STATISTICAL

Stochastic Approach to Epidemic Spreading

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Abstract

We analyze four models of epidemic spreading using a stochastic approach in which the primary stochastic variables are the numbers of individuals in each class. The stochastic approach is described by a master equation and the transition rates for each process such as infection or recovery are set up by using the law of mass action. We perform numerical simulations as well as numerical integration of the evolution equations for the average number of each class of individuals. The onset of the epidemic spreading is obtained by a linear analysis of the disease free state, from which follows the initial exponential increase of the infected and the frequency of new cases. The order parameter and the variance in the number of individuals are also obtained characterizing the onset of epidemic spreading as a critical phase transition.

Keywords Stochastic epidemic models · Epidemic spreading models · SIR model · SIS model

1 Introduction

The theoretical study of the epidemic spreading [1–5] started with the employment of ordinary differential equations of the first order in time, which became known as the deterministic approach [1]. The individuals of a population are classified in accordance with their condition in relation to the infectious disease and these equations give the evolution equations on the number of individuals belonging in each class. The deterministic approach, however, does not describe, in an explicit manner, the random fluctuations occurring in a real epidemic spreading. This observation may have given way to the need of a stochastic approach to the epidemic spreading as that developed by Bartlett [6, 7] and by Bailey [8, 9].

A stochastic version of the deterministic model proposed by Kermack and McKendrick [10] was developed by Bartlett in 1949 [7]. The model, called susceptible-infective-removed, describes the spread of an infectious disease in a community of individuals who acquire permanent immunization. There are three classes of individuals: the susceptible, the infective, and the recovered. The approach advanced by Bartlett treated the numbers of individuals in each class as stochastic variables from which he developed a time evolution equation for the generating function corresponding to the probability distribution of these variables.

The evolution equation for the probability distribution, or master equation, of the model analyzed by Bartlett was obtained by Bailey [9]. The stochastic approach they employed was based on the use of a continuous time Markov process in a discrete space in which the variables increase or decrease by one unit. In 1955, Whittle [11] presented a stochastic version of the Kermack and McKendrick theorem [1, 10] concerning the outbreak of an epidemic. According to this theorem, if the density of the susceptible is smaller than a certain value, the epidemic does not outbreak.

Stochastic versions of deterministic models can be obtained by transforming the numbers of individuals in each class into stochastic variables, as was the case of the deterministic susceptible-exposed-infective-removed model proposed by Dietz [12] which was transformed into a stochastic model allowing its Monte Carlo simulation [13]. One way of achieving the stochastic versions is to set up a master equation in which case one is left with the problem of finding the transition rates. Another way is to add noise in the deterministic equations, transforming them into Langevin equations. In this case, the problem is reduced to finding the appropriate type of noise. The transition rates and noises, once established, lead to the several approaches used in the study of epidemic and population models [14–20].

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The approach we use here to analyze four epidemic models considers the number of individual in each class as the primary stochastic variables. It is based on the use of a master equation and on the law of mass action to set up the transition rates. This is accomplished by using the analogy of the processes in which the individuals change classes with chemical reactions. After that, an expansion method was used to transform the master equation into a Fokker-Planck equation [21, 22].

More detailed stochastic approaches can be conceived if one wishes to take into account the spatial structure where the individuals live. In this case, we may, for instance, associate to each individual a stochastic variable that takes values corresponding to the condition of an individual in relation to the disease. This will not be pursued here but models of this type have in fact been studied by several authors owing to their relevance to the spreading of disease in space and because of their critical behavior [23–34].

2 Evolution Equations

2.1 Master Equation

The description of the time evolution of a system by a stochastic approach needs first of all the specification of the variables that will be used as primary stochastic variables. A detailed approach such as that employed in spatial stochastic model could be used. Here, we follow a less detailed approach, which uses as primary stochastic variables the numbers of individuals belonging in each class. A class of individuals is its condition with respect to the infectious disease that we are about to study. Examples are the classes of susceptible, infected, removed, and exposed.

To properly set up the stochastic approach, we start by considering that the individuals of a community interact with each other in such a way that the epidemic will spread in the population. One individual does not interact with every person of the community but interacts with a certain number N of individuals, which is not small but is smaller than the total number of individuals of the community. In accordance with the approach we will use, it suffices to focus on a neighborhood with N individuals. Its reciprocal $\varepsilon = 1/N$ is understood as a parameter of the present stochastic approach.

We denote by n_i the number of individuals of the ith class within the neighborhood, and by n the vector whose components are the variables n_i. The vector n is identified as a state of the system. At each time step of the dynamics, the state n changes to a new value n' and the stochastic dynamics becomes defined by the transition rates $W_r(n'|n)$ from state n to state n' corresponding to each process involving the change of an individual class. The equation

that governs the evolution of the probability distribution $P(n, t)$ of n at time t, the master equation, is [22, 35]

$$\frac{d}{dt} P(n) = \sum_r \sum_{n'} \{ W_r(n|n') P(n') - W_r(n'|n) P(n) \}, \quad (1)$$

where the first summation is over the several processes and the second summation is over the variables n_i' of all classes.

Next, we have to set up the transition rates. To this end, we use the analogy of the present problem with that of chemical kinetics. A class of individuals is analogous to a chemical species, and a process of changing class is analogous to a chemical reaction. As an example of the analogy, we consider the process that is always present in the evolution of an infectious disease. It is the process of infection of a susceptible (S) individual, who becomes exposed (E), by an infective (I) individual, represented by

$$S \xrightarrow{I} E, \quad (2)$$
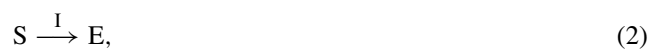
and understood as the catalytic reaction that transforms an S into one E by the catalyst I. In this reaction, the number n_1 of the susceptible decreases by one unit, the number of the infective n_2 remains invariant, and the number of the exposed increases by one unit. The infection transition rate is

$$W_{\text{inf}} = bN \left(\frac{n_1}{N} \right) \left(\frac{n_2}{N} \right), \quad (3)$$

where b is the infection rate constant.

