A Summary of Stochastic Epidemic Models of Influenza

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1 Introduction

1.1 Background

Last year, an estimated 80,000 individuals died from influenza in the United States alone [2]. Influenza, more well-known as the flu, is a common contagious viral infection that affects millions of people worldwide every year. This infectious disease mainly affects the upper respiratory system, with the common symptoms including fever, chills, cough, rhinorrhea, congestion, and sore throat. Symptoms typically occur 2 days after exposure and last about one week, with a contagious period of 3-5 days. Although the flu causes similar symptoms to other viruses, like the common cold, the severity of the symptoms can range from mild to severe, which can sometimes lead to hospitalization and/or death. Death, however, usually results from complications from the flu, such as pneumonia or exacerbation of chronic diseases like asthma, rather than the flu virus itself. This makes the young, elderly, and immunocompromised individuals in society the most at risk of acquiring the flu.

Contracting the influenza virus is not a very difficult task. What makes the flu dangerous is how easily it is spread. The influenza virus can spread from person to person simply through droplets, which are often transferred through the air or through direct contact with infected surfaces. This is why the flu always spreads in yearly outbreaks, typically beginning in the fall and peaking in winter. At these specific times of the year, people are more likely to stay indoors due to the colder weather and thus, have increased opportunities for close contact, which promotes disease transmission. This contagious characteristic of the flu has led to several large epidemics throughout history. In these epidemics, the influenza virus spread extremely rapidly and was hard to control. One of the most lethal outbreaks of the flu was the Spanish flu pandemic in 1918, in which 50-100 million people died. A majority of these outbreaks, like the Spanish flu, were caused by a specific type of flu, influenza A [3].

There are 3 main types of the flu that affect humans: type A, type B, and type C. Type A is the most virulent of all of the flu types and variations in this species, specifically variations in the surface proteins, leads to many different serotypes. Some well-known serotypes include H1N1, also known as the swine flu, H2N2, the Asian flu, and H5N1, the bird flu. The other two types of the flu are less common and induce less severe symptoms in humans. There

are many more subtypes and strains of influenza, which are a result of mutations within the antigenic structure of the virus. Mutation of the virus occurs readily and is what makes it possible for people to get the flu more than once. The virus undergoes up to $8x10^{-}3$ point mutations per year [3]. More information about the biology of influenza and how it infects humans can be found in [6].

While a lot is known about the flu virus and its different strains and genetic structure, treatment of the flu is still extremely limited. Since it is a viral infection, treatments largely consist of just symptom control. There are few antiviral drugs, namely oseltamivir (Tamiflu), that can decrease the severity of the symptoms of the flu if taken within a short time window after onset of symptoms, but the most effective form of treatment is not a drug, but prevention. One of the best preventative methods to protect against the flu is the flu vaccine. The flu vaccine is prepared annually and protects against several strains of the flu that are predicted to be most prominent that given season. Since there are so many different types of the virus, the flu vaccine needs to be updated every year. Additionally, promotion of good health habits, such as regularly washing hands, covering your mouth and nose when coughing or sneezing, staying at home when sick, etc, helps reduce the spread of the flu.

All of these precautions are needed because the influenza virus is highly contagious. From a public health standpoint, it is important to prevent a large outbreak of the virus, or worst case scenario, a pandemic outbreak of the disease. Pandemic influenza viruses have consistently emerged about every 8 to 41 years over the past several centuries /citestats. This is why it is advantageous to understand the dynamics of the spread of influenza in order to better control and prepare against it.

1.2 Overview

Mathematical models have been used to study many scientific processes and also have the potential to provide valuable insight into the dynamics of influenza. In this paper, I will review the well-known deterministic discrete SIR model and then extend the analysis of influenza beyond this model, studying several other derived SIR models based on several other papers [1], [4], [5]. These models include the deterministic continuous SIR model and its stochastic counterparts, the continuous time Markov chain model and stochastic differential equation model. I will compare the deterministic and stochastic approaches and discuss properties of each model in the context of providing unique information about the dynamics of the influenza virus. Finally, I will analyze the effects of using different influenza control methods.

2 Discrete SIR Model

One of the most well-known mathematical models of infectious disease is the SIR model. The SIR is a compartmental model that breaks down a population into three compartments: the susceptible (S), the infected (I), and the recovered (R). In the context of the flu, during

each flu season, every individual is susceptible to contracting the virus and once contract, becomes infected, moving from the susceptible compartment to the infected compartment. Then, after a short period of time, about 1-2 weeks (the duration of the flu), the infected individuals recover and attain immunity against the flu for the rest of the season. This cycle is depicted in Figure 1. In order to effective the following season, a different mutation of the flu will need to be spread since the number of susceptible individuals will be much smaller the season following the outbreak. This model works well for modeling other diseases in which individuals who become infected and acquire immunity, including chickenpox, mumps, and measles.

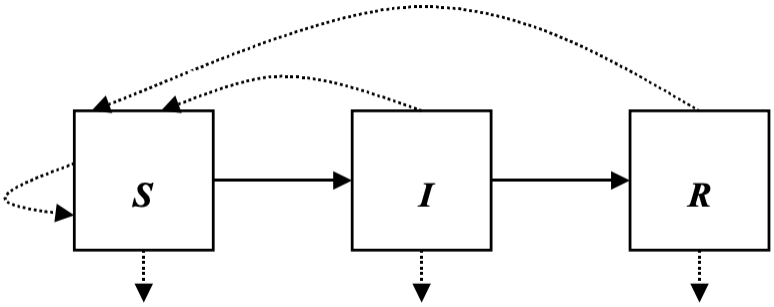

Figure 1: SIR Model

The SIR model is deterministic, meaning that there is no randomness in the system and that the system will behave the same way for a given set of initial conditions and parameters. While it may not be as realistic as a stochastic model, which will be explored later, it provides a good starting point for understanding flu dynamics as it is easy to interpret. The discrete form of the SIR model, where n is the number of weeks after the beginning of the flu season is shown in Equation 1.

$$\begin{aligned} S_{n+1} &= S_n - \frac{\beta}{N} S_n I_n \\ I_{n+1} &= I_n + \frac{\beta}{N} S_n I_n - \gamma I_n \\ R_{n+1} &= R_n + \gamma I_n \end{aligned} \tag{1}$$

The simplest version of this model makes simplifying assumptions that make analyzing the dynamics much easier. It assumes that the population, N, is constant, with no births or deaths. Represented mathematically, $N = S + I + R$. To further simplify this system of equations, since the first two equations are independent of R and R can be rewritten as a function of the other variables: $R = N - S - I$, the system of three equations can be reduced to just two equations that only depend on the two variables, S and I.

The SIR model has several important parameters that need to be defined. For each iteration (each week of the evolution of the disease), $\frac{\beta}{N}$ is the contact rate between susceptible and infected individuals in the population. The number of susceptible decreases at a contact rate

$\frac{\beta}{N}$ and, as a result, the number of infected individuals increases at the same rate. γ is the rate at which infected individuals are cured. The number of infected individuals decreases at a recovery rate γ and the number of recovered increases at this same rate.

