NUMERICAL DISEASE MODELING WITH THE 4TH ORDER RUNGE-KUTTA METHOD AND MONTE CARLO METHOD

SIRS-MODEL

Anna Eliassen, Aron Jansson Nordberg and Kristina Othelia Lunde Olsen
Department of Physics, University of Oslo, Norway
{anna.eliassen, aronjn, koolsen}@fys.uio.no

December 19, 2020

Abstract

This report studies the spread of an infectious disease by applying the SIRS model to four isolated populations, with a set rate of transmission and immunity loss, as well as a varying rate of recovery $b = [1, 2, 3, 4]$. The basic SIRS model is extended to be more realistic by including vital dynamics, seasonal variations and vaccinations. The 4th order Runge-Kutta method and the Monte Carlo method is used to numerically solve the differential equations from the SIRS model. The two methods gave the overall same results, with some more fluctuations for the Monte Carlo method. Both methods showed that the disease would establish itself in the population if the recovery rate were less than the transmission rate. When using vital dynamics, birth- and death rates were implemented, resulting in an increasing population despite the disease, when $b > 1$ and the birth rate were higher than the death rate. When including a oscillating time dependent transmission rate, the model gained seasonal variations, where the spread of the disease were higher during winter months. As expected when fighting an infectious disease, the implementation of distributing a vaccine resulted in a decrease of infected people in all populations.

1 Introduction

When writing this report, the world is undergoing the Coronavirus disease (COVID-19) pandemic, making the understanding of the evolution of infectious diseases over time more important than ever. For scientists, an informative and important method to achieve this understanding is to make compartmental models, which simplify the mathematical modelling of infectious diseases [1].

Infection models are dynamic, as a person or group of people, may affect other peoples' chances of getting infected. This is why modelling infectious diseases are so difficult. The most commonly used models of longtime predictions are build on a mechanical framework, where the disease is tracked from the time of entering a population of susceptible people, until it is no longer transmitted from person to person. The purpose of this report is to study a well-known compartmental model, the SIRS model, which consists of three population groups (Susceptible, Infected and Recovered). This model allows us to have some knowledge of how the disease transmits in the given population, but simplify the behaviour of the disease by three rates. The rate of transmission a, the rate of recovery b and the rate of immunity loss c. With these parameters, the model can be expressed by ordinary differential equations (ODEs), as well as transition probabilities. Solving the equations numerically, we can predict whether or not a certain disease has the capacity to establish itself within the population [2]. Two methods will be implemented in `Python` to simulate the evolution of the disease. The two methods are the 4th order Runge-Kutta (RK4) differential equation model, which assumes an continuous population, and a discrete model solved by a Monte Carlo (MC) method. The last method use transition probabilities to move one individual person between the susceptible, infected and recovered groups.

With better predictions of how, and how fast, a infectious disease is spreading through the population, the faster necessary measures and precautions can be made. If the disease is severe enough, a vaccine will have to be developed. We will investigate the impact vital dynamics have on the SIRS model, i.e. making a more realistic model with births and deaths in the population. We will also investigate the impact of seasonal variations and vaccination.

In section 2 we present the theoretical background needed for the SIRS model and the numerical differential equation solvers RK4 and MC. In section 3, we explain our approach for solving the model with both methods, and which parameters we used when introducing vital dynamics, seasonal variations and vaccination to the SIRS model. Our results are presented in section 4, where we discuss the finding of our RK4 and MC simulations of disease evolution. Finally, section 5 contains our conclusion.

All figures and `Python` code used for this report can be found on our GitHub page [3].

2 Theory

2.1 The SIR model

The SIR model is used to model the spread of a disease within a population. It is a compartmental model, meaning that every member of the population is included in only one population group at any given time. The model considers an isolated population of N-people which are divided into separate groups [1]. The SIR model is named after the three categories Susceptible, Infected and Recovered, as summarized in table 1. In this basic SIR model a person can move from one group to the next as implied by the model name, $S \rightarrow I$ and $I \rightarrow R$.

Table 1: The three population groups in the SIR-model (Susceptible-Infected-Recovered) [2].

Group		Explanation
Susceptible	S	The people without immunity to the disease.
Infected	I	The people who are currently infected with the disease.
Recovered	R	The people who have been infected in the past and have developed an immunity to the disease.

2.2 The SIRS model

For our disease modeling, we want a slightly more advanced and realistic version of the SIR model, so we will use the SIRS model. The SIRS model is an improved, or extended, version of the basic SIR model. In this model a person can move from one group to another in a cyclical fashion as indicated by the name. This is illustrated in figure 1, where a is the rate of transmission from $S \rightarrow I$, b is the rate of recovery from $I \rightarrow R$ and c is the rate of immunity loss from $R \rightarrow S$. This means that people who have recovered are not permanently immune to the disease, as there is a rate c of the recovered who will become susceptible again.

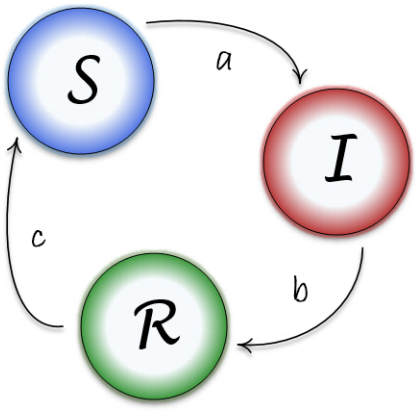

Figure 1: The SIRS model, where S is the Susceptible group, I is the Infected group and R is the Recovered group. a is the rate of transmission from $S \rightarrow I$, b is the rate of recovery from $I \rightarrow R$ and c is the rate of immunity loss from $R \rightarrow S$.

We will assume that the population is mixed homogeneously and that the total population will remain constant so that

$$N = S(t) + I(t) + R(t) \tag{1}$$

This is justified if we consider a disease run that is much shorter than the average lifespan. This means the epidemic will not be effected by births and (natural) deaths, and these rates are therefore ignored. This justifies (1) as a restriction in our model. From these assumptions, the change in the S, I and R group can be constructed by a set of coupled differential equations [4]

$$S' = cR - \frac{aSI}{N} \tag{2}$$

$$I' = \frac{aSI}{N} - bI \tag{3}$$

$$R' = bI - cR \tag{4}$$

Where the apostrophe signifies the infinitesimal change in the quantity over an infinitesimal time step, e. g. $S' = dS/dt$. Each equation can be understood intuitively. For the population group S the change will come from people losing their immunity, the number of which is cR. Another change is the number of people who are infected with the disease. This is given by aS, but also by I/N, as the number of people being infected is proportional to the fraction of the total population who are already infected. More infected people means it spreads more quickly. The I group changes by receiving the number of people who are infected, given by aSI/N and loses the people who recover, given by bI. In a similar way, the R group loses people who become susceptible again, cR and gains people who recoved from the disease bI. The set of differential equation (2)-(4) does not have an analytical solution. However, we can use the additional restriction from equation (1) to reduce the number of equations to just two. We eliminate R and get

$$\begin{aligned} S' &= c(N - S - I) - \frac{aSI}{N} \\ I' &= \frac{aSI}{N} - bI \end{aligned} \tag{5}$$

R can then easily be solved by $R = N - S - I$. Because the SIRS model allows of continuous movement between the categories, it will eventually reach an equilibrium state. It can be shown that in order for the disease to establish itself, the transmission rate must be greater than the recovery rate, that is $a > b$. See Appendix A.

This basic SIRS model can be expanded to include more details about the population, the disease and the other facts surrounding the epidemic. We will consider vital dynamics (birth and death rates), seasonal variations in transmission rates, and the effect of a vaccine. This will make our model more realistic.

2.2.1 Vital dynamics

Vital dynamics is added to the model so that we can describe the spread of diseases over a longer period of time. This means we include *Birth* and *Death* groups in our model, as seen in figure 2.

