Disease Modeling Using the Classical SIRS Model

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December 19, 2020

Abstract

We develop a Monte Carlo simulation of the spread of an infectious disease. The aim of creating such a simulation is to investigate how a disease spreads throughout a given population over time. As a learning exercise, we include a deterministic approach using the fourth-order Rung-Kutta method. Four populations with three modifications to the simple SIRS model are studied; vital dynamics, seasonal variations and vaccine. Our simulations/findings show ...

1 Introduction

Nothing has killed more human beings than infectious diseases. Whenever they happen, they significantly impact global economies and public health. Therefore, it is of great significance to use mathematical models to analyze the transmission and control of infectious diseases. By using these models, we can make predictions about e.g. the total number infected, the duration of an epidemic, or whether or not a certain disease has the capacity to establish itself within the population. These analyzes may help governments decide which public health interventions to set in place to mitigate the disease.

Compartment models can be used to simplify the mathematical modelling of infectious diseases. In these models, the population is assigned to different compartments, e.g. S, I or R (Susceptible, Infectious, or Recovered) which people may progress between. We investigate the SIRS model. Here, the order of labels shows the flow pattern between the compartments; susceptible, infectious, recovered than susceptible again.

The aim of this work is to develop a Monte Carlo simulation of the spread of an infectious disease for the purpose of investigating how a disease spreads throughout a given population over time. We begin with running the SIRS model with ordinary differential equations (which are deterministic), which we solve using the fourth-order Runge-Kutta method. Once this is done, we switch to Monte Carlo methods to use randomness to solve the problem. This is a more realistic approach but it is much more complicated to analyze. Further, we modify or simple SIRS model to add vital dynamics, seasonal variations or vaccination. We run these modified models using both the fourth-order Runge-Kutta method and Monte Carlo methods.

The outline of this report is as follows: Section 2 gives a short review of the SIRS model and some modifications to include more details in the model. In section 3, we briefly explain how we have implemented the methods and which parameters we have used. Then we present our results in section 4, before we discuss our findings in section 5. Last, some concluding remarks are given in section 6.

2 Theory

2.1 The SIRS model for transmission of an infectious disease

The SIRS model is a modification of the traditional SIR model for epidemics introduced by Kermack and McKendrick [1] almost 100 years ago, where immunity lasts only for a short period of time. We consider an isolated population of N people which are divided into three classes:

- Susceptible (S): represents the individuals not yet infected with disease at time, t, or those susceptible to the disease of the population.
- Infected (I): denotes the individuals of the population who are currently infected with the disease and are capable of spreading the disease to those in the susceptible category.
- Recovered (R): represents those who have been infected in the past and have developed an immunity to the disease. Thus, the people in this category are not able to be infected again or to transmit the infection to others.

The flow of this model is cyclic and may be considered as follows: $\mathcal{S} \to \mathcal{I} \to \mathcal{R} \to \mathcal{S}$. We use the rate of transmission, a, the rate of recovery, b, and the rate of immunity loss, c, to help describe the flow of people moving between the three classes. The population is assumed to be fixed and mix homogeneously so that

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

First, we assume that the dynamics of the epidemic occur during a time scale much smaller than the average person's lifetime. Hence, we neglect the effect of the birth and death rate of the population. With these assumptions, we construct a set of coupled differential equations from the classical SIRS model:

$$\begin{aligned} S^{'} &= cR - \frac{aSI}{N} \\ I^{'} &= \frac{aSI}{N} - bI \\ R^{'} &= bI - cR \end{aligned} \tag{2}$$

Note that if we study a small population, we may choose to write aSI instead of $\frac{aSI}{N}$ since the number of susceptible which become infected depend more on the absolute number of infected people rater than the infected fraction of the population.

