

Early die out events in SIR epidemic models

Jonathan Ballone

Montclair State University, jonathandballone@gmail.com

Follow this and additional works at: <https://scholar.rose-hulman.edu/rhumj>

Recommended Citation

Ballone, Jonathan (2010) "Early die out events in SIR epidemic models," *Rose-Hulman Undergraduate Mathematics Journal*: Vol. 11 : Iss. 1 , Article 3.

Available at: <https://scholar.rose-hulman.edu/rhumj/vol11/iss1/3>

Early die out events in SIR epidemic models.

Jonathan Ballone

May, 2010

Abstract

We are interested in the behavior of an SIR epidemic model with respect to low-probability events. Specifically, we want to identify the probability of the early die out of a disease. Ordinary differential equations are commonly used to model SIR systems. However, this approach fails to describe the spontaneous die out event. We develop a Markov SIR model from which the probability of early die out can be captured. Additional simulations reveal that this model agrees closely with the ODE solutions when this low-probability event is ignored.

1 Introduction

In this article, we compare three algorithms for modeling the spread of a disease through a population. Markov chains can be used to model systems that switch among several states, with the next state depending probabilistically only on the current state. Because we are modeling a discrete random process, the Markov chain does not predict the exact state of the system in the future. However, it does approximate the probability of the system transitioning to each of the possible states at a future time. An agent-based model, which is a Monte Carlo simulation using random variables, can be used to simulate how a system switches from state to state in time. To determine if an event has occurred, a random number with uniform distribution is generated and compared to the probability of the event occurring. The general behavior of the system can be obtained by averaging the results of many simulations, but there are few analytic tools for this method and computational time can be costly. Ordinary differential equations capture the average behavior of a system by approximating how the system changes in

time. Solutions are continuous and there are well defined methods to analyze their stability and long term behavior. All three methods are commonly used to study epidemiology.

The goal of this study is to identify low probability events, which are not captured by the use of ordinary differential equations. In particular, we are interested in an early or spontaneous die-out of a disease. While differential equations capture the average behavior of a system for a large population, this approach cannot capture spontaneous die out. Markov models and agent-based simulations can be used to understand the conditions for which the early die-out event is most likely to occur and can aid in identifying strategies that minimize or eradicate a disease from a population.

This work extends the results of [2], in which the authors consider modeling the spread of a computer virus, with the possibility of reinfection after the removal of the virus. The authors compare the methods of Markov chains, differential equations, and agent-based simulations. In that scenario, each computer could be classified as either susceptible or infected. Since the state of the network is fully defined by the number of computers in either class, the dynamics can be captured by one-dimensional model. Comparing the methods revealed differences in the behavior that each model can simulate.

In this paper, we extend that approach to consider any disease spread in which life-long immunity is conferred after infection. For this type of system each node must be in one of three conditions: susceptible, infectious or recovered; this is commonly referred to as the SIR model. The state of a population is determined by the number of nodes in any two of these classes and two-dimensional models are required for analysis. The SIR pattern is usually found in childhood diseases, such as measles, mumps, and chickenpox. It is well known that for the SIR model, all solutions will limit to the disease free solution asymptotically. We are interested in the maximum number of infectives for an average realization and if that number can be decreased.

2 The Markov Model

In this model, a disease is introduced into the network so that one or more individuals are initially infected. We assume that all others start in the susceptible condition (although some could begin as recovered, perhaps to simulate previous vaccinations). In one time step, an infective individual can transmit the infection to a susceptible or become recovered. No individ-

ual becomes recovered without first becoming infected. Once an individual becomes recovered, they will remain in that condition. The state of the network is characterized by the number of individuals in each condition. Using a Markov model approach, we calculate the probability of transitioning from each state to every other. The probability distributions are collected in a matrix, which is used to generate the expected value for the number of infected individuals over a given number of time steps.

We consider a population with a total of N individuals. The individuals in this population are not uniquely identified. However, the average connectivity in the population, c , is known. The state of the network is given by the triple (S, I, R) where S , I , and R are the number of individuals in the conditions susceptible, infective and recovered, respectively. Since the size of the network is fixed, note that each state is fully defined by the pair (S, I) , with $R = N - (S + I)$ implied.