The modified differential equations are given by

$$S' = cR - \frac{aSI}{N} - dS + eN \tag{6}$$

$$I' = \frac{aSI}{N} - bI - dI - d_I I \tag{7}$$

$$R' = bI - cR - dR \tag{8}$$

where e is the birth rate, d is the natural death rate, and d_I is the death rate of infected people due to the disease [2]. All newborns are assumed susceptible to the disease, and that all natural deaths can occur in all groups with the same constant rate.

2.2.2 Seasonal variation

Many diseases, like influenza, are affected by the seasonal variations, making the transmission rate dependent of the time of year. The population behaves differently during seasons, warmer months are spent more outside, while colder months are spent more inside. The temperature also affects our immune systems. Spending more time inside, also means closer proximity to other people. All this results in a transmission rate a that is now a function of time $a(t)$. We can implement a periodic function to model the seasonal variations.

$$a(t) = A\cos(\omega t) + a_0 \tag{9}$$

a_0 is the average transmission rate, A is the maximum deviation from a_0 and ω is the frequency of the oscillation.

2.2.3 Vaccination

The last factor we want to explore in our disease simulation is the effect of introducing vaccination against the disease. When there is a vaccine to a disease, susceptible people who have taken the vaccine can move directly from S to R. This rate of vaccination is indicated as f in figure 2.

The rate of people getting vaccinated will in reality be dependent on a number of things. Availability of the vaccines, the number of people already taking it, information about it (awareness) and so on. We will assume that the rate of people in S getting vaccinates is a constant f, but it will only be applied after a certain time has gone by. This models the reality that a vaccine will be developed in response to the disease, so it will not be available from the beginning. The system of differential equations with vaccination are now given by [2]

$$S' = cR - \frac{aSI}{N} - fS \tag{10}$$

$$I' = \frac{aSI}{N} - bI \tag{11}$$

$$R' = bI - cR + fS \tag{12}$$

where we can see a number of people fS going directly from S to R. There is no separate group for the vaccinated, meaning they will get susceptible at the same rate as before. So the system will still reach an equilibrium.

2.2.4 Combined model

The combined model is the SIRS model with vital dynamics, seasonal variations and vaccination. This combined model is illustrated in figure 2, and the full differential equations becomes

$$S' = cR - \frac{aSI}{N} - dS + eN - fS \tag{13}$$

$$I' = \frac{aSI}{N} - bI - dI - d_I I \tag{14}$$

$$R' = bI - cR - dR + fS \tag{15}$$

with a as defined in equation (9).

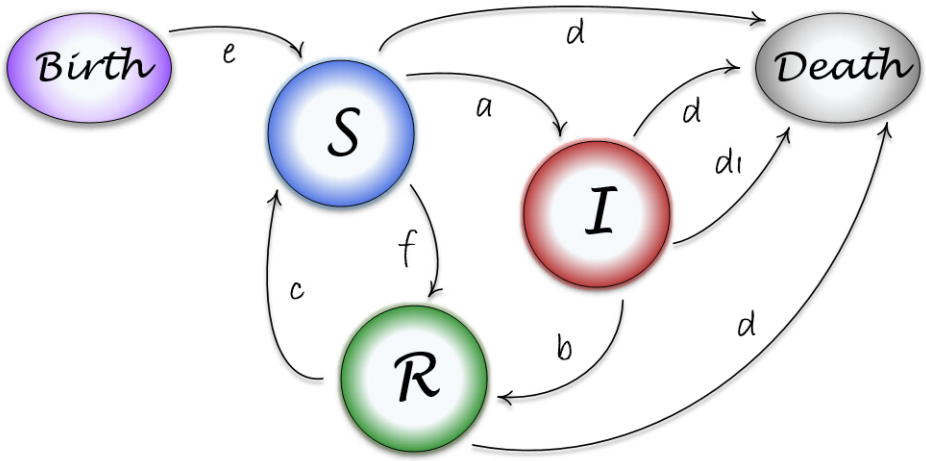

Figure 2: The base of the model is the SIRS model, where S is the Susceptible group, I is the Infected group and R is the Recovered group. a is the rate of transmission from $S \to I$, b is the rate of recovery from $I \to R$ and c is the rate of immunity loss from $R \to S$. From the vital dynamics we have a group *Birth*, with birth rate e, and a group for *Death*, with a death rate d that comes from the S, I and R group. There is also the death rate of infected people due to the disease d_I that only comes from group I. Lastly, there is a group of people that goes directly from S to R after receiving a vaccine, indicated by the vaccination rate f.

2.3 4th order Runge-Kutta method

The 4th order Runge-Kutta (RK4) method is a numerical algorithm to solve 1st order ordinary differential equations (ODEs). It best understood as a generalization of the simpler forward Euler method. Consider an initial value problem

$$\frac{dy}{dt} = f(t, y), \qquad y(t_0) = y_0, \tag{16}$$

where y is an unknown function, to be determined, and y_0 is a known value for when $t = t_0$. [5]. In order to solve (16) numerically we first create a grid of n points where we will compute the values for y. Thus we define $y_i = y(t_i)$ for $i \in (0, 1, 2, \ldots, n-1)$. The general description of the forward Euler method can be derived from the definition of the derivative

$$\frac{dy}{dt} = \lim_{t\to\infty} \frac{y(t+\Delta t) - y(t)}{\Delta t}$$
$$\frac{dy}{dt} \approx \frac{y(t+\Delta t) - y(t)}{\Delta t}$$
$$y(t+\Delta t) \approx y(t) + \frac{dy}{dt}\Delta t$$

This is also a first-degree Taylor expansion of $y(t)$, which allows us to determined the local truncation error as $O(h^3$. Now let $h = (t_{n-1} - t_0)/n$ be the step size in our grid, then we can describe the forward Euler scheme as follows

$$y_{i+1} = y_i + f(t_i, y_i)h$$
$$t_{i+1} = t_i + h$$

If we now have an initial value y_0, we can compute each y_{i+1}, one step at a time. Each step is determined by the previous one in a recurrence relation. In the step from t_i to t_{i+1} we compute the value of y_i and the slope $f(t_i, y_i)$ at t_i and approximate the function $y(t)$ as a straight line with slope $f(y_i, t_i)$. This method is intuitive, but it suffers from some problems. It suffers from taking the slope at the beginning of every step, which will normally cause it to "overshoot" when the function it approximates is curved, so the errors accumulate.

In forward Euler we only use one point to compute the slope, namely in the beginning of the step. What makes RK4 a more advanced method is the computing of additional 3 estimates of the increase. One at the end and two in the middle, with increased importance given to the two in the middle. Here we will present the algorithm in its entirety, and the explanation will follow below.

$$y_{i+1} = y_i + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4),$$
$$t_{i+1} = t_i + h,$$

for $i = 0, 1, 2, ...$ The slopes k_1, k_2, k_3 and k_4 are defined as

$$k_1 = f(t_i, y_i)$$
$$k_2 = f(t_i + \frac{h}{2}, y_i + \frac{k_1}{2})$$
$$k_3 = f(t_i + \frac{h}{2}, y_i + \frac{k_2}{2})$$
$$k_4 = f(t_i + h, y_i + k_3)$$

Notice that computing k_1 is exactly the same as for Euler's method. It is the slope at the beginning of the interval $[t_i, t_i + 1]$.

k_2 computes the slope at the half-way point $t_i + h/2$ using the point $y_i + k_1/2$, which is the point on the line from t_i with slope k_1.

k_3 also computes the slope at the halfway point $t_i + h/2$, but it uses the point $y_i + k_2/2$, which is the point along a line from t_i with the slope k_2 instead of k_1.

k_4 is the slope at the end point of the interval, $t_i + h$, on the point $y_i + k_3$ which a line from t_i with the slope k_3.

Finally a weighted mean is taken of all these four estimates, with greater importance placed on the two in the middle of the interval, k_2 and k_3. These facts are summarized in figure 3. The local error is the order of $O(h^5)$, while the global error goes as $O(h^4)$ [4]. This method needs more numerical operations per time step than the forward Euler method, but it has better stability and greater accuracy. The cost-benefit of RK4 makes it very popular in scientific applications.

