UNIVERSITY OF AMSTERDAM

# REPORT

## Mathematical Modeling of Infectious Diseases: SIR, SEIR and Vaccination Strategies

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# Contents

# 1 Introduction

Infectious diseases have been present in the human world for centuries [1]. Currently, despite the major advances in vaccination programs, antibiotics, and global surveillance systems, emerging and reemerging infectious diseases pose a significant problem around the world. In recent years, a growing range of factors have contributed to the faster and wider spread of an infection when an outbreak occurs [6]. These factors include, but are not limited to, growth of the global population and its density, worldwide migration, greater accessibility of travel, and global climate changes. An outbreak is characterized by a sudden increase in infectious cases above the endemic level (that is, an expected baseline), and when such an outbreak spreads further to a larger geographical area, it is considered an epidemic [9]. When uncontrolled, an epidemic can further spread globally and be considered a pandemic. Examples of pandemics are the 1918 Spanish influenza, the 2003 severe acute respiratory syndrome (SARS), and most recently, the 2019 Covid-19 pandemic [7]. Pandemics have far-reaching consequences for societies, ranging from economic, social, health and psychological risks [3,5]. Epidemics and pandemics are highly complex phenomena, and the mechanisms underlying sudden outbreaks and their further stages are not always fully understood. Given their profound impact on the society and public health, is it crucial for policymakers to understand the dynamical mechanisms underlying the spread of an infectious disease, to design strategies that effectively prevent the outbreaks and their further escalations.

To advance such knowledge, mathematical models and advanced computational tools are used to uncover mechanisms underlying the dynamics of an infectious disease [11], such as conditions for an outbreak escalation, projected case numbers or thresholds for effective vaccination programs. One of the most widely used models for epidemiological modeling is the SIR model (susceptible-infected-recovered). In the previous research, SIR has been used in studying vaccination strategies in epidemics that show seasonal variations [12]. It has also been used in Covid-19 pandemic research [2, 8]. In addition to modeling infectious diseases, the SIR model can be applied to other domains than epidemiology, such as online social networks or diffusion of ideas [13].

## 1.1 Classic SIR Model

We simulated the classic SIR model under different initial and parameter conditions to study the outbreak dynamics and the long-term behavior of the system. We applied this model to the influenza outbreak data from a boys' school and estimated the model parameters. Lastly, we performed simulation experiments to test two different vaccination strategies on the fitted model to assess their effectiveness.

## 1.2 SIR Models with Demography

Next, we included natural birth and death rates to the classic SIR model and studied the system's long-term behavior. Further, we added the infection-induced mortality to study the dynamics when the population is no longer constant. We performed a Fourier transform to quantify the oscillatory behavior of the infectious class $I$.

## 1.3 SEIR Model

Lastly, we explored the dynamics of the SEIR Model (susceptible–exposed–infectious–recovered). To this model, we added seasonal effects in the form of temporal forcing to influence the transmission rate.

This report is organized as follows: **Section 2: Theory** introduces the SIR model and its extensions, the SEIR model, temporal forcing and Fourier transform. **Section 3: Numerical**

**Methods** explains the numerical methods and their implementation in this study. **Section 4: Results** presents the findings and discusses their meaning and interpretation. Finally, **Section 5: Conclusions** concludes the work and and discusses the key findings.

# 2 Theory

## 2.1 Classic SIR Model

The SIR model is a compartmental model used to describe the temporal dynamics of the spread of an infectious disease in a population [10]. In the SIR model and its derivatives, the sequence of letters demonstrates the flow of individuals between compartments. We define the compartments $X$, $Y$, $Z$ to represent the following:

- $X$: Number of people in the susceptible class. When a susceptible individual contracts the disease, they transition to the Infectious class.
- $Y$: Number of individuals who are currently infectious. Infectious individuals transmit the disease to susceptibles. When infectious individuals recover, they move to the Recovered class.
- $Z$: Number of people who have recovered and developed immunity to the disease.

In the classic SIR model, we make the following assumptions:

- Once individuals recover from the disease, they develop lifelong immunity and cannot contract the disease again.
- The population, $N$, is constant. Therefore, $X + Y + Z = N$.

Two key parameters determine the dynamics of the model:

- $\beta$: the transmission rate, which represents the rate at which infectious individuals spread the disease per unit of time.
- $\gamma$: the recovery rate, which represents the rate at which infectious individuals recover and move to the recovered compartment.

