PAPER

Effect of immunization through vaccination on the SIS epidemic spreading model

IOP Publishing Journal of Physics A: Mathematical and Theoretical
J. Phys. A: Math. Theor. **55** (2022) 275602 (12pp) https://doi.org/10.1088/1751-8121/ac7116

Effect of immunization through vaccination on the SIS epidemic spreading model

Tânia Tomé* **and Mário J de Oliveira***

Instituto de Física, Universidade de Sãoo Paulo, Rua do Matão, 1371, 05508-090 São Paulo, São Paulo, Brazil

E-mail: ttome@if.usp.br and oliveira@if.usp.br

Received 17 September 2021, revised 23 March 2022
Accepted for publication 18 May 2022
Published 14 June 2022

Abstract

We analyze the susceptible–infected–susceptible model for epidemic spreading in which a fraction of the individuals become immune by vaccination. This process is understood as a dilution by vaccination, which decreases the fraction of the susceptible individuals. For a nonzero fraction of vaccinated individuals, the model predicts a new state in which the disease spreads but eventually becomes extinct. The new state emerges when the fraction of vaccinated individuals is greater than a critical value. The model predicts that this critical value increases as one increases the infection rate reaching an asymptotic value, which is strictly less than the unity. Above this asymptotic value, the extinction occurs no matter how large the infection rate is.

Keywords: SIS model, diluted contact process, stochastic lattice epidemic models, vaccination models

1. Introduction

In the second edition of his book on the prevention of malaria [1], Ross developed an analytical theory of dynamics of infectious diseases through the use of differential equations of the first order in time [1, 2]. He called the approach the theory of happenings, understood as events that transform the condition of an individual such as a disease or a vaccination. The time evolution of the number z of infected individuals in a population p that remains constant, he represented by the equation

$$\frac{\mathrm{d}z}{\mathrm{d}t} = cz(p - z) - rz, \tag{1}$$

and called c the infection rate and r a factor that is related to the reversion or recovery of the individuals. He also solved this equation and found a sigmoid curve for z, that is, a monotonic

*Authors to whom any correspondence should be addressed.

increasing function of time with a finite asymptotic value, and a bell shaped curve of new cases, which is related to z by $cz(p-z)$.

A simple epidemic model involving only the infection process was later developed by Bailey [3, 4]. In this model all susceptible individuals eventually become infected. The evolution equation for the number y of susceptible individuals was written as

$$\frac{\mathrm{d}y}{\mathrm{d}t} = -\beta y(n+1-y), \tag{2}$$

where β is the infection rate and n is the number of susceptible individuals at $t=0$. He also developed a stochastic version of this model where the number of susceptible and infected individuals are treated as stochastic variables. This simple model was extended by Weiss and Dishon through the introduction of a recovery process [5]. Symbolically, the two processes of their model were represented by

$$S + I \xrightarrow{\alpha} 2I, \qquad\qquad I \xrightarrow{\beta} S, \tag{3}$$

where α is the infection rate and β the recovery rate. The evolution equation for the number x of infected individuals was written as

$$\frac{\mathrm{d}x}{\mathrm{d}t} = \alpha x(N-x) - \beta x, \tag{4}$$

which is identical to the Ross equation (1). They also developed a stochastic approach to this model where the numbers of susceptible and infected individuals are treated as stochastic variables.

The model represented by the reaction equation (3) is known as the susceptible–infected–susceptible (SIS) model and the equations (4) or (1) describe the deterministic version of this model [6–9]. The model represented by the first reaction equation alone is known as the susceptible–infected (SI) model and the equation (2) describes the deterministic version of the model. The stochastic versions are understood generically as a random walk on a space where the axes are the number of the various classes of individuals [10, 11].

The models for epidemic spreading and in general models that describe population dynamics can also be defined as having a spatial structure and evolving through a stochastic dynamics. When the spatial structure is represented by a lattice, these models are referred to as stochastic lattice models [12–26]. In these models each site of a lattice is in one of several possible states. The change of the state of a given site follows a stochastic dynamics in accordance with the processes represented by the reaction equations. An interesting feature of the stochastic lattice models is that the deterministic version as well as the stochastic version referred to above may be obtained from the stochastic lattice models through an approximation where the correlations are entirely or partially neglected [16, 20, 22, 23]. This is the procedure we will follow here to obtain the deterministic equations of the SIS epidemic model with vaccination.

