Susceptible–infected–recovered model with recurrent infection

Flávia M. Ruziska, Tânia Tomé *, Mário J. de Oliveira
Instituto de Física, Universidade de São Paulo, Rua do Matão, 1371, 05508-090 São Paulo, SP, Brazil

ARTICLE INFO
Article history:
Received 2 August 2016
Received in revised form 4 September 2016
Available online 5 October 2016

Keywords:
Epidemic models
Nonequilibrium phase transitions
Dynamic percolation
SIR model

ABSTRACT
We analyze a stochastic lattice model describing the spreading of a disease among a community composed by susceptible, infected and removed individuals. A susceptible individual becomes infected catalytically. An infected individual may, spontaneously, either become recovered, that is, acquire a permanent immunization, or become again susceptible. The critical properties including the phase diagram is obtained by means of mean-field theories as well as numerical simulations. The model is found to belong to the universality class of dynamic percolation except when the recovering rate vanishes in which case the model belongs to the directed percolation universality class.

© 2016 Elsevier B.V. All rights reserved.

1. Introduction

The spreading of diseases and, more generally, the dynamics of populations are described by models that can be characterized as being deterministic or stochastic, space structured or non-space structured [1–9]. An important model that has been studied by each one of these approaches is the so-called susceptible–infected–recovered (SIR) model [5–22]. The SIR model describes the spreading of a disease in a community in which individuals acquire permanent immunization. A susceptible (S) individual becomes infected (I) by the contact with an infected individual, a process interpreted as a catalytic reaction, $S + I \rightarrow I + I$. An infected individual may eventually recover from the disease becoming recovered (R) forever, acquiring thus a permanent immunization, a process described by the spontaneous reaction $I \rightarrow R$.

Starting from a single infected individual in a community of susceptible individuals, a disease described by the SIR model will spread if the rate of infection is high enough. The infected individuals will eventually become recovered and the whole community will be composed by the recovered individuals and the remaining susceptible individuals. If the infection rate is small the disease will not spread. The passage from non spreading to spreading regime is regarded as a phase transition from a phase with a vanishing density of recovered, in the thermodynamic limit, to a phase with a nonzero density of recovered. The critical behavior of this transition places the SIR model into the universality class of the dynamical percolation (DyP)
We remark that the stationary critical behavior of models belonging to the DyP universality class is the same as that of the isotropic percolation [20–23]. The SIR model can be extended by the addition of one more process in several ways [8], by including, for instance, processes describing vaccination [8], or the influence of resource constraints [24,25]. If we include the spontaneous reaction \( R \rightarrow S \), which means that the individuals do not acquire a permanent immunization, it becomes the susceptible–infected–recovered–susceptible (SIRS) model [18]. If we include the spontaneous reaction \( I \rightarrow S \), it becomes a model in which the infected individuals may spontaneously recover from the disease becoming again susceptible, that is, a model in which the infection is recurrent [15], which is the object of our analysis, here.

In the model we analyze, illustrated in Fig. 1, a susceptible becomes infected by a catalytic reaction, \( S + I \rightarrow I + I \), with infection rate \( b \). Some infected individuals become recovered spontaneously, \( I \rightarrow R \), with a recovering rate \( c \), acquiring a permanent immunization; and some become again susceptible spontaneously, \( I \rightarrow S \), with a recurrence rate \( a \). When \( c = 0 \), there is no recovered individuals and the model reduces to the susceptible–infected–recovered–susceptible model (SIRS), which is equivalent to the contact process [9,16,26]. The critical behavior of the contact process belongs to the universality class of directed percolation (DP) [16,26] and we may say that when \( c = 0 \), the critical behavior of the present model belongs to the DP universality class. When \( c \neq 0 \) and \( a \neq 0 \), the model is expected to be in the same universality class of the SIR model, that is the DyP universality class. Our results indeed confirm this expectation.