The system of ordinary differential equations describing the classic SIR model is given by:

$$\frac{dX}{dt} = -\frac{\beta XY}{N} \tag{1}$$
$$\frac{dY}{dt} = \frac{\beta XY}{N} - \gamma Y \tag{2}$$
$$\frac{dZ}{dt} = \gamma Y \tag{3}$$

Since the population $N$ is constant, we can express the SIR model in terms of fractions instead of numbers of individuals. Let:

$$S = \frac{X}{N}, \quad I = \frac{Y}{N}, \quad R = \frac{Z}{N}.$$

Then, we have the new fractional compartments that satisfy S + I + R = 1:

- $S$: The fraction of susceptible individuals.

- $I$: The fraction of infectious individuals.
- $R$: The fraction of recovered individuals.

The system of ordinary differential equations becomes:

$$\frac{dS}{dt} = -\beta SI \tag{4}$$
$$\frac{dI}{dt} = \beta SI - \gamma I \tag{5}$$
$$\frac{dR}{dt} = \gamma I \tag{6}$$

A fundamental quantity derived from the parameters of this model is the basic reproductive ratio $R_0$, defined as:

$$R_0 = \frac{\beta}{\gamma},$$

which represents the average number of secondary infectious cases produced by a single infectious individual in an entirely susceptible population. If $R_0 > 1$, the infection will spread in the population, potentially leading to an epidemic. However, if $R_0 < 1$, an infection will eventually die out.

## 2.2 SIR Models with Demography

### 2.2.1 Natural Mortality

In the classic SIR model, only two mechanisms are involved in the system dynamics – infection and recovery. The extension of the classic SIR model includes the addition of the demographical variables that represent birth and natural death of individuals in the population. A simplifying assumption is made that the birth and death rates in a population are equal. Thus, the population $N$ stays constant. We define:

- $\mu$: the rate at which new susceptibles are born, and at which individuals (in any epidemiological compartment) suffer natural mortality.

This extended SIR model with demography is described by the following ordinary differential equations:

$$\frac{dS}{dt} = \mu - \beta SI - \mu S \tag{7}$$
$$\frac{dI}{dt} = \beta SI - \gamma I - \mu I \tag{8}$$
$$\frac{dR}{dt} = \gamma I - \mu R \tag{9}$$

The expression for the basic reproductive ratio $R_0$ for this model is:

$$R_0 = \frac{\beta}{\gamma + \mu},$$

which corresponds to the ratio of new disease transmissions over the length of time an individual spends in the infectious compartment.

By setting the derivatives (7), (8), (9) to zero, we obtain the following equilibrium state:

$$(S^*, I^*, R^*) = \left(\frac{1}{R_0}, \frac{\mu}{\beta}(R_0 - 1), 1 - \frac{1}{R_0} - \frac{\mu}{\beta}(R_0 - 1)\right). \tag{10}$$

### 2.2.2 Infection-induced Mortality

An extension of the previously described SIR model with demography is a model in which we include an infection-induced mortality factor $\rho$:

- $\rho$: probability of an individual in the $I$ class dying from the infection before either recovering or dying from natural causes.

In the previous SIR models, the population $N$ was constant, and therefore the variables $S$, $I$, and $R$ could be rescaled to represent the fractions of the population. However, with the addition of an extra loss term to the Infectious compartment only, the population $N$ is no longer constant. Thus, the compartments are now given in terms of the absolute number of individuals in each class:

- $X$: The number of susceptible individuals.
- $Y$: The number of infectious individuals.
- $Z$: The number of recovered individuals.

Accordingly, the total population at time $t$ is given by:

$$X(t) + Y(t) + Z(t) = N(t)$$

The system of ordinary differential equations describing this SIR model with infection-induced mortality is given by:

$$\frac{dX}{dt} = \mu N - \frac{\beta XY}{N} - \mu X \tag{11}$$

$$\frac{dY}{dt} = \frac{\beta XY}{N} - \frac{\gamma + \mu}{1 - \rho} Y \tag{12}$$

$$\frac{dZ}{dt} = \gamma Y - \mu Z \tag{13}$$

## 2.3 SEIR Model

The SEIR model extends the classic SIR model by introducing an additional compartment $E$, which represents the fraction of individuals who have been exposed to the pathogen but are not infectious yet. This latent period reflects the time when the pathogen is present in the host but not yet abundant enough to transmit to others. In this model, we assume that $S+E+I+R = 1$, so the total population $N$ is constant. The compartments are defined:

- $S$: The fraction of susceptible individuals.
- $E$: The fraction of exposed individuals.
- $I$: The fraction of infectious individuals.
- $R$: The fraction of recovered individuals.

