A Look Into Analytical and Stochastic SEIR Modelling

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Abstract

The object of this paper is to analytically and stochastically model the spread of a disease for the endemic and epidemic cases based on the classic SEIR model. We will examine the location and stability of fixed points based on variations in the parameter R_0 with phase plane analysis and numerical simulations. We will also analyze the effect of vaccinations for the stochastic models for both epidemics and endemics. We find that values of $R_0 > 1$ mean that the disease will never be eradicated. We also find that vaccinations significantly reduce the exposed and infected population sizes at steady state, highlighting the importance of vaccines in lowering disease rates.

1. Introduction

Infectious diseases have been a major cause of suffering and distress in humanity's history. For instance, Hethcote in [6] asserts that Europe suffered enormous social, economic, and religious impacts during the 14th century as a result of the black plague. The population suffered a 25% decrease due to the disease. The author also mentions the 1960s confidence boosts that arose due to vaccination efforts, eventually leading up to the focus on other illnesses, such as chronic diseases like cancer and cardiovascular diseases. However, from 1960 to 1990, multiple infectious and previously unknown diseases appeared, in which he concludes that the battle against epidemics is far from being settled. A clear example of this is the Covid-19 pandemic, an unprecedented health issue that has plagued the world with economic, social, and health problems.

One way to approach the issue is by utilizing mathematical models to determine the behavior of an infection under a certain number of assumptions. Models serve as useful tools to understand the consequences of such infectious diseases and examine the effectiveness of public health strategies and the

personal measures we can apply. For instance, Adiga et al. [1] and Carcione et al. [5] examine various key simplifications made to analyze the spread of Covid-19 in a different set of scenarios and successfully evaluate the effectiveness of vaccination and quarantine to methods contain the disease.

The exact dynamics of a disease hold immense complexity. All of the factors englobed by society can drastically influence the outcome of an epidemic and should be accounted for. Theoretically, an ideal model would include the different genetics of every single individual, exact weather dynamics, the exact geography of the earth, and many other specific factors. However, the butterfly effect makes it an impossible task for human computation. Through certain assumptions, we can model simplified scenarios which still provide valuable insight into the real world dynamics of disease spread. Tom Britton argues that the benefits of the simplified methods are that the connections between basic quantities become clearer, forcing emphasis on which quantities have the most influence on predictions, with the added conclusion that uncertainties in these numbers transfer over to high uncertainty in forecasts [3]. As such, our emphasis will be on simplicity and transparency in our models, with the aim of providing an intuitive idea of the role of each part of the model.

As Fred Brauer discusses in [2], the first works on mathematical epidemiology date back to the 16th century, in which the mathematician Daniel Bernoulli calculated variations in life expectancy with respect to variolation in smallpox (inoculation with a mild strain of the virus). However, it wasn't until the 1870s that compartmental models were introduced, which quickly became the standard for studying the spread of infectious diseases. The field was first developed not by mathematicians, but by health physicians. One of the most illustrative and pioneering works on compartmental models was given by Sir R.A. Ross, a physician that studied malaria on mosquitoes and introduced the concept of the basic reproduction number, a threshold to determine the severity of the spread of the disease. This threshold is the dividing line between the infection dying out and the onset of an epidemic [2]. Moreover, in the late twenties and early thirties, Kermack and McKendrick published a series of three papers containing the basic compartmental models describing the transmission of communicable diseases. These include the well-known SIR and SEIR models, and we will focus on analyzing the latter. Since Kermack and McKendrick's last paper of 1933 [8], there has been a great deal of work on compartmental disease transmission models, with generalizations in many directions, but the main idea of compartmental models remains the same.

This paper will cover two versions of the SEIR model and explore their differences in contrasting scenarios. We will examine a continuous SEIR model, calculate the basic reproduction number to dif-

ferentiate an endemic from a pandemic, and finally, study the stochastic version of the model including additional parameters such as vaccination rates and mutations.

2. Methods

2.1. Analytical SEIR Model

The main method used for the analysis of the classic SEIR model for endemics is phase plane analysis to determine the stability of the equilibria. The theoretical results will be contrasted with numerical simulations using MATLAB's `ode45` method. Different parameters will be used to survey a variety of different scenarios. The values are picked in accordance with the reproduction number R_0, which determines the long-term behavior of the solutions.

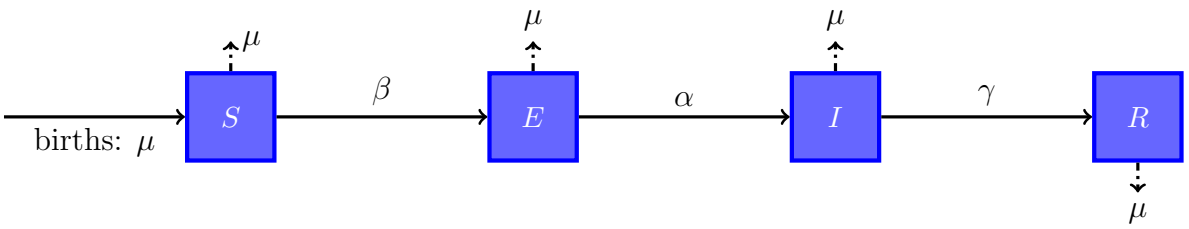


Figure 1: Endemic SEIR Compartmental Diagram

The compartmental model examined stands for **S**usceptible → **E**xposed → **I**nfected → **R**ecovered. The transition from one state to the other will depend on a set of different rates, which are described below and graphically depicted in Fig. 1. For a matter of notational convention, we will use the same symbols as Brown and Hethcote in [4, 6].