We consider the model defined on a regular lattice where each site of the lattice is occupied by just one individual that can be susceptible, infected or recovered. We analyze the model by the use of mean-field theories, at the level of one-site and two-site approximations. We also performed numerical simulations on a square lattice and construct the phase diagram.

2. Model

The model studied here is a stochastic lattice model defined as follows. Each site of a regular lattice can be occupied by a susceptible (\( S \)) individual, or by an infected (\( I \)) individual or by a recovered (\( R \)) individual. A susceptible individual becomes infected catalytically with rate proportional to \( b \). More precisely, if a site \( i \) of the lattice is in state \( S \), then it becomes \( I \) with rate \( bn_i/k \) where \( n_i \) is the number of infected individuals in the neighborhood of site \( i \), defined as the nearest neighbor sites of site \( i \), and \( k \) is the number of sites of the neighborhood. An infected individual becomes spontaneously either recovered with rate \( c \) or susceptible with rate \( a \).

When \( a = 0 \) and \( c \neq 0 \), the model becomes the susceptible–infected–recovered (SIR) model, for which an infected individual eventually recovers from the disease acquiring a permanent immunization. The recovered individual does not transmit the disease, remaining forever in this condition. When \( c \neq 0 \) and \( a \neq 0 \), an infected individual may become again susceptible, but, eventually, an infected individual may recover from the disease becoming immune forever. In particular, when \( a \gg c \), the processes \( S = I \) will occur several times and eventually \( I \) becomes \( R \).

Once the transition rates are defined, we may set up the master equation that gives the time evolution of the probability of \( P(\eta) \) of a given configuration \( \eta \), defined as the collection of variables \( \eta_i \) that give the state of each site of the lattice. The variable \( \eta_i \) takes the values 0, 1 or 2, according to whether site \( i \) is occupied by a susceptible, an infected or a recovered individual, respectively. From the master equation it is possible to write down the time evolution of several marginal probability distributions, \( P(\eta_j) \), \( P(\eta_i, \eta_j) \), and so on. The time evolution equations for \( P_S \), \( P_I \) and \( P_R \), which are the densities of susceptible, infected and recovered individuals, are given by

\[
\frac{d}{dt} P_S = a P_I - b P_{SI},
\]

\[
\frac{d}{dt} P_I = -(a + c) P_I + b P_{SI},
\]

\[
\frac{d}{dt} P_R = c P_I,
\]

where \( P_{SI} \) denotes the probability that a pair of neighboring sites: one occupied by a susceptible and the other by an infected. These equations can be understood by considering each one of the three reactions that define the model. The spontaneous reaction \( I \rightarrow S \) gives a contribution \( a P_I \) to the increase in \( P_S \) and the same contribution to the decrease in \( P_I \). Similarly, the spontaneous reaction \( I \rightarrow R \) gives a contribution \( c P_I \) to the increase in \( P_R \) and the same contribution to the decrease in \( P_I \). To
Fig. 2. (a) Densities of susceptible $x$, infected $y$ and recovered $z$ individuals versus time $t$, and (b) epidemic curve, $\Gamma = -\frac{dx}{dt}$ versus $t$, obtained from simple mean-field approach for $a = 0.2$, $b = 0.4$ and $c = 0.1$. The starting condition is $x = 1 - h$, $y = h$ and $h = 0.001$.

We find the contribution coming from the catalytic reaction $S \rightarrow I$ to the increase in $P_I$ we argue as follows. Each neighbor of a site of type $I$ gives a contribution equal to $(b/k)P_{IS}$. Since the number of neighbors is $k$, the total contribution of this reaction is $bP_{IS}$. A similar reasoning will lead us to the same contribution to the decrease in $P_S$.

The present model has been defined as having three parameters: $a$, $b$, and $c$. By rescaling the time it is possible to reduce the number of parameters to two. We thus introduce the parameters $p$ and $q$, understood as the effective immunization and recovery rates, defined by

$$p = \frac{a}{b}, \quad q = \frac{c}{b},$$

and when convenient we use these two parameters instead of $a$, $b$ and $c$.