This set (equation (2)), does not have analytic solutions like the closely-related SIR model, but we can easily obtain the equilibrium solutions. We use the constraint in equation (1) to reduce this three dimensional system into a two dimensional one, so that we can omit the equation for $R^{'}$:

$$\begin{aligned} S^{'} &= c(N - S - I) - \frac{aSI}{N} \\ I^{'} &= \frac{aSI}{N} - bI. \end{aligned} \tag{3}$$

The steady state solution is found by setting both equations in equation (3) equal to zero. We let s, i and r denote the fractions of people in S, I and R, respectively. Then we find that the fractions of people inn each group at equilibrium are:

$$\begin{aligned} s^* &= \frac{b}{a}, \\ i^* &= \frac{1-\frac{b}{a}}{1-\frac{b}{c}}, \\ r^* &= \frac{b}{c}\frac{1-\frac{b}{a}}{1+\frac{b}{c}}. \end{aligned} \tag{4}$$

Note that the asterisk is used to signify that these fractions are at equilibrium. Each fraction must be a number between 0 and 1, and the three fractions must add up to 1. Hence, the equations in equation (4) suggest that the disease establishes itself in the population only if $b < a$.

In the following, we extend our simple model to include more details about the population and disease.

2.1.1 Vital dynamics

We add vital dynamics to our system so that the model can describe the spread of diseases which occur over longer stretches of time. Let e be the birth rate. d the death rate, and d_I be the death rate of infected people due to the disease, then the modified differential equations are given by:

$$\begin{aligned} S^{'} &= cR - \frac{aSI}{N} - dS + eN \\ I^{'} &= \frac{aSI}{N} - bI - dI - d_I I \\ R^{'} &= bI - cR - dR \end{aligned} \tag{5}$$

Where we have assumed that all babies born into the population are initially susceptible.

2.1.2 Seasonal variation

Some diseases are seasonal, or put differently: The rate of transmission depends largely on the time of year. Common cold viruses are more prevalent during winter and childhood diseases are strongly correlated with the school calendar. As a consequence, for many diseases, one should consider a rate of transmission which oscillates. We can a periodically varying contact rate by letting the rate of transmission be given by

$$a(t) = A\cos(\omega t) + a_0. \tag{6}$$

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

2.1.3 Vaccination

For diseases with available vaccinations, people can move directly from S to R, breaking the cyclic structure of the SIRS model. We need to make a couple of assumptions. First, we assume that a susceptible individual's choice to take the vaccine does not depend on how many other susceptibles are vaccinated. Further, we assume that the rate of vaccination, f can depend on time, since this rate may oscillate during the course of a year and/or increase as awareness and medical research increases. Our system of differential equations now become

$$\begin{aligned} S^{'} &= cR - \frac{aSI}{N} - f \\ I^{'} &= \frac{aSI}{N} - bI \\ R^{'} &= bI - cR + f. \end{aligned} \tag{7}$$

where we define

$$f = f(t) = min\{f_0, f_0\frac{t - f_t}{10}\}, \quad t \geq f_t, \tag{8}$$

as our time dependent vaccination function, with f_0 as the initial rate of vaccination and f_t as the time at which the vaccination begins.

3 Method

3.1 Populations

We investigate the effect of increasing the rate of recovery b and crossing the "threshold" in four different populations. Each population consists of 400 people, 100 of which were initially infected and 300 of which were initially susceptible. The rate of transmission and rate of immunity loss is fixed between populations. We let the rate of recovery be varied because it is the one parameter which can be reasonably controlled by the actions of human society. Table 1 lists the set of parameters for the four different populations. We treat the population as a continuous variable.

Table 1: Parameters for the investigated populations.

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

3.2 Fourth order Runge-Kutta method

We use the well known fourth-order Runge-Kutta method to solve the differential equations. For a function $f(t, y)$, the fourth-order Runge-Kutta method is given by

$$y_{i+1} = y_i + \frac{1}{6}(k_1 + 2k_2 + 2k_3 + k_4), \tag{9}$$

where

$$
\begin{aligned}
k_1 &= hf(t_i, y_i) \\
k_2 &= hf(t_i + \frac{1}{2}h, y_i + \frac{1}{2}k_1) \\
k_3 &= hf(t_i + \frac{1}{2}h, y_i + \frac{1}{2}k_2) \\
k_4 &= hf(t_i + h, y_i + k_3).
\end{aligned} \tag{10}
$$

