Simulating and Comparing Compartmental Models of Epidemics

Mathematical Modelling: Non-linearity,
Uncertainty and Computational Methods
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January 14, 2024

1 Introduction

Epidemiology, which examines the distributions and patterns of diseases in populations, is crucial for understanding, controlling, and preventing their spread. This report explores deterministic and stochastic models for disease dynamics. Utilizing the computational and plotting capabilities of MATLAB, we will perform simulations of these models for comprehensive analysis and comparisons.

2 Deterministic SIR model

Consider a continuous population of susceptible, infected and recovered individuals. Influenced only by transmission, recovery and mortality, the progression between these groups can be modelled with the following ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= -\beta \frac{SI}{N}, \\ \frac{dI}{dt} &= \beta \frac{SI}{N} - \gamma I - \mu I, \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{1}$$

where $N(t) = S(t) + I(t) + R(t)$ represents the population. Rate parameters are transmission β, recovery γ and mortality μ. While the terms for recovery and mortality are straightforward, the transmission term $\beta \frac{SI}{N}$ is less intuitive. $\beta \frac{SI}{N}$, signifies the interactions between susceptible and infected individuals in a well-mixed population. β represents the general interactions per unit time multiplied by the chance of transmission during each encounter.

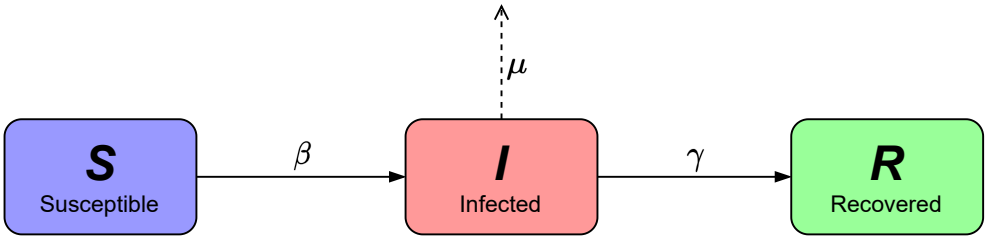


Diagram of the standard SIR model illustrating the compartments and flow between states.

2.1 Simulation under standard initial conditions

To test the dynamics of the standard deterministic SIR model, we encoded the system of ODEs (Equation 1) as a function, `deter_sir()` (Listing 1). Using another function, `ode45()`, which performs numerical integration, we computed solutions to the system under initial conditions: $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters: $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. The generated plot (Figure 1a) confirmed the successful encoding onto MATLAB, and displayed patterns of epidemics typical of standard SIR models. We can observe an initial surge in infections, followed by a gradual decline during recovery. Though this model is effective, there are notable limitations. Foremost that it uses continuous values for individuals, which are especially unrealistic of outcomes seen in smaller populations. Consequently, the ODEs can indicate that transmission and recovery rates never quite reach zero but approach it.

2.2 Sensitivity analysis of the transmission rate

When employing strategies to combat the spread of infectious disease, understanding their effects on the system is useful. As previously discussed, the parameter for transmission rate, β, governs how quickly the infection spreads. During the COVID pandemic, many vital measures were taken in order to reduce β such as social distancing, reducing interactions between individuals, and mask wearing, lowering the chance of infection upon exposure with infected. With our same initial conditions but using several values of β, we solved the system and plotted against the number of deaths for comparison (Figure 1b). From our simulations, we found that increasing β above 1 did not lower the plateau number of deaths but only reach it faster. Values below 1 did, however, reduce the maximum deaths in the epidemic simulation. This was because the low transmission meant that infection would die off before spreading to individuals, i.e. $\|\frac{dI}{dt}\| > \|\frac{dS}{dt}\|$.

2.3 Effects of vaccination: the SIRV model

Imagine that a vaccine, developed at the start of the epidemic, prevents transmission to vaccinated individuals. To model this, we extended the SIR model by adding a vaccinated compartment, V, and susceptible members are vaccinated at a constant rate ν. The extended SIRV model is now:

$$\begin{aligned} \frac{dS}{dt} &= -\beta\frac{SI}{N} - \nu, \\ \frac{dI}{dt} &= \beta\frac{SI}{N} - \gamma I - \mu I, \\ \frac{dR}{dt} &= \gamma I \\ \frac{dV}{dt} &= \nu \end{aligned} \tag{2}$$

where $\nu = 0$ when $S = 0$ means vaccination ceases when there are no susceptible. This was encoded onto MATLAB with another function `vaccine_deter_sir()`. After solving for solutions and generating the plot (Figure 1c) you can see that the peak number of infected is significantly smaller compared to the standard SIR (Figure 1a). Vaccination causes a faster decrease in susceptible members and allows numbers to reach zero, ending the epidemic. Further sensitivity analysis on ν shows the parameter's strong influence on the epidemic death toll plateau. As opposed to β where values above 1 only change how fast the death plateau is reached, increasing the vaccination rate is a definitive way to reduce the number of deaths.

