Susceptible-infectious-recovered models revisited: From the individual level to the population level

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ABSTRACT

The classical susceptible-infectious-recovered (SIR) model, originated from the seminal papers of Ross [51] and Ross and Hudson [52,53] in 1916–1917 and the fundamental contributions of Kermack and McKendrick [36–38] in 1927–1932, describes the transmission of infectious diseases between susceptible and infective individuals and provides the basic framework for almost all later epidemic models, including stochastic epidemic models using Monte Carlo simulations or individual-based models (IBM). In this paper, by defining the rules of contacts between susceptible and infective individuals, the rules of transmission of diseases through these contacts, and the time of transmission during contacts, we provide detailed comparisons between the classical deterministic SIR model and the IBM stochastic simulations of the model. More specifically, for the purpose of numerical and stochastic simulations we distinguish two types of transmission processes: that initiated by susceptible individuals and that driven by infective individuals. Our analysis and simulations demonstrate that in both cases the IBM converges to the classical SIR model only in some particular situations. In general, the classical and individual-based SIR models are significantly different. Our study reveals that the timing of transmission in a contact at the individual level plays a crucial role in determining the transmission dynamics of an infectious disease at the population level.

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1. Introduction

Mathematical modeling in epidemiology started with the pioneering work of Bernoulli [10] in 1760 in which he aimed at evaluating the effectiveness of inoculation against smallpox. The model of Bernoulli described the susceptible and recovered classes and already incorporated the chronological age of individuals (see [20,21]). The susceptible-infectious-recovered (SIR) model as we know today takes its origin in the fundamental works on "*a priori* pathometry" by Ross [51] and Ross and Hudson [52,53] in 1916–1917 in which a system of ordinary differential equations was used to describe the transmission of infectious diseases between susceptible and infected individuals. In 1927–1933, Kermack and McKendrick [36–38] extended Ross's ideas and model, proposed the cross quadratic term βIS linking the sizes of the susceptible (S) and infective (I) populations from a probabilistic analysis of the microscopic interactions between infective agents and/or

vectors and hosts in the dynamics of contacts, and established the threshold theorem. Since then epidemic models have been extensively developed in several directions, we refer to the monographs of Bailey [7], Bartlett [9], Muench [45], Anderson and May [4], Busenberg and Cooke [13], Capasso [14], Murray [46], Daley and Gani [16], Mode and Sleeman [47], Brauer and Castillo-Chavez [11], Diekmann and Heesterbeek [19], Thieme [59], and Keeling and Rohani [35] on these topics.

In order to focus on the dynamical properties of an infectious disease itself, here we neglect the demography, namely the birth and death processes, and the immigration/emigration process. The classical SIR model takes the following form [4]:

$$\begin{cases} S' = -\beta \frac{SI}{N} \\ I' = \beta \frac{SI}{N} - \eta_R I \\ R' = \eta_R I, \end{cases} \tag{1.1}$$

where $S(t)$ is the number of susceptible individuals, $I(t)$ is the number of infective individuals (i.e. individuals who are infected and capable to transmit the disease), $R(t)$ is the number of recovered individuals at time t, respectively, and N is the total number of individuals in the population. The parameter $\beta > 0$ is called the

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infection rate (i.e. the contact rate times the probability of infection, see [59]), and $\eta_R > 0$ is the recovery rate (i.e. the rate at which infective individuals recover). The SIR model has been used successfully to describe several epidemics (see for example [15]), but as far as we understand, this rate of infection is only derived empirically, namely by comparison of the model with real data.

When one neglects the demography, an epidemic model becomes a combination of the following aspects:

(a) a rule of contacts between individuals;
(b) a rule of transmission per contact;
(c) a rule of development of the infection at the level of individuals.

Since the development of an infection is not instantaneous, rule (c) can be described by introducing a latency between the transmission of the pathogen and the moment at which an exposed individual becomes capable to transmit the infection (namely becomes infective). This latency can be described by using either an extra exposed class (when the time of latency follows an exponential law), which leads to SEIR models, or an age of infection (i.e. the time since infection), which leads to age-structured models, we refer to [61,60,33,59,41] for details on this topic. In this article, we will neglect the aspect (c) and focus only on (a) and (b).

In an epidemic of an infectious disease, the graph of contact plays a crucial role in the transmission of the disease. It is usually admitted (see [4,30]) that the SIR model (1.1) is derived by using a "*fully mixed*" population. This means that all individuals have the same probability to contact with any other individuals in the population. Here we will see that even with a fully mixed population, the SIR model may fail to reproduce the dynamics of the epidemic. Actually we will see that more sophisticated models are needed to understand the dynamical property of an epidemic.

Of course in most epidemics, the contacts between individuals will arise only locally in space. Therefore more general graphs of contact are needed, we refer to [26,48,24,25,43,8] (and references therein) for more information on this subject. Actually the space can be incorporated by using different approaches: it can be regarded as a continuous domain (see [50,54,55]) or again as a network (see [6] and references therein). In this article, we will neglect the space in order to focus on the classical SIR model.

Stochastic individual-based models (IBM) have been extensively used to investigate threshold conditions and to evaluate the efficacy of disease control measures in which each host is viewed as an individual agent whose status changes based on probabilistic events occurring over time. IBM are particularly suitable to describe the transmission of infectious diseases in a small population in which the individual behavior plays an important role in the spread of diseases [18,29,40,34]. Studies have been performed to compare different types of IBM. For instance, [57] compared two different types of individual-based models, one assumes random mixing without repetition of contacts and the other assumes that the same contacts repeat day-by-day. They tested and compared how the total size of an outbreak differs between these model types depending on the key parameters such as transmission probability, number of contacts per day, duration of the infectious period, different levels of clustering and varying proportions of repetitive contacts. If the number of contacts per day is high or if the per-contact transmission probability is high, as seen in typical childhood diseases such as measles, they showed that random mixing models provide acceptable estimates of the total outbreak size. If the number of daily contacts or the transmission probability is low, such as the infection of meticillin-resistant *Staphylococcus aureus* (MRSA), they found that particular consideration should be given to the actual structure of potentially contagious contacts when designing the model. See also the comparison of a stochastic agent-based model and a structured metapopulation stochastic model for the progression of a baseline pandemic event in Italy by Ajelli et al. [1].

We should mention that the Gillespie algorithm or Doob-Gillespie algorithm (see [22,23,27,28]) provides a method to run random Monte-Carlo simulations associated to ordinary differential equations (see [5,39]). This method was successfully used for chemical or biochemical systems of reactions. In epidemics, we will see in this article that changing the moment of pathogen's transmission from the beginning to the end of contact may influence the dynamical property of the equations.

The main issue to be addressed in this article is the comparison between the classical deterministic SIR model and its computer stochastic versions. The stochastic models will be derived by using Monte Carlo simulations or IBM. The increase in behavioral details provided by IBM, however, leads to much greater computational intensity and much greater difficulty in analyzing the significance of parameters. Some comparisons between deterministic models and IBM have been performed by Pascual and Levin [49] (in the context of predator–prey), D'Agata et al. [17] (in the context of epidemics), Hinow et al. [32] (in the context of cell population dynamics), and Sharkey [56] (in the context of epidemics in networks). But as we will see, even with rather simple rules (a) and (b), the comparison between the SIR model (1.1) and the IBM derived from these stochastic rules (at the individual level) is not clear in general. Actually we will see that more general classes of SIR models are necessary to derive a comparison with the IBM.

The paper is organized as follows. In Section 2 we make some assumptions about the rules of contacts between susceptible and infective individuals, the rules of transmission of diseases through these contacts, and the time of transmission during contacts. In Section 3 we analyze the transmission driven only by susceptible individuals and compare the numerical simulations between the classical SIR model and the IBM. In Section 4, the transmission driven only by infective individuals is modeled and analyzed. Our analysis and simulations demonstrate that in both cases, the IBM converges to the classical SIR model only in some particular situations. In general, the classical SIR model and the IBM are significantly different. A brief discussion is given in Section 5.

2. Rules of contacts and transmission

In this section we present the stochastic process describing contacts between individuals. This process will lead to the construction of a simple deterministic model. The contacts are supposed to be arbitrarily given at an initial time, and in order to describe the evolution of the contacts with time, we will use the following rules.

We would like to point out that the evolution of the contact network is indeed dynamic since it changes with time. We define the rules of contacts, the rules of transmission, and the time of transmission for the purpose of numerical and stochastic simulations of the SIR model. These rules may not affect the outcome of an epidemic from a deterministic modeling point of view. However, they are important in numerical and stochastic simulations and produce dramatically different results.

2.1. Rules of contacts

Firstly, we make some assumptions on the rules of contacts.

(a) At any time each individual has initiated exactly one contact with an individual in the population (possibly himself).
(b) The duration of a contact follows an exponential law and the average duration of a contact is $T_C > 0$.

(c) At the end of a given contact the initiating individual randomly chooses a new individual within the population and the duration for this contact is determined.

The rules of contacts are chosen to be as simple as possible, since our goal is to obtain a comparison between an epidemic model using the above rules for contacts and the usual SI model. The duration of a given contact is the time from when the contact begins until the individual who initiated the contact concludes it and begins a new contact. Therefore the average contact duration $T_C > 0$ can be estimated in practice. Indeed, $v_C = 1/T_C$ is the average number of contacts per unit of time.

In real epidemics, participants may have no choice on contact. Here the terminology "choose" or "initiate" means an individual of one type (either S or I type) is tracked through a time course of paired-contact with other individuals. In the following we will consider the extreme case where one follows either only the susceptible-individuals (abbr. S-individuals) or only the infectious-individuals (abbr. I-individuals). As we will see the model outcomes can be very different if the probability of transmission depends on the class of individuals that is tracked.

To further clarify the terminology, the above assumption also means that the graph of contact (in which the nodes or vertices are the individuals) is oriented. Moreover, we assume that each individual has one and only one outward arrow (possibly directed to himself) and at the end of a given oriented contact the new partner is chosen randomly (within the population). Oriented graphs have been used previously in the literature to describe epidemics (see for example [44]). The definition of "choose" or "initiate" is contextual in the description of the graph (or the IBM).