- β: Represents the infection rate, i.e. how quickly the virus spreads through the population.
- α: Represents the latency rate i.e. how long it takes an exposed individual to be infectious. The period is α^{-1}.
- γ: Recovery rate of an individual.
- μ: Birth and death rate due to natural causes. Note that this is a simplified model since birth and death rates are not typically equivalent.

2.1.1. Analysis of Equlibria

Let us first analyze the stability of the disease-free fixed point. In the case of being stable, a small population of infectious individuals will not be enough to trigger a widespread infection and the solution will go back to a healthy, disease-free equilibrium.

$$\frac{dS}{dt} = \mu N - \beta(S \cdot I) - \mu S \tag{1}$$
$$\frac{dE}{dt} = \beta S \cdot I - (\mu + \gamma)E \tag{2}$$
$$\frac{dI}{dt} = \gamma E - (\mu + \alpha)I \tag{3}$$
$$\frac{dR}{dt} = \alpha I - \mu R \tag{4}$$

By inspection of the above model equations obtained from Martcheva in [9], we see that in the initial case where all the population is susceptible, and no one has the virus, we have equilibria. Clearly, we need at least one infectious individual to start propagating the disease. As we assume that the total population N remains constant, we'll denote the disease-free equilibrium as $e^* = (N, 0, 0, 0)$. Solving for the other fixed point, we equal the derivatives to 0, so

$$0 = \mu N - \beta(S \cdot I) - \mu S \tag{5a}$$
$$0 = \beta S \cdot I - (\mu + \gamma)E \tag{5b}$$
$$0 = \gamma E - (\mu + \alpha)I \tag{5c}$$
$$0 = \alpha I - \mu R \tag{5d}$$

From (5c), we see $E = \frac{\mu+\alpha}{\gamma} I$, which by substituting into (5b) yields:

$$S = \frac{(\mu + \gamma)(\mu + \alpha)}{\beta\gamma}$$

Plugging this result into (5a) as S, we have

$$I^* = \frac{\mu(N - S)}{\beta S} = \frac{\mu}{\beta}\left(\frac{N - S}{S}\right) = \frac{\mu}{\beta}\left(\frac{N}{S} - 1\right) = \frac{\mu}{\beta}\left(\frac{N\beta\gamma}{(\mu + \alpha)(\mu + \gamma)} - 1\right) \tag{6}$$

For simplicity, we define $R_0 = \frac{N\beta\gamma}{(\mu+\alpha)(\mu+\gamma)}$ to be the reproduction number, and with this notation the

algebra simplifies.

Now, we solve for R^* in (5d) and E^* in (5b) and obtain:

$$R^* = \frac{\alpha}{\mu} I = \frac{\alpha}{\beta}(R_0 - 1) \qquad \text{and} \qquad E^* = \frac{\mu + \alpha}{\gamma}\frac{\mu}{\beta}(R_0 - 1) \tag{7}$$

Let us now analyze the stability of the disease-free and endemic equilibrium through phase plane analysis. As discussed by Martcheva in [9], the characteristic polynomial is too complicated to explicitly solve for the conditions needed for stability, and thus a different approach is taken.

1. **Disease-free equilibrium** $p_1 = (N, 0, 0, 0)$. Considering the Jacobian, we see

$$J = \begin{pmatrix} -\beta I - \mu & 0 & -\beta S & 0 \\ \beta I & -(\mu+\gamma) & \beta S & 0 \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix} \qquad J(N,0,0,0) = \begin{pmatrix} -\mu & 0 & -\beta N & 0 \\ 0 & -(\mu+\gamma) & \beta N & 0 \\ 0 & \gamma & -(\mu+\alpha) & 0 \\ 0 & 0 & \alpha & -\mu \end{pmatrix}$$

We now look for $\lambda \in \mathbb{C}$ such that $\det(J - \lambda I) = 0$. To simplify the determinant computation we notice the matrix is close to sparse and therefore use the method of minors to simplify the calculation:

$$\begin{vmatrix} -\mu-\lambda & 0 & -\beta N & 0 \\ 0 & -(\mu+\gamma)-\lambda & \beta N & 0 \\ 0 & \gamma & -(\mu+\alpha)-\lambda & 0 \\ 0 & 0 & \alpha & -\mu-\lambda \end{vmatrix} = -(\mu+\lambda) \begin{vmatrix} -(\mu+\gamma)-\lambda & \beta N & 0 \\ \gamma & -(\mu+\alpha)-\lambda & 0 \\ 0 & \alpha & -\mu-\lambda \end{vmatrix} \tag{8}$$

$$= (\mu+\lambda)^2 \begin{vmatrix} -(\mu+\gamma)-\lambda & \beta N \\ \gamma & -(\mu+\alpha)-\lambda \end{vmatrix} = 0 \tag{9}$$

Thus, it is clear that $\lambda = -\mu$ and has an algebraic multiplicity of two. Next we determine the sign of the other eigenvalues. To do so, we look at the trace and determinant of

$$A = \begin{pmatrix} -(\mu+\gamma) & \beta N \\ \gamma & -(\mu+\alpha) \end{pmatrix}$$

Hence $\operatorname{tr} A = -(2\mu + \gamma + \alpha)$ and $\det A = (\mu + \alpha)(\mu + \gamma) - \beta\gamma N$. For negative eigenvalues and thus stability, we require $\det A > 0$ and $\operatorname{tr} A < 0$, which means that

$$\frac{\det A}{\operatorname{tr} A} = \frac{(\mu + \alpha)(\mu + \gamma) - \beta\gamma N}{-(2\mu + \gamma + \alpha)} < 0 \tag{10}$$

Note that this could also arise from $\det A < 0$ and $\operatorname{tr} A > 0$, but this case is covered as the trace is always negative. As all parameters are positive, we must have $(\mu + \alpha)(\mu + \gamma) - \beta\gamma N > 0 \implies \beta\gamma N < (\mu + \alpha)(\mu + \gamma)$ or

$$R_0 = \frac{\gamma\beta N}{(\mu + \alpha)(\mu + \gamma)} < 1$$

.