We make a list, q , of all possible states for the network. Let k be the length of this list, so that q is a k by 3 matrix with each row storing the triple for one state. States are referenced by their row index. The rows of q are generated with an assignment loop of the following form:

$$[S, I, R] = [i, j, N - (i + j)], \text{ for } 0 \leq i \leq N \text{ and } 0 \leq j \leq N - i. \quad (1)$$

In the first state, all individuals are susceptible. Successive states are organized in blocks, by decreasing the value of S and then I . The following is an example of q with $N = 2$:

$$\begin{pmatrix} 2 & 0 & 0 \\ 1 & 1 & 0 \\ 1 & 0 & 1 \\ 0 & 2 & 0 \\ 0 & 1 & 1 \\ 0 & 0 & 2 \end{pmatrix}. \quad (2)$$

The length of q can be found in the following way. For every value of S , there are $N - S + 1$ possible values for I , each of which fully defines a state. Using (2) as an example, we see that k is the sum of the first $N + 1$ positive integers, also known as the $N + 1$ triangular number. This value can be computed directly with a binomial coefficient: $k = \binom{N+2}{2}$.

Organizing the states in this way has a major programming benefit. Consider the probability of transitioning from a state i to a state j . With $i \leq j$,

this is the probability of a certain number of individuals moving from S to I and I to R . With $i > j$, the probability of a transition is always zero, because such a transition would involve some individuals moving from I to S or from R to I or from R to S . So as i increases, there are fewer states for which valid transitions are defined and must be calculated.

We use the list of all possible states for the network, q , to index the rows and columns of a transition matrix Q . In this way, each entry of Q is associated with a unique pair of states: (S, I, R) and (S', I', R') . The entry $Q(i, j)$ corresponds to the probability of transitioning from state (S, I, R) to state (S', I', R') .

The transition probabilities in Q are derived in the following way. At a given time, let $x = S - S'$ be the number of individuals that become infected ($S \Rightarrow I$) and let $z = R' - R$ be the number of individuals that become recovered ($I \Rightarrow R$). Let μ be the probability of one individual becoming infected and δ be the probability of one individual becoming recovered. Then $Q(i, j)$ is given by

$$\binom{S}{x} (\mu^x) (1 - \mu)^{S-x} \binom{I}{z} (\delta^z) (1 - \delta)^{I-z}. \quad (3)$$

We use the product of independent binomial distributions to calculate the probability for any x individuals to become infected and any z individuals to become recovered, in the same time step.

The value of μ is not a constant. It is a function of the probability of transmitting the virus, β , and the connectivity of the network, c . Let y be the number of infected neighbors for an individual. Then the probability of a individual becoming infected by at least one connected infective neighbor is given by the following formula, which was derived in [2]:

$$\mu = \sum_{y=1}^I (1 - (1 - \beta)^y) \binom{I}{y} (c^y) (1 - c)^{1-y}. \quad (4)$$

The size of Q is k^2 , which becomes large very quickly. For example, with $N = 100$, Q has 5151^2 entries. The brute-force approach to generating Q involves calculating a sparse matrix with many small entries. However, the structure of q guarantees that Q is an upper diagonal matrix. So Q is initially a matrix of zeros and entries are generated one row at a time, beginning with $Q(i, i)$. Since each row contains a probability distribution,

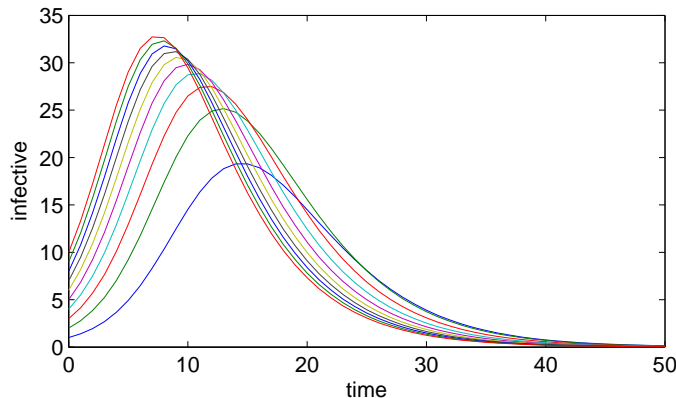


Figure 1: Time series of expected values for number of infective individuals using the Markov model. Parameters are set to $N = 100$, $\beta = 0.12$, $c = 0.0505$, and $\delta = 0.2$. Initial conditions are sampled from $I = 1, 2, \dots, 10$.

the sum along each completed row is 1. As j increases, states i and j become significantly different. The result is that entries farther off the diagonal are generally smaller. For example, it is not likely that everyone in the population will become infective if currently only one person is infective. In practice, we keep a running sum of the entries in the current row and neglect the remaining entries once the sum becomes close enough to 1 as specified by an acceptable tolerance.