3. Simple mean-field approximation

Eqs. (1)–(3) do not form a closed set of equations for $P_S$, $P_I$ and $P_R$ due to the presence of the two site probability $P_{SI}$ on the right-hand side of these equations. However, we may close the equations by a truncation scheme which consists in the simple mean-field approximation

$$P(\eta_1, \eta_2) = P(\eta_1)P(\eta_2).$$

Using the definitions $P_S = x$, $P_I = y$, and $P_R = z$ we may write $P_{SI} = P_S P_I = xy$, and the time evolution equations become

$$\frac{dx}{dt} = ay - bxy,$$  

$$\frac{dy}{dt} = -(a + c)y + bxy,$$  

$$\frac{dz}{dt} = cy.$$  

Notice that $x + y + z = 1$ implying that only two equations are independent.

We start our analysis by numerically integrating the set of Eqs. (5)–(7). A typical solution for $x$, $y$ and $z$ as functions of time, is shown in Fig. 2(a), for a given set of values of $a$, $b$ and $c$, corresponding to the spreading of disease regime. The density of infected individuals has a bell shape and vanishes for large enough times. The densities of susceptible and recovered individuals, on the other hand, approaches finite values when $t \to \infty$, as seen in Fig. 2(a). Another quantity that characterizes the dynamics of the spreading of the disease is the quantity defined by $\Gamma = -\frac{dx}{dt}$, which is understood as the difference between the fraction of susceptible sites becoming infected and the infected ones becoming susceptible. The plot of $\Gamma$ versus time, the epidemic curve, is shown in Fig. 2(b) and has also a bell shape, with $\Gamma$ vanishing when $t \to \infty$.

Now we turn to the stationary solutions of the set of Eqs. (5)–(7). One stationary solution corresponds to an absorbing state with no infected nor recovered individuals, that is, $x = 1$, $y = 0$ and $z = 0$, characterizing a non-spreading regime. A linear stability analysis of this state leads to the conclusion that this absorbing state is stable as long as $b < a + c$. When this condition is not met another stationary solution appears for which $x \neq 1$, $y = 0$ and $z \neq 0$, as we will see below. This state corresponds to a spreading regime. Thus we are faced with a phase transition that occurs when $b = a + c$, or $p + q = 1$, between an absorbing state, identified as the non-spreading regime, and an active state, identified as the spreading regime.

To solve the evolution equations for the densities, it is convenient to divide Eq. (6) by (5) to get

$$\frac{dy}{dx} = -\frac{q}{x - p} - 1.$$
Fig. 3. Density of recovered individuals $z$, at the stationary state, as a function of $q = c/b$ obtained from simple mean-field approximation, for $q/p = c/a = 2$ and for several values of $h$. From bottom to top the values of $h$ are: 0+ (see text), 0.001, 0.01, and 0.05.

Assuming that initially there is no recovered individual but only susceptible and infected individuals then at $t = 0$, $x = 1 - h$, $y = h$ and $z = 0$, where $h$ is the initial density of infected. Solving Eq. (8) with this initial condition we find

$$y = q \ln \frac{x - p}{1 - h - p} - x + 1. \tag{9}$$

an expression that may be understood as a constant of motion. To get the stationary solution it suffices to remember that in the limit $t \to \infty$, the infected individuals disappear, that is, $y \to 0$, as long as $c \neq 0$ ($q \neq 0$), so that the stationary density of susceptible individuals are given by the equation

$$x = q \ln \frac{x - p}{1 - h - p} + 1. \tag{10}$$

In the context of mean-field approach, the order parameter is defined as the density of recovered individuals $z$, which in the stationary state is related to the density of susceptible individuals $x$ by $z = 1 - x$. From Eq. (10) we find the following expression for the order parameter at the stationary state,

$$z = -q \ln \frac{1 - x - p}{1 - h - p}. \tag{11}$$

valid for $q \neq 0$. In Fig. 3, we plot $z$ versus $q$ for several values of $h$. It is worth mentioning that the phase transition occurs only when $h \to 0$, a limit that should be understood as follows. First we obtain $z$ for a nonzero value of $h$ and after that we take the limit $h \to 0$, a solution indicated in Fig. 3 by $h = 0^+$. This procedure amounts to say that at the beginning there is an infinitesimal small seed of infected individuals.

