Assignment 1: SIR model (ODE)

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Abstract

This report introduces computational modeling of infectious diseases using the (extended) SIR model, which – despite its simplicity, can provide many insights into the spread and eradication approaches of various infectious diseases. We analyze the model and its dynamics for endemic and epidemic conditions, fit it to observed historical data, and investigate its dynamics.

1 Introduction

Computational Science deals with developing models that are useful for prediction and planning. Especially since the outbreak of the worldwide SARS Covid-2 pandemic, accurate models of infectious diseases have been of great interest. In this report, we use the simple, yet powerful SIR model for modeling the spread of infectious diseases. The latter model describes the the changes in susceptible (S), infected (I), and recovered (R) individuals of a population over time through the following set of coupled first order differential equations (ODE's):

$$\begin{aligned}\frac{\delta S}{\delta t} &= -\beta SI \\ \frac{\delta I}{\delta t} &= \beta SI - \gamma I \\ \frac{\delta R}{\delta t} &= \gamma I\end{aligned}$$

[KR11]

Individuals are initially susceptible and get infected at the rate of infection β and subsequently recover at the rate of recovery γ. Once recovered, they are assumed to be immune and no longer susceptible.

This report is organized as follows. In Section 2, we analyze the basic SIR model for states of an epidemic and no epidemic (2.1), fit it to observed historical data (2.2) and propose a vaccination plan (2.3). In Section 3, the use the extended demographic SIR model to include natural birth and death rates into the model and analyze its oscillatory behaviour (3.1) before then further adding an

infection induced mortality term to the model (3.2). Finally, Section 4 discusses a variation of the SIR model that takes latent periods and seasonality into account.

2 Numerical integration of the basic model

In the following, we will consider the basic SIR model without demographics (namely natural births and deaths) and use the frequency dependent model where S, I, R represent fractions of the total closed population $S + I + R = 1$.

2.1 Numerical integration

Despite its simplicity, the ordinal differential equations of the SIR model can not be solved analytically. Therefore, numerical integration using e.g. Euler's method is required. In order to numerically integrate, appropriate starting values (initial conditions) must be used. In Figures 2 and 1, we show the phase space diagram for states of and epidemic and no epidemic, respectively, with their parameters β, γ and initial conditions $S(0), I(0), R(0)$.

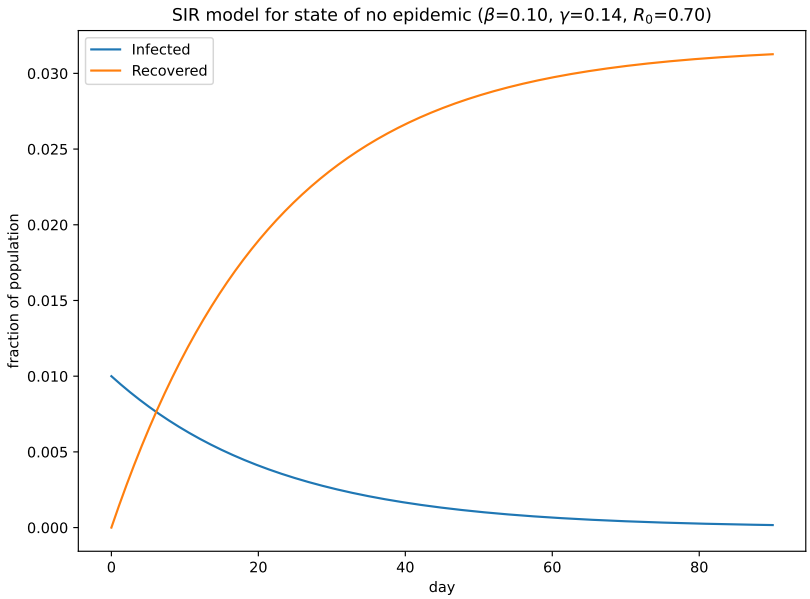


Figure 1: Phase space diagram for the state of no epidemic. $(I(0) = 0.01, S(0) = 0.99, R(0) = 0)$

The critical epidemiological parameter that determines if an infectious disease will invade and develop into an epidemic is the basic reproductive ratio $R_0 = \frac{\beta}{\gamma}$[KR11], because it defines the average number of secondary cases an average primary case produces in a totally susceptible population. By looking at the equation, one can immediately see that it describes the proportion of the infection rate and the recovery rate, such that the fraction of infected individuals of the population is increasing for $R_0 > 1$, decreasing for $R_0 < 1$, and constant (in a state of equilibrium) for $R_0 = 0$ (endemic). The