In this model, an S-individual may have many contacts directed to him, in particular many directed contacts from an I-individual and vice versa. The directed network approach may look at first more complicated, but it provides an advantage to construct an associated mathematical model to the epidemic considered (i.e. to evaluate the contacts between S and I individuals). One may also observe that similar treatment can be used for disease involving a criss-cross transmission between two populations (e.g. malaria, nosocomial infections, etc.). In such a situation, the probability of transmission may depend on which population is transmitting the pathogen. But the mathematical model associated with the problem will remain similar to the ones constructed here. Therefore, here it makes sense to look at the population divided into two subpopulations S and I.

In order to describe the epidemics, we will classify the population into the class S of susceptible individuals (i.e. capable to become infected by contact) and the class I of infective individuals (i.e. capable to transmit the disease by contact). The transmission can only occur at the end of a contact between an S-individual and an I-individual. Moreover, we use the following rules of transmission. In order to take into account new infections, it will be appropriate to distinguish two processes:

(i)   an S-individual chooses an I-individual for a contact;
(ii)   an I-individual chooses an S-individual for a contact.

Depending on the disease, there may be an asymmetry between these processes and an important difference in their likelihood of leading to disease transmission. For example, if the infection is severe enough to immobilize infective individuals, then the transmission can only take place when susceptible individuals initiate contacts with infective individuals. On the other hand, for the common seasonal diseases, such as influenza, infected individuals continue to initiate contacts and they are very likely to play an important role in the transmission of the diseases.

As a first attempt to model this process, we first distinguish the number S_C of S-individuals who choose a contact with an infective individual, the number S_F of S-individuals who choose a contact with a susceptible individual, the number I_C of I-individuals who choose a contact with a susceptible individual, and the number I_F of I-individuals who choose a contact with an infective individual. A diagram of the contact network is given in Fig. 1. The arrows are pointing the individuals who are chosen for the contact. $T_1, T_2, \ldots, T_5$ are the times remaining to the end of the contact for each individual. For this graph of contacts, $(S_c, S_f, I_c, I_f) = (1, 2, 1, 1)$. The individuals in S_c and I_c can be spotted by finding the arrows between susceptible and infective nodes.

Under the Rules of contacts, we obtain the following model

$$\begin{cases} S_C' = v_C \frac{I}{S+I}(S_C + S_F) - v_C S_C \\ S_F' = v_C \frac{S}{S+I}(S_C + S_F) - v_C S_F \\ I_C' = v_C \frac{S}{S+I}(I_C + I_F) - v_C I_C \\ I_F' = v_C \frac{I}{S+I}(I_C + I_F) - v_C I_F \end{cases} \tag{2.1}$$

with

$$S = S_C + S_F \quad \text{and} \quad I = I_C + I_F.$$

In system (2.1), the fraction $\frac{S(t)}{S(t)+I(t)}$ (resp. $\frac{I(t)}{S(t)+I(t)}$) is the probability (in a "well mixed" population) that an individual initiates a new contact (resp. stops a contact) to a susceptible individual (resp. an infective) at a given time t. The quantity $v_C S_C(t)$ (resp. $v_C S_F, v_C I_C, v_C I_F$) is the flux of individuals interrupting a contact at time t in the class of S_C (resp. S_F, I_C, I_F). Thus, in the absence of new infections, the rate that susceptible individuals forming new contacts is $v_C(S_C + S_F)$. So the rate at which susceptible individuals form new contacts with infective individuals is $v_C \frac{I}{S+I}(S_C + S_F)$, the flux into S_C. Similarly, the flux into S_F is $v_C \frac{S}{S+I}(S_C + S_F)$. The equations for I_C' and I_F' can be explained similarly.
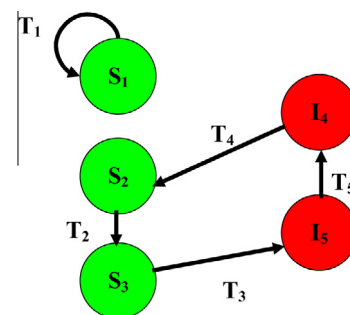
The following lemma is readily proved.

Lemma 2.1. *For solutions of system* (2.1), *$S(t)$ and $I(t)$ are constant and*
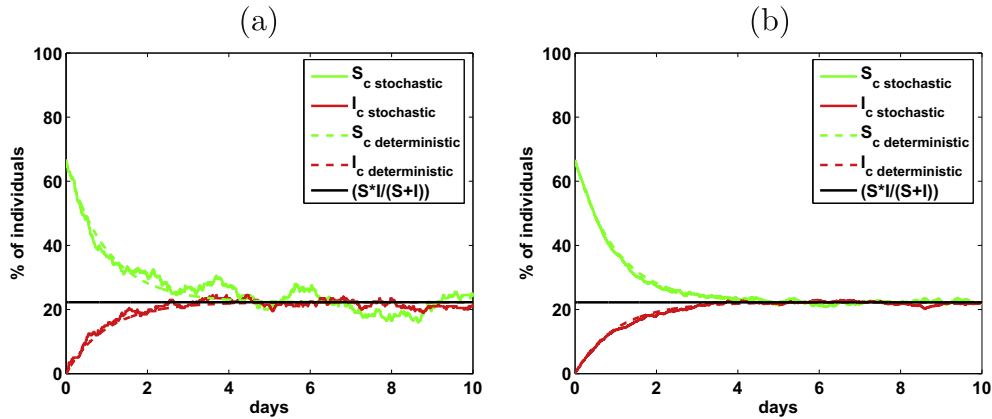
$$(S_C(t), S_F(t), I_C(t), I_F(t)) \rightarrow \left(\frac{SI}{S+I}, \frac{S^2}{S+I}, \frac{SI}{S+I}, \frac{I^2}{S+I} \right) \quad \text{as } t \rightarrow +\infty,$$

where the convergence is exponential.

The comparison between the ODE model (2.1) and Monte Carlo simulations of the model is given in Fig. 2. Here a Monte Carlo simulation means we run a stochastic computer program where we make a simulation of the above assumptions for N individuals. In this figure we fix $v_c = 1, I = N/3, S = 2N/3, I_c = 0$ and $S_c = S$ at time $t = 0$. Moreover, the number of individuals N varied from $N = 200$

Fig. 1. Diagram of the contact network at a given time $t \geqslant 0$. The first S-individual (S_1) only contacts with himself. The second S-individual (S_2) chooses to contact with a third S-individual (S_3) who in turn contacts with an I-individual (I_5). I_5 contacts another I-individual (I_4) who in turn chooses S_2 for contact.

Fig. 2. The comparison between solutions of the ordinary differential equation model (2.1) and Monte Carlo simulations of the model. The solutions of the stochastic model converge to the equilibrium solutions of the ODE model. Here $v_c = 1, I = N/3, S = 2N/3, I_c = 0$ and $S_c = S$ at time $t = 0$. (a) $N = 200$ and (b) $N = 2000$.

in (a) up to $N = 2000$ in (b). Therefore, when N increases the solutions $S_c(t)$ and $I_c(t)$ of the stochastic simulations converge to the trajectories of the ordinary differential equation model (2.1).

We now specify the rules for disease transmission.

2.2. Rules of transmission

We make assumptions about the rules of transmission: During a given contact between an S-individual and an I-individual, the probability of transmission is

(a) $p_S \in [0, 1]$ if the contact was initiated by an S-individual;
(b) $p_I \in [0, 1]$ if the contact was initiated by an I-individual.

We would like to make some remarks about p_S and p_I. It is important to understand that the word "choosing" here serves only to construct the diagram of contact. For most cases (i.e. for non vector-borne diseases) there is no reason to assume that the transmission is oriented. Therefore, for non vector-borne diseases it will be natural to assume that $p_S = p_I$. Nevertheless in order to count the number of contacts between S- and I-individuals it will be convenient to keep the word "choosing". For vector-borne or sexually transmitted diseases, p_S and p_I might be different. For example, it has been reported that [31] the man-to-woman transmission rate of HIV/AIDS is different from the woman-to-man transmission rate. Also, in hospitals the contamination rate of health care workers and the colonization rate of patients for nosocomial infection are usually different. Thus, for the sake of generality we assume that p_S and p_I are different.

The next two sections are divided according to the following special cases:

(i) $p_S > 0$ and $p_I = 0$ (which will be called transmission driven by the susceptibles);
(ii) $p_S = 0$ and $p_I > 0$ (which will be called transmission driven by infectives).

One may observe that case (i) actually corresponds to the fact that the contact initiated by I-individuals plays no role in the transmission. Therefore assumption (i) is also equivalent to supposing that only S-individuals are initiating contacts. Case (ii) is similar and this assumption is also equivalent to assuming that only I-individuals are initiating contacts. As we will see, even if these two cases look symmetric at first, these two scenarios are fairly different in terms of mathematical models. We also would like to note

that p_S and p_I are used as the probabilities for all contacts during a simulation.

2.3. Time of transmission

Finally we would like to address the issue on the time of transmission during a given contact. For a given contact between a susceptible individual and an infective individual, the transmission of the disease occurs (with a probability p_S or p_I) only at one of the following two moments:
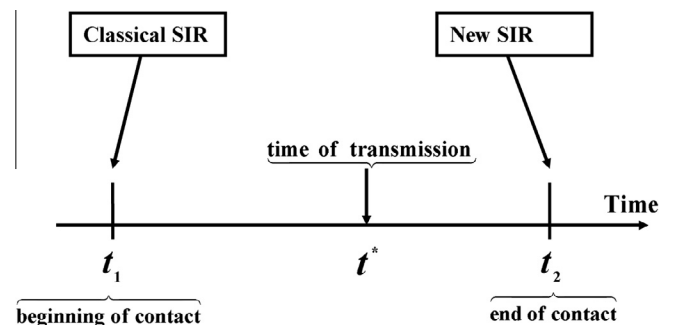
(c) the beginning of the contact;
(d) the end of the contact.

This is illustrated in Fig. 3. In subsections 3.4 and 4.4 (on numerical simulations), we will examine the following two "extreme" cases. With the notations of Fig. 3, case (c) describes the situation where $t^* = t_1$ while case (d) corresponds to the situation where $t^* = t_2$. Both cases may look very similar at first, in reality they are not. As we will see in Sections 3.4 and 4.4, case (c) corresponds to the classical SIR model (1.1) while case (d) describes new classes of SIR models which will be presented in Sections 3 and 4.

3. Transmission driven only by S-individuals

Under the Rules of contacts and Rules of transmission, we further assume that

$$p_S > 0 \quad \text{and} \quad p_I = 0.$$

Fig. 3. Time of transmission. (a) Transmission occurs at the beginning of a contact as assumed in the classical SIR model and (b) transmission occurs at the end of a contact as assumed in the new SIR model. In general, transmission can occur at any moment in between.