If the product of the reaction in (2) is the catalyst itself, that is,

$$S \xrightarrow{I} I, \quad (4)$$

then the reaction is auto-catalytic, but the infection rate is still given by (3).

Another example is the process in which an infective (I) becomes recovered (R), represented by the spontaneous reaction

$$I \longrightarrow R, \quad (5)$$

in which the number of infected n_2 decreases by one unit and the number of recovered increases by one unit. The recovered transition rate is

$$W_{\text{rec}} = cN \left(\frac{n_2}{N} \right), \quad (6)$$

where c is the recovery rate constant.

The rule that we use to set up a transition rate W_r, which is the reaction rate corresponding to a certain reaction, is understood as the application of the law of mass action [22], and is given by $W_r = N w_r$ where

$$w_r = k_r q_r, \quad (7)$$

where k_r is the rate constant, and q_r is the product of the fractions n_i/N of each class of individual appearing as a reactant, including the catalyst if the reaction is catalytic.

2.2 Simulation

Let us discretize the time in intervals equal to τ. If we denote by $P(n)$ and $P'(n)$ the probability distribution at time t and $t + \tau$, respectively, then the master equation can be written in the discretize form as

$$P'(n) = \sum_{n'} T(n'|n) P(n), \qquad (8)$$

where

$$T(n'|n) = \sum_{r} p_r q_r(n'|n), \qquad (9)$$

$p_r = \tau k_r N$, and the sum of p_r equals 1.

The numerical simulation of the master equation is carried out as follows. At each time step, we choose which reaction to perform. The reaction is chosen with a probability p_r which, as we have seen above, is proportional to the corresponding reaction rate constant k_r. After the reaction has been chosen, it will be in fact executed with a probability equal to q_r. If this is the case, then the numbers n_i will change according to the chosen reaction. This procedure is repeated a number of times and a sequence of states is generated, starting from an initial state.

2.3 Fokker-Planck Equation

According to the law of mass action, the transition rate $W_r(n'|n)$ associated with a certain reaction is always written as $W_r = N w_r$ where w_i is a fraction or a product of fractions $x_i = n_i/N$. In the example given by (3), $w_{\text{inf}} = b x_1 x_2$ and in the example given by (6), $w_{\text{rec}} = c x_2$. This allows us to write the the master (1) in terms of x,

$$\frac{d}{dt}\rho(x) = N \sum_{r}\sum_{x'} \{w_r(x|x')\rho(x') - w_r(x'|x)\rho(x)\}. \quad (10)$$

Usually, the transition rates $W_r(n'|n)$ are such that the differences $n'_i - n_i$ are small numbers, and in fact, in the cases that we consider here the differences are ± 1 or 0. This means that the difference $x'_i - x_i$ is of the order $\varepsilon = 1/N$, a result that allows us to expand the quantities on the right-hand side of (10), around the state x. Performing this expansion up to second order in ε, the result is the following Fokker-Planck equation

$$\frac{\partial \rho}{\partial t} = -\sum_{i}\frac{\partial f_i \rho}{\partial x_i} + \frac{\varepsilon}{2}\sum_{ij}\frac{\partial^2 h_{ij}\rho}{\partial x_i \partial x_j}, \qquad (11)$$

where f_i and h_{ij} are functions of x determined from the transition rates. The first is related to w_r by

$$f_i = \sum_{r} v_{ir} w_r, \qquad (12)$$

where the coefficient v_{ir} is the variation of n_i in the reaction r, and the second is related to w_r by

$$h_{ij} = \sum_{r} v_{ir} v_{jr} w_r. \qquad (13)$$

We point out that the Fokker-Planck (11) is equivalent to the set of Langevin equations

$$\frac{dx_i}{dt} = f_i + \xi_i, \qquad (14)$$

where ξ_i are stochastic variables with the following properties: $\langle \xi(t) \rangle = 0$ and

$$\langle \xi_i(t)\xi_j(t') \rangle = \varepsilon h_{ij}\delta(t - t'). \qquad (15)$$

As h_{ij} may depend on x_i, the random variables ξ_i represent a multiplicative noise.

2.4 Evolution of the Averages

The time evolution of the averages of the various quantities is obtained from the Fokker-Planck as follows. Let us consider the average

$$\langle x_i \rangle = \int x_i \rho \, dx. \qquad (16)$$

We multiply both sides of the Fokker-Planck equation by x_i and integrate in x to get

$$\frac{d}{dt}\langle x_i \rangle = \langle f_i \rangle, \qquad (17)$$

where we have performed appropriate integration by parts and considered that ρ vanishes quickly as the limits of the integral is approached.

Next, we determine the time evolution of the covariances $C_{ij} = \langle x_i x_j \rangle - \langle x_i \rangle\langle x_j \rangle$. To this end, we find first the time evolution of the average $\langle x_i x_j \rangle$. We proceed in the same way as above to get the result

$$\frac{d}{dt}\langle x_i x_j \rangle = \langle x_i f_j \rangle + \langle x_j f_i \rangle + \varepsilon \langle h_{ij} \rangle, \qquad (18)$$

from which we find, with the help of (17)

$$\frac{d}{dt}C_{ij} = \langle x_i f_j \rangle - \langle x_i \rangle\langle f_j \rangle + \langle x_j f_i \rangle - \langle x_j \rangle\langle f_i \rangle + \varepsilon \langle h_{ij} \rangle. \quad (19)$$

(17) does not consist of a closed set of equations for the averages $\langle x_i \rangle$. However, if ε is small, we may replace the average $\langle f(x) \rangle$ by $f(\langle x \rangle)$ on the right-hand side of (17) and the set of equations become closed. The corrections will be of the order ε and can thus be neglected. The reasoning to reach this result is as follows. In the limit $\varepsilon \to 0$, the probability distribution ρ becomes sharped around x_i, giving way to assume that it is a Gaussian distribution with mean $\langle x_i \rangle$ and covariances C_{ij}, proportional to ε. This

assumption allows to replace x in the average $\langle f_i(x) \rangle$ by $f(\langle x \rangle)$ so that (17) becomes the equation

$$\frac{d}{dt}\bar{x} = f_i(\bar{x}), \tag{20}$$

where we used the simplified notation $\bar{x}_i = \langle x_i \rangle$. We see that the evolution (20) is now closed equation for the averages $\bar{x}_i$.