Some other important terms that are derived from this set of equations is the average length of infectious period of the disease, $\frac{1}{\gamma}$, and the basic reproduction ratio, $R_0 = \frac{\beta}{\gamma}$. The basic reproduction ratio is a measure for how rapidly the virus will spread and how much of the population will be affected by the virus. Intuitively, an R_0 ¿ 1 implies that the disease is spreading (outbreak) since more people are getting the disease than recovering, and an $R_0 < 1$ implies that the disease is dying out since less people are getting the disease than recovering. The larger the magnitude of these values, the stronger the effect.

Because the discrete SIR model is a simple system of equations, one can determine the equilibria by setting the equations for $S = 0$ and $I = 0$. Linearizing about the equilibria provides information about the stability of the different equilibria, with the second eigenvalue of the Jacobian matrix determining the stability (Equations 2 and 3).

$$I_e = 0, 0 \leq S_e \leq N \tag{2}$$

$$\begin{bmatrix} S_{n+1} \\ I_{n+1} \end{bmatrix} = \begin{bmatrix} 1 & -\frac{\beta}{N} S_e \\ 0 & 1 - \gamma + \frac{\beta}{N} S_e \end{bmatrix} \begin{bmatrix} S_n \\ I_n \end{bmatrix} \tag{3}$$

3 Continuous SIR Model

The continuous SIR model is almost identical to the discrete SIR model with the difference being that the continuous SIR model is defined on a continuous time scale instead of a discrete one. The S, I, and R state variables, however, still remain discrete. Like with the discrete model, we are assuming a simple SIR model with no births or deaths, meaning a constant total population size. The total population size, $N = S(t) + I(t) + R(t)$. The disease-free equilibrium occurs when $S = N$ and when $I = R = 0$.

$$\begin{aligned} \frac{dS}{dt} &= \frac{\beta}{N} SI \\ \frac{dI}{dt} &= \frac{\beta}{N} SI - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{4}$$

Similarly, since this is a simple system of ordinary differential equations, one can determine the equilibria and stability by setting each equation equal to 0 and solving for the Jacobian matrix and its eigenvalues similar to the method shown for the discrete SIR model in the previous section. Details for this calculation are omitted.

3.1 Implementation

The continuous SIR model is a deterministic model and so a given set of initial conditions and parameters will yield distinct behavior. Numerical simulations will be computed for later stochastic models, but for this model, it is more valuable to fit the model to real-world data.

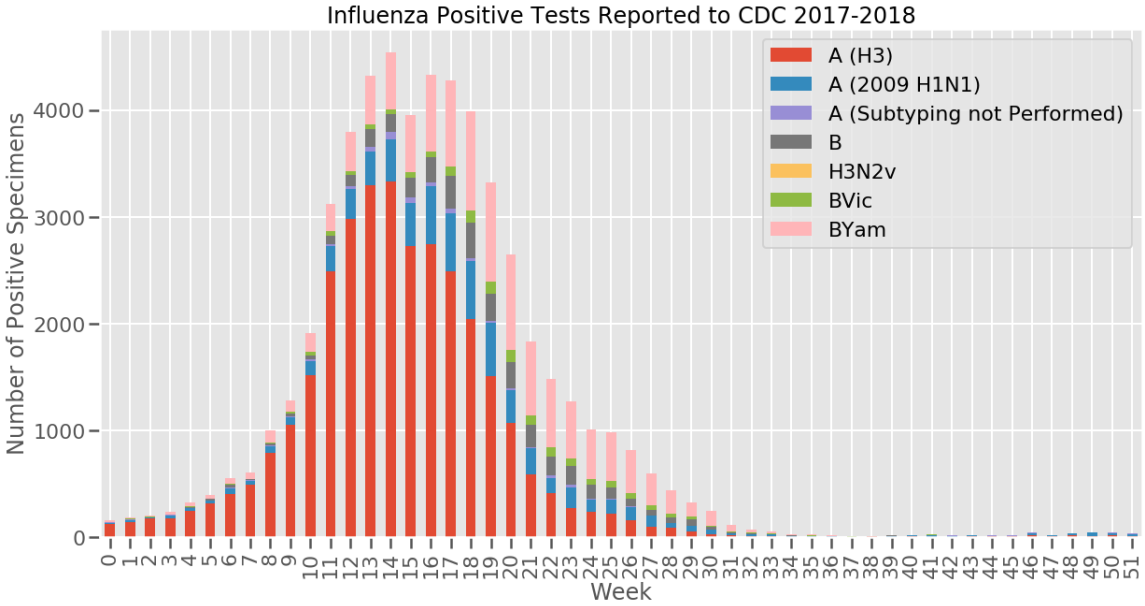

Figure 2: CDC Influenza Data from 2017-2018

One can determine the set of initial conditions and parameters, namely β and γ, that fit the real-world data best. The dataset used to fit the continuous SIR model will be the latest influenza data form CDC form 2017-2018 (Figure 2) [2], which provides weekly measures of the number of positive flu specimens for each tested strain versus the number of individuals sampled. Within the context of this fit, it can be assumed that the total population, N, will be the total number of individuals sampled and that the sum of positive flu specimens for each strain will be the total number of infected individuals, I, for a given week. The best fit will be calculated by minimizing the sum of squared errors between the model and the data at each of the discrete data points (Figures 3 and 4). For each of the following simulations in this paper, time is measured in weeks after the start of the current flu season to match with CDC data.