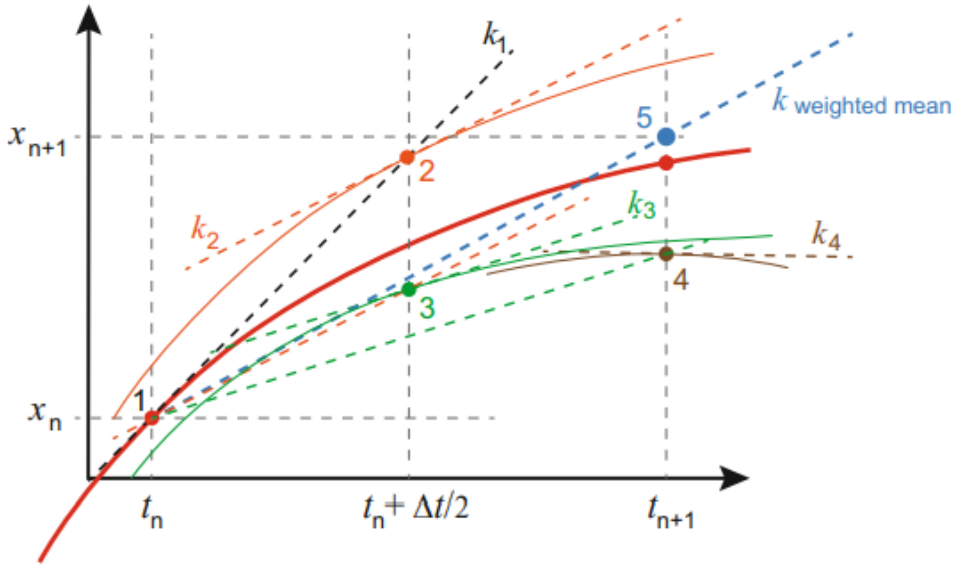

Figure 3: Visual representation of RK4. Follow the steps 1, 2, and so on and one can see how the different slopes are computed. Source: [6]

3 Method

For this project, we used two numerical methods to solve the system of differential equations derived from the SIRS model, and the various extensions presented in section 2.2. The first method is the deterministic RK4 method and the second one is the stochastic MC simulations. We implemented both methods using the programming language `Python`, with extensive use of the `numpy` package for numerical work and `matplotlib.pyplot` for visualization.

3.1 4th order Runge-Kutta method

The system of differential equations that are derived from the basic SIRS model, (2)-(4), were solved directly by the RK4 method as described in section 2.3. As the method iterated over the grid points in the time interval, each quantity S, I and R were updated simultaneously as each of their time evolution depended on the two others. As we investigated the various scenarios detailed in 3.3, the only part of the algorithm that had to be modified were the right-hand side of the equations.

To verify our results, we calculated the analytical fractions of the different groups for each population according to Appendix A, as seen in table 2. These values were obtain by setting equations (5) equal to zero, and using the constraint of having a constant number of total people in the population. For each value of b, the fractions of the different groups adds up to 1. These fractions were used to compare to the values of S, I and R after they reached equilibrium in the numerical computations.

Table 2: The analytical fractions of the different groups at equilibrium for the SIRS-model, considering transmission rate of $a = 4$ and immunity loss of $c = 0.5$.

	b=1	**b=2**	**b=3**	**b=4**
s*	0.25	0.5	0.75	1
i*	0.25	0.1	~ 0.036	0
r*	0.5	0.4	~ 0.214	0
SUM	1	1	1	1

3.2 Monte Carlo simulations

This following section is based on the *Part b), moving on to Monte Carlo simulation* chapter in the project description [2].

The set of differential equations derived from the basic SIRS model treats the quantities S, I, and R as continuous variables. In reality, however, they represent groups of people, and would therefore be discrete. A straight-forward solver of the set of differential equations, such as RK4, will allow for fractions of people. One remedy is to make us of Monte Carlo simulations. Instead of using equation (2)-(4) as deterministic descriptions of how S, I and R evolve with time, we considered them as a stochastic process. In each time step, there was a finite probability that one person will go from one group to the next. This stochastic process was modelled using random numbers and the transition probabilities derived from the SIRS model.

If we consider the set of differential equations from (2)-(4), we could see that for a small time step, Δt, the number of people moving from group S to I are approximately $\frac{aSI}{N}\Delta t$. Also, the number of people moving from group I to R are approximately $bI\Delta t$ and $cR\Delta t$ for R to S [2]. Next, Δt had to be set small enough for only one person to be moved from a given group to another, so we got

$$\max\left\{\frac{aSI}{N}\Delta t\right\} = \frac{a}{N}\left(\frac{N}{2}\right)^2 \Delta t = \frac{aN}{4}\Delta t \tag{17}$$

$$\max\left\{bI\Delta t\right\} = bN\Delta t \tag{18}$$

$$\max\left\{cR\Delta t\right\} = cN\Delta t \tag{19}$$

The time step were then given by three options, where we were interested in the smallest one for each iteration in our simulation, so that

$$\Delta t = \min\left\{\frac{4}{aN}, \frac{1}{bN}, \frac{1}{cN}\right\} \tag{20}$$

Then we interpret (18)-(19) as transition probabilities.

$$P(S \to I) = \frac{aSI}{N}\Delta t \tag{21}$$

$$P(I \to R) = bI\Delta t \tag{22}$$

$$P(R \to S) = cR\Delta t \tag{23}$$

Then, our simulation computed the probabilities of each transition happening for each time step. A random number between 0 and 1 were drawn, and if the number were less than the given probability, the transition of one individual person from the group to another would happen.

3.3 Population and scenarios

When studying the SIRS model and the possible extensions we could add, we thought the most instructive way was to study each extension separately. We dedicated a scenario for each extension, as described in the following sections, where all have starting point in Scenario 1. In the fifth scenario, we combined the SIRS model with all extensions we studied, giving us a combined model where we could study the combined effect by adding vital dynamics, seasonal variations and vaccination to the SIRS model.

In each scenario, we investigated four different population compositions. We used a initial population of 400 people, where 300 people were susceptible and 100 people were infected. The rate of transmission and the rate of immunity loss were fixed for all four populations, but the recovery rate were varied, see table 3, the rates have units of inverse time. The reason we let the recovery rate vary was because it represent the action that can be taken by society during a pandemic. Regulations of personal contact, hygiene, public events etc. is something that each separated population can handle differently.

Table 3: A set of parameters for the four different populations; A, B, C and D. a is the rate of transmission, b is the rate of recovery and c is the rate of immunity loss.

Rate	A	B	C	D
a	4	4	4	4
b	1	2	3	4
c	0.5	0.5	0.5	0.5

3.3.1 Scenario 1: The SIRS model

The first scenario is the basic SIRS model, as described by equation (2)-(4) in section 2.2. When solving them with RK4 we used the reduced set of equations, given by (5). For MC simulations we made direct use of the transition probabilities given by equation (22)-(23). As mentioned in section 3.2, this was done by drawing one random number for each step in time, and evaluate if it was smaller than the transition probabilities. If that was the case for the transition from S to I, we subtracted 1 from S and added 1 for R in the next time step. The same was done for the transition from I to R and R to S.

For both methods, we studied the scenario for all four populations A, B, C, and D as described in table 3. We studied the differences between the two methods and the impact of the varying recovery rate.

For the results of our MC simulation, we made a table of the mean number of people, and the corresponding standard deviation, for each S, I and R group after the system reached equilibrium. Because the system reached equilibrium at different times, the equilibrium time were set manually for each population. The times were set to $T = [8, 9, 10, 10]$ for populations A, B, C and D, respectively.

