1 Introduction

In the 14th century, the Black Death killed nearly 100 million people worldwide. Diseases as aggressive and easily transferrable as the bubonic plague are studied from a biological perspective. Biologists often ask questions about the nature of the virus or bacteria responsible for the disease and study the pathways by which the disease moves through a population. The goal being not only understanding but control; ensuring that we, the population, are safe from the effects of the disease. Equally important in the characterization of the disease is the mathematical study of the outbreak and its subsequent demise; modeling the epidemic.

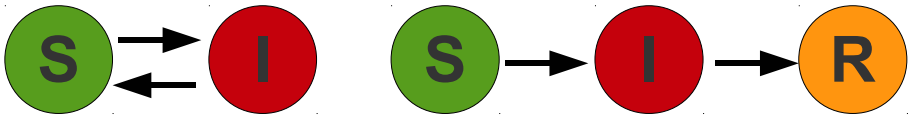
Mathematical epidemiology aims to understand the movement of a disease through a population. Models of this movement are compartmental; the population is divided into compartments and transfer rates between the compartments are measured from the best available data. Models span the space from relatively simple ordinary differential equations to complex dynamical systems requiring numerical techniques to solve. The purpose of this paper is to cite the results from two canonical models and apply the techniques from these models to a slightly more complicated case.

2 A couple models

The modeling of epidemics follows from a few simple considerations:

- the population under consideration can be sufficiently compartmentalized into distinct groups, and
- there is a rate (fixed or variable) at which individuals can move between compartments.

These two ideas provide a framework for the rate equations that govern the population for a particular model. For this paper, we will consider only the two simplest models and extend one of them to an interesting case.



(a) Movement in the SIS model (b) Movement in the SIR model

Figure 1: Compartment diagrams for the SIS and SIR models

2.1 The SIS Model

The Susceptible-Infected-Susceptible (SIS) model describes an epidemic in which healthy individuals (Susceptibles) can become infected with the disease in question (Infected). The infected individuals can be cured but retain no natural immunity to the disease (i.e., become susceptible again). Fig. 1(a) illustrates the compartments and flux of individuals in the SIS model. Such models are suitable for bacterial infections.

The rate at which susceptibles are infected is related to the contact rate (β). The recovery rate (α) describes how quickly individuals are cured. The rate equations that govern this model are two ordinary non-linear differential equations. Here we have neglected the birth and death rates of the

populations by making the approximation the epidemic occurs very quickly. The total number of individuals in the population is N.

$$\frac{dS}{dt} = -\frac{\beta}{N}SI + \alpha I \tag{1a}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - \alpha I \tag{1b}$$

We solve these equations in Sec. 3.1.

2.2 The SIR Model

The Susceptible-Infected-Recovered (SIR) model describes an epidemic in which healthy individuals (Susceptibles) can become infected with the disease in question (Infected). The infected individuals can be cured and retain a natural immunity to the disease (Recovered). Fig. 1(b) illustrates the compartments and flux of individuals in the SIR model. Such models are suitable for viral infections. It is this model that we modify in Sec. 4.1 to include the flux of individuals from the Recovered compartment into the Infected compartment.

The transfer rates can be defined for the SIR model as was for the SIS model. The rate equations that govern this model are three ordinary non-linear differential equations. Again, we have neglected the birth and death rates of the populations by making the approximation the epidemic occurs very quickly.

$$\frac{dS}{dt} = -\frac{\beta}{N}SI \tag{2a}$$

$$\frac{dI}{dt} = \frac{\beta}{N}SI - \alpha I \tag{2b}$$

$$\frac{dR}{dt} = \alpha I \tag{2c}$$

We solve these equations in Sec. 3.2.

2.3 Why use a Stochastic Model?

The ordinary differential equations that describe our models are sufficient to get a sense for the epidemic “on average”. However, by using a stochastic model, we can get a true sense for the average. In addition, a stochastic model might produce some trajectories (e.g., for the Infected compartment) that diverge from the deterministic model. Stochastic models can give a measure of the probability of an outbreak rather than the simple result that all deterministic models produce. It might also be possible to measure the average temporal extent of the outbreak.