Now we use the assumption that the distribution is a sharped Gaussian distribution to determine the first terms on the right-hand side of (19). But before we expand

$$\langle x_i f_j \rangle - \langle x_i \rangle \langle f_j \rangle = \sum_k f_{jk}(\bar{x}) C_{ik}, \tag{21}$$

$$\langle x_j f_i \rangle - \langle x_j \rangle \langle f_i \rangle = \sum_k f_{ik}(\bar{x}) C_{jk}, \tag{22}$$

where $f_{jk} = \partial f_j / \partial x_k$. Replacing these results in (19), we find

$$\frac{d}{dt} C_{ij} = \sum_k \{ f_{jk}(\bar{x}) C_{ik} + f_{ik}(\bar{x}) C_{jk} \} + \varepsilon h_{ij}(\bar{x}), \tag{23}$$

which is the equation that determines C_{ik} once we have determined $\bar{x}_i$, and confirms that the variances are indeed proportional do ε. Due to this dependence, it is convenient to define a reduced covariance χ_{ij} by $C_{ij} = \varepsilon \chi_{ij}$, which obeys the equation

$$\frac{d}{dt} \chi_{ij} = \sum_k \{ f_{jk}(\bar{x}) \chi_{ik} + f_{ik}(\bar{x}) \chi_{jk} \} + h_{ij}(\bar{x}). \tag{24}$$

The expansion in ε that we have carried out above allowed us to find the Fokker-Planck (11) and its associate Langevin (14), and to reach (20) and (24) by assuming that the solution of the Fokker-Planck equation is a Gaussian with variances proportional to ε. Such an expansion was possible because the transition rates w_r depend only on the fractions n_i/N, a result that follows from our use of the law of mass action. The expansion of the master equation in a small parameter was developed by van Kampen in 1961 by assuming that the solution of the master equation is a Gaussian with variances proportional to the expanding parameter [35–37]. It was applied to an epidemic model by McNeil [38] and also considered by Nisbet and Gurney [14] in population dynamics under the name of diffusion approximation.

3 Critical Behavior

The outbreak of an epidemic is characterized as being a critical event. If the density of infective individuals is small, there is no spread. But if the density increases, it will reach a critical density above which the epidemic spreads, the increase of the infectious individuals being exponential in time. This fundamental idea was used by Ross in his studies on the transmission of malaria [39, 40] and was introduced by Kermack and McKendrick in a clear form as the threshold theorem [1, 10].

To determine the onset of the spread, we perform a stability analysis of the disease free state, which is the state without infective individuals. This state is always present because the infective individuals are created catalytically. If the infective are absent, the system remains forever in the disease free state, and for this reason, it is called absorbing state in stochastic approaches.

In the present approach, the stability analysis can be performed by using the evolution equations for the fractions $\bar{x}_i$ because these equations are closed equations for these averages. We consider that the disease free state is a state full of susceptible individuals so that the fraction of the susceptible equals 1. The linearization of (20) gives

$$\frac{d\bar{x}_i}{dt} = \sum_j f_{ij} \, \bar{x}_i, \tag{25}$$

where $f_{ij} = \partial f_i / \partial x_j$ and is calculated at the disease free state. The equation for the susceptible is excluded from this set because (20) is not in fact all independent as the sum of the fractions $\bar{x}_i$ equals 1.

From the linearized equations, it follows that the time behavior of $\bar{x}_i$ is

$$\bar{x}_i = x_{i0} \, e^{\alpha t}, \tag{26}$$

where α is the largest eigenvalue of the matrix with elements f_{ij}. The onset of spreading occurs when $\alpha = 0$. When $\alpha > 0$, the increase in $\bar{x}_i$ is exponential.

The largest eigenvalue α of the stability matrix has a relationship with the reproduction number, used to characterize the epidemic spreading. This quantity is related to the number of individuals that can be infected by one individual in a population of susceptible. It is defined more precisely as follows. Let N_a be the number of new cases occurring in a time interval Δt, which is given by $N_a = N \bar{f} \Delta t$, where f is the frequency of new cases, that is, f is the fraction of individuals that are being infected per unit time.

The frequency of new cases comes from all reactions of the type

$$A \xrightarrow{I} B, \tag{27}$$

where A represents an individual free of disease and B an individual that has been infected. Since this reaction is catalytic and the infective is the catalyst, the reaction rate is proportional to the fraction of the infective. Therefore, f is proportional to the fraction of the infective y, that is, $f = gy$, where g depends on the fractions of the other classes but not on y.

Next, we have to determine the number N_b of infective individuals that have infected the N_a individuals in the
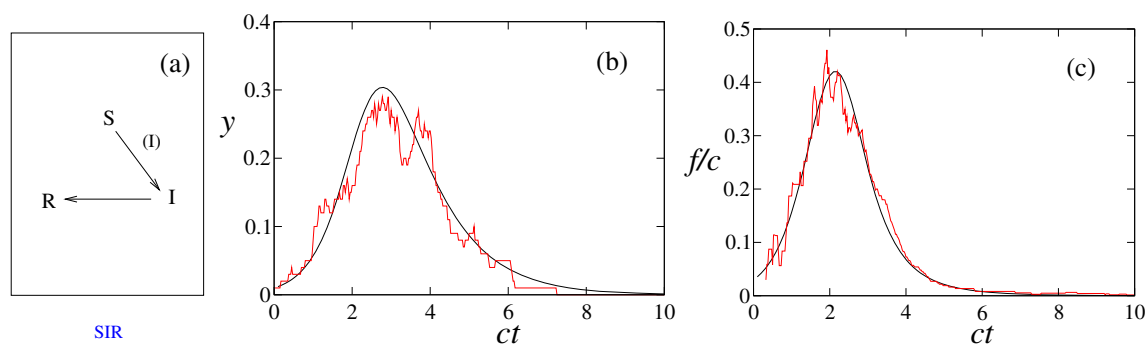
Fig. 1 SIR model. **a** The processes composing the model. **b** Fraction of infective individuals at a given time versus time t, obtained from numerical simulation of the master equation and its average. **c** Epidemic curve from simulation and its average. All curves were obtained for $b/c = 3$. The simulations were performed using $\varepsilon = 0.01$

