MAT 196 Laboratory Exercise No. 5

Jeanne Marie B. Quiñanola

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1 Introduction

The Susceptible-Infected-Recovered (SIR) model is a cornerstone in epidemiological modeling, offering a framework to understand the dynamics of infectious disease spread within a population. This model divides the population into three compartments: susceptible individuals (S_t), infected individuals (I_t), and recovered individuals (R_t). The transitions between these compartments are governed by parameters such as the infection rate (β), recovery rate (γ), and the vaccination rate given by (p).

In its discrete-time formulation, the SIR model is represented by the following system of difference equations:

$$\begin{cases} S_{t+1} = (1-p)S_t - \dfrac{\beta}{N} I_t S_t + b(R_t + I_t) \\ I_{t+1} = \dfrac{\beta}{N} I_t S_t + (1 - b - \gamma) I_t \\ R_{t+1} = (1-b)R_t + \gamma I_t + pS_t \end{cases}$$

where $0 < p + \beta < 1$ and $0 < b + \gamma < 1$ with the following parameters:

- S_t: Number of susceptible individuals at time t
- I_t: Number of infected individuals at time t
- R_t: Number of recovered individuals at time t
- N: Total population size
- β: Contact rate
- γ: Probability of recovery
- p: Proportion of vaccinated individuals
- b: Probability of birth

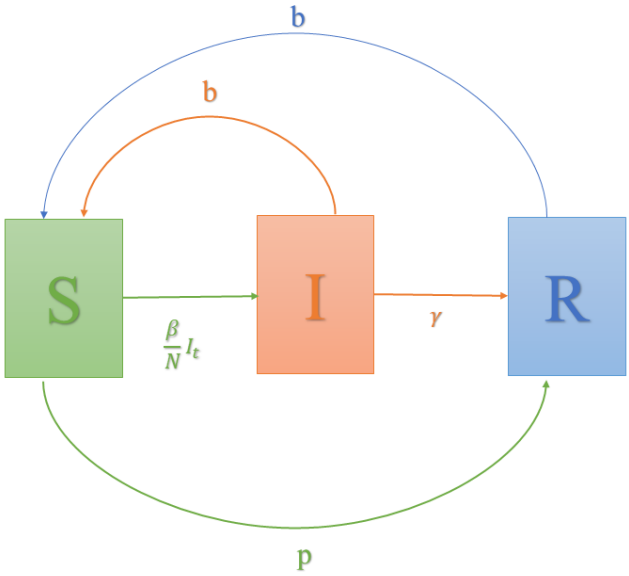

Figure 1: State flow diagram of the SIR model

The Susceptible-Infected-Recovered (SIR) model is a foundational tool in epidemiology, providing a simplified framework to understand the spread of infectious diseases. However, its simplicity can lead to oversights in capturing the complexities of real-world disease dynamics. For instance, a study published in *Nature* examined the inefficiencies of traditional SIR models in forecasting the COVID-19 epidemic, highlighting the challenges in accurately predicting disease spread using these models [1]. Additionally, research in *Frontiers in Epidemiology* discussed the limitations of compartmental models, including the SIR model, in capturing the complexities of disease transmission, emphasizing the need for more nuanced approaches [2].

Despite these limitations, the SIR model remains a foundational tool in epidemiology. Its simplicity allows for the derivation of key metrics such as the basic reproduction number (R_0), which indicates the average number of secondary infections produced by a single infected individual in a fully susceptible population. Understanding R_0 is crucial for assessing the potential for disease spread and evaluating control measures.

Moreover, the SIR model has been adapted and extended to incorporate various factors influencing disease dynamics. For example, a study in *Scientific Reports* presented a modified age-structured SIR model that accounts for social contact patterns and distancing measures, providing a more detailed analysis of COVID-19 transmission [1].

Similarly, research has developed an SIR model to study the effects of vaccination adherence and health protocols on epidemic dynamics, underscoring the model's versatility in evaluating intervention strategies[3].

In summary, the SIR model serves as a fundamental framework in epidemiology, offering valuable insights into disease transmission dynamics. While it has certain limitations, ongoing research continues to refine and adapt the model to better capture the complexities of infectious disease spread and to inform effective public health interventions.