In addition we assume that the transmission occurs at the end of the contact period (i.e. Time of transmission (d) is satisfied). By combining these assumptions and using model (2.1), we obtain the following epidemic model

$$
\begin{cases}
S_C' = v_C\left(\frac{I}{S+I}(S_C + S_F - p_S S_C) - S_C\right) \\
S_F' = v_C\left(\frac{S}{S+I}(S_C + S_F - p_S S_C) - S_F\right) \\
I' = v_C p_S S_C,
\end{cases}
\tag{3.1}
$$

which yields

$$
\begin{aligned}
S' &= -v_C p_S S_C \\
I' &= v_C p_S S_C.
\end{aligned}
$$

The terms in Eqs. (3.1) involving p_S come from the fact that the rates at which new contacts of susceptible individuals and infective individuals have changed due to the inclusion of disease transmission in (3.1). The flux of S-individuals ending a contact at time t and in contact with an infective individual is $v_C S_C$. Therefore the flux of S-individuals becoming infected must be $p_S v_C S_C$. The rate at which susceptible ones are forming new contacts is now $v_C(S_C + S_F - p_S S_C)$.

3.1. Model with recovery

We consider the case that the population is divided into three groups: susceptible S, infective I, and recovered R. We also assume that only susceptible individuals initiate the contact.

Assumption 3.1. The duration of an infection follows an exponential law and the average duration of an infection is $T_R > 0$.

Under the above assumption, the rate at which I-individuals are recovering is $\eta_R := \frac{1}{T_R}$ and we obtain the following model

$$
\begin{cases}
S_C' = v_C\left(\frac{I}{N}[S_F + (1 - p_S)S_C] - S_C\right) \\
S_F' = v_C\left(\frac{S+R}{N}[S_F + (1 - p_S)S_C] - S_F\right) \\
I' = v_C p_S S_C - \eta_R I \\
R' = \eta_R I,
\end{cases}
\tag{3.2}
$$

where $N = S + I + R$. The fluxes between the compartments of the model (3.2) are described in Fig. 4.

The parameters and state variables of the model are listed in Table 1.

3.2. Asymptotic behavior

Set $s = S/N, i = I/N, r = R/N, s_c = S_C/N$, and $s_f := S_F/N$. Then model (3.2) is equivalent to

$$
\begin{cases}
s_c' = v_C\{i(s - p_S s_c) - s_c\} \\
s_f' = v_C\{(s + r)(s - p_S s_c) - s_f\} \\
i' = v_C p_S s_c - \eta_R i \\
r' = \eta_R i.
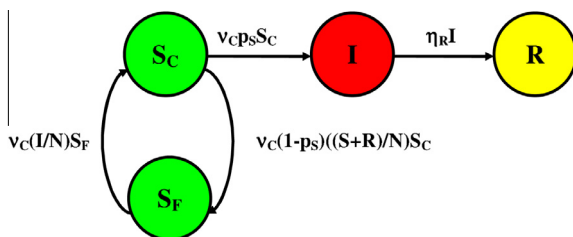\end{cases}
\tag{3.3}
$$

Fig. 4. The flux diagram of model (3.2).

By summing up the first two equations in system (3.3) we obtain the following model

$$
\begin{cases}
s' = -v_c p_S s_c \\
i' = v_c p_S s_c - \eta_R i \\
r' = \eta_R i \\
s_c' = v_c\{i(s - p_S s_c) - s_c\}
\end{cases}
\tag{3.4}
$$

with $s(0) \geqslant 0, i(0) \geqslant 0, r(0) \geqslant 0$ and

$$
s(0) + i(0) + r(0) = 1 \quad \text{and} \quad s_c(0) \in [0, s(0)].
\tag{3.5}
$$

Now consider $\bar{s}_c = \frac{s_c}{s}$ which is the probability (or the fraction) of S-individuals in contact with an I-individual within the population of S-individuals. By using the first equation of systems (3.3) and (3.4), we have

$$
\begin{aligned}
\bar{s}_c' &= \frac{s_c'}{s} - \frac{s_c}{s}\frac{s'}{s} = v_c \frac{1}{s}\{i(s - p_S s_c) - s_c\} + \frac{s_c}{s}\frac{v_c p_S s_c}{s} \\
&= v_c\{i(1 - p_S \bar{s}_c) - \bar{s}_c + p_S \bar{s}_c^2\}.
\end{aligned}
$$

Hence

$$
\bar{s}_c' = v_c(i - \bar{s}_c)(1 - p_S \bar{s}_c).
\tag{3.6}
$$

So we obtain the following system

$$
\begin{cases}
s' = -v_c p_S \bar{s}_c s \\
i' = v_c p_S \bar{s}_c s - \eta_R i \\
r' = \eta_R i \\
\bar{s}_c' = v_c(i - \bar{s}_c)(1 - p_S \bar{s}_c),
\end{cases}
\tag{3.7}
$$

where the initial values satisfy

$$
s(0) = s_0 \geqslant 0, \quad i(0) = i_0 \geqslant 0, \quad r(0) = r_0 \geqslant 0 \quad \text{and} \quad \bar{s}_c(0) = \bar{s}_{c0}
$$
$$
\in [0, 1)
$$

with $s_0 + i_0 + r_0 = 1$. We can also see that

$$
s(t) + i(t) + r(t) = 1, \quad \forall t \geqslant 0.
\tag{3.8}
$$

Moreover, we have the following inequality

$$
\bar{s}_c'(t) \leqslant v_c(1 - \bar{s}_c(t))(1 - p_S \bar{s}_c(t))
$$

whenever $\bar{s}_c(t) \leqslant 1$. Hence we deduce that

$$
\bar{s}_c(t) \in (0, 1), \quad \forall t > 0.
\tag{3.9}
$$

The last tool to complete the description of the asymptotic behavior is the following equality

$$
\frac{d}{dt}\left\{s + i - \frac{\eta_R}{p_S v_c}[\ln(1 - p_S \bar{s}_c) + \ln(s)]\right\} = 0.
\tag{3.10}
$$

Remark 3.2. The model does not coincide with the classical SIR model. Indeed, if $\eta_R = 0$ then we have $s + i = 1$ (for both models), it is clear that the equations

$$
\bar{s}_c' = v_c(i - \bar{s}_c)(1 - p_S \bar{s}_c)
$$

and

$$
i' = \beta(1 - i)i
$$

do not coincide. If $\eta_R > 0$, Eq. (3.10) provides a formula for $\bar{s}_c$ which is not proportional to i.

As for the classical SIR model, equality (3.10) is the main tool to determine the limits of the s and r components in model (3.7). The equilibria of system (3.7) satisfy

$$
s^* + r^* = 1 \quad \text{and} \quad i^* = \bar{s}_c^* = 0
$$

Table 1
List of parameters and variables of the model.

Symbol	Interpretation
v_C	Rate of contact
η_R	Rate of recovery
$T_C = 1/v_C$	Average duration of contacts
$T_R = 1/\eta_R$	Average duration of infection
p_S	Probability of infection at the end of a contact whenever a susceptible chooses an infective
p_I	Probability of infection at the end of a contact whenever an infective chooses a susceptible
S_C	Number of susceptibles in contact with an infective
S_F	Number of susceptibles contact free with an infective
I_C	Number of infectives in contact with a susceptible
I_F	Number of infectives contact free with a susceptible
$S := S_C + S_F$	Number of susceptibles
$I := I_C + I_F$	Number of infectives
R	Number of recovereds

By using the previous observations (3.8)–(3.10), we obtain the following proposition.

Proposition 3.3. *Assume that $p_S \in (0,1), v_c > 0, \eta_R > 0$, and further suppose that*

$$s_0 > 0 \quad \text{and} \quad i_0 > 0.$$

Then all solutions of model (3.7) *and their limits satisfy the following property for initial values with $i_0 > 0$*

$$\lim_{t \to +\infty} \begin{pmatrix} s(t) \\ i(t) \\ r(t) \\ \bar{s}_c(t) \end{pmatrix} = \begin{pmatrix} s^* \\ 0 \\ r^* \\ 0 \end{pmatrix}$$

with the equilibrium satisfying

$$s^* + r^* = 1$$

and s^* being the unique solution in $\left(0, \frac{\eta_R}{p_S v_c}\right]$ of the equation

$$s^* - \frac{\eta_R}{p_S v_c} \ln(s^*) = \left\{ s_0 + i_0 - \frac{\eta_R}{p_S v_c} [\ln(1 - p_S \bar{s}_{c0}) + \ln(s_0)] \right\}. \quad (3.11)$$

Proof. Since $t \to s(t)$ is decreasing and $t \to r(t)$ is increasing, and both functions are bounded by 1, we deduce that both functions converge (when t goes to $+\infty$), respectively, to $s^* \geq 0$ and $r^* \geq 0$. Moreover, since $s + i + r = 1$ we deduce that $t \to i(t)$ also converges (when t goes to $+\infty$) to some $i^* \geq 0$. Now by using the r-equation one deduces that $i^* = 0$ (otherwise $r(t)$ would be unbounded). Moreover, by using (3.10) one deduces that $s^* > 0$ and satisfies (3.11). Now it remains to prove that s^* belongs to $\left(0, \frac{\eta_R}{p_S v_c}\right]$. Assume that

$$s^* > \frac{\eta_R}{p_S v_c}. \quad (3.12)$$

Without loss of generality we assume that $i_0 > 0$ and $\bar{s}_{c0} > 0$. Otherwise if $\bar{s}_{c0} = 0$, since $i_0 > 0$ and

$$\bar{s}'_c = v_c(i - \bar{s}_c)(1 - p_S \bar{s}_c),$$

replacing $(i_0, \bar{s}_{c0})$ by any $(i(t), \bar{s}_c(t))$ for $t > 0$ we obtain the desired property.