interval Δt. If the number of infective remain the same in the interval Δt, then N_b would be equal to N_a. However, the number of infective may have increased by an amount N_c in the interval Δt, in which case $N_b = N_a - N_c$. As $N_c = N(d\bar{y}/dt)\Delta t$ and

$$N_b = N\bar{f}\Delta t, \tag{28}$$

we get

$$N_b = N\left(\bar{f} - N\frac{d\bar{y}}{dt}\right)\Delta t, \tag{29}$$

and the reproduction number $R = N_a/N_b$ becomes

$$R = \frac{\bar{f}}{\bar{f} - d\bar{y}/dt}. \tag{30}$$

In the early stages of the epidemic, the reproduction number is called basic reproduction number, denoted R_0. In this case, the fraction of infective behaves exponentially with time, $\bar{y} = y_0 e^{\alpha t}$ and

$$R_0 = \frac{\bar{f}}{\bar{f} - \alpha\bar{y}} = \frac{\bar{g}}{\bar{g} - \alpha}, \tag{31}$$

where the second equality was obtained by recalling that $\bar{f} = \bar{g}\bar{y}$, and $\bar{g}$ is calculated using the disease free solution. The onset of spreading occurs when $\alpha = 0$, that is, when $R_0 = 1$. When $\alpha > 0$, that is, when $R_0 > 1$, the epidemic spreads whereas when $\alpha < 0$, that is, when $R_0 < 1$, it does not. The epidemic spreading occurs when the basic reproduction number is larger than 1.

4 SIR Model

The susceptible-infective-recovered (SIR) model consists of three classes of individuals, susceptible, infective, and recovered. The recovered individuals acquire permanent immunization and cannot be infected again. The model has two processes. The first is the infection of a susceptible by

an infective, represented by the auto-catalytic reaction

$$S \xrightarrow{\text{I}} I, \tag{32}$$

occurring with an infection rate constant b, and the second is the spontaneous recovery of an infective, represented by

$$I \longrightarrow R, \tag{33}$$

occurring with a recovery rate constant c. In Fig. 1a, we show a representation of the model involving these two processes.

We denote by x, y, and z the fractions of the susceptible, the infected, and the recovered, respectively. The rate of the infection process is

$$w_{\text{inf}} = bxy, \tag{34}$$

whereas the rate of the recovery process is

$$w_{\text{rec}} = cy. \tag{35}$$

According to the rules above, the equations that give the time evolution of the averages $\bar{x}$, $\bar{y}$, and $\bar{z}$ are

$$\frac{d\bar{x}}{dt} = -b\bar{x}\bar{y}, \tag{36}$$

$$\frac{d\bar{y}}{dt} = b\bar{x}\bar{y} - c\bar{y}, \tag{37}$$

$$\frac{d\bar{z}}{dt} = c\bar{y}. \tag{38}$$

We remark that these three equations are not independent because $\bar{x} + \bar{y} + \bar{z} = 1$.

We have solved numerically this set of equation and obtained $\bar{x}$, $\bar{y}$, and $\bar{z}$ as functions of t. In Fig. 1b, we show $\bar{y}$ as a function of t together with y obtained from a simulation of the master equation obtained with $\varepsilon = 0.01$. The infective increases exponentially, reaches a maximum, and then decreases towards 0.

The fraction of individuals that are being infected per unit time f, or frequency of new cases, is obtained from the infection process (32) and is given by $f = bxy$. From the

Fig. 2 SIR model. **a** Order parameter s, which is the area under the epidemic curve, as a function of b/c. **b** Variance χ related to the susceptible as a function of b/c
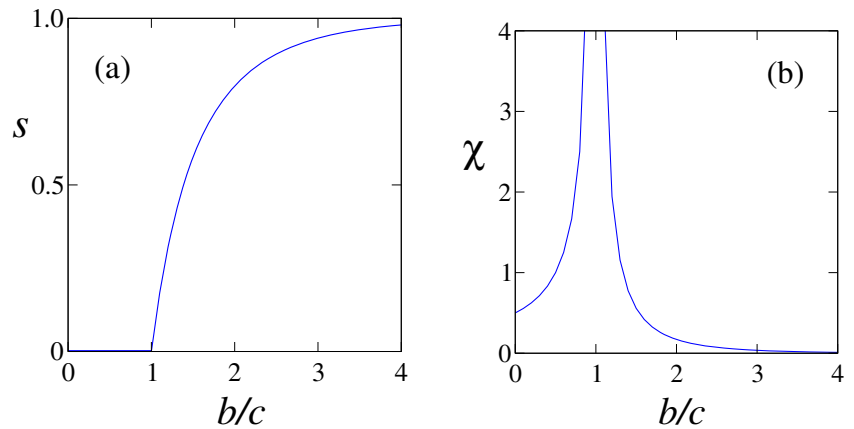
simulation, we have obtained f which is shown in Fig. 1c together with its average $\bar{f} = b\bar{x}\bar{y}$ as a function of time, the epidemic curve. The frequency of new cases increases exponentially, reaches a maximum, and then decreases towards 0, indicating that the disease became extinct.

The initial exponential increase in the fraction of infected, and thus in the frequency of new cases, is shown by a stability analysis of the disease free state. This state corresponds to the absence of disease, and all individuals are susceptible. That is, $\bar{x} = 1$, $\bar{y} = 0$, and $\bar{z} = 0$, which is a stationary solution of the set of equations above. As only two equations are independent, we will use only the last two, which after linearization gives

$$\frac{d\bar{y}}{dt} = \alpha\bar{y}, \tag{39}$$

$$\frac{d\bar{z}}{dt} = c\bar{y}, \tag{40}$$

where $\alpha = b - c$. The solution of the first equation gives

$$\bar{y} = y_0 e^{\alpha t}, \tag{41}$$

and we see that if $\alpha > 0$, then $\bar{y}$ increases exponentially. The value $\alpha = 0$, that is, $b = c$, gives the onset of the spread because if $\alpha < 0$, then $\bar{y}$ dies out.