2 Analysis and Discussion

2.1 Algebraic Solution

Consider the system of difference equations given by:

$$\begin{cases} S_{t+1} = (1-p)S_t - \dfrac{\beta}{N} I_t S_t + b(R_t + I_t), \\ I_{t+1} = \dfrac{\beta}{N} I_t S_t + (1-b-\gamma) I_t, \\ R_{t+1} = (1-b)R_t + \gamma I_t + pS_t. \end{cases}$$

Since the total population $N = S_t + I_t + R_t$ remains constant over time, $N_t = N$. We have,

$$\begin{aligned} S_{t+1} + I_{t+1} + R_{t+1} &= (1-p)S_t - \frac{\beta}{N} I_t S_t + b(R_t + I_t) + \frac{\beta}{N} I_t S_t \\ &\quad + (1-b-\gamma) I_t + (1-b)R_t + \gamma I_t + pS_t \\ &= S_t + I_t + R_t \\ &= N. \end{aligned}$$

Thus, I_{t+1} and S_{t+1} do not depend on R_t . The given system of difference equation can be reduced to the following :

$$\begin{cases} S_{t+1} = (1-p)S_t - \dfrac{\beta}{N} I_t S_t + b(N - I_t - S_t + I_t), \\ I_{t+1} = \dfrac{\beta}{N} I_t S_t + (1-b-\gamma) I_t. \end{cases}$$

Simplifying further:

$$\begin{cases} S_{t+1} = (1-p)S_t - \dfrac{\beta}{N} I_t S_t + b(N - S_t) = f(S_t, I_t), \\ I_{t+1} = \dfrac{\beta}{N} I_t S_t + (1-b-\gamma) I_t = g(S_t, I_t). \end{cases}$$

Now, the equilibrium solutions satisfy $f(\bar{S}, \bar{I}) = \bar{S}$ and $g(\bar{S}, \bar{I}) = \bar{I}$. That is,

$$\begin{aligned} (1-p)\bar{S} - \frac{\beta}{N} \bar{I}\bar{S} + b(N - \bar{S}) &= \bar{S}, \\ \frac{\beta}{N} \bar{I}\bar{S} + (1-b-\gamma)\bar{I} &= \bar{I}. \end{aligned}$$

Simplifying we get:

$$-p\bar{S} - \frac{\beta}{N} \bar{I}\bar{S} + b(N - \bar{S}) = 0, \tag{1}$$

$$\frac{\beta}{N} \bar{I}\bar{S} - (b+\gamma)\bar{I} = 0. \tag{2}$$

Adding (1) and (2):

$$\begin{aligned} -p\bar{S} - \frac{\beta}{N} \bar{I}\bar{S} + b(N - \bar{S}) + \frac{\beta}{N} \bar{I}\bar{S} - (b+\gamma)\bar{I} &= 0, \\ b(N - \bar{S}) &= (b+\gamma)\bar{I} + p\bar{S}. \end{aligned} \tag{3}$$

Substituting (3) into (1) we have:

$$\begin{aligned} -p\bar{S} - \frac{\beta}{N} \bar{S} + (b+\gamma)\bar{I} + p\bar{S} &= 0, \\ \bar{I}\left(-\frac{\beta}{N} \bar{S} + b + \gamma \right) &= 0. \end{aligned}$$

Thus, either:

$$\bar{I} = 0 \quad \text{or} \quad \bar{S} = \frac{(b+\gamma)N}{\beta}.$$

If $\bar{I} = 0$, then substituting into (1) we have:

$$\begin{aligned} -p\bar{S} - \frac{\beta}{N}(0)\bar{S} + b(N - \bar{S}) &= 0, \\ -p\bar{S} + b(N - \bar{S}) &= 0, \\ -p\bar{S} + bN - b\bar{S} &= 0, \\ \bar{S}(p + b) &= bN \\ \bar{S} &= \frac{bN}{p + b}. \end{aligned}$$

If $\bar{S} = \frac{(b+\gamma)N}{\beta}$, then substituting into (1) we have:

$$\begin{aligned} -p\left(\frac{b+\gamma}{\beta}\right)N - \frac{\beta}{N}\bar{I}\left(\frac{b+\gamma}{\beta}\right)N + b\left(N - \left(\frac{b+\gamma}{\beta}\right)N\right) &= 0, \\ -p\left(\frac{b+\gamma}{\beta}\right)N - \bar{I}(b+\gamma) + b\left(\frac{N\beta - (b+\gamma)N}{\beta}\right) &= 0 \\ \bar{I}(b+\gamma) &= -p\left(\frac{b+\gamma}{\beta}\right)N + b\left(\frac{N\beta - (b+\gamma)N}{\beta}\right) \\ \bar{I}(b+\gamma) &= b + \gamma\left(-\frac{pN}{\beta} - \frac{bN}{\beta}\right) + bN \\ \bar{I} &= bN\left(\frac{\beta - (b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta} \end{aligned}$$