The average duration of the latent period is given by $1/\sigma$. The SEIR model with natural mortality $\mu$ is described by the following system of ordinary differential equations:

$$\frac{dS}{dt} = \mu - (\beta I + \mu)S \tag{14}$$
$$\frac{dE}{dt} = \beta SI - (\mu + \sigma)E \tag{15}$$
$$\frac{dI}{dt} = \sigma E - (\mu + \gamma)I \tag{16}$$
$$\frac{dR}{dt} = \gamma I - \mu R \tag{17}$$

The endemic fixed point of the system of the equations (14), (15), (16), (17) is:

$$S^* = \frac{(\mu + \gamma)(\mu + \sigma)}{\beta\sigma} = \frac{1}{R_0}, \tag{18}$$
$$E^* = \frac{\mu(\mu + \gamma)}{\beta\sigma}(R_0 - 1), \tag{19}$$
$$I^* = \frac{\mu}{\beta}(R_0 - 1), \tag{20}$$

with $R^* = 1 - S^* + E^* + I^*$.

### 2.4 Temporal Forcing for SEIR

For many infectious diseases, the transmission rate is affected by external seasonal factors, such as school terms or weather patterns. These fluctuations can lead to substantially different infection dynamics depending on the time of year. Temporal forcing captures this seasonality by allowing the transmission rate to vary with time, making the model more realistic for pathogens with seasonal patterns.

The constant transmission rate $\beta$ is replaced with a time-dependent rate:

$$\beta(t) = \beta_0\big[1 + \beta_1 \cos(\omega t)\big],$$

where:

- $\beta_0$ is the average transmission rate,
- $\beta_1$ is the amplitude of seasonal variation and $0 < \beta_1 < 1$,
- $\omega = \frac{2\pi}{T}$ is the angular frequency of forcing, with $T$ representing the period of the seasonal cycle.

The SEIR equations with temporal forcing are then:

$$\frac{dS}{dt} = \mu - (\beta(t)I + \mu)S \tag{21}$$
$$\frac{dE}{dt} = \beta(t)SI - (\mu + \sigma)E \tag{22}$$
$$\frac{dI}{dt} = \sigma E - (\gamma + \mu)I \tag{23}$$
$$\frac{dR}{dt} = \gamma I - \mu R \tag{24}$$

The basic reproductive ratio for SEIR model with temporal forcing [15], defined in terms of the average transmission rate, is:

$$R_0 = \frac{\beta_0\,\sigma}{(\sigma+\mu)(\gamma+\mu)}.$$

### 2.5 Discrete Fourier Transform (DFT)

The Discrete Fourier Transform (DFT) is a numerical method used to analyze the frequency content of a discrete time series [14]. Given the values $x[n]$ taken at equally spaced time intervals $\Delta t$, the DFT transforms the signal into the sum of sinusoidal components:

$$X[k] \triangleq \sum_{n=0}^{N-1} x[n]\, e^{-j\frac{2\pi}{N}kn}, \quad k = 0, 1, \ldots, N-1,$$

where:

- $N$ is the total number of data points,
- $n$ is the current sample index,
- $k$ is the frequency index, with $k \in [0, N-1]$,
- $x[n]$ is the value at sample $n$.

## 3 Numerical methods

The dynamics of the SIR model and its extensions were studied using numerical integration of the systems of ordinary differential equations (ODEs). All simulations were performed in Python using the `solve_ivp` function from SciPy. To assess the oscillatory dynamics in the SIR model with natural mortality, we applied Fourier transformation using the Fast Fourier Transform (FFT) from the `fftpack` module in SciPy.

### 3.1 General Approach to SIR Models

The explicit Runge–Kutta method of order 5(4) (RK45), which is the default for the `solve_ivp`, was used for general simulations of the SIR model and its extensions:

```
sol = solve_ivp(
    sir_model, t_span, z0, # z0 = [s0,i0,r0]
    args=(beta, gamma),    # params
    t_eval=t_eval          # eval points
)
```

Here, the initial conditions (`z0`) and model parameters (`beta`, `gamma`) were passed as arguments, with solutions evaluated at uniformly spaced points `t_eval`.

### 3.2 Damped Oscillations

To capture oscillatory dynamics in the SIR model with demographics (natural mortality), the same RK45 method was used with stricter tolerances:

```
sol2 = solve_ivp(
    sir_with_demo,         # model
    t_span=t_span,         # time span
    y0=z0,                 # z0 = [s0, i0, r0]
    args=(B, y, u),        # params
```

```
    t_eval=t_eval,          # eval points
    method='RK45',
    rtol=1e-6, atol=1e-8   # tolerances
)
```

Parameters: $\beta$ (transmission rate), $\gamma$ (recovery rate), $\mu$ (natural mortality and birth rate). Initial conditions: $z_0 = [S_0, I_0, R_0]$, where $S_0 = 0.1$, $I_0 = 2.5 \times 10^{-4}$, and $R_0 = 1 - S_0 - I_0$. Time span: 0–60 years, with 1000 evaluation points.

