A Discrete SIRS Model with Kicked Loss of Immunity and Infection Probability

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Abstract. A discrete-time deterministic epidemic model is proposed with the aim of reproducing the behaviour observed in the incidence of real infectious diseases, such as oscillations and irregularities. For this purpose we introduce, in a naïve discrete-time SIRS model, seasonal variability in the loss of immunity and in the infection probability, modelled by sequences of kicks. Restrictive assumptions are made on the parameters of the models, in order to guarantee that the transitions are determined by true probabilities, so that comparisons with stochastic discrete-time previsions can be also provided. Numerical simulations show that the characteristics of real infectious diseases can be adequately modeled.

1. Introduction

The dynamics of epidemics are generally based on the SEIR or SIR models [1], in which the host population is partitioned into categories containing susceptible, infected but not yet infectious (exposed), infectious, and recovered individuals. These models have endemic asymptotically stable equilibria or the disease dies out. Because oscillations are observed in the incidence of many infectious diseases (such as measles, mumps, rubella and influenza), it is of interest to determine how oscillating solutions can arise in epidemiological models.

Usually, epidemic models are analysed in the field of continuous systems. In the deterministic framework, periodic solutions exist if the transmission rate is allowed to vary seasonally or spatial heterogeneity is included [2, 3]. It is also suggested that non linear incidence rate [4], temporary immunity (through which an individual’s return to susceptibility can be delayed) [5], the introduction of a quarantine class [6], a constant removal rate of the infective individuals [7] are mechanisms that can induce oscillations in the occurrence of diseases.

Discrete models have also been used to study infectious disease dynamic. Discrete-time models are useful for several reasons. The discrete time-step approach is justified because, on one hand, it is an approximation to the continuous time case, while on the other hand, our every day life has a certain periodicity, e.g., seasonal changes. Moreover, it provides a substantial reduction of computational time, and the entire collection of tools developed for the study of maps can be advantageously used.

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In discrete-time deterministic SIRS models periodic solutions are found when weak conditions on the force of infection are assumed [8], or the models parameters are not let to vary within realistic ranges of values [9], or unreasonable discrete-time steps are introduced [10]. In more realistic approaches, the interactions within the population are described by small world networks [11], and/or dynamics are described by delay difference equations [11,12]. In the small word networks approach [11], for a finite value of the disorder of the network (ranging from ordered lattices to random graphs), a transition from a stationary state (fixed point, with fluctuations) to self-sustained oscillations in the size of the infected population was found. In the time-delay approach [12], individuals spend a fixed amount of time in a generalized immune class; and oscillations occur for a specific ranges of immunity duration and infected individual exposure.

The conditions that lead to periodicity in the infectious disease was also investigated in reference [13] by a naïf discrete SIRS model, and it was found that the epidemic oscillates when a small fraction of individuals became not permanently immunised. In fact, the inclusion in a SIR model of a positive feedback, from the removed class to a susceptible one, in a very narrow range of the corresponding control parameter, has the effect of periodically enhancing the spread of disease. The smaller is the probability that a recovered becomes susceptible, generally the larger is the period of the oscillations in the infected population. This observation together with results in [11,12] suggest that oscillations can be the result of two factors: the manner in which the infection takes place, and residence time in the class of immunized.

In this paper we add to the discrete SIRS model of ref. [13] a seasonal variability, introduced by means of a sequence of kicks, which change periodically (i) the fraction of recovered individuals, which lose immunity in the sampling intervals, and/or (ii) the infection probability. Our goal is understanding and reproducing the oscillations of real diseases propagation. The simplicity of the model makes it possible to easily verify the correctness of above assumptions.

The outline of the paper is the following. We introduce the SIRS model in Sec. 2. In Sect. 3 seasonality in the loss of immunity parameter is introduced by means of a sequence of kicks. We discuss dynamical properties of the system when the infection probability is perturbed, in Sect. 4. Finally, we give conclusions in Sect. 5.

2. Epidemic SIRS model

We consider a population consisting of susceptible (S), infected (I) and recovered (R) individuals. As a consequence, the total population is

\[ N = S + I + R. \]  

(1)

Susceptible elements can pass to the infected state through contagion by an infected one. Infected elements can pass to the recovered state, and recovered elements can return to the susceptible state by loss of immunity. This kind of system is usually called SIRS, for the cycle that a single element goes over. The contagion is possible only during the S phase, and only by an I element. During the R phase, the elements are immune and not infectious.

SIRS models are excitable systems known to display relaxation oscillations in mean field or well-mixed approaches. In spatially extended versions space-time oscillations can occur, due to the interaction between neighbouring elements.