Thus, there are two equilibrium solutions:

- Disease-free equilibrium:

$$(\bar{S}, \bar{I}) = \left(\frac{bN}{p+b}, 0\right)$$

- Endemic Equilibrium:

$$(\bar{S}, \bar{I}) = \left(\frac{(b+\gamma)N}{\beta}, bN\left(\frac{\beta - (b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta}\right)$$

2.2 Stability analysis

The Jacobian matrix J of the model is given by:

$$J = \begin{pmatrix} \frac{\partial f}{\partial S} & \frac{\partial f}{\partial I} \\ \frac{\partial g}{\partial S} & \frac{\partial g}{\partial I} \end{pmatrix} = \begin{pmatrix} 1 - p - \frac{\beta}{N}I - b & -\frac{\beta}{N}S \\ \frac{\beta}{N}I & \frac{\beta}{N}S + 1 - b - \gamma \end{pmatrix}.$$

Let J_1 be the Jacobian matrix evaluated at the disease-free equilibrium $(\bar{S}, \bar{I}) = (\frac{bN}{p+b}, 0)$.

$$J_1 = \begin{pmatrix} 1 - p - \frac{\beta}{N}(0) - b & -\frac{\beta}{N}\left(\frac{bN}{p+b}\right) \\ \frac{\beta}{N}(0) & \frac{\beta}{N}\left(\frac{bN}{p+b}\right) + 1 - b - \gamma \end{pmatrix}$$

$$J_1 = \begin{pmatrix} 1 - p - b & -\frac{\beta b}{p+b} \\ 0 & \frac{\beta b}{p+b}S + 1 - b - \gamma \end{pmatrix}$$

J_1 is locally asymptotically stable if $|\lambda_{1,2}| < 1$.

$$\begin{aligned} &\det(J_1 - \lambda I) = 0 \\ &\det\begin{pmatrix} 1 - p - b - \lambda & \frac{-\beta b}{p+b} \\ 0 & \frac{\beta b}{p+b} + 1 - b - \gamma - \lambda \end{pmatrix} = 0 \\ &(1 - p - b - \lambda)\left(\frac{\beta b}{p+b} + 1 - b - \gamma - \lambda\right) = 0 \\ &\lambda_1 = 1 - p - b \quad \text{or} \quad \lambda_2 = \frac{\beta b}{p+b} + 1 - b - \gamma. \end{aligned}$$

Hence,

$$\begin{aligned}|\lambda_1| < 1 \Leftrightarrow |1-p-b| < 1 &\Leftrightarrow -1 < 1-(p+b) < 1\\ &\Leftrightarrow -2 < -(p+b) < 0\\ &\Leftrightarrow 2 > p+b > 0\\ &\Leftrightarrow 0 < p+b < 2\end{aligned}$$

Meanwhile,

$$\begin{aligned}|\lambda_2| < 1 &\Leftrightarrow \left|\frac{\beta b}{p+b} + 1 - b - \gamma\right| < 1\\ &\Leftrightarrow -1 < \frac{\beta b}{p+b} + 1 - b - \gamma < 1\\ &\Leftrightarrow \frac{\beta b}{p+b} - b - \gamma < 0\\ &\Leftrightarrow \frac{\beta b}{p+b} < b + \gamma\\ &\Leftrightarrow \frac{\beta b}{(p+b)(b+\gamma)} < 1.\end{aligned}$$

Thus, we have the basic reproduction number given by:

$$\mathcal{R}_\prime = \frac{\beta b}{(p+b)(b+\gamma)}.$$

If $\mathcal{R}_\prime < 1$, there will be no epidemic.