We see that the algorithm consists in first calculating k_1 with t_i, y_1 and f as inputs. Then, the step size is increased by $h/2$ and k_2, k_3 and finally k_4 is calculated. The global error goes as $\mathcal{O}(h^4)$

3.3 Monte Carlo simulation

In our Monte Carlo simulation we use the idea of randomness and define a set of transition probabilities for the possible moves a person can take from one state to another. From equation (2) we see that in a small time step, Δt, the number of people moving from $\mathcal{S}$ to $\mathcal{I}$ is approximately $\frac{aSI}{N}\Delta t$. Likewise, the number of people moving from $\mathcal{I}$ to $\mathcal{R}$ is approximately $bI\Delta t$ and $cR\Delta t$ move from $\mathcal{R}$ to $\mathcal{S}$. We assume that *at most* one person moves from a given group to another

$$
\begin{aligned}
\max\left\{\frac{aSI}{N}\Delta t\right\} = \frac{a}{N}\left(\frac{N}{2}\right)^2\Delta t = \frac{aN}{4}\Delta t, \\
\max\left\{\frac{aSI}{N}\Delta t\right\}, \\
\max\left\{\frac{aSI}{N}\Delta t\right\},
\end{aligned} \tag{11}
$$

where the time step is given by

$$
\Delta t = \min\left\{\frac{4}{aN}, \frac{1}{bN}, \frac{1}{cN}\right\}. \tag{12}
$$

We reinterpret the values $\frac{aSI}{N}\Delta t$, $bI\Delta t$ and $cR\Delta t$ as transition probabilities:

$$
\begin{aligned}
P(S \to I) &= \frac{aSI}{N}\Delta t, \\
P(I \to R) &= bI\Delta t, \\
P(R \to S) &= cR\Delta t.
\end{aligned} \tag{13}
$$

A random number between 0 and 1 is generated, and if the number is less than the probability for the move, the move is taken.

4 Results

We will here present the results of our simulations. In all simulations done, a fixed set of parameters were used, whose values can be found in table (**??**).

Parameter	S_0	I_0	R_0	a	c
Value	300	100	0.0	4	0.5

Table 2: The set of parameters that stayed constant during simulations.

4.1 Simple SIRS model

As explained in section 3.1, we want to see how a disease spreads in four identical populations when the only parameter that changes is the infection rate b of the disease. The results can be seen in figure (1), where we compare both a simulation based on the Runge Kutta 4th order method and a method using Monte Carlo simulation.

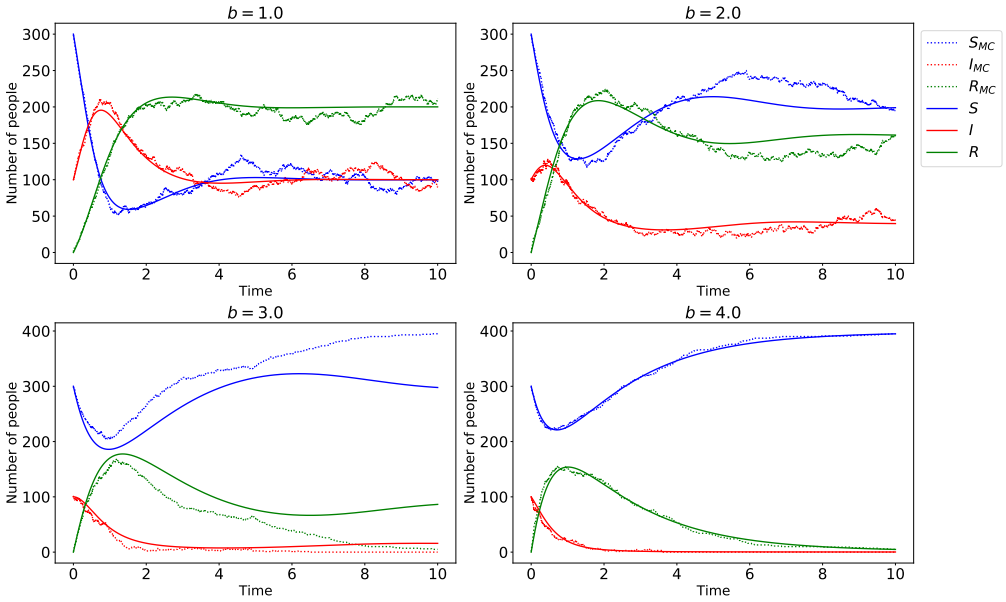

Figure 1: Monte Carlo and RK4 simulation of base SIRS model for varying b values.