Next by using the i-equation in (3.7) we obtain

$$\begin{cases} i' \geq \eta_R(\bar{s}_c - i), \\ \bar{s}'_c = v_c(i - \bar{s}_c)(1 - p_S \bar{s}_c). \end{cases}$$

We deduce that

$$(i(t), \bar{s}_c(t)) \geq (x(t), y(t)), \quad \forall t \geq 0,$$

where (x, y) is the solution of the monotone system (see [58])

$$\begin{cases} x' = \eta_R(y - x) \\ y' = v_c(x - y)(1 - p_S y) \end{cases} \quad (3.13)$$

with

$$x(0) = i_0 > 0 \quad \text{and} \quad y(0) = \bar{s}_{c0} > 0.$$

Set

$$\varepsilon = \min(i_0, \bar{s}_{c0}).$$

We conclude that

$$\lim_{t \to +\infty} i(t) \geq \varepsilon \quad \text{and} \quad \lim_{t \to +\infty} \bar{s}_c(t) \geq \varepsilon,$$

since $(\varepsilon, \varepsilon)$ is an equilibrium of system (3.13), we obtain a contradiction with the fact that

$$\lim_{t \to +\infty} i(t) = \lim_{t \to +\infty} \bar{s}_c(t) = 0.$$

This completes the proof. □

3.3. Comparison with the classical SIR model

Let $\beta_S > 0$ and $\eta_R > 0$ be fixed. Set $v_c = \frac{1}{\varepsilon}, p_S = \beta_S \varepsilon$, where $\varepsilon \in (0, \beta_S^{-1}]$ is a small parameter. By using this rescaling, system (3.7) becomes the following system parameterized by ε:

$$\begin{cases} s'_\varepsilon = -\beta_S \bar{s}_{c\varepsilon} s_\varepsilon \\ i'_\varepsilon = \beta_S \bar{s}_{c\varepsilon} s_\varepsilon - \eta_R i_\varepsilon \\ r'_\varepsilon = \eta_R i_\varepsilon \\ \bar{s}'_{c\varepsilon} = \frac{1}{\varepsilon}(i_\varepsilon - \bar{s}_{c\varepsilon})(1 - \beta_S \varepsilon \bar{s}_{c\varepsilon}) \end{cases} \quad (3.14)$$

with

$$s_\varepsilon(0) = s_0 > 0, \quad i_\varepsilon(0) = i_0 > 0, \quad r(0) = r_0 \geq 0 \quad \text{and} \quad \bar{s}_{cc}(0) = \bar{s}_{c0}$$
$$\in [0, 1).$$

Indeed when ε goes to 0 we obtain a singular perturbation problem.

The main question to be addressed in this section is the convergence of the first three components of the system.

Now consider the classical SIR model

$$\begin{cases} s' = \beta_S is \\ i' = \beta_S is - \eta_R i \\ r' = \eta_R i \end{cases} \quad (3.15)$$

with

$$s(0) = s_0 > 0, \quad i(0) = i_0 > 0, \quad r(0) = r_0 \geq 0.$$

Recall now the classical result of the SIR model (see [30]). By using the fact that

$$\frac{d}{dt}\left[s + i - \frac{\eta_R}{\beta_S} \ln(s) \right] = 0 \quad (3.16)$$

and the same argument as above one has the following result.

Proposition 3.4. *Assume that*

$$s_0 > 0 \quad \text{and} \quad i_0 > 0,$$

then

$$\lim_{t \to +\infty} \begin{pmatrix} s(t) \\ i(t) \\ r(t) \end{pmatrix} = \begin{pmatrix} s^* \\ 0 \\ r^* \end{pmatrix},$$

where s^* is the unique solution in $\left(0, \frac{\eta_R}{\beta_S}\right]$ of the equation

$$s^* - \frac{\eta_R}{\beta_S} \ln(s^*) = \left\{ s_0 + i_0 - \frac{\eta_R}{\beta_S} \ln(s_0)] \right\}. \qquad (3.17)$$

The main result of this section is the following theorem on the convergence of the solution.

Theorem 3.5 (*Uniform convergence in time*). *Under the above assumptions, if*

$$s_\varepsilon(0) = s(0) > 0, i_\varepsilon(0) = i(0) > 0, r_\varepsilon(t) = r(0) \geqslant 0,$$

then

$$\begin{pmatrix} s_\varepsilon(t) \\ i_\varepsilon(t) \\ r_\varepsilon(t) \end{pmatrix} \rightarrow \begin{pmatrix} s(t) \\ i(t) \\ r(t) \end{pmatrix}$$

as $\varepsilon \to 0$ uniformly with respect to t in $[0, +\infty)$.

In other word, for each constant $\delta > 0$, we can find $\widehat{\varepsilon} = \widehat{\varepsilon}(\delta) > 0$, such that

$$|s_\varepsilon(t) - s(t)| \leqslant \delta, \quad |i_\varepsilon(t) - i(t)| \leqslant \delta \quad \text{and} \quad |r_\varepsilon(t) - r(t)| \leqslant \delta$$

for each $\varepsilon \in (0, \widehat{\varepsilon}]$ and each $t \geqslant 0$.

In order to prove this result, we start with a convergence result for a finite time.

Lemma 3.6. *Let $\tau > 0$. Then*

$$\begin{pmatrix} s_\varepsilon(t) \\ i_\varepsilon(t) \\ r_\varepsilon(t) \end{pmatrix} \rightarrow \begin{pmatrix} s(t) \\ i(t) \\ r(t) \end{pmatrix}$$

as $\varepsilon \to 0$ uniformly with respect to t in $[0, \tau]$.

Proof. Let $\tau > 0$ be fixed. It is clear the

$$i_\varepsilon(t) + s_\varepsilon(t) \leqslant 1, \quad \forall t \in [0, \tau],$$

and by construction we have

$$0 \leqslant s_{\varepsilon c}(t) \leqslant 1 \quad \text{and} \quad 0 \leqslant i_{\varepsilon c}(t) \leqslant 1.$$

We also have

$$s'_\varepsilon = -\beta_S \bar{s}_{c\varepsilon} s_\varepsilon$$
$$i'_\varepsilon = \beta_S \bar{s}_{c\varepsilon} s_\varepsilon - \eta_R i_\varepsilon$$
$$r'_\varepsilon = \eta_R i_\varepsilon.$$

Thus

$$\left| i'_\varepsilon(t) \right| + \left| s'_\varepsilon(t) \right| + \left| r'_\varepsilon(t) \right| \leqslant 2(\beta_S + \eta_R), \quad \forall t \in [0, \tau].$$

Therefore, by the Arzela–Ascoli theorem, for each sequence $\varepsilon_n \to 0$ we find a subsequence (denoted with the same index) such that

$$s_{\varepsilon_n}(t) \to s(t), \quad i_{\varepsilon_n}(t) \to i(t) \quad \text{and} \quad r_{\varepsilon_n}(t) \to r(t) \quad \text{as } n \to +\infty,$$

uniformly with respect to t in $[0, \tau]$. Moreover,

$$\bar{s}'_{c\varepsilon} = \frac{1}{\varepsilon}(i_\varepsilon - \bar{s}_{c\varepsilon})(1 - \beta_S \varepsilon \bar{s}_{c\varepsilon}).$$

$$= \frac{1}{\varepsilon}[i_\varepsilon(1 - \beta_S \varepsilon \bar{s}_{c\varepsilon}) - \bar{s}_{c\varepsilon}(1 - \beta_S \varepsilon \bar{s}_{c\varepsilon})],$$

so

$$\bar{s}'_{c\varepsilon} = -\frac{1}{\varepsilon}\bar{s}_{c\varepsilon} + \frac{1}{\varepsilon}[i_\varepsilon - \beta_S \varepsilon(\bar{s}_{c\varepsilon} - \bar{s}_{c\varepsilon}^2)].$$

Therefore,

$$\bar{s}_{\varepsilon c}(t) = e^{-\frac{t}{\varepsilon}}\bar{s}_{c0} + \int_0^t \frac{1}{\varepsilon}e^{-\frac{(t-l)}{\varepsilon}}i_\varepsilon(l)dl - \int_0^t e^{-\frac{(t-l)}{\varepsilon}}\beta_S[\bar{s}_{c\varepsilon}(l) - \bar{s}_{c\varepsilon}^2(l)]dl.$$

Since

$$\int_0^t \frac{1}{\varepsilon_n}e^{-\frac{1}{\varepsilon_n}(t-l)}i_{\varepsilon_n}(l)dl \to i(t) \quad \text{as } n \to +\infty \quad \text{in } L^1(0, \tau)$$

and

$$e^{-\frac{t}{\varepsilon_n}}\bar{s}_{c0} - \int_0^t e^{-\frac{1}{\varepsilon_n}(t-l)}\beta_S[\bar{s}_{c\varepsilon}(l) - \bar{s}_{c\varepsilon}^2(l)]dl \to 0 \quad \text{as } n \to 0 \quad \text{in } L^1(0, \tau),$$

the result follows. $\square$

Proof of Theorem 3.5. The proof is to combine the (monotone) convergence of $t \to s(t)$ and $t \to r(t)$, Lemma 3.6, and the convergence of the equilibrium as $\varepsilon \to 0$ (i.e. formula (3.11) and formula (3.17)). $\square$

3.4. Numerical simulations

In order to compare these models numerically we consider some extreme cases. The codes used for the numerical simulations can be downloaded at http://www.math.u-bordeaux1.fr/pmagal/SIR/SIR.htm.

Actually the IBM are rather delicate to run since some parameters and terms need to be more specific. Here we specify the rules of transmission between an *S*-individual and an *I*-individual.

Assumption 3.7. We further assume that

  (a) **(New SIR Model)** For a given pair of (S, I)-individuals in contact, if the individual initiating the contact is an *S*-individual, then the transmission only occurs at the end of the contact with the probability p_S.
  (b) **(Classical SIR Model)** For a given pair of (S, I)-individuals in contact, if the individual initiating the contact is an *S*-individual, then the transmission only occurs at the beginning of the contact with the probability p_S.

We will use Assumption 3.7 (a) to run an IBM (called IBM$_{11}$ here) which corresponds to model (3.2), while Assumption 3.7 (b) will be used to run an IBM (called IBM$_{21}$ here) which corresponds to the classical SIR model (1.1). There are four cases in total which are summarized in Table 2. The two other cases will be studied in subsection 4.4. As we will see, replacing one model by the other might lead to a large bias in the prediction. Therefore one must be very careful in using IBM to simulate an epidemic.

