The $R_0$ Approach to Epidemic-non-Epidemic Phases Revisited

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Abstract

In this work, we revisit the basic reproduction rate $R_0$ definition for analysis of epidemic-non-
epidemic phases describing the dynamics of the discrete stochastic version of the epidemic SIR
model based on the Master Equation formalism. One shows that it is a very precise and efficient
way to determine the epidemic threshold; using its most primitive concept, we can find exact
results.
Introduction - The basic reproduction rate, $R_0$, is the most fundamental parameter used by epidemiologists [1]. It has raised interest of physicists because of analogies between epidemic and percolation systems [2, 3, 4, 5], and its generality, that permits, for example, analyze spreading of viruses in a structured scale-free network [6]. The definition of $R_0$ is “the average number of secondary cases caused by an infectious individual in a completely susceptible population” [7]. This simple idea had a profound effect on epidemic theory. It may be a global insight that cuts through the details of the transmission process, because it originated from consideration of deterministic models of homogeneous population with random mixing [8]. However, putting more details in the model one can extend its definition to heterogeneous mixing. In this way, naturally the efforts were in analytical calculations of $R_0$ for continuous deterministic or stochastic models [2, 9, 10]. The stochastic framework, although more realistic in principle, it is more complex to analyze because of detail required [11, 12, 13]. Commonly, simulations helped to confirm theoretical assumptions. Improved machine technology has spread the use of computationally intensive methods to solve a great diversity of epidemics, and so simulation technics, as Dynamical Monte Carlo (DMC) [14, 15, 16, 17], are becoming more popular in this subject. Taken on the advantage of the DMC method, we can calculate $R_0$ straightforward. Thus, we adopted this approach to characterize the separation between the epidemic and non-epidemic phases, because its efficiency and simplicity that enables a facile finding of exact results.

Throughout this Letter, we shall consider the classical $SIR$ (Susceptible, Infected, Removed) epidemic model, originally based on the chemical “mass action” principle (see [18] and references therein), to illustrate the discrete stochastic approaches. Our model encloses the deterministic one as a particular case. Based on the Master equation formalism we generalize the $SIR$ model and $R_0$ for discrete stochastic systems. Also, we describe the local epidemic model with an exact result to $R_0$ using its primitive concept. Finally, we show a phase diagram of the local − $SIR$ model, with deterministic-like behavior, where no homogeneous mixing is considered, and dynamical Monte Carlo simulation results.

Generalized SIR model and $R_0$ - Stochastic process approaches could simulate non-equilibrium systems, even the deterministic ones, introducing random variables to describe them in a microscopic scale. The macroscopic behavior of some system is resulting from averages of its microscopic properties. One can describes the evolution of the distribution
of probabilities, for markovian processes, with the *Master Equation*:

\[
\frac{dP_i(t)}{dt} = \sum_j w_{j\rightarrow i} P_j - \sum_j w_{i\rightarrow j} P_i, \tag{1}
\]

where \(P_i\) is the probability to find the system at the state \(i\) at the time \(t\), and \(w_{i\rightarrow j}\) is the transition probability per unity of time. Considering \(T_{ij}\) the probability of transition from \(i\) to \(j\), we may write \(w_{i\rightarrow j} = \frac{T_{ij}}{\tau_i} \tag{19}\), where \(\tau_i\) is a characteristic time constant (*lifetime*) of the state \(i\).

We now start by choosing a convenient physical extensive microscopic quantity \(A_i\), which depends only of the system’s state \(i\). Since the time must change for every successful event, we will consider only counting events related quantities. To *SIR* epidemic systems the number of infected individuals, for example, is an adequate quantity because it represents the balance between the number of infection and removal events. The mean value for a given quantity at the time \(t\) is

\[
A(t) = \langle A \rangle = \sum_i P_i(t) A_i. \tag{2}
\]

This equation represents a continuous physical macroscopic quantity \(A(t)\). We can differentiate both sides of the equation above, with respect to \(t\). After that, using (1), and by defining \(\Delta A_{ij} = A_i - A_j\), we get

\[
\frac{dA(t)}{dt} = \sum_i \sum_j w_{j\rightarrow i} P_j \Delta A_{ij}. \tag{3}
\]