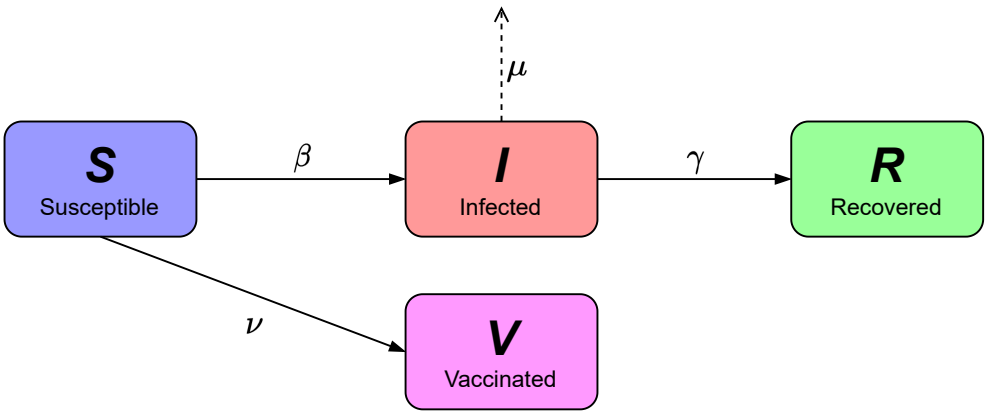


Diagram of the SIRV model.

2.4 Effects of reinfection: the SIRS model

The previous models assume permanent immunity in the recovered state R. For certain diseases, notably the common cold, immunity is temporary and recovered individuals can become susceptible for reinfection. In the real world, this occurs as the pathogen evolves to maintain its infectious capabilities. The dynamic nature of these infection-immunity interactions between pathogens and hosts often being described as an evolutionary arms race. Allowing that an individual from the recovered pool can become susceptible again. Allowing for recovered individuals to become susceptible again, the altered ODEs are as follows:

$$\begin{aligned} \frac{dS}{dt} &= -\beta\frac{SI}{N} + \theta R, \\ \frac{dI}{dt} &= \beta\frac{SI}{N} - \gamma I - \mu I, \\ \frac{dR}{dt} &= \gamma I - \theta R \end{aligned} \tag{3}$$

with θ as the rate interpreted as the chance, per each recovered individual, of becoming susceptible again per unit time. Note that this is different from the SIS model which does not assume a temporary immunity period. The addition of this reinfection term creates a constant cycling of infections and recovery, meanwhile each cycle results in more deaths. The effect of this model is that the population does not plateau and will eventually die out (Figure 1d).

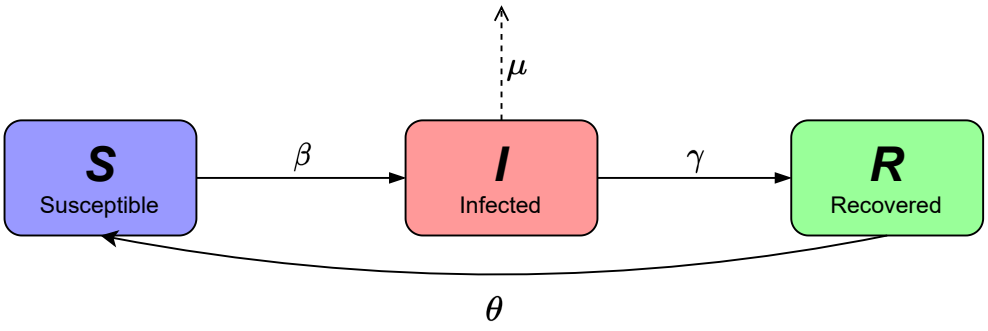


Diagram of the SIRS model.