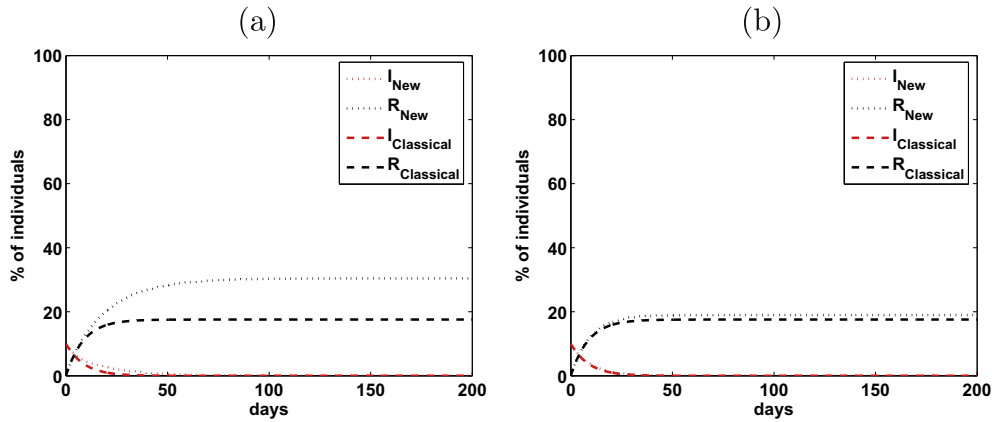### 3.4.1. Simulations with a fully random graph of connection at time $t = 0$

In Fig. 5 we compare the new SIR model (3.2) and the classical SIR model (1.1) with $\beta = \beta_S = p_S/T_C$ fixed. The initial value $S_C = SI/N$ at time $t = 0$, therefore the contacts are assumed to be already stabilized. We observe large bias in comparing both models. The deviation is confirmed in the numerical simulations of the IBM in Fig. 5. As predicted by Theorem 3.5, we first observe numerically the convergence of solutions of the new SIR model (3.2) to the solutions of the classical SIR model (1.1) when the average time of contact T_C goes to zero. Note that the curve of $R(t)$, the number of recovered individuals, is influenced by the parameter T_C. Let R_∞ be the limit of $R(t)$ when t goes to $+\infty$. Recall that R_∞ is the total number of cases produced by an epidemic. We can see that R_∞ is influenced by the parameter T_C. In Fig. 5(a) the total numbers of cases of the new SIR model (3.2) and the classical SIR model (1.1) are fairly different. Here $S = 0.9, I = 0.1, S_c = SI/(S + I + R)$ at time

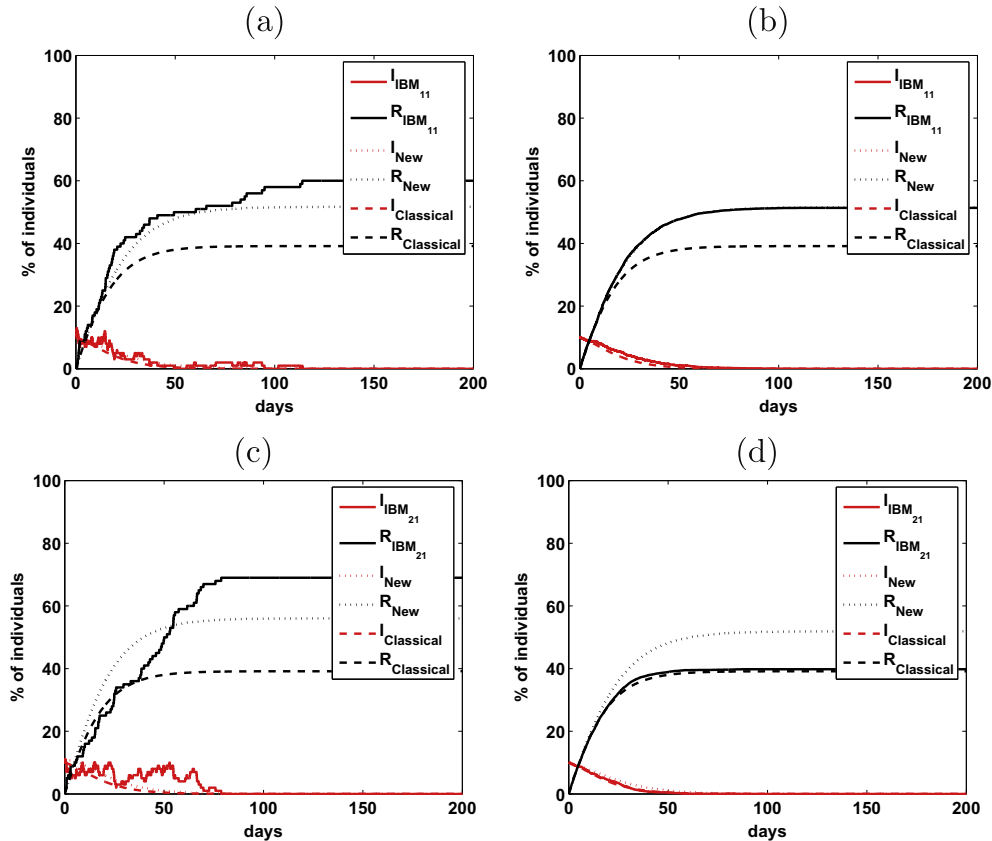**Table 2**
List of four IBMs.

	Transmission driven by S	Transmission driven by I
End of contact	IBM_{11}(First new SIR)	IBM_{12} (Second new SIR)
Beginning of contact	IBM_{21} (Classical SIR)	IBM_{22} (Classical SIR)

$t = 0$. $T_R = 5$ and $\beta_S = 0.1$ were fixed. In (a) (resp. (b)), $T_C = 10$ and $p_S = \beta_S T_C = 1$ (resp. $T_C = 1$ and $p_S = \beta_S T_C = 0.1$) were fixed.

Comparisons of the IBM, the new SIR model (3.2), and the classical SIR model (1.1) are presented in Fig. 6. Here $I = (1/10)N, S = (9/10)N$, with $S_c = SI/(S + I + R)$ at time $t = 0$. $T_C = 5, T_R = 5, \beta_S = 0.2$ and $p_S = \beta_S T_C = 1$ were fixed. $v_c = 1/T_C = 1/5, v_R = 1/T_R = 1/5$. N (the total number of individuals in the IBM) varies from $N = 100$ in (a) and (c) to $N = 10,000$ in (b) and (d). In (a) (resp. (b)) the IBM_{11} was run to simulate the transmission at the end of the contact for $N = 100$ (resp. $N = 10,000$). In (c) (resp. (d)) the IBM_{21} was run to simulate the

Fig. 5. Comparison of the new SIR model (3.2) (dotted curves) and classical SIR model (1.1) (dashed curves) with $\beta = \beta_S = p_S/T_C, S = 0.9, I = 0.1, S_c = SI/(S + I + R)$ at time $t = 0$. $T_R = 5$ and $\beta_S = 0.1$ were fixed. (a) $T_C = 10$ and $p_S = \beta_S T_C = 1$. (b) $T_C = 1$ and $p_S = \beta_S T_C = 0.1$. In both cases the solutions of the new SIR model converge to the solutions of the classical SIR model.

Fig. 6. Comparison of simulations of the IBM (solid curves), the new SIR model (3.2) (dotted curves), and the classical SIR model (1.1) (dashed curves) with $\beta = \beta_S, I = (1/10)N, S = (9/10)N$ with $S_c = SI/(S + I + R)$ at time $t = 0$. $T_C = 5, T_R = 5, \beta_S = 0.2$ and $p_S = \beta_S T_C = 1$ were fixed. $v_c = 1/T_C = 1/5, v_R = 1/T_R = 1/5$. In (a) and (b) the IBM_{11} was run to simulate the transmission at the end of the contact for $N = 100$ and $N = 10,000$, respectively. In (c) and (d) the IBM_{21} was run to simulate the transmission at the beginning of the contact for $N = 100$ and $N = 10,000$, respectively.

transmission at the beginning of the contact for $N = 100$ (resp. $N = 10,000$).

In order to illustrate the random fluctuations occurring in IBM_{11} and IBM_{21}, we provide more numerical simulations of these two models with 50 runs. For the sake of simplicity, we only plot the simulations of the recovered class in see Fig. 7. In Fig. 7(a) (resp. (b)) the IBM_{11} was run 50 times to simulate the transmission at the end of the contact for $N = 100$ (resp. $N = 10,000$). In Fig. 7(c) (resp. (d)) the IBM_{21} was run 50 times to simulate the transmission at the beginning of the contact for $N = 100$ (resp. $N = 10,000$). All parameter values are the same as in Fig. 6.

3.4.2. Simulations with a non-fully random graph of connection at time $t = 0$

In this subsection we run some simulations assuming that $S_C = S$ at time $t = 0$. This means that all S-individuals choose randomly a contact with an I-individual at $t = 0$, and for $t > 0$ all individuals choose randomly a contact within the all population (i.e. including the S, I and R individuals). This also means that the contacts are not yet stabilized at time $t = 0$.

In Fig. 8 we observe a large deviation between the new SIR model (3.2) and the classical SIR model (1.1), but the former still predicts the IBM_{11} when the number of individuals increases; that is, the solutions of the IBM_{11} converge to the equilibrium solutions of the new SIR model (3.2) when the number of individuals increases. In both (a) and (b) the classical SIR model (1.1) (with $\beta = \beta_S := p_S/T_C$) fails to predict the total number of cases produced by IBM_{11} (even when $T_C = 0.1$). The classical SIR model (1.1) cannot predict this case, because the contacts are not yet stabilized, and it takes count of the evolution of contacts between individuals. Here $S = 0.9$, $I = 0.1$, $S_c = S$ at time $t = 0$. $T_R = 5$ and $\beta_S = 0.1$ are

fixed. In (a) (resp. (b)) $T_C = 10$ and $p_S = \beta_S T_C = 1$ (resp. $T_C = 0.1$ and $p_S = \beta_S T_C = 0.1$) are fixed. In (a) and (b), the total number of individuals is fixed at $N = 100$ and $N = 1000$ respectively for the IBM_{11}. Of course by taking T_C smaller enough and $p_S = \beta_S T_C$, the total number of cases will finally be predicted by the classical SIR model. This question has to be explored further in order to derive some practical evaluation of the time of contacts and the probability of transmission in order to use the classical SIR model.

