Asymmetric percolation drives a double transition in sexual contact networks

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Abstract modeling of epidemics on networks remains an active field, because some of the most basic features of epidemics are still misunderstood. The classic model is quite simple (1): disease spreads stochastically with a fixed transmission probability, \( T \), through contacts around a given patient zero. The outbreak dies quickly if \( T \) is too small but spreads to a macroscopic fraction \( S \) of the entire population if \( T \) is larger than a threshold \( T_c \). At \( T_c \), most of the typical insights from phase transition theory are valuable. For instance, the sizes of microscopic outbreaks follow a power law distribution, such that the expected size of microscopic outbreaks, \( S \), indicates the position of a phase transition. Indeed, as \( T \) increases, \( S \) monotonically increases, diverges exactly at \( T_c \), and then monotonically goes down; meanwhile, the expected macroscopic epidemic size, \( S \), starts increasing monotonically at \( T_c \).

However, simple modifications to this model can dramatically alter its phenomenology. The epidemic threshold can vanish in networks with a scale-free degree distribution (2) or in growing networks (3). The phase transition can be discontinuous in the case of complex contagions with threshold exposition or reinforcement (4), interacting epidemics (5, 6), or adaptive networks (7–9). Recently, a unique phenomenon of double-phase transitions has also been observed numerically when networks have a very heterogeneous and clustered structure (10, 11).

The current Zika virus (ZIKV) epidemic exhibits two unique properties. First, while the main transmission pathway for ZIKV is through a mosquito vector [predominantly *Aedes aegypti* or *Aedes albopictus* (13, 14)], a feature which has its own type of well-studied model and behavior (14–16), it can also spread through sexual contacts (17, 18). Second, the probability of sexual transmission is highly asymmetric between males and females. Although this is also true for other sexually transmitted infections, such as HIV (19), it reaches an extreme level of asymmetry in the case of ZIKV. Indeed, males can be infectious for over 180 days (20), while females are infectious for less than 20 days (21). Assuming a symmetric risk of transmission per contact, males would be 10 times more likely to transmit to a partner than females. This is, however, a rather conservative estimate, since male-to-female transmissions tend to be more likely than the opposite (19, 22).

The dynamics of the ZIKV epidemic is well-understood in countries where the vector-borne pathway dominates (23). However, with travelers moving to and from endemic regions, the potential of ZIKV as an emerging sexually transmitted infection (STI) in regions without the mosquito vector remains to be fully assessed. Indeed, with only few reported cases of sexual transmission of ZIKV—including male to male, male to female, and female to male (18)—the scientific community still struggles to reach a consensus on the impact of sexual transmission of ZIKV (24, 25). It is, therefore, imperative to investigate the extent to which canonical knowledge about emerging infectious diseases applies to the threat assessment of ZIKV as an STI.

We model the ZIKV sexual transmission through asymmetric percolation on random sexual contact networks and solve it exactly using a multitype (multivariate) generating function formalism (26). We then show how the asymmetric percolation leads to a double transition. Interestingly, the formulation of our model allows us to provide a first analytic framework for the aforementioned numerical results on double transitions. More importantly, this allows us to identify two different thresholds for ZIKV to be endemic as an STI in regions where the mosquito vector is absent but where travelers to/from endemic regions can bring the virus. Our results also shed light on a class of processes on random networks by providing a complete analysis of dynamics with multiple critical points.

Zika virus (ZIKV) continues to be a threat to countries with conditions suitable for transmission, namely adequate temperatures and the presence of competent mosquito vectors. Estimates of risk in other countries based on the sexual transmission of ZIKV may be underestimated because of inadequate surveillance. Here, we formulate random network models of sexual transmission of ZIKV with asymmetric transmission (men being infectious for longer than women) and show that, contrary to previous work, there exists two epidemic thresholds and that certain men who have sex with men communities could sustain transmission on their own. Our results also provide insight into the effectiveness of sexual networks by providing a complete analysis of dynamics with multiple critical points.