Meanwhile, let J_2 be the Jacobian matrix evaluated at the endemic equilibrium:

$$(\bar{S}, \bar{I}) = \left(\frac{(b+\gamma)N}{\beta},\, bN\left(\frac{\beta-(b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta}\right).$$

Then, we have:

$$\begin{aligned}J_2 &= \begin{pmatrix} 1-p-\left(\frac{\beta}{N}\left(bN\left(\frac{\beta-(b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta}\right)\right) - b & -\frac{\beta}{N}\left(\frac{(b+\gamma)N}{\beta}\right) \\ \frac{\beta}{N}\left(bN\left(\frac{\beta-(b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta}\right) & \frac{\beta}{N}\left(\frac{(b+\gamma)N}{\beta}\right) + 1 - b - \gamma \end{pmatrix}\\ &= \begin{pmatrix} 1 - b\left(\frac{\beta-(b+\gamma)}{b+\gamma}\right) - b & -(b+\gamma) \\ b\left(\frac{\beta-(b+\gamma)}{b+\gamma}\right) - p & 1 \end{pmatrix}.\end{aligned}$$

Writing J_2 in terms of $\mathcal{R}_\prime = \frac{\beta b}{(p+b)(b+\gamma)}$:

$$J_2 = \begin{pmatrix} 1 - \mathcal{R}_\prime(p+b) & \frac{-\beta b}{(p+b)\mathcal{R}_\prime} \\ \mathcal{R}_\prime(p+b) - b - p & 1 \end{pmatrix}.$$

The following are the trace and determinant of J_2, respectively.

$$\begin{aligned}\mathrm{Tr}(J_2) &= 1 - \mathcal{R}_\prime(p+b) + 1 = 2 - \mathcal{R}_\prime(p+b),\\ \det(J_2) &= 1 - \mathcal{R}_\prime(p+b) + \frac{\beta b\left(\mathcal{R}_\prime(p+b) - b - p\right)}{(p+b)\mathcal{R}_\prime}.\end{aligned}$$

By Theorem 2.17, J_2 is locally asymptotically stable if and only if:

$$|\mathrm{Tr}(J_2)| < 1 + \det(J_2) < 2.$$

Substituting,

$$\begin{aligned}|2 - \mathcal{R}_\prime(p+b)| &< 2 - \mathcal{R}_\prime(p+b) + \frac{\beta b\left(\mathcal{R}_\prime(p+b) - b - p\right)}{(p+b)\mathcal{R}_\prime} < 2\\ 2 - \mathcal{R}_\prime(p+b) &< 2 - \mathcal{R}_\prime(p+b) + \frac{\beta b\left(\mathcal{R}_\prime(p+b) - b - p\right)}{(p+b)\mathcal{R}_\prime} < 2\end{aligned}$$

$$0 < \frac{\beta b\left(\mathcal{R}_\prime(p+b) - b - p\right)}{(p+b)\mathcal{R}_\prime} < \mathcal{R}_\prime(p+b)$$
$$0 < Bb\left(1 - \frac{1}{\mathcal{R}_\prime}\right) < \mathcal{R}_\prime(p+b).$$

Since $\mathcal{R}_\prime > 1$ in the endemic equilibrium,

$$\mathcal{R}_\prime(p+b) > 1 > \beta b\left(1 - \frac{1}{\mathcal{R}_\prime}\right).$$

From the assumption,

$$2 - \mathcal{R}_\prime(p+b) > 0,$$
$$\mathcal{R}_\prime < \frac{2}{p+b}.$$

Combining this with $\mathcal{R}_\prime > 1$, we have $1 < \mathcal{R}_\prime < \frac{2}{p+b}$. Hence, the endemic equilibrium given by $(\bar{S}, \bar{I}) = \left(\frac{(b+\gamma)N}{\beta}, bN\left(\frac{\beta-(b+\gamma)}{\beta(b+\gamma)}\right) - \frac{pN}{\beta}\right)$ is locally asymptotically stable if $1 < \mathcal{R}_\prime < \frac{2}{p+b}$.

3 Numerical Simulations

We consider the following cases with the total population $N = 100$, initial value of susceptible and infected cases to be $S(0) = 70$ and in $I(0) = 30$, respectively.