A stochastic lattice model which is equivalent to the SIS model was in fact advanced by Harris, who called it a *contact process* [12]. Each site of the lattice is occupied by a particle or is empty. A particle is created on an empty site, with a certain creation rate, if a nearest neighbor is occupied. Particles disappear spontaneously with a certain annihilation rate. By identifying a particle with an infected individual and an empty site with a susceptible individual, the contact process becomes equivalent to the stochastic lattice SIS model.

Here we are concerned with the analytical description of the process of vaccination on the SIS model and the effects it produces on the spreading of the infectious disease. Vaccination is a powerful tool for the mass prevention of infection by inducing in the body an immune reaction which in turn generates immunity to infections [27]. The immunity may be only partial but here we assume that the immunity is permanent. To properly describe this process we add a third class of individuals to represent the vaccinated individuals. In addition to the class of susceptible (S) and infected (I), we consider also the class of vaccinated (V) individuals, which have acquired permanent immunization through vaccination. We consider also that the process of vaccination starts at the beginning of the spreading of the disease and that in the long run a fraction k of the individuals are vaccinated.

The SIS model presents two behaviors according to the strength of the infection rate. For small infection rate there is no spreading. When the infection rate is greater than a certain value, the spread of the epidemic sets in, and the disease becomes endemic, or persistent, which means that in the long run there is a nonzero fraction of infected individuals. In accordance with the model of vaccination analyzed here, if the fraction of vaccinated individuals is large enough the epidemic eventually disappears, or that the disease becomes extinct, meaning that the fraction of infected vanishes in the long run. The effect of vaccination is to turn the sigmoid curve of infected individuals into a bell shaped curve. Our analysis shows that the critical value of the fraction of vaccinated individuals above which the disease disappears increases with the infection rate, reaching an asymptotic value which we found to be strictly less that the unity. In other words, if the fraction of vaccinated is larger than this asymptotic critical value, the extinction of the disease occurs no matter how large the infection rate is.

The process of vaccination is usually approached by a spontaneous process represented by the reaction equation $\mathrm{S} \to \mathrm{V}$ which occurs with a certain vaccination rate [28–36]. If we apply this approach to a deterministic model, the whole population will eventually become vaccinated. We wish here to describe a process of vaccination in which only a fraction k of the individuals will be vaccinated. We have previously reported a mean-field model containing this idea of a fraction of vaccinated individuals [37]. Here, however, we wish to apply this idea to a lattice model. The implementation is original and not trivial, as we shall see. We remark in addition that the mean-field approximation that we use here to analyze the lattice model gives equations that are distinct from that of the previous model [37]. The way in which we introduce the vaccination process is explained in the following.

2. Vaccination process

The epidemic model with vaccination that we analyze is defined on a lattice, each site of which is occupied by an individual that can be in various conditions with respect to the infectious disease. We first analyze the case in which the individuals are sedentary, remaining forever in the same sites of the lattice. Here it suffices to consider two classes of individuals. The class of individuals that have been vaccinated (V) and the class of individuals that have not been vaccinated (N). The vaccination process that we wish to set up is such that in the long run a certain fraction k of individuals are vaccinated. A natural way of setting up this process is to choose randomly a certain set $\mathcal{A}$ of k sites of the lattice whose individuals will be vaccinated. Initially, all sites are in state N.

The sites of $\mathcal{A}$ are subject to the process $\mathrm{N} \to \mathrm{V}$ which we assume to occur with a rate a. The sites of the complementary set of sites will never be in state V, remaining forever in state N. As the sites of $\mathcal{A}$ are randomly chosen *a priori* and do not change in time, the set $\mathcal{A}$ is

understood as comprising a *quenched disordered* set. In accordance with the reaction N → V, the probability $P_{\rm V}$ that a site of $\mathcal{A}$ is in state V evolves in time according to

$$\frac{\rm d}{{\rm d}t}P_{\rm V} = aP_{\rm N}, \tag{5}$$

where $P_{\rm N}$ is the probability that the site is in state N. As $P_{\rm N} = 1 - P_{\rm V}$, this equation becomes an equation in $P_{\rm V}$ whose solution is

$$P_{\rm V} = 1 - e^{-at}, \tag{6}$$

where we are considering that at $t = 0$ the site is in state N. From this result it follows that the fraction v of all sites of the lattice that are in the state V varies in time according to

$$v = k(1 - e^{-at}), \tag{7}$$

where k is the number of sites of $\mathcal{A}$ and is the fraction of vaccinated individuals in the long run.