The continuous phase transition happens when $p + q = 1$, or $a + c = b$. To see this, we write $h$ as a function of $z$,

$$h = 1 - p - (1 - z - p) e^{2/q}, \tag{12}$$

and then expand the expression on the right-hand site of Eq. (12) in powers of $z$ Up to second order in $z$, we get

$$h = \frac{1}{q} (p + q - 1) z + \frac{1}{2q} z^2. \tag{13}$$

If $h > 0$, this equation has one positive and one negative solution. The positive is given by

$$z = (1 - p - q) + \sqrt{(1 - p - q)^2 + 2hq}. \tag{14}$$

If now we take the limit $h \to 0$, we find $z \to 0$ if $p + q \geq 1$ but $z = 2(1 - p - q) > 0$ if $p + q < 1$, as shown in Fig. 3. Writing $z \sim (1 - p - q)^\beta$, we see that $\beta = 1$. At the critical point, we find from (13) that $h = z^2/2q$. Writing, $z \sim h^{1/\delta}$, we see that $\delta = 2$.

4. Pair mean-field approximation

A better approach can be obtained by the use of the pair mean-field approximation [21]. In this approach, unlike to what happens in the simple mean-field approximation, the possible correlation between neighboring sites are properly taken
into account. We start by writing down the evolution equations of the two-site correlations $P(\eta_i, \eta_j)$ where $i$ and $j$ are two nearest neighbor sites of the regular lattice. From the master equation, we find the following equations

\begin{align}
\frac{d}{dt} P_{SI} &= aP_{II} - aP_{SI} - cP_{SI} - b(1-f)P_{SI} + bfP_{SSI} - bfP_{SR}, \\
\frac{d}{dt} P_{SR} &= aP_{IR} + cP_{SI} - bfP_{ISR}, \\
\frac{d}{dt} P_{IR} &= -aP_{IR} + cP_{II} - cP_{IR} + bfP_{ISR},
\end{align}

where $f = (k-1)/k$ and $k$ is the lattice coordination number.

To understand how the terms on the right-hand side of these equations are obtained, let us take a look at the contributions of each one of the three reactions that define the model. We will consider only the contributions to the equation for $P_{SI}$, the probability that site 0 of the lattice is in state S and site 1 is in state I. A similar reasoning can be used to get the contributions related to the other equations. The spontaneous reaction $I \rightarrow S$ gives a contribution equal to $aP_{II}$ to the increase in $P_{SI}$, and a contribution equal to $bfP_{SI}$ to the decrease in $P_{SI}$. The spontaneous reaction $I \rightarrow R$ gives a contribution equal to $cP_{CI}$ to the decrease in $P_{SI}$.

To find the contribution to the increase in $P_{SI}$ coming from the catalytic reaction $S \rightarrow I$ we argue as follows. Let site 0 be in state S, and site 1 in state S. Each neighbor of site 0 which is in state S gives a contribution equal to $(b/k)P_{SSI}$. Since the number of neighbors is $k-1$, the total contribution is $bfP_{SSI}$. The contribution to the decrease in $P_{SI}$ is obtained by setting site 0 in state S and site 1 in state I. Each neighbor of site 0 which is in state I, except site 1, gives a contribution equal to $(b/k)P_{ISR}$. But the number of neighbors is $k-1$, and we get a contribution equal to $bfP_{ISR}$ to the decrease in $P_{SI}$. The neighboring site 1, being in state I, gives a contribution equal to $(b/k)P_{SI} = (1-f)P_{SI}$ to the decrease in $P_{SI}$.