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Zika virus (ZIKV) exhibits unique transmission dynamics in that it is concurrently spread by a mosquito vector and through sexual contact. Due to the highly asymmetric durations of infectiousness between males and females—it is estimated that males are infectious for periods up to 10 times longer than females—we show that this sexual component of ZIKV transmission behaves akin to an asymmetric percolation process on the network of sexual contacts. We exactly solve the properties of this asymmetric percolation on random sexual contact networks and show that this process exhibits two epidemic transitions corresponding to a core–periphery structure. This structure is not present in the underlying contact networks, which are not distinguishable from random networks, and emerges because of the asymmetric percolation. We provide an exact analytical description of this double transition and discuss the implications of our results in the context of ZIKV epidemics. Most importantly, our study suggests a bias in our current ZIKV surveillance, because the community most at risk is also one of the least likely to get tested.
regions can spark a sexual epidemic when they return/visit. We also find that, in the large interval of parameter space between those two thresholds, the asymmetric percolation creates a core–periphery structure in a system where there was none. Finally, we discuss the implications of this core–periphery structure for the surveillance and control of the ZIKV epidemic and provide policy guidelines.

**Results**

Inspired by the sexual transmission of ZIKV, we investigate the effect of asymmetry on bond percolation on networks and show that it yields outcomes akin to the double-phase transitions observed numerically in other contexts (10, 11). To isolate the effect of asymmetry alone and thus, provide a clear proof of concept, we consider a very simple model, in which nodes belong to one of six types based on their sex and sexual orientation (i.e., female/male and homo-/bi-/heterosexual). Each node is assigned a number of contacts, \( k \), independent of its type (i.e., all nodes have the same degree distribution \( \{ p_k \}_{k \geq 0} \)), and links are created randomly via a simple stub-matching scheme constrained by the sexual orientations (12, 26). For instance, bisexual males choose their partners randomly in the pools of heterosexual females, bisexual males and females, and homosexual males. This implies that there is no correlation between the type of a node and its number of contacts and consequently, no core–periphery structure. In fact, this model generates well-mixed contact networks that are indistinguishable from networks generated with the configuration model and the same degree distribution (SI Appendix).

Although these networks are originally undirected, asymmetric percolation implies that links can be more likely to exist (i.e., transmit) in one direction than in the other, thus inducing an effective semidirected structure to the networks (27). In other words, \( T_{ij} \neq T_{ji} \) in general, with \( T_{ij} \) being the probability of transmission from a node of type \( i \) to a node of type \( j \) (hereafter, we denote \( \mathcal{N} \) as the set of the six possible types of nodes). In particular, we set \( T_{ij} = T \) for every \( i, j \in \mathcal{N} \) except when \( i \) corresponds to a female, in which case we set \( T_{ij} = T/a \) to enforce asymmetric probabilities of transmission (i.e., females are \( a \) times less likely to transmit ZIKV than males).

We adapt the formalism presented in ref. 26 to compute the epidemic threshold and the expected final size of outbreaks in the limit of large networks. It is worth pointing out that, since asymmetric percolation (i.e., whenever \( a \neq 1 \)) induces an effective semidirected structure to the networks, the probability for the existence of an extensive connected component (i.e., an epidemic) does not equal to its relative size as for symmetric, traditional bond percolation (i.e., \( a = 1 \)). Here, we focus on the relative size for the sake of conciseness; we refer to SI Appendix for full details of the analysis and numerical validation.

To obtain the relative size of the extensive component, we define \( v_i \) as the probability that a neighbor of type \( i \) is not in the extensive component, which we solve by a self-consistent argument. If the neighbor of a node is not in the extensive component, then none of its other neighbors should be in it either. With the probability that the neighbor has a degree equal to \( k \) being \( k p_{k_1}/(k) \), with \( \langle k \rangle = \sum_k k p_{k_1} \), this self-consistent argument can be written as

\[
v_i = \frac{\sum_k k p_{k_1} \sum_{j \in \mathcal{N}} \alpha_{ij} (1 - T_{ji} + T_{ji} v_j) \langle k \rangle^{k-1}}{\sum_k k p_{k_1} \sum_{j \in \mathcal{N}} \alpha_{ij} (1 - T_{ji} + T_{ji} v_j) \langle k \rangle^k}, \tag{1}
\]