We now suppose that the individuals are not sedentary but move on the lattice without keeping their positions. As a consequence, the sites belonging to $\mathcal{A}$ change with time. If we focus on a specific site which in a certain instant of time is in state V, we see that now this site may change to the state N, a impossibility in the case of a quenched disorder set. Therefore, if we consider a *specific site* of the lattice, which is the case when we set up the stochastic process on a lattice, we have to consider also the process V → N in addition to the process N → V. Denoting by γ and β the transition rates of the former and latter processes, the time evolution of $P_{\rm V}$ is

$$\frac{\rm d}{{\rm d}t}P_{\rm V} = -\gamma P_{\rm V} + \beta P_{\rm N}. \tag{8}$$

Again, as $P_{\rm N} = 1 - P_{\rm V}$, this equation can be solved with the solution

$$P_{\rm V} = \frac{\beta}{\beta + \gamma}[1 - e^{-(\alpha+\beta)t}]. \tag{9}$$

We next assume that $P_{\rm V}$ is the same for all sites and as a consequence it is equal to the fraction v of vaccinated sites. We also assume that v has the same form of the previous quenched approach given by (7). Comparing (9) with (7), we see that the rates α and β are related to the vaccination rate a and to the fraction k of vaccinated individuals by $\beta = ak$ and $\gamma = a(1 - k)$. Using these relations, equation (8) is written as

$$\frac{\rm d}{{\rm d}t}P_{\rm V} = a(k - P_{\rm V}), \tag{10}$$

and (9) as

$$P_{\rm V} = k(1 - e^{-at}). \tag{11}$$

The first approach to vaccination, which is associated to the quenched disorder, is appropriate to describe a situation in which the individuals are fixed in space, an example being the spread of an epidemic occurring in an orchard where the trees are attached to the ground. In the epidemic spreading occurring in a community of moving individuals, the appropriate approach is that associated with the process of *dilution by vaccination* described by the equation (10).

Here we consider just the second approach, applying it to the SIS model. It is worth mentioning that the stationary properties of the SIS model with the first approach to vaccination are identified with contact process under quenched dilution [38–41].

3. Model

We present now the SIS model under vaccination appropriate to describe an epidemic spreading on a community of moving individuals. The sites of a lattice are occupied by three types of individuals: susceptible (S), infected (I), or vaccinated (V). A susceptible becomes infected through an auto-catalytic process S $\to$ I with rate b. An infected become susceptible through a spontaneous reaction I $\to$ S with rate c. These two processes comprise the ordinary SIS model. In accordance with the dilution approach to vaccination explained above, the processes involving the vaccinated individuals are as follows. An S or an I become V with rate ak and V becomes S with rate $a(1-k)$. These processes are represented by the reaction equations S $\to$ V and I $\to$ V, each one occurring with rate $\beta = ak$, and V $\to$ S occurring with rate $\gamma = a(1-k)$, where a is the rate of vaccination and k is the fraction of individuals to be vaccinated.

These considerations allow us to set up a stochastic process on a lattice, which is described by a master equation that governs the time evolution of the probability of the global state of the system [42]. From the master equation we may derive the evolution equations for the various marginal probabilities [42]. The evolution equations for the probabilities $P_{\rm S}$, $P_{\rm I}$, and $P_{\rm V}$ of a site being occupied by a susceptible, an infected, and a vaccinated individuals, respectively, are

$$\frac{\mathrm{d}}{\mathrm{d}t}P_{\rm S} = -bP_{\rm SI} + cP_{\rm I} - \beta P_{\rm S} + \gamma P_{\rm V}, \tag{12}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}P_{\rm I} = bP_{\rm SI} - cP_{\rm I} - \beta P_{\rm I}, \tag{13}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}P_{\rm V} = -\gamma P_{\rm V} + \beta(P_{\rm S} + P_{\rm I}), \tag{14}$$

where $P_{\rm SI}$ is the probability of a site being in state S and one of its neighbors in state I. These equations are not all independent because $P_{\rm S} + P_{\rm I} + P_{\rm V} = 1$.