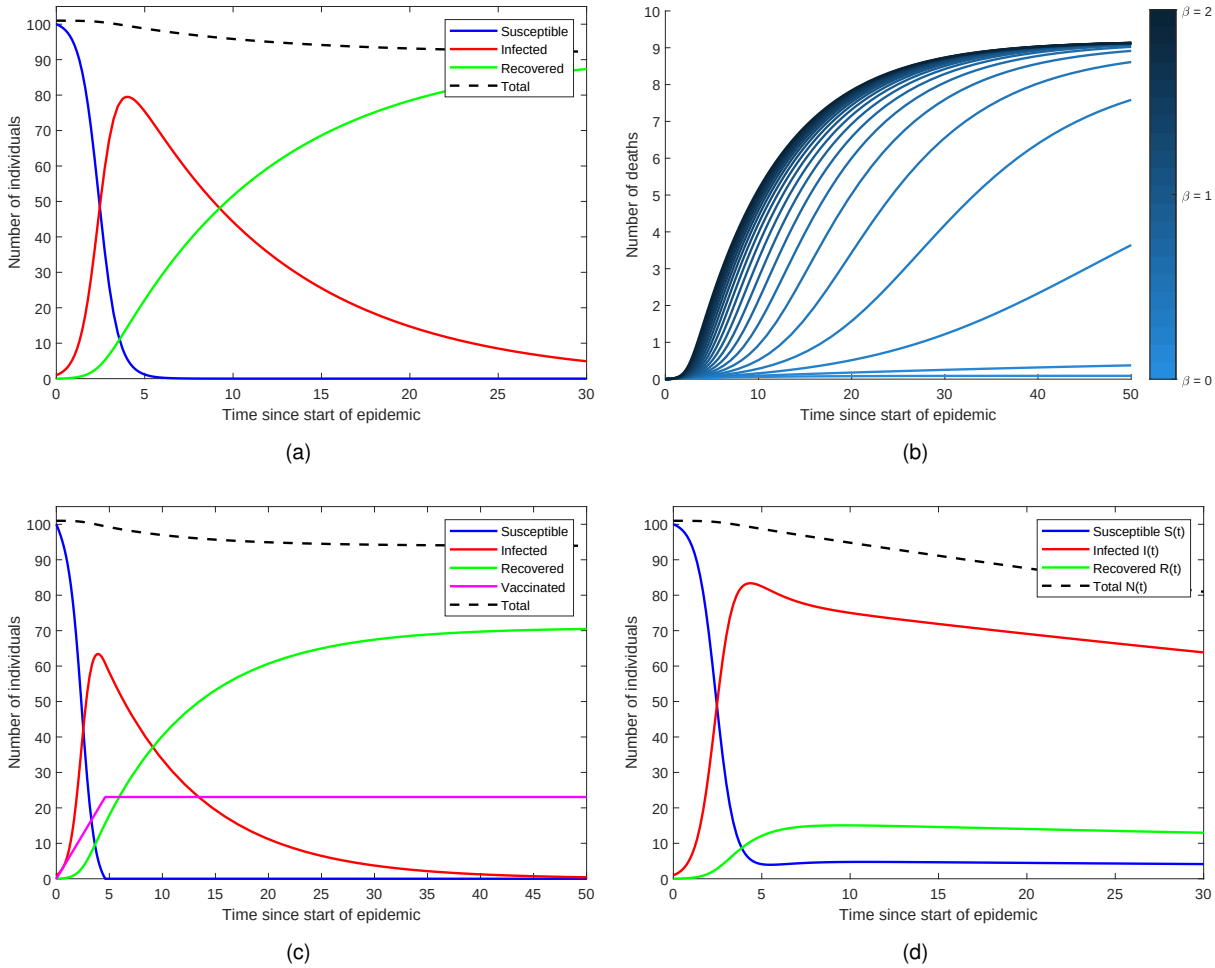


Figure 1: **(a)**: Graph showing the epidemic dynamics in the standard deterministic SIR model. Infected curve peaks at (4.07, 79.5). Generated via script `ex_1a.m`. **(b)**: Number of deaths over time plotted with different transmission parameters β. Other initial conditions kept the same. Generated via script `ex_1b.m`. **(c)**: Graph showing the epidemic dynamics in the deterministic SIRV model using vaccination rate ν = 5 vaccinations per unit time. Infected curve peaks at (3.96, 63.4). Generated via file `ex_1c_i.m`. **(d)**: Graph showing epidemic dynamics in the deterministic SIRS model with reinfection chance θ = 0.5 per individual per unit time. Infected curve peaks at (4.33, 83.4). Generated via script `ex_1c_ii.m`.

Unless otherwise stated, the initial conditions are $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. Scripts for generating these plots can be found in the `code/ex1` folder.

