Mathematical models of SIR disease spread with combined non-sexual and sexual transmission routes

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ABSTRACT

The emergence of Zika and Ebola demonstrates the importance of understanding the role of sexual transmission in the spread of diseases with a primarily non-sexual transmission route. In this paper, we develop low-dimensional models for how an SIR disease will spread if it transmits through a sexual contact network and some other transmission mechanism, such as direct contact or vectors. We show that the models derived accurately predict the dynamics of simulations in the large population limit, and investigate $\mathscr{R}_0$ and final size relations.

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1. Introduction

The recent emergence of Ebola (Christie et al., 2015; Mate et al., 2015) and Zika (Foy et al., 2011; Musso et al., 2015) demonstrates that diseases which spread primarily through other means can also have a sexual component to their spread.

Zika is a mosquito-borne virus which can cause birth defects if a pregnant woman is infected (Oliveira Melo et al., 2016; Ventura et al., 2016). Although more is being learned, it appears that Zika causes self-limiting infections (Duffy et al., 2009) and it is likely that infected individuals recover with immunity (Lessler et al., 2016).

Ebola is a directly transmitted disease which causes extreme morbidity and mortality (Baron, McCormick, & Zubeir, 1983; Emond, Evans, Bowen, & Lloyd, 1977; Heymann et al., 1980). It is spread through direct contact with bodily fluids from an infected individual. Individuals who survive appear to gain immunity (McElroy et al., 2015).

For both Zika and Ebola, evidence suggests that sexual transmission is possible (Christie et al., 2015; Foy et al., 2011; Mate et al., 2015; Musso et al., 2015). Further, it appears that individuals may be able to transmit the viruses sexually even after they appear to no longer be infectious through the usual mechanisms (Mackay, & Arden, 2015; Deen et al., 2015; Nicastri et al., 2016).

To better predict the spread of such diseases, we need to capture these different transmission modes into a model. Unfortunately, many existing mathematical models of disease spread through networks require a large number of equations (Kiss, Miller, & Simon, 2017; Miller & Kiss, 2014). However, in the specific case of Susceptible—Infected—Recovered (SIR) diseases such as Ebola and Zika, a low dimensional model, the Edge-based compartmental model, exists which can capture a diverse set of assumptions about the network structure (Miller, Slim, & Volz, 2012; Miller & Volz, 2013). However, it is

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structurally very different from the usual models used for other transmission mechanisms. Consequently it is not immediately obvious that we can combine the different transmission models into a single low-dimensional mathematical model.

Our goal in this paper is to develop simple mathematical models which capture a range of different transmission mechanisms combined with a sexual mode of transmission, and to provide enough examples to show how various assumptions can be combined into simple, low-dimensional models.

We begin by revisiting existing models for an SIR disease spreading through mass-action mixing and for an SIR disease spreading through a sexual contact network. We explore a number of models of the spread of an SIR disease through a sexual contact network combined with another transmission mechanism. In all of the models we build, we assume that the sexually infectious period lasts longer than the period of infectiousness through the other mechanism. It is straightforward to modify this assumption. We divide the models presented into two broad classes:

- We consider a random static sexual network (a "configuration model" network (Newman, 2003)) combined with some other transmission model, in particular
  - mass action mixing,
  - vector-borne transmission,
  - or social contact network.
- We then consider a simple mass-action transmission model combined with a more complex sexual transmission network including:
  - dynamic partnership changes
  - preferential mixing.

2. Models with configuration model networks

Throughout we assume that S, I, and R (and any subdivisions of these classes) represent the proportion of the population in the susceptible, infected, or recovered state. We assume the outbreak is initialized with a fraction ρ of the population chosen uniformly at random and infected at time $t = 0$.

2.1. The standard models

We briefly review the mass-action SIR model (Kermack & McKendrick, 1927) and the Edge-based compartmental model (Miller et al., 2012; Miller, 2011). Although these models appear structurally different, a simple change of variables shows that they are closely related. This close relation will allow us to combine mixtures of the models.

2.1.1. The mass-action model

We begin with the mass-action SIR model. An infectious individual transmits infectious doses as a Poisson process with rate β. Each infectious dose is received by an individual randomly chosen from the population. If the recipient is susceptible, then she becomes infectious. Infected individuals recover as a one-step Poisson process with rate γ. Once recovered they are immune to future infection. The diagram in the top-left of Fig. 1 leads us to the equations

$$\dot{S} = -\beta IS \tag{1a}$$

$$\dot{I} = \beta IS - \gamma I \tag{1b}$$

$$\dot{R} = \gamma I \tag{1c}$$

with initial conditions

$$S(0) = 1 - \rho$$
$$I(0) = \rho$$
$$R(0) = 0.$$

Fig. 2 shows that the solutions of system (1) accurately predicts the outcome of stochastic simulation in the large-population limit. The fit is excellent. Indeed, the equations correctly predict the large-population limit of the simulations. The convergence can be understood through the results of (Kurtz, 1971).

2.1.2. Edge-based compartmental model

For the simplest network-based model, we assume that a function $P(k)$ is known which gives the probability a randomly chosen individual has k partners (its *degree*). If we assume that partners are randomly chosen, then the probability a random partner of a random individual has degree k is $P_n(k) = kP(k)/\langle K \rangle$ where $\langle K \rangle = \sum kP(k)$ is the average degree.