Consider now the nearest-neighbor states \(j\) of a given state \(i\); if we measure the “distance” between the states, say by the quantity \(|\Delta A_{ij}|\), such that the non-null minimum value is \(|\Delta A_{ij}| = a\), we may approach the equation (3) by:

\[
\frac{dA(t)}{dt} = \sum_{(ij)} w_{j\rightarrow i} P_j a \delta_{ij}, \tag{4}
\]

where the symbol \((ij)\) denotes a nearest-neighbor pair of states, and \(\delta_{ij} = \Delta A_{ij}/|\Delta A_{ij}|\). Now we consider another physical quantity \(A^\dagger\) that represents a source for the quantity \(A\). Thus, we can rewrite (4) as:

\[
\frac{dA(t)}{dt} = \sum_j r_j^+ P_j A_j^\dagger - \sum_j r_j^- P_j A_j, \tag{5}
\]

where \(r_j = < w_{j\rightarrow i} >_i\) are the averaged transition probabilities per unity of time over the ensemble of the nearest-neighbor states \(i\) of \(j\) at some time \(t\), i.e., the *mesoscopic* rates.
Here, the word ensemble means a set of configurations accessible at a finite (small) time around a time \( t \); in this sense we are using a time dependent ergodicity idea \[14\], and so generally the systems are non ergodic in nonequilibrium states. The superscripts “+” and “−” mean respectively the contributions to increasing and to decreasing the quantity \( A(t) \) \[20\].

Based on (5), we formulated the GSIR model through the following set of stochastic differential equations and inter-classes rates:

\[
\frac{dS}{dt} = \sum_j r_{RS}^j P_j R_j - \sum_j r_{SI}^j P_j S_j, \quad (6)
\]

\[
\frac{dI}{dt} = \sum_j r_{SI}^j P_j S_j - \sum_j r_{IR}^j P_j I_j, \quad (7)
\]

\[
\frac{dR}{dt} = \sum_j r_{IR}^j P_j I_j - \sum_j r_{RS}^j P_j R_j. \quad (8)
\]

The mesoscopic rates are \( r_{SI}^j \), \( r_{IR}^j \) and \( r_{RS}^j \), for each state \( j \), respectively, from \( S \to I \), \( I \to R \) and \( R \to S \). To satisfy the SIR condition the set \( \{r_{RS}^j\} \) is null. Note that we meant that, for example, if \( A = I \), then \( A^\dagger = S \) in the equation (5). The conservation law with the total number of individuals \( N = S(t) + I(t) + R(t) \) is satisfied. One may obtain the reproduction rate, \( \mathcal{R}_0 \), directly from the equations (6–8) with the epidemic condition \( \frac{dI}{dt} \geq 0 \); where the equality is the threshold and it is set to \( t_0 = 0 \), the initial time. Thus, we can do \( \sum_j r_{SI}^j P_j S_j - \sum_j r_{IR}^j P_j I_j \geq 0 \), what implies that \( \sum_j r_{SI}^j P_j S_j \geq \sum_j r_{IR}^j P_j I_j \). One can thus write the reproduction rate as

\[
\mathcal{R}_0 = \frac{\sum_j r_{SI}^j P_j S_j}{\sum_j r_{IR}^j P_j I_j}, \quad (9)
\]

for stochastic processes. As the condition to \( \mathcal{R}_0 \) to the epidemic threshold must be valid to the ensemble average of initial states \( j_0 \) that gives the same initial condition \( S_0, I_0 \) and \( R_0 \), we may define

\[
\mathcal{R}_0 = \frac{< r_{SI} >_0 S_0}{< r_{IR} >_0 I_0}; \quad (10)
\]

the average number of the secondary cases produced by \( I_0 \) infected initially. Note that if we do \( < r_{SI} >_0 = bI_0 \) and \( < r_{IR} >_0 = a \) we recover the deterministic case \[12\]. One must observe that if some initial configuration is fixed, one does not need the averages in (10), but only obtain the rates, \( r_{SI} \) and \( r_{IR} \); so, of course, generally, the \( \mathcal{R}_0 \) depends on the initial configuration choice. In many practical situations this is important because it will determine
an epidemic or not. We can easily adapt the result above to other models, as the SIS model,[6] for example, to analysis of the epidemic threshold.