- Case 1: $\beta = 0.2, b = 0.05 = \gamma$, and $p = 0.25$
- Case 2: $\beta = 0.2, b = 0.05 = \gamma$, and $p = 0.50$
- Case 3: $\beta = 0.2, b = 0.05 = \gamma$, and $p = 0.75$
- Case 4: $\beta = 0.2, b = 0.05 = \gamma$, and $p = 0$

If we calculate the $\mathcal{R}_\prime$ for each cases we have the following:

- Case 1:
$$\mathcal{R}_\prime = \frac{(0.2)(0.05)}{(0.25+0.05)(0.05+0.05)} = 0.\bar{3} < 1$$
- Case 2:
$$\mathcal{R}_\prime = \frac{(0.2)(0.05)}{(0.5+0.05)(0.05+0.05)} = 0.1\bar{8} < 1$$
- Case 3:
$$\mathcal{R}_\prime = \frac{(0.2)(0.05)}{(0.75+0.05)(0.05+0.05)} = 0.125 < 1$$
- Case 4:
$$\mathcal{R}_\prime = \frac{(0.2)(0.05)}{(0+0.05)(0.05+0.05)} = 2 > 1$$

For the first three cases, $\mathcal{R}_\prime < 1$, the disease will eventually die over time. That is, S_t and I_t will approach the disease disease-free equilibrium as $t \to \infty$.

Meanwhile, if there is no vaccination ($p = 0$), $\mathcal{R}_\prime > 1$. The disease will persist, that is, S_t and I_t will approach the endemic equilibrium as $t \to \infty$.

This is shown in the succeeding graphs.

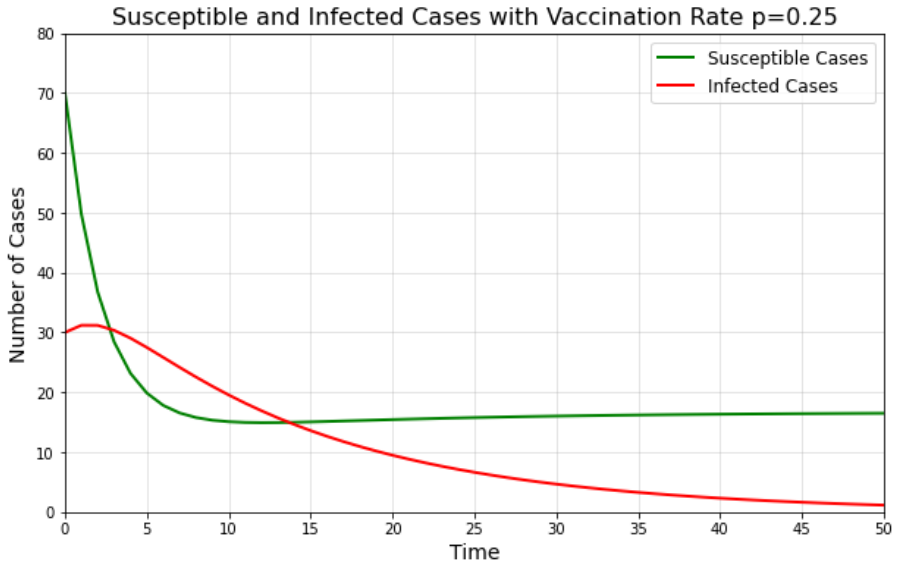

Figure 2: Susceptible and Infected Cases with Vaccination Rate p=0.25

```
import numpy as np
import matplotlib.pyplot as plt

T = 50  # Time steps from 0 to 50
index_set = range(T + 1)
S = np.zeros(len(index_set))
I = np.zeros(len(index_set))

N = 100  # Total population
beta = 0.2  # Transmission rate
b = 0.05  # Birth/Death rate
gamma = 0.05  # Recovery rate
p = 0.25  # Vaccination rate

# Initial values
S[0] = 70
I[0] = 30

# Iterative calculations for S and I
for t in index_set[0:T]:  # Iterate up to T
    S[t+1] = (1 - p) * S[t] - (beta/N) * I[t] * S[t] + b * (N - S[t])
    I[t+1] = (beta/N) * I[t] * S[t] + (1 - b - gamma) * I[t]

# Plotting S and I
plt.figure(figsize=(10, 6))
plt.plot(index_set, S, color='green', linewidth=2, label="Susceptible Cases")
plt.plot(index_set, I, color='red', linewidth=2, label="Infected Cases")
plt.xlabel("Time", fontsize=14)
plt.ylabel("Number of Cases", fontsize=14)
plt.title(f"Susceptible and Infected Cases with Vaccination Rate p={p}", fontsize=16)
plt.grid(alpha=0.4)
plt.legend(fontsize=12)
plt.xticks(np.arange(0, T+1, 5))
plt.xlim(0, T)
plt.ylim(0, max(S.max(), I.max()) + 10)
plt.show()
```