### 3.3 Fourier Analysis

To quantify oscillatory behavior of the system, the infectious class time series $I(t)$ was analyzed using the Fast Fourier Transform (FFT), which was performed using the SciPy `fftpack`. The dominant frequency and amplitude were identified from the peak of the positive-frequency spectrum.

## 4 Results and discussion

### 4.1 Numerical Integration of the SIR Model

We performed numerical integration of the classic SIR model with two sets of parameters, resulting in two distinct system dynamics: without an epidemic, and with an epidemic. The parameter that determines whether a system will evolve into an epidemic is $R_0$, according to the theory presented in Section 2.1. The evolution of each compartment over time is shown in Figure 1. Additionally, the phase space diagrams for both scenarios are presented in Figure 2.

As seen in Figures 1 and 2, in the no-epidemic scenario, infected individuals cannot replace themselves, so the infection dies out quickly. In the epidemic scenario, the infectious class grows rapidly; however, after reaching a peak, it declines and eventually goes to 0. This decline occurs because the number of susceptible individuals decreases, causing each infectious individual to infect fewer than one new person on average. The phase space diagram shows that, in the epidemic scenario with $R_0 > 1$, the system approaches an equilibrium in which some or no susceptibles remain, but there are no infectious individuals in the population.

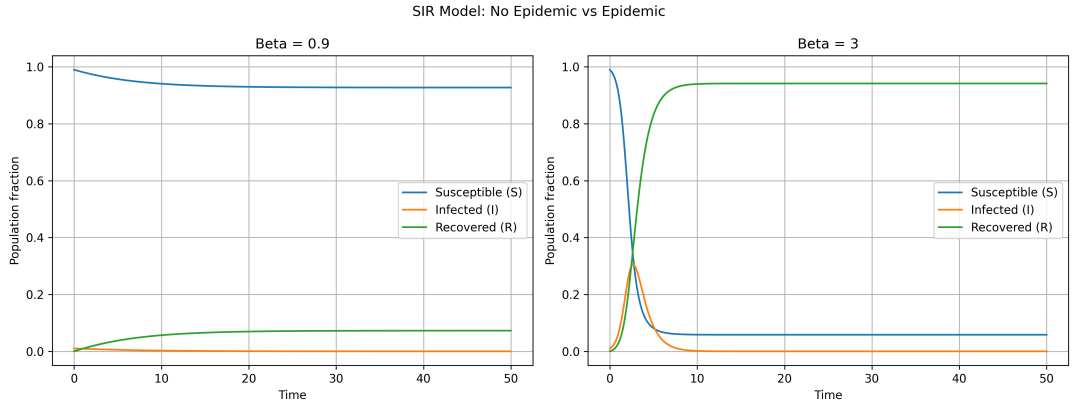


Figure 1: Time evolution of the SIR compartments for the two scenarios: no epidemic (left) and epidemic (right). Initial conditions for both scenarios were $i_0 = 0.01$, $s_0 = 0.99$, and $r_0 = 0$. The models were evaluated over a time span of 0–50 with 1000 equally spaced intervals.

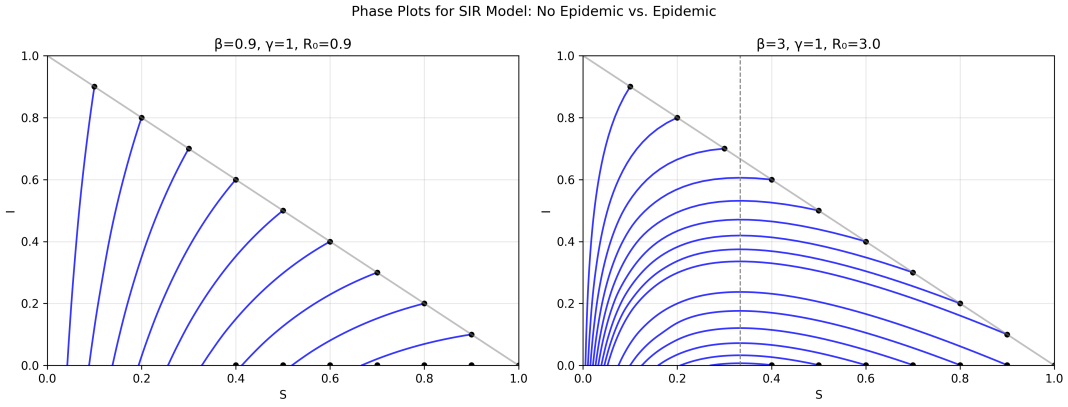


Figure 2: Phase space diagrams of the SIR model for the two scenarios: no epidemic (left) and epidemic (right). Initial conditions are indicated with dots. For both scenarios, points on the diagonal line satisfy $S + I = 1$, meaning there are no recovered individuals initially. On the horizontal line, the fraction of infected individuals is always 0.001.