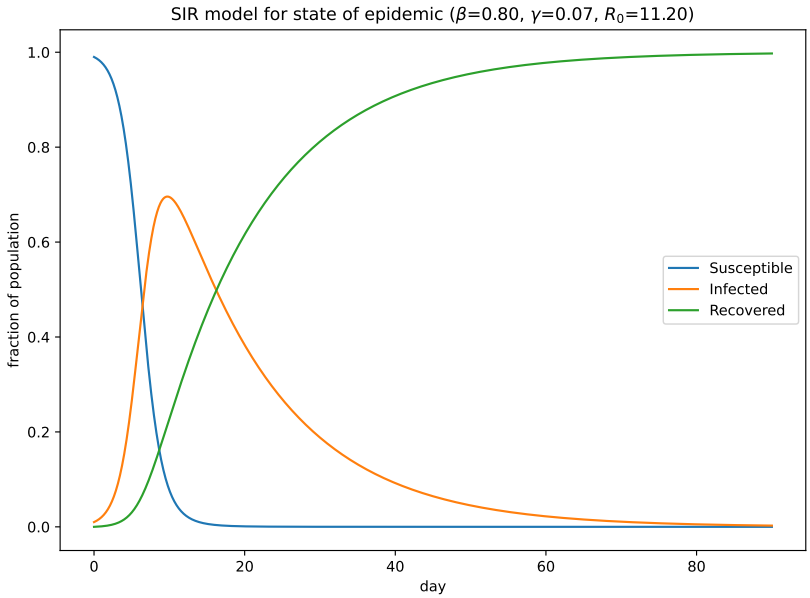


Figure 2: Phase space diagram for the state of an epidemic. $(I(0) = 0.01, S(0) = 0.99, R(0) = 0)$

parameter can also be used to determine the threshold level of vaccination required for eradication or the long-term proportion of susceptible individuals for an endemic.

This can be observed in Figure 1, which shows the phase space diagram for the state of no epidemic with $R_0 = 0.7 < 1$. In this case, the fraction of infected individuals is decreasing and the number of recovered (hence immune) individuals is increasing. We note that the fraction of susceptible individuals has been omitted for better readability as $S = 1 - R - I$.

On the other hand, Figure 2 shows the phase space diagram for the state of epidemic with $R_0 = 11.2 > 0$ due to increased infection rate ($\beta = 0.8$ compared to $\beta = 0.1$) and decreased recovery rate ($\gamma = 0.07$ compared to $\gamma = 0.14$). In this case, the fraction of infected individuals is rapidly increasing from $I(0) = 0.01$ to about $I(15) = 0.7$ of the population at its peak before decreasing.

2.2 Fitting the SIR model

We now continue to fit historical data from a case of an influenza outbreak in a boys school over a period of 3 weeks using the SIR model.

We again assume a total closed population $S + I + R = 1$. Given $N_{pop} = 763$ and the assumption that one infected boy started the epidemic, we choose $I(0) = \frac{1}{N_{pop}} = \frac{1}{763}$, $S(0) = 1.0 - \frac{1}{763}$ and $R(0) = 0$ as the initial conditions.

To fit an SIR model, we initially used a bounded differential evolution with bounds $[0, 2]$ to obtain an initial guess for β and γ with the mean squared error as an error measure. Subsequently, ordinary least squares was used to fit the curve, resulting in parameters $\beta = 1.55$ and $\gamma = 0.42$ ($R_0 = 3.72$).

Figure 3 shows the absolute number of infected boys $N_{pop}I$ together with the historical data

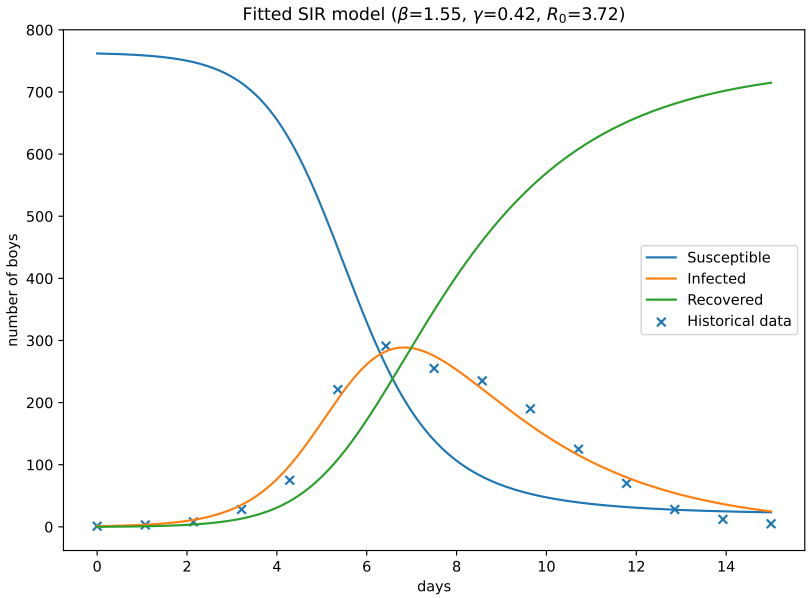


Figure 3: Phase space diagram for the SIR model fitted on historical data.
$(I(0) = \frac{1}{763}, S(0) = 1.0 - \frac{1}{763}, R(0) = 0)$

points. As one can see, the orange graphs very closely matches the historical data points and we conclude that this is a clear example of a (local) epidemic where on average, each infected individual caused $R_0 = 3.72$ secondary cases of infection. In the following section, a vaccination strategy to avoid such an epidemic outbreak will be discussed.

