Monte Carlo simulations of the SIRS model

Gunnar Thorsen Liahjell

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Abstract

A Monte Carlo implementation of the SIRS-model is implemented and compared to the solving of the SIRS models ODEs by Runge-Kuttas 4th order solver. The MC-implementation is found to be in good agreement with the classical solutions provided by RK4, yet it is found to be sensitive to the size of the population in question.

Introduction

The SIR-model was proposed by Kermack and McKendrick in 1927 [4] to model the spread of diseases. The model was successful in modelling outbreaks of diseases on a large scale, but as these equations considers populations as continuous and yields perfect steady state solutions the introduction of monte carlo simulations may be a more realistic model. With Monte Carlo modelling one can make use of discrete and random transitions to better capture the dynamics of disease spreading. The goal of this paper is to produce such an algorithm, that keeps the already useful aspects of the classical SIR, but improves upon them and gives more information. Both MC and the classical ODEs solved by RK4 will be implemented and a test population with an initial state of 400 people where 300 are healthy and 100 infected, will be used to test both methods,to see how they differ in handling inputs like vital dynamics, variation and vaccinations will be compared.

The sir model is presented in the Theory section, an overview of the general concepts that form the basis for the programs used are worked through in the methods section,the results and interpretations are presented in Results and Discussion before the findings are discussed in Conclusions.

Theory

The SIRS model used in this paper is a slight modification of the compartmental SIR model. Both models split the population in question into three compartments, namely

1. Susceptible (S): those susceptible to infection
2. Infected (I): those who are currently infected with the disease
3. Recovered (R), those who have been infected in the past and have developed an immunity to the disease

The difference between the two, as the names suggest is the flow of people between the compartments. In the SIR model one can go through the model as $S \rightarrow I \rightarrow R$ witch fits

for diseases like i.e measles and rubella that result in a lifelong immunity after the infection. Whereas the SIRS model flows like $S \to I \to R \to S$ allowing for the loss of immunity, resulting in a better model for diseases like the flu.

Assuming a rate of transmission a, a rate of recovery b and a loss of immunity c one can construct a set of coupled differential equations that describe the flow between the compartments:

$$S' = cR - \frac{aSI}{N}$$
$$I' = \frac{aSI}{N} - bI$$
$$R' = bI - cR$$

Given initial conditions these equations can be approximated by a numerical ODE solver.

This is the simplest version of SIRS but a perk of this model is that it can very easily modified. The parameters a , b and c can be made functions of time and extra terms can be added. For example can a be made into a sine function accounting for seasonal variations in infection pressure. The transmission rate, b, can be lowered due to increased awareness about a disease which was the case with i.e the governmental campaigns around the world during pig flu. the rate of recovery can be made better by i.e medical advancement or worse by for instance overuse of antibiotics.

Vital dynamics

For studying populations over longer periods of time one needs to take in to account the vital dynamics of the populations, that is, the birth, and death rate, and also the possibly increased death rate of infected individuals. This can be done by introducing three new variables to the ODE's. By adding d, the death rate, e, the birth rate and d_i the increased death rate of infected people the new equations reads

$$S' = cR - \frac{aSI}{N} + eN - dS$$
$$I' = \frac{aSI}{N} - bI - (d + d_i)I$$
$$R' = bI - cR - dR$$

Seasonal variation

Many diseases like the flu have seasonal fluctuations increasing and decreasing the infection pressure as the conditions for the diseases spread gets better or worse. To take this into account in our model one simply allows a to be a function of time. For instance setting

$$a = a_0 + sin(t\omega)$$

Vaccination

A third thing that can have an impact on the spreading of a disease is vaccination. This will move people from S to R. The vaccination parameter f is here set to be a function of time and is a fraction of the total number of people as this seems to be a reasonable assumption [2], making the final equations:

$$S' = cR - \frac{aSI}{N} + eN - dS - fN$$
$$I' = \frac{aSI}{N} - bI - (d + d_i)I$$
$$R' = bI - cR - dR + fN$$

Note on time

Time in this paper is of arbitrary units, as the coefficients of the model (a,b,c..) are of inverse time, they will decide the timescale. The coefficients are in turn decided by the disease and the population in question. HIV would for instance have a meaningful timescale of years[1], while the flu (one season) would have a much shorter timescale, and the spread of flu in a densely populated area will be faster than in a sparse population.

