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Basic Reproduction Number in a Growing Population

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Abstract The basic reproduction number of a fast disease epidemic on a slowly growing network may increase to a maximum then decrease to its equilibrium value while the population increases, which is not displayed by classical homogeneous mixing disease models. In this paper, we show that, by properly keeping track of the dynamics of the per capita contact rate in the population due to population dynamics, classical homogeneous mixing models show similar non-monotonic dynamics in the basic reproduction number. This suggests that modeling the dynamics of the contact rate in classical disease models with population dynamics may be important to study disease dynamics in growing populations.

Keywords Basic reproduction number, Population dynamics.

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1. Introduction

Contact networks are graphs representing the person-to-person contact structure in a population, in which the nodes represent individuals and the edges represent contacts (see, e.g., [1] and [8, Chapter 17]). They are more realistic than homogeneous mixing models to describe the heterogeneous and long-term nature of human contacts. Not surprisingly, they give predictions that cannot be easily reproduced in homogeneously mixing models. For example, on networks, the Susceptible-Infectious Susceptible models (or SIS, for diseases without acquired immunity) have a larger basic reproduction number than that of the Susceptible-Infectious-Removed models (or SIR, for diseases with lifetime acquired immunity) with the same disease and contact parameters [7], due to that fact that the long-term contacts allows multiple transmissions along an edge for SIS models, but not for SIR models. In addition, network SIS models introduces correlations in the infection status of neighboring nodes [4]. These cannot be observed in homogeneous mixing models.

Interestingly, Yuan et al. [10] shows another feature of network disease models, that the basic reproduction number $\mathcal{R}_0$ may display non-monotonic behavior in a growing population, i.e., $\mathcal{R}_0$ may increase to a maximum then decrease to an equilibrium value while the population increases to an equilibrium. There have been extended studies in homogeneous mixing disease models with population dynamics

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(see, e.g., [5,6] and [2, Section 10.2]). These studies assume that the per-capita contact rate either is a constant, or scales monotonically with the population size. Yet, none has shown such non-monotonic dynamics in the basic reproduction number as predicted by the network models.

We conjecture that this non-monotonic behavior is the result of precise counting of contacts in networks models, which has always been neglected in homogeneous mixing models. Specifically, for a population with N individuals and a per capita contact β, i.e., a total contact rate if βN in the population, when one individual leaves, his/her contacts are also removed. Because contacts are mutual, this must cause the peers of his/her contacts to have a lower contact rate. On the other hand, if one extra individual comes in with a contact rate β, he must contact others who already have a contact rate β, and thus increase their contact rate. We propose that if we properly count for the change in per capita contact rate by counting the dynamics of total contact rate in the population, then the homogeneous mixing models will show similar non-monotonic behavior in $\mathcal{R}_0$.

In Section 2, we construct a mathematical model that incorporates the dynamics of the per capita contact rate due to a slow population dynamics, and study the basic reproduction number of a fast disease dynamics (during which the population size can be regarded as a constant). This model is analyzed in Section 3 and is shown to have a non-monotonic dynamics for $\mathcal{R}_0$. Concluding remarks are given in Section 4.

2. Model

The main goal of this paper is to model the change of the per capita contact rate in a growing homogeneously mixed population. To do so, we assume a simple population dynamics: the births (or immigrations) are assumed to be a constant λ, and the per capita death rate μ is also a constant. Thus, the population size $N(\tau)$ at time τ can be modeled as

$$\frac{dN}{d\tau} = \lambda - \mu N\,.$$

Here we model the total contact rate in the population at time τ, $C(\tau)$, and then compute the per capita contact rate $\beta(\tau)$ from $C(\tau)$ as

$$\beta(\tau) = \frac{C(\tau)}{N(\tau)}\,,$$

because we assume a homogeneous population, and thus every individual has the same per capita contact rate at time τ. This idea is well summarized in [3, Page 33].