2.3 Vaccination Plan

Among other forms of infectious disease control such as quarantine or contact tracing, vaccination is one of the key strategies to avoid the outbreak of an epidemic, as it can provide long-lasting immunity to the infection, preventing both transmission and disease.

Under the assumption that vaccination offers lifelong protection, we propose to vaccinate a large proportion p of newborns. To model the effect that constant long-term vaccination of a fraction p of newborns against an infection with a basic reproductive ratio R_0 has, we replace the transmission rate β with $\beta(1-p)$ [KR11]. Note that when dealing with the vaccination of newborns it makes sense to add a birth (and death) rate μ to the model, which will be discussed in more detail in Section 3 and can be ignored for now.

This yields the following SIR model equations with vaccination:

$$\begin{aligned}\frac{\delta S}{\delta t} &= -\beta(1-p)SI + \mu - \mu S \\ \frac{\delta I}{\delta t} &= \beta(1-p)SI - \gamma I - \mu I \\ \frac{\delta R}{\delta t} &= \gamma I - \mu R\end{aligned}$$

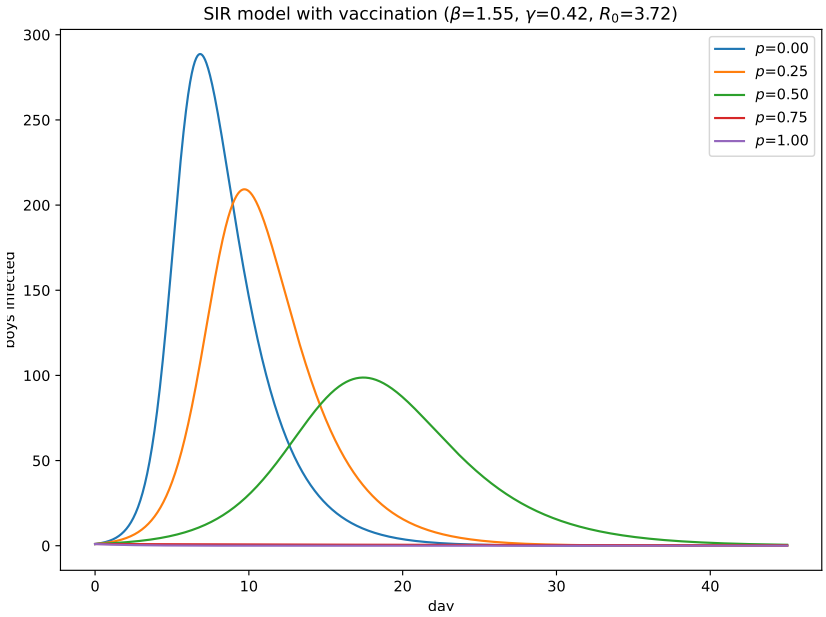


Figure 4: Number of infected boys for the SIR model for different vaccination proportions p.
$(I(0) = \frac{1}{763}, S(0) = 1.0 - \frac{1}{763}, R(0) = 0)$

In order to avoid an epidemic, one must ensure that $R'_0 = (1-p)R_0 < 1$, which gives rise to the critical proportion of newborns $p_c = 1 - \frac{1}{R_0}$ that need to vaccinated to avoid an epidemic [KR11]. Even if not all individuals have been vaccinated and $p_c < 1$, it is enough to establish what is commonly known as *herd immunity*.

Given the concrete case of the epidemic in the boys school discussed before, we know that $p_c = 1 - \frac{1}{R_0} = 1 - \frac{1}{3.72} = 0.73$ and therefore at least 73% of the 763 students would have to be vaccinated. If we can motivate at least 73% to get vaccinated, Figure 4 shows the success of our proposed vaccination strategy. In Figure 4, the same epidemic outbreak is plotted for different parameters of p using the same parameters otherwise. One can clearly see that for all $p < p_c = 0.73$ the disease can invade and develop into an epidemic. However, for $p = 0.75$ and $p = 1.0$, the disease is successfully eradicated within a very short time.

3 Demographic SIR

We will incorporate the parameter μ as the natural birth and death rate into the model. Adding a constant natural birth and death rate μ, we get the following set of ordinary differential equations (ODE's) for the demographic SIR model:

$$\begin{aligned}\frac{\delta S}{\delta t} &= -\beta SI + \mu - \mu S \\ \frac{\delta I}{\delta t} &= \beta SI - \gamma I - \mu I \\ \frac{\delta R}{\delta t} &= \gamma I - \mu R\end{aligned}$$

[KR11]

3.1 Oscillatory behaviour of the demographic SIR model

A property of the demographic SIR model is that it stabilizes in either the disease-free equilibrium state or the endemic equilibrium state. In the first one, the disease is eradicated, while in the second one, the fraction of infected individuals oscillates around, and eventually converges to its long-term limit.