Methods

RK-4

To solve the differential equations produced by the SIRS model, a Runge-Kutta-4 algorithm is implemented. The Runge-Kutta 4 method makes use of taylor expansions and simpsons rule to make an algorithm with an error that goes as $O(h^4)$ ([3],p 251-252).

The general procedure goes as

1. Compute the slope at t_i: $k_1 = f(t_i, y_i)$
2. Use k_1 to approximate the slope at the midpoint: $k_2 = f(t_i + h/2, y_i + k_1/2)$
3. Use k_2 to adjust the slope at the midpoint: $k_3 = f(t_i + h/2, y_i + k_2/2)$
4. Use k_3 to approximate the slope at y_{i+1}: $k_2 = f(t_i + h, y_i + k_3)$
5. The slope of y_i is approximated by the weighted average of these slopes and $y_{i+1} = y_i + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4)$

MC-simulation

The Monte-carlo implementation is extracted from the differential equatioins by observing that in a small time step the amount of people moving from one compartment is given by

$$S \to I = \frac{aSI}{N}\Delta t$$
$$I \to R = bI\Delta t$$
$$R \to S = cR\Delta t$$

by making the size of the time step so that at most one person can move from one compartment to another by seeing that

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

$$max\Big\{bI\Delta t\Big\} = bN\Delta t$$

$$max\Big\{cR\Delta t\Big\} = cN\Delta t$$

and defining the timestep as

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

one can interpret the values as transition probabilities:

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

$$P(I \to R) = bI\Delta t$$

$$P(R \to S) = cR\Delta t$$

Adding vital dynamics and vaccination is done by the same procedure and adding

$$P(Birth) = eN\Delta t$$

$$P(Death_S) = dS\Delta t$$

$$P(Death_I) = (d + d_i)I\Delta t$$

$$P(Death_R) = dR\Delta t$$

$$P(Vaccination) = fN\Delta t$$

where N is the whole population, assuming that everyone gives birth to a healthy child.

Implementation

All programs used in the report can be found at https://github.com/gunnartl/fys4150/tree/master/project5