Listing 1: Code snippet for `deter_sir()`

```
function f = deter_sir(t, X)
% ODEs of the deterministic SIR Model
global beta gamma mu % Initialising globals
dSdt = -beta * X(1) * X(2) / sum(X);
dIdt = beta * X(1) * X(2) / sum(X) - X(2)*(gamma + mu);
dRdt = gamma * X(2);
f = [dSdt; dIdt; dRdt];
end
```

3 Stochastic SIR models

Now picture the epidemic as the random sequence of events: infection, recovery and death. Captured in the function `stoch_sir()` (Listing 2), we now define infection, recovery and

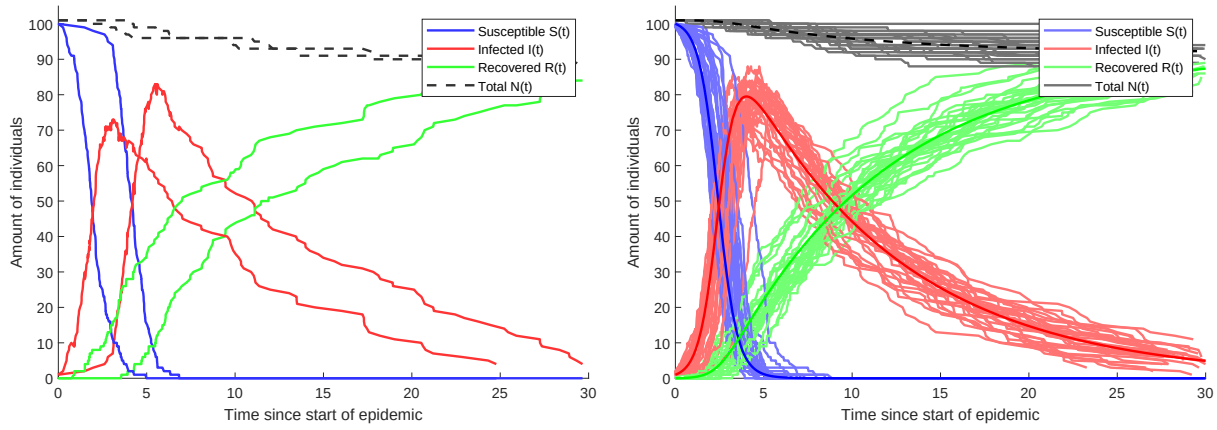
death, as Poisson processes. These events occur at rates akin to terms in ODEs of the deterministic model with $\beta SI/N$ for infection, γI for recovery and μI for death. The interval until the next infection, recovery and death are thus random variables which follow exponential distributions. By using the function `exprnd()`, which generates random numbers from the exponential distribution, we decide the next event by which time interval generated was smallest among them. The ensuing event alters the counts of susceptible, infected, and recovered individuals in the system, prompting updates to the corresponding rate terms. We then iterated this process, recording the counts of S, I and R each time, for a given length of time. After plotting, you can see that the trajectories of the stochastic model (Figure 2a) closely resemble the dynamics of the deterministic model (Figure 1a). Simulations also revealed many cases in which the infection would not spread. In particular, the system capped at one or two infected individuals which would then recover or die before infecting more individuals. It is important to note of these 'small epidemic' cases for the following sections.

3.1 Epidemic duration, death toll and peak infected count

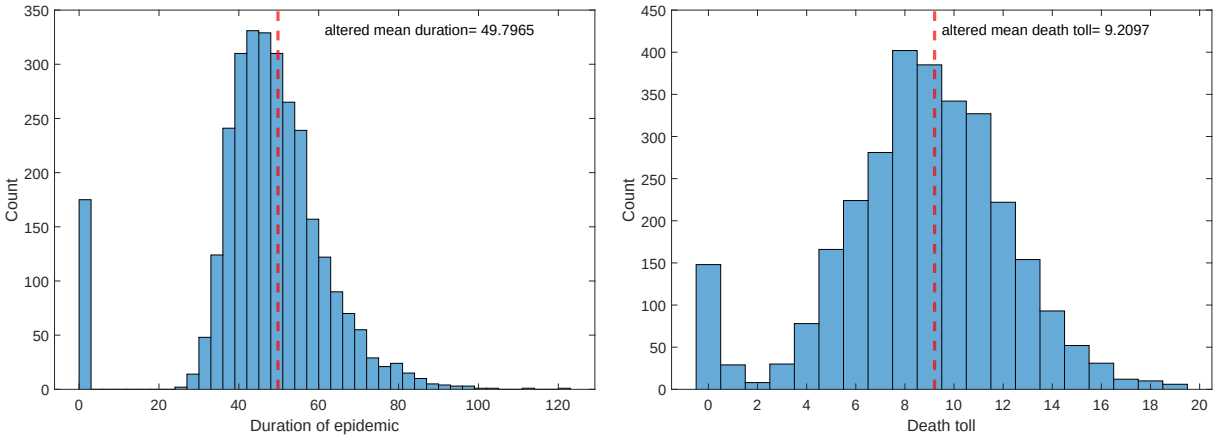
Compared to the deterministic model, simulations of the stochastic model exhibited a finite epidemic duration, ending when the number of infected individuals reached zero. To find how this is distribution, we simulated 3000 epidemics and recorded their duration on a histogram (Figure 2b). 'Small epidemic' scenarios are clearly illustrated by the peak on the left. The computed mean duration was 46.9 including these 'small epidemics', and 49.80 after excluding them. Other statistics such as the average death toll, peak number of infected and also time taken to reach peak infected were also computed using a sample size of 3000.