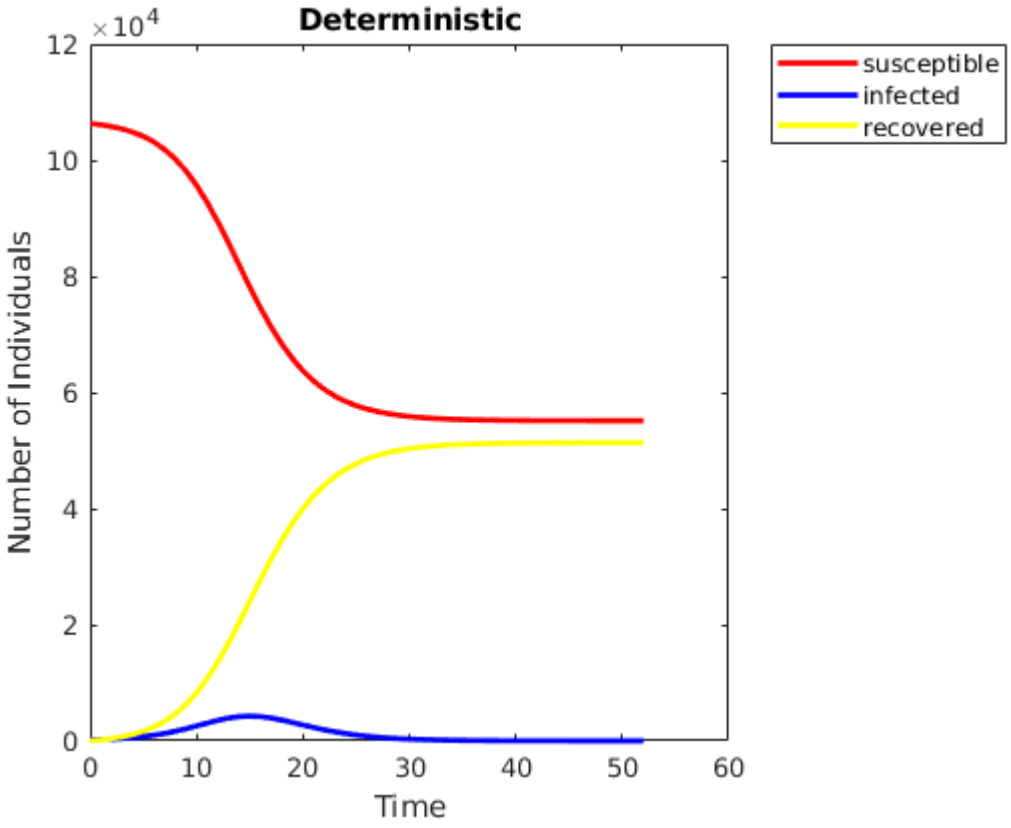

Figure 3: Continuous SIR Model (Best Fit to CDC Data)

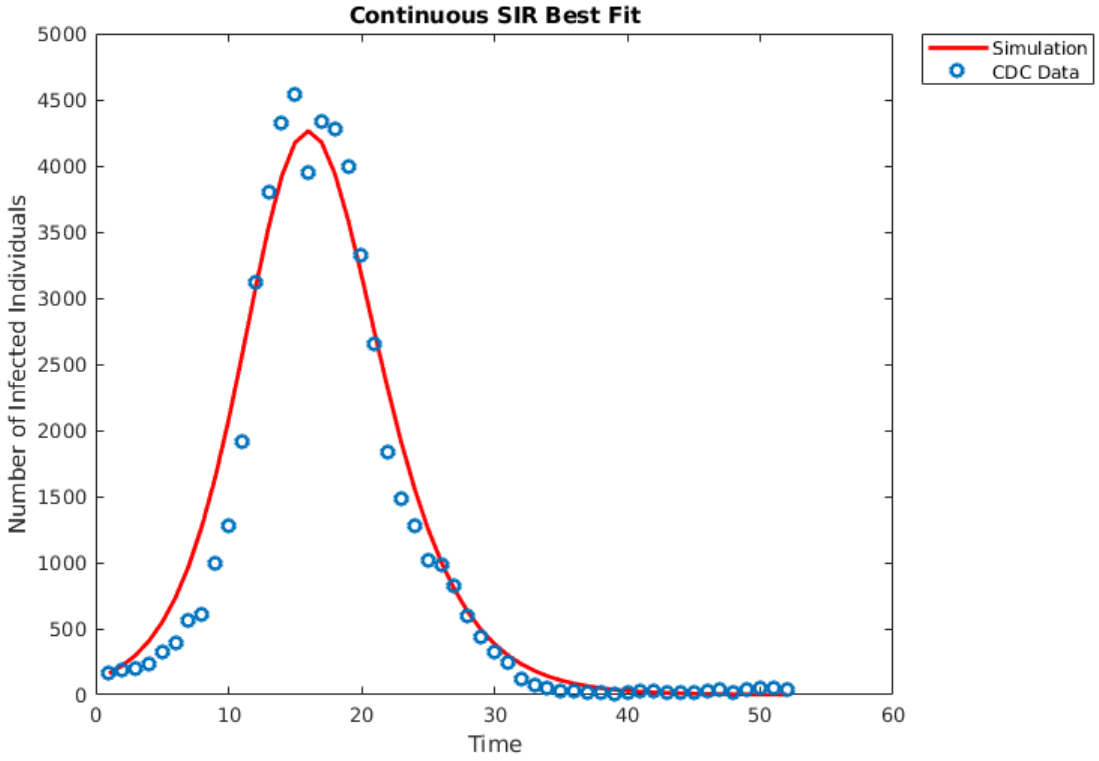

Figure 4: Continuous SIR Model (Best Fit to CDC Data)

For this best fit, the best fitting values are: $\beta = 1.1837$ and $\gamma = 0.8691$. Interpreting these values, this means that the average number of susceptible contacts is 1.1837 contacts per infected individual and average length of infectious period for influenza is $\frac{1}{\gamma} = 1.1506$ weeks. $R_0 = \frac{\beta}{\gamma} = 1.3620$, which means more people are getting the disease than recovering from it, implying the spread of the disease.

The total sum of squared errors is 3699300. Visually, the model fits the data relatively well and the magnitude of the sum of squared errors can be attributed to the large population sizes used for the fit (a small discrepancy between the model and data will be magnified for a squared error). In both the data and fit, there is a rapid increase and peak in the disease at around 15 weeks and then a rapid decline to the disease-free equilibrium thereafter. However, the fit does not quite reach the maximum peak indicated in the data and slightly overestimates the number of infected individuals in the first 10 weeks. Besides these small discrepancies, the fit aligns closely to the data points has depcits the same trends. These values, along with the initial values of $S_0 = 106487$ and $I_0 = 161$, will be used for the later numerical simulations.

The deterministic and continuous SIR models provide a good understanding of the dynamics of influenza, but it will be further insightful to model the dynamics stochastically. Stochastic models will be more realistic since the spread of the virus is an intrinsically probabilistic process.

4 Continuous Time Markov Chain Model

The continuous time Markov chain model (CTMC) extends the continuous SIR model by including a stochastic process. The CTMC epidemic processes are defined on a continuous time scale and the states $S(t), I(t)$, and $R(t)$ are discrete random variables (discrete number of individuals in each state). Furthermore, since R can be rewritten in terms of S and I, this model is a bivariate process with independent random variables, S and I. The dynamics of the random variables S and I will be determined by transition probabilities of two events: infection and recovery. The graph of this cycle looks identical to the one for the deterministic counterpart with the addition that the transitions are determined probabilistically (Figure 5).

The two possible events at a given time t: a susceptible individual becomes infected $(S-1, I+1, R)$ or an infected individual recovers $(S, I-1, R+1)$. The sum of the these transitions equals one because these transitions represent all possible changes in the state during the time interval Δt. Also, because the CTMC model is a Markov process, the transition probability at time $t_n + 1$ only depends on the most recent time t_n. The transition probabilities are shown in Equation 5 [1]. These probabilities are derived from the terms in the corresponding deterministic model. For simplicity, $b = 0$ is used for the CTMC simulations.