As one increases the infection rate constant b, from a small value, it will reach a critical value $b_c = c$ at which the spread occurs. The order parameter s of the epidemic spreading phase is the area under the epidemic curve, that is,

$$s = \int_0^\infty \bar{f}\,dt. \tag{42}$$

In the present case, $\bar{f} = b\bar{x}\bar{y}$ and from (36) we see that $\bar{f} = -d\bar{x}/dt$ and we may conclude that

$$s = 1 - x^*, \tag{43}$$

where x^* denotes the value of $\bar{x}$ for long times and we have taken into account that at initial times $\bar{x}$ equals 1.

The basic reproduction number is obtained from (31) and considering that $\bar{f} = b\bar{x}\bar{y}$, we find

$$R_0 = \frac{b}{b - \alpha} = \frac{b}{c}, \tag{44}$$

where we have taken into account that for the disease free state $\bar{x} = 1$ and in the second equality we have used the result $\alpha = b - c$.

If we divide (38) and (36), we find

$$\frac{d\bar{z}}{d\bar{x}} = -\frac{c}{b\bar{x}}, \tag{45}$$

which after integrating gives

$$\bar{z} = -\frac{c}{b}\ln\bar{x}, \tag{46}$$

where the integration constant was found by using the disease free state $\bar{x} = 1$ and $\bar{z} = 0$. If we denote by x^*, y^*, and z^* the values of the fractions for large times, we see that $x^* + z^* = 1$ because $y^* = 0$. Therefore, an equation for z^* is obtained by replacing z by z^* and x by $1 - z^*$ in (46). But s equals $1 - x^* = z^*$ as we have seen above, so that

$$s = -\frac{c}{b}\ln(1 - s). \tag{47}$$

This equation gives the order parameter as a function of b and is shown in Fig. 2a. If $b \leq c$, s vanishes. For $b > c$, s is nonzero and for b near its critical value $b_c = c$, it is given by

$$s = \frac{2}{c}(b - c). \tag{48}$$

The order parameter s increases monotonically with infection rate b from its zero value at the critical point $b_c = c$, approaching the asymptotic value $z = 1$.

The use of a stochastic approach allows us to determine the fluctuations in the variables x, y, and z. A measure of the fluctuations are given by the covariances. Using the formula (24) for the reduced covariances, we find the following
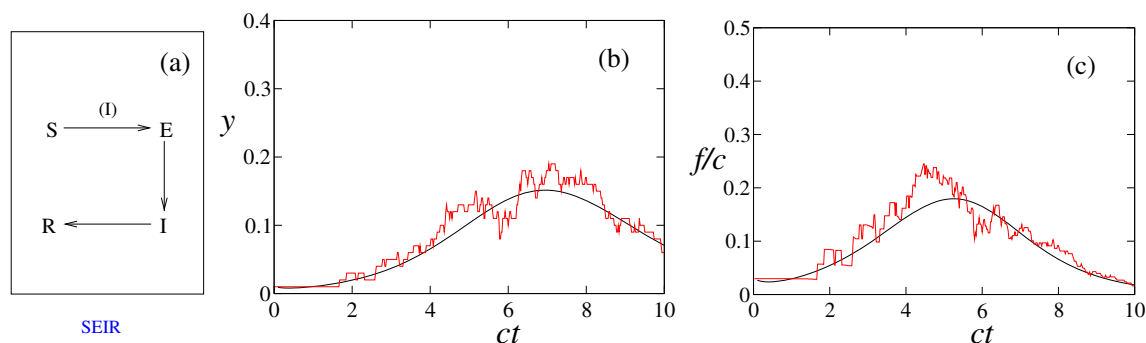
Fig. 3 SEIR model. **a** The processes composing the model. **b** Fraction of infective individuals at a given time versus time t, obtained from numerical simulation of the master equation and its average. The fraction of the infective vanishes for long times. **c** Epidemic curve, or frequency of new cases as a function of time, from simulation, and its average. All curves were obtained for $b/c = 3$ and latent period $\ell = c/k = 1$. The value of b/c is the same as that used in Fig. 1 but due to a nonzero value of the latent period the curves present a flattening when compared with those of Fig. 1. The simulations were performed using $\varepsilon = 0.01$

expression for the reduced variance χ of the fraction of the susceptible at the stationary state

$$\chi = \frac{c(1-s)}{2(bs - b + c)}. \tag{49}$$

A plot of χ versus b is shown in Fig. 2b. Near the critical point, it diverges as

$$\chi = \frac{c}{2|b-c|}. \tag{50}$$

5 SEIR Model

There are some diseases such that the susceptible individuals that have been infected take a certain time to be infective. These individuals, who have got the disease but are not capable of infect others, are called exposed. The model susceptible-exposed-infective-recovered (SEIR) is similar to the SIR model but there is an intermediate step before a susceptible becomes infective as shown in Fig. 3a. The process of infection is represented by

$$S \xrightarrow{\text{I}} E, \tag{51}$$

occurring with an infection rate constant b, the process of becoming infective is represented by

$$E \longrightarrow I, \tag{52}$$

occurring with a rate constant k, and the process of recovering is represented by

$$I \longrightarrow R, \tag{53}$$

occurring with a recovering rate constant c. The inverse of the rate constant k is a measure of the latent period ℓ of the exposed individual. When the latent period vanishes, $\ell = 0$, the present model reduces to the SIR model, in which a susceptible that has been infected becomes infective immediately.

We use the same notation as that of the SIR model, namely, x, y, and z for the fraction of susceptible, infective, and recovered, and u for the fraction of the exposed. The rate of the infection process is

$$w_{\text{inf}} = bxy, \tag{54}$$

the rate of the process becoming infective is

$$w_{\text{ive}} = ku, \tag{55}$$

and the rate of the recovery process is

$$w_{\text{rec}} = cy. \tag{56}$$

According to the rules, the evolution equation for the averages of these quantities are

$$\frac{d\bar{x}}{dt} = -b\bar{x}\bar{y}, \tag{57}$$

$$\frac{d\bar{u}}{dt} = b\bar{x}\bar{y} - k\bar{u}, \tag{58}$$

$$\frac{d\bar{y}}{dt} = k\bar{u} - c\bar{y}, \tag{59}$$

$$\frac{d\bar{z}}{dt} = c\bar{y}. \tag{60}$$

These equations are not all independent because $\bar{x} + \bar{u} + \bar{y} + \bar{z} = 1$.