where \( \alpha_{ij} \) is the probability that a neighbor of a node of type \( i \) is of type \( j \) (i.e., \( \sum_j \alpha_{ij} = 1 \) for any \( i \)). Solving this equation for every \( i \in \mathcal{N} \), the probability that a node of type \( i \) is part of the extensive component, \( S_i \), corresponds to the probability that at least one of its neighbors is in it as well:

\[
S_i = 1 - \sum_k p_k \left[ \sum_{j \in \mathcal{N}} \alpha_{ij} (1 - T_{ji} + T_{ji} v_j) \right]^k. \tag{2}
\]

The relative size of the extensive component is then

\[
S = \sum_{i \in \mathcal{N}} w_i S_i,
\]

where \( w_i \) is the fraction of the nodes that are of

![Fig. 1.](image)
type $i$. Below the epidemic or percolation threshold, every $v_i$ is equal to one, since there is no extensive component. The percolation threshold corresponds to the point where the largest eigenvalue of the Jacobian matrix of Eq. 1 equals one.

The distribution of the composition of the small, nonextensive components can be computed in a similar fashion (full details are in SI Appendix). Let us define the probability-generating function (pgf) $H_i(x)$, with coefficients that correspond to the probability that a neighbor of type $i$ leads to a small component of a given composition (i.e., the number of nodes of type $j$ is given by the exponent of $x_j$). Invoking the same self-consistency argument as above, the pgfs are the solution of

$$H_i(x) = x_i \sum_k \frac{p_k}{(k)} \sum_{j \in \mathcal{N}} \alpha_{ij} \left[1 - T_0 + T_0 H_j(x)\right]^{k-1},$$

where the extra $x_i$ has been added to account for the neighbor of type $i$ itself. Similarly, the small component that can be reached from a node of type $i$ is, therefore, given by

$$K_i(x) = x_i \sum_k p_k \left[ \sum_{j \in \mathcal{N}} \alpha_{ij} \left[1 - T_0 + T_0 H_j(x)\right] \right]^k.$$

The distribution of the composition of the small components is $K(x) = \sum_{i \in \mathcal{N}} v_i K_i(x)$. It is worth noting that, whenever $S > 0$, the distribution generated by $K(x)$ is no longer normalized, $K(1) < 1$, such that the average number of nodes of type $i$ in the small components is

$$\langle s_i \rangle = \frac{1}{K(1)} \frac{dK(x)}{dx} \bigg|_{x=1}.$$

An example of the general phenomenology is shown in Fig. 1. Unlike the classic epidemic transition picture, where $\langle s \rangle$ diverges at the epidemic threshold where the macroscopic epidemic emerges, we now find two peaks in $\langle s \rangle$. This double transition is similar to numerical results from ref. 10 but here observed without the need for either strong clustering or heterogeneity in degree distribution. In fact, we used the homogeneous Poisson degree to ensure that the asymmetry in the transmission is the only salient feature of the model. Interestingly, as shown in Fig. 2, $T_c(1)$ and $T_c(2)$ are virtually equal for small values of the asymmetry. As asymmetry increases, the peak separates, thus yielding a double transition corresponding to an effective core-periphery organization in the network of infections. The core then corresponds to the men having sex with men (MSM) population, where infections are more frequent than in the remaining population. Fig. 3 shows the network of who infected whom for two values of $T$. For $T_c(1) < T < T_c(2)$, the extensive component is mostly composed of one type of nodes, and any spillover in the other types quickly dies out. However, at $T = T_c(2)$, these spillovers now cause cascades into other types with truncated power law-distributed sizes (Fig. 4). For $T > T_c(2)$, the extensive component recovers the well-mixed structure of the original underlying network.