### 4.2 Fitting the SIR Model

We fitted the classical SIR model to data from an influenza outbreak at a boys' school, presented in Table 1. The initial conditions were as follows: $i_0 = 1/763$, $s_0 = 762/763$, and $r_0 = 0$. Using these initial conditions, we obtained the following optimal parameters for the model fit: $\beta_{\text{opt}} = 1.6648$, $\gamma_{\text{opt}} = 0.4480$. The model fit is shown in Figure 3. As can be seen, the model follows the observed outbreak dynamics closely, capturing the rise and decline of the infected population.

| Day | 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Number | 1 | 3 | 8 | 28 | 75 | 221 | 291 | 255 | 235 | 190 | 125 | 70 | 28 | 12 | 5 |

Table 1: Influenza outbreak data from 14 days.

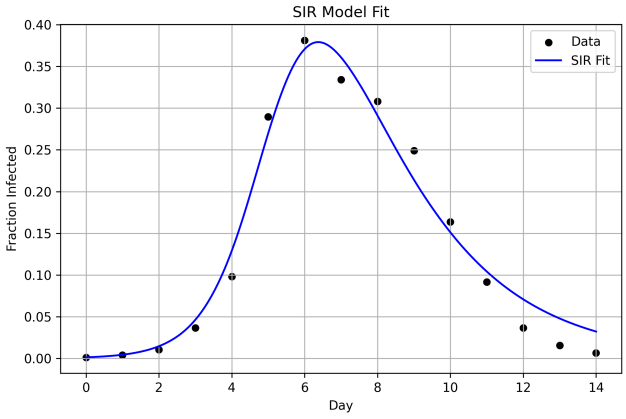


Figure 3: Model fit of the SIR model to the influenza outbreak data. The fitted curve closely follows the observed infection counts over time.

### 4.3 Vaccination Strategies

To explore potential vaccination strategies, we formulated two hypotheses:

1. Vaccinating approximately 25 individuals per day will prevent the epidemic.
2. Vaccinating half of the population within the first three days will prevent the epidemic.

The first strategy represents a realistic scenario with limited vaccination capacity, while the second strategy represents an aggressive, less realistic scenario designed to vaccinate a large portion of the population before the epidemic peak starts (before day 4). We simulated the system using the optimized parameters and applied both vaccination strategies. The results of these simulations are shown in Figure 4.

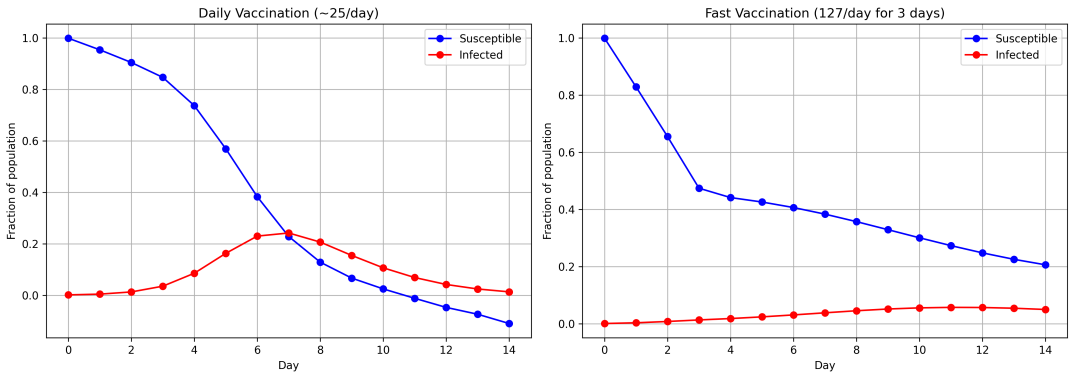


Figure 4: SIR model simulations with two vaccination strategies. Vaccination of 25 individuals per day (left). Vaccination of half the population within the first three days (right). The first strategy slows the epidemic but does not fully prevent it; the second strategy effectively prevents the epidemic.

### 4.4 SIR Models with Demography

We performed numerical integration of the SIR model with a natural birth and death rate $\mu$. The evolution of the fractional compartments over time is shown in Figure 5. Figure 6 presents the corresponding phase space diagrams. As can be seen from both figures, the system converges to a fixed point regardless of the initial conditions.