4. Transmission driven only by I-individuals

4.1. Gain and loss of contacts

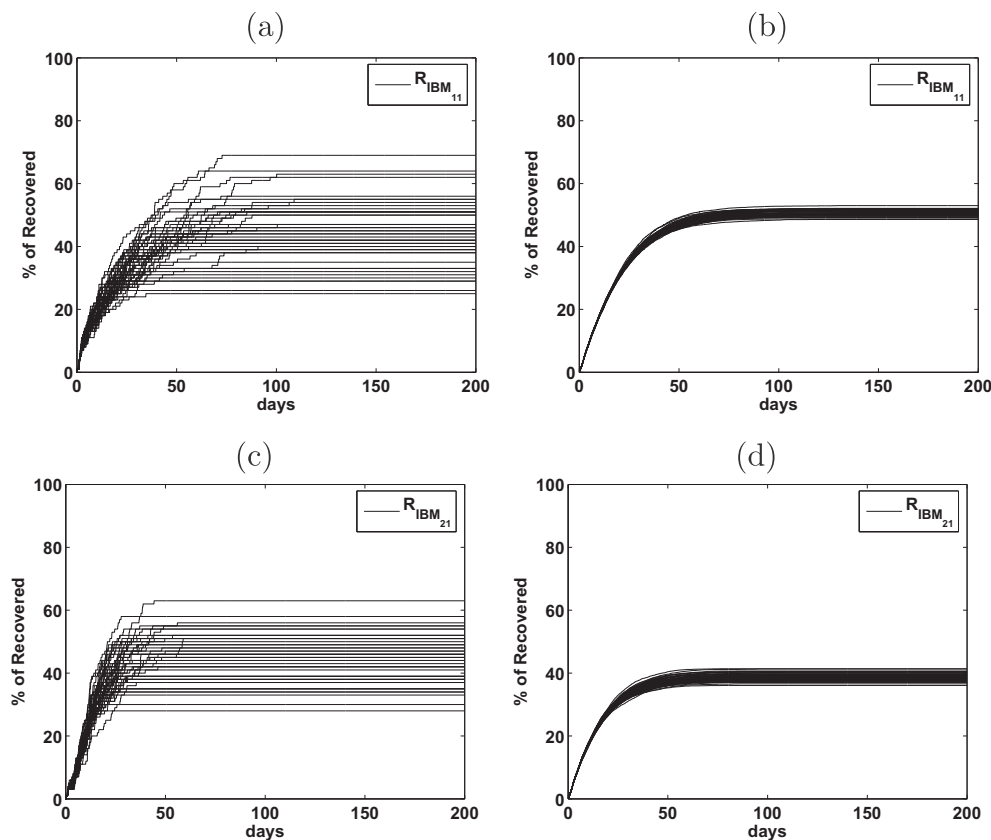Under the Rules of contacts and Rules of transmission, we assume in addition that

$$p_S = 0 \quad \text{and} \quad p_I > 0.$$

This is also equivalent to assuming that only the I-individuals are building some contacts with other individuals chosen randomly in the population.
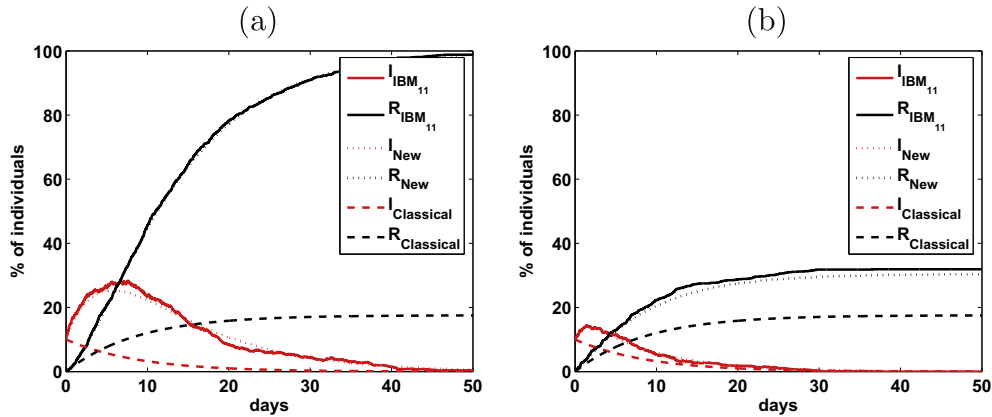
We define $S_n(t)$ as the number of S-individuals that have been chosen n times for a contact by I-individuals. So each S-individual in class S_n has been chosen exactly n times for a contact by exactly n different infective individuals. The total number of S-individuals is given by

$$S := \sum_{n=0}^{+\infty} S_n. \tag{4.1}$$

The structured diagram of the population in term of contacts is given in Fig. 9. Recall that each I-individual chooses at most one S-individual. Since I-individuals are choosing randomly an

Fig. 7. Simulations of the recovered class in the IMB. In (a) and (b) the IBM_{11} was run 50 times to simulate the transmission at the end of the contact for $N = 100$ and $N = 10,000$, respectively. In (c) and (d) the IBM_{21} was run 50 times to simulate the transmission at the beginning of the contact for $N = 100$ and $N = 10000$, respectively. All parameters are the same as in Fig. 6.

Fig. 8. Comparison of simulations of the IBM_{11} (solid curves), the new SIR model (3.2) (dotted curves), and the classical SIR model (1.1) (dashed curves) with $\beta = \beta_S$ and a non-fully random graph of connection at time $t = 0$. Here $S = 0.9, I = 0.1, S_c = S$ at time $t = 0$. $T_R = 5$ and $\beta_S = 0.1$ are fixed. (a) $T_C = 10$ and $p_S = \beta_S T_C = 1$. (b) $T_C = 0.1$ and $p_S = \beta_S T_C = 0.1$.

individual in the population at the end of each contact, an S-individual can be chosen from 0 to a number up to the number of I-individuals. The arrows are pointing to the group of S-individuals which have been chosen for a contact by I-individuals. S_0 is the group of S-individuals which has not been chosen by I-individuals, and S_n is the group of S-individuals which has been chosen n times by I-individuals, where $n = 1, 2, \ldots$

In order to present the model, we first consider separately the following two processes for S-individuals: (1) the gain contacts with I-individuals; (2) the loss contacts with I-individuals. To describe the processes, we first make the following assumption.

Gain of contacts: Assume that, at any given time $t \geqslant 0$, each S-individual can gain at most one contact with an I-individual.

Under this assumption the model describing the gains of contacts is given by the following infinite system of ordinary differential equations:

$$\begin{cases} S'_0 = v_c[S_1(t)] \\ S'_1 = v_c[2S_2(t) - S_1(t)] \\ S'_2 = v_c[3S_3(t) - 2S_2(t)] \\ \ldots \\ S'_n = v_c[(n+1)S_{n+1}(t) - nS_n(t)] \\ \ldots \end{cases} \qquad (4.2)$$

For S-individuals, the total number of contacts with I-individuals is given by $\sum_{n=0}^{+\infty} nS_n(t)$ and its variation is

$$\frac{d}{dt}\sum_{n=0}^{+\infty} nS_n(t) = v_c \frac{I(t)}{N}\left[\sum_{n=1}^{+\infty} n(S_{n-1}(t) - S_n(t))\right]$$

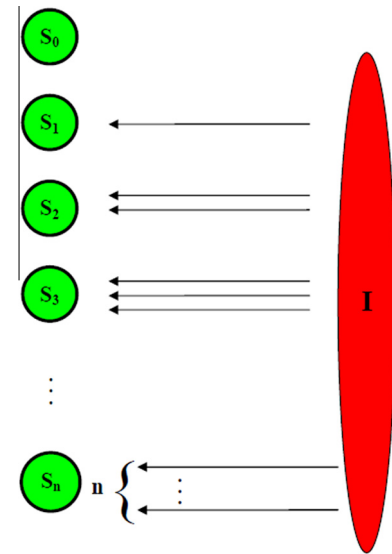$$= v_c \frac{I(t)}{N}\left[\sum_{n=0}^{+\infty}(n+1)S_n(t) - \sum_{n=1}^{+\infty} nS_n(t)\right].$$

Hence

$$\frac{d}{dt}\sum_{n=0}^{+\infty} nS_n(t) = v_c \frac{I(t)}{N}S(t).$$

Now we shall remember that the variation of the number of contacts with I-individuals is $v_c I$. Since by assumption I-individuals are choosing their new contacts randomly, we shall consider the probability of finding an S-individual within the population (namely $\frac{S}{N}$). Therefore the rate at which S-individuals gain contacts with I-individuals must be $v_c I \frac{S}{N}$. This fully justifies model (4.2).

We also make the following additional assumption.

Loss of contacts: Assume that, at any given time $t \geqslant 0$, each S-individual can lose at most one contact with an I-individual.

Fig. 9. Structured diagram of the population in term of contacts indicating the number of S-individuals chosen for a contact by I-individuals. S_n is the group of S-individuals which has been chosen n times by I-individuals, where $n = 0, 1, 2, \ldots$

Then the model describing the loss of contacts of S-individuals with I-individuals is given by the following infinite system of ordinary differential equations:

$$\begin{cases} S'_0 = v_c[S_1(t)] \\ S'_1 = v_c[2S_2(t) - S_1(t)] \\ S'_2 = v_c[3S_3(t) - 2S_2(t)] \\ \ldots \\ S'_n = v_c[(n+1)S_{n+1}(t) - nS_n(t)] \\ \ldots \end{cases} \qquad (4.3)$$

Some explanations are in order at this level. Consider an S-individual with n contacts. Let $\tau_1, \tau_2, \ldots, \tau_n$ be the random variable durations of contact. Then each random variable τ_i follows an exponential law with mean $1/v_c$. Moreover, since $\tau_1, \tau_2, \ldots, \tau_n$ are independent random variables, the probability is

$$P(\tau_1 \in [t, +\infty), \tau_2 \in [t, +\infty), \ldots, \tau_n \in [t, +\infty))$$

$$= \underbrace{\exp(-v_c t) \ldots \exp(-v_c t)}_{n \text{ times}} = \exp(-nv_c t).$$

This justifies the term $-v_V n S_n(t)$ in the model, where $v_V n$ is the rate at which S_n-individuals are losing a contact with an I-individual.