Thus, if the reproduction number is below 1, we get negative eigenvalues and the disease-free equilibrium is stable, which means that a small initial infected population wouldn't be a major issue. In the case where $R_0 = 1$, we see that $\det A = 0$, and thus our eigenvalues are 0, which implies the system is non-hyperbolic, as explained in [9], and it is hard to draw conclusions from a simple linearization. Finally, with $R_0 > 1$, we get positive valued real parts for our eigenvectors and the disease-free equilibrium is unstable, so the disease would turn into an endemic, which brings us to the other fixed point.

2. **Endemic equilibrium**. After having derived the fixed point, we rewrite it to simplify the notation with R_0. Note that all values are positive as reaching this point would imply $R_0 > 1$, so $R_0 - 1 > 0$.

$$p_2 = (S^*, E^*, I^*, R^*) = \left(\frac{(\mu + \gamma)(\mu + \alpha)}{\beta\gamma},\ \frac{\mu + \alpha}{\gamma}\frac{\mu}{\beta}(R_0 - 1),\ \frac{\mu}{\beta}(R_0 - 1),\ \frac{\alpha}{\beta}(R_0 - 1) \right) \tag{11}$$

At this point we see:

$$J(p_2) - \lambda I = \begin{pmatrix} -\beta I^* - \mu - \lambda & 0 & -\beta S^* & 0 \\ \beta I^* & -(\mu + \gamma) - \lambda & \beta S^* & 0 \\ 0 & \gamma & -(\mu + \alpha) - \lambda & 0 \\ 0 & 0 & \alpha & -\mu - \lambda \end{pmatrix} \tag{12}$$

It is clear that from the last column, the value $\lambda = -\mu$ yields a singular matrix, so we are left with:

$$\begin{vmatrix} -\beta I^* - \mu - \lambda & 0 & -\beta S^* \\ \beta I^* & -(\mu+\gamma) - \lambda & \beta S^* \\ 0 & \gamma & -(\mu+\alpha) - \lambda \end{vmatrix} = (\beta I^* + \mu + \lambda)\big(\gamma\beta S^* - (\mu+\gamma+\lambda)(\mu+\alpha+\lambda)\big) - \beta S^*\big(\beta I^*\gamma\big) = \tag{13}$$

Thus, the remaining roots satisfy the following equation:

$$(\beta I^* + \mu + \lambda)\big(\gamma\beta S^* - (\mu+\gamma+\lambda)(\mu+\alpha+\lambda)\big) = \beta S^*\big(\beta I^*\gamma\big) \tag{14}$$

With basic algebra, we simplify:

$$(\mu+\lambda)\big(\gamma\beta S^* - (\mu+\gamma+\lambda)(\mu+\alpha+\lambda)\big) = (\beta I^*)(\mu+\gamma+\lambda)(\mu+\alpha+\lambda) \tag{15}$$

$$\big(\mu+\lambda\big)(\mu+\gamma)(\mu+\alpha) = [\mu+\gamma+\lambda][\mu+\alpha+\lambda]\big[\beta I^* + \mu + \lambda\big] \tag{16}$$

To prove stability, we assume $\mathrm{Re}(\lambda) \geq 0$ has a solution to the previous equation and derive a contradiction. Thus, assume $\mathrm{Re}(\lambda) \geq 0$. As $\lambda \neq -\mu$, we divide (5) by $(\mu+\lambda)$, grouping all λ on the right side and obtain:

$$0 < (\mu+\gamma)(\mu+\alpha) = \frac{|\mu+\gamma+\lambda||\mu+\alpha+\lambda|\big|\beta I^* + \mu + \lambda\big|}{|\mu+\lambda|} \tag{17}$$

We take the absolute value of the expression and drop the bars on the left side as all the parameters are positive and real-valued. Here, we note that the complex absolute value is the 2-norm of the vector $\mathbf{x} \in \mathbb{R}^2$ with $\mathbf{x} = (x, y)$ associated with the complex number $x + yi$. Letting $\lambda = a + bi$ we see $\beta I^* > 0$, and thus $|\beta I^* + \mu + \lambda| = \sqrt{(\beta I^* + \mu + a)^2 + b^2}$ and $|\mu + \lambda| = \sqrt{(\mu+a)^2 + b^2}$. By assumption, $\mathrm{Re}(\lambda) = a \geq 0$ so $\sqrt{(\beta I^* + \mu + a)^2 + b^2} > \sqrt{(\mu+a)^2 + b^2}$, and thus $|\beta I^* + \mu + \lambda| > |\mu + \lambda|$, or $\frac{|\beta I^* + \mu + \lambda|}{|\mu+\lambda|} > 1$. With this result in hand, the contradiction follows directly:

$$\frac{|\mu+\gamma+\lambda||\mu+\alpha+\lambda|\big|\beta I^* + \mu + \lambda\big|}{|\mu+\lambda|} > |\mu+\gamma+\lambda||\mu+\alpha+\lambda| \geq (\mu+\gamma)(\mu+\alpha). \tag{18}$$

This means that the right-hand side of (17) is always greater than the left-hand side when $\mathrm{Re}(\lambda) \geq 0$, so there is no solution to the equation and we have a contradiction. It is then safe to conclude that

the eigenvalues must have a negative real part, which implies stability of the endemic equilibrium. Note that the above trick usually isn't possible for complex systems, and a more general approach is suggested, such as the use of the Routh-Hurwitz Criterion, which is explained by Martcheva in [9].