We also identify the following patterns in generating Q that save time computationally. Consider that the number of possible values for $\binom{S}{x}$ is the same as those for $\binom{I}{z}$, there are $N + 1$ of them, with each component of the binomial coefficient ranging from 0 to N , so all of these values can be stored in a square matrix of size $N + 1$. However, all of the entries on the diagonal, $S = x$, are 1 and entries above the diagonal, $S < x$ are always zero. So instead of calculating two binomial coefficients for each entry in Q , as required for 3, we generate a table of binomial coefficients and reference them as needed. Similar tables can be generated for the rest of the elements of the product, as well as the values of μ . This approach involves more start-up time and space, but as N increases, the savings in time also increase.

The matrix Q can be used to generate a time-series for the expected number of infected individuals given an initial condition. The initial condition

is the state of the network at time $t = 0$. An initial condition vector v_0 is of length k and has a one in the position that corresponds to the initial state of the network, indicating with 100% probability that it is the initial state of the network. The probability of how the system will evolve in one time step is given by $v_1 = Qv_0$. The entries in v_1 represent the probability of the system realizing each state, at the next time step, given the initial condition. If we continue in time, the probability distribution at time $t = n$ is $v_n = Q^n v_0$. We find the expected number of infective individuals at time $t = i$ by multiplying the probabilities in v_i by the number of infectives in each associated state (second column of q) and taking the sum of that vector. See Figure 1 for an example time series of expected values for the number of infective individuals, sampling initial conditions of 1 to 10 infective in a population of $N = 100$.

3 The Differential Equation Model

One must be careful in generating the matrix Q for large values of N , because memory requirements will impose a limitation on the size of that matrix. Therefore, we continue with the most common and fastest approach to finding the dynamics of an SIR model with a large population. Using a continuous time perspective, a system of ordinary differential equations can describe the process that allows the SIR system to change states [1, 8]. The number of infected individuals increases by the number of susceptible nodes that get infected. This rate is captured by $\mu SI/N$, noting the dependence of μ on I as shown in (4). The number of infected nodes decreases by the number of infected nodes that are cured, δI .

Let $s = S/N$, $i = I/N$, and $r = R/N$ be the proportion of susceptible, infective, and recovered nodes respectively. The system can be described by the following system of equations:

$$\begin{aligned} \frac{ds}{dt} &= -\mu_N si, \\ \frac{di}{dt} &= \mu_N si - \delta i, \\ \frac{dr}{dt} &= \delta i, \end{aligned} \tag{5}$$

with μ_N defined as μ in (4), but using the next smallest integer I of N

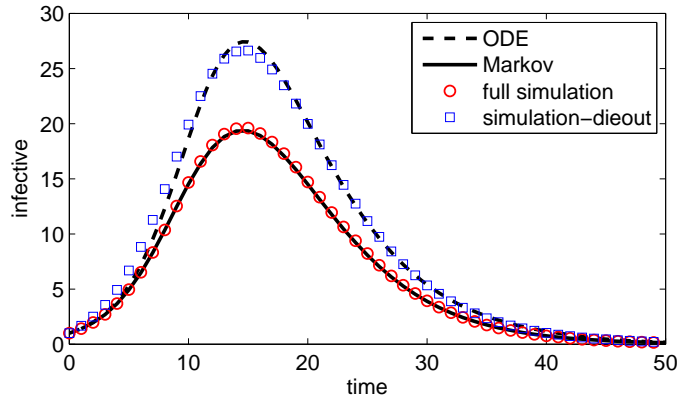


Figure 2: Time series of expected values for number of infective individuals using the Markov model and the ordinary differential equations model. Overlaid are simulations with and without early die out, time series averaged over 10,000 realizations. See text for details. Parameters are set to $N = 100$, $\beta = 0.12$, $c = 0.0505$, $\delta = 0.2$, and initial condition $I = 1$.

nodes infected. Computationally, we approximate μ_N as a piecewise constant function on a fixed time interval.

As shown in Figure 2, the ODE solution exhibits a larger outbreak than the solution for the Markov model. We attribute the difference to the occurrence of early die out events, which can be clearly defined by the distribution of maximum outbreak sizes in an agent-based simulation.