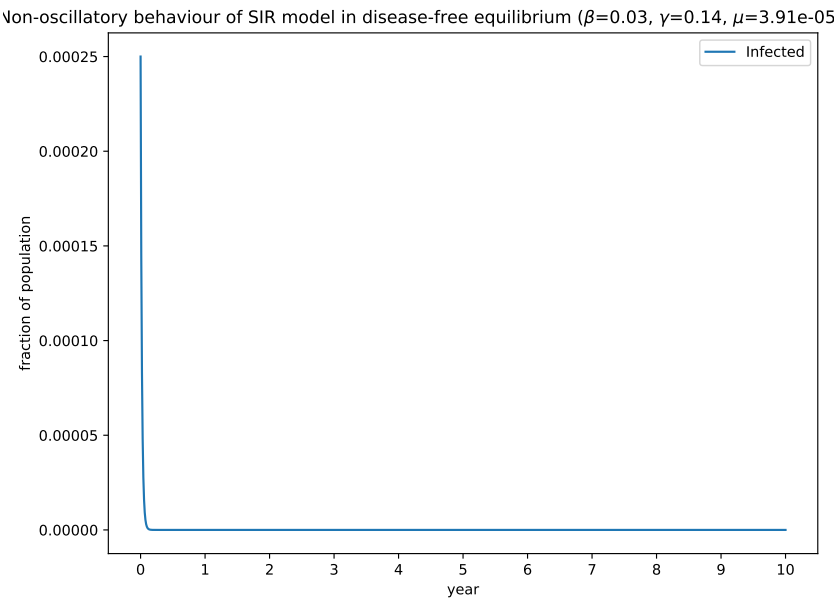


Figure 5: Non oscillatory behaviour for the demographic SIR model in disease-free equilibrium. $(I(0) = 2.5 \cdot 10^{-4}, S(0) = 0.1, R(0) = 1.0 - I(0) - S(0))$

A configuration of the disease-free equilibrium state is given in Figure 5, where no oscillatory behaviour can be observed and the disease is eradicated due to a low basic reproductive ratio $R_0 < 1$.

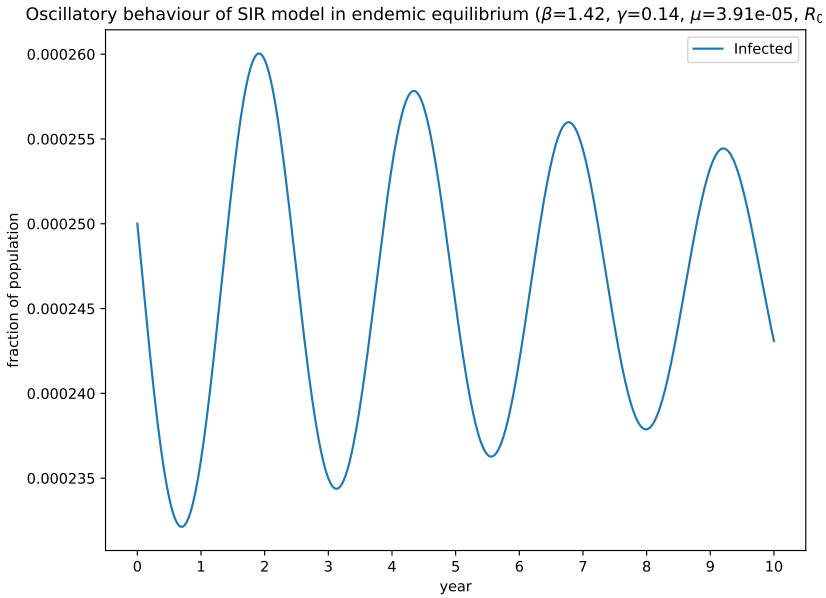


Figure 6: Harmonic oscillatory behaviour for the demographic SIR model in endemic equilibrium. $(I(0) = 2.5 \cdot 10^{-4}, S(0) = 0.1, R(0) = 1.0 - I(0) - S(0))$

In Figure 6 we give one sample configuration of an endemic equilibrium state where we can clearly see oscillatory behaviour around a median with decreasing amplitudes for every period. The oscillatory behaviour can always be observed for the endemic equilibrium, where the mean number of infected people of the population converges to some equilibrium $I > 0$. The requirement for an endemic equilibrium is that $R_0 = 1$. Recall that R_0 determines the number of secondary infections per index case in a naive population of susceptible individuals and is defined as $R_0 = \frac{\beta}{\gamma+\mu}$ for the demographic SIR model (assuming that the entire population is susceptible such that $S = 1 - I$) [KR11].

Furthermore, the period of oscillation T is then given by

$$T \sim 2\pi\sqrt{AG} \tag{1}$$

with $A = \frac{1}{\mu(R_0-1)}$ denoting the mean age of infection and $G = \frac{1}{\mu+\gamma}$ denoting the typical period of a host's infectivity [KR11].

