Universitiet van Amsterdam

Introduction to Computational Science

Assignment 2: Stochastic and Spatial Models

Group 15

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1 Stochastic Model

1.1 Introduction

In Assignment 1 which covered the SIR-model in detail, the underlying assumption was that the dynamics of the model were deterministic. This is, however, a simplification, as systems involving human behaviour are seldom deterministic. Implementing stochasticity into an epidemic model intended for studying transmissions of pathogens among humans can bring it closer to reality. Keeling and Rohani (2011) explain that there are two ways in which this can be done: inclusion of process noise and the event driven approach. The process noise approach is a more intuitive way to incorporate noise by introducing it directly into the deterministic equations. As such, the dynamics at each point in time are subject to some random variability and this variability is propagated forward in time by the underlying equations. Here the focus is on the latter approach, and in particular on the Gillespie's Direct Algorithm application of it.
We prepare for the experiments by implementing stochastic SIR models with demography and comparing them with the equivalent deterministic SIR model (1.2). In continuation, we implement different parameter sets to find out what influence that has on the simulation outcomes, especially with regards to simulation variability and covariance between X and Y (1.3). Next, we investigate stochastic resonance and transients (1.4). Finally, we conduct experiments to show the effect of R_0 and N on the probability of extinction (1.5).

1.2 General Methods: Implementing a Stochastic SIR Model

We have chosen for the Gillespie's Direct Algorithm as described in box 6.3 of Keeling and Rohani (2011, p. 201). In this algorithm, we first determine the types of events that could take place in the system. In our case this includes: {births, disease transmission, disease recovery, death of susceptible, death of infectious, death of recovered}.
Secondly, the rates of these events are determined. These are respectively: $\{\mu \cdot N, \beta \cdot \frac{X \cdot Y}{N}, \gamma \cdot Y, \mu \cdot Z, \mu \cdot Y, \mu \cdot Z\}$ with $X =$ the number of susceptible, $Y =$ the number of infectious and $Z =$ the number of recovered individuals.
Thirdly, we determine the sum of these event-rates and call it R_{total}. This is done by

$$R_{\text{total}} = \sum_{m=1}^{n} R_m$$

where n is the number of events (which is six in our case).
Fourthly, we determine the time it takes until the next event takes place. We do this by the following equation

$$\delta t = \frac{-1}{R_{\text{total}}} \cdot \log(\text{Random})$$

where Random is a uniformly random number between 0 and 1.
Next, we generate another random number between 0 and 1 and multiply it to R_{total}. We call this number P.
In continuation, we determine that an event e takes place if

$$\sum_{m=1}^{e-1} R_m < P \leq \sum_{m=1}^{e} R_m.$$

After this step, the time variable is updated, which means that δt is added up to the previous time. Also, event e occurs. Finally, we iterate this sequence from step 2 onward, until a termination value has been met.
For reference we also implemented the deterministic model as incorporated in Assignment 1 using the standard set of SIR differential equations with birth/death rates.
In Figure 1, a stochastic implementation of the SIR model using Gillispie's Direct Algorithm is plotted together with the deterministic simulation. The outcomes of the stochastic simulation are very similar to the deterministic simulation, implying that Gillispie's Direct Algorithm is correctly implemented.

1.3 Experiment 1: Variability and Covariance

The goal of this section is to investigate how varying the model parameters changes the behaviour of the stochastic dynamics established in prior section. In particular, we want to evaluate how they relate to variance between runs and how they impact negative covariance between S and I.
We do this by i) evaluating the means of stochastic outcomes - mean variance of the number of infectious individuals, in particular, against parameter value sets of rates of infection, recovery and birth/death (β, γ, μ), (see Figure 2), and mean covariance in the number of susceptible and infectious individuals against the same parameters (see Figure 3), ii) comparing the means of stochastic simulations with the equivalent outputs from the deterministic models. Here we

