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Abstract

Human sexual networks exhibit a heterogeneous structure where few individuals have many partners and many individuals have few partners. Network theory predicts that the spread of sexually transmitted infections (STI) on such networks should exhibit striking properties (e.g. rapid spread). However, these properties cannot be found in epidemiological data. Current network models typically assume a constant STI transmission risk per partnership, which is unrealistic because it implies that sexual activity is proportional to the number of partners and that individuals have the same activity with each partner. We develop a framework that allows us to weight any sexual network based on biological assumptions. Our results indicate that STI spreading on the resulting weighted networks do not have heterogeneous-related properties, which is consistent with data and earlier studies.

Summary

Networks reflecting human sexual contacts over several years in a population have been shown to exhibit a high variance in the number of sexual partners per individual (Liljeros et al., 2001, Hamilton et al., 2008). The exact nature of the distribution of partners per individual is debated: some argue that it tends to follow a power-law (PL; Liljeros et al., 2001, Schneeberger et al., 2004, Hamilton et al., 2008), others argue that it tends to follow a negative-binomial (NB; Handcock and Jones, 2004, Hamilton et al., 2008). At any rate, both of these distributions are the appanage of highly heterogenous networks in which a few nodes in a network (i.e. individuals) are highly connected (i.e. have many partners), while the others nodes have few connections. Theory predicts that epidemics spreading on heterogeneous networks (either PL or NB) should exhibit specific properties such as a very low epidemic threshold, a rapid spread or in the case of PL, a doubling time that decreases with population size (Newman, 2003, Keeling and Eames, 2005). There is an ongoing debate in the literature concerning the relevance of these predictions for biological systems (especially the absence of epidemic threshold; May, 2006, Hamilton et al., 2008). For instance, specific epidemiological properties of highly heterogeneous networks are not observed generally in the data (Liljeros, 2004, Handcock and Jones, 2006, Britton et al., 2007, Hamilton et al., 2008).

Most network-based epidemiological studies consider unweighted networks. In the context of sexually transmitted infections (STI), it means that the risk of infection is the same on all the edges (i.e. for all the interactions) in the network (but see Eames et al., 2009). While these assumptions may be justifiable for diseases such as flu or tuberculosis that only require short-term interactions between hosts, they are unrealistic for STI. Here, we develop a new framework to add sex acts explicitly on sexual contact networks and show that such a biologically-relevant weighting has major epidemiological consequences.

Modelling the spread of STI on unweighted networks makes two implicit assumptions. First, the total number of sex acts of an individual per unit of time is assumed to be strictly proportional to his/her number of sexual partners. Second, these sex acts are assumed to be partitioned equally among all the partners. That individuals with 5 sexual partners generally have 5 times more sex acts per unit of time than individuals with one partner is unrealistic and contradicts empirical data (Blower and Boe, 1993, Nordvik and Liljeros, 2006, Britton et al., 2007).

Several studies point out the importance of weighted networks in general (see e.g. Barrat et al. (2004)'s seminal work). Some studies consider the spread of STIs on weighted networks of infinite size. Newman (2002) already showed that assuming that the transmission rate from one infected individual to another is not constant

but rather a function of node degree (i.e. number of partners) can affect disease spread. More precisely, he shows that for epidemics to spread rates of transmission need to fall off slower than inversely with node degree (independently of the network structure). Wang et al. (2007) approximate an infinite size so-called 'scale free' network with mean field equations and show that assuming that the transmission from one node to another is proportional to the node degree affects epidemic spreading. Joo and Lebowitz (2004) use a similar approach but consider a more elaborate transmission function, which they allow to saturate with increasing node degree. They find that such a saturation further decreases the speed of disease spread. Recently, Britton et al. (2011), study infinite size weighted sexual contact networks, where weights are drawn in a distribution that is allowed to depend on node degree. They show that if nodes (vertices) with high degree tend to have a low weight, then it is harder for an epidemics to take off. The two main limitations of these models is that they consider infinite size networks and/or that they do not consider any biological basis for the weighting they use. Note also that in most of these formalisms, the transmission rate from an individual A to an individual B differs from the transmission rate from B to A if they have different number of partners. In (Britton et al., 2011) the assumption is slightly different because a node can only be linked to a node that has the same weight (which means the weighting constraints the shape of the network).