In the pair mean-field approximation we use the approximation $P(\eta_i, \eta_j, \eta_k) = P(\eta_i, \eta_j)P(\eta_j, \eta_k)/P(\eta_j)$ where $i$ and $k$ are distinct nearest neighbor sites of site $j$. Using the notation $x = P_S$ and $y = P_I$ as before and $u = P_{SR}$, $v = P_{SI}$ and $w = P_{IR}$, we write

$P_{SSI} = v^2/x$, $P_{SSI} = (x-v-u)v/x$, $P_{ISR} = vu/x$, where we have take into account that $P_{SS} = P_S - P_{SI} - P_{SR} = x - v - u$. Eqs. (1) and (2) are written as

\begin{align}
\frac{dx}{dt} &= ay - bv, \\
\frac{dy}{dt} &= -(a + c)y + bv, \\
\frac{dv}{dt} &= a(y - 2v - w) - cv - b(1-f)v + bf(x - 2v - u)
\end{align}

whereas Eqs. (15)–(17) are written as

\begin{align}
\frac{dv}{dt} &= a(y - 2v - w) - cv - b(1-f)v + bf(x - 2v - u)
\end{align}

where we have taken into account that $P_{SI} = P_I - P_{SS} - P_{IR} = y - v - w$. We are left with five equations in five unknowns.

To find the phase transition line we analyze the stability of the trivial stationary solution $x = 1, y = 0, u = 0, v = 0$ and $w = 0$. Up to linear terms, the set of evolution equations is written as

\begin{align}
\frac{dx}{dt} &= ay - bv, \\
\frac{dy}{dt} &= -(a + c)y + bv, \\
\frac{dv}{dt} &= ay + (2bf - 2a - b - c)v - aw, \\
\frac{du}{dt} &= cv + aw, \\
\frac{dw}{dt} &= cy - cv - (a + 2c)w.
\end{align}

From the eigenvalues of the Jacobian matrix, we see that this solution becomes unstable when

\[(k - 1)p + (k - 2)q = k(p + q)^2.\]
Fig. 4. (a) Phase diagram in the variable \( q = c/b \) versus \( p = a/b \), for simple mean-field (smf), pair mean-field (pmf) with \( k = 4 \) and simulation (sim) on a square lattice. The absorbing state, or non-spreading regime, is located above the transition line and the active state, or spreading regime, below. (b) Critical line obtained by numerical simulations in the variables \( \ln q \) versus \( \ln (p_0 - p) \) where \( q = c/b \) and \( p = a/b \), and \( p_0 = 0.606532 \) is the critical value of \( p \) at \( c = 0 \).

Therefore, Eq. (28) gives the transition line from the spreading to non-spreading regime of the present model in the pair mean-field approximation, as shown in Fig. 4. When \( q = 0 \), the critical point is given by \( p = (k - 1)/k \) which gives the critical point of the contact model in the pair approximation. When \( p = 0 \), the critical point is given by \( q = (k - 2)/k \), which gives the critical point of the SIR model in the pair approximation. As usual, the pair approximation gives a phase diagram with the absorbing phase occupying a larger region of the phase diagram when compared with the simple mean field approximation.

5. Numerical simulations

We have performed numerical simulations of the model on a square lattice with periodic boundary conditions. The initial condition is a lattice full of susceptible individuals except for one site occupied by an infected individual placed at the center of the lattice. The algorithm we used was defined as follows. At each time step an infected site is chosen from a list of infected sites. (1) With probability \( c' \), the chosen site I becomes R; (2) with probability \( a' \), the chosen site I becomes S; (3) with the complementary probability \( b' = 1 - a' - c' \), a neighboring site of the chosen site is chosen at random. If the neighboring site is S then it becomes I; otherwise nothing happens. After performing this procedure the time is increased by a value that is equal to the inverse of the number of sites in the list of infected sites and the list is updated.