The damped oscillations of the infected fraction $I(t)$, along with the corresponding Fourier analysis showing the dominant frequency and amplitude, are presented in Figure 7. The dominant frequency of the oscillations is 0.416 1/yr, corresponding to a dominant period of 2.40 years, with an amplitude at the peak of 0.002. These results indicate that the oscillations are small and gradually decay over time.

### 4.5 Infection-induced Mortality

We numerically integrated the SIR model with demography including an infection-induced mortality parameter $\rho$. The results of three simulations with different values of $\rho$ are shown in Figure 8. As seen from the graphs, as $\rho$ approaches 1, the epidemic becomes less likely to occur. This occurs because infected individuals die rapidly due to the infection and therefore have less opportunity to transmit the disease to susceptible individuals.

### 4.6 SEIR Model

We numerically integrated the SEIR model under different parameter conditions to explore its diverse dynamic behaviors.

In the first scenario, the model exhibits a prolonged epidemic, characterized by a slower increase in the infectious cases. This scenario could represent an endemic state - after the

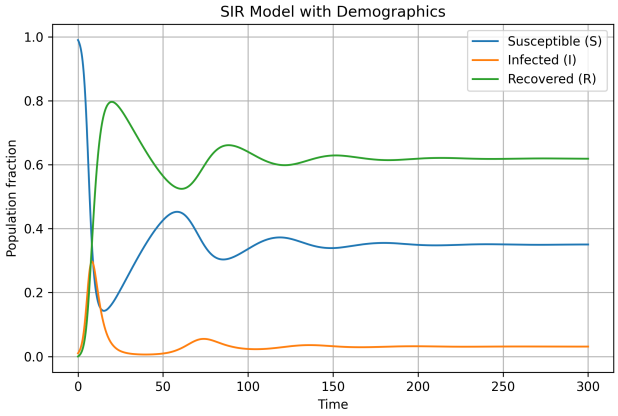


Figure 5: Time evolution of the SIR model with demography. Parameters: $\beta = 1$, $\gamma = 1/3$, $\mu = 1/60$.

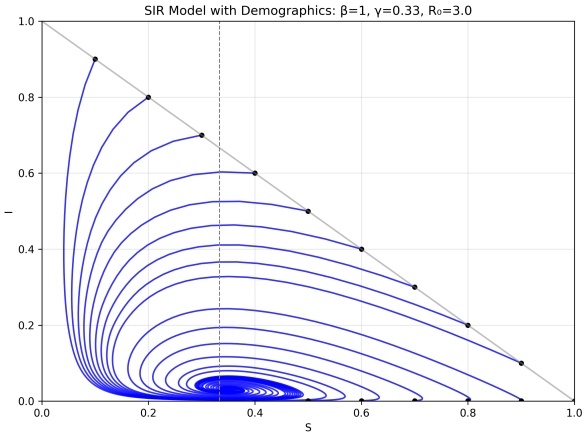


Figure 6: Phase space diagram of the SIR model with demography. The system converges to a fixed point regardless of initial conditions.

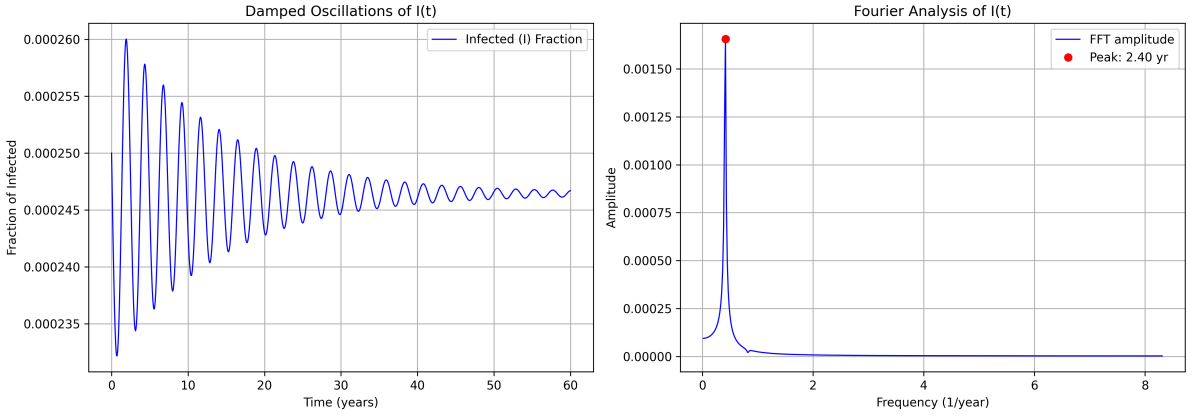


Figure 7: Damped oscillations of the infected fraction $I(t)$ (left). Fourier analysis showing the dominant frequency (0.416 1/yr), dominant period (2.40 years), and peak amplitude (0.002) (right).

outbreak, the fraction of infectious individuals stabilizes at around 0.1, meaning the disease remains present in the population.