Listing 2: Code snippet for `stoch_sir()`

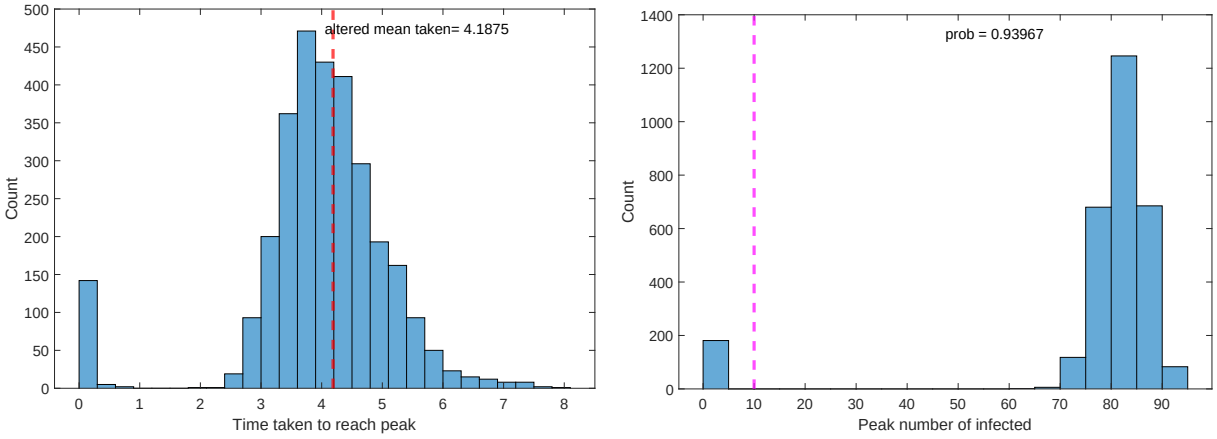
```
function [t, SIR] = stoch_sir(T, X)
% Stochastic SIR model as the sequence of independent poisson processes.
global gamma beta mu
[S_n, I_n, R_n] = deal(X(1), X(2), X(3));
SIR = [S_n, I_n, R_n];
t = 0;
tvec = 0;
while t<T % Runs simulation until time T reached
    rates = [beta * S_n * I_n / (S_n + I_n + R_n), mu * I_n, gamma * I_n]; %
        Updates rates
    [tau, event] = min(exprnd(1./rates)); % time till next event
    if event == 1 % infection happens next
        S_n = S_n - 1;
        I_n = I_n + 1;
    elseif event == 2 % death happens next
        I_n = I_n - 1;
    else % recovery happens next
        I_n = I_n - 1;
        R_n = R_n + 1;
    end
    t = t + tau;
    SIR = [SIR; S_n, I_n, R_n]; % Updates SIR count
    tvec = [tvec, t];
end
```



(a)



(b) (c)



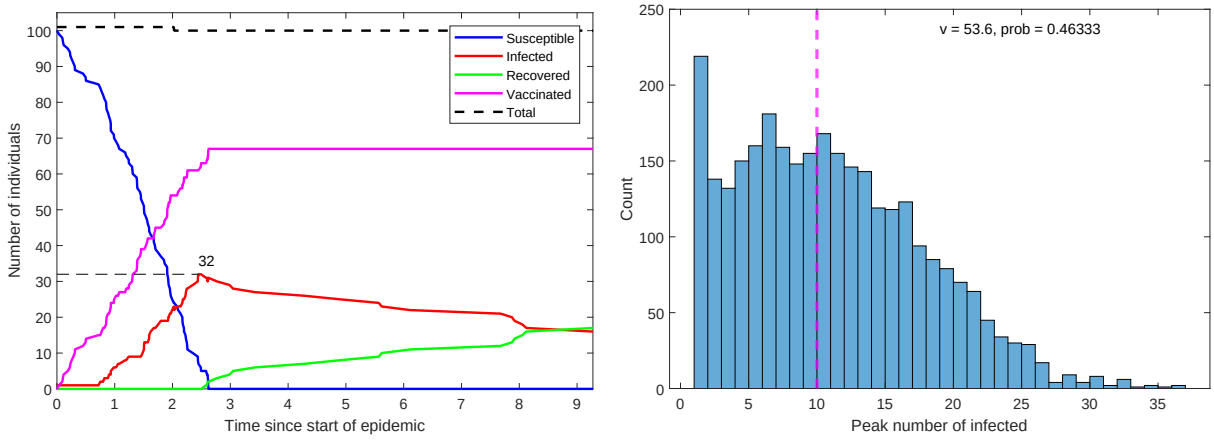
(d) (e)