Results and discussion

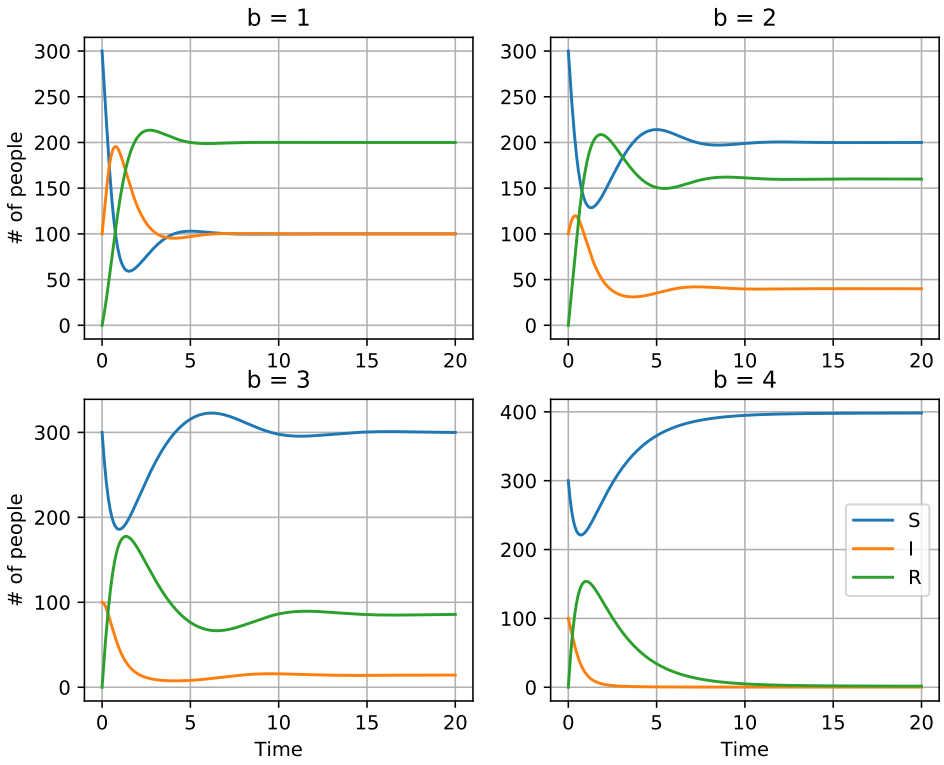

Figure 1: Development in the SIR compartments over time with increasing values of b, with the differential model

Figure 1 shows how different values of the recovery rate b affect the equilibrium values. As the recovery rate goes up, the infected part of the population at equilibrium goes down until it goes to zero at b = 4.

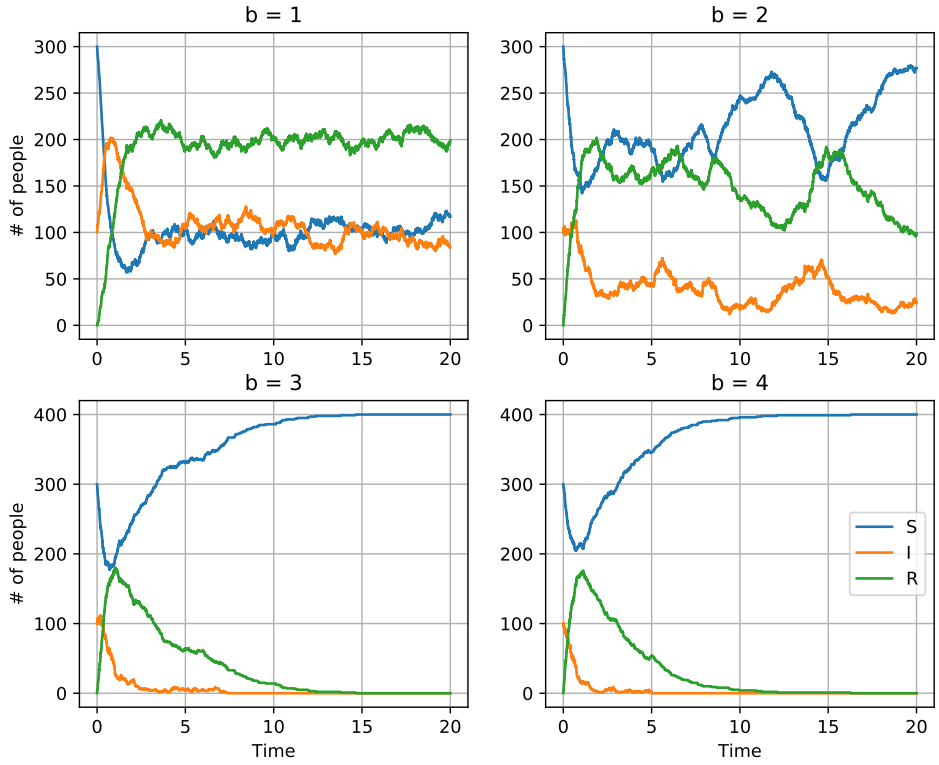

Figure 2: An example of a development in the SIR compartments over time with increasing values of b, with the Monte Carlo modell, and one cycle

Table 1: Table of expectation values and corresponding standard deviations for increasing b values. The values are calculated as an average over 1000 monte carlo runs of time = 150 and after allowing a equlibration time of 50

b	S	SD_s	I	SD_I	R	SD_R
1.00	100.27	11.56	99.75	10.48	199.98	9.15
2.00	205.43	26.47	39.10	11.72	155.47	20.29
3.00	400.00	0.00	0.00	0.00	0.00	0.00
4.00	400.00	0.00	0.00	0.00	0.00	0.00

Figure 2 and the values of table 1 for b values of 1,2 and 4 correspond rather well to the values produced by RK4 in figure 1, the expectation values are close to the ones in equilibrium state runge kutta and the fluctuations produced by the random walk in the Monte carlo simulation are described by the standard deviation. The monte carlo simulation with $b = 3$ goes to zero.