We have solved numerically this set of equation and obtained $\bar{x}$, $\bar{y}$, $\bar{z}$, and $\bar{u}$. In Fig. 3b, we show $\bar{y}$ as a function of time together with y obtained from the simulation of the master equation with $\varepsilon = 0.01$. The infective growth exponentially attains a maximum and then decreases towards the zero value. In Fig. 3c, we show the frequency of new cases f which comes from the infective process (51) and is given $f = bxy$. Its average is $\bar{f} = b\bar{x}\bar{y}$ and is also shown in the same figure.

We determine now the conditions for the outbreak of the epidemic. To this end, we employ a stability analysis of the

disease free state, which is $\bar{x} = 1$, $\bar{y} = 0$, $\bar{z} = 0$, and $\bar{u} = 0$. After linearizing (58) and (59) become

$$\frac{d\bar{u}}{dt} = b\bar{y} - k\bar{u}, \tag{61}$$

$$\frac{d\bar{y}}{dt} = k\bar{u} - c\bar{y}. \tag{62}$$

Assuming solutions of the type $\bar{y} = y_0 e^{\alpha t}$ and $\bar{u} = u_0 e^{\alpha t}$, we find

$$-ku_0 + by_0 = \alpha u_0, \tag{63}$$

$$ku_0 - cy_0 = \alpha y_0, \tag{64}$$

which is a set of eigenvalues equations. The largest eigenvalue is

$$\alpha = \frac{1}{2}\{-(k+c) + \sqrt{(k-c)^{2+4bk}}\}. \tag{65}$$

The epidemic spreads when $\alpha > 0$ which occurs when $b > c$, and the threshold of spread occurs when $\alpha = 0$, that is, when $b = c$, results that are independent of k. We see that the process S→E, which occurs with a rate constant k, does not change the outbreak of the epidemic but yields a flattening of the epidemic curve as seen in Fig. 3.

Although the presence of a latent period induces a flattening of the epidemic curve, its area does not change and is the same as that of the SIR model. To show this result, we recall that the frequency of new cases is $\bar{f} = b\bar{x}\bar{y}$ and from (57) we see that $\bar{f} = -d\bar{x}/dt$. Therefore,

$$s = \int_0^\infty \bar{f} dt = 1 - x^*. \tag{66}$$

Now we have to show that x^* does not depend on k. Dividing (60) and (57),

$$\frac{d\bar{z}}{d\bar{x}} = -\frac{c}{b\bar{x}}, \tag{67}$$

which after integration gives

$$\bar{z} = -\frac{c}{b} \ln \bar{x}, \tag{68}$$

and we recall that $\bar{x} + \bar{y} + \bar{z} + \bar{u} = 1$. For large times, the infective as well as the exposed disappears, $y^* = 0$ and $u^* = 0$ and $x^* = 1 - z^*$. Replacing this last result in (68) we get an equation for x^* that does not depend on k. The equation for $s = 1 - x^*$ follows immediately and is

$$s = -\frac{c}{b} \ln(1 - s), \tag{69}$$

and does not depend on k and is the same as that of the SIR model.

As we have seen above, the frequency of new cases comes from the infection reaction (51) as is given by $f = $

bxy. The basic reproduction number is obtained from (31) and given by

$$R_0 = \frac{b}{b - \alpha}. \tag{70}$$

Replacing α given by (65), we obtain R_0 in terms of the rate constant b, c, and k. The onset of the epidemic spreading occurs when $\alpha = 0$, that is, when $R_0 = 1$. When $\alpha > 0$, the value of R_0 is greater than 1. It should be remarked that R_0 is smaller that the basic reproduction number for the SIR model. To reach this result, it suffices to recall that $\alpha_{\mathrm{sir}} = b - c$ and that we can show from the expression (65) that $\alpha \leq b - c$ if $b \geq c$. The depression on the basic reproduction number is a consequence of the time it takes for the exposed to become infective.

6 SIS Model

In the two models that we have analyzed above, the infective as well as the frequency of new cases vanishes in the long term. The disease becomes extinct within the population. In the susceptible-infective-susceptible (SIS) model, the disease does not disappear, becoming endemic. For long times, the infective does not disappear and the frequency of new cases is nonzero. The SIS model has only two classes, the susceptible and the infective, and two processes, as shown in Fig. 4a. The first is the infection process represented by

$$S \xrightarrow{\mathrm{I}} I, \tag{71}$$

occurring with an infection rate constant b, and the recovering process

$$I \longrightarrow S, \tag{72}$$

occurring with a recovery rate constant c.

We denote by x and y the fraction of susceptible and infective, respectively. The rate of the infection process is

$$w_{\mathrm{inf}} = bxy, \tag{73}$$

and the rate of the recovery process is

$$w_{\mathrm{rec}} = cy. \tag{74}$$

According to the rules, the evolution equations for the averages of these quantities are