3 Stochastic Modeling

We can use the deterministic models to derive the stochastic difference equations (SDEs). The approach involves two basic assumptions:

- the model obeys the Markov property, and
- the time step is small enough that the change in each compartment is approximately normally distributed.

With these assumptions, the SDEs will have the form

$$X(t+\Delta t) = X(t) + E(\Delta X(t)) + \sqrt{\mathbf{V}(\Delta X(t))} \tag{3}$$

where $E(\Delta X(t))$ is the expectation value of the change in the compartments and $\mathbf{V}(\Delta X(t))$ is the covariance of the change in the compartments.

Let $\vec{\lambda}_j = [\lambda_{1,j}, \lambda_{2,j}, ...\lambda_{n,j}]^\mathsf{T}$ represent a change (i.e., the j_{th} change) in the compartments $\Delta X = [\Delta X_1, \Delta X_2, ..., \Delta X_n]^\mathsf{T}$ that occurs with probability p_j. Then we can write the expectation and covariance of ΔX,

$$E(\Delta X) = \sum_{j=1}^{m} p_j \vec{\lambda}_j \Delta t \tag{4}$$

$$\mathbf{V}(\Delta X) = \sum_{j=1}^{m} p_j \vec{\lambda}_j (\vec{\lambda_j})^\mathsf{T} \Delta t. \tag{5}$$

The $\vec{\lambda}_j$'s and p_j's can be easily read off from the rate equations as we will see in Secs. 3.1, 3.2, and 4.1.

3.1 The Stochastic SIS model

The focus of this stochastic model is the number of infected individuals $I(t)$. In the SIS model, the number of healthy individuals can easily be calculated, $S(t) = N - I(t)$. Hence, only the rate equation for I is necessary to formulate the model,

$$\frac{dI}{dt} = \frac{\beta}{N}(N-I)I - \alpha I. \tag{6}$$

From Eqn. 6 we see that the number of infected individuals increases by 1 with probability $\frac{\beta}{N}(N-I)I$ and decreases by 1 with probability αI. Table 1 summarizes the results.

$\vec{\lambda}_j$	p_j
$[1]^\mathsf{T}$	$\frac{\beta}{N}(N-I)I$
$[-1]^\mathsf{T}$	αI

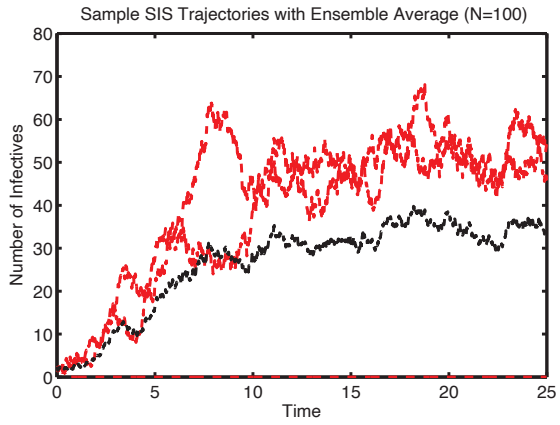
Table 1: The changes in infected population, $\vec{\lambda}_j$ occur with probability p_j in the SIS model.

The problem is one-dimensional so that the expectation and variance are scalars, $\mu(I)$ and $\sigma^2(I)$. The infinitesimal mean and variance at a time t are $\mu(I) = \frac{\beta}{N}I(N-I) - \alpha I$ and $\sigma^2(I) = \frac{\beta}{N}I(N-I) + \alpha I$. We can use these results to model ΔI over a small Δt by assuming it is approximately normally distributed. Thus,

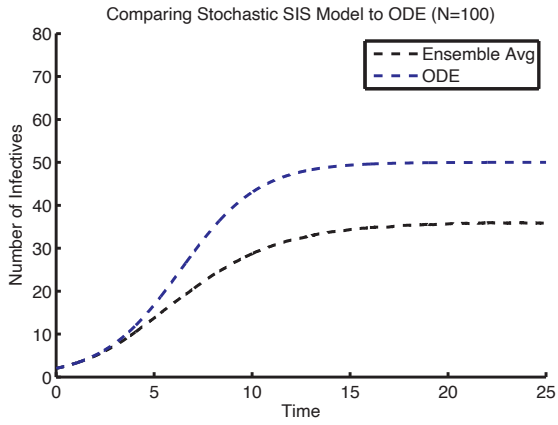
$$I(t+\Delta t) = I(t) + \mu(I)\Delta t + \sigma(I)\sqrt{\Delta t}\,\eta$$

where $\eta \sim \mathbf{N}(0,1)$, a random variable that is normally distributed between zero and one.