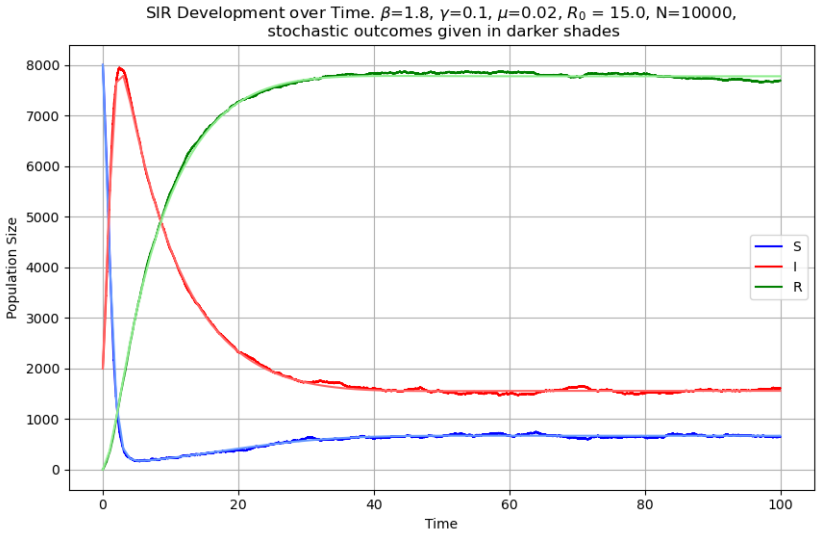

Figure 1: Joint representation of a deterministic SIR model outcome and the outcome of a stochastic SIR model obtained using Gillespie's Direct Algorithm, and the following parametrisation: population size N = 10000, rate of infection $\beta = 1.8$, recovery rate $\gamma = 0.1$, and a birth / death rate being jointly $\mu = 0.02$ - resulting in the basic reproduction number $R_0 = 15$.

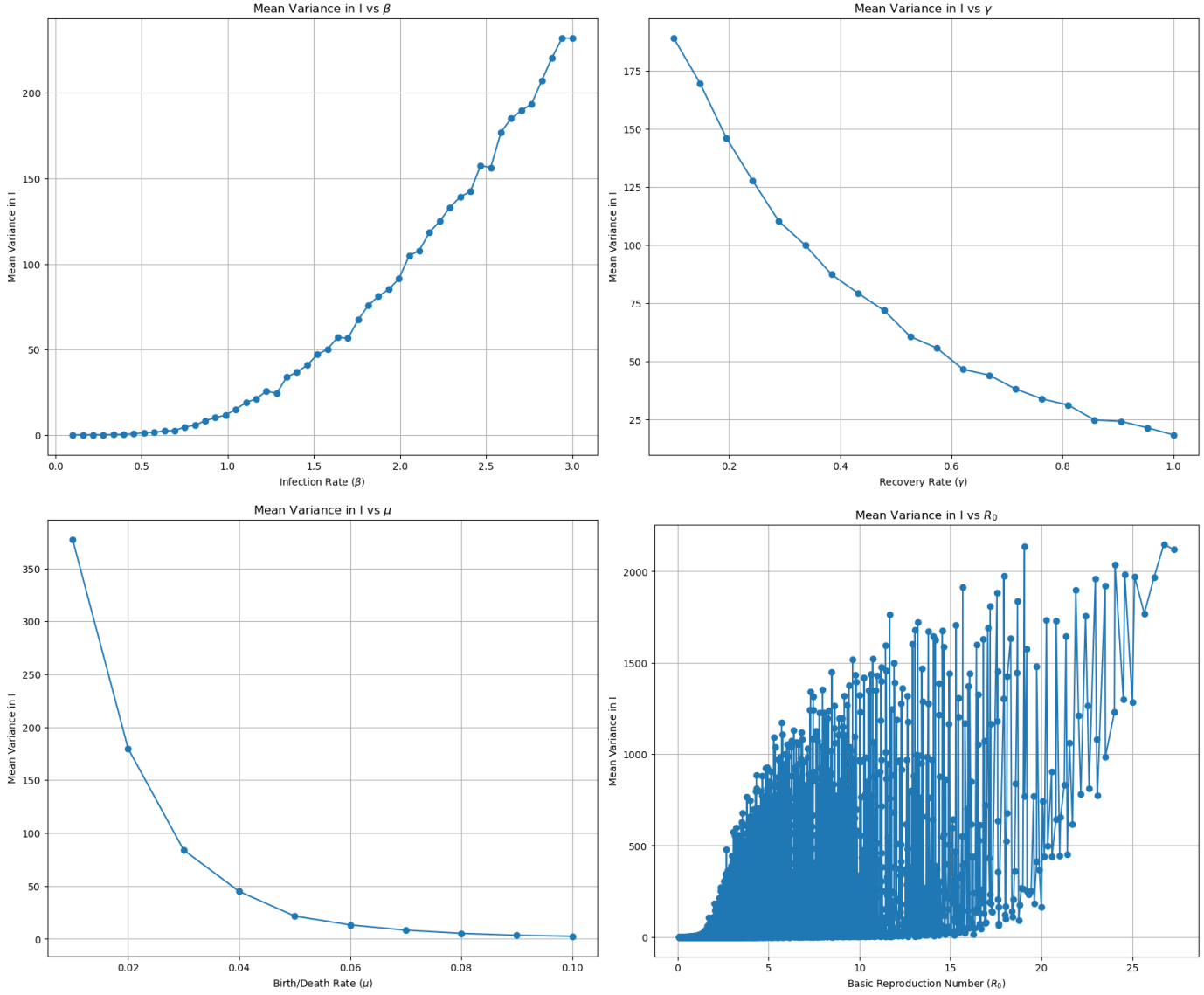

Figure 2: Mean variance in the number of infectious individuals obtained in simulations of the stochastic SIR spread over the population of the size $N = 1000$, as a function of i) infectious rate β in $[0, 1, 3]$, recovery rate γ in $[0.1, 1]$ and birth/death rate μ in $[0.01, 01]$, and the resulting basic reproductive number R_0 in $[0, 27.27]$. Ten simulations were ran for each parameter combination (β, γ, μ).