The basic discrete-time deterministic (SIRS) model has the form

\[
\begin{align*}
S_{n+1} &= qS_n + cR_n \\
I_{n+1} &= (1-q)S_n + bI_n \\
R_{n+1} &= (1-c)R_n + (1-b)I_n
\end{align*}
\]  

(2)

where, during each sampling interval \( n \), \( q \) denotes the probability that a susceptible avoids the infection, \( b \) is the proportion of individuals which remains infected (\( 0 \leq b \leq 1 \)), \( c \) (\( 0 \leq c \leq 1 \)) the fraction of recovered individuals, which lose immunity.
The probability $q$ can be modelled in different ways [13]. The probability $q$ is an arbitrary function $0 \leq q(S, I) \leq 1$ with the property $q(S, 0) = 1$. It depends on the particular form of propagation of the disease. Let us denote by $p$ ($0 < p < 1$) the probability of the infection effectively transmitting through a time of contact. We assume the population has a homogeneous spatial distribution and the probability per unit time of a contact between two individuals is the same for any pair. Then $I_n / N$ (proportion of infected in a population) represents the probability that an infective individual is in contact with a susceptible, and $p I_n / N$ is the probability that a susceptible remains infected. As a consequence we obtain the following expression

$$q = 1 - pI / N$$

for the standard probability that a susceptible avoids the infection.

2.1. Periodic incidence of the disease

Let us consider the propagation of an infection on a population of constant size $N$ in the SIRS model with the standard probability (3). When $c = 0$, the epidemic equations reduce to the SIR equations ones. So $c \neq 0$ was used throughout what follows.

By utilizing the relation (1) and inserting equation (3) in the system (2) one obtains

$$S_{n+1} = (1 - pI_n / N)S_n + c(N - S_n - I_n)$$
$$I_{n+1} = (pS_n / N + b)I_n$$

The system has two fixed-point:

$$S^* = N, I^* = 0,$$

$$S^* = N \frac{1-b}{p}, I^* = N \frac{c(p+b-1)}{p(1-b+c)}.$$

![Figure 1.](image.png)

**Figure 1.** a) Fraction of susceptible, infected and recovered individuals as a function of time for $p = 0.8$, $b = 0.5$ and $c = 0.01$. An initial fraction of 0.1 infected, and the rest susceptible, was used. The system evolves through damped oscillations toward the endemic equilibrium (6). b) Magnification of infected population time series.

The fixed point (5) is stable if $p + b < 1$, otherwise it is unstable. Quite the opposite, the fixed point (6) is stable if $p + b > 1$, otherwise it is unstable. Thus, the fixed points change their stability on different sides of $p + b = 1$, and the system undergoes a transcritical bifurcation [13]. So, for $p + b > 1$ the dynamics correspond to that of an endemic infection, with a persistent fraction of infected individuals.
Typical time series are shown in figure 1. An initial fraction of 0.1 infected, and the rest susceptible, was used in all the results shown here.

In reference [13] it is shown that for each fixed pair of values $p$ and $b$ such that $p + b > 1$, there exists a value $c_M$ of $c$ such that oscillations are observed in the infected population, if we take $0 < c < c_M$. By reducing the value of $c < c_M$, periodic incidence of the disease is observed at higher values of time (figure 2).

These results highlight the importance of properly modelling the “‘loss of immunity’” step, for observations of periodic epidemic outbreaks. A second issue is obviously the mode of infection spread between infected and healthy individuals.

![Figure 2. As in figure 1, but for $c = 0.001$.](image)

We wish to insert some degree of variability both in the recovery parameter $c$ and in the infection probability $p$. Epidemiological justifications can, for example, reside in the immunity memory response or pathogen mutation effects. As we are mainly interested in introducing some form of discontinuity in the dynamics, we merely add a perturbation in the form of a series of periodic kicks.

### 3. Perturbation of the recovery class

We evaluate now the hypothesis that the oscillations occur if the duration of immunity has a limited duration or if the fraction of recovered individuals suddenly change over time. In our model this can be easily simulated by perturbing the fraction of recovered individuals which lose immunity by a sequence of kicks with amplitude $k$ and period $t$. We therefore assume that

$$c = c_0 + k\delta_{n,t}.$$  \hspace{1cm} (7)

We start by considering

$$\delta_{n,t} = \begin{cases} 1 & \text{if } n/t = \text{integer} \\ 0 & \text{if not} \end{cases}.$$  \hspace{1cm} (8)

Under these assumptions, the system’s dynamics are quite different. Most notably, as the parameter $k$ increases from 0, sustained oscillations emerge in time series. The plots of figure 3 was obtained by increasing $c_0$ of 0.3 one time step every 100 time steps. Calculations show also that at high values of $k$ the amplitude of the oscillations becomes larger.

The time series is periodic, with an almost defined period (equal to $t$) and negligible variation in amplitude. Epidemiologically, this behaviour is qualitatively similar to the epidemic patterns typical of diseases such as influenza [14].

The mechanism, by which the oscillations emerge, is easily explained: while the fraction of the population that is infected is close to a steady state, the transition from immune to susceptible class of
a suitable number of individuals increases the possibility of infection, and consequently the size of epidemic raises. Then the system moves back toward the steady state and the process is repeated.