Assume that each incoming individual brings in β_0 contacts per unit time, to random individuals in the population. Thus, the total $\lambda\beta_0$ incoming contact rate causes an increase of the same amount in the total contact rate $C(\tau)$ among the original individuals the population. Thus, $C(\tau)$ increases by $2\lambda\beta_0$ per unit time. On the other hand, when an individual leaves (or dies), he/she takes away his/her $\beta(\tau)$ contacts per unit time, and at the same time, causing a decrease of $\beta(\tau)$ in the total contact rate $C(\tau)$ among the remaining individuals. Thus the total rate of contacts in the population decreases by an amount

$$2\mu N(\tau)\beta(\tau) = 2\mu C(\tau)\,.$$

per unit time. In summary,

$$\frac{dC}{d\tau} = 2\lambda\beta_0 - 2\mu C\,.$$

This gives us the dynamics of the per capita contact rate $\beta(t)$:

$$\frac{d\beta}{d\tau} = \frac{d}{d\tau}\left(\frac{C}{N}\right) = \lambda\frac{2\beta_0 - \beta}{N} - \mu\beta\,.$$

We assume that the disease dynamics is much faster than the population dynamics, i.e., during an epidemic, the population can be regarded constant. For simplicity, and to compare with the network model results, we assume an SIR disease dynamics. In addition, because of the assumed separation of timescales, the births and deaths are neglected during the disease epidemic. Assuming that the disease invades at time τ in the slow (population dynamics) timescale, which corresponds to time $t = 0$ in the fast (epidemic) timescale. Letting $S(t)$, $I(t)$, an $R(t)$ be the number of susceptible, infectious and recovered individuals in the population at time t, respectively, the SIR model can be written as

$$\frac{dS}{dt} = -\beta(\tau)\frac{SI}{N}\,, \tag{2.1a}$$

$$\frac{dI}{dt} = \beta(\tau)\frac{SI}{N} - \gamma I\,, \tag{2.1b}$$

$$\frac{dR}{dt} = \gamma I\,, \tag{2.1c}$$

where γ is the recovery rate (i.e., the infectious period is exponentially distributed with mean $1/\gamma$). The basic reproduction number $\mathcal{R}_0(\tau)$ for a disease introduced at time τ of the slow timescale, can be computed as [9]

$$\mathcal{R}_0 = \frac{\beta(\tau)}{\gamma}\,, \tag{2.2}$$

which is proportional to $\beta(\tau)$. That is, the dynamics of $\beta(\tau)$ completely determines that of $\mathcal{R}_0(\tau)$. Specifically, we choose the proper timescale so that $\gamma = 1$, i.e., the unit time in the disease dynamics is the mean infectious period, then $\mathcal{R}_0(\tau) = \beta(\tau)$. We thus use $\beta(\tau)$ and $N(\tau)$ to describe the dynamics of the basic reproduction number $\mathcal{R}_0(\tau)$ for a disease introduced at time τ of the population growth process, which can be summarized as

$$\frac{dN}{d\tau} = \lambda - \mu N\,, \tag{2.3a}$$

$$\frac{d\beta}{d\tau} = \lambda\frac{2\beta_0 - \beta}{N} - \mu\beta\,. \tag{2.3b}$$

3. Analysis

The system (2.3) has a unique equilibrium ($N^* = \lambda/\mu, \beta^* = \beta_0$).

To analyze the dynamics of (2.3), non-dimensionalize the system with the transformation $s = \mu\tau$, $N = N^*x$, $\beta = \beta^*y$, giving

$$\frac{dx}{ds} = 1 - x\,, \tag{3.1a}$$

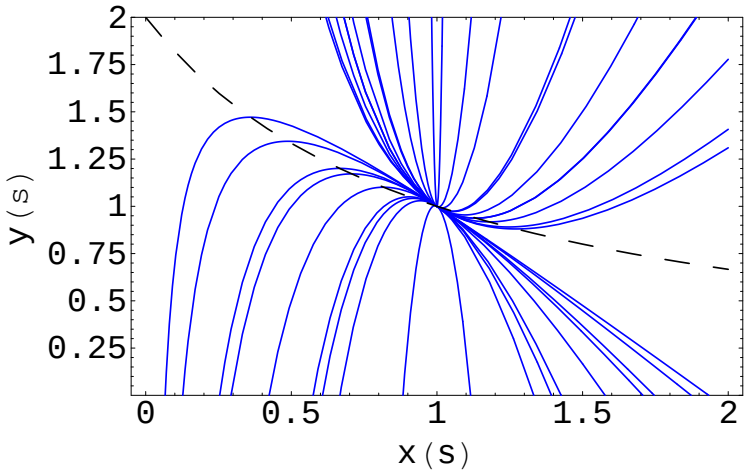

Figure 1. The phase plot of the non-dimensionalized model (3.1). The solid lines are trajectories starting from different initial conditions, and the dashed line is the y-nullcline of (3.1). The scaled average contact rate $y(s)$ first increases to a maximum then decreases to its equilibrium if the initial population size $x(0)$ is less than the equilibrium population size.