Altogether, the second peak in the average size of outbreaks, $\langle s \rangle$, corresponds to a transition between subcritical and supercritical spillover in a less susceptible subpopulation but not to a second phase transition in the classic sense. Indeed, the analytical nature of our results allows us to confirm the null critical exponent observed in ref. 10 for the scaling of the height of the second susceptibility peak with regards to system size. Even in the infinite system considered by our calculations, the peak saturates, which is the only possible outcome for a system with an order parameter that is already nonzero. Interestingly, a critical power law-like behavior is nonetheless observed in the heterosexual population at both thresholds. Moreover, our results suggest that the asymmetry in transmission probability is reflected in the asymmetric prevalence within the male and female heterosexual populations, which is reminiscent of recent empirical results (6).

Based on our results, we can summarize the phase diagram of the ZIKV epidemic in three possible outcomes. First, with $T < T_c(1)$, all outbreaks are microscopic, quickly die out, and mostly infect MSM. Second, with $T_c(1) < T < T_c(2)$, we now see a macroscopic epidemic within the network of homosexual contacts between males, with microscopic spillover into the rest of the population via bisexual males. Third, with $T > T_c(2)$, we now find a more classic epidemic scenario in the sense that it is of macroscopic scale in most of the population. It is also worth mentioning that this phenomenology is robust to the presence of multiple infectious seeds sparking outbreaks (SI Appendix). Our results are thus valid beyond ZIKV for any infections with asymmetry in probabilities of direct transmission, regardless of whether there is also vector transmission.

**Discussion**

We developed a network model of ZIKV transmission highlighting the importance of asymmetric sexual transmission between males and females. We find a double transition generated by a core group of MSM that could maintain ZIKV transmission without the presence of a viable mosquito vector, such as in regions where people may have brought back ZIKV with them after a...
trip to endemic regions. These results are unique, because previous models showing double transitions relied on the need for strong clustering and heterogeneity in degree distribution.

Our study carries important consequences for the ongoing ZIKV epidemic and stresses the large knowledge gap in the sexual transmission of ZIKV (25). The aim of our work is to present the epidemiological consequences of possible sustained sexual transmission. While there are many unknowns, recent work shows (i) multiple anecdotal cases of sexual transmission of ZIKV in humans (25, 29–31), (ii) multiple separate animal models showing sexual transmission (32–34), (iii) strong asymmetries between durations of ZIKV shedding in semen and vaginal secretions (20, 21), and (iv) differential risk between sexes for ZIKV infection in sexually active populations. Indeed, recent work has identified 90% more ZIKV infections in women between 15 and 65 years old than in men of the same age in Rio de Janeiro (28) adjusted for gender-related health-seeking behavior and pregnancy status. Importantly, this risk difference was not seen in women <15 or >65 years of age, indicating the potentially large impact of sexual transmission of ZIKV in a country with known ongoing vectored transmission of ZIKV. A similar situation has also been observed in Colombia (35) and the Dominican Republic (36). Although more research on the epidemiological impacts and basic biology of sexual ZIKV transmission is needed, there is compelling need to be prepared with epidemiological studies examining transmission on a population scale.

We showed that potential ZIKV persistence in MSM, even if barely critical within that subpopulation, could cause subcritical but dramatic spillover into the heterosexual community. ZIKV
infections in adults are largely asymptomatic (37), and therefore, most testing occurs in the roughly 20% of cases that are symptomatic or individuals seeking to have children (38). The vast majority of these individuals will be outside of the MSM community (38). This means that the community most at risk is also one of the least likely to get tested. To avoid underestimating the spread of ZIKV, it is, therefore, important for health officials and policymakers to keep its unique behavior and phenomenology in mind.

Given the extent of foreign travel to locations endemic with ZIKV, public health practitioners should be aware of the potential for infectious introduction into local MSM communities. Travel history as well as sexual history should be used when evaluating an occult fever. Cities which have a viable vector for ZIKV should be doubly aware of the potential transmission routes of ZIKV. As it stands, current estimates of the basic reproductive number, R0, of ZIKV may be too low, because they fail to account for sustained sexual transmission (17, 39, 40). Important future work will be to accurately estimate R0 of ZIKV across various settings with differing sexual practices and mosquito fauna.

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