We thus conclude by saying that $R_0 < 1$ yields a stable, disease-free equilibrium, whereas $R_0 > 1$ implies the disease will spread and the long-term behavior of the system will tend towards the stable endemic equilibrium.

2.1.2. Numerical Simulations

We survey three distinct scenarios which give rise to drastically different long-term solutions. The chosen initial population is $N = 500$ and the parameters for the models are:

1. $\beta = 0.0075$, $\alpha = \frac{1}{8}$, $\mu = 1/1000$, $\gamma = \frac{1}{2}$, $(S, E, I, R)_{t=0} = (450, 0, 50, 0)$.
2. $\beta = 0.002$, $\alpha = \frac{1}{2}$, $\mu = \frac{1}{10}$, $\gamma = \frac{1}{8}$, $R_0 = 0.93$, $(S, E, I, R)_{t=0} = (450, 0, 50, 0)$.
3. $\beta = 0.0045$, $\alpha = \frac{2}{5}$, $\mu = \frac{1}{10}$, $\gamma = \frac{1}{25}$, $R_0 = 0.89$, $(S, E, I, R)_{t=0} = (450, 0, 50, 0)$.

2.2. Stochastic Methods to Solve Epidemic SEIR-V

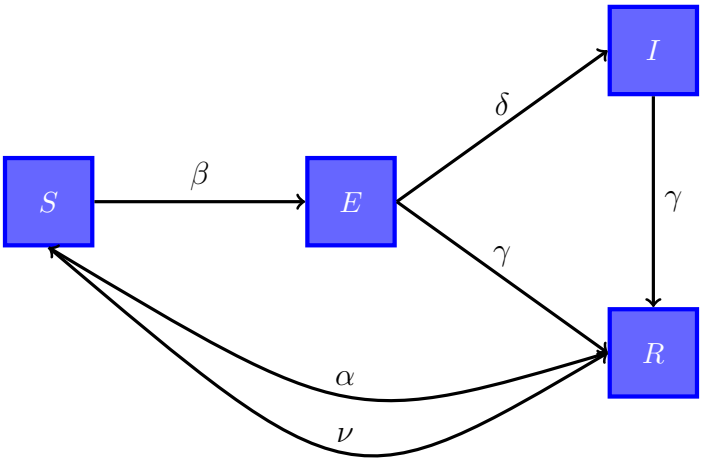


Figure 2: SEIR-V Epidemic Compartmental Diagram

Now we solve the SEIR-V model with stochastic methods, specifically Markov Chains following the method used by Kamboj in [7] for the simple SIR-V case for this higher order model. In order to perform

this analysis we begin with the simplifying assumption that there are no births or deaths (i.e. this is an epidemic case, not an endemic case). We will, however, examine the effect of vaccinations on the overall model dynamics in this case. To simplify the model slightly, we will assume that only the susceptible class is able to get vaccinated, so individuals do not leave the exposed or infected classes due to vaccinations. We use Fig. 2 to make the following observations to derive the transition matrix and equations for this model (note that the variables and notation used are not the same as in previous sections, and will be redefined for the stochastic methods as indicated later in this section):

- Most individuals, with the exception of those who are vaccinated or recovered (i.e. they have recently been exposed or infected and therefore have temporary immunity), are part of the susceptible class. A susceptible individual can become exposed or get vaccinated and leave the susceptible class. However, due to mutations in the virus, a certain percentage of the population may go from the recovered class back to the susceptible class.

$$S_{n+1} = S_n + \alpha R_n - \beta S_n - \nu S_n \tag{19}$$

- An individual can go from being susceptible to being exposed and enter the exposed class. They may also leave the exposed class by becoming infected or recovering. Exposed individuals are contagious but not infected.

$$E_{n+1} = E_n + \beta S_n - \delta E_n - \gamma E_n \tag{20}$$

- An individual can enter the infected class by going from being exposed to infected. Infected individuals may also recover and leave the class.

$$I_{n+1} = I_n + \delta E_n - \gamma I_n \tag{21}$$

- Recovered individuals have temporary immunity from being exposed or infected. An individual can join the recovered class by going from exposed directly to recovered, infected to recovered, or by getting vaccinated. Individuals in this class can leave this class due to mutations in the virus and become susceptible again.

$$R_{n+1} = R_n + \gamma E_n + \gamma I_n + \nu S_n - \alpha R_n \tag{22}$$

Putting the equations derived above into matrix notation gives:

$$\vec{x}_n = \begin{bmatrix} 1-\nu-\beta & 0 & 0 & \alpha \\ \beta & 1-\delta-\gamma & 0 & 0 \\ 0 & \delta & 1-\gamma & 0 \\ \nu & \gamma & \gamma & 1-\alpha \end{bmatrix} \begin{bmatrix} S \\ E \\ I \\ R \end{bmatrix} \tag{23}$$

Here,

- $\vec{x}_n$ is a 4x1 column vector containing the the percentage of the total population that is susceptible, exposed, infected, or recovered respectively on day n from day 0.
- α represents the percentage of people who become susceptible after recovery per day due to mutations in the virus.
- β represents the percentage of people who become exposed to the virus per day.
- δ represents the percentage of people who get infected per day.
- γ represents the percentage of people who recover after being infected or exposed per day.
- ν represents the daily percentage rate of people getting vaccinated.
- S, E, I, and R represent the percentage of the total population that is susceptible, exposed, infected, or recovered respectively on day n-1 from day 0. This column vector can alternatively be defined as $\vec{x}_{n-1}$.