**Local epidemic model** - Generically, the temporal and spatial evolution characterize any epidemics, where in each part of the system the density of the elements can vary with the time. One can analyze this process through a two-dimensional lattice, in which each site, representing an individual of the population, receives own attributes as susceptibility and interactivity referring each site with the others. We will analyze a model with local contact only. The elements are all fixed, i.e., no populational mobility is considered. The main reason to study a such particular model is that we have more fluctuations, and so it is a good test to the efficiency of our approach.

The probability of individuals contracts the illness, in transmitting a disease by contact, depends on the status (susceptible, infected or removed) in which they meet its neighbors; its chance of getting sick will depend on the number of sick neighbors. Thus, considering an element possesses \( n \) infective neighbors, and an infection chance, \( p_0 \), due to each neighbor, the probability of its change in a sick element (through \( n \) effective contacts) will be [18]:

\[
\omega_{SI} = \Lambda[1 - (1 - p_0)^n].
\]

Therefore, \((1 - p_0)^n\) is the probability of no infection of a susceptible (individual) if it has \( n \) infected neighbors, thus \( 1 - (1 - p_0)^n \) is the probability of infection of a susceptible if it has \( n \) infected neighbors. The \( \Lambda \) parameter gives the \( \omega_{SI} \) as inverse of time units. When \( n = 0 \), that is, when no neighbor is contaminated, the probability of contamination due to contact is zero, so \( \omega_{SI} \) increases when the number of effective contacts, \( n \), increases. A global removal rate determines the infectious period, and an infected individual turn immune (removed) stochastically. So the infectious period for each individual fluctuates over an average number given by the inverse of the removal rate, like in the mean field approach [21]. In this sense our definition of \( R_0 \) is more general than that gave in the reference [13]. Also has a difference that it is considered instantaneously instead during the infectious period, so it follows close the classical definition. However, we considered a range of initial values to the number of infected individuals, and did an analysis with an initial random distribution of immunes. Note that for the considered model in a square lattice, when \( I_0 = 1 \), we have the trivial exact result

\[
R_0 = \frac{n_{\text{max}} \Lambda p_0}{w_{IR}},
\]

(12)
where \( n_{\text{max}} \) is the maximum number of contacts. One can see that the exact result to a
more thorough model, including the homogeneous mixing (mean field), is straightforward. The reference [13] shows this result as an analytical approximation to \( R_0 \).

**Results and final remarks** - For practical purposes one distributes the individuals on a
square lattice of \( N = M \times M \) sites. All the individuals at the lattice boundary have their
statuses fixed at susceptible status. One considered only two lattice sizes, \( M = 10 \) and
200, because increasing \( M \) reaches smooth curves near to the \( M = 200 \) rapidly. We did
\( r_{IR} = q = 1 \), constant independent of the configuration, and \( r_{SI} = \langle w_{SI} \rangle_j \), averaged over
all individual probabilities that appear in any random configuration \( j \); with \( w_{SI} \) modeled
with a purely local interaction with \( \Lambda = 1 \), so \( w_{SI} = 1 - (1 - p_0)^n \). The initial condition
for the number of infectives, \( I_0 \), to the system, for \( M = 10 \), was varying from 1 to 99, and,
for \( M = 200 \), from 1 up to 38000 randomly distributed on the lattice. One occupies the
remaining sites by \( S_0 = N - I_0 \) susceptibles, so \( R_0 = 0 \) for both cases. We did the variable
\( n \) as an integer ranging from 0 to 8, since the first and second nearest infected neighbors
are indistinguishably considered for each susceptible.