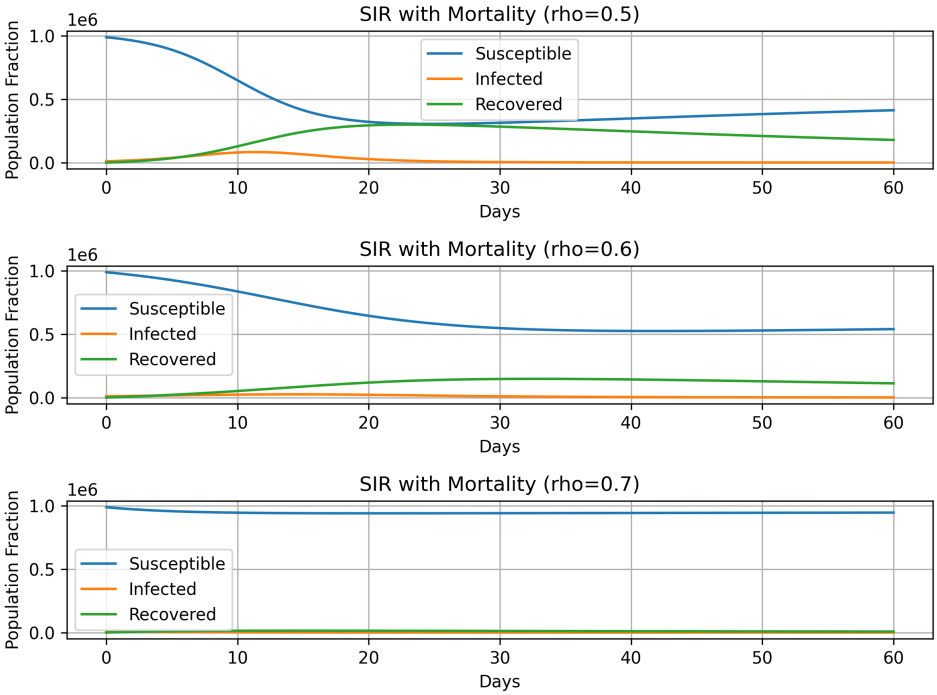


Figure 8: Time evolution of the SIR model with infection-induced mortality for three different values of $\rho$.

In the second scenario, a rapid and severe epidemic occurs because the basic reproductive rate is high ($R_0 = 3/0.2$). Most susceptibles become infected quickly, and individuals rapidly move to the recovered class. Although new susceptibles are continuously introduced through births, they are quickly infected, and recovered individuals also die at a comparable rate. As a result, the system stabilizes without significant accumulation of susceptible, infectious or recovered individuals, maintaining an approximate steady state.

In the third scenario with a moderate $R_0$ ($R_0 = 1/0.3$), infections spread slower than in the second scenario. The delay in transmission allows for small fluctuations in the number of susceptibles and infected individuals before the system approaches a stable equilibrium.

The results of these three scenarios are presented in Figure 9. Each subplot illustrates the time evolution of the Susceptible, Exposed, Infected, and Recovered compartments under the corresponding parameter conditions.

### 4.7 SEIR Model with Temporal Forcing

We extended the SEIR model by introducing temporal forcing in the transmission rate to account for seasonal differences. Numerical integration was performed with initial conditions $S_0 = 0.06$, $E_0 = 10^{-3}$, $I_0 = 10^{-3}$, $R_0 = 0$, parameters corresponding to a high basic reproduction number ($R_0 = 10$), and a period $T = 2.8$ years.

The results are shown in Figure 10. Temporal forcing results in seasonal outbursts of infected individuals, and periodic variations in all compartments.

## 5 Conclusions

In this study, we evaluated the classic SIR model and its several extensions, including vaccination, demographic effects and infection-induced mortality. Across all cases, the results confirmed the central theoretical role of the basic reproductive ratio $R_0$: when $R_0 < 1$, infections die out,