This system can be solved iteratively given the initial conditions of the S,E,I,R vector on day 0 and be used to analyze the progression of the virus per day based on different initial condition. The 4×4 matrix above is our transition matrix. Since each column of this matrix is a proportion of the whole, we can verify the results of the derivation by ensuring that all columns individually sum up to one.

2.3. Stochastic Methods to Solve Endemic SEIR-V

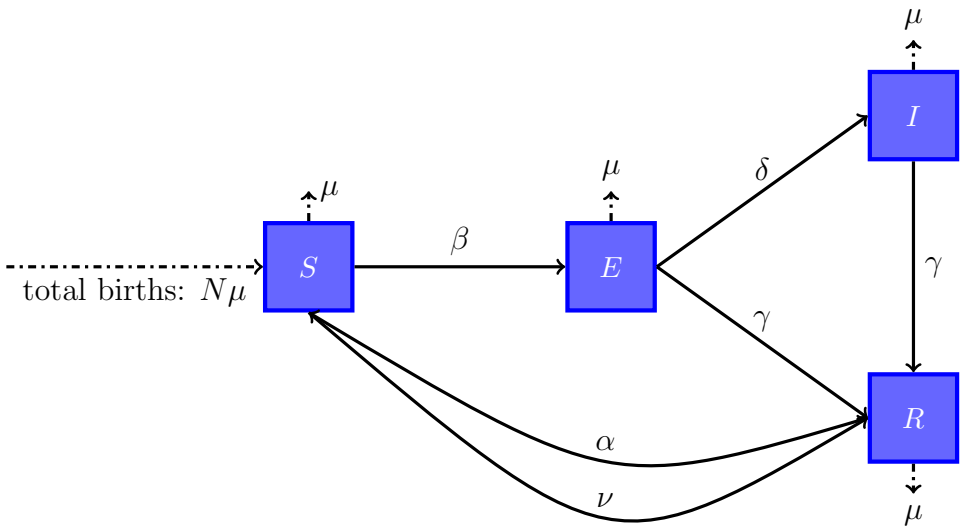


Figure 3: SEIR-V Endemic Compartmental Diagram

For the expanded endemic case, we use the epidemic model as a base, but now introduce a new variable, μ, which represents the daily birth and death rate as depicted in Fig. 3. Note that in this case, since the rate of births equals the rate of deaths, the total population remains the same, but the new input of susceptible individuals due to births ensures that the susceptible class does not decrease to zero over time, as observed in the epidemic case. We assume that all classes give birth and the newborns join the susceptible class. We also assume each class loses members of its class due to the death rate.

Here,

$$\vec{x}_n = \begin{bmatrix} 1-\nu-\beta & \mu & \mu & \alpha+\mu \\ \beta & 1-\delta-\gamma-\mu & 0 & 0 \\ 0 & \delta & 1-\gamma-\mu & 0 \\ \nu & \gamma & \gamma & 1-\alpha-\mu \end{bmatrix} \begin{bmatrix} S \\ E \\ I \\ R \end{bmatrix} \tag{24}$$

Note that the first term in the transition matrix is actually $1-\nu-\beta+\mu-\mu$ in order to account for births and deaths in the susceptible class. Since the birth and death rate is the same, the effects of births and deaths within the susceptible class cancel out. However, the susceptible class is still getting new additions due to the contributions of the births from the exposed, infected, and recovered classes.

3. Results

3.1. Continuous, Endemic SEIR Results

3.1.1. Phase Plane Analysis

The eigenvalue computations for the Jacobian evaluated at both fixed points gave the same kind of insight.