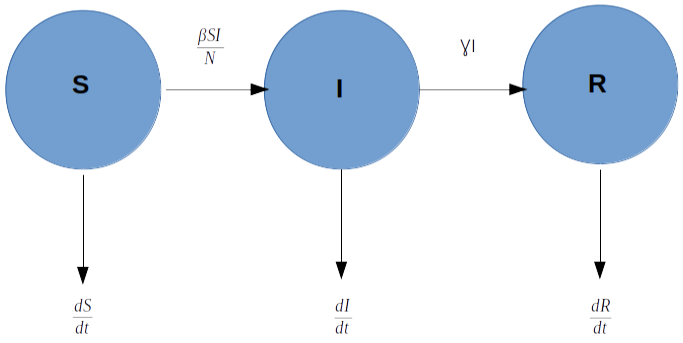

Figure 5: CTMC Visual Graph

$$p_{(S+k,I+j),(S,I)}(\Delta t) = \begin{cases} \beta\frac{SI}{N}\Delta t, (k,j) = (-1,1) \\ \gamma I\Delta t, (k,j) = (0,-1) \\ bI\Delta t, (k,j) = (1,-1) \\ b(N-S-I)\Delta t, (k,j) = (1,0) \\ 1 - \frac{\beta SI}{N}\Delta t - [\gamma I + b(N-S)]\Delta t, (k,j) = (0,0) \\ 0, otherwise \end{cases} \tag{5}$$

These transition probabilities can be applied to all of the possible ordered pairs of states at a given time to obtain a difference equation that can be used to predict future dynamics (at time $t + \Delta t$) that is Equation 6.

$$\begin{aligned} p_{(S,I)}(t+\Delta t) = p_{(S+1,I-1)}(t)\frac{\beta}{N}(I-1)(S+1)\Delta t + p_{(S,I+1)}(t)\gamma(I+1)\Delta t \\ + p_{(S-1,I+1)}(t)b(I+1)\Delta t + p_{(S-1,I)}(t)b(N-S+1-I)\Delta t \\ + p_{(S,I)}(t)(1 - [\frac{\beta}{N}SI + \gamma I + b(N-S)]\Delta t) \end{aligned} \tag{6}$$

Finally, a differential equation can be derived by subtracting $p(i(t))$ from both sides taking the limit as Δt goes to 0. This differential equation known as the forward Kolmogorov differential equation (Equation 7). There is also a backward Kolmogorov differential equation that can be used to estimate the probability of reaching a given state [4].

$$\begin{aligned} \frac{dp_{(S,I)}}{dt} = p_{(S+1,I-1)}(t)\frac{\beta}{N}(I-1)(S+1) + p_{(S,I+1)}(t)\gamma(I+1) \\ + p_{(S-1,I+1)}(t)b(I+1) + p_{(S-1,I)}(t)b(N-S+1-I) \\ - p_{(S,I)}(t)[\frac{\beta}{N}SI + \gamma I + b(N-S)] \end{aligned} \tag{7}$$

An important thing to note is that $I = 0$ is an absorbing state for this model [4]. This means that once the number of infectious individuals reaches 0, it continues to remain at 0 for the remaining future states, which results in a disease-free state. Logically, if there are no more infectious individuals left in a population, no additional individuals can be infected. States that are otherwise not absorbing are referred to as transient states.

4.1 Implementation

Like with other stochastic processes, it is much easier to numerically simulate stochastic processes rather than finding an analytical solution for the transition probabilities. The Gillespie algorithm will used to simulate the CTMC model, using uniform random numbers to simulate the change in state.

One random number, u_1 is used to determine interevent time (τ), which is the amount of time in between different events (the amount of time that the system remains unchanged at a given state) [1]. It will be assumed, due to it being Markov process, that the interevent time follows an exponential distribution so that $\tau = -\frac{lnu_1}{\lambda}$

The second random number, u_2 is used to determine which event occurs at time t, with the random numbers subdivided into intervals corresponding with the transition probabilities of each event (Equation 8).

$$\begin{aligned} p_{(S-1,I+1,R)} &= \frac{\frac{\beta SI}{N}}{\frac{\beta SI}{N} + \gamma I} \\ p_{(S,I-1,R+1)} &= \frac{\gamma I}{\frac{\beta SI}{N} + \gamma I} \end{aligned} \tag{8}$$

Using the same parameters as determined from the continuous SIR model best fit, three different simulations were calculated using the above equations and plotted against the continuous SIR model and CDC data (Figure 8).