3.3.2 Scenario 2: Vital dynamics

The second scenario is the SIRS model with vital dynamics as described in section 2.2.1. Here, we have assumed that all newborns are susceptible. We chose a birth rate e, death rate d and a death rate of infected people due to the disease d_I as follows

$$\begin{aligned} e &= 0.25 \\ d &= 0.2 \\ d_I &= 0.35 \end{aligned}$$

We updated our RK4 code with the modified differential equations (6)-(8). For MC we needed additional transition probabilities. This was done straight forwardly by using that the probability of one person dying in, for example, the S group is simply $dS\Delta t$. We therefore get the additional transition probabilities

$$\begin{aligned} P(S \to \text{death}) &= dS\Delta t \\ P(I \to \text{death}) &= (dI + d_I I)\Delta t \\ P(R \to \text{death}) &= cR\Delta t \\ P(\text{birth} \to S) &= eS\Delta t \end{aligned}$$

We tried out different ways to draw the random number, meaning using the same random number for all transitions in each time step, or by using a different random number for one or more of the transition probabilities in vital dynamics. We ended up with drawing one random number for the S, I and R transitions (as implemented in the basic SIRS model), and using another random number which all vital dynamic transitions were evaluated by. For the interpretation and visualization, we added a line of the total population in our plots, as we no longer have a constant population.

3.3.3 Scenario 3: Seasonal variations

The third scenario is the SIRS model with seasonal variation. Here we used the SIRS equations from (2)-(4), with the time dependent a as described in section 2.2.2 and equation (9). As scenario 3 includes seasonal variation, but not vital dynamics, we will have a constant population. The variables were set as follows

$$\begin{aligned} A &= 4 \\ \omega &= 0.5 \\ a_0 &= 4 \end{aligned}$$

so that the time dependent transmission rate becomes $a(t) = 4\cos(0.5t) + 4$. We model the disease spread over a year, so we chose $\omega = 0.5$. This gives a periodic

function that is at its max at the beginning and end of the computations. This corresponds to maximum transmission during winter (beginning and end of year) and the minimum transmission during summer. For MC this also meant that the time step Δt had to be determined at each step in the simulation, as given by (20). The transitions between the different groups were evaluated the same way as in the basic SIRS-model.

3.3.4 Scenario 4: Vaccination

The fifth scenario is the SIRS model with vaccination as described in section 2.2.3. We chose to start the vaccination after $T/2$, which is after $T = 6$.

$$f(t) = \begin{cases} 0 & \text{for } t < 6 \\ 0.5 & \text{for } t > 6 \end{cases}$$

We updated the differential equations in our code to equations (10)-(12) for RK4. For MC, we again needed to implement new transition probability, now given by

$$P(S \to R) = fS\Delta t.$$

As mentioned in the introduction of this chapter, we wanted to study the effects of the extensions separately, so we did not include vital dynamics or seasonal variation at this point. By viewing plots from different runs of MC, and comparing with RK4, we decided to evaluate the probability of getting a vaccine with the same random number that we drew for the SIRS-transitions in the current time step.

3.3.5 Scenario 5: Combined model

In the last scenario, we investigated the outcome when simulating the SIRS model with vital dynamics, seasonal variations and vaccination. We decided to use the same parameters as described in section 3.3.2 to section 3.3.4. We will have a non-constant population, so a total population line were added to the plots to better observe the change in population. In our RK4 implementation, we updated the differential equations to the equations (13)-(15). For the MC simulation we included all the transition probabilities introduced in the previous scenarios at once. As in the previous cases, we ended up using the same random number drawn at each time step for evaluating the SIRS-transitions and vaccination probability, and a separate one for evaluating the transitions related to vital dynamics.

4 Results and Discussion

From our RK4 and MC simulations, there are separate plots for $b = [1, 2, 3, 4]$ for each population A, B, C and D on our GitHub page under the *Result* folder. The different scenarios are divided into separate sub-folders, where *5a* and *5b* correspond to scenario 1; while *5c/2_rdm*, *5d/omega_05_rapport* and *5e/1_rdm* correspond to scenarios 2, 3 and 4. *CombinedModel/own_rdm_vital_report* corresponds to scenario 5.

4.1 Disease evolution using RK4 and MC

Figure 4 shows the time development as computed by the RK4 method for our disease evolution in scenario 1, where each population has a different recovery rate b. Figure 5 displays the same from our MC simulation. We can see from the figures that the two methods produce the same overall trends, but MC have more smaller fluctuations and is less smooth than RK4. This is to be expected as RK4 treats S, I and R as continuous functions of t, so they will be smooth functions, determined uniquely by the differential equations. MC on the other hand treats them as discrete, and the change in each time step is an integer. In addition, when running the script several times, the formation of S, I and R will be slightly different, causing "the time of equilibrium" to vary more with MC than for RK4.

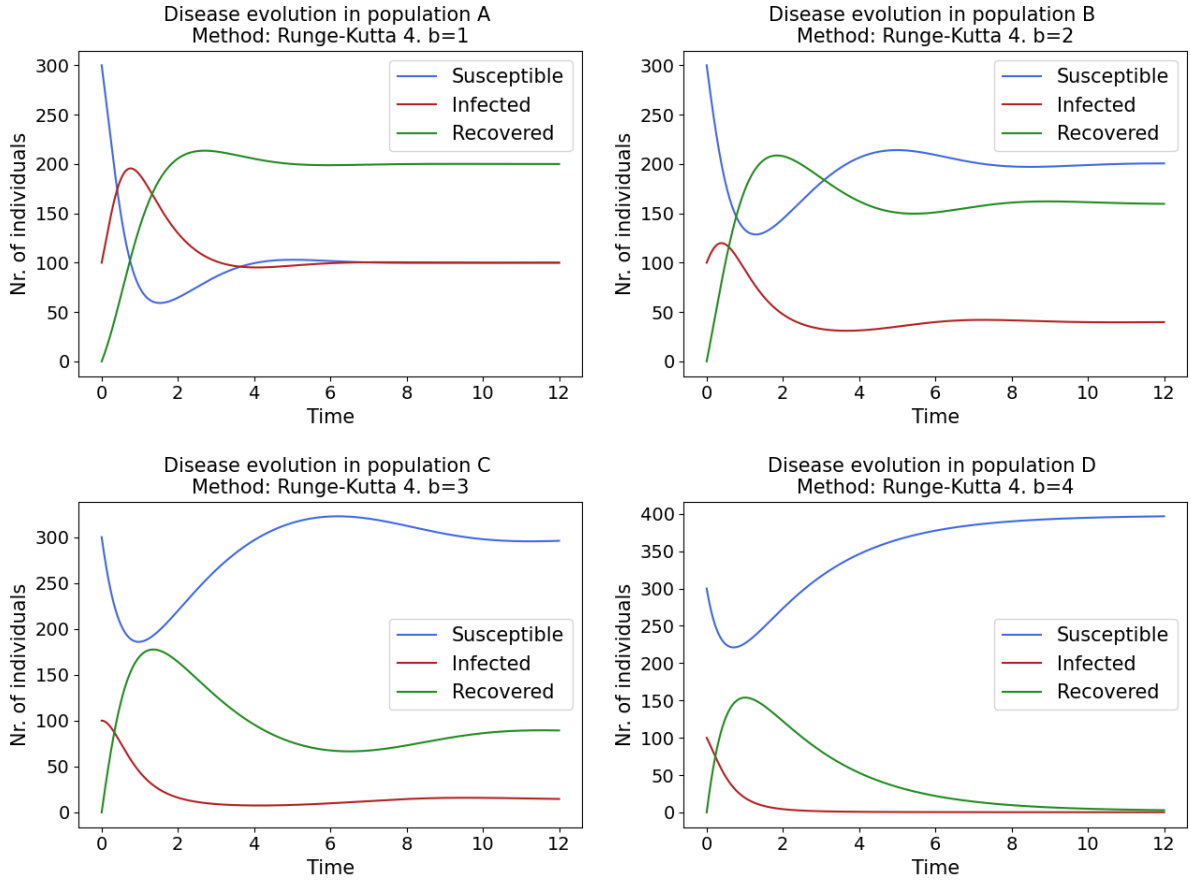

Figure 4: Time evolution of number of susceptible, infected and recovered individuals in population A, B, C and D, simulated using Runge Kutta 4 and $b = [1, 2, 3, 4]$.

For population A and B, we observe an initial rise in the number of infected people, and therefore a dip in the number of susceptible people. The number of susceptible reaches its minimum slightly after the infected-peak. This is because the infected individuals recovers after some time, and a fraction of recovered individuals will become susceptible again because of the rate of immunity loss.