More biologically-oriented approaches develop individual-based models to understand how sexual contact networks emerge (see e.g. Althaus et al., 2010) or pair-approximation models but without taking into account sex acts (Ferguson and Garnett, 2000). Among these studies, two consider the effect of sexual activity on the spread of an STI more explicitly. Röttingen and Garnett (2002) study the association between STI risk and number of sex acts without a network-based approach by fitting the relationship between the HIV transmission probability per-partnership and the number of sex acts. However, their model ignores epidemiological feedbacks, thus assuming for instance that all sexual partners of one individual have the same probability to be infected (whereas this probability should at least depend on the number of partner these partners have). More recently, Britton et al. (2007) model the spread of an STI in a heterogeneous population (in terms of number of sexual partners) where individual of each class can have both 'steady' or 'casual' partnerships. The latter partnerships have higher transmission probabilities than the former and the ratio of each type of partnership is inferred from empirical data on the number of sex acts per partner. They show that the basic reproduction number (R_0) can be over-estimated when different types of partnerships are not considered.

For sexual contact networks, the weight of an edge between two nodes corresponds to the number of sex acts that are actually realised between two individuals per unit of time (here weeks). In the following, we refer to these as realised sex acts (RSA). We derive the number of RSA between any interacting pair (i.e. all the weights of the network edges) from the network topology based on two simple biological assumptions. First, each individual is allocated a number of potential sex acts (PSA). PSA can be seen as a quantification of the sexual activity of an individual. Second, each individual partitions his/her PSA among his/her sexual partners. The number of RSA between two individuals i and j depends on the number of PSA that i attributed to j and vice versa.

We consider three models that describe the link between the number of PSA allocated to an individual and the number of partners of this individual: a linear relationship (the linear allocation model A_{lin}), a saturating relationship (the saturating allocation model A_{sat}), or a constant relationship where each individual receive the same number of PSA (the constant allocation model, A_{cst}). Classical models implicitly make the strong assumption that PSA are allocated linearly with the number of sex partners, which is at odds with empirical data (Blower and Boe, 1993, Nordvik and Liljeros, 2006).

In our study, PSA can then be partitioned by an individual among his/her partners in three ways: i) equally (P_{equ}), where all the partners get the same share, ii) randomly (P_{rand}), where the fraction each partner gets is random and iii) to maximise his/her number of RSA (P_{max}). Classical models implicitly assume an equal or a maximising partitioning, but the random partitioning arguably better reflects current sexuality, where the majority does not act in terms of maximising sex budgets (Foucault, 1976).

To the best of our knowledge, our allocation-partitioning model is first to present a biologically plausible relationship between total number of sex acts and total number of partners derived from explanatory behavioural hypotheses. In earlier studies (e.g. Newman, 2002, Joo and Lebowitz, 2004), this relationship is imposed without any mechanistic justification.

Our framework stands out compared to earlier studies because we use a finite size network inferred from contact tracing data, contrary to earlier models that are based on infinite 'scale-free' networks (Joo and Lebowitz, 2004, Wang et al., 2007) or that use degree distributions without explicit network topology (Röttingen and Garnett, 2002, Britton et al., 2007). This allows us to model explicitly how individual sexual behaviours affect the network weighted topology and, hence disease spread. Earlier studies that account for sex acts either do not include epidemiological feedbacks (Röttingen and Garnett, 2002) or only model a heterogenous population with two types of partnerships and therefore cannot keep track of individual behaviours (Britton et al., 2007). Our framework also allows us to model sex acts explicitly on any network type. This allows us to show that the underlying assumptions made when modelling disease spread on unweighted networks lead to correlation patterns between number of partners and number of sex acts that are at odds with empirical data (Blower and

Boe, 1993, Nordvik and Liljeros, 2006). Overall, we show that minimal deviations from implicit assumptions regarding sexual behaviour made in earlier models can strongly affect diseases spread. Adopting a more realistic (weighted) network affects epidemiological dynamics in a way that is consistent with epidemiological data on HIV. Furthermore, these results hold for theoretical power-law networks and negative-binomial networks.

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