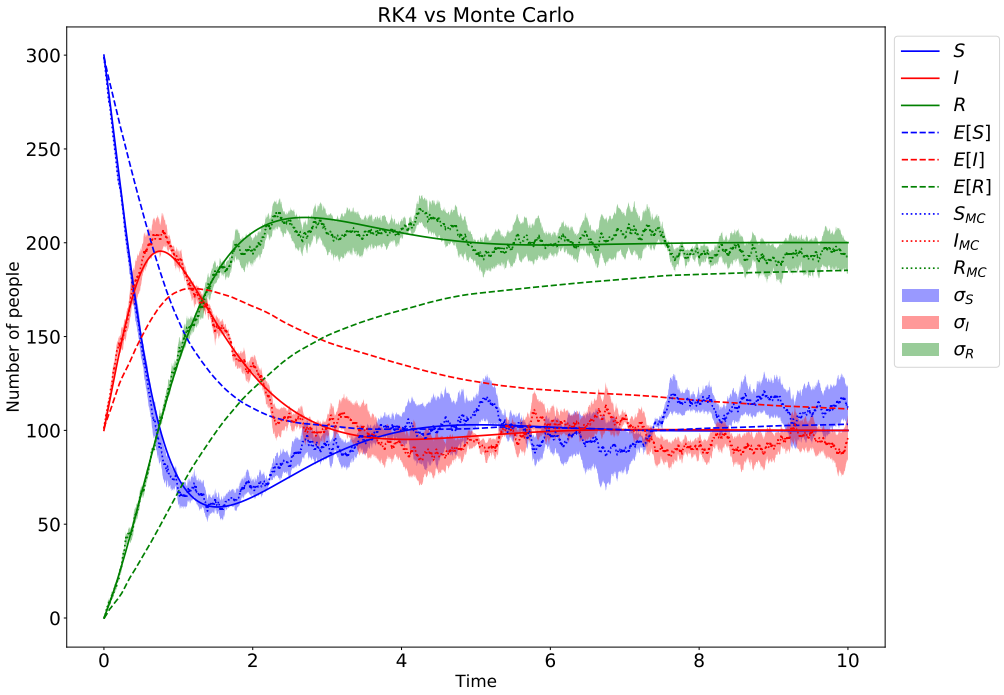

Figure 2: A closer look at the results of RK4 and MC for a sample population with $b = 1$. Also shown is the standard deviation σ over several simulations using the MC method and the corresponding expectation values E.

4.2 SIRS model with vital dynamics

We know want to look at what would happen to a sample population where vital dynamics have been added. In this case, we wish to see how the deadliness of the disease impacts the population as a whole.