Figure 2: (**a**): Graphs showing simulations of the stochastic SIR model with 2 on the left and 30 on the right. Graph on right also plotted with the deterministic SIR model for comparison. (**b**): Histogram showing distribution of epidemic duration. Mean before was 47.2. (**c**): Histogram showing distribution of death tolls. Mean before was 8.68. (**d**): Histogram showing time taken to reach the peak number of infected individuals. Mean before was 3.98. (**e**): Histogram showing the peak number of infected individuals. Dashed line at 10 individuals. The 0-5 bar has a count of 181.

For these figures, the initial conditions are $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. Altered means are after removing small epidemic cases. Scripts for generating these plots can be found in the `code/ex2` folder.

3.2 Ideal vaccination rates

We have shown that even with a single initially infected individual, substantial epidemics are still very likely to occur. Small epidemics, where infected individuals remain under 10, occur in only 181 out of 3000 simulations (Figure 2e). This approximates to a probability of 0.939 for the peak infected count to exceed 10. We also found that the small epidemic cases would have a maximum of two infected and no epidemics peaked at values between 3 and 60 infected. This implies that with 3 infected, a substantial epidemic was almost certain, leading to at least 60 more infections. We then wanted to know what rate of vaccination would half the current likelihood of such substantial epidemics. By creating an altered version of `stoch_sir()` called `vaccine_stoch_sir()` we were able to implant a constant vaccination rate similar to the deterministic SIRV model. Utilising a `while` loop that performed a sensitivity analysis on vaccination rate v, were able to deduce a minimum v = 53.6 such that the likelihood of infected growing to more than 10 was halved.



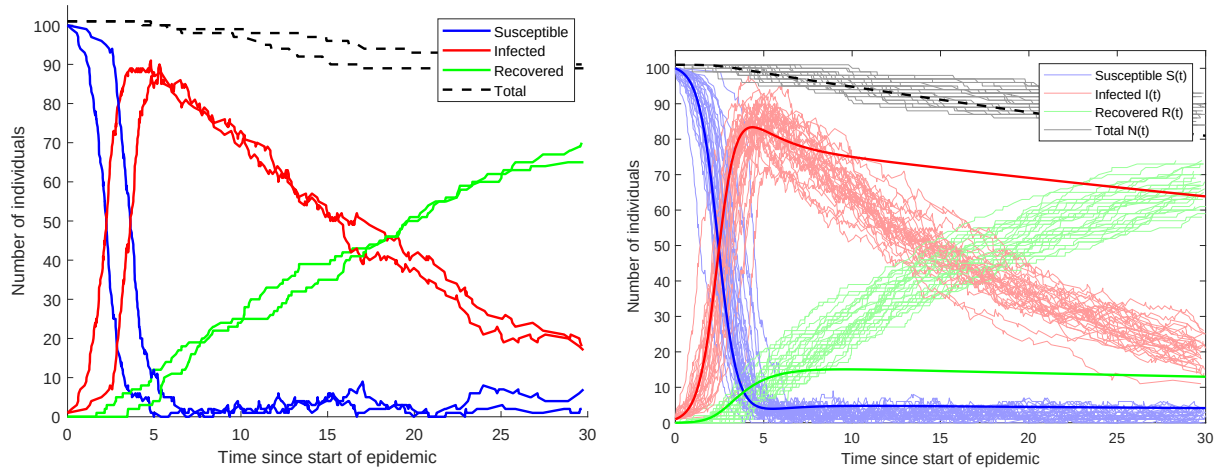
(a) (b)

Figure 3: (**a**): Graph showing an example simulation of the stochastic SIRV model with a vaccination rate of 53.6. (**b**): Histogram showing the peak infected distribution in 3000 simulations of the stochastic SIRV model. Value 'prob' is an approximation of the probability that a simulation will result in a peak of under 10 infected. Dashed line at 10 infected.