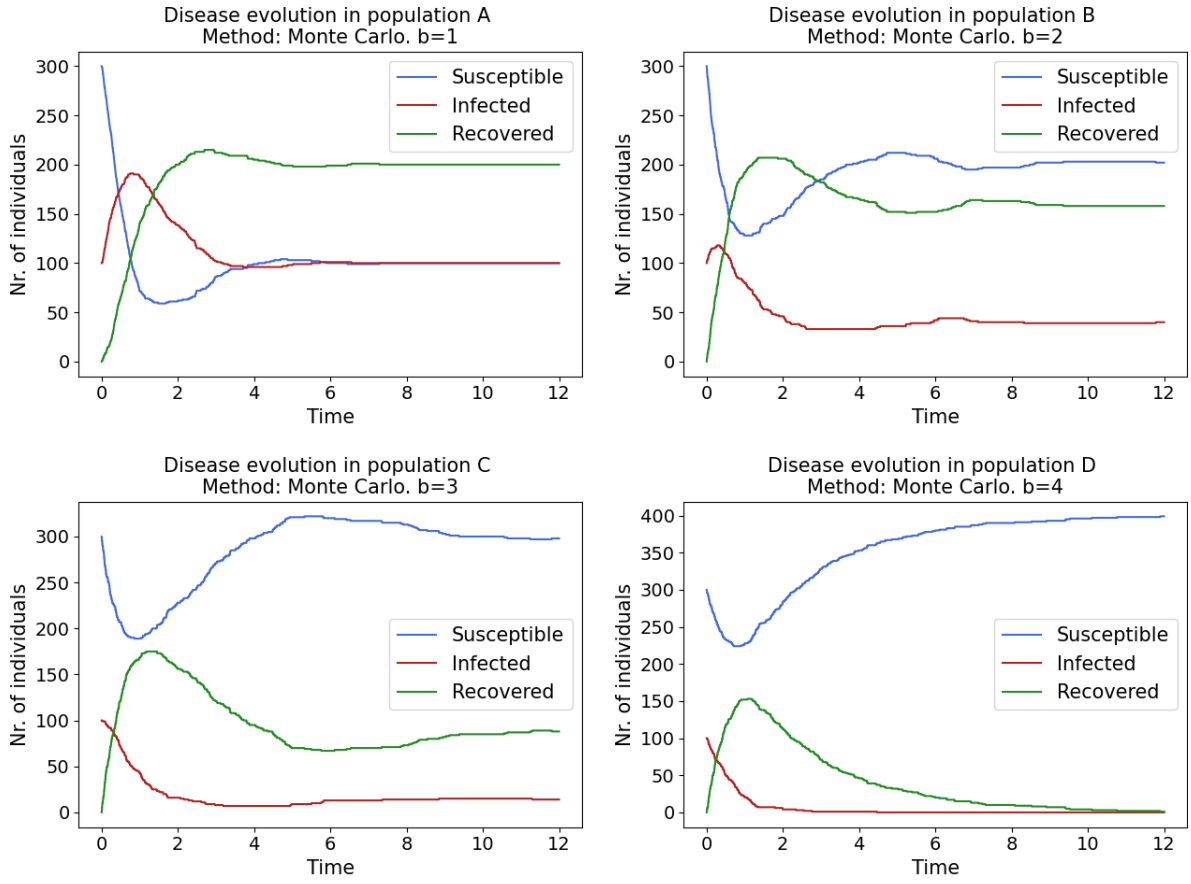

Figure 5: Time evolution of number of susceptible, infected and recovered individuals in population A, B, C and D, computed using Monte Carlo and $b = [1, 2, 3, 4]$.

We can see that for A and B, the main difference between them are the magnitude of the number of infected and susceptible people. When b increases, I decreases while S increases. This effect is even more visible for C, where the initial population have almost no infected individuals after $T = 2$. It also reaches a much higher number of susceptible individuals, and therefore the number of recovered individuals are few. This is intuitive since there are almost none infected. A higher value for b means that people recover faster and spend less time in I.

As discussed in appendix A, the quantities S, I and R eventually reach an equilibrium state, where their time derivatives become zero. Here we see a difference in the end-state of the diseases in our model. When $b < a$, the disease will establish itself in the population. This is reflected in both figures 4 and 5 when $b = 1, 2, 3$. Even though the disease gets established in all 3 populations, we observe that a higher b-value leads to a slower establishment, and the number of constantly infected individuals in the population will be much lower when b increases.

For population D, we have $b = a$ and observe an instant drop of infected. Because of the recovery rate being equally high as the transmission rate, the infected people recover too fast for the disease to spread trough the population. We see how the number of infected converge towards zero as the system reaches equilibrium, and the disease is prevented from establishing itself in the population.

Table 4 shows the equilibrium values for the S, I and R group for all populations. The numerically calculated values correspond well to the analytical values, which is easily obtain by the fractions s*, i* and r* in table 2 and multiplying them by $N = 400$. The sum for each population is as expected 400, as the population is constant. Table 5 contains a set of mean values of people belonging to each group after steady-state is reached, with corresponding standard deviation, σ for the MC method.

Table 4: Number of individuals in each S, I and R group for all populations after the system reached equilibrium. Table corresponds to figure 4, RK4.

	A	**B**	**C**	**D**
S	100.02	200.60	269.04	369.81
I	99.99	39.78	14.64	0.22
R	199.99	159.62	88.32	2.96
SUM	400.00	400.00	400.00	400.00

Table 5: The mean number of individuals in each group, and the corresponding standard deviation, for the MC method in figure 5 after the system reached equilibrium. The time of equilibrium are $T = [8, 9, 10, 10]$ for populations A, B, C and D, respectively.

Population	**A**	**B**	**C**	**D**
Group	mean $\pm \sigma$	ean $\pm \sigma$	mean $\pm \sigma$	mean $\pm \sigma$
S	100.78 ± 0.41	202.21 ± 0.42	295.66 ± 1.37	397.09 ± 0.84
I	100.00 ± 0.00	39.20 ± 0.40	16.74 ± 0.44	0.00 ± 0.00
R	199.22 ± 0.41	158.60 ± 0.64	87.60 ± 1.71	2.91 ± 0.84

4.2 Disease evolution with vital dynamics

The results from the time development of scenario 2 is shown in figure 6. Population A is to the left in the figure and population C to the right. Top row is RK4 and bottom row is MC. We will compare this to the basic SIRS case, given in figures 4 and 5.

Since our birth rates are higher than the (natural) death rates, that is $e > d$, every population will have a gradual increase over time, in the absence of the disease. The group I has an additional death rate d_I, which means that in populations were the recovery rate is lower, more people will die because they stay there longer than with higher b. For population A, which has a low recovery rate, the total population has declined by the time the system reaches equilibrium.

For population C, the time spent with the disease is lower and so fewer people will die and the total population will have increased when the system reached equilibrium. At that time the disease has established itself, but it is very low, and the population of C will increase over time.