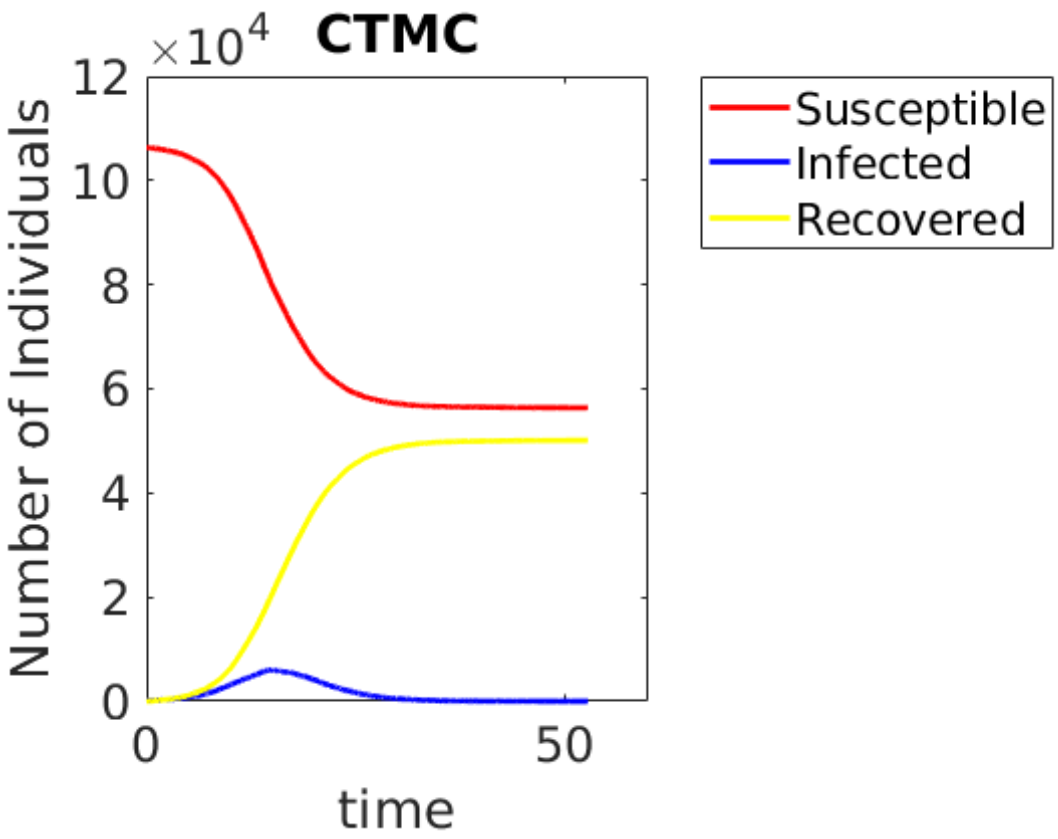

Figure 6: One Simulation of the CTMC Model

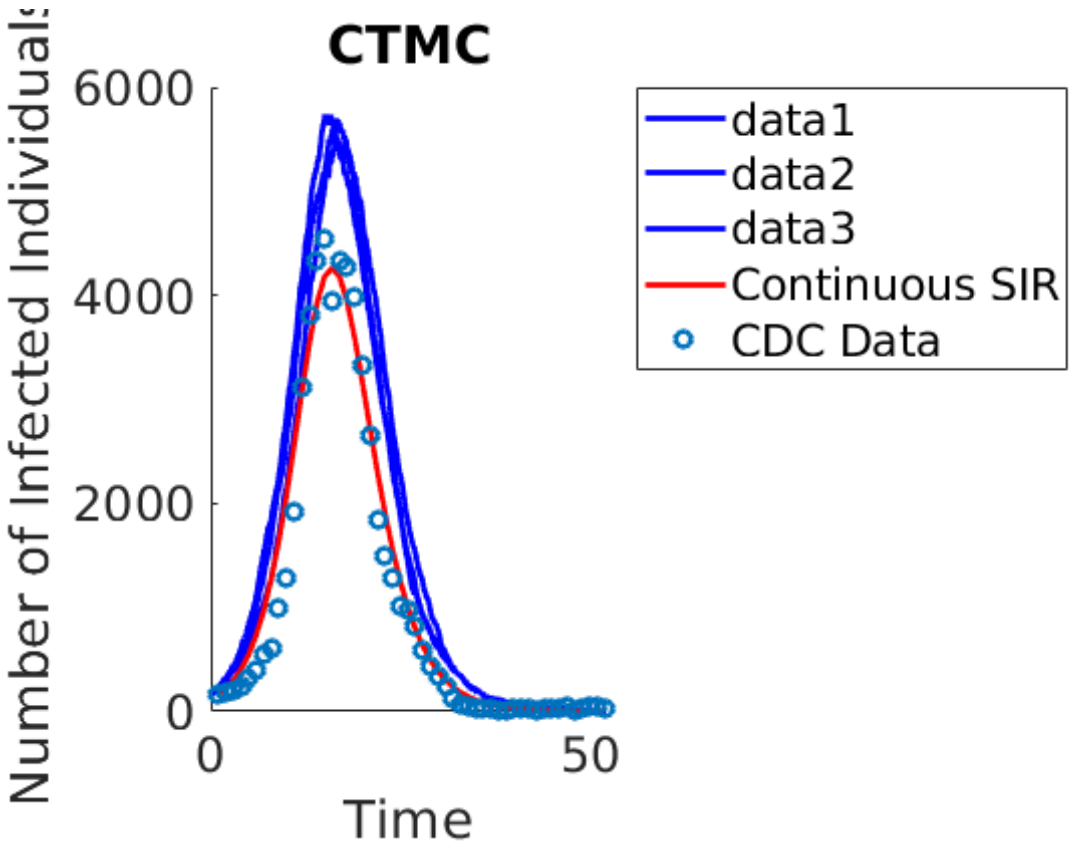

Figure 7: Three Different Simulations of the CTMC Model vs Continuous SIR Model (Infectious Individuals Only)

Comparing the CTMC model with the continuous SIR model, it is clear that both model the same general trends. However, it appears that the CTMC model consistently overestimates that peak number of infected individuals compared to the CDC data by about 2000 individuals. This may be due to the small interevent times, resulting in more events and thus more individuals infected at each time step. Based on this fit, the continuous SIR model is a better fit to the data. The sole advantage is that, unlike the deterministic model in which only one outcome is possible for a given set of parameters, the stochasticity captured in the CTMC model provides many possible simulation trajectories.

The possibility of different trajectories give rise to new possible calculations about the dynamics of the system. Because the CTMC model is a probabilistic model, it can be used to calculate the probability of an outbreak using the branching process approximation. Using this approximation, it is possible to estimate the probabilities of two possible scenarios, a minor outbreak, in which there is a slight increase in the number of infected individuals, and a major outbreak, in which there is a substantial increase in the number of cases (Equation 10) [1]. For these probabilities to be valid, it is assumed that each infected individual is independent from another individual with the same probabilities of infecting and recovering, and that there is a large susceptible population.

$$p_{minor} = \begin{cases} (\frac{1}{R_0})^I, R_0 > 1 \\ 1, R_0 < 1 \end{cases} \quad p_{major} = \begin{cases} 1 - (\frac{1}{R_0})^I, R_0 > 1 \\ 0, R_0 < 1 \end{cases} \tag{9}$$

Calculated previously, the $R_0 = 1.3620$ and so at the given initial conditions, the probability of a minor epidemic is $1.34x10^{-22}$ and the probability of a major epidemic is about 1. This fits with our understanding of the SIR model since an $R_0 > 1$ means that more individuals are being infected than recovering. For other parameter values, these probabilities will likely be less definite. These probabilities provide a more interpretable understanding than just a raw R_0 value, helping to predict how a given influenza virus will spread in a population under certain set initial conditions.

5 Stochastic Differential Equation Model

The final model studied in this paper is the stochastic difference equation model (SDE). This model is similar to the CTMC model except that the model follows a diffusion process in which the random variables are continuous instead of discrete. The SDE model uses the same transition probabilities as derived in the CTMC model and uses the probabilities to calculate the mean and covariance matrix, assuming that the random variables are independent and identically distribution for small enough delta t and approximate a normal distribution for large enough n (small enough time steps). This approximation is valid due to the central limit theorem [5].

The change in the random variables can be approximated by a system of what are known as Ito SDEs (Equation 11) [5]. In the Ito SDEs, B is the covariance matrix, which is determined using E, the expected values of the random variables Equation 10.

$$E = \begin{bmatrix} -\frac{\beta SI}{N} \\ \frac{\beta SI}{N} - \gamma I \end{bmatrix}$$
$$B = \begin{bmatrix} -\sqrt{\frac{\beta SI}{N}} & 0 \\ \sqrt{\frac{\beta SI}{N}} & -\sqrt{\gamma I} \end{bmatrix} \tag{10}$$

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta}{N} SI + B_{11}\frac{dW_1}{dt} + B_{12}\frac{dW_2}{dt} \\ \frac{dI}{dt} &= \frac{\beta}{N} SI - \gamma I + B_{21}\frac{dW_1}{dt} + B_{22}\frac{dW_2}{dt} \end{aligned} \tag{11}$$

Each of the differential equations has a term, W, that represents independent Wiener process. Independent Wiener processes, also called Brownian motion process, is a continuous-time stochastic process in which W is a random variable with a Gaussian distribution with zero mean and unit variance [7].