Using this model, we computed several sample trajectories (for population size $N = 100$) to illustrate typical time series. What is unique about this approach is that it predicts trajectories that do not result in an epidemic (see Fig. 2(a)). That is, after a short time the number of infected individuals drops to zero and remains so for the integration time.



(a) Sample trajectories of Infected population using the stochastic SIS model (red) are plotted along with their ensemble average (black). One trajectory decays to zero very quickly resulting in no infected individuals at the final integration time.



(b) The ensemble average (10 000 simulations) of the stochastic model (black) predicts a lower number of infected individuals compared to the numerical solution of the SIS ODEs (blue). In the stochastic model a signficant number of trajectories decays to zero very quickly resulting in no infected individuals at the final integration time.

Figure 2: A few trajectories from the stochastic SIS model are plotted and the stochastic model is compared to the deterministic model.

In addition, we find that integrating 6 over-estimates the number of infected individuals because the effect of these non-epidemic final states is significant (see Fig. 2(b)). In fact, running a simulation consisting of 10 000 realizations produces a moderate probability of a non-infected final state, $p(I = 0, t = 25) \approx 0.28$.

With these striking results from this model, we turn next to the stochastic SIR model.

3.2 The Stochastic SIR model

The Stochastic SIR model can be derived in a similar manner to the SIS model; where the process is now bivariate. The solution will again be modeled using Eqn. 3, where $\Delta X = [\Delta S, \Delta I]^{\mathsf{T}}$.

We can construct the expectation and covariance of ΔX using Eqn. 2. We can neglect the equation for R as it does not appear in any of the other ODEs. However, in doing so we must include both the equations for S and I since the $N = S + I + R$. Table 2 summarizes the results.

$\vec{\lambda}_j$	p_j
$[-1, 1]^{\mathsf{T}}$	$\frac{\beta}{N} SI$
$[0, -1]^{\mathsf{T}}$	αI

Table 2: The changes in susceptible and infected populations, $\vec{\lambda}_j$ occur with probability p_j in the SIR model.

The expectation and covariance of ΔX are then constructed. Notice, as expected, the expectation value is a 1x2 matrix and the covariance is 2x2.

$$E(\Delta X(t)) = \begin{pmatrix} -\frac{\beta}{N}SI \\ \frac{\beta}{N}SI - \alpha I \end{pmatrix} \Delta t \tag{7}$$

$$\mathbf{V}(\Delta X(t)) = \begin{pmatrix} \frac{\beta}{N}SI & -\frac{\beta}{N}SI \\ -\frac{\beta}{N}SI & \frac{\beta}{N}SI + \alpha I \end{pmatrix} \Delta t \tag{8}$$

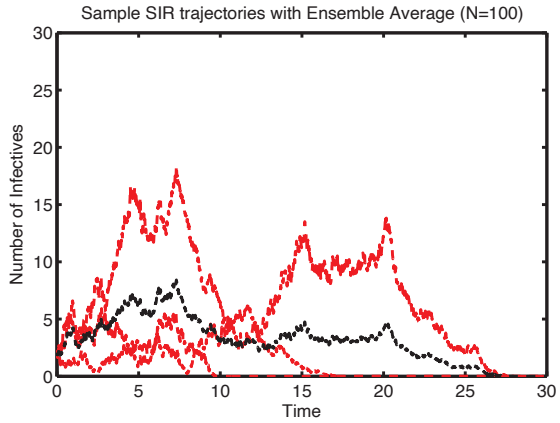
Let $\mathbf{B}\sqrt{\Delta t} = \sqrt{\mathbf{V}}$, the resulting system of stochastic difference equations is as follows:

$$S(t + \Delta t) = S(t) + E_1 + B_{11}\eta_1 + B_{12}\eta_2 \tag{9a}$$

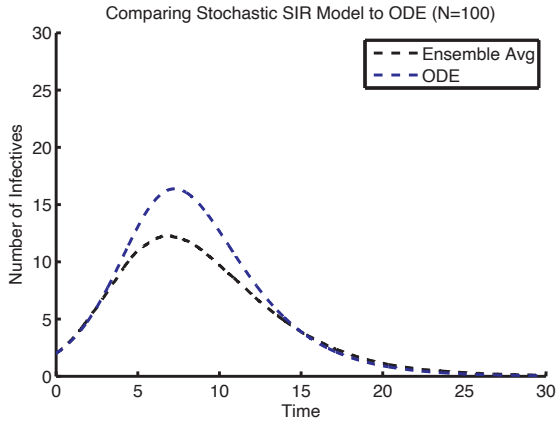
$$I(t + \Delta t) = I(t) + E_2 + B_{21}\eta_1 + B_{22}\eta_2 \tag{9b}$$

where η_1 and η_2 are independent normally distributed random variables.

Using this model, we computed several sample trajectories (for population size $N = 100$) to illustrate typical time series. What is unique about this approach is that it predicts trajectories that do not result in an epidemic and trajectories that cycle briefly (see Fig. 3(a)).



(a) Sample trajectories of Infected population using the stochastic SIR model (red) are plotted along with their ensemble average (black). Two trajectories decay to zero very quickly resulting in no infected individuals at the final integration time.



(b) The ensemble average (1 000 simulations) of the stochastic model (black) predicts a lower number of infected individuals at the peak compared to the numerical solution of the SIR ODEs (blue).

Figure 3: A few trajectories from the stochastic SIR model are plotted and the stochastic model is compared to the deterministic model.

In addition, we find that integrating Eqn. 2 over-estimates the maximum number of infected individuals because the effect of low infection states is significant (see Fig. 3(b)).

4 A novel problem

Consider a modified SIR model in which individuals in recovery, R compartment, can return to the infected population, I compartment. We could modify Fig. 1(b) by including an arrow that points from the R compartment to the I compartment. There are few diseases that have this characteristic. One such “disease” is zombification, the resurrection of the dead as flesh-eating monsters. For this example, it is more instructive (i.e., less confusing) to refer to this model as the Susceptible-Zombie-Removed (SZR) model.

4.1 The SZR model

This SZR model treats the population as compartmentalized into the 3 groups: Susceptible (S), Zombie (Z), and Removed (R). Movement between these groups is illustrated in Fig. 4. Susceptibles can become zombies through an encounter with a zombie. Zombies can move to the removed compartment by being destroyed in classic manners (e.g., Dawn of the Dead). Susceptibles can move to the removed compartment through death by a non-zombie encounter. Finally, removed individuals can become zombies through typical resurrection techniques (e.g., Live and Let Die).

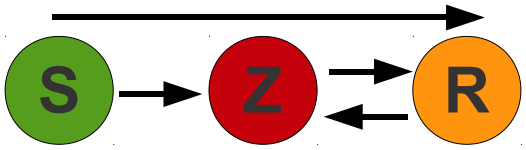


Figure 4: Movement in the SZR model

The deterministic model includes transfer rates we have seen before, but we include the rates of transfer from susceptible to removed (δ) and removed to zombie (ζ).

$$\frac{dS}{dt} = -\beta SZ - \delta S \tag{10a}$$

$$\frac{dZ}{dt} = \beta SZ + \zeta R - \alpha SZ \tag{10b}$$

$$\frac{dR}{dt} = \delta S + \alpha SZ - \zeta R \tag{10c}$$

To model these rate equations stochastically, we utilize the method presented in Sec. 3 and used in Secs. 3.1 and 3.2. We propose that the changes in the compartments, $\Delta X = [\Delta S, \Delta Z, \Delta R]^\mathsf{T}$, is approximately distributed normally so we can use Eqn. 3. Notice in this model all the equations are relevant because they are all coupled. Table 3 summarizes the results.