4.2. The SIR model

The SIR model can be derived as before. Taking into account the fact that the flux of an S-individual losing one contact is given by $v_c \sum_{n=1}^{+\infty} n S_n(t)$, the flux of an S-individual becoming infective is given by $v_c p_I \sum_{n=1}^{+\infty} n S_n(t)$, and using Assumption 3.1 again, we obtain the following SIR model:

$$\begin{cases} S_0' = v_c\left[(1-p_I)S_1(t) - \frac{I(t)}{N}S_0(t)\right] + \eta_R S_1(t) \\ S_1' = v_c\left[\frac{I(t)}{N}S_0(t) + (1-p_I)2S_2(t) - S_1(t) - \frac{I(t)}{N}S_1(t)\right] + \eta_R[2S_2(t) - S_1(t)] \\ \cdots \\ S_n' = v_c\left[\frac{I(t)}{N}S_{n-1}(t) + (1-p_I)(n+1)S_{n+1}(t) - nS_n(t) - \frac{I(t)}{N}S_n(t)\right] + \eta_R[(n+1)S_{n+1}(t) - nS_n(t)] \\ \cdots \\ I' = v_c p_I \sum_{n=1}^{+\infty} n S_n(t) - \eta_R I(t) \\ R' = \eta_R I(t). \end{cases} \tag{4.4}$$

The flowchart of model (4.4) is described in Fig. 10.

One may first observe that the SIR model (4.4) contains the usual term $\eta_R I$ in both the I-equation and the R-equation. This term describes the fact that the time spend by individuals in the infective class follows an exponential law with mean $T_R = 1/\eta_R$. However, since the I-individuals are becoming R-individuals, the numbers of S_n-individuals (for $n \geqslant 1$) are also affected by this process. Remembering that S_n is the number of S-individuals who have been chosen n times by an I-individual, the fact that some I-individuals are leaving will also induce a flux from the class S_n into the class S_{n-1}. By using the same idea as the one used above for the loss of contacts, we deduce that the term $\eta_R[(n+1)S_{n+1}(t) - nS_n(t)]$ is needed in the S_n-equation.

By renormalizing the distributions, namely by setting

$$s_n = \frac{S_n}{N}, \quad i = \frac{I}{N} \quad \text{and} \quad r = \frac{R}{N},$$

we obtain the system

$$\begin{cases} s_0' = v_c[(1-p_I)s_1(t) - i(t)s_0(t)] + \eta_R[s_1(t)] \\ s_1' = v_c[i(t)s_0(t) + (1-p_I)2s_2(t) - s_1(t) - i(t)s_1(t)] + \eta_R[2s_2(t) - s_1(t)] \\ \cdots \\ s_n' = v_c[i(t)s_{n-1}(t) + (1-p_I)(n+1)s_{n+1}(t) - ns_n(t) - i(t)s_n(t)] + \eta_R[(n+1)s_{n+1}(t) - ns_n(t)] \\ \cdots \\ i' = v_c p_I \sum_{n=1}^{+\infty} n s_n(t) - \eta_R i(t) \\ r' = \eta_R i(t). \end{cases} \tag{4.5}$$

Moreover, we have

$$s_n \geqslant 0, \quad \forall n \geqslant 0$$

and

$$\sum_{n=0}^{+\infty} s_n + i + r = 1.$$

Furthermore, the quantity $\frac{\sum_{n=0}^{+\infty} n s_n}{\sum_{n=0}^{+\infty} s_n}$ is the average number with an I-individual. Therefore, it is natural to impose that

$$\sum_{n=0}^{+\infty} n s_n < +\infty.$$

The analysis of model (4.5) will be presented elsewhere. We refer to [42] for results about the well posedness of similar classes of infinite differential equations.

4.3. Formal singular limit to the classical SIR model

By using again the rescaling

$$v_c = \frac{1}{\varepsilon}, \quad p_I = \beta_I \varepsilon,$$

where $\varepsilon \in (0, \beta_S^{-1}]$ is supposed to be a small parameter of the system, then system (4.5) can be rewritten as

$$\begin{cases} s_0' = \frac{1}{\varepsilon}[(1-\beta_I\varepsilon)s_1(t) - i(t)s_0(t)] + \eta_R[s_1(t)] \\ s_1' = \frac{1}{\varepsilon}[i(t)s_0(t) + (1-\beta_I\varepsilon)2s_2(t) - s_1(t) - i(t)s_1(t)] + \eta_R[2s_2(t) - s_1(t)] \\ \cdots \\ s_n' = \frac{1}{\varepsilon}[i(t)s_{n-1}(t) + (1-\beta_I\varepsilon)(n+1)s_{n+1}(t) - ns_n(t) - i(t)s_n(t)] + \eta_R[(n+1)s_{n+1}(t) - ns_n(t)] \\ \cdots \\ i' = \beta_I \sum_{n=1}^{+\infty} n s_n(t) - \eta_R i(t) \\ r' = \eta_R i(t). \end{cases} \tag{4.6}$$

This singular perturbation problem describing the convergence of the above problem to the classical SIR model turns to be technical since

$$\alpha_\varepsilon := (1 - \beta_I \varepsilon) \to 1 \quad \text{as } \varepsilon(>0) \to 0.$$

Here we consider this problem formally.

Formal singular limit. Set $\varepsilon = 0$ in system (4.6). Then we obtain

$$\begin{cases} 0 = [s_1(t) - i(t)s_0] \\ 0 = [i(t)s_0(t) + 2s_2(t) - s_1(t) - i(t)s_1(t)] \\ 0 = [i(t)s_1(t) + 3s_3(t) - 2s_2(t) - i(t)s_2(t)] \\ \cdots \\ 0 = [i(t)s_{n-1}(t) + (n+1)s_{n+1}(t) - ns_n(t) - i(t)s_n(t)] \\ \cdots \\ i' = \beta_I \sum_{n=1}^{+\infty} n s_n(t) - \eta_R i(t) \\ r' = \eta_R i(t). \end{cases} \tag{4.7}$$

Hence the susceptible distribution must satisfy

$$\begin{cases} i s_0 = s_1 \\ (i+1)s_1 = is_0 + 2s_2 \\ (i+2)s_2 = is_1 + 3s_3 \\ \cdots \\ (i+n)s_n = is_{n-1} + (n+1)s_{n+1} \\ \cdots \end{cases}$$

Therefore, we have

$$s_1 = i s_0$$

$$s_2 = \frac{1}{2}[(i+1)s_1 - is_0] = \frac{[(i+1)i - i]}{2}s_0 = \frac{i^2}{2}s_0$$

$$s_3 = \frac{1}{3}[(i+2)s_2 - is_1] = \frac{1}{3}\left[(i+2)\frac{i^2}{2} - i^2\right] = \frac{i^3}{3!}s_0$$

$$\cdots$$

$$s_n = \frac{1}{n}[(i+(n-1))s_{n-1} - is_{n-2}] = \frac{1}{n}\left[(i+(n-1))\frac{i^{n-1}}{(n-1)!} - \frac{i^{n-1}}{(n-2)!}\right] = \frac{i^n}{n!}s_0$$

$$\cdots$$

By induction we obtain

$$s_n = \frac{i^n}{n!}s_0, \quad \forall n \geqslant 0.$$

Thus the fraction of susceptible is

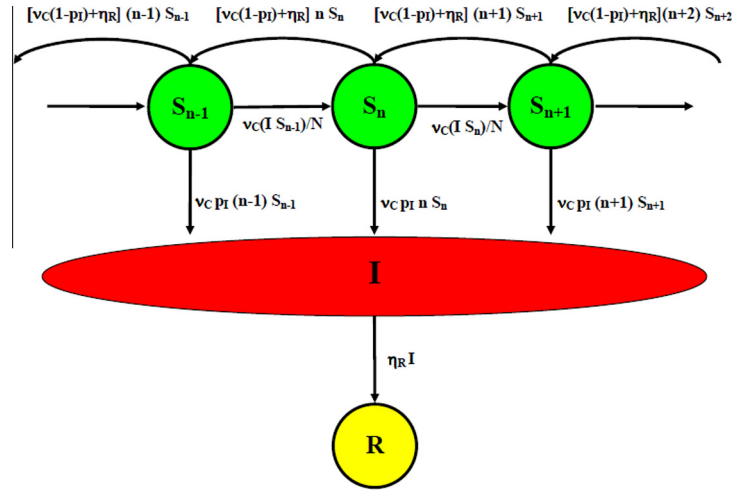$$s := \sum_{n=0}^{+\infty} s_n = \sum_{n=0}^{+\infty} \frac{i^n}{n!}s_0 = s_0 \exp(i).$$

Fig. 10. Flux diagram of model (4.4).

Since $s + i + r = 1$, we obtain

$$s_0 = s \exp(-i) = (1 - i - r) \exp(-i).$$

It follows that $\frac{s_n}{s}$ is a Poisson distribution with parameter i, which means that

$$\frac{s_n}{s} = \frac{i^n}{n!} \exp(-i), \quad \forall n \geqslant 0.$$

We can interpret the quantity

$$s_n = \frac{i^n}{n!}(1 - (i + r)) \exp(-i) \tag{4.8}$$

as the probability that an S-individual has been chosen by n I-individuals.

Since s_n is the proportion of S-individuals which have been chosen by n I-individuals, the average number of contacts per S-individual is

$$\sum_{n=1}^{+\infty} n s_n = \left(\sum_{n=0}^{+\infty} \frac{i^n}{n!} s_0 \right) i = si.$$

Hence, formally as the singular limit, we obtain the SIR type model

$$\begin{cases} s' = -\beta_I si \\ i' = \beta_I si - \eta_R i \\ r' = \eta_R i. \end{cases} \tag{4.9}$$

4.4. Numerical simulations

In order to perform numerical simulations of system (4.4), we truncate the system at the order $(n^* + 1) > 1$. That is, we neglect the terms $S'_k(t)$ for $k \geqslant (n^* + 2)$ and consider the following system:

$$\begin{cases} S'_0 = v_c \left[(1 - p_I) S_1(t) - \frac{I(t)}{N} S_0(t) \right] + \eta_R S_1(t) \\ S'_1 = v_c \left[\frac{I(t)}{N} S_0(t) + (1 - p_I) 2 S_2(t) - S_1(t) - \frac{I(t)}{N} S_1(t) \right] + \eta_R [2 S_2(t) - S_1(t)] \\ \cdots \\ S'_{n^*-1} = v_c \left[\frac{I(t)}{N} S_{n^*-2}(t) + (1 - p_I) n^* S_{n^*}(t) - (n^*-1) S_{n^*-1}(t) - \frac{I(t)}{N} S_{n^*-1}(t) \right] + \eta_R [n^* S_{n^*}(t) - (n^*-1) S_{n^*-1}(t)] \\ S'_{n^*} = v_c \left[\frac{I(t)}{N} S_{n^*-1}(t) - n^* S_{n^*}(t) - \frac{I(t)}{N} S_{n^*}(t) \right] - \eta_R [n^* S_{n^*}(t)] \\ I' = v_c p_I \sum_{k=1}^{n^*} k S_k(t) - \eta_R I(t) \\ R' = \eta_R I(t). \end{cases} \tag{4.10}$$

At time $t = 0$, we will take the following initial distributions to simulate the above model

$$N > I > 0, \quad R = 0$$

and

$$S_n = N \frac{i^n}{n!} \exp(-i)(1 - i), \quad \forall n = 0, \ldots, n^*,$$

with

$$i = \frac{I}{N}.$$

As before, we need to specify further the rules of transmission between an S-individual and an I-individual.