4 The Agent Based Model

An agent-based simulation of the infection process tracks the state of N individuals in time. At each time step we simulate an event by generating a random number between 0 and 1 and seeing if it falls in the probability defined by the model. For example, we start by determining if a connection exists between a particular infective and a particular susceptible. The probability of this event is given by c . Then we test to see if the disease was transmitted (β). Finally, we test each infective individual for a recovery event (δ). We update the individuals' conditions and repeat for the next time step. The times series collects the average number of infectives, at each

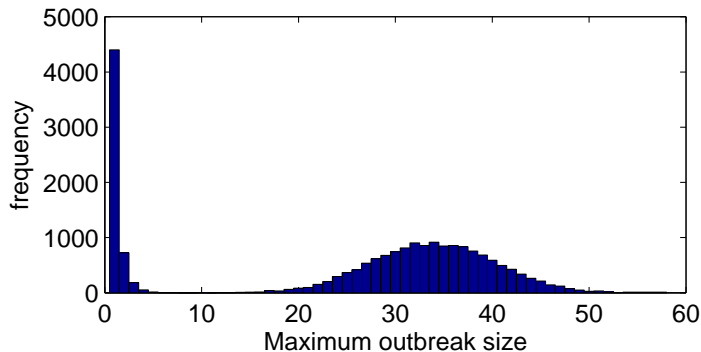


Figure 3: Associated distribution of total number of infective individuals over the time series for 20,000 realizations.

time step, over a large number of simulations. See Figure 2 for an example of $N = 100$ and an initial condition of 1 infected individual. Averaged over 20,000 realizations, we see that this agent-based simulation approach (circles) agrees with the Markov process (solid curve).

If we examine the distribution of maximum outbreak sizes from the simulation realizations, we see two distinct behaviors as shown in Figure 3. The first is a grouping of very small maximums, mostly less than five. The other is a grouping of large maximums, mostly between 25 and 45. We call the first grouping the early die out group, because the disease quickly dies out without infecting many individuals. Removing the early die out group from consideration results in an average behavior that is close to the ODE solution, as shown by the squares in Figure 2.

5 Conclusions

We conclude by discussing the need for additional analysis of early die out realizations, which are missed by deterministic ODE models. Most endemic diseases exhibit oscillations, which have low minimums in between outbreak years. In this paper, we simulate the random events in the state variables (S , I , R), to show the variability in realizations for low numbers of infectives. Markov models provide an analytical way to identify the behavior of the early die out events. The state variables are only one source that could facilitate an

early die out event in these low years, commonly called fade-outs in epidemiology [5]. Parameters such as the contact rate (including seasonal forcing [11]), birth rate [6, 10], age structure [3], and different demographic and geographic patterns [9, 4, 7] have all been considered as possible candidates to elicit the onset of complex fluctuations in this class of models. Further analysis of Markov models and stochastic differential equations, might give insight into the circumstances under which noise may be more easily ignored versus where it must be included to fully understand the early die out events in these systems. These results could influence future vaccination strategies.

6 Acknowledgments

Research was supported by the National Science Foundation (Award No. DMS-0806218) and the National Institute of General Medical Sciences (Award No. R01GM090204). The content is solely the responsibility of the the authors and does not necessarily represent the official views of the National Institute of General Medical Science or the National Institutes of Health.

References

- [1] R. M. Anderson and R. M. May. *Infectious diseases of humans: Dynamics and Control*. Oxford University Press, Oxford, 1991.
- [2] L. Billings, W. M. Spears, and I. B. Schwartz. A unified prediction of computer virus spread in connected networks. *Physics Letters A*, 297(3-4):261–266, 2002.
- [3] B. Bolker. Chaos and complexity in measles models - a comparative numerical study. *IMA J. Math. Appl. Med.*, 10:83–95, 1993.
- [4] B. M. Bolker and B. T. Grenfell. Chaos and biological complexity in measles dynamics. *P. Roy. Soc. B - Biol. Sci.*, 251:75–81, 1993.
- [5] D. A. T. Cummings, I. B. Schwartz, L. Billings, L. B. Shaw, and D. S. Burke. Dynamic effects of antibody-dependent enhancement on the fitness of viruses. *Proc. Natl. Acad. Sci.*, 102:15259–15264, 2005.

- [6] D. J. D. Earn, P. Rohani, B. M. Bolker, and B. T. Grenfell. A simple model for complex dynamical transitions in epidemics. *Science*, 287:667–670, 2000.
- [7] B. T. Grenfell, B. M. Bolker, and A. Kleczkowski. Seasonality and extinction in chaotic metapopulations. *P. Roy. Soc. B - Biol. Sci.*, 259:97–103, 1995.
- [8] H. W. Hethcote. The mathematics of infectious diseases. *SIAM Review*, 42:599–653, 2000.
- [9] A. L. Lloyd and R. M. May. Spatial heterogeneity in epidemic models. *J. Theor. Biol.*, 179:1–11, 1996.
- [10] I. B. Schwartz. Small outbreaks in seasonally driven epidemics. *J. Math. Biol.*, 30:473–491, 1992.
- [11] I. B. Schwartz and H. L. Smith. Infinite subharmonic bifurcations in an SEIR epidemic model. *J. Math. Biol.*, 18:233–253, 1983.