Replacing $P_{\rm S} + P_{\rm I} = 1 - P_{\rm V}$ in equation (14), it becomes

$$\frac{\mathrm{d}}{\mathrm{dt}}P_{\rm V} = a(k - P_{\rm V}), \tag{15}$$

which is in accordance with equation (10). The solution of this equation with the initial condition with no vaccinate individuals is

$$P_{\rm V} = k(1 - e^{-at}), \tag{16}$$

which is in accordance with (11). Equations (15) and (16) are a consequence of the independence of each site with respect to the dynamics of the vaccinated individuals, a relevant property of the present model. In general, the probability that in a set of n sites, m sites are in state V and the remainder are not in state V is the product $P_{\rm V}^m(1-P_{\rm V})^{n-m}$.

Similarly we may write the evolution equations for the pair correlations P_{SI}, which is given by

$$\frac{\mathrm{d}P_{\mathrm{SI}}}{\mathrm{d}t} = -cP_{\mathrm{SI}} - b(1-r)P_{\mathrm{SI}} - brP_{\mathrm{ISI}} + cP_{\mathrm{II}} + brP_{\mathrm{SSI}}$$

$$-2\beta P_{\mathrm{SI}} + \gamma P_{\mathrm{IV}}, \tag{17}$$

where $r = (\kappa - 1)/\kappa$ and κ is the coordination number of the lattice.

The right-hand side of equation (17) is obtained by considering the several transitions that contribute to the decrease or increase of P_{SI}. For instance, the transition $\mathrm{I} \to \mathrm{S}$ occurring with rate c transforms the pair II into the pair SI, giving a positive contribution $+cP_{\mathrm{SI}}$. The same transition transforms the pair SI into the pair SS, giving a negative contribution $-cP_{\mathrm{SI}}$. The contributions from other transitions are similarly obtained. A care must be taken in the case of the catalytic transition $\mathrm{S} \to \mathrm{I}$, in which case we must consider a cluster of three neighboring sites instead of two. In a square lattice there are two two types of these clusters. In one of them the tree sites are collinear and in the other they form a right angle. However, in view of the approximative approach that we will use here, we have considered them to be equal to one another.

The set of evolutions equations are to be solved for the initial condition such that all sites are in the S state except a small number of them which is in the I state. The set of equations are not closed equations and to solve them we resort in two approximations.

4. First approximation

We consider here an approximation in which the probability P_{SI} is replaced by the product $P_{\mathrm{S}}P_{\mathrm{I}}$, which is understood as a full break of the correlations. Using the notation $x = P_{\mathrm{S}}$, $y = P_{\mathrm{I}}$, and $v = P_{\mathrm{V}}$, the equation (13) becomes

$$\frac{\mathrm{d}y}{\mathrm{d}t} = bxy - (c+\beta)y, \tag{18}$$

where $x = 1 - y - v$ and v is considered to be a function of time given by

$$v = k(1 - e^{-at}), \tag{19}$$

where $a = \beta + \gamma$. This equation is to be solved for an initial condition such that y is small at $t = 0$.

One solution of this equation corresponds to the absence of infected individuals, that is, $y = 0$. To determined the stability of this solution, we linearize (18) around this solution to get

$$\frac{\mathrm{d}y}{\mathrm{d}t} = b(1-v)y - (c+\beta)y. \tag{20}$$

At the initial times the fraction of vaccinated individuals is zero, $v = 0$, and equation (20) becomes

$$\frac{\mathrm{d}y}{\mathrm{d}t} = (b - c - \beta)y. \tag{21}$$

The solution of this equation tell us that y decreases if $b < b_1$, where

$$b_1 = c + ak. \tag{22}$$

Therefore, if $b < b_1$ there is no spreading of the epidemic, the epidemic is absent, a state we denote by A.

In the opposite case, $b > b_1$, y grows initially, and in the long run it reaches an asymptotic value. We envisage two behaviors for the time evolution of y. In one of them, y stops increasing and asymptotically reaches a nonzero value, indicating that the disease becomes endemic, or persistent, a state we call P. In the other, y stops increasing, then decreases and reaches a zero value which means that the disease becomes extinct, a state we call E.