The relation between \( a', b', c' \) and the rates \( a, b, c \) are as follows

\[
a' = \frac{a}{a + b + c}, \quad b' = \frac{b}{a + b + c}, \quad c' = \frac{c}{a + b + c},
\]

and the relation with the parameters \( p \) and \( q \) are \( p = a'/b', \ q = c'/b' \).

Several quantities were measured, such as \( n_S, n_I \) and \( n_R \), the numbers of susceptible, infected and recovered sites, respectively. The density \( z = \langle n_R \rangle/L^2 \) of recovered individuals at the stationary state versus \( q \) is shown in Fig. 5(a) for several values of \( L \). According to finite size scaling theory developed in Ref. [22], the density \( z \) at the stationary state is expected to behave around the critical point as

\[
z = L^{-2\beta/\nu_\perp} \Phi_0(\epsilon L^{1/\nu_\perp}),
\]

where \( \Phi_0 \) is a universal function and \( \epsilon \) is the deviation from the critical point, and \( \beta \) and \( \nu_\perp \) are the critical exponents related to the order parameter and the spatial correlation length, respectively. Fig. 5(b) shows \( z \) versus \( L \) for several values of the parameter \( q \) along \( q = 2p \). The slope of the log–log plot at the critical point equals \( -2\beta/\nu_\perp \). As we will see below, the critical point along \( q = 2p \) occurs at \( q_c = 0.17288(3) \). From the data points of Fig. 5(b) we find \( 2\beta/\nu_\perp = 0.209(3) \) which should be compared with the exact value for isotropic percolation in two dimensions, \( 2\beta/\nu_\perp = 5/24 = 0.20833 \).

Another useful quantity, related to the second moment of \( n_R \) is the ratio

\[
U = \frac{\langle n_R^2 \rangle}{\langle n_R \rangle^2}.
\]

According to finite size scaling theory developed in Ref. [22], the quantity \( U \) behaves as

\[
U = L^{\beta/\nu_\perp} \Phi_1(\epsilon L^{1/\nu_\perp}),
\]
Fig. 5. (a) Density $z$ of recovered individuals, at the stationary state, as a function of $q$, along the line $q = 2p$ of the phase diagram, for several values of $L$ indicated. (b) $z$ versus $L$ for several values of $q$ indicated.

Fig. 6. (a) $U$ versus $L$ and (b) $P$ versus $L$ for several values of $q$ indicated along $q = 2p$.

where $\Phi_1$ is a universal function. Fig. 6(a) shows $U$ versus $L$ for several values of $q$ along $q = 2p$. The slope at the critical value $q_c = 0.17288(3)$ gives $\beta/\nu_\perp = 0.105(1)$ which should be compared with the exact value for isotropic percolation in two dimensions, $\beta/\nu_\perp = 5/48 = 0.10416$.

We have also determined the quantity $P$, interpreted as the order parameter, and defined as follows [22]. Let $B$ be the number of runs such that the border of the final cluster of recovered sites reaches the boundaries of the square lattice. The quantity $P$ is defined as the ratio $B/R$ where $R$ is total number of runs. In the case of periodic boundary conditions, used here, the boundaries are interpreted as the two vertical and horizontal lines at a distance $L/2$ from the center of the lattice, where an infected site was placed at the beginning. Around the critical point $P$ behaves as

$$P = L^{-\beta/\nu_\perp} \Phi_2(\epsilon L^{1/\nu_\perp}),$$

(33)

where $\Phi_2$ is a universal function. Fig. 6(b) shows $P$ versus $L$ for several values of $q$ along $q = 2p$. The slope at the critical value $q_c = 0.17288(3)$ gives $\beta/\nu_\perp = 0.105(1)$ which should be compared with the exact value for isotropic percolation in two dimensions, $\beta/\nu_\perp = 5/48 = 0.10416$.