Assumption 4.1. We further assume that

(a) **(New SIR model)** For a given pair of (S, I)-individuals in contact, if the individual initiating the contact is an I-individual, then the transmission only occurs at the end of the contact with the probability p_I.

(b) **(Classical SIR Model)** For a given pair of (S, I)-individuals in contact, if the individual initiating the contact is an I-individual, then the transmission only occurs at the beginning of the contact with the probability p_I.

We will use Assumption 4.1(a) to run an IBM (called here IBM$_{12}$) which corresponds to the new model (4.10), while Assumption 4.1(b) will be used to run an IBM (called here IBM$_{22}$) which corresponds to the classical SIR model (1.1). The comparison between the new model (4.10) and the classical SIR model (1.1) with $\beta = \beta_I$ is given in Fig. 11. We can observe numerically the convergence of the new SIR model (4.10) to the classical SIR model (1.1) when T_C goes to zero and $p_I = \beta_I T_C$. Similarly, we can also see that the curve of the recovereds is influenced by the parameter T_C. Let R_∞ the limit of $R(t)$ when t goes $+\infty$. In (a) the total numbers of cases for the new SIR model (4.10) and the classical SIR model (1.1) are different. Once again one must be very careful in using IBM to simulate an epidemic, since replacing one model by the other might lead to a large bias in prediction. Here $S/N = 0.9, I/N = 0.1, S_c = I_c = SI/N$ at time $t = 0$. $T_R = 10$ and $\beta_I = 0.2$ are fixed and in (a) (resp. (b)) $T_C = 5$ and $p_I = \beta_I T_C = 1$ (resp. $T_C = 0.5$ and $p_I = \beta_I T_C = 0.1$) are also fixed.

The comparisons of the IBM, the new SIR model (4.10), and the classical SIR model (1.1) are given in Fig. 12. Here $S/N = 0.9, I/N = 0.1, S_c = I_c = SI/N$ at time $t = 0$. $T_R = 10, \beta_I = 0.2, T_C = 5$, and $p_I = \beta_I T_C = 1$ are fixed. In (a) (resp. (b)) the IBM$_{12}$ was run to simulate the transmission at the end of the contact for $N = 100$ (resp.
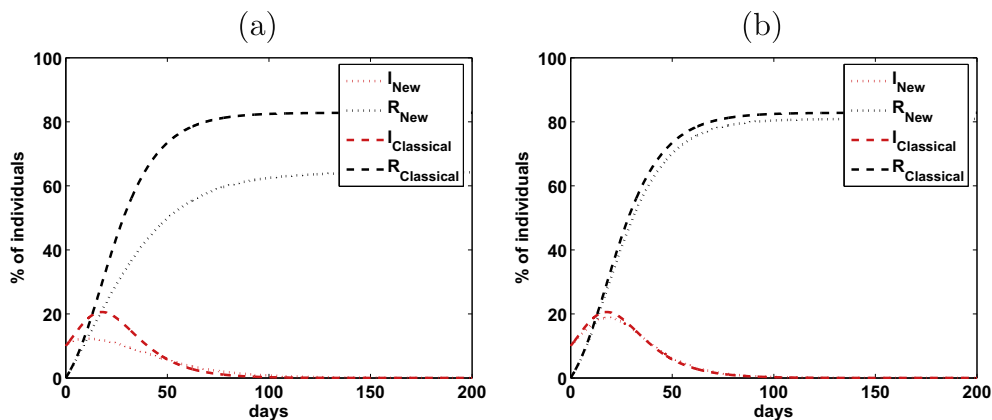
$N = 10,000$). In (c) (resp. (d)) the IBM$_{22}$ was run to simulate the transmission at the beginning of the contact for $N = 100$ (resp. $N = 10,000$).
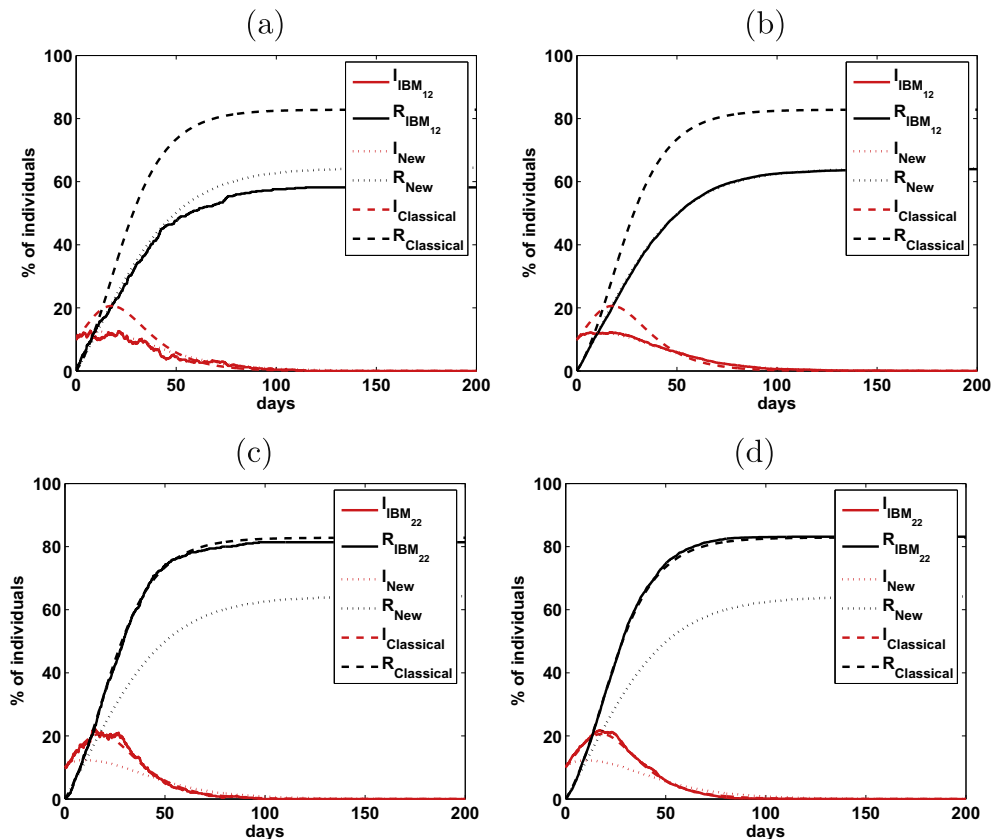
5. Discussion

Stochastic individual-based models (IBM) use continuum dynamics to track relatively small numbers of individuals based on the change rates of time and view individuals as individual agents whose status changes based on probabilistic events occurring over time. Such models are particularly suitable to describe the transmission dynamics of infectious diseases in a small population in which the individual behavior plays an important role in the spread of diseases. Most stochastic IBM simulations are based on the framework of certain deterministic epidemic models, in particular the classical susceptible-infective-recovered (SIR) model.

The purpose of this article was to compare the stochastic IBM and the classical SIR model and to examine how the behavior at

Fig. 11. Comparison of the new SIR model (4.10) (dotted curves) and the classical SIR model (1.1) (dashed curves) with $\beta = \beta_S$. (a) The total number of recovered individuals in the new and classical SIR models are significantly different as time increases. (b) The solution of the new SIR model converges to the solution of the classical SIR model. Here $S/N = 0.9, I/N = 0.1, S_c = I_c = SI/N$ at time $t = 0, T_R = 10$ and $\beta_I = 0.2$ (a) $T_C = 5$ and $p_I = \beta_I T_C = 1$. (b) $T_C = 0.5$ and $p_I = \beta_I T_C = 0.1$

Fig. 12. Comparison of the IBM (solid curves), the new SIR model (4.10) (dotted curves), and the classical SIR model (1.1) (dashed curves) with $\beta = \beta_I, S/N = 0.9, I/N = 0.1, S_c = I_c = SI/N$ at time $t = 0$. $T_R = 10, \beta_I = 0.2, T_C = 5$, and $p_I = \beta_I T_C = 1$ are fixed. In (a) and (b) the IBM$_{12}$ was run to simulate the transmission at the end of the contact for $N = 100$ and $N = 10000$, respectively. In (c) and (d) the IBM$_{22}$ was run to simulate the transmission at the beginning of the contact for $N = 100$ and $N = 10000$, respectively.

the individual level affects the eventual transmission dynamics of infectious diseases at the population level. We first made some assumptions about the rules of contacts between susceptible and infective individuals, the rules of transmission of diseases through these contacts, and the time of transmission during contacts. For the sake of comparison, we distinguished two types of transmission processes: that initiated by susceptible individuals and that driven by infective individuals. We then studied the transmission driven only by the susceptible individuals and compared the numerical simulations between the IBM and the classical SIR models. The transmission initiated only by infective individuals was also modeled and analyzed and the comparison of stochastic IBM simulations and the classical SIR model was presented. Our analysis and simulations demonstrate that the IBM converges to the classical SIR model only in some particular situations. In general, the individual-based and the classical SIR models are significantly different. Moreover, our study reveals that the timing of transmission in a contact at the individual level plays a crucial role in the transmission dynamics of a disease at the population level.

Stochastic SIR epidemics models have been studied extensively and we refer to [2,3,5,12] (and references therein) for details on stochastic epidemic models. However, as far as we understand the derivations of most of these stochastic SIR models are not very clear and poorly understood using individual stochastic rules. This is left for future consideration.

There are several issues which deserve further investigation. Firstly, the stochastic process introduced in Section 2 needs to be studied in detail. Secondly, we assumed that the time of transmission of diseases was either at the beginning or at the end of a contact, in reality the time of transmission probably occurs at certain moment between the beginning and the end of a contact. Therefore, modeling the disease transmission with such a timing of transmission and comparing these models is useful. Thirdly, both the spatial structure and the age of infection play very important roles in the spread of infectious diseases, it will be very interesting to include these features into the individual stochastic modeling.

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