This is because of the random fluctuations in the monte carlo simulation. The general trend

is the same in 1,2, and 4 and as the SDs from 1 and 2 sugests the fluctuations are quite large compared to the population and in figure 3 they are almost as large as the part of the population that are infected. This results in a rather high chance of the infected population to get wiped out, which is the case with $b = 3$. If the compartments in the monte carlo gets to zero it Will stay zero, being that the transition probability is exactly zero. In the RK4 simulation the compartments can be between 0 and one an thereby survive longer and have a rise later.

Figure 3 shows that the developments are different for equal parameters a, b and c, but different populations sizes. Here the development in the larger population corresponds much better to the results produced by RK4 as the relative size of the fluctuations now are much smaller and the probability for the infected population to simply fluctuate to zero is much smaller.

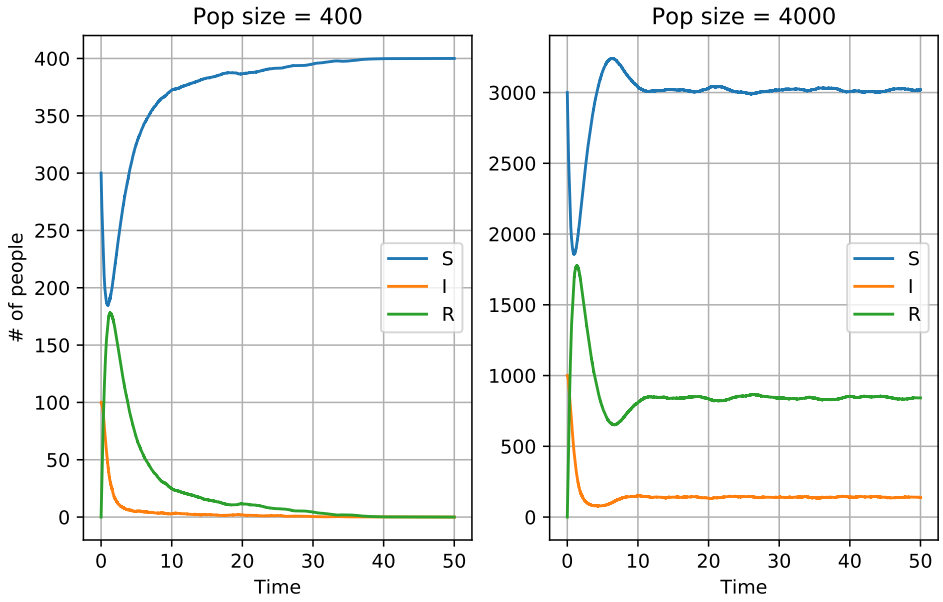

Figure 3: Average over 100 MC cycles with $a = 4$, $b = 3$ and $c = .4$, for populations of 400 and 4000