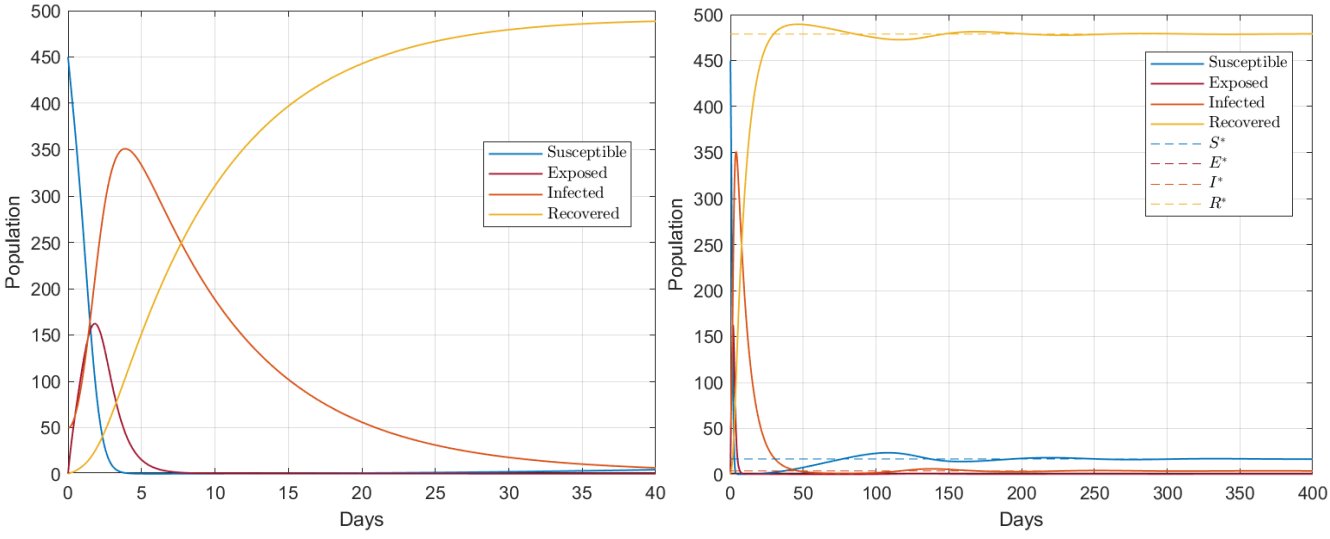
1. For the disease-free equilibrium it was clear that $R_0 < 1$ meant it was stable, so initial conditions close enough to the fixed point would imply the exposed, infected, and recovered population would tend to 0, and only leave susceptible individuals. However, $R_0 > 1$ yielded positive eigenvalues, so the disease would spread towards an endemic, in which the disease would survive indefinitely.

2. The condition $R_0 > 1$ implies the disease would become stable with a non-zero number of infected individuals in the long-run.

3.1.2. Numerical Simulation

1. The first scenario is the following:

   The chosen parameters are $\beta = 0.0075$, $\alpha = \frac{1}{8}$, $\mu = 1/1000$, $\gamma = \frac{1}{2}$, $(S, E, I, R)_{t=0} = (450, 0, 50, 0)$. The reproduction number is $R_0 = \frac{N\beta\gamma}{(\mu+\alpha)(\mu+\gamma)} = \frac{500 \cdot 0.0075 \cdot \frac{1}{8}}{(0.001+0.5)(0.001+0.125)} \approx 7.43$. The results of this simulation are shown below in Fig. 4

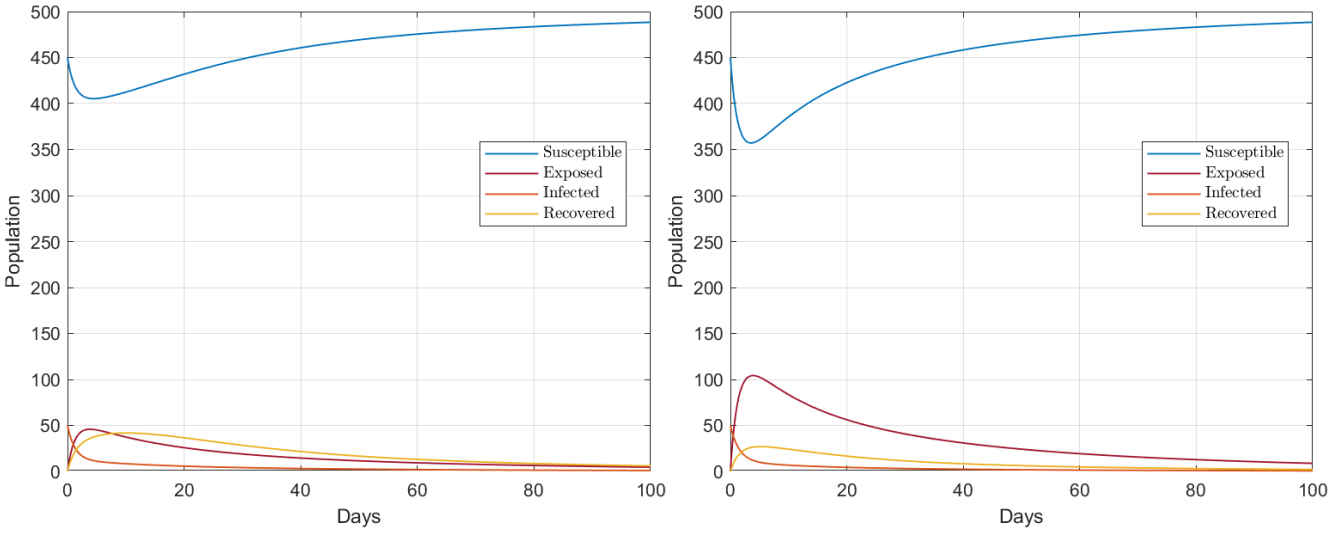


(a) Short term behavior

(b) Long-term behavior

Figure 4: Graphs with $R_0 > 1$

2. We now illustrate with Fig. 5 two examples in which the disease-free fixed point is stable, and the disease is contained.