In an applied implementation, the Wiener process can be represented by a random number generated from a normal distribution. One can observe that removing the Wiener process terms returns the same ODEs in the original continuous SIR model. Essentially, these added Wiener processes add the element of stochasticity into the SIR model.

5.1 Implementation

Simulating SDEs can be done using the Euler-Maruyama method, which uses a finite-difference approximation (equation 12).

$$X(t + \Delta t) = X(t) + f(X(t), t)\Delta t + G(X(t), t)\eta\sqrt{\Delta} \tag{12}$$

Each of the Weiner processes is approximated independently using the standard normal random number generator and used in each stochastic differential equation. Especially for large population sizes, SDE numerical simulation computations perform much faster than for CTMC models since it easier to solve numerically [1]. For instance, the interevent time is not required to be calculated and instead, a fixed length time step is chosen.

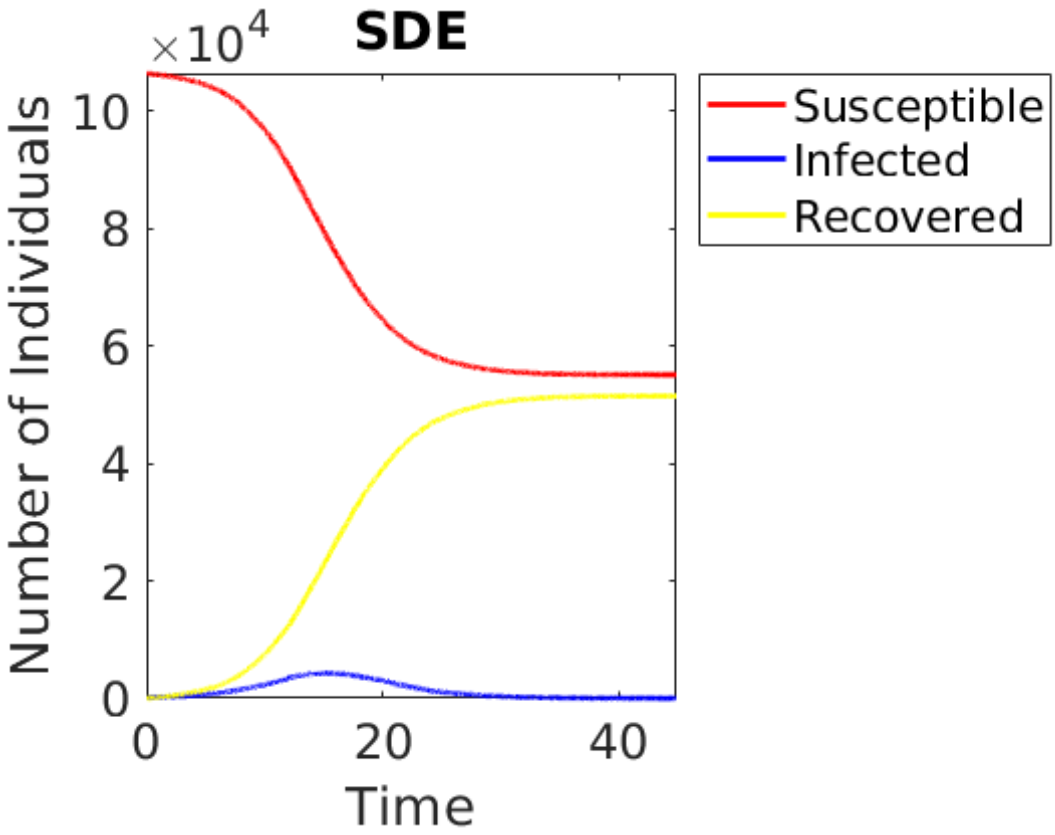

Figure 8: One Simulation of the SDE Model

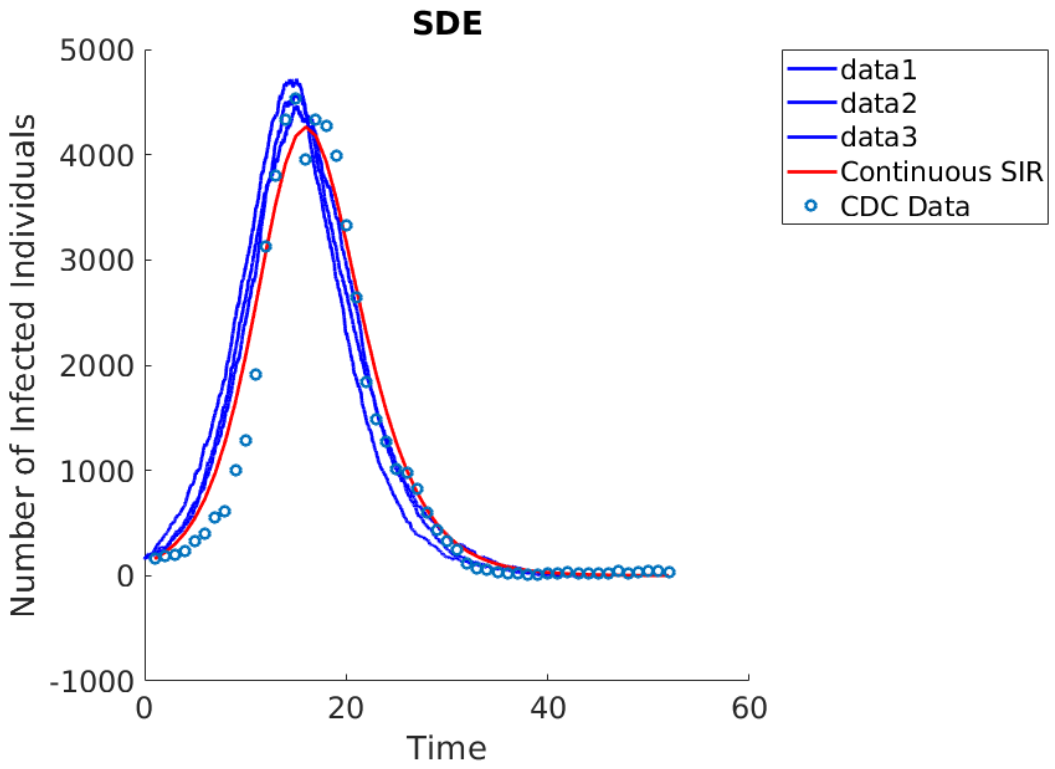

Figure 9: Three Different Simulations of the SDE Model vs Continuous SIR Model (Infectious Individuals Only)

Comparing the SDE model to the continuous SIR model, it appears that the SDE simulations fit the SIR model and data very well, more so than the CTMC model. The different simulations slightly overestimate the peak, but not by a significant margin, and matches the continuous SIR model otherwise. And again, like with the CTMC model, the disease-free equilibrium is the absorbing state. So, For other parameter values, while the deterministic model may reach an endemic equilibrium, the SDE model will still approach the disease-free one.