Considering that the sum of the “microscopic influences” creates a rate \( r_{SI} \), we calculated
by a sample average the initial mean rate \( \langle r_{SI} \rangle_0 \). Of course in the practice of the simulation we drew randomly only a few configurations to estimate the averages and we
used the equation (10) to elaborate the phase diagrams for the SIR model showed in the
figure 1. We got the values for \( \langle r_{SI} \rangle_0 \) using 100 and \( 4 \times 10^4 \) configurations for \( M = 200 \)
and 10 respectively. Observe that the maximum \( R_0 \) happens when the probability \( p_0 \) reaches
its largest value in a great population of susceptible. We found for \( M = 10 \), the epidemic
threshold in the interval \( R_0 = 1.03 - 1.05 \). For \( M = 200 \), the interval reduced to a very thin
line defined at \( R_0 = 1 \), as expected to infinite systems. Note that the \( R_0 \) contour lines for
\( M = 10 \) are noisy because of the small size of the lattice. For cases when \( R_0 > 1 \), happen
epidemic bursts in average, and when \( R_0 < 1 \) does not. We did, also, experiments with a
non-zero initial immune individuals number, \( R_0 \), and its qualitative effect on \( R_0 \) is the same
as for \( R_0 = 0 \), since we have a random initial distribution of immunes. Quantitatively, for
the initial infectives number fixed, we need to increase the probability per unit of time, \( w_{SI} \),
to have epidemics; as expected, has a critical value to \( R_0 \) that no epidemic occurs, the so
known herd immunity effect [17]. For the considered parameters of our model, we are at the
epidemic threshold when
\[
< r_{SI} >^t_0 = \frac{I_0}{S_0}.
\] (13)

After several fittings we found the reasonable expression to the initial average rate: 
\[
< r_{SI} >^t_0 = \alpha[1 - (1 - p_0)\beta],
\]
with the real numbers \(\alpha \leq 1\) and \(0 \leq \beta \leq 8\). The critical value for \(R_0\) is so: 
\[
R_0 = N - I_0[1 + 1/ < r_{SI} >^t_0].
\]
Note that if the condition \(I_0 > \alpha S_0\) is satisfied we have no epidemics for any value of \(p_0\). At first sight it might be a strange result, however, if the initial state has most infected individuals, the removal chance of some is high; so it decreases faster than the number of infections itself.

The figure 2 show some epidemic and non epidemic cases obtained by Dynamical Monte Carlo. This direct method was used to find the threshold line of the phase diagrams showed in the figure 1. The very beginning of the process is sufficient to determine \(R_0\), but for completeness we showed the epidemic curves in an extended time.

In summary, we presented a stochastic version for the parameter \(R_0\), based on the description of the SIR model, by means of the Master equation formalism. This way, the predictive power \(R_0\) is transported from deterministic to the stochastic one, generalizing the concept. In fact, a very defined result to the threshold curve was earlier found to deterministic systems; it is interesting that we can have this, also, for stochastic systems. It is consequence of the definition of \(R_0\), whose difference with that one of already cited recent work \[13\] did decrease the threshold. Fluctuations to favor epidemics in our case when \(R_0 < 1\) are smaller than those favor non-epidemics, i.e., the number of non-epidemic cases prevails, even for finite small systems; the same happens with the opposite case. Complex geometries can be included in this model, since have no restrictions to the model in this sense, and the system geometry is an important factor to change the threshold. If the system is strongly dependent on the initial conditions, the averages are not appropriated to predict an epidemic. We believe that this definition of \(R_0\) open the doors for new investigations and calculations of \(R_0\) for more realistic systems because we used a general microscopic description to get a parameter of macroscopic nature.

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FIGURE CAPTIONS

Figure 1. Shows a phase diagram for the local - SIR model with parameters $q = 1.0$, $\frac{S_0}{N} = (0.05 - 0.95)$ and $p_0 = (0.05 - 0.95)$. The values of $R_0$ larger than 1 in the smooth contour lines ($M = 200$) allow that the number of infected increases with the time characterizing an epidemic outbreak, for $R_0$ smaller than 1 the infection fade-out. The threshold to the noisy contour map ($M = 10$) is in the interval 1.03 – 1.05 that is too small to show in the diagram.

Figure 2. Epidemic curves. Show the number of Infectives evolving with the time. The numerical values for the model parameters are $q = 1$, $M = 200$ and $I(0) = 5000$. A total of 20 experiments was done to get the averages. The figure shows two cases, some curves those represent epidemic outbreaks ($R_0 > 1$) and others in that the infection fade-outs ($R_0 < 1$).
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Figure 2

\[ I(t) \times 10^{-3} \]

\[ \text{Time (days)} \]

| \( p_0 \) | \( R_0 \) |
|---------|---------|
| 0.05    | 0.33    |
| 0.10    | 0.66    |
| 0.16    | 1.01    |
| 0.19    | 1.25    |
| 0.25    | 1.52    |