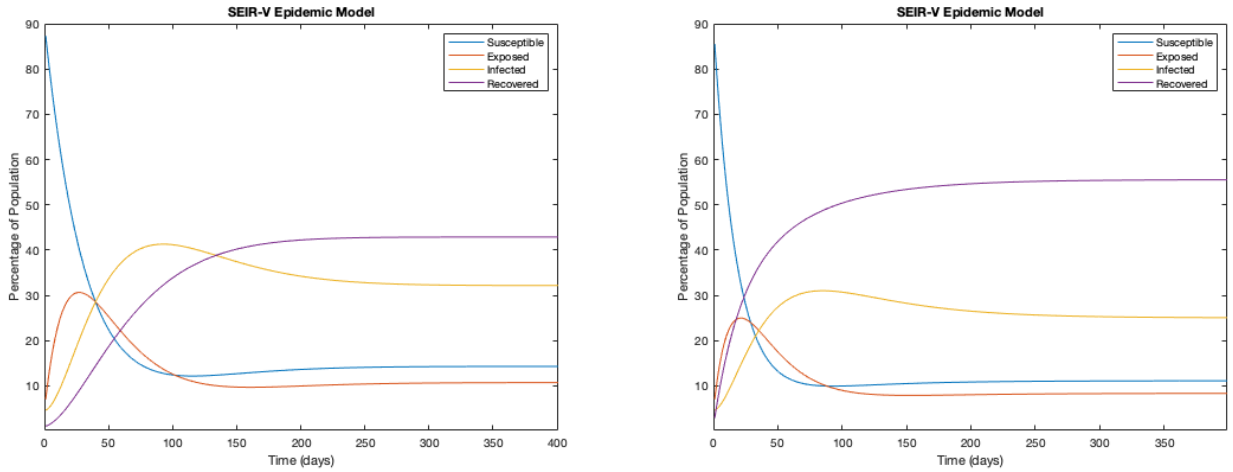


(a) $\beta = 0.002$, $\alpha = \frac{1}{2}$, $\mu = \frac{1}{10}$, $\gamma = \frac{1}{8}$, $R_0 = 0.93$ (b) $\beta = 0.0045$, $\alpha = \frac{2}{5}$, $\mu = \frac{1}{10}$ $\gamma = \frac{1}{25}$, $R_0 = 0.89$

Figure 5: Graphs with $R_0 < 1$

3.2. Stochastic Epidemic SEIR-V Results

For the analysis used to produce Fig. 6, the values of the constants used were: $\vec{x}_n = \begin{bmatrix} 0.9 & 0.045 & 0.045 & 0.01 \end{bmatrix}^T, \alpha = 1/100, \beta = 3/100, \delta = 3/100, \gamma = 1/100, \nu = 0, 2/100$. $\nu = 0$ was used to model the case of an epidemic where there was no vaccine available, while $\nu = 2/100$ was used to model the case where the daily percentage of the susceptible population getting vaccinated was 2 %. As depicted in Fig. 6a, once steady state is reached for the case of no vaccinations, around 40 % of the population is recovered while about 45 % of the population is infected or exposed. In contrast, with the introduction of vaccinations as in Fig. 6b, the steady state value of recovered individuals rises to 55 % and the amount of exposed or infected individuals drops to around 33 %. This is a significant reduction in the number of sick individuals, which highlights the benefits of vaccinations on a societal level.



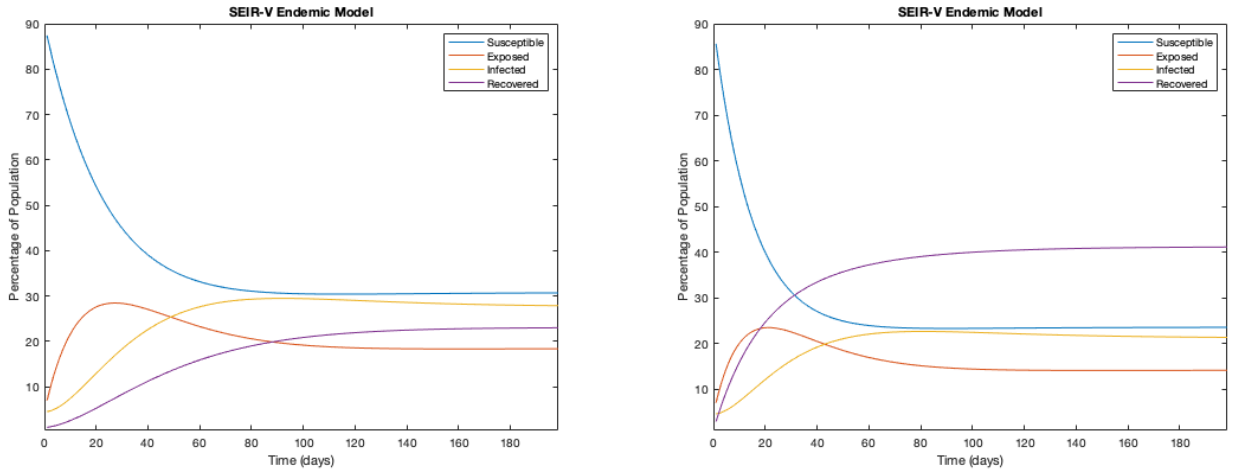
(a) No Vaccinations

(b) With Vaccinations

Figure 6: Epidemic SEIR-V Stochastic Models

3.3. Stochastic Endemic SEIR-V Results

For the analysis used to produce Fig. 7, the values of the constants used were the same as in the epidemic case discussed above, with the exception of introducing $\mu = 1/100$. As depicted in Fig. 7a, for the endemic case without vaccinations, once steady state is reached, around 23 % of the population is recovered while about 46 % of the population is infected or exposed. In contrast, with the introduction of vaccinations as in Fig. 7b, the steady state value of recovered individuals rises to 41 % and the amount of exposed or infected individuals drops to around 35 %. Again, the case of vaccinations has a much lower percentage of exposed and infected individuals. Further, for the case of no vaccinations the population of susceptible individuals exceeds the population of recovered individuals, whereas for the case of vaccinations, this is reversed. It is preferable to have a greater population of recovered individuals than susceptible individuals in terms of public health. Therefore, we again see the benefits of vaccinations on a societal level.