Including Vital dynamics

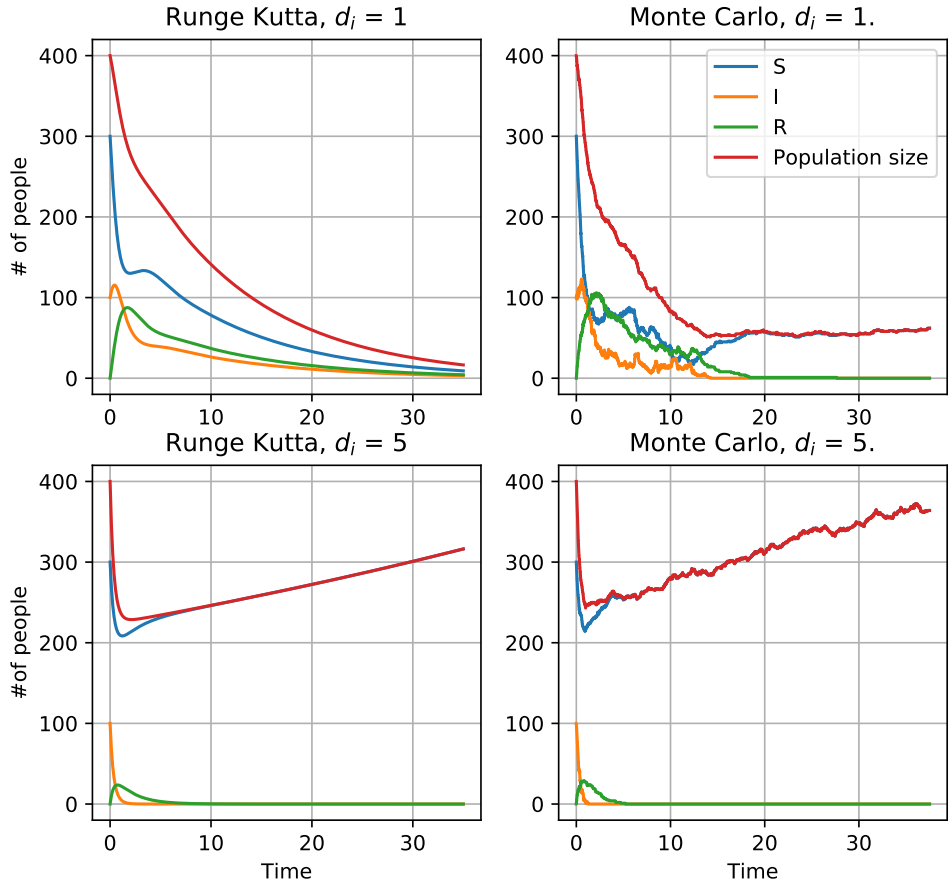

Figure 4: Development in the compartments and the size of the population with included vital dynamics. Parameters $a = 4$,$b = 1$,$c = .5$,$e = 0.04$, $d = 0.03$ and varying $d_i = 1$ and 5

Figure 4 shows some effects of adding vital dynamics to the MC- and RK4 solvers. These examples show in both cases, a very deadly infection spreading in a population. With $d_i = 1$ the population decreases rapidly and the RK4 model predicts the population getting whiped out, while the monte carlo simulation disagrees and predicts around 50 people surviving. The lower figures show a even more deadly disease rapidly decreasing the population then followed by an increase as both models agrees upon.

The difference in the upper figures are caused by the same effects as in the previous segment. The population goes down and discrete steps in Monte carlo kills off the infected before the whole population goes down.

The difference between the different diseases, the lower and upper row, is caused by the balance of the parameters. With $d_i = 1$ people die rapidly of the disease, but still infect people

before they die, with $d_i = 5$ people wont have enough time to infect others before dying from the disease themselves, thereby removing the disease from the population, and leaving births and natural deaths to control the size of the population causing the exponential rise after the disease is gone.