$\vec{\lambda}_j$	p_j
$[-1, 1, 0]^\mathsf{T}$	βSZ
$[-1, 0, 1]^\mathsf{T}$	δS
$[0, 1, -1]^\mathsf{T}$	ζR
$[0, -1, 1]^\mathsf{T}$	αSZ

Table 3: The changes in susceptible, zombie, and removed populations, $\vec{\lambda}_j$ occur with probability p_j in the SZR model.

The results from Table 3 helps to construct the stochastic difference equations. Notice the expectation value is a 1x3 matrix and the covariance matrix is 3x3 as expected.

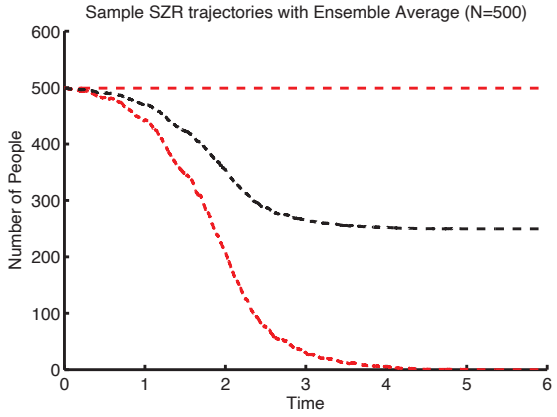
$$E(\Delta X(t)) = \begin{pmatrix} -\beta SZ - \delta Z \\ \beta SZ + \zeta R - \alpha SZ \\ \delta S + \alpha SZ - \zeta R \end{pmatrix} \Delta t \tag{11}$$

$$\mathbf{V}(\Delta X(t)) = \begin{pmatrix} \beta SZ + \delta S & -\beta SZ & -\delta S \\ -\beta SZ & \beta SZ + \zeta R + \alpha SZ & -\zeta R - \alpha SZ \\ -\delta S & -\zeta R - \alpha SZ & \delta S + \zeta R + \alpha SZ \end{pmatrix} \Delta t \tag{12}$$

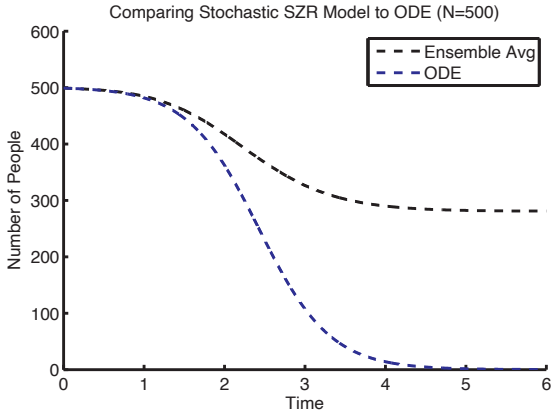
Let $\mathbf{B}\sqrt{\Delta t} = \sqrt{\mathbf{V}}$, the resulting system of stochastic difference equations is as follows:

$$S(t+\Delta t) = S(t) + E_1 + B_{11}\eta_1 + B_{12}\eta_2 + B_{13}\eta_3 \tag{13a}$$
$$Z(t+\Delta t) = Z(t) + E_2 + B_{21}\eta_1 + B_{22}\eta_2 + B_{23}\eta_3 \tag{13b}$$
$$R(t+\Delta t) = R(t) + E_3 + B_{31}\eta_1 + B_{32}\eta_2 + B_{33}\eta_3 \tag{13c}$$

where η_1, η_2, and η_3 are independent normally distributed random variables.



(a) Sample trajectories of the Susceptible population using the stochastic SZR model (red) are plotted along with their ensemble average (black). One trajectory decays to zero (all susceptibles die out) while the healthy remain populous for the integration time (almost all susceptibles live) in another. These simulations started with one zombie in the population.



(b) The ensemble average (500 simulations) of the stochastic model (black) is compared to the numerical solution of the SZR ODEs (blue). In the stochastic model a significant number of trajectories result in the zombie population dying out quickly and thus the healthy population remains. The SZR ODEs predict no such result for any non-zero starting number of zombies. These simulations started with one zombie in the population.