The nonzero asymptotic value of y is given by the stationary solution of equation (18). For large times, we replace v by its asymptotic value $v = k$ and the equation (18) becomes

$$\frac{\mathrm{d}y}{\mathrm{d}t} = b(1-k-y)y - (c+\beta)y. \tag{23}$$

In the stationary state

$$y = (1-k) - \frac{c+\beta}{b}. \tag{24}$$

This solution occurs as long $y > 0$, that is, as long as $b \geqslant b_2$, where

$$b_2 = \frac{c+ak}{1-k}. \tag{25}$$

Alternatively, we may consider the stability of the solution $y = 0$ in the regime of large times in which case we replace v in the linearized equation (20) by k, and equation (20) becomes

$$\frac{\mathrm{d}y}{\mathrm{d}t} = b(1-k)y - (c+\beta)y. \tag{26}$$

From this equation we conclude that the solution $y = 0$ is stable if $b < b_2$.

In figure 1(a) we show the phase diagram in the variables b and k which displays the various states A, E and P according to the present approximation. The line $b = b_1$ determines the AE transition line and $b = b_2$ determines the EP transition line. According to this approximation, the effect of immunization through vaccination is to cause the disappearance of the disease, or its extinction, when the fraction k of vaccinated individuals is large enough.

5. Second approximation

Now we wish to consider a second approximation in which the correlations are partially broken [22]. The approximation we use now is to break the three-site correlations by using the replacements: $P_{\mathrm{ISI}} = P_{\mathrm{SI}}^2/P_{\mathrm{S}}$ and $P_{\mathrm{SSI}} = P_{\mathrm{SS}}P_{\mathrm{SI}}/P_{\mathrm{S}}$. The equation (17) becomes

$$\frac{\mathrm{d}P_{\mathrm{SI}}}{\mathrm{d}t} = -cP_{\mathrm{SI}} - b(1-r)P_{\mathrm{SI}} - br\frac{P_{\mathrm{SI}}^2}{P_{\mathrm{S}}} + cP_{\mathrm{II}} + br\frac{P_{\mathrm{SI}}P_{\mathrm{SS}}}{P_{\mathrm{S}}} - 2\beta P_{\mathrm{SI}} + \gamma P_{\mathrm{IV}}, \tag{27}$$

We use the notations $P_{\mathrm{S}} = x$, $P_{\mathrm{I}} = y$, $P_{\mathrm{V}} = v$, $P_{\mathrm{SI}} = u$, the approximations $P_{\mathrm{IV}} = yv$ and $P_{\mathrm{SV}} = xv$, and the identities $P_{\mathrm{II}} + u + yv = y$ and $P_{\mathrm{SS}} + xv + u = x$ to write the equation

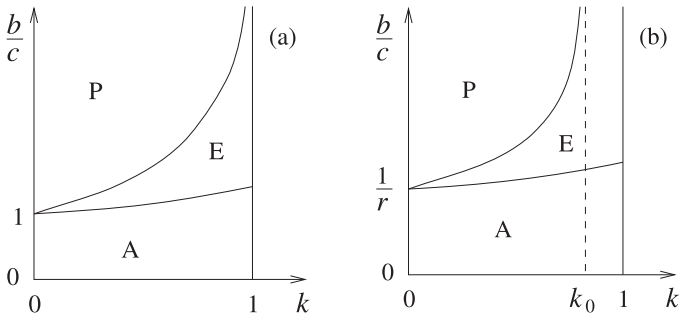

Figure 1. Diagram showing the behavior of the SIS model with vaccination, according to the first approximation (a) and to the second approximation (b) where b is the infection rate, c is the recovery rate, and k is the fraction of individuals that have acquired permanent immunization by vaccination, where k_0 is strictly less than 1. In the region A there is no spreading of the disease, whereas in P and E the disease spreads. In P it becomes persistent, with a nonzero fraction of infected, and in E it becomes extinct, with a zero fraction of infected individuals. The figures were drawn by considering that vaccination rate a is fixed.

above in the form

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -cu - b(1-r)u - br\frac{u^2}{x} + c(y-u-yv) + br\frac{u}{x}(x-u-xv) - 2\beta u + \gamma yv. \tag{28}$$

Equation (28) together with equation (13), which we write in the form

$$\frac{\mathrm{d}y}{\mathrm{d}t} = -cy + bu - \beta y, \tag{29}$$

constitute a close set of equations for y and u, and we recall that v is a function of time given by (19), and that $x = 1 - y - v$.