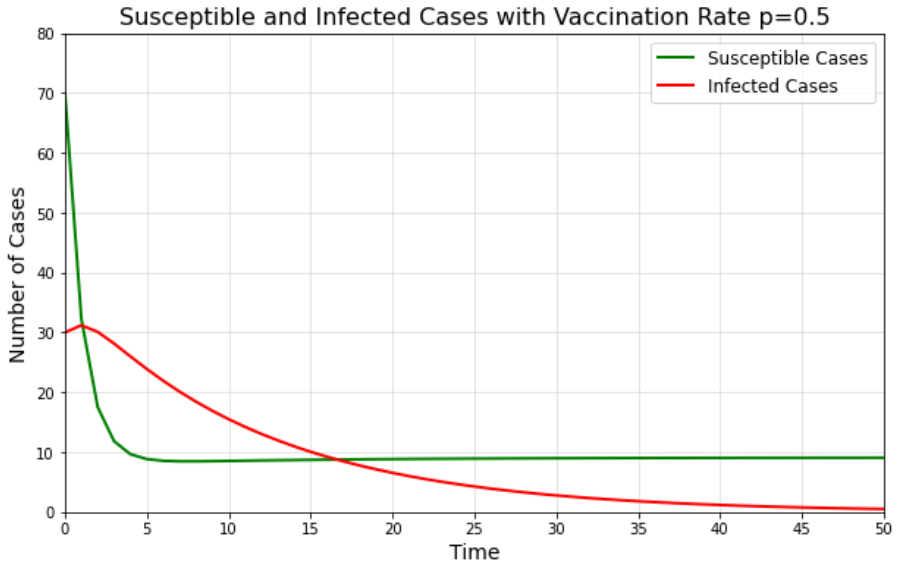

Figure 3: Susceptible and Infected Cases with Vaccination Rate p=0.50

```
import numpy as np
import matplotlib.pyplot as plt

T = 50  # Time steps from 0 to 50
index_set = range(T + 1)
S = np.zeros(len(index_set))
I = np.zeros(len(index_set))

N = 100  # Total population
beta = 0.2  # Transmission rate
b = 0.05  # Birth/Death rate
gamma = 0.05  # Recovery rate
p = 0.50  # Vaccination rate

# Initial values
S[0] = 70
I[0] = 30

# Iterative calculations for S and I
for t in index_set[0:T]:  # Iterate up to T
    S[t+1] = (1 - p) * S[t] - (beta/N) * I[t] * S[t] + b * (N - S[t])
    I[t+1] = (beta/N) * I[t] * S[t] + (1 - b - gamma) * I[t]

# Plotting S and I
plt.figure(figsize=(10, 6))
plt.plot(index_set, S, color='green', linewidth=2, label="Susceptible Cases")
plt.plot(index_set, I, color='red', linewidth=2, label="Infected Cases")
plt.xlabel("Time", fontsize=14)
plt.ylabel("Number of Cases", fontsize=14)
plt.title(f"Susceptible and Infected Cases with Vaccination Rate p={p}", fontsize=16)
plt.grid(alpha=0.4)
plt.legend(fontsize=12)
plt.xticks(np.arange(0, T+1, 5))
plt.xlim(0, T)
plt.ylim(0, max(S.max(), I.max()) + 10)
plt.show()
```