$$\frac{d\bar{x}}{dt} = -b\bar{x}\bar{y} + c\bar{y}, \tag{75}$$

$$\frac{d\bar{y}}{dt} = b\bar{x}\bar{y} - c\bar{y}. \tag{76}$$

These equations are not all independent because $\bar{x} + \bar{y} = 1$. It is convenient to replace $\bar{x}$ in the second equation to
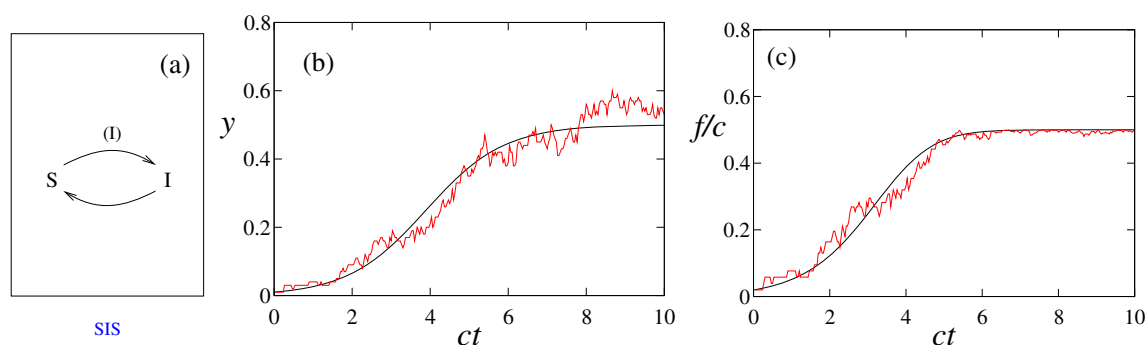
Fig. 4 SIS model. **a** The processes composing the model. **b** Fraction of infective individuals at a given time versus time t, obtained from numerical simulation of the master equation and its average. The fraction of the infective approaches a nonzero asymptotic value.

c Epidemic curve, or frequency of new cases as a function of time, from simulation, and its average. For long times it approaches a nonzero value. All curves were obtained for $b/c = 2$. The simulations were performed using $\varepsilon = 0.01$

obtain just one equation in $\bar{y}$,

$$\frac{d\bar{y}}{dt} = \alpha \bar{y} - b\bar{y}^2, \qquad (77)$$

where $\alpha = b - c$.

The solution for $\bar{y}$ can be given in closed form,

$$\bar{y} = \frac{\alpha y_0}{by_0 + (\alpha - by_0)e^{-\alpha t}}. \qquad (78)$$

In Fig. 4b, we show $\bar{y}$ as a function of t together with y obtained from simulations of the master equation using $\varepsilon = 0.01$. We see that the fraction of infective does not decrease, remaining finite at large times. We also show in Fig. 4c the frequency of new cases $f = bxy = b(1 - y)y$ obtained from simulations as well as its average $\bar{f} = b(1 - \bar{y})\bar{y}$ where $\bar{y}$ is given by the solution (78). The frequency of new cases does not decrease for long times and remains finite.

The linear exponential increase of $\bar{y}$ can be perceived from the closed solution. Alternatively, we may obtain this behavior by the linearization of (77) around the disease free solution $\bar{y} = 0$,

$$\frac{d\bar{y}}{dt} = \alpha \bar{y}, \qquad (79)$$

from which follows the solution

$$\bar{y} = y_0 e^{\alpha t}. \qquad (80)$$

Thus, if $\alpha > 0$, that is, if $b > c$, the epidemic spreads; otherwise, it does not. If one increases b from small values, it will reach a critical value $b_c = c$ which determined the onset of spread. The basic reproduction number is obtained from (31) and considering that $\bar{f} = b\bar{x}\bar{y}$, and that $\alpha = b - c$, we find

$$R_0 = \frac{b}{c}. \qquad (81)$$

In the limit $t \to \infty$, $\bar{y}$ does not vanish but reaches the value

$$y^* = \frac{b - c}{b}. \qquad (82)$$

This value is obtained either by taking the limit $t \to \infty$ in (78) or by setting to zero the right-hand side of (77), and is identified as the order parameter s. Therefore, s is given by

$$s = \frac{b - c}{b}, \qquad (83)$$

and is shown in Fig. 5a as a function of the infection rate b.

Applying the formula (24) for the present case, we find the following expression for the reduced variance χ of the fraction of the infective at the stationary state

$$\chi = \frac{c}{b}, \qquad b > c, \qquad (84)$$

$\chi = 0$ for $b < c$, and $\chi = 1/2$ when $b = c$. A plot of χ versus b is shown in Fig. 5b.

7 SIRS Model

In the model we consider now, the infective and the frequency of new cases do not vanish in the long term and in this sense it is similar to the SIS model. The susceptible-infective-recovered-susceptible (SIRS) model has three classes of individuals like the SIR mode, susceptible, infective, and recovered, and one more process than the SIR model. The processes are are shown in Fig. 6a and are as follows. The infection of a susceptible individual,

$$S \xrightarrow{I} I, \qquad (85)$$

occurring with a rate constant b, the spontaneous recovery,

$$I \longrightarrow R, \qquad (86)$$

occurring with a rate constant c, and the spontaneous loss of immunity,

$$R \longrightarrow S, \qquad (87)$$

Fig. 5 SIS model. **a** Order parameter s, which is the final value of the fraction of the infective, as a function of b/c. **b** Variance χ related to the infective a a function of b/c
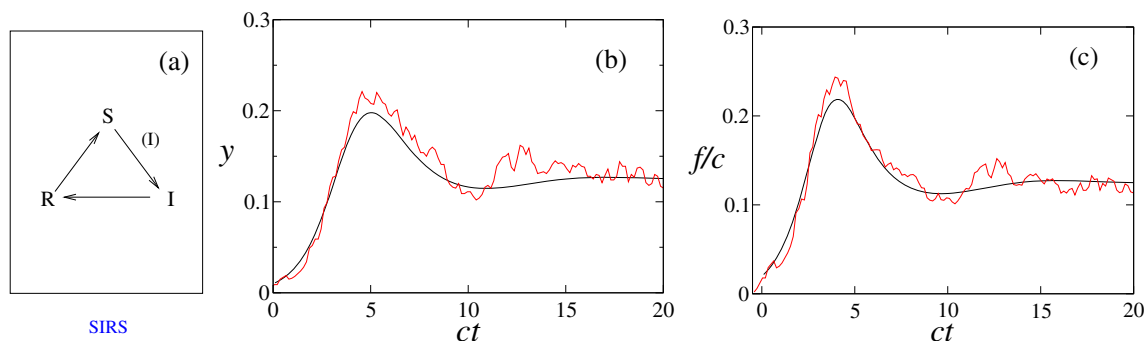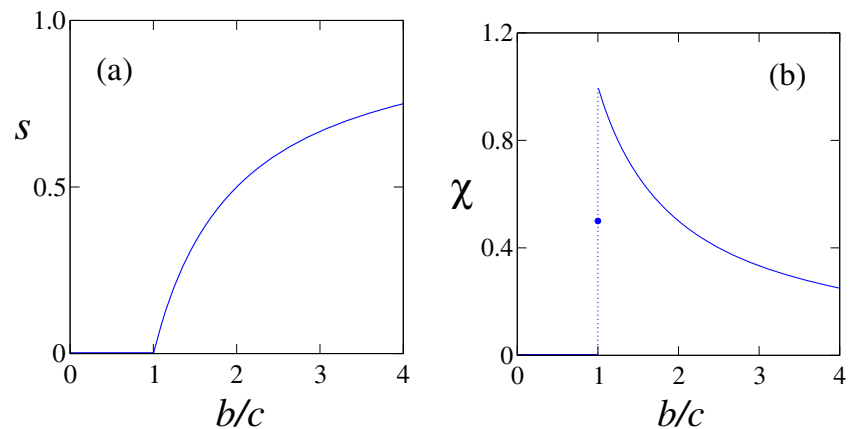