The trivial solution of these equations is $y = 0$, $u = 0$, which corresponds to the absence of spreading. To determine its stability, we linearize equation (28) to find

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -cu - b(1-r)u + c(y-u-yv) + bru(1-v) - 2\beta u + \gamma yv. \tag{30}$$

At the initial times $v = 0$ and this equations become

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -cu - b(1-2r)u + c(y-u) - 2\beta u. \tag{31}$$

The stability analysis is carried out by writing the equations (29) and (31) in the matrix form

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix} y \\ u \end{pmatrix} = \begin{pmatrix} -c-\beta & b \\ c & -2c-b+2br-2\beta \end{pmatrix}\begin{pmatrix} y \\ u \end{pmatrix}. \tag{32}$$

After determining the eigenvalues of the square matrix we find that the largest eigenvalue becomes zero when $b = b_1$ where

$$b_1 = \frac{2(c+ak)^2}{2rc + (2r-1)ka}. \tag{33}$$

If the solution $y = 0$ and $u = 0$, which characterize the state without spreading of the disease, is stable then the eigenvalues of the square matrix are nonnegative which occurs when $b < b_1$. Thus, the epidemic will not spread if $b < b_1$.

For long times, v approaches k and the linearized equation (30) becomes

$$\frac{\mathrm{d}u}{\mathrm{d}t} = -cu - b(1-r)u + c(y-u-yk) + bru(1-k) - 2\beta u + \gamma yk. \tag{34}$$

The stability analysis is carried out as before. Now the matrix equation equivalent to (29) and (34) are

$$\frac{\mathrm{d}}{\mathrm{d}t}\begin{pmatrix} y \\ u \end{pmatrix} = \begin{pmatrix} -c-\beta & b \\ c - ck + \gamma k & -2c - b + 2br - bkr - 2\beta \end{pmatrix}\begin{pmatrix} y \\ u \end{pmatrix}. \tag{35}$$

The largest eigenvalue of the square matrix vanishes when $b = b_2$ where

$$b_2 = \frac{2(c+ak)}{2r-k-rk}. \tag{36}$$

The solution corresponding to extinction state is thus stable when $b < b_2$. When $b > b_2$, there emerges a state where the disease becomes endemic.

The phase diagram predicted by this second approximation is qualitatively similar to that of the first approximation as can be seen in figure 1(b). However, there is an important quantitative difference. If the fraction k of vaccinated individuals is larger that k_0, which is strictly less that the unity, the disease disappears, or becomes extinct no matter how large the rate of infection is. From the line $b = b_2$ we find

$$k_0 = \frac{2r}{1+r}. \tag{37}$$

For a square lattice $r = 3/4$ and $k_0 = 6/7 = 0.857$.

6. Numerical simulations

We have simulated the SIS model with vaccination on a square lattice with periodic boundary conditions with $L \times L$ sites, with L ranging from 10 to 80 and 10^6 Monte Carlo steps. The transition rules that we used are as follows. At each time step, a site of the lattice is chosen at random. With probability ε, the process of vaccination is carried out, otherwise, with the complementary probability $1-\varepsilon$, the SIS processes are preformed. In the first case, if the chosen site is in state S or I, it becomes V with probability k, and if it is in state V, it becomes S with probability $1-k$. In the second case, if the chosen site is in state I then it becomes S with probability $1-p$. If it is in state S, then one of its nearest neighbors is chosen at random, and if the chosen neighboring site is in state I, then the chosen site becomes I with probability p.

We have initially determined the stationary values of the fraction y of infected individuals. The vanishing of y determines the transition line between the states E and P as shown in the phase diagram of figure 2(a) in the variables p and k for $\varepsilon = 0.01$. We recall that p is related to the rate of infection and k is the fraction of vaccinated individuals in the long run. To find the transition line between the A and E states, we have calculated y as a function of time with an initial condition with all sites in state S except one which is in the state I. The transition point occurs when y begins to increase with time as one increases the value of p at a fixed value of k.