In regards to computational efficiency and fit, it seems to outperform the CTMC model. However, the CTMC model offers an advantage in allowing more interprability of the epidemic process, such as allowing us to approximate the probability of a minor or major epidemic occurring near a disease-free equilibrium.

6 Discussion

6.1 Control of Influenza

As mentioned in a previous section, there are several somewhat effective methods to help control the rate of infection and spread of the influenza virus: vaccination, public health

education/quarantining, and antiviral medication. In terms of the mathematical model, each of these methods affects a specific parameter in the model:

1. Flu vaccine: Decreases S_0 by moving S_0 to R_0.
2. Quarantine/Public Health Education: Decreases the contact rate for susceptible individuals and thus decreases β.
3. Antiviral (Oseltamivir/Tamiflu): Increases the rate of recovery for infected individuals by shortening the time with symptoms (decreasing the period of infectivity) and thus increases γ.

Below are plots comparing the effect of each control method at a 5% rate (only plotting the infected individuals) versus their control (Figures 8, 9, and 10). Comparing the three different models, it is clear that each control method has a similar impact on reducing the number of infected individuals in the population, which the vaccination control being slightly more effective for the continuous SIR and CTMC models. There are pros and cons for each control method in terms of practical implementation. Vaccinations, for instance, are expensive to manufacture and distribute to every individual in a large population, whereas reducing the number of contacts is the most cost-effective alternative. On the other hand, vaccinations typically have a high rate of success in preventing influenza while reducing the number of contacts can only be accomplished succesfully to a certain extent. Tamiflu, the third option, is the most expensive option and can only be used if the flu is properly diagnosed within 2 days of showing symptoms. One would need to obtain a flu swab from a medical clinic and within a limited time window, both non-trivial tasks to fulfill and tasks that most individuals probably would not accomplish.

