Simulation of epidemic dynamics based on the effective contact process

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1 Introduction

The stochastic model described hereunder aims at representing the epidemic dynamic of an infectious disease that spreads within a population. In particular, the model focuses on the description of the infectiousness over the course of the infectious period, accounting for features that affect and modify the probability of observing infection events.
Infectious diseases spread through interactions between hosts, called transmission routes or contacts, which are pathogen-specific and depend on the specific host population. Not all of these interactions lead to observe infection events, depending on the couple of hosts having such a contact and on the infecitivity and susceptibility level. In addition, the contact rates can vary over the course of the infectious period: the onset of symptoms can cause infectives to change their normal behavior reducing the number of social interactions (e.g., staying at home)[1]. Therefore, it is important to distinguish between the contact behavior, and a viral component.
The infectivity measures, defines the probability that an individual i infects a specific individual j when i and j are having a contact. We assume this function to be time-dependent and we represent the shape of such a curve using viral load observation. An important assumption in this model is that the higher is the viral load value, the higher is the infectivity measure. Since the infectivity measure can be defined on specific couple (i, j) it is possible to include other features that can characterize the transmission probability given a contact, e.g. risk perceived by j after noticing the onset of symptom in i.
The present model aims at establishing a framework in which components that could affect either the infectivity, or the contact rate, can be included. It is flexible, allowing to track changes at the individual level and it can easily be adapted to include the desired components. The stochastic nature makes the model suitable to investigate the aforementioned effects in small populations. Complexity makes the code computationally intense and requires a careful implementation to represent correctly the desired dynamic.

2 Theoretical description of the model

Consider a specific pathogen and the underlying contact network in a closed population of size n. Let i, j be distinct individuals in this population that have contact between themselves. We assume that the contact process among i and j is described by a Poisson Process of rate $r_{i,j}$. When one of the two, say i, becomes infectious, at time t_i, he or she can spread the infection

to j, as well as to all the other individuals is in contact with. Contacts, are accepted, i.e. lead to an infection event, according to a probability value that is the product of the infectivity measure $\nu_{i,j}(t)$, i.e. which quantifies the level of infectivity over the infectious period, and the total infectivity $q_{i,j}$, i.e. the proportion of the average number of effective contacts over the daily contact rate [2]. The former quantity is a positive measure with unitary mass along the infectiousness period while the latter is assumed to be a constant value. If individual j, at the time of the contact, is not susceptible, this probability has value zero. In this terms, the effective contact between i and j results to be described by a non-homogeneous Poisson process of rate $r_{i,j}q_{i,j}\nu_{i,j}(t)$. The mean number of effective contact between i and j is thus given by:

$$\bar{\mathcal{R}}_0 = \int_0^{\mu_i} r_{i,j}q_{i,j}\nu_{i,j}(t)dt = r_{i,j}q_{i,j}$$

where μ_i is the length of the infectious period of individual i. For a fixed value of the contact rate, the implementation is correct as long as $q_{i,j}\nu_{i,j}(t) \leq 1$ for every time t. This mathematical description is also known as thinning of a Poisson Process. We assume that the effective contact processes among i and the other members of the population are independent. Therefore, the rate at which i makes effective contacts is:

$$\sum_{j \neq i} r_{i,j}q_{i,j}\nu_{i,j}(t)$$

Note that this framework allows to define different contact rates between different individuals as well as different acceptance probability functions. The mean number of effective contact individual i makes during the infectious period is given by:

$$\bar{\mathcal{R}}_0 = r_{i,j}q_{i,j} \tag{1}$$

We remark here that the number of effective contacts is an approximation of the basic reproduction number. In fact, in the former cases effective contacts could be realized with the same person. The two quantities coincide when the population is infinite and individuals are contacted with the same probability. In this case, the probability of contacting two time the same individual is zero.

2.1 Mass-action model

In a mass-action model the population is assumed to be homogeneous and the contact processes to be independent. The contact rate is scaled by the population size, i.e.

$$r_{i,j} = \frac{\bar{r}}{n-1}$$

According to this formulation, the expected number of effective contacts an infectious individual makes is given by:

$$\bar{\mathcal{R}}_0 = rq \tag{2}$$

We remark here that this formulation is valid for every distribution of the infectious period length, since the infectiousness measure has unitary mass along the infectious period of each individual.

2.2 Infectivity measure as viral load function

With this model we describe the epidemic dynamic of infectious diseases. At first, we assume that the infectiousness measure $\nu(t)$ resembles the shape of the viral load curve. This assumption is motivated by the fact that a virus, before establishing in a host individual, has to overcome the external immune system. The higher is the viral load, the higher the amount of virus spreads in a contact with a consequent higher probability of observing an infection event. However, as mentioned before, perceive risk by susceptibles or antiviral can affect the infectiousness measure. This is model substituting the original infectivity measure $\nu(t)$ that represents the viral load, with another infectiousness measure $\bar{\nu}(t)$ that describes the decrease thereof. If the change in the infectivity measure happens at time t^* for all individuals independently from the length of the infectious period, the mean number of effective contact is given by

$$\bar{\mathcal{R}}_0 = \int_0^\infty \left[\int_0^{t^*} rq\nu(t)dt + \int_{t^*}^{s} rq\bar{\nu}(t)dt \right] f(s)ds$$

where $f(s)$ is the infectious length density function.
An example of a change in the infectiousness measure is given in the simulation scripts in that account for the effect of an antiviral.

2.3 Decrease of contact rate due to control measures

Next to a decrease in the infectivity measure, a decrease in the contact rate reduces the expected number of secondary cases. When isolation or quarantine are included, the contact rate decreases accordingly to the efficacy of the control measure. Assume that a change in the contact rate, from r to $\bar{r}$ happens at time t^* for all the individuals, and the infectiousness measure is always the same. In this case the mean number of effective contacts results to be:

$$\bar{\mathcal{R}}_0 = \int_0^\infty \left[\int_0^{t^*} rq\nu(t)dt + \int_{t^*}^{s} \bar{r}q\nu(t)dt \right] f(s)ds$$

where $f(s)$ is the distribution of the infectious period length. An example of the reduction in contact rate is given in the scripts that account for isolation and quarantine measures.

References

[1] P. H. Rhodes, M. E. Halloran, and I. M. Longini Jr. Counting process models for infectious disease data: distinguishing exposure to infection from susceptibility. *Journal of the Royal Statistical Society: Series B (Methodological)*, 58(4):751–762, 1996.

[2] Å. Svensson. A note on generation times in epidemic models. *Mathematical biosciences*, 208(1):300–311, 2007.