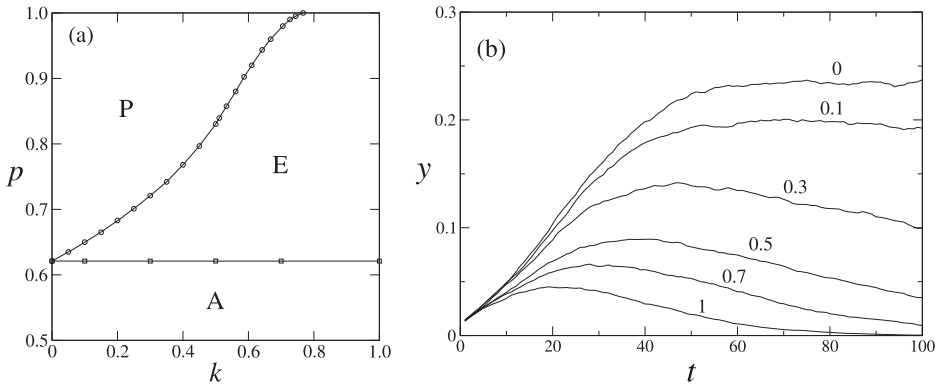

Figure 2. (a) Phase diagrams obtained by numerical simulations in the plane p versus k. The states are persistent (P), extinct (E) and no spreading (A). (b) Fraction y of infected individuals as a function of time t obtained by numerical simulations for $p = 0.7$ and several values of the fraction k of vaccinated individuals. For $k = 0.3$, 0.5, 0.7 and 1, y vanishes in the long run.

The line $k = 0$ corresponds to the ordinary SIS model which has a transition from the disease free state A to the persistent state P occurring at $p = 0.622\,46$ [42]. From this point, two transition lines emerge, as shown in figure 2(a). The first line separates the state A and the extinction state E, where the disease becomes extinct. In the state E, the fraction of infected individuals increases, reaches a maximum and then decreases and vanishes in the long run as can be seen in figure 2(b). A second line separates the E and P states. This line ends at a point along the line $p = 1$ at the value of k equal to $k_0 = 0.760(2)$ for $\varepsilon = 0.01$. From these results we draws the following conclusions. For a given value of p, and thus of the infection rate, there is a critical value of the fraction k of vaccinated individuals above which the disease becomes extinct. The critical value increases with the infection rate and reaches the value k_0 at the maximum possible value of the infection rate which corresponds to $p = 1$. If the value of k is larger than k_0 the disease becomes extinct no matter how large the infection rate is, a result that we have found also within the second approximation.

Usually in mean field approximation we expect that the regions of the phase diagram corresponding to the ordered state is larger when compared to simulations. That is what is happening here. For instance, the critical point at $k = 0$ for numerical simulation is $p = 0.622\,46$, which is larger than $p = 4/7 = 0.571\,43$, the value corresponding to the second approximation, which in turn is larger $p = 0.5$, corresponding to the first approximation.

7. Conclusion

We have analyzed an extension of the SIS model by the inclusion of the process of vaccination. To this end a third class of individuals, the ones that have acquired permanent immunization by vaccination, were added to the classes of susceptible and infected. The process of vaccination is understood as a dilution of the system by transforming the susceptible into vaccinated, and thus decreasing the fractions of the susceptible and infected. For a nonzero fraction of vaccinated individuals, the model predicts a new state in which the disease spreads but eventually becomes extinct, in addition to the two states of the ordinary SIS model, the absence of epidemic spreading and the endemic state.

As one increases the fraction k of vaccinated individuals, there is a critical value of k above which the disease becomes extinct. The critical value increase with the strength of the infection rate. However, as the infection rate increases without bounds, the critical fraction approaches a value k_0 which is strictly less than the unity. This result says that no matter how large the infection rate is, the disease disappears if the fraction of individuals is larger than k_0. From the numerical simulations of the model on a square lattice, we have found $k_0 = 0.760(2)$ for $\varepsilon = 0.01$.

A relevant feature of the process of immunization by vaccination that we used here is that the dynamics of the vaccination is independent of the dynamics of the other processes. In addition the dynamics related to a specific site is independent of other sites. Let us denote by N a site that is either in state S or I and ask for the probability that each site of the lattice is in a certain given state, either V or N. This probability is equal to the product $P_{\rm V}^n P_{\rm N}^m$, where $P_{\rm N} = 1 - P_{\rm V}$, n is the number of sites in state V, and m is the number of sites in state N.

Acknowledgments

We wish to take this opportunity to congratulate Robert Ziff on his seventieth birthday.

Data availability statement

No new data were created or analysed in this study.