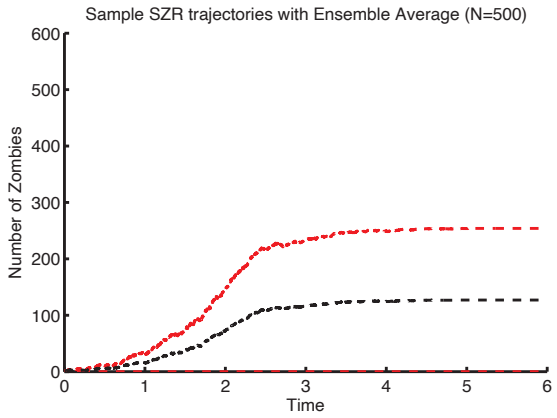
Figure 5: A few trajectories of healthy population from the stochastic SZR model are plotted and the stochastic model is compared to the deterministic model.

Using this model, we computed several sample trajectories (for population size $N = 500$ and $Z(0) = 1$) to illustrate typical time series. This approach is unique because it predict trajectories in which the zombies die out and healthy individuals remain populous (see Figs. 5(a) and 6(a)). The deterministic model (i.e., Eqn. 13) predicts that healthy individuals die or become zombies for any non-zero starting number of zombies (see Fig. 5(b)).

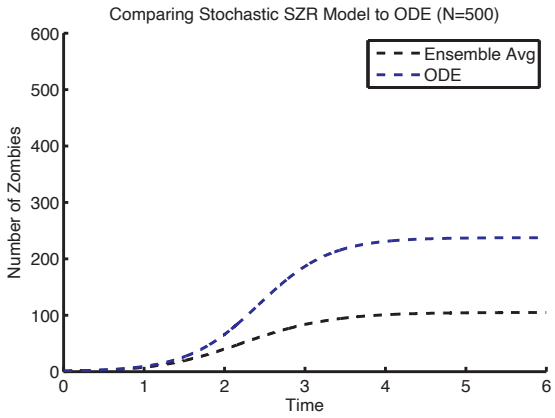
The probability of surviving a zombie outbreak predicted byt Eqn. 13 is zero for any non-zero starting number of zombies. In the deterministic model, zombies eventually dominate the population and the healthy individuals die out or become zombies. Using the stochastic model, we find that the probability of survivial is not so grim. In fact, an ensemble average of this model predicts that the survival rate is relatively high, $p \approx 0.66$. However, it should be noted that the population starts off with only one zombie, so maybe the result is grim.

5 Conclusion

By including the probabilistic effects in the modeling of diseases, a new picture starts to form. Individual trajectories might produce unforeseen effects and the resulting ensemble average might differ greatly from the “average” produced by rate equations.



(a) Sample trajectories of the Zombie population using the stochastic SZR model (red) are plotted along with their ensemble average (black). One trajectory decays to zero (zombies die out) while the zombies remain populous for the integration time (almost all susceptibles live) in another. These simulations started with one zombie in the population.



(b) The ensemble average (500 simulations) of the stochastic model (black) is compared to the numerical solution of the SZR ODEs (blue). In the stochastic model a significant number of trajectories result in the zombie population dying out quickly. The SZR ODEs predict no such result for any non-zero starting number of zombies. These simulations started with one zombie in the population.

Figure 6: A few trajectories of the zombie population from the stochastic SZR model are plotted and the stochastic model is compared to the deterministic model.

Modeling diseases stochastically also provides the added benefit calculating survival probabilities and mean infection times. While ODEs might provide this feature, it's clear from the examples presented in this paper that is not always the case. The effect was most dramatic in the SZR model which predicts a moderate survival probability for an ODE model that predicts no possibility of survival.

While the work presented in this paper is entirely numerical, it's possible that analytic techniques that applicable to the models presented in this paper could provide interesting and complementary results.

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[2] Brauer, F. and Van den Driessche, P. and Wu, J. and Allen, L.J.S. *Mathematical epidemiology*, Springer, 2008.

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