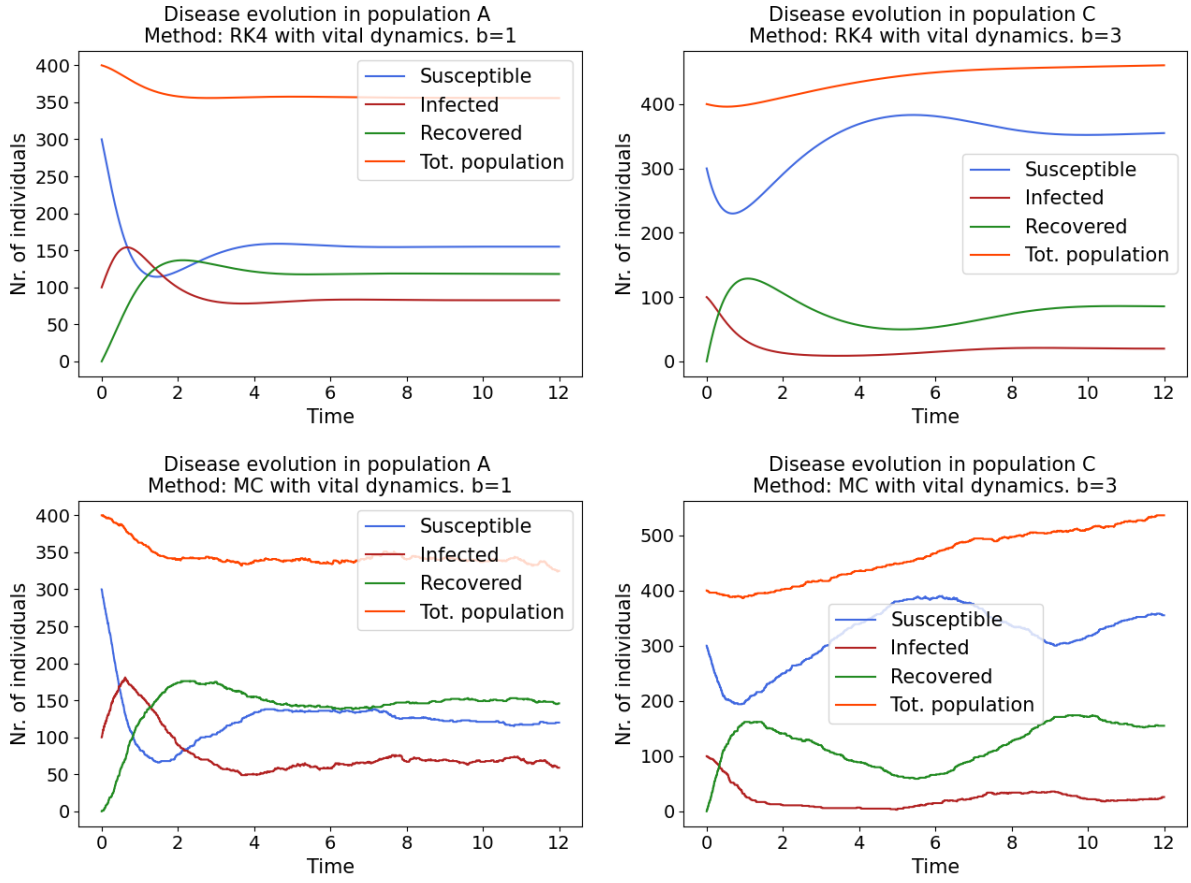

Figure 6: Time evolution of number of susceptible, infected and recovered individuals in population A and C for $b = 1$ and $b = 3$ (rate of recovery), computed using Runge-Kutta 4 and Monte Carlo. The disease evolution is a SIRS model with vital dynamics, with birth rate $e = 0.25$, death rate $d = 0.2$ and death rate of infected people due to the disease $d_I = 0.35$.

4.3 Disease evolution with seasonal variations

The results from the time development of scenario 3 is shown in figure 7. Population A is to the left and population C to the right. Top row is RK4 and bottom row is MC.

With the time dependent transmission rate, we observe that we have peaks for I in the beginning and end, corresponding to the winter months, and a minimum around the middle, which would be summer time. Correspondingly, the S category reaches its peak around the same time when I is at its minimum, as expected. This means the spread of the disease is high during winter, which is the time when the weather would be cold and the population spend more time inside in close approximation to others. This is in good agreement with the theory that the transmission rate is high during winter and lower during summer. A result of the frequency $\omega = 0.5\ 1/\text{year}$. Further, we observe that the seasonal variation are more distinct with a lower b value. This is because a lower b means lower recovery value, so people are in the infected group for longer than when b is higher. We can see for when in population A with $b = 1$ the infection rates are at their peaks in the beginning and end of the year, and lowest during the middle of the year. For population C when $b = 3$ this is not the case. It seems the lower rates during summer completely killed off the disease and it did not establish itself at all by the time the system reached equilibrium.

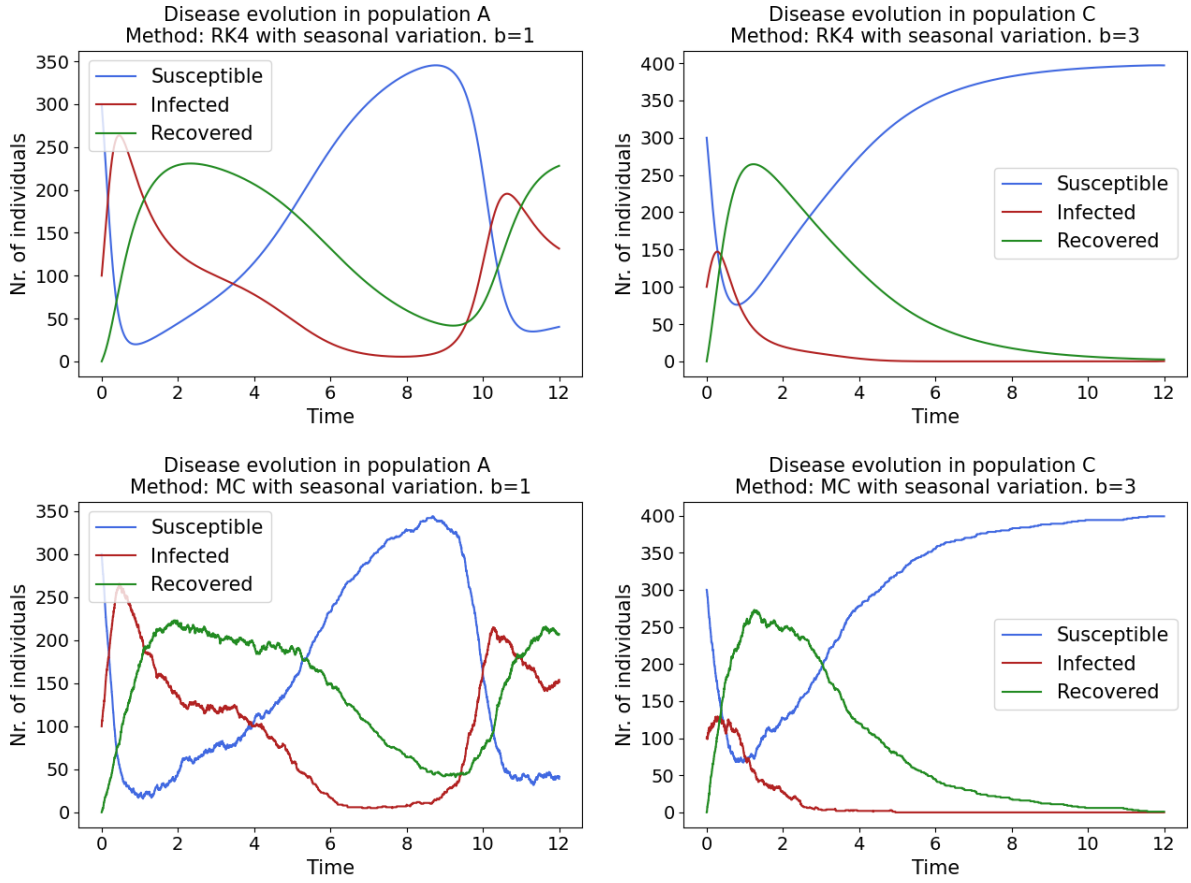

Figure 7: Time evolution of number of susceptible, infected and recovered individuals in population A and C for $b = 1$ and $b = 3$ (rate of recovery), computed using Runge-Kutta 4 and Monte Carlo. The SIRS model includes seasonal variation, where the time dependent transmission rate is given by $a(t) = 4\cos(0.5t) + 4$.

4.4 Disease evolution with vaccination

The results from the time development of scenario 4 is shown in figure 9, with corresponding labels in figure 8. Population A is to the left in the figure and population C to the right. Top row is RK4 and bottom row is MC.

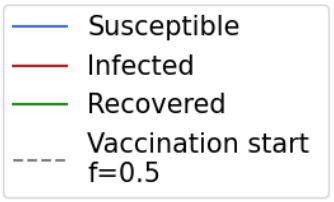

Figure 8: Labels for *Disease evolution with vaccination*

The time the vaccine is introduced to the populations is marked with a striped line at $T = 6$. The results show a clear difference before and after the vaccination is introduced to the populations. Since vaccinations takes people directly from S to R, they are mirrored in their interactions. The number of recovered increase just as the number of susceptible decrease.