Adding seasonal variance

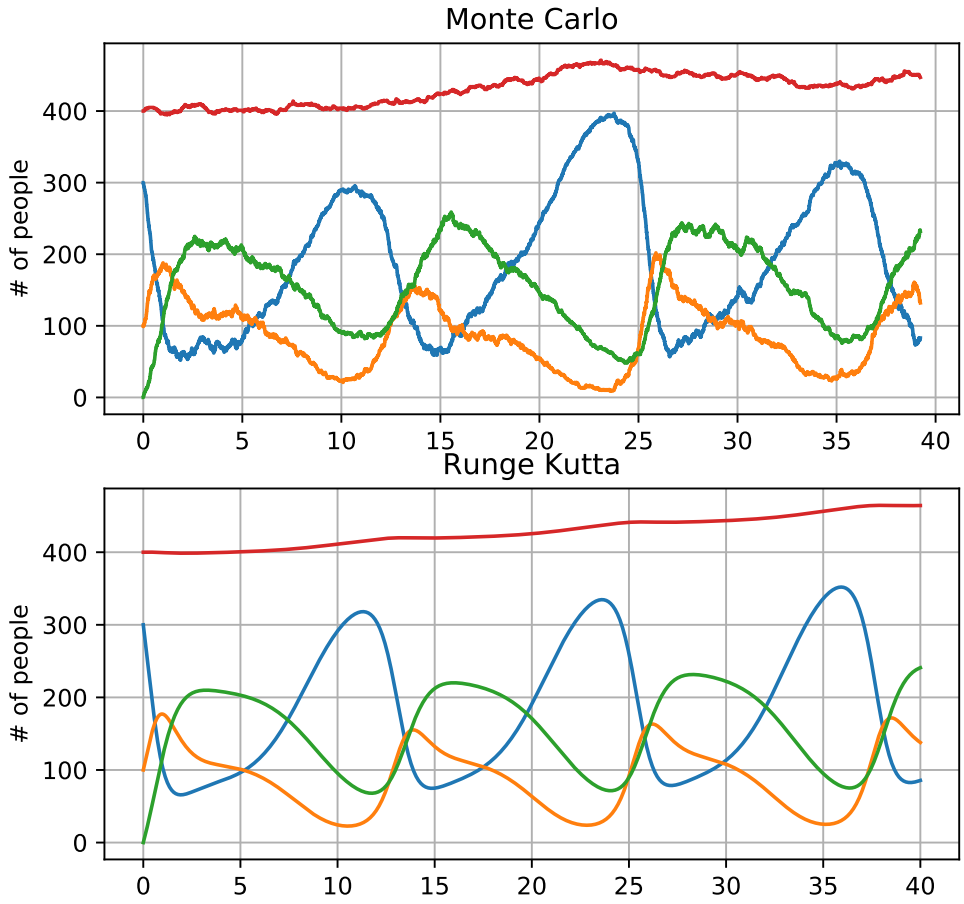

Figure 5: Development of the compartments with $a = 3 + 2sin(t\omega)$, $b = 1$ and $c = 0.5$

Figure 5 shows a possible development of a disease with seasonal variance in the infection pressure. If you think of the time axis as months it can fit fairly well with a the flu. 10 months beeing summer, and the top at around 13-14 being fall, followed by and increase in immunity and less sickness in winter-spring. Both models agree pretty good on the development over time.