For the configuration of the dynamical system shown Figure 6, we expect $T_{expected} \sim 2\pi\sqrt{AG} = 887.10$ days and frequency of oscillation $f_{expected} = \frac{1}{T_{expected}} = 0.00113$ from Equation 1. We will confirm this empirically by finding the frequencies and amplitudes of the oscillation in Figure 6 using Fourier analysis and compare it to the expected values.

Figure 8 shows the absolute amplitudes for the frequencies in the interval $(0, 0.014]$ after decomposition using real discrete Fourier analysis. We see that the dominant frequency of the oscillation signal is $f = 0.00110$ $(T = 912.50)$ with an amplitude of $\varphi_f = 1.93290$, which is close enough to the expected solutions and matches the graph of Figure 6.

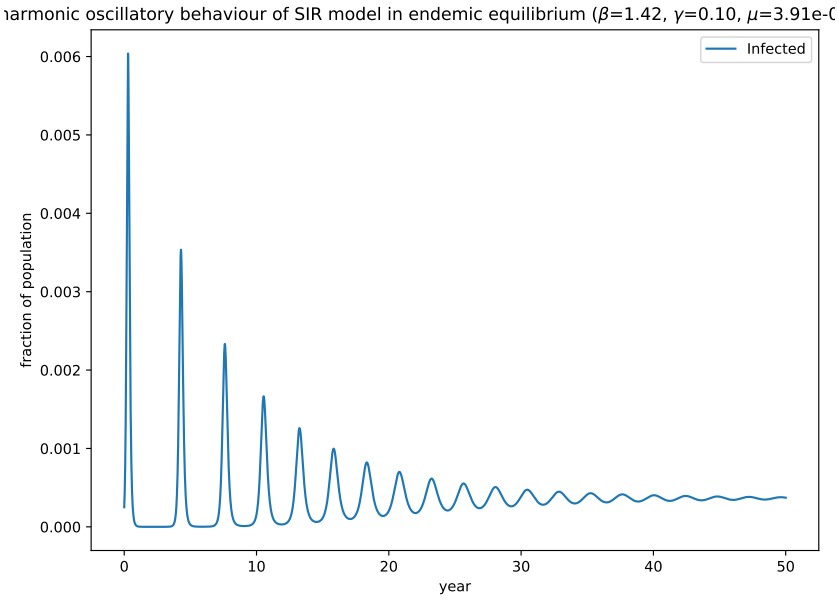


Figure 7: Non-harmonic oscillatory behaviour for the demographic SIR model in endemic equilibrium. $(I(0) = 2.5 \cdot 10^{-4}, S(0) = 0.1, R(0) = 1.0 - I(0) - S(0))$

Finally, we want to note that not all oscillations in the state of endemic equilibrium are perfectly harmonic, as the number of infected people can not be negative. Figure 7 shows such a configuration.

3.2 Infection induced mortality

We introduce ρ as probability of dying from the infection, which gives a new total rate of death

$$m = \frac{\rho}{1-\rho}(\gamma + \mu)$$

[KR11]

With rate m, the updated equation for $\frac{\delta I}{\delta t}$ are described by

$$\frac{\delta I}{\delta t} = \beta SI - (\gamma + \mu)I - \frac{\rho}{1-\rho}(\gamma + \mu)I$$

[KR11]

Analytically, one can already see that

$$\lim_{\rho \to 1} \frac{\rho}{1-\rho}(\gamma + \mu)I = \infty$$

for $\gamma + \mu > 0$ and $I > 0$, hence the number of infections will rapidly decrease as $\rho \to 1$.

Figure 9 empirically shows that the number of infections rapidly drops and the disease eradicates into the disease-free equilibrium as the probability of dying of infection ρ approaches 1.