At the time before $T = 6$, the system is the basic SIRS model, and as expected behaves the same as in section 4.1. When the vaccination starts however, we observe that for A, the fraction of recovered increases. The number of susceptible drop for a short period of time, but ends up at around the same equilibrium value as without a vaccine. Correspondingly, the number infected decreases. The fraction of people having the disease after it establishes itself becomes less.

For population C the effect is more dramatic. Because the recovery rate is already higher than for A, the vaccines will help end the disease quickly after it is introduced. The number of infected go to zero, while the rest go to either R and S. Instead of the susceptible and recovered flattening out at quite different equilibrium values, they quickly move towards an equal fraction of individuals at equilibrium.

It is interesting to note that after our model reaches an equilibrium state, with I going to zero, we will still have people continuously move between R and S. The reason for this is that in the basic case, R only received people from I and when the disease died $I = 0$, there were no new people, and R gradually lost everyone back to S which would converge towards the total population N. Now R and S exchange people independent of I, so they will reach a stable value even after the disease is over. This is obviously not realistic, as vaccination programs would be ended if the disease is eradicated.

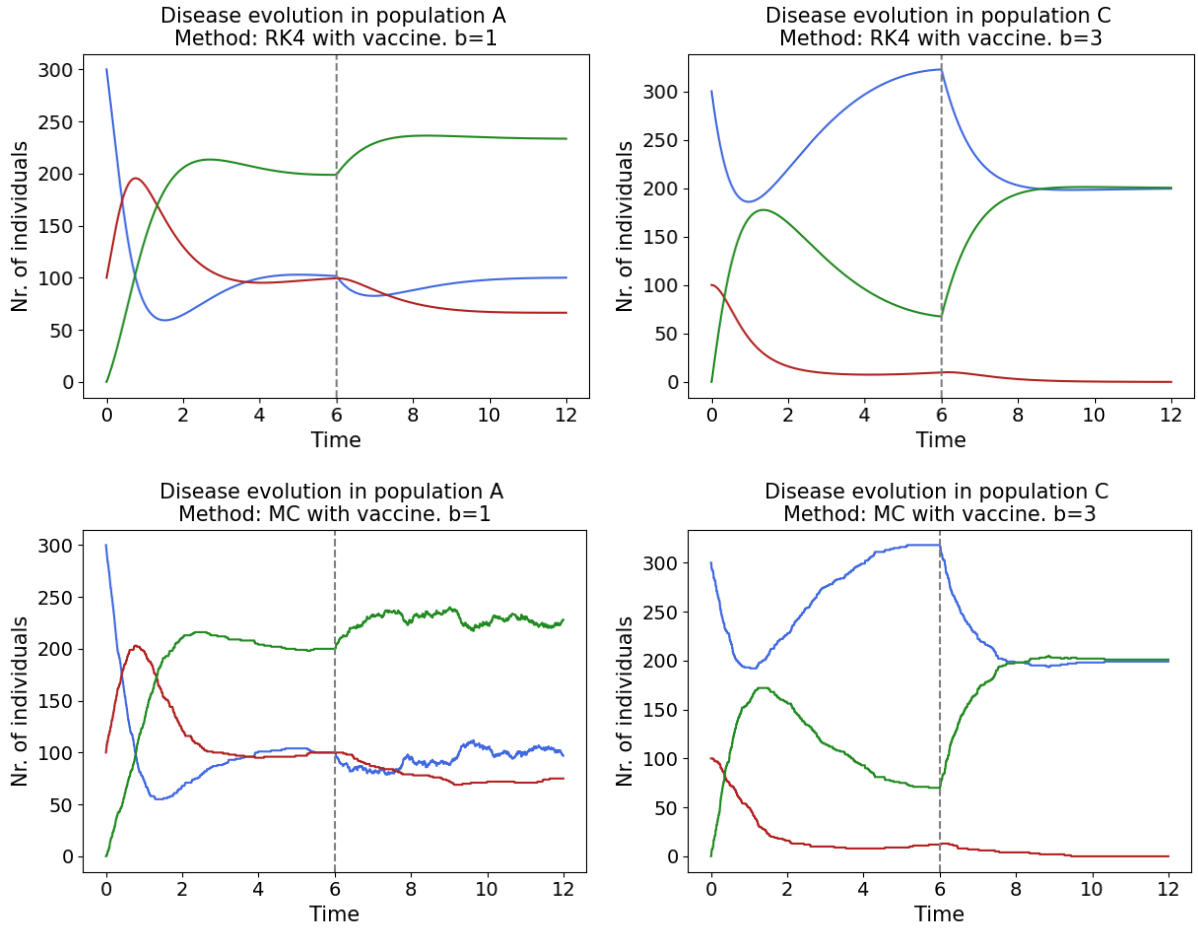

Figure 9: Time evolution of number of susceptible, infected and recovered individuals in population A and C for $b = 1$ and $b = 3$ (rate of recovery), computed using Runge-Kutta 4 and Monte Carlo. The SIRS model with vaccination, where $f = 0.5$ after $T = 6$.

4.5 Disease evolution with a combined model

The results from the time development of scenario 5 is shown in figure 11, with label explanations in figure 10. To the left we have population A, and population C to the right. Top row is RK4 and bottom row is MC. As stated in section 3.3.5, the parameters used are the same as for scenario 2, 3 and 4.

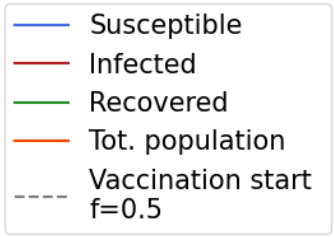

Figure 10: Labels for *Disease evolution with a combined model*

First, we notice the effect of vital dynamics. In the beginning the population will decline both A and C. This is due to the higher infection rates given by seasonal variation. However, the population will rise again as infection rates decline. This is because we have a birth rate that is higher than the normal death rate. The seasonal variation is more visible in population A. The population begin to decline towards the end, as the infection rates once again rise for winter time. This is most noticeable using the RK4 model, but the MC model hint about the same tendency. In addition, we clearly observe that the infection rates are at their highest in the beginning and end of the season, for both

RK4 and MC. In population C, with a three times larger recovery rate, the disease does not manage to establish itself. The seasonal variations become irrelevant, as the number of infected goes to zero, and they are unable to further spread the disease. The population will just continue to increase naturally.

We can clearly see the great effect of a vaccine. In population A it seems to reduce the infection rate greatly. For population C, it was possibly what killed off the disease completely. The infection rates were already very low by the time the vaccines were introduced (middle of summer). This means it only affected the interaction between susceptible and recovered, which is a feature of this model, and not necessarily realistic, as explained in the previous section. In our model we started the vaccination at $T/2$, which for e.g. a flu is rather late. The earlier a vaccine is distributed, the faster will the transmission of the disease decrease.