Adding vaccination

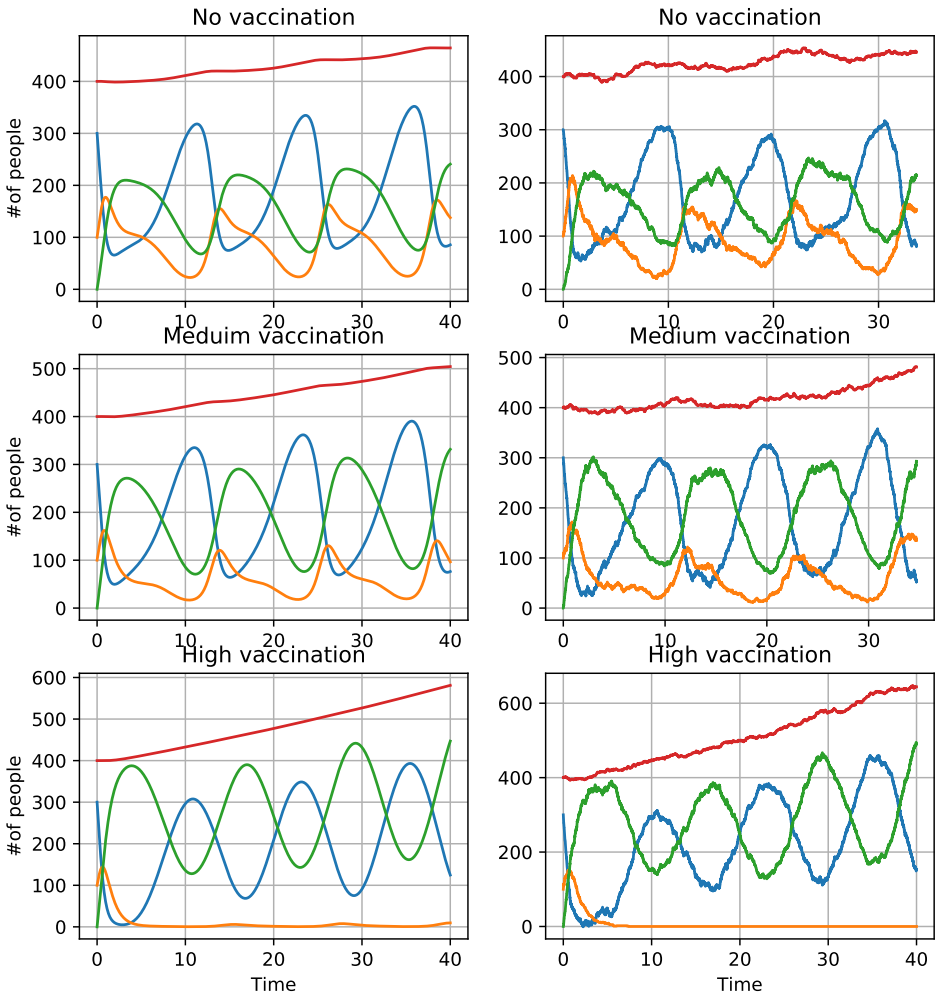

Figure 6: Devolpment of compartments with different vaccination plans. The smooth graphs to the left are from RK4 and to the right are the mean of a 100 MC- runs with the same parameters as in 5 and a vaccination parameter f. The top have no vacination, the middle has vaccinaition going as $f = 0.1 + 0.1sin(t\omega)$, and the bottom has a vaccanating functon $f = 0.3 + sin(t\omega)0.2$ where ω is the same as in the seasonal fluctuations of the disease

Figure 6 shows how increasing levels of vaccination affects the compartments. The upper plots show the development with no vaccination. The second row with a small sine function in phase with the infection pressure, and the third row with a stronger sine function and a constant.

As seen in the middle row a low vaccination vaccination lowers the amount of infected people each year slightly, but the infection still remains in the population.

In order for the vaccination to take real effect, the susceptible group has to be at almost zero, at the same time as the infected group goes to zero, as is the case in the lower row.

Being that in the model all people interact with all people, vaccination is not effective before everyone is vaccinated as herd immunity relies on separating the susceptible from each other which is not possible in this model as it is.

Conclusions

The monte carlo implementation agrees on a large part with the classical solving of the ODE's set up by the SIRS model. The main differences lie in the handling of population sizes. The discrete nature of monte carlo captures information about the population size, and being that rk4 operates with (semi-)real numbers and transition fractions it does not. The change in the compartments are decided by fractions of the population and would yield the same graphs for all population sizes while the relative magnitude of the discrete transition in monte carlo makes it sensitive to the population size. Where the SIR model might predict if a disease manages to establish itself in a population, and if it does how large fractions of the populations are left infected, it does not take into account the random fluctuations that would occur in such populations nor the consequences of such fluctuations. The monte carlo stochastic model does, and can be use to model differences of development patterns in populations of different sizes.

Future work

Use the Montecarlo method can be used to extract expectation values for the survival time of diseases in small populations.

Use bigger populations in MC modeling and implement som sort of encounter-simulation to see if one can recreate herd immunity as this model assumes that everyone is in contact with everyone.

References

[1] F Baryarama, Joseph Mugisha, and Livingstone Luboobi. Mathematical model for hiv/aids with complacency in a population with declining prevalence. *Computational and mathematical methods in medicine*, 7:27–35, 03 2006.

[2] Folkehelseinstituttet. Flere tar influensavaksine, men fortsatt langt til mål. https://www.fhi.no/historisk-arkiv/nyheter/2017/flere-tar-influensavaksine-men-fortsatt-ikke-mange-nok/, 2017.

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[4] A.G. McKendrick W.O Kermack. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society A*, 115:700–721, Aug 1927.