Fig. 6 SIRS model. **a** The processes composing the model. **b** Fraction of infective individuals at a given time versus time t, obtained from numerical simulation of the master equation and its average. The fraction of the infective approaches a nonzero asymptotic value.

c Epidemic curve, or frequency of new cases as a function of time, from simulation, and its average. For long times, it approaches a nonzero value. All curves were obtained for $b/c = 2$ and $a/c = 1/3$. The simulations were performed using $\varepsilon = 0.001$

occurring with a rate constant a. The recovered individual has only partial immunity in contrast to the SIR model where the recovered individual has permanent immunity.

The fractions of susceptible, infective, and recovered are denoted by x, y, and z, respectively. The rate of the infection process is

$$w_{\text{inf}} = bxy, \tag{88}$$

and the rate of the recovery process is

$$w_{\text{rec}} = cy, \tag{89}$$

and the rate of the loss of immunity is

$$w_{\text{los}} = az. \tag{90}$$

According to the rules, the evolution equations for the averages of these quantities are

$$\frac{d\bar{x}}{dt} = -b\bar{x}\bar{y} + a\bar{z}, \tag{91}$$

$$\frac{d\bar{y}}{dt} = b\bar{x}\bar{y} - c\bar{y}, \tag{92}$$

$$\frac{d\bar{z}}{dt} = c\bar{y} - a\bar{z}, \tag{93}$$

and they are not all independent because $\bar{x} + \bar{y} + \bar{z} = 1$.

We have solved numerically this set of equations and obtained $\bar{x}$, $\bar{y}$, and $\bar{z}$. In Fig. 6b, we show $\bar{y}$ as a function of time together with y obtained from the simulation with $\varepsilon = 0.001$. The infective increases exponentially, and then after reaching a maximum, it shows a damping oscillation towards a nonzero value. On Fig. 6c, we show the epidemic curve, which follows the same behavior with time as y. The frequency of new cases is $f = bxy$ and was obtained from numerical simulation. Its average $\bar{f} = b\bar{x}\bar{y}$ was also obtained from the numerical solutions of $\bar{x}$ and $\bar{y}$.

The linearization of (92) and (93) around the disease free solution, $\bar{y} = 0$, $\bar{x} = 1$, and $\bar{z} = 0$, gives

$$\frac{d\bar{y}}{dt} = \alpha\bar{y}, \tag{94}$$

$$\frac{d\bar{z}}{dt} = c\bar{y} - a\bar{z}. \tag{95}$$

The solution for $\bar{y}$ is

$$\bar{y} = y_0 e^{\alpha t}, \tag{96}$$

where $\alpha = b - c$. The spread occurs when $\alpha > 0$, that is, when $b > c$. Increasing the infection rate constant b from small values, the threshold of the spread happens when b reaches $b_c = c$, independent of a. The basic reproduction

number is obtained from (31) and considering that $\bar{f} = b\bar{x}\bar{y}$, and that $\alpha = b - c$, we find

$$R_0 = \frac{b}{c}. \tag{97}$$

The asymptotic values x^* and y^* of $\bar{x}$ and $\bar{y}$ are obtained by setting to zero the right-hand side of (91) and (92), and recalling that the $\bar{z} = 1 - \bar{x} - \bar{y}$. The result is

$$x^* = \frac{a}{b}, \qquad y^* = \frac{a(b-a)}{b(a+c)}. \tag{98}$$

A stability analysis of this solution can also be performed. It is possible to show that the eigenvalue related to the stability matrix has, for some values of the parameter, an imaginary part, which together with a negative real part indicates a damped oscillations. This is the behavior shown in Fig. 6 not only for the fraction of the infective but also for the epidemic curve.

The order parameter s for the present model is identified as the fraction y^*, as in the case of the SIS model, and is given by

$$s = \frac{a(b-c)}{c(a+c)}. \tag{99}$$

8 Conclusion

We have analyzed four models of epidemic spreading using a stochastic approach in which the primary stochastic variables are the numbers of individuals in each class. The individuals are classified in accordance with its condition with respect to the infectious disease. The process of changing from one class to the other is understood as being analogous to a chemical reaction. This analogy allowed to use the laws of mass action to set up the rate of several processes taking place in an epidemic spreading.

We have determined the onset of the epidemic spreading by a linear analysis of the disease free state. From this analysis, we have determined the critical infectious rate, above which the diseases spread. By solving the evolution equations, we determined the time behavior of the fraction of the infected and the frequency of new cases. These two quantities were also determined by numerical simulations of the master equation.

A relevant feature of the present approach is that the evolution equation for the average in the number of individuals is similar to the evolution equation employed in certain deterministic approaches. For instance, (36), (37), and (38) for the averages of the fractions of individuals are identical to those introduced by Kermack and McKendrick. The similarity or in some cases the equality of the equations

allows to take the point of view according to which the stochastic approach and the deterministic are not in opposition. Quite the contrary, they can be understood as being consistent views of the same problem.

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