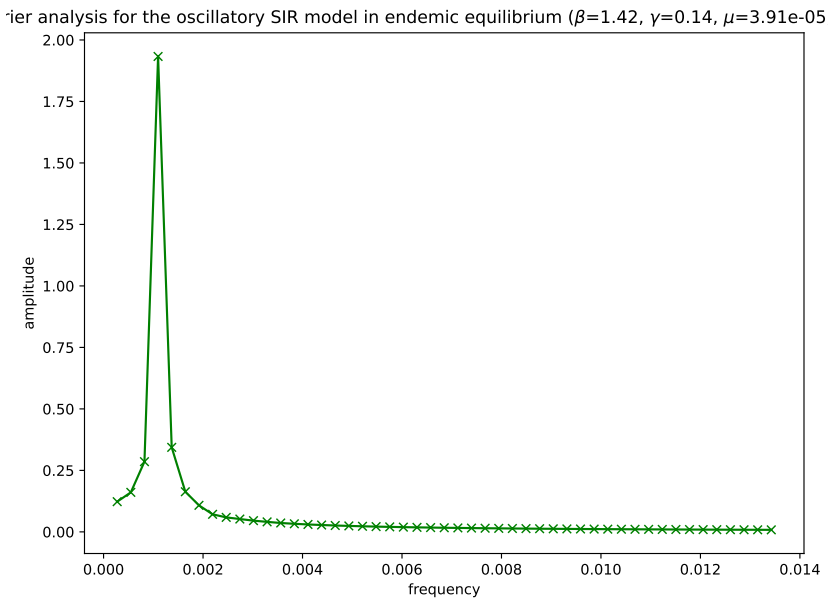


Figure 8: Non-harmonic oscillatory behaviour for the demographic SIR model in endemic equilibrium. $(I(0) = 2.5 \cdot 10^{-4}, S(0) = 0.1, R(0) = 1.0 - I(0) - S(0))$

4 Variation of the SIR model

In the following, we will analyse the SEIR (SIER) model of infection, which takes into account the latent period of exposed (E) individuals which are infected but not yet infectious.

Introducing the rate of exposure σ corresponding to the average duration of the latent period $\frac{1}{\sigma}$, the SEIR model is described by the following set of coupled ordinary differential equations (ODE's):

$$\begin{aligned}
\frac{\delta S}{\delta t} &= -\beta SI + \mu - \mu S \\
\frac{\delta E}{\delta t} &= \beta SI - \sigma E - \mu E \\
\frac{\delta I}{\delta t} &= \sigma E - \gamma I - \mu I \\
\frac{\delta R}{\delta t} &= \gamma I - \mu R
\end{aligned}$$

[KR11]

Note that except for σ, the parameters are the same as for the SIR model with demographics (natural birth and death rates). Note that $\frac{\delta I}{\delta t}$ had to be changed to account for the group of exposed population E.

4.1 Effects of the latent period of exposed individuals

To better understand the dynamics of the SEIR model, we first provide an intuition for the effect that the latent period of exposed individuals from the group E has on the dynamics of the model.

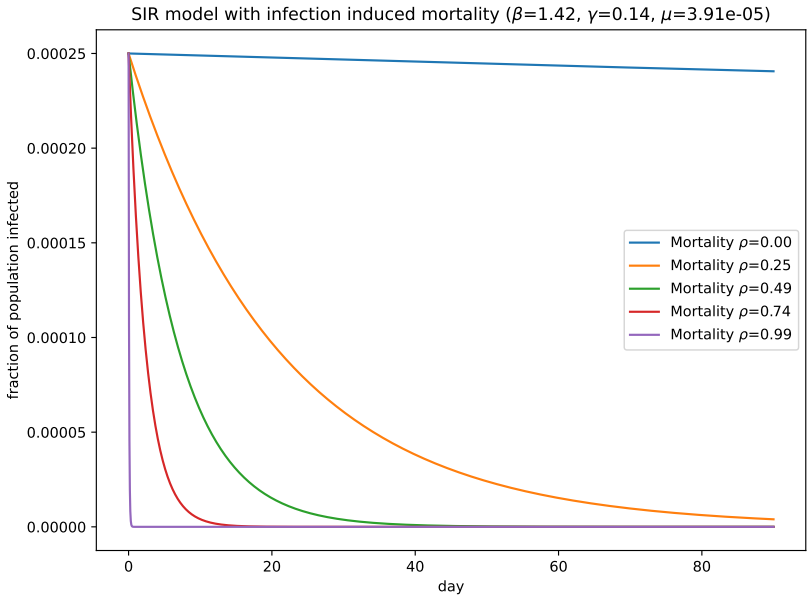


Figure 9: Effect of changing the probability of dying from the infection ρ on the demographic SIR model with infection induced mortality. $(I(0) = 2.5 \cdot 10^{-4}, S(0) = 0.1, R(0) = 1.0 - I(0) - S(0))$

As shown in Figure 10, the longer the latent period $\frac{1}{\sigma}$ in which the individual is exposed (infected but not yet infectious), the more latency is introduced to the model's dynamics.

4.2 Seasonal changes to the rate of infection

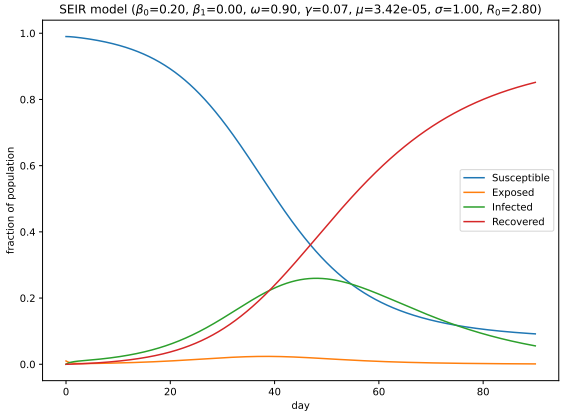
Having established the SEIR model and some of its characteristics, we wish to extend the model to take seasonal effects into account. Such models are called *temporally forced*. This makes a lot of practical sense, because it is commonly known that transmission rates increase during autumn and winter when people are closer to each other in poorly ventilated rooms. The same applies to childhood infections which are peaking at the start of the school year and declining in the summer. Moreover, we could imagine this to potentially also be useful to model infectious diseases for which the pathogen units (e.g. bacterial cells or virions) are highly sensitive to differences in temperature and might not withstand cold or heat.

