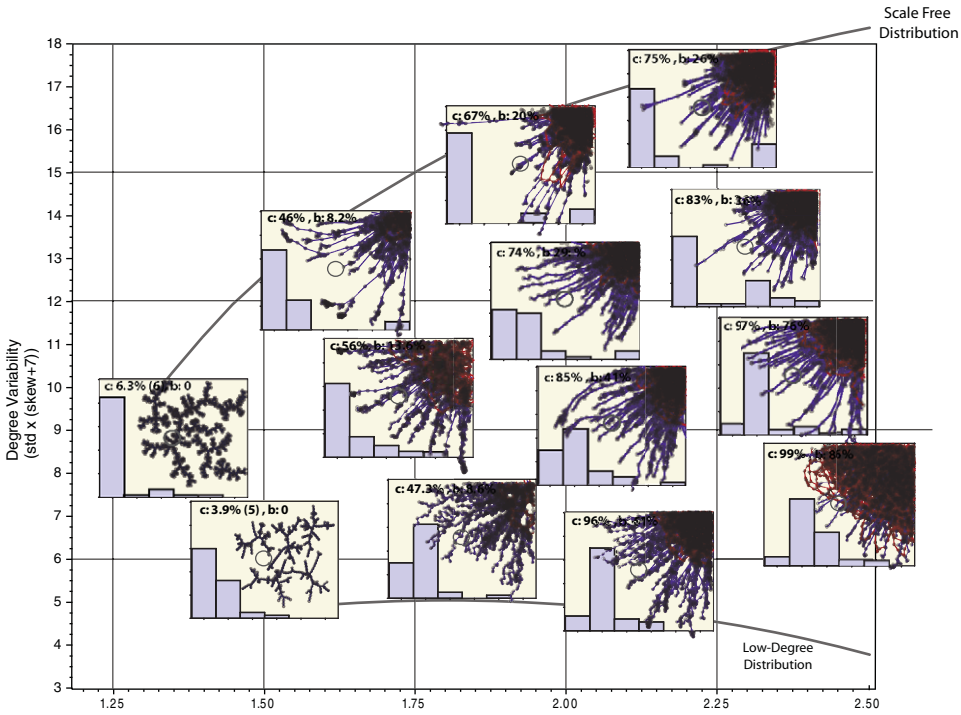

Fig. 5. Select degree distribution and partial sociograms for simulated giant components. Histogram bars are for degree = 1 to 6 from left to right; a bar reaching the top of a subpanel would be 100%. The number of nodes in components of at least 1% is given in the upper left of each subfigure. If there is more than one such component, the number is given in parentheses following the count. The count for size of the largest bicomponent is also given. For each network figure, blue nodes are in the largest component, red in the largest bicomponent.

when variability is high the transition to larger components is smooth (contour lines far apart).

Epidemic potential is highest in the reconnected core, represented in the right panel of Figure 4. The shape of the surface defined by the z-axis rises much more sharply in the lower right-hand portion of the space, and we only see giant bicomponents in this low-variability region. This result means that in the rest of the space, the large single connected components have either no bicomponent (purple and dark blue) or comparatively small bicomponents (light-blue to cyan), suggesting a setting with fragile epidemic potential.[10] That is, networks with large components but small bicomponents are characterized primarily by long single-connectivity paths, which are comparatively easy to disrupt, as any point can block transmission at the nodes (e.g., by using condoms) or across edges (e.g., via inconsistent temporal order). In contrast, we find more robust networks, capable of carrying disease even in the face of transmission disruption, when the degree distribution has low variably.

[10] These sparsely connected areas are the sections of the distribution space where relational timing, and concurrency in particular, will be most important (Moody & Benton, 2016).

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To gain a qualitative sense of how the shapes of these distributions relate to the structure of the resulting networks, we plot sample degree histograms from across the space (exact point of the sample distribution marked by the "◯" within each subpanel) along with an exemplar sociogram colored by component (blue) and bicomponent (red) membership in Figure 5. As these are largely core-like structures (since there is no mixing feature other than degree), we display only the "south-west" corner of the sociogram.

The dominating robust connectivity is clear in the lower right of Figure 5, where it is evident that nearly everyone is re-connected by multiple paths, compared to the long "tendrils" of single connected paths in the long-tail distribution portion of the space. In these reconnected settings, it is difficult to prevent STI flow unless blocking activity (condom use or vaccination, for example) is extremely widespread.

6 Conclusions

In this paper, we explored network connectivity in the low-volume sexual activity space that can be used as a qualitative model for moderate-term sexual networks (Carnegie & Morris, 2012; Helleringer & Kohler, 2007; Young et al., 2014). We used simulations to examine nearly 98,000 different degree distributions within this space and then identified the size of the largest component and bicomponent as indicators of potential epidemic extent and robust connectivity, respectively. We find that the large components necessary for widespread diffusion can emerge at low average degree, but that the proportion of the population reached is highly sensitive to both the volume and the shape of the degree distribution. Long-tailed distributions can create extensive connectivity at lower average volume, but these tend to be fragile single-connected networks and ultimately reach fewer people than in short-tailed distributions with similar volume. We also find that the volume transition threshold for networks with large and robust epidemic potential is sharper for short-tailed distributions than for long-tailed degree distributions.