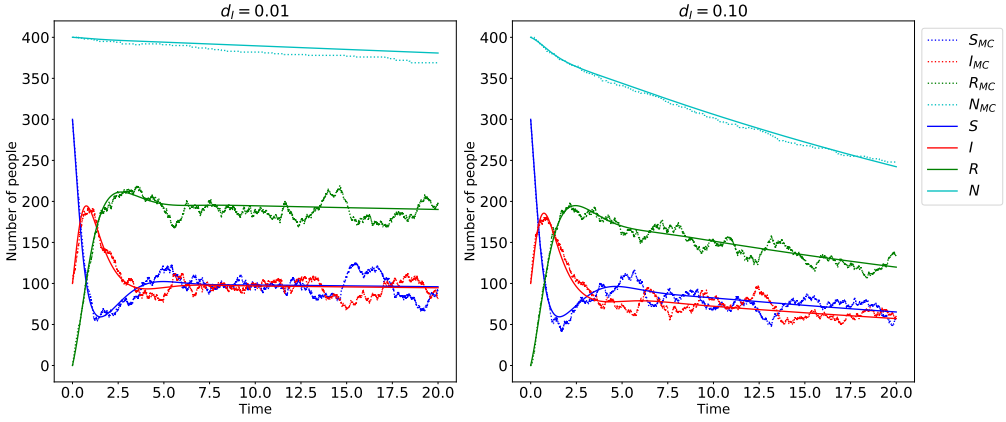

Figure 3: Monte Carlo and RK4 simulations of SIRS model with vital dynamics for varying d_I. $b = 1$, $d = 0.0003$ and $e = 0.0005$ for both populations. N is the total population.

4.3 SIRS model with seasonal variation

We now look at the effect of adding a cyclic function dependent on time t that governs the rate of transmission $a = a(t)$ as described in equation (6).

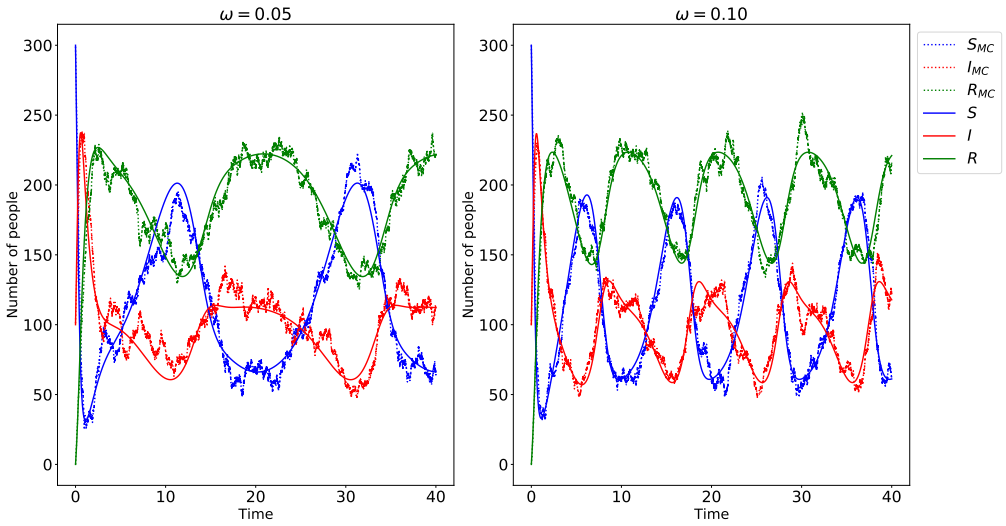

Figure 4: Monte Carlo and RK4 simulations of SIRS model with seasonal variation on the rate of transmission for varying w. $A = 2$ for both populations.

4.4 Vaccination

Finally, we want to see how the addition of a vaccine would impact the spread of a disease, and the recovery of the population. Here we are using the linear function f as described by equation (8).

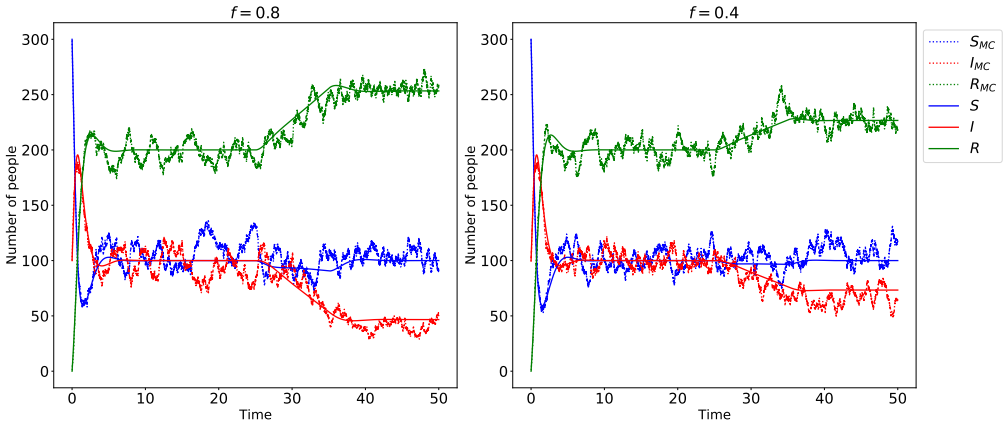

Figure 5: A comparison of SIRS model with added vaccination parameter f, using both RK4 and MC simulation. Vaccination starts at $f_t = 25$