Having established the usefulness of a time varying rate of infection β, we model β as a function of time t:

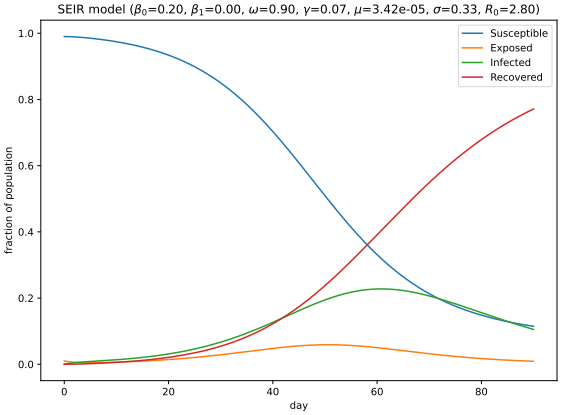
$$\beta(t) = \beta_0(1 + \beta_1 \cos(\omega t))$$

[KR11]

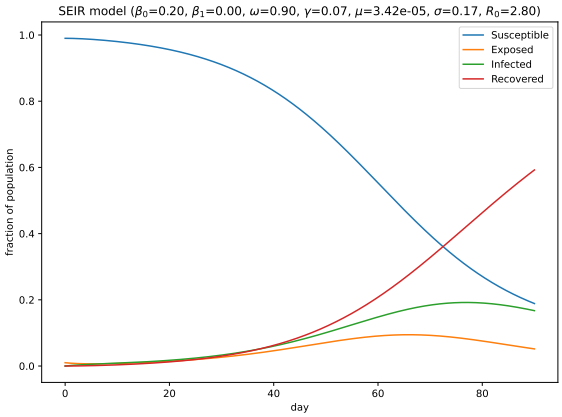
Here, the parameter β_0 denotes the baseline transmission rate, ω denotes the period of the seasonal forcing, and β_1 denotes the amplitude of the seasonality ($0 \leq \beta_1 \leq 1$). Figure 11 gives an intuition on the parameters of $\beta(t)$.



(a) $\sigma = \frac{1}{1}$



(b) $\sigma = \frac{1}{3}$



(c) $\sigma = \frac{1}{6}$

Figure 10: Phase space diagram for a latent periods $\sigma = \frac{1}{1}$,$\sigma = \frac{1}{3}$,$\sigma = \frac{1}{6}$ of one, three, and 6 days on the SEIR model. $(E(0) = 0.01, I(0) = 0.0, S(0) = 0.99, R(0) = 0.0)$