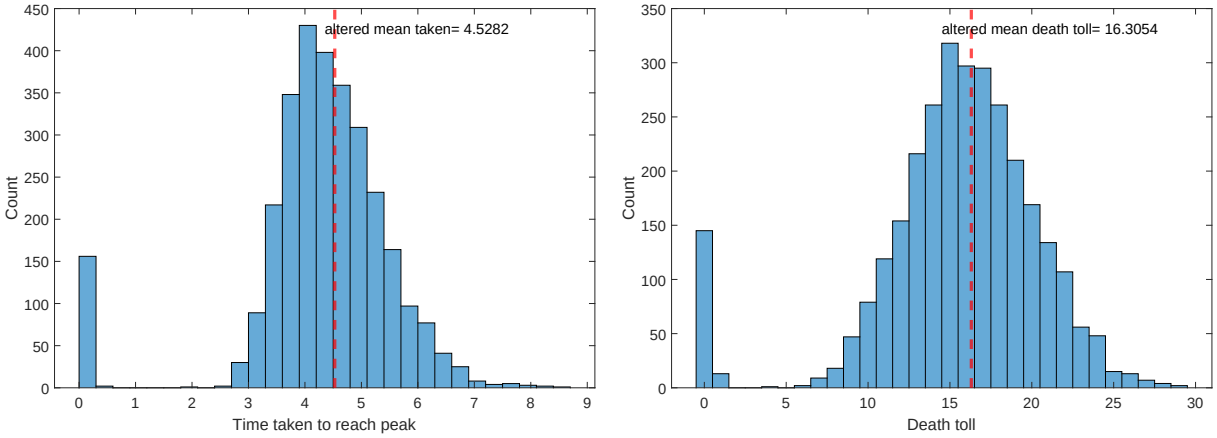
For these figures, the initial conditions are $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. Scripts for generating these plots can be found in the `code/ex2` folder.

3.3 Comparison with a modified model with reinfection.

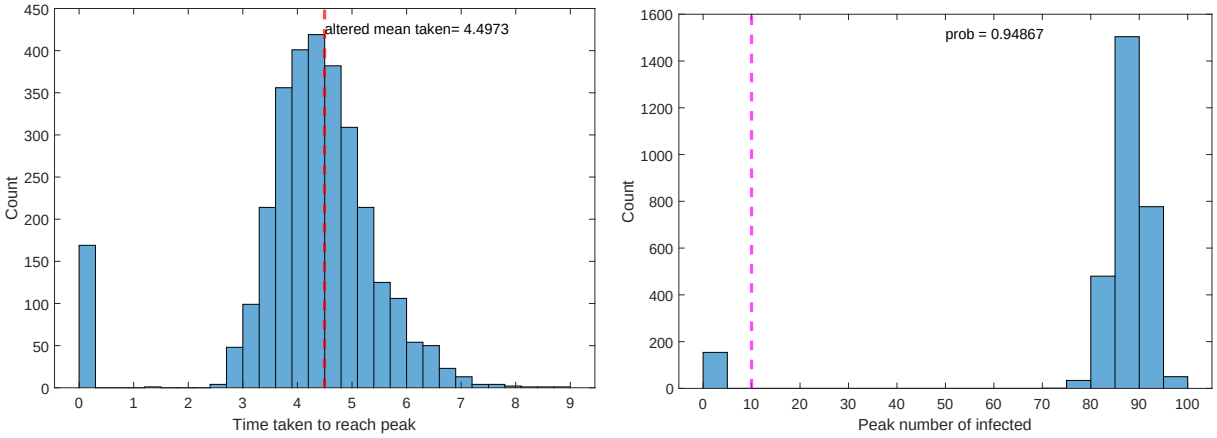
Revisiting the concept of reinfection, we created a modified SIRS model (Listing 3), in which individuals have a 50% chance of becoming susceptible again upon recovery. Unlike the SIRS model, this model lacks a temporary immunity period, resembling the continuous susceptible-infected-susceptible (SIS) cycle with added permanent immunity probability. Then we conducted the same analyses as before with the standard stochastic model (Figure 4a-4e), and also with the stochastic SIRV model (Figure 5a) requiring another vaccination model. The result is an epidemic lasting almost twice as long with a higher death toll (Figure 5c, 5d) compared to the standard stochastic model.The continuous cycling of infected individuals back to susceptible prevents the infected count from reaching zero (Figure 5a) and provides more opportunities for mortality events. To half the likelihood of having more than 10 infected individuals at the peak, the adjusted model also necessitated a higher vaccination rate of v = 54 compared to v = 53.6 in the standard stochastic SIRV model. We argue this model better reflects real epidemic dynamics, where recovery outcomes vary; e.g., antibiotics may treat but not guarantee immunity, unlike natural recovery with antibody production.