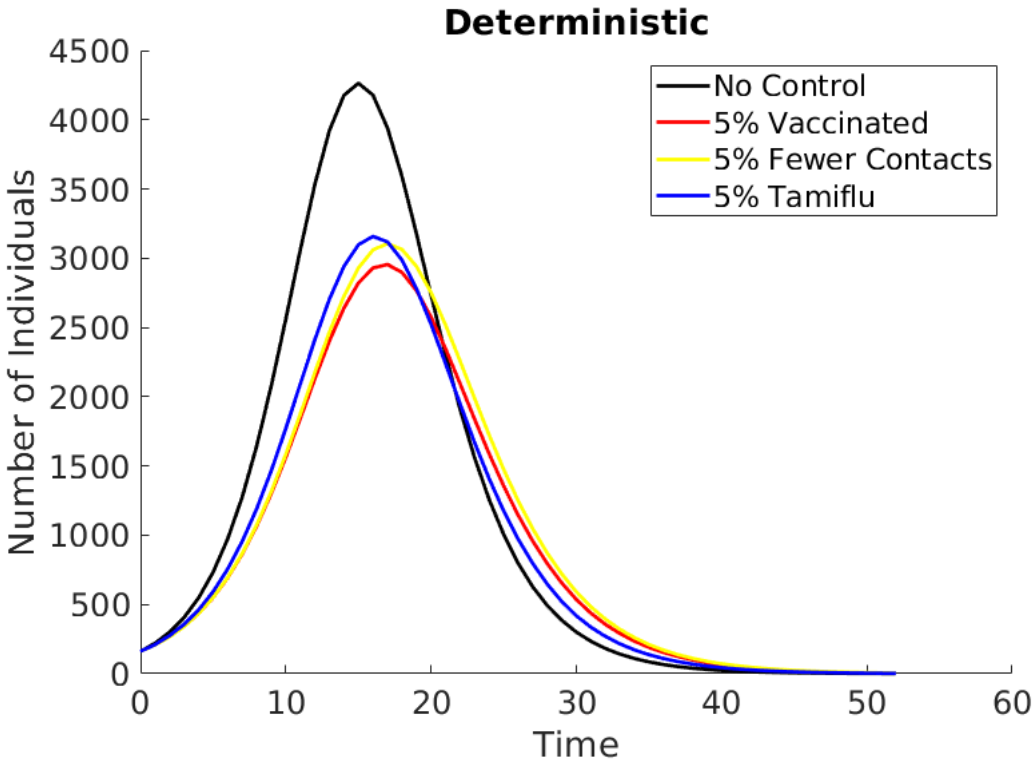

Figure 10: Continuous SIR Model

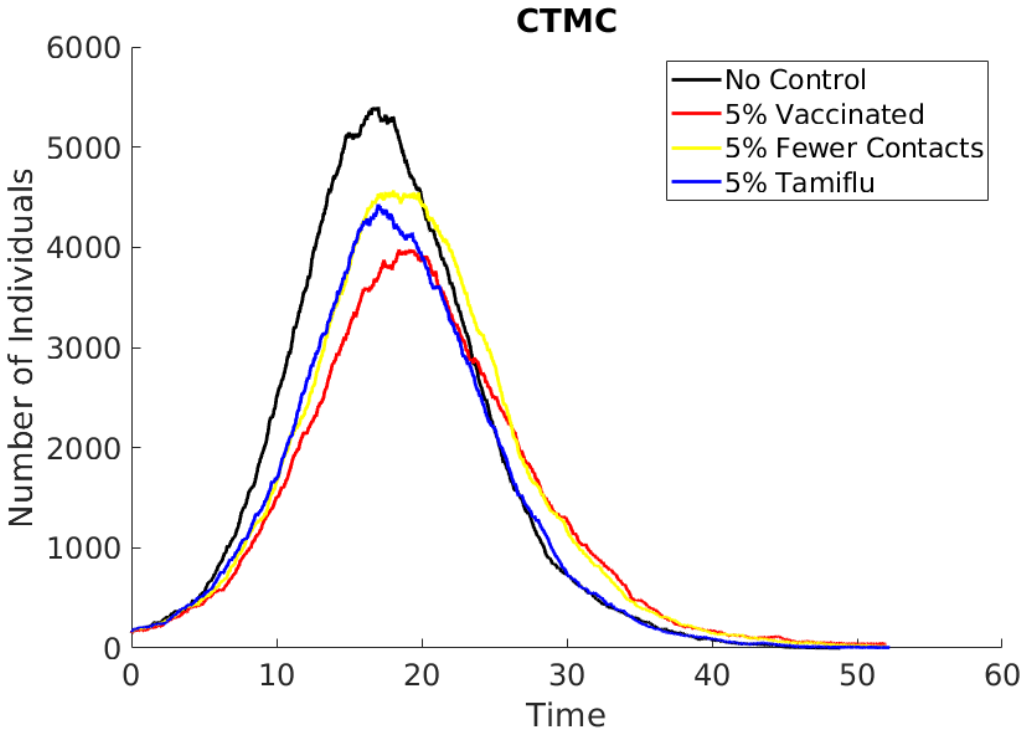

Figure 11: CTMC Model

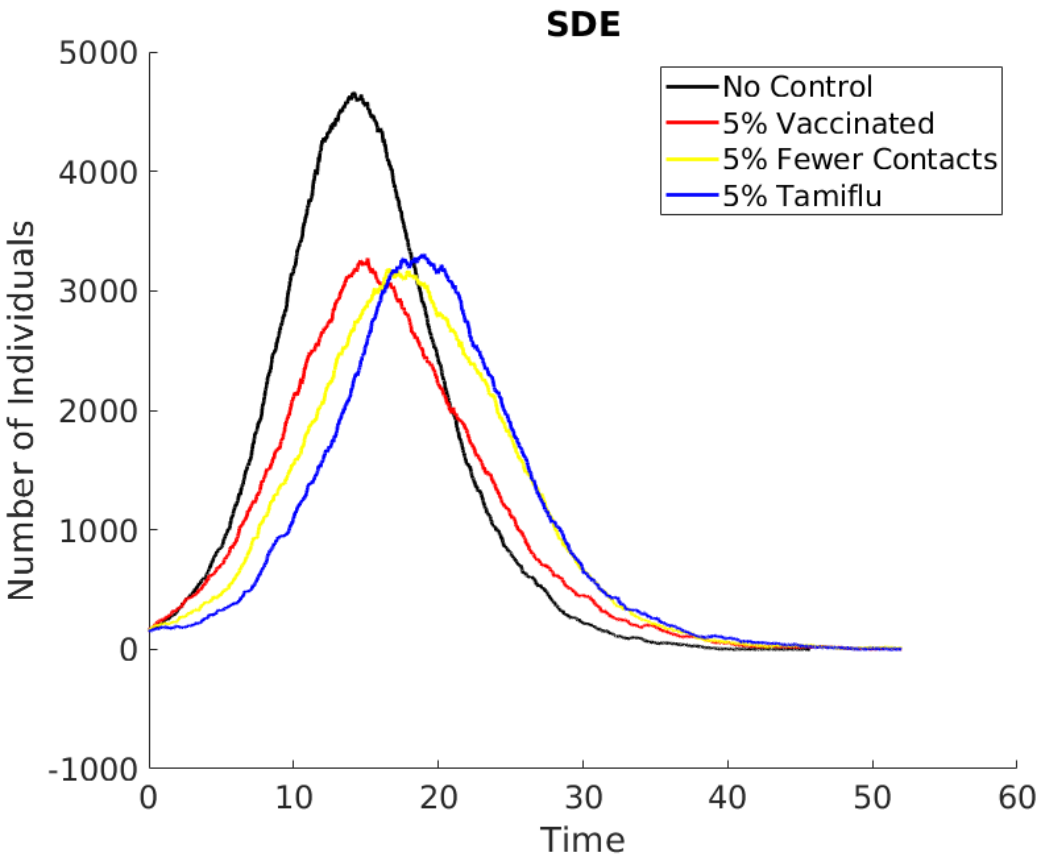

Figure 12: SDE Model

6.2 Evaluation

The deterministic and the stochastic models each capture important information about the dynamics of influenza. Each model, as shown in the previous sections, fits the real data well and captures the important trends. The continuous SIR model is arguably the simplest model to implement, but does not take into account chance like the stochastic models do. Taking into account probabilities of infection and recovery model real-world influenza behavior much closer and so the stochastic models are superior in that aspect, able to yield different simulation trajectories. Even so, the stochastic models do not deviate far from the deterministic model. Comparing the two stochastic models, the SDE model outperforms the CTMC model in terms of fit and calculation efficiency.

Besides having variable trajectories, another important difference between the deterministic and stochastic epidemic models is their asymptotic dynamics. Eventually stochastic solutions (sample paths) converge to the disease-free state even though the corresponding deterministic solution converges to an endemic equilibrium. Another property that is unique to the stochastic epidemic models include the probability of an outbreak. Additionally, other characteristics can be calculated from the stochastic models including the quasistationary

probability distribution, the final size distribution of an epidemic, and the expected duration of an epidemic [1].

6.3 Further Analysis

Although it is beyond the scope of this paper, it would be interesting to see how varying the birth rate will influence each model. The Matlab code for each of the given models is included in the appendix and can take an input for the rate of birth, b. Changing the value of b can increase the number of susceptible individuals at any given time, which can increase the number of infected individuals over a given flu season. Analysis of the dynamics of influenza focusing on the effect of having a non constant population, which is more realistic, can provide insightful results.

There are also other alternatives to the SIR model such as the SEIR model and SEIRS model, which includes an exposed population in addition to the susceptible, infected, and recovered populations. It may be interesting to see how these models compare to the SIR models and to implement a stochastic version of these models.

7 Conclusion

These different methods for modeling the dynamics of influenza, the deterministic SIR models, the CTMC model, and the SDE models, all have certain strengths and weaknesses. One thing they all have in common is that all of the models fit real-world data well. The deterministic SIR models are simpler to implement, but can yield endemic equilibria instead of disease-free equilibria. The stochastic models incorporate probability, which captures the disease process more realistically. It can be more difficult to analyze, but approximations, such as the branching process approximation, can be used to provide insight.

The impact of vaccination, reducing contact rate, and using Tamiflu, can be seen in each model. All of the control methods yield a similar effect on reducing the number of infected individuals in a population. From a public health standpoint, while each method has its pros and cons, it will be important to encourage the use of all three methods to best control the spread of influenza.

Beyond just influenza, these deterministic and stochastic SIR models can be extended to simulate the dynamics of other infectious disease and determine probabilities for epidemics. For example, one can study the dynamics of malaria or even Ebola. For diseases that fit within the regime of the SIR process, the SIR model can be adjusted to model the dynamics well. The difficult part of simulating the dynamics is to first determine the governing equations and the transition probabilities that are derived from those equations.

Mathematical modeling helps us understand the dynamics of influenza on a deeper level, but that does not mean that there is not more left to be done to improve the accuracy of modeling techniques. More importantly, it is vital to focus on figuring out more effective

methods for preventing influenza. There does not seem to be a long-term solution to the threat of influenza on a global health scale and there is a persisting concern of a pandemic. Although there are no immediate cures or treatment for the flu, public health entities can continue to promote vaccination to decrease the rate of infection and spread of disease. While it may be more cost effective to shift the focus to public health education, vaccination is not a significant cost and can have a much stronger effect, but only if the predicted formula accurately covers individuals against the specific virus for that season.

Until a better solution emerges, it seems that influenza will continue to be an active concern each winter. So in the meantime, let us be diligent in washing our hands frequently and encouraging others to vaccinate.

Note: Matlab code is included in the appendix of this paper for each of the mathematical models discussed.

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