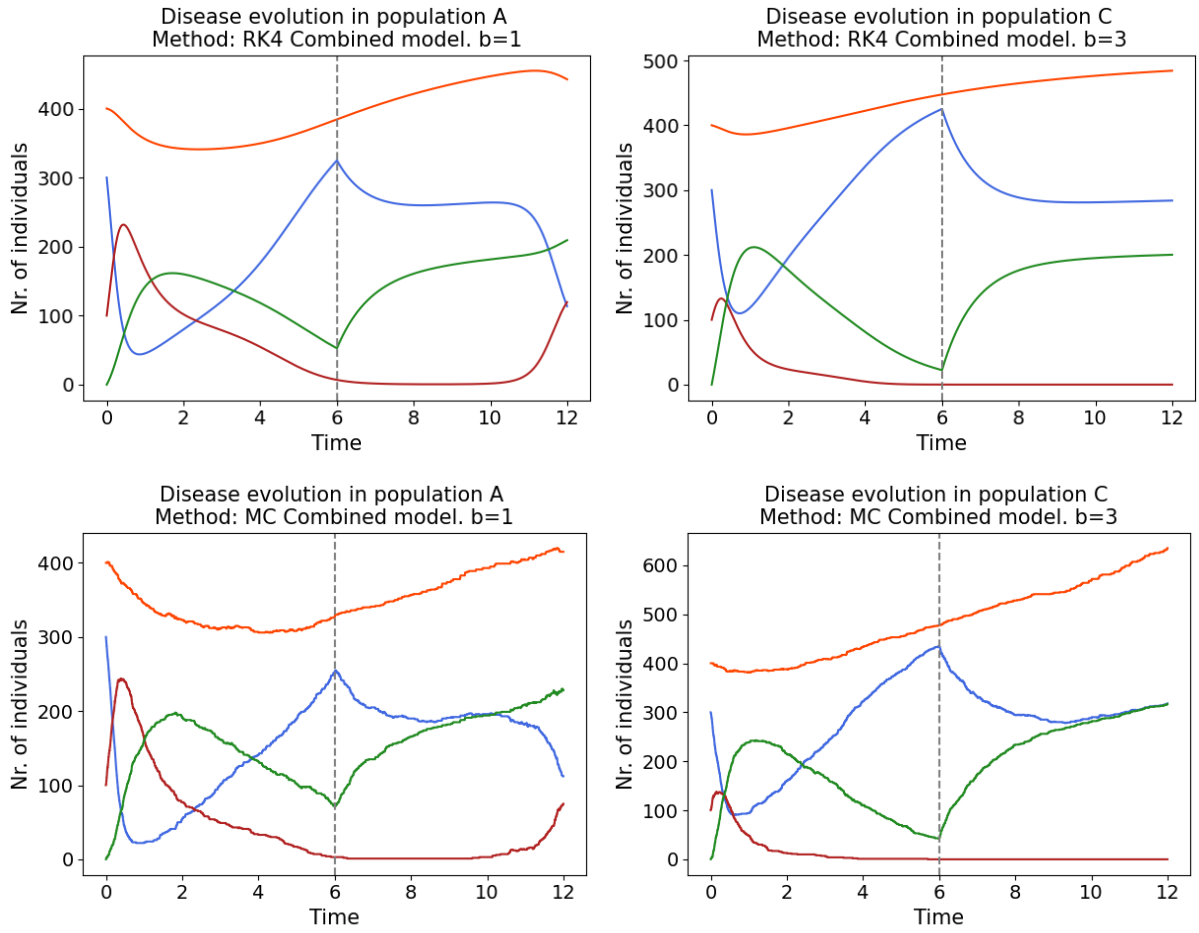

Figure 11: Time evolution of number of susceptible, infected and recovered individuals in population A and C for $b = 1$ and $b = 3$ (rate of recovery), computed using Runge-Kutta 4 and Monte Carlo. The disease evolution is a SIRS model with vital dynamics, seasonal variation and vaccination, with birth rate $e = 0.25$, death rate $d = 0.2$, death rate of infected people due to the disease $d_I = 0.35$, $a(t) = 4\cos(0.5t) + 4$ and a vaccination start after $T = 6$ with $f = 0.5$.

5 Conclusion

In this project we have looked into the SIRS-model, modelling it with both an ODE deterministic approach using 4th order Runge-Kutta, and a stochastic Monte-Carlo model which may be a more realistic approach when encountering an unpredictable disease. For the simplest version of the SIRS-model, both methods achieved to provide equilibrium values of the three groups (S, I and R), corresponding to the ones calculated analytically. We also observed that criteria $b < a$ were accurate in our simulation, that the disease did not establish itself in the population when b were high enough.

Then, we looked into the effect that different extensions had when applied separately to the SIRS-model. With vital dynamics the population were no longer constant, and because of a higher birth rate than death rate, the population grew despite the disease, when $b > 1$. The evolution of the disease were fairly similar as the basic SIRS model. With seasonal variation we observed an expected pattern of a higher infection peak at the beginning and end of the season, at least when the recovery rate was not too high. For a higher recovery rate, $b = [3, 4]$, the seasonal impact actually prevented the disease from establishing itself in the population, and we obtained an result similar to the basic SIRS-model. Another appliance which turned out to be very effective to fight the disease when $b < a$, was vaccination. The effects were seen immediately, and it clearly reduced the fraction of infected people if/when the disease were established in the population. Like seasonal variations, the disease died out for $b = [3, 4]$. However, the different groups reached a steady-state much faster. Lastly, we studied a disease evolution with a combined model. As expected, the more complex the model becomes, as with the combined model, the harder it was to to keep track of the individual impacts. Overall, it is the introduction of a vaccine that has he biggest impact in stopping the transmission of the infectious disease. The earlier the vaccine is distributed, the better.

A weakness with our model is that the populations are fairly small, and the disease data is not based on a real disease. Our results can therefore not be directly compared to a real disease, and it is hard to determine if our model is able to predict how a disease actually behave over a period of time. However, we believe the learning outcome is still transferable and useful for understanding simple diseases. A further development of the work is to make simulations for larger populations. Future work should implement data from real epidemics for adapting our model, possibly using methods from supervised learning to adjust our input parameters, in particular the different rates of transfer between the compartments in the model.

The work done for this report have been interesting and educational, and seem even more valuable at a time were most of us get to feel the vulnerability of living in a global society undergoing a pandemic. The frequency of epidemics and/or pandemics will probably just increase as the population of the world continues to grow. Developing new, and better disease models, that can be applied on new/unknown/unpredictable infectious diseases is therefore of great importance and necessity.

References

[1] Wikipedia. *Compartmental models in epidemiology*. Visited: 09.12.20 https://en.wikipedia.org/wiki/Compartmental_models_in_epidemiology.

[2] Morten Hjorth-Jensen (UiO). *FYS4150 Project 5: Disease Modeling*. http://compphysics.github.io/ComputationalPhysics/doc/Projects/2020/Project5/DiseaseModeling/pdf/DiseaseModeling.pdf.

[3] Aron J. Nordberg Anna Eliassen and Kristina Othelia L. Olsen. *FYS4150: Project 5 - GitHub*. https://github.com/kristinaothelia/FYS4150/tree/master/Project5.

[4] Morten Hjorth-Jensen. *Computational Physics Lectures: Ordinary differential equations*. 05.10.2017 pp.(8-9) http://compphysics.github.io/ComputationalPhysics/doc/pub/ode/pdf/ode-print.pdf.

[5] Wikipedia. *The Runge–Kutta method*. Visited: 10.12.20 https://en.wikipedia.org/wiki/Runge%E2%80%93Kutta_methods#The_Runge%E2%80%93Kutta_method.

[6] Arnt Inge Vistnes. "Physics of Oscillations and Waves". In: Springer International Publishing AG, 2018, p. 68. ISBN: 9783319723136.

Appendix A System equilibrium

This appendix is based on the section *Theoretical background and description of the system* chapter in the project description [2].

The SIRS model allows for continuous flow of individuals from one category to the next in a cyclical way. $S \to I \to R \to S$. This means that after a certain time the system will stabilize and reach an equilibrium state. We will now investigate what requirements there are of the different rates that would result in the establishment of a disease within a population.

The steady state of the SIRS model is found by setting both equations in (5) equal to zero. Let $s^* = S/N$, $i^* = I/N$ and $r^* = I/N$ be the fractions of each group of the population after reaching equilibrium. We then get

$$\begin{aligned} s^* &= \frac{b}{a}, \\ i^* &= \frac{1 - \frac{b}{a}}{1 + \frac{b}{c}}, \\ r^* &= \frac{b}{c}\frac{1 - \frac{b}{a}}{1 + \frac{b}{c}}. \end{aligned} \tag{24}$$

Each fraction must be a number between 0 and 1, and the three fractions must add up to 1. Thus the equations in (24) suggest that the rate of recovery b must be less than the rate of transmission a for the number of infected people at equilibrium to be greater than zero. In other words, the disease establishes itself in the population only if $b < a$. This is intuitive as the disease must transmit faster within the population than the population recovers from the disease.