5 Discussion

5.1 Spread of disease in a sample population

In figure (1) we can see how the value of b affects the equilibrium states of the systems. When $b < a$, the disease can infect more people, which will in turn lead to more recovered and less susceptible people, until enough time has elapsed and the system reaches a natural equilibrium. As we increase b and $b \to a$, we can see how the number of infections drops until it reaches zero, effectively killing the disease. Of course, as the levels of infected drop, the amount of people who will eventually catch the disease and then later recover will naturally decrease, and after enough time has passed, you will be left only with people who are susceptible, as immunity is gradually lost in the population. This is an expected outcome, and all four sub plots support this.

We can also observe the inherent randomness of the Monte Carlo simulation, but although the Monte Carlo lines some random noise, it is still possible to see that the Monte Carlo simulation more often then not follows the lines made by the Runge Kutta method quite well.

Looking closer at the Monte Carlo method in comparison to the Runge Kutta 4 method in (2), we can see how the Monto Carlo simulations may vary for each simulation, but will generally stay similar to ODE simulation, with only a few cases where there is no overlap between the two methods. This lack of overlap in some places might be because the number of simulations done was not enough, and it is possible that more runs would yield a bigger standard deviation. It is also possible to see what looks like the expectation values converging. In general, one would expect the Monte Carlo method to be slightly more useful for real life simulations of diseases, as the stochastic nature of the method allows for calculations of a range of possible outcomes, instead of a single deterministic one.

5.2 Addition of vital dynamics

When we add the possibility of a fluctuating total population due to natural deaths, infected deaths and the birth of new members, we would expect to see either an increase or decrease in population, depending of course on the rates at which people die and new babies are born, and of course the rate at which an infection kills its host. In the case of figure (3), we can see how a disease with a $d_I = 10\%$ can devastate a population in a short amount of time. When $d_I = 1\%$ however, the infection is more manageable, even though some part of the population will die. In either case, the loss of population should continue until the system reaches equilibrium. As is suggested by the plot for $d_I = 0.1$, when the population reduces, so must the number of infected and the susceptibles, as there is naturally less people in the system.

5.3 Seasonal variations

Many diseases will have transmission rates which vary with the seasons. This could be due to a plethora of factors, but it is usually because of what method a disease uses to spread itself, and whether or not said method is affected by outside temperature, air humidity or other such seasonal changes. Our results, as seen in (4), show how a disease could periodically vary with time. In our case, since we are using a cyclic function to model our variations, we see that the disease in question has a cyclic nature, reaching a low in infected I and a high in susceptibles S around every 10 units of time.

5.4 Vaccination

Introducing vaccinations to combat a disease should more often than not significantly reduce the number of infected, and also reduce the number of susceptibles at any on time. Our data somewhat supports these thoughts. Looking at the results obtained in figure (5), we can see how after a vaccine is introduced at time $f_t = 25$, the number of infected is reduced accordingly with the vaccination rate and correspondingly, the amount of recovered people increases. This does make sense, as anyone infected should recover after being administered with a vaccine. The number of susceptibles does not decrease however. From our model, it seems that only those that are infected get the vaccine.

6 Conclusion

References

[1] W. O. Kermack, A. G. McKendrick, and G. T. Walker, "A contribution to the mathematical theory of epidemics", *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character*, vol. 115, no. 772, pp. 700–721, 1927. DOI: 10.1098/rspa.1927.0118. eprint: https://royalsocietypublishing.org/doi/pdf/10.1098/rspa.1927.0118. [Online]. Available: https://royalsocietypublishing.org/doi/abs/10.1098/rspa.1927.0118.

A Appendix

A.1 Source code

Github repository with codes and figures can be found at `https://github.com/idadue/ComputationalPhysics/tree/master/project5`.