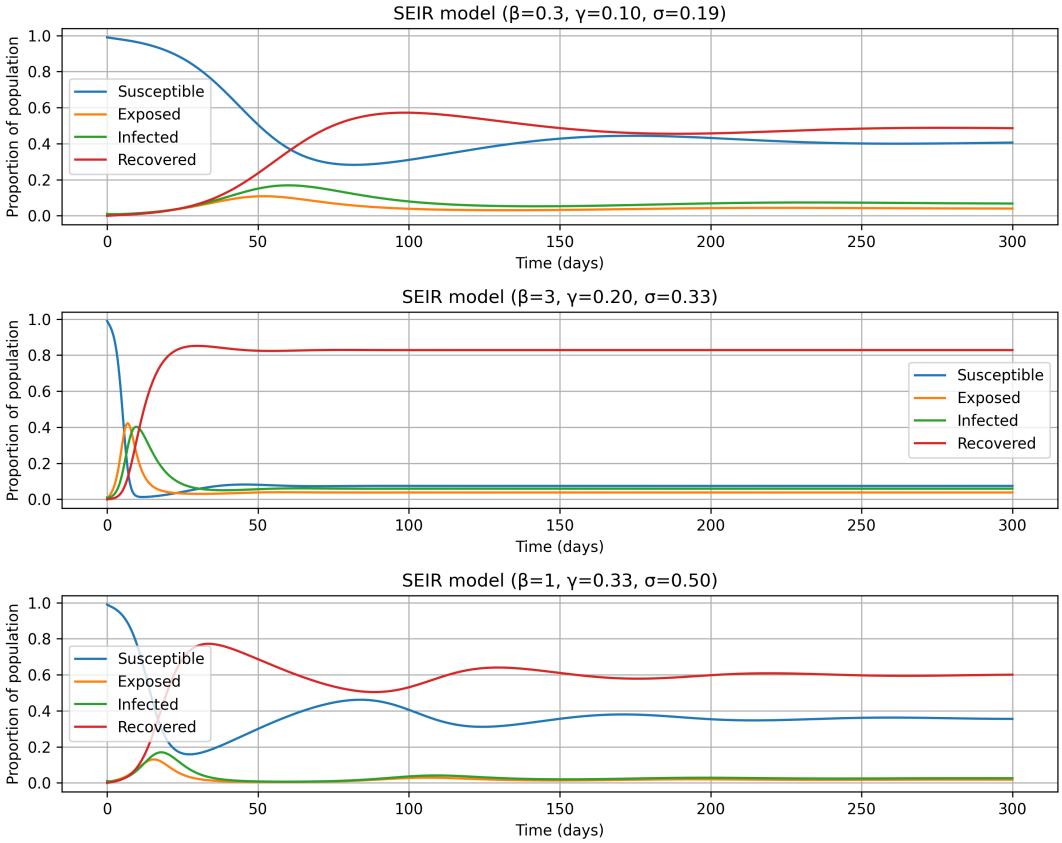


Figure 9: Time evolution of the SEIR model under three different parameter scenarios. Endemic state (top). Rapid, severe epidemic with high $R_0$ (center). Milder scenario with moderate $R_0$ (bottom).

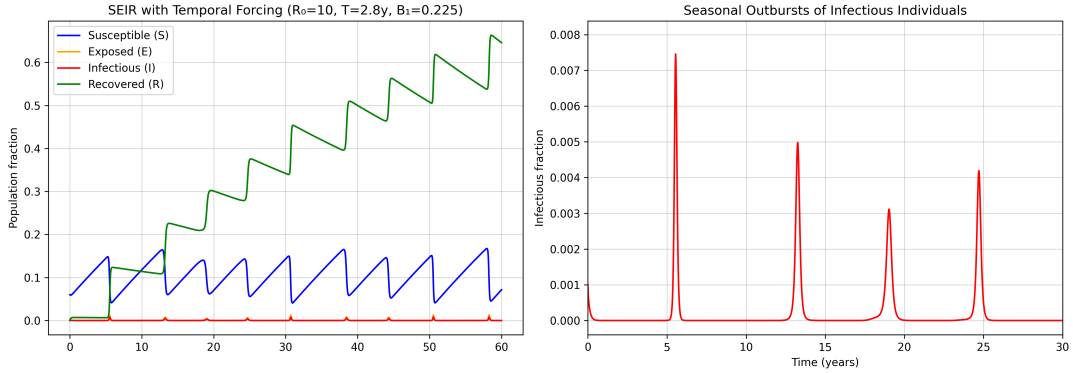


Figure 10: SEIR model with temporal forcing. Time evolution of all compartments (S, E, I, R) (left). Seasonal outbursts in infectious compartment $I(t)$ (right).

while with $R_0 > 1$, an epidemic can occur. Vaccination simulations showed that realistic daily vaccination rates slow but do not prevent epidemics, whereas rapid large-scale vaccination can suppress them entirely. Adding the birth and death rates showed that births can supply new susceptibles and therefore prolong infections, whereas deaths, especially together with infection-induced mortality, can limit the transmission rates. Lastly, we observed the dynamics of the SEIR model with various parameters, and showed how adding temporal forcing to the model results in seasonal outbursts of infections. Future work could apply stochastic effects and age-structured populations to the models to study their long-term dynamics and assess the effect

on vaccination strategies.

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