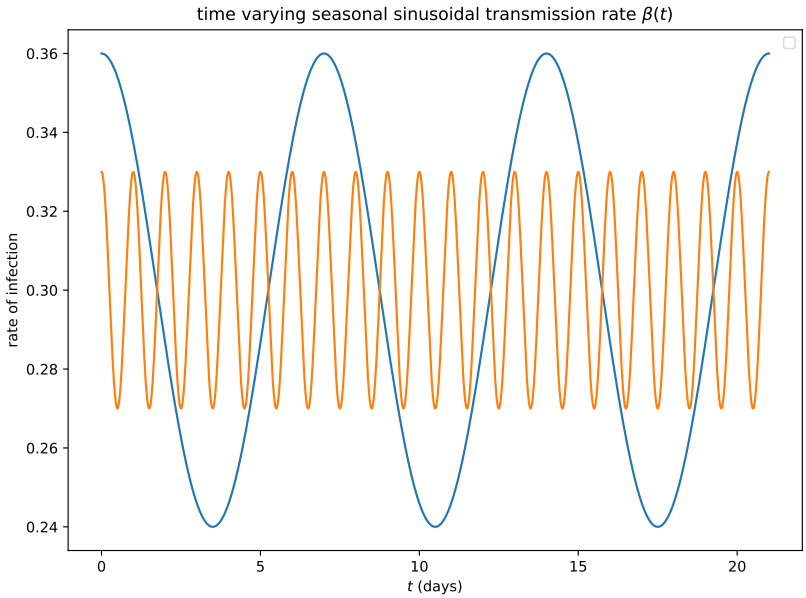


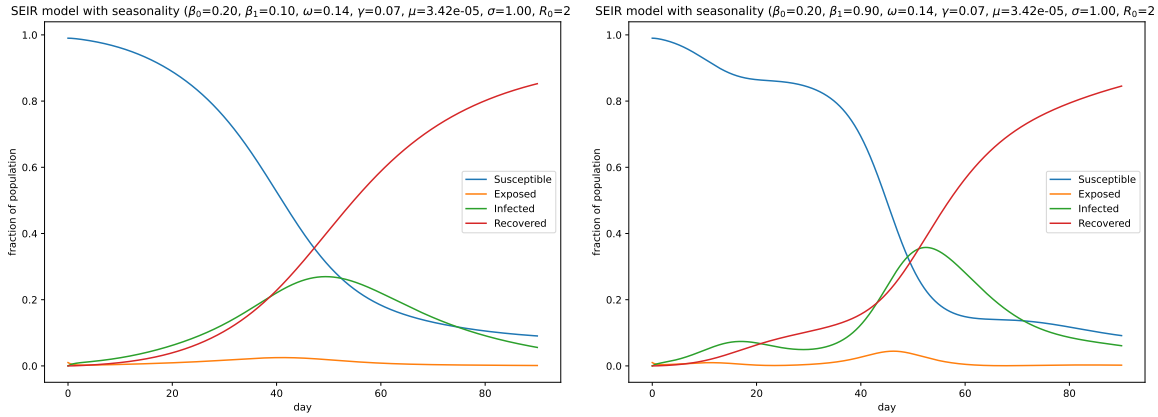
Figure 11: Time varying seasonal sinusoidal transmission rate $\beta(t)$. For the blue graph, $\beta_1 = 0.2$ and $\omega = \frac{2\pi}{7}$ (weekly). For the orange graph, $\beta_1 = 0.1$ and $\omega = \frac{2\pi}{1}$ (daily). In both cases $\beta_0 = 0.2$.

Given the seasonal rate of infection, Figure 12 shows the effect of increasing β_1 from $\beta_1 = 0.1$ to $\beta_1 = 0.9$ without altering the other parameters. As can be seen, the seasonal term does not radically change the dynamics of the model, but causes considerable variations.

Finally, we investigate the pattern of recurring epidemics that can be observed on temporally forced models as opposed to unforced models, for which oscillations are damped toward equilibrium as shown in Section 3.1. Figure 13 gives an example of a configuration with seasonally recurring epidemics without a decrease in amplitude. The conditions under which those arise are similar to the SIR model, in such that the disease-free equilibrium does not produce oscillations or recurring epidemics. However, recurring epidemics are more find as they are based on the interplay between the spread of the disease which requires a certain threshold of susceptible individuals in the population. Therefore, the seasonal rate of infection is closely tied to the harmonic resonance of the fraction of susceptible individuals due to an influx via the natural birth rate and a balance has to be found.

References

[KR11] Matt J Keeling and Pejman Rohani. *Modeling infectious diseases in humans and animals*. Princeton university press, 2011.



(a) $\beta_1 = 0.1$ (b) $\beta_1 = 0.9$

Figure 12: Phase space diagram for the same SEIR model for different degrees of seasonal influence β_1 on the infection rate. $(E(0) = 0.01, I(0) = 0.0, S(0) = 0.99, R(0) = 0.0)$

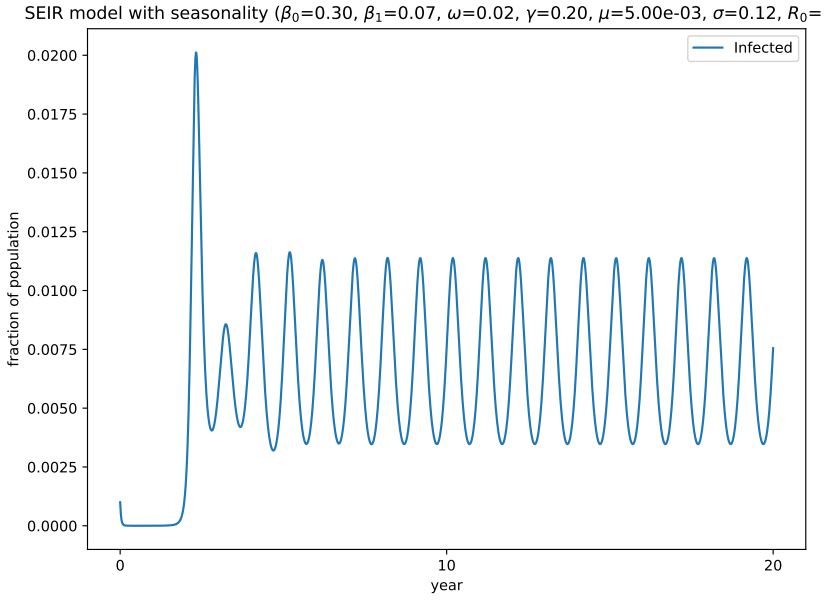


Figure 13: Recurring epidemics of the temporally forced SEIR model.
$(E(0) = 10^{-3}, I(0) = 10^{-3}, S(0) = 6 \cdot 10^{-2}, R(0) = 1.0 - I(0) - S(0) - E(0))$