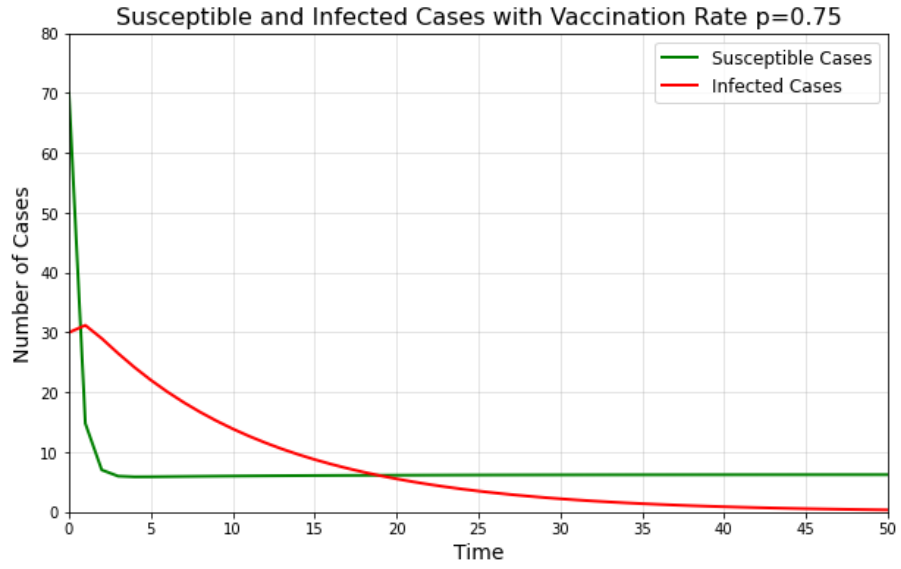

Figure 4: Susceptible and Infected Cases with Vaccination Rate p=0.75

```
import numpy as np
import matplotlib.pyplot as plt

T = 50  # Time steps from 0 to 50
index_set = range(T + 1)
S = np.zeros(len(index_set))
I = np.zeros(len(index_set))

N = 100  # Total population
beta = 0.2  # Transmission rate
b = 0.05  # Birth/Death rate
gamma = 0.05  # Recovery rate
p = 0.75  # Vaccination rate

# Initial values
S[0] = 70
I[0] = 30

# Iterative calculations for S and I
for t in index_set[0:T]:  # Iterate up to T
    S[t+1] = (1 - p) * S[t] - (beta/N) * I[t] * S[t] + b * (N - S[t])
    I[t+1] = (beta/N) * I[t] * S[t] + (1 - b - gamma) * I[t]

# Plotting S and I
plt.figure(figsize=(10, 6))
plt.plot(index_set, S, color='green', linewidth=2, label="Susceptible Cases")
plt.plot(index_set, I, color='red', linewidth=2, label="Infected Cases")
plt.xlabel("Time", fontsize=14)
plt.ylabel("Number of Cases", fontsize=14)
plt.title(f"Susceptible and Infected Cases with Vaccination Rate p={p}", fontsize=16)
plt.grid(alpha=0.4)
plt.legend(fontsize=12)
plt.xticks(np.arange(0, T+1, 5))
plt.xlim(0, T)
plt.ylim(0, max(S.max(), I.max()) + 10)
plt.show()
```