Figure 3. a) Fraction of susceptible, infected and recovered individuals as a function of time for $p = 0.8$, $b = 0.5$ and $c_0 = 0.01$. The fraction of recovered, which in each time-step are returned to the susceptible class, is perturbed by a sequence of kicks, as described by equation (8). Other parameters are $k = 0.3$, $t = 100$. Self-sustained oscillations develop. b) Magnification of infected population dynamic.

The shape of the oscillations depends on the values of $c_0$ and $k$, the period depends on $t$. In figure 4 a) simulations are shown for different values of these parameters. The number of infected individuals grows with $c_0$.

Figure 4. a) Fraction of infected individuals as a function of time for $p = 0.8$, $b = 0.5$, $t = 100$, and different values of $c_0$ and $k$. b) As in a), but with $k = 0$.

Increasing the value of $c_0$, the system returns faster to steady state corresponding to $k = 0$, so that the peaks in the size of the infected population appear separated in time. While increasing the value of $c_0$ involves oscillations with peaks above the steady states, his decrease works in the opposite direction, and the oscillations are a result of a decrease in the number of infections compared to the value of the steady states, which are shown in figure 4 b), for a comparison. The $k$ value determines the height of the peaks, $t$ the oscillations period. Decreasing only $t$, a situation equal to that of figure 4 a) is restored, but with smaller oscillations periods.

The kicks can be continued for more time steps. In this case the kicking is modelled as follows:
\[
\delta_{n,t,s} = \begin{cases} 
1 & \text{for } n/t, n/t + 1, \ldots, n/t + s - 1 (n/t, s = \text{integer}) \\
0 & \text{otherwise}
\end{cases} 
\tag{9}
\]

where the parameter \(s\) is the number of consecutive times that a kick works (duration of kicks). For \(s = 1\) equation (9) becomes equivalent to equation (8) (1 kick every \(t\)).

Figure 5. \(a)\) The dynamics of infection as a function of the number of time steps, for different values of \(s\), when formula (9) is used for kicking. Parameters are: \(p = 0.8\), \(b = 0.5\), \(t = 100\), \(c_0 = 0.01\), \(k = 0.1\). \(b)\) An enlargement.

Figures 5 illustrate the effect of kicking during \(s\) successive time steps. The dynamics of infection as a function of the number of kicks \(s\) are the following: by increasing \(s\) also the time interval of persistence of the infection increases; starting from about \(s = t/2\), increasing still \(s\) a secondary minimum occurs immediately after the primary one and the oscillations can have a plateau, which stabilizes around the steady state corresponding the value \(c = c_0 + k\).

4. Perturbation of the probability of infection

We now address the second hypothesis that links the occurrence of oscillations to changes in the probability of infection. For this purpose we introduce perturbations similar to the previous, but this time referring to \(p\):

\[
p = p_0 + k\delta_{n,t,s}
\tag{10}
\]

where \(\delta_{n,t,s}\) is given by (9).

Numerical calculations with \(s = 1\) show that the discontinuity introduced on the infection probability also produces periodic oscillations of period \(t\) (figure 6). The fraction of infected has a peak in correspondence of each kick, (i.e. when \(\delta_{n,t,s} = 1\)). Besides, the average number of infected elements increases with \(c\).

At constant values of \(c\), variations in the value of the parameter \(s\) produce two main effects: a change of peaks amplitude and a shifting or a frequency variation of the time series between the peaks. An example of this behaviour is reported in figure 7. From the epidemiological point of view, different infection patterns can be obtained qualitatively similar to the real ones.

Therefore, the duration \(s\) of the kick is an important factor, affecting the structure of the temporal evolution of the epidemic. Indeed, reversing symmetrically the duration of the kicks/no kicks time steps (with \(p_0 = 1.0\), \(k = -0.2\), you get again, apart from an initial phase, the pattern of figure 7.
The relevant fact that emerges from the preceding discussion is that the basic phenomenon that underlies on the presence of oscillations in the dynamics is the discontinuity in $p$. This is also confirmed by the result shown in the figure 8, where for the discontinuity the value $p = 0$ was used.

5. Conclusions
To understand the evolution of many infectious disease systems, we introduced the effects of seasonality in a discrete SIRS model. We have shown that the introduction of seasonal forcing in loss of immunity and in transmission rate can well explain the periodicity observed in the outbreaks of diseases.
We have quantified the effect of contact rate and immunity duration on oscillatory behaviour by modelling their variability by means of sequences of kicks, acting on the corresponding model parameters.

Epidemiologically, oscillations in the number of infected individuals in a population could be due essentially or to a mutating pathogen or to a seasonal enhancement of the infection probability. Model behaviors are qualitatively similar to the epidemic patterns typical of diseases such as influenza [14]. Moreover, they emphasize the importance of the interplay between seasonal infection and recruitment and suggest that our simulation patterns are meaningful in different epidemic scenarios.

6. References
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