$$\frac{dy}{ds} = \frac{2}{x} - (\frac{1}{x} + 1)y\,, \tag{3.1b}$$

with an equilibrium $(x^* = 1, y^* = 1)$.

The phase plot of this simple system is illustrated in Figure 1 for different initial conditions. This system is solvable analytically. With the initial condition $x(0) = x_0$, (3.1a) gives

$$x(s) = 1 + (x_0 - 1)e^{-s}\,, \tag{3.2}$$

which can then be then substituted in (3.1b), together with $y(0) = y_0$,

$$y(s) = \frac{1}{1 + (x_0 - 1)e^{-s}} + (x_0 y_0 - 1)\frac{e^{-2s}}{1 + (x_0 - 1)e^{-s}}\,. \tag{3.3}$$

Note that, with $0 < x_0 < 1$, $x(s)$ monotonically increases to $x = 1$. On the other hand, dy/ds is positive for $2 > (x + 1)y$ and negative for $2 < (x + 1)y$. Thus with $y_0 < 1$, $y(s)$ may either increase monotonically to reach a maximum on $(x+1)y = 2$ then decrease to $y = 1$ or increase monotonically to $y = 1$. Consequently, the behavior of $y(s)$ is uniquely determined by the sign of dy/ds about the equilibrium $(1, 1)$. As $s \to \infty$,

$$\frac{dy}{ds} = \frac{(x_0 - 1)e^{-s}}{[1 + (x_0 - 1)e^{-s}]^2} + o(e^{-s})\,.$$

Hence, if $x_0 < 1$, then $\frac{dy}{ds} < 0$ about the equilibrium, and thus $y(s)$ reaches the equilibrium from above. In this case, $y(s)$ reaches its maximum with above $y = 1$ and then decreases to $y = 1$. On the other hand, if $x_0 > 1$, then $y(s)$ increase monotonically to $y = 1$.

This is the same kind of dynamics on basic reproduction number that we observed in network models in [10].

4. Concluding Remarks

In the introduction we have conjectured that the non-monotonic dynamics of the basic reproduction number as a function of the invasion time in a growing network is not observed in a homogeneous population, because network models keep tract of the dynamics of the contacts more precisely. On the other hand, popular homogeneous mixing models naively assume that the per capita contact rate either remains constant or scales non-monotonically with the population size in a changing population. We have also hypothesized that, by properly tracking the dynamics of contact rate in the population, the same type of non-monotonic dynamics in the basic reproduction number will emerge in homogeneous mixing models.

To prove these, we formulated a simple homogeneously mixing population model that keeps track of the dynamics of the per capita contact rate by counting each contact that an incoming individual brings per unit time, and each contact that an outgoing individual takes away per unit time. For a simple SIR epidemic model on a fast timescale (so that the population remains approximately constant), this per capita contact rate is the basic reproduction number with a proper time unit (the mean infectious period).

The basic reproduction number shows non-monotonic behavior as the population size increases to its equilibrium as long as the initial population size is less than its equilibrium (i.e., in a growing population). This is in analogous to the uniform attachment scheme in a growing network, but unlike the preferential attachment scheme which requires that the initial total contacts is only a small fraction of the total equilibrium contacts [10].

Assuming that individuals in the population have a constant contact rate $\beta(\tau)$ is an over simplification. It would be an interesting extension to study the dynamics of the distribution of the contact rates in the population while still assuming homogeneous mixing. The resulting model will be a partial differential equation on $N(\tau, \beta)$ for the number of individuals with contact rate β at time τ.

In summary, keeping track of the change of the per capita contact rate in classical homogeneous mixing disease models may be necessary for understanding the invasion risks of emerging diseases such as Ebola and Zika virus in growing populations.

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