The main implication of these results is to make clear the highly non-linear and contingent nature of connectivity substrates in sexual networks. Any policy that targets degree will likely shift both volume and shape, and we should be cognizant of what that implies for epidemic potential. We find that epidemic potential is contingent on both the volume and shape of the degree distribution. Degree volume captures how much sex is happening in the population overall while the degree variability describes the concentration of who is having sex. Substantively, one can assume that the volume dimension in a setting is thus an imperfect indicator of population demand; while the distribution shape, particularly the length of the upper tail, is largely about the concentration of supply. Social interventions (either as policy aimed at health interventions or stigma-enhancing police enforcement) that focus disproportionately on high-degree nodes will largely have the effect of shifting who has sex without changing volume. With respect to our results, this is equivalent to moving the population "down" the y-axis of Figure 4 at a fixed point along the x-axis (volume), which will lead to *greater* connectivity in the right-hand portion of the space, and thus likely increase epidemic potential. In contrast, interventions targeted to lower demand for commercial sex uniformly shifts the population to types of networks with lower connectivity (down and to the left in Figure 4).

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Similarly, policies aimed at effectively removing transmission-relevant edges—such as widespread condom use or lowering numbers of concurrent partnerships—will have the substantive effect similar to lowering average degree; pushing the population toward safer regions of the space (left on the x-axis in Figure 4). While volume-lowering interventions seem to always help (reducing network connectivity), the returns are highly non-linear depending on the shape of the distribution, and as such, the realized effectiveness will vary widely across populations. Since public policies tend to be blunt instruments, it is important to ask how different incentives shape the structure of the degree distribution. By assessing where populations sit in this degree space, health policy investigators might also have a better sense of what sort of network structure is active, helping them evaluate achieved trace samples.[11]

These results are based on simulations and focus on a single dimension of the underlying networks—namely the degree distribution—and as such are not without their limitations in making inferences to epidemic potential. While our findings are based on random networks, one can easily extend the analytic results to more realistic networks for STIs, and we would expect qualitatively similar outcomes (in fact our networks appear similar to those observed in real-world settings, see Helleringer & Kohler, 2007). For example, clustering features—such as strong assortative mixing based on sexual preference, number of partners, or race—might prevent giant components from forming by isolating subpopulations, but the restrictions would need to be nearly absolute. Anything less than absolute isolation will simply increase path redundancy within the assortative groups, creating localized disease cores rather than the simple core-periphery shape identified here. Degree assortativity has well-known effects on the extent of reachability in networks; future work should extend these results to assess whether such effects are contingent jointly on volume and variability. Additionally, our approach assumes distributions result from a single underlying behavior (sex). Future work should address how different actors (e.g., men vs. women), or different ties (e.g., sex vs. needle-sharing) differential and sometimes complementary contributions to those distributions (see e.g., adams et al., 2013) alter our results.

We have enumerated degree distributions across a somewhat narrow range, and one could extend along both dimensions, though there is no *a priori* reason to expect a discontinuity in the shape of the response surface found in Figure 4. That is, extending to higher maximum degree (beyond 6) or higher volume (beyond 2.5) should continue with the surface shape identified here, and is well beyond most empirical estimates over disease-relevant time frames.

Finally, more realistic transmission simulations could more precisely target policy toward disease specific transmission times and windows, as a key limitation of this work is that we ignore relationship temporal dynamics. Timing features have at least two implications. First, realistic transmission dynamics would respect the infectivity window of each disease (and the population demographics and behaviors). The effect of this, however, is largely to shorten the relevant duration within which partners can contribute to connectivity, effectively lowering volume, and variability. As a result, the effective transmissibility networks will be sparser than those examined here and likely

[11] We thank a reviewer for pointing this out.

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with shorter tails. Second, continued transmission depends on a temporal sequence of relations to create a continually connected set of future susceptible cases linked to currently infectious nodes (Moody, 2002; Morris et al., 2009). This effectively limits exposure to the forest of paths existing after transmission, further limiting the relevant degree distribution to the lower volume portion examined here. In general, there are complex interactions between volume, variability, and relationship timing, particularly in open populations, that make identifying reachability complicated. However, analytic work on identifying expected size of the forward reachable path—the set of people downstream in time in an open population—has begun (see Morris et al., 2010), which holds great promise for understanding the joint interaction of structure and timing. Our initial simulation forays into this work suggest a general correspondence between the shape of the response surface in Figure 4 when relational timing is built in, so we expect the results to be robust to timing in general shape, if not level, though a full examination of this is beyond the scope of the current paper.

Since adding such features come at considerable computational cost, and depend crucially on setting-specific transmission details, we leave that to future work. Any transmission effects must occur within a subset of edges and paths identified here. We therefore anticipate that the contours of high epidemic potential in such work would correlate highly with the z-axis of Figure 4 here, but with lower absolute levels.

The main contribution of this paper has been to highlight a qualitative tradeoff in network connectivity between the shape and volume of sexual degree distributions. In very low-volume settings, long-tailed degree distributions generate higher connectivity, as high-degree hubs are the only way to generate any connectivity, though much of that connectivity is fragile. As total relational volume increases, however, we see a shift, where short-tailed degree distributions generate higher total connectivity than long-tailed distributions. Short-tailed distributions reach more people, and do so more robustly than long-tailed distributions, at higher volumes.

If both highly skewed and short-tailed networks can sustain high connectivity, the public health question becomes empirical and context specific: What do sexual networks look like in the populations and time-spans of interest? The short infectious period of many STDs requires large components to emerge relatively quickly and maintain in the face of rapid population churn to sustain transmission. If the volume levels in such settings create relatively sparse contact networks, concurrency will be crucial for maintaining the temporal connectivity needed for disease spread (Moody & Benton, 2016, Morris et al., 2009). Armed with a better sense of how changes in individual behavior might affect contact structure, researchers can next turn our attention more directly to questions of how timing and population turnover affect disease spread within these structures.

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