(a) No Vaccinations

(b) With Vaccinations

Figure 7: Endemic SEIR-V Stochastic Models

4. Discussion

4.1. Analysis of Continuous SEIR Results and Limitations

The numerical simulations and the stability of the fixed points from the phase plane analysis perfectly agree. We see the reproduction number is a very clear threshold to determine the severity of the epidemic caused by the disease. Note that the reproduction number is directly proportional to the total population, and thus large populations require much stronger conditions to ensure containment of the disease. Note also that the only case in which one of the parameters tends to zero is with $R_0 < 1$. In the endemic case, there is always a positive, non-zero amount of susceptible individuals left, which is due to the fact that there is a constant influx of births. In the case where $\mu = 0$, there is no scenario in which the susceptible compartment reaches an equilibrium, as there only is an out-flux $\beta \cdot S \cdot I$ which depends on itself. We can see that in this case, the only possibility of equilibrium is a strict, nonexistent amount of disease, making the epidemic case of $\mu = 0$ unrealistic, as everyone will be infected in the long run. In regard to limitations, the simplicity of the model aids with the fixed point stability computations and offers insight into the bigger picture, but fails to capture other complex dynamics of real-life scenarios. For a more accurate representation, one could account for time-dependent vaccination and age-stratified compartments, in which analytical tools usually fail and we must resort to numerical approximations.

4.2. Comparison of Epidemic and Endemic Stochastic SEIR-V Results and Limitations of These Methods

First, we note that the steady state results for the total number of infected and exposed individuals do not vary significantly between the epidemic and endemic cases when comparing the cases of vaccinations and no vaccinations with each other for each model. The major difference in these models is the percentage of the population that is susceptible has a much higher steady state value in the endemic case than the epidemic case due to constantly being replenished by the birth rate. In the epidemic case, the susceptible population slowly gets drained and the only additions to it are from the low chance of mutations making recovered individuals susceptible again. Next, we notice that because the percentage of the population that is susceptible is higher for the endemic case, and the exposed and infected percentage remains about the same, we must see a decrease in the number of recovered individuals at steady state given that the population size remains static. Thus, the unfortunate difference between the epidemic and endemic cases is that the recovered population has a lower steady state value for these long-term diseases such as Covid-19. Another difference between the epidemic and endemic models is that the endemic model reaches a steady state slightly faster than the epidemic case, however, the reason for this is unclear. Of course, having an equivalent birth and death rate is not realistic, so in actuality, these population percentages would vary. Particularly, there would likely be a different death rate for each class based on the higher likelihood of an infected or exposed individual dying than a recovered or susceptible individual. Further, the chance of a vaccinated individual getting susceptible again is likely different than a recovered but not vaccinated individual so these rates would also not be equivalent as they are in this model. These complications highlight the weaknesses of the simple SEIR-V models derived above, but also provide further directions future researchers could take to refine these models.

4.3. Comparison of Continuous SEIR and Stochastic SEIR-V Results

Even though the stochastic SEIR-V model accounts for vaccination rates, the numerical methods indicate the models have similar long-term behaviors. While the continuous SEIR model doesn't include a rate relating the exposed to the recovered, or a mutation rate from the recovered back into the susceptible class, we can see that in the long run, both show an endemic equilibrium. Though no techniques have been employed to derive a reproduction number for 6a, the fact that the curves even out with a non-zero number of infectious individuals indicate the reproduction number of the stochastic model is greater than

one as predicted with the analytical model. To achieve similar effects in the continuous model, such as the decrease of infected individuals due to the vaccine in the stochastic model, the main parameter that should be controlled would be β, i.e. decrease the infection rate through isolation or masking (a decrease in β corresponds to a decrease in R_0).

5. Conclusions

In this paper we analyzed the SEIR model both analytically and stochastically for the case of epidemics and endemics. We found the fixed points of the SEIR model and analyzed their stability, finding that the stability of the fixed points is dependent on the value of the parameter R_0. Specifically, we found that if $R_0 < 1$, the disease will eventually be eradicated, however, if $R_0 > 1$, the disease will perpetuate and turn from an epidemic into an endemic. We also examined the SEIR-V model stochastically and refined the analysis to include vaccination rates and virus mutations. We found that regardless of the case of an epidemic or an endemic, vaccinations are a critical parameter to reduce the number of exposed or infected individuals in the population. Disease modelling is a complex endeavor with real world consequences if there are inaccuracies. It requires constant refinement of previous models in order to better serve public health and safety policy creation. The simple methods used to derive these results represent a good start but require further refinement in order to produce truly accurate results. As discussed earlier, future refinements to the stochastic models could include accounting for a birth rate which is different than the death rate, and perhaps even varies amongst the different classes of the population. Another consideration could be the likelihood of a vaccinated person becoming susceptible as compared to the likelihood of a person who has recovered from the virus becoming susceptible again. Further research is likely also required to understand why the stochastic endemic models reach steady state faster than stochastic epidemic models. Due to the limited scope of this paper, only a few cases were tested for each model, and only cases with and without vaccinations were tested, rather than a range of vaccination rates. Future researchers could examine if there is a value of the vaccination parameter at which point increasing the rate of vaccinations does not significantly alter the steady state population results. A preliminary version of this analysis has already begun as shown by Randolph in [10], but more work is required. This could provide insight into how long vaccination campaigns for a particular disease should be a primary public health policy concern.

6. References

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