(a)



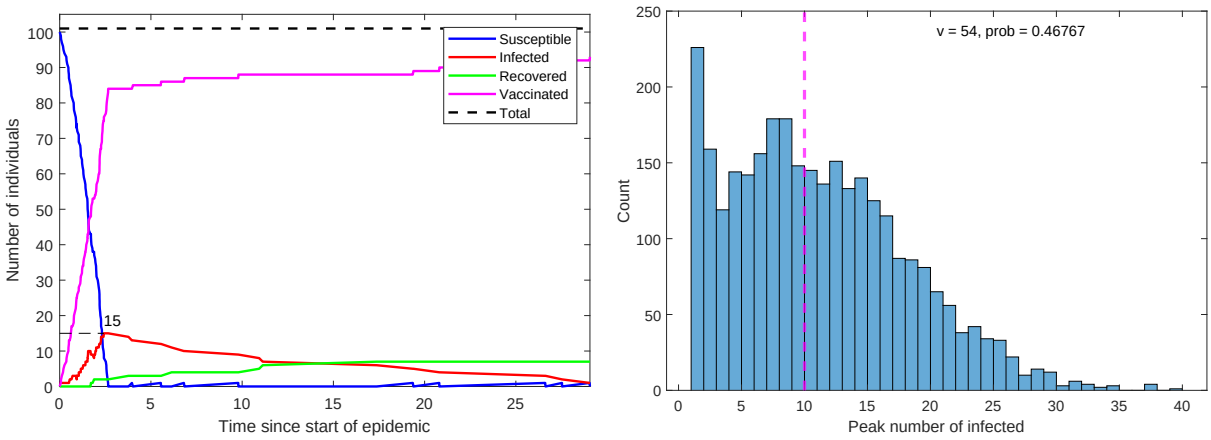
(b) (c)



(d) (e)

Figure 4: (**a**): Graphs showing simulations of the modified stochastic SIR model with 2 on the left and 30 on the right. Graph on right also plotted with the deterministic SIRS model for comparison. (**b**): Histogram showing distribution of epidemic duration. Mean before was 74.4. (**c**): Histogram showing distribution of death tolls. Mean before was 15.5. (**d**): Histogram showing time taken to reach the peak number of infected individuals. Mean before was 4.29. (**e**): Histogram showing the peak number of infected individuals. Dashed line at 10 individuals. The 0-5 bar has a count of 181.

For these figures, the initial conditions are $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. Altered means are after removing small epidemic cases. Scripts for generating these plots can be found in the `code/ex3` folder.



(a) (b)

Figure 5: (**a**): Graph showing an example simulation of the stochastic SIRV model with a vaccination rate of 53.6. (**b**): Histogram showing the peak infected distribution in 3000 simulations of the stochastic SIRV model. Value 'prob' is an approximation of the probability that a simulation will result in a peak of under 10 infected. Dashed line at 10 infected.

For these figures, the initial conditions are $S(0) = 100$, $I(0) = 1$, $R(0) = 0$; and parameters $\beta = 2$, $\gamma = 0.1$, $\mu = 0.01$. We also defined a 50% chance an individual that would have recovered, became susceptible again. Scripts for generating these plots can be found in the `code/ex3` folder.

Listing 3: Code snippet for `mod_stoch_sir()`

```
...
rates = [beta * S_n * I_n / (S_n + I_n + R_n), mu * I_n, gamma * I_n]; % Updates
    rates
[tau, event] = min(exprnd(1./rates)); % time till next event
if event == 1 % infection happens next
    S_n = S_n - 1;
    I_n = I_n + 1;
elseif event == 2 % death happens next
    I_n = I_n - 1;
else % recovery happens next
    I_n = I_n - 1;
    flip = rand; % RNG for individual's fate
    if flip > 0.5;
        R_n = R_n + 1 % Recovers
    else
        S_n = S_n + 1 % Susceptible again
    end
end
...
```

4 Conclusion

In summary, we have constructed and applied both deterministic and stochastic compartmental SIR models to simulate epidemics and analyze their dynamics. With its element of randomness, our stochastic simulations revealed cases of 'small epidemics', in which infected immediately die or recover before disease has spread. We have also reviewed several extensions to the models which include the effects of vaccination and reinfection. By doing further sensitivity analysis of transmission and vaccination parameters, we have proposed values which would reduce the spread of the disease. The project highlighted the capability of MATLAB as a modelling tool, and also gave insight into the techniques for modelling real systems.