Taking into account the critical behavior of $U$ and $P$, given by Eqs. (32) and (33), it follows that the behavior of $UP$ around the critical point is [22]

$$UP = \Phi(\epsilon L^{1/\nu_\perp}),$$

(34)

where $\Phi = \Phi_1 \Phi_2$ is a universal function. We may conclude that the curves of $UP$ as a function of the parameter $p$ and $q$, for different values of $L$, will cross at the critical point, if $L$ is large enough [22]. In other words, $\Phi(0) = \Phi_c$ does not depend on $L$ as can be seen in Fig. 7. From this figure we obtain that the critical value is $q_c = 0.17288(3)$ and $UP = 1.0160(5)$, in agreement with that of the SIR model and other isotropic percolation models $UP = 1.0167(1)$ [22].

The crossing of $UP$ procedure was used to determine the critical line shown in Fig. 4. The values of $q$ and $p$ at the critical line are shown in Table 1 together with the corresponding value of $UP$, which is a universal quantity at the critical point. As can be seen in this table the critical values of $UP$ are, within the statistical errors, in agreement with that of the SIR model.
The critical behavior of the present model shows that it belongs to the DyP universality class, except when \( q = 0 \). In this case the model reduces to the contact process which belongs to a different universality class, namely the DP universality class. We have also performed numerical simulations along \( q = 0 \) and determined the stationary properties. The critical properties are found to be identical to the contact model \([9,16]\). In particular the critical point \( p_0 \) is found to be \( p_0 = 0.605532(1) \). We remark that our numerical results indicate that the critical line, shown in Fig. 4 goes continuously into this point when \( q \to 0 \). Assuming that is indeed the case, it is worthwhile to find the behavior of the critical line around the point corresponding to the contact critical point \((p, q) = (p_0, 0)\). We assume the following behavior

\[
q \sim (p_0 - p)\phi,
\]

where \( \phi \) is the crossover exponent given. A fitting on the data points of Fig. 4 using an expression of the type \( \ln q = \phi \ln(p_0 - p) - k_1 - k_2(p_0 - p)^{k_3} \) gives \( \phi = 1.7(1) \), implying that the axis-\( p \) is tangent to the critical line.

### 6. Conclusion

We have analyzed a stochastic lattice model for the spreading of disease which is a modification of the SIR model by the introduction of recurrent infection. In addition to the two processes of the SIR model, \( S + I \to I + I \) and \( I \to R \), the model displays a third process \( I \to S \). The phase diagram was obtained by means of simple and pair mean field approximations as well as numerical simulations on a square lattice. When the recurrence rate is nonzero the infected individual may become again a susceptible individual, but, eventually, an infected individual may recover from the disease becoming immune forever. In epidemiology terms, this model for \( a \gg c \) can be seen as a model which can be understood as a simplified version for the spreading of a disease in which the process \( S \to I \) occurs many times before the occurrence of the process \( I \to R \), in which case the individual acquires a permanent immunization. The recurrent infection is relevant to those diseases for which the full immunization is achieved after the infection has been acquired more than once.

It is found that the critical properties place the model in the DyP universality class except when the rate \( c \) of reaction \( I \to R \) vanishes, in which case the model reduces to the SIS, equivalent to the contact model. In this case, the model belongs to the DP universality class. If we interpret the model studied here as the contact process with the addition of the process \( I \to R \), then we may conclude that the parameter \( c \) is a relevant parameter. That is, for \( c \neq 0 \), the model leaves the DP universality class.

The DyP universal critical behavior of the model studied here confirms the idea that some modifications of a model does not change the critical behavior. Here, the modification is the introduction of recurrent infection in the SIR model. In the present case we can use moreover the argument of Grassberger \([12]\) according to which the SIR model and slight modifications of the SIR model have stationary behavior similar to isotropic percolation, including the critical behavior.
References