ORCID iDs

Tânia Tomé https://orcid.org/0000-0002-5030-4518

References

[1] Ross R 1911 *The Prevention of Malaria* 2nd edn (London: Murray)
[2] Ross R 1915 *Br. Med. J.* **1** 546
[3] Bailey N T J 1950 *Biometrika* **37** 193
[4] Bailey N T J 1957 *The Mathematical Theory of Epidemics* (New York: Hafner)
[5] Weiss G H and Dishon M 1971 *Math. Biosci.* **11** 261
[6] Kryscio R J and Lefèvre C 1989 *J. Appl. Probab.* **26** 685
[7] Mollison D (ed) 1995 *Epidemic Models* (Cambridge: Cambridge University Press)
[8] Keeling M J and Rohani P 2008 *Modeling Infectious Diseases* (Princeton, NJ: Princeton University Press)
[9] Tomé T and J Oliveira M 2020 *Rev. Bras. Ensino Fís.* **42** e20200259
[10] Nisbet R M and Gurney W C S 1982 *Modelling Fluctuating Populations* (Caldwell: Blackburn)
[11] Tomé T and de Oliveira M J 2020 *Braz. J. Phys.* **50** 832
[12] Harris T E 1974 *Ann. Probab.* **2** 969
[13] Grassberger P 1983 *Math. Biosci.* **63** 157
[14] Grassberger P and de La Torre A 1983 *Ann. Phys.* **122** 373
[15] Ohtsuki T and Keyes T 1986 *Phys. Rev.* A **33** 1223
[16] Satulovsky J E and Tomé T 1994 *Phys. Rev.* E **49** 5073
[17] Durrett R 1995 *Epidemic Models* ed D Mollison (Cambridge: Cambridge University Press) p 187
[18] Antal T, Droz M, Lipowski A and Ódor G 2001 *Phys. Rev.* E **64** 036118
[19] Dahmen S M and Hinrichsen H 2003 *Phys. Rev.* E **68** 016114
[20] Tomé T and de Oliveira M J 2009 *Phys. Rev.* E **79** 061128
[21] de Souza D R and Tomé T 2010 *Physica* A **389** 1142

[22] Tomé T and de Oliveira M J 2011 *J. Phys. A: Math. Theor.* **44** 095005
[23] de Souza D R, Tomé T, Pinho S T R, Barreto F R and de Oliveira M J 2013 *Phys. Rev.* E **87** 012709
[24] Wada A H O, Tomé T and de Oliveira M J 2015 *J. Stat. Mech.* P04014
[25] Ruziska F M, Tomé T and de Oliveira M J 2017 *Physica* A **467** 21
[26] Silva A T C, Assis V R V, Pinho S T R, Tomé T and de Oliveira M J 2017 *Physica* A **468** 131
[27] Keeling M, Tildesley M, House T and Danon L 2013 *Math. Today* 40
[28] Kribs-Zaleta C M and Velasco-Hernández J X 2000 *Math. Biosci.* **164** 183
[29] Alexander M E, Moghadas S M, Rohani P and Summers A R 2006 *J. Math. Biol.* **52** 290
[30] Shaw L B and Schwartz I B 2010 *Phys. Rev.* E **81** 046120
[31] Wang Z, Bauch C T, Bhattacharyya S, d'Onofrio A, Manfredi P, Perc M, Perra N, Salathé M and Zhao D 2016 *Phys. Rep.* **664** 1
[32] Pires M A and Crokidakis N 2017 *Physica* A **467** 167
[33] Khanjanianpak M, Azimi-Tafreshi N and Castellano C 2020 *Phys. Rev.* E **101** 062306
[34] Kuga K, Tanaka M and Tanimoto J 2021 *Proc. R. Soc.* A **477** 20200769
[35] Chen S, Small M and Fu X 2020 *IEEE Trans. Netw. Sci. Eng.* **7** 1583
[36] Lv W, Ke Q and Li K 2020 *Nonlinear Dyn.* **99** 1507
[37] Tomé T, Silva A T C and de Oliveira M J 2021 *Braz. J. Phys.* **51** 1853
[38] Moreira A G and Dickman R 1996 *Phys. Rev.* E **54** R3090
[39] Vojta T and Dickison M 2005 *Phys. Rev.* E **72** 036126
[40] de Oliveira M M and Ferreira S C 2008 *J. Stat. Mech.* P11001
[41] Wada A H O and de Oliveira M J 2017 *J. Stat. Mech.* 043209
[42] Tomé T and de Oliveira M J 2015 *Stochastic Dynamics and Irreversibility* (Berlin: Springer)