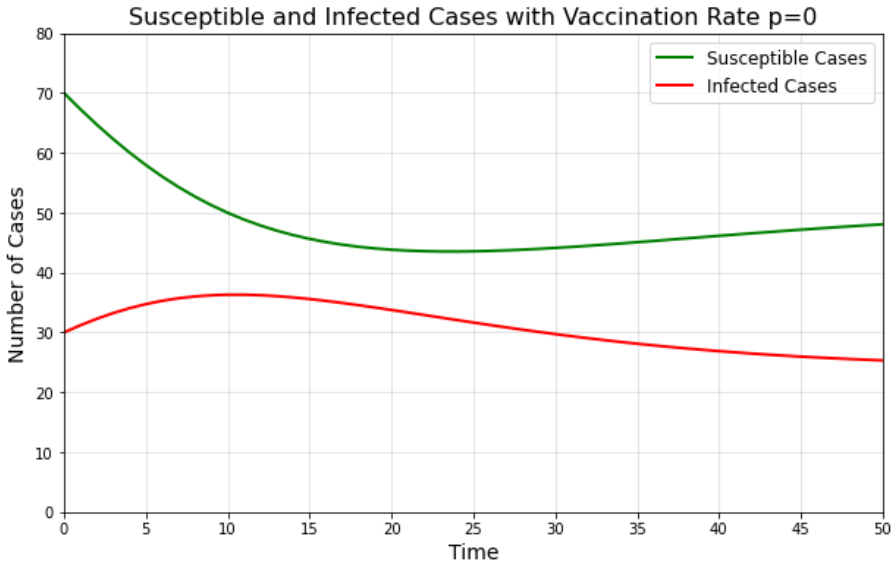

Figure 5: Susceptible and Infected Cases with Vaccination Rate p=0

```
import numpy as np
import matplotlib.pyplot as plt

T = 50  # Time steps from 0 to 50
index_set = range(T + 1)
S = np.zeros(len(index_set))
I = np.zeros(len(index_set))

N = 100  # Total population
beta = 0.2  # Transmission rate
b = 0.05  # Birth/Death rate
gamma = 0.05  # Recovery rate
p = 0  # Vaccination rate

# Initial values
S[0] = 70
I[0] = 30

# Iterative calculations for S and I
for t in index_set[0:T]:  # Iterate up to T
    S[t+1] = (1 - p) * S[t] - (beta/N) * I[t] * S[t] + b * (N - S[t])
    I[t+1] = (beta/N) * I[t] * S[t] + (1 - b - gamma) * I[t]

# Plotting S and I
plt.figure(figsize=(10, 6))
plt.plot(index_set, S, color='green', linewidth=2, label="Susceptible Cases")
plt.plot(index_set, I, color='red', linewidth=2, label="Infected Cases")
plt.xlabel("Time", fontsize=14)
plt.ylabel("Number of Cases", fontsize=14)
plt.title(f"Susceptible and Infected Cases with Vaccination Rate p={p}", fontsize=16)
plt.grid(alpha=0.4)
plt.legend(fontsize=12)
plt.xticks(np.arange(0, T+1, 5))
plt.xlim(0, T)
plt.ylim(0, max(S.max(), I.max()) + 10)
plt.show()
```

4 Summary and Conclusion

This study explores the SIR (Susceptible-Infected-Recovered) model given by the following system of difference equations, focusing on equilibrium solutions and their stability.

$$\begin{cases} S_{t+1} = (1-p)S_t - \dfrac{\beta}{N} I_t S_t + b(R_t + I_t) \\ I_{t+1} = \dfrac{\beta}{N} I_t S_t + (1-b-\gamma) I_t \\ R_{t+1} = (1-b)R_t + \gamma I_t + pS_t \end{cases}$$

By incorporating parameters such as contact rate (β), probability of recovery (γ),proportion of vaccinated individuals (p), probability of birth (b), the derived equations reveal two equilibrium solutions:

- Disease-free equilibrium:

$$(\bar{S}, \bar{I}) = \left(\frac{bN}{p+b}, 0 \right)$$

- Endemic Equilibrium:

$$(\bar{S}, \bar{I}) = \left(\frac{(b+\gamma)N}{\beta}, bN \left(\frac{\beta - (b+\gamma)}{\beta(b+\gamma)} \right) - \frac{pN}{\beta} \right)$$

Additionally, the calculation of the basic reproduction number, $\mathcal{R}_\prime$, that is the average number of secondary infections caused by a single infectious individual given by:

$$\mathcal{R}_\prime = \frac{\beta b}{(p+b)(b+\gamma)}$$

provides valuable insights into the dynamics of disease transmission under different vaccination scenarios.

In the first three cases, where $\mathcal{R}_\prime < 1$, the disease cannot sustain itself within the population, leading to its eventual eradication. This outcome demonstrates that with sufficient vaccination coverage, the system will stabilize at a disease-free equilibrium, where the susceptible (S_t) and infected (I_t) populations approach steady-state values over time without the presence of the infection.

Moreover, as the vaccination rate (p) increases, $\mathcal{R}_\prime$ decreases further, highlighting the direct and proportional relationship between vaccination coverage and the reduction in disease transmission.

Conversely, where vaccination is absent ($p = 0$), $\mathcal{R}_\prime > 1$, indicating that the disease will persist within the population. Under these conditions, the system stabilizes at an endemic equilibrium, where the infection remains present over time.

This result emphasizes the critical role of vaccination in controlling infectious diseases and underscores the threshold effect of $\mathcal{R}_\prime = 1$ as a tipping point between disease eradication and persistence.

Overall, the findings validate the importance of vaccination programs in reducing $\mathcal{R}_\prime$ below 1 to achieve a disease-free state. They also highlight that insufficient vaccination coverage allows the disease to persist, posing a continuous public health threat. These results provide a mathematical basis for setting vaccination targets and reinforce the necessity of sustained efforts to maintain high vaccination rates to prevent outbreaks and ensure long-term population health.

References

[1] T Liu, J Huang, Z He, Y Zhang, N Yan, CJP Zhang, and WK Ming. A real-world data validation of the value of early-stage sir modelling to public health. sci. rep. 2023, 13, 9164.

[2] Omar Melikechi, Alexander L Young, Tao Tang, Trevor Bowman, David Dunson, and James Johndrow. Limits of epidemic prediction using sir models. *Journal of Mathematical Biology*, 85(4):36, 2022.

[3] Jasmin Nunuvero, Angelique Santiago, Moshe Cohen, and Anca Radulescu. Modeling the effects of adherence to vaccination and health protocols in epidemic dynamics by means of an sir model. *arXiv preprint arXiv:2308.01038*, 2023.