evaluate the sum of mean square errors (MSEs) between mean stochastic outcome and the deterministic outcome for each parameter set (β, γ, μ) (see Figure 4) as a measure of closeness between the two sets of results.
Observing the Figure 2, it can be noted that mean variance in the number of infectious individuals increases non-linearly with increases in infection rate β, while it decreases also non-linearly with increases in recovery rate γ and birth/death rate μ, where the latter has a more stronger non-linear negative impact on the variance. The increase in birth rate would imply an increase in the susceptible population, increasing the covariance between S and I by expanding the pool of potential new infectious. Under the assumption that the equal death rate affects all compartments, the covariance between the S and I by uniformly removing individuals from both compartments. The increase in the basic reproduction number R_0 has a bit noisier but still positive relationship with the variance in the number of infectious individuals - with values of R_0 higher than 15 always producing non-zero mean variance in the number of infectious individuals.
The covariance between the number of susceptible individuals (S) and the number of infectious individuals (I) provides insight into how these model compartments tend to vary together over time. For SIR model with birth/death rate the covariance between the number of susceptible (S) and number of infectious individuals is negative simply as a consequence of the fact the increase in the latter population can only be achieved by a decrease in the former. Magnitude of covariance in absolute terms cannot be straightforwardly interpreted as the measure of the strength of the relationship between the two variables (as only a standardization would secure this interpretation), but it is a clear pointer in that direction.
As it can be seen in Figure 3, increasing the rate of infection β should increase the rate at which susceptible individuals become infected. The higher rate of infections would lead to initially increasing covariance between S and I - because of the significant changes in both compartments. With the decreasing population of susceptibles later on, the covariance may also decrease. The simulations actually show that the mean covariance steadily increases (in absolute terms) with the value of β.
Increasing the recovery rate γ should lead to quicker recovery times, and hence the number of infectious individuals should decrease faster relative to changes in the number of susceptibles (i.e. there's less time for infectious to infect the susceptibles) - decreasing the covariance between the two. This is also observed in Figure 3. Increase in birth /death rate μ again points to relatively swift decrease in covariance between the two compartments (in absolute terms).
The actual impact of covariance on the simulation outcomes will be a result of complex interactions between the parameters and the stochastic nature of the model. The actual realisations of the model could have fluctuations due to random variations that are behaving differently than the mean values shown in Figures 2 and 3. The covariance can inform us about the disease dynamics, such as whether an increase in infected individuals is likely to correspond with a decrease in the number of susceptible (negative covariance) or whether the relationship is not straightforward (near-zero or positive covariance).
To evaluate the impact of the SIR parametrisation (β, γ, μ) on the closeness between mean stochastic realisation and a deterministic realisation with the same parameters, we can refer to the results presented in the Figure 4. There we see that increases in infection rate β lead to steady but non-linear increase in divergence between the two outcomes, whereas the increase in the recovery rate γ has the opposite effect. The impact of birth/death rate is non-linear in that that after the initial spike in differentiation (for , essentially, higher birth rates) in drives the differentiation down with any further increase in value ($\mu > 0.03$). The increase in basic reproductive number R_0 has a strong impact on the differentiation as soon as it becomes larger than 1, but the impact of this variable on the differentiation gradually stabilises for values higher than 15.
Finally, we add a number of specific evaluations of the stochastic and deterministic outcomes to showcase the options. Figure 5 shows the example with the highest values of MSE sums obtained across the parameter sets ($\$beta, \$gamma, \$mu$).

1.4 Experiment 2: Stochastic Resonance and Transients

The goal of this section is to show how the stochastic model can induce stochastic resonance around the equilibrium and how that resonance relates to the model parameters (e.g., N,), etc). We are looking for i) examples of increased transience away from deterministic equilibrium, as well as the parameter values (β, γ, μ) which lead to largest transients.
In order to be able to determine the effect of different parameter sets on stochastic resonance and transients at equilibrium, we first simulated the deterministic model for 1000 time steps as an indicator of the equilibrium point of the model with the given parameter values. Then we simulated the stochastic, as well as the deterministic models for additional 100 time steps. Consequently, we plotted the simulation outcomes where the difference between the minimum and the maximum value of the infectious population was greater than 100 - as an indicator of large transients.
Figures 6 shows a selection of plots with large transients. In the subplot 1.3.8, the values of the stochastic model stay around the equilibrium values. In the other presented cases we can see stochastic resonance too. These specific plots show that greater stochastic resonance is achieved with lower values of R_0. It is worth noting that all four figures have quite high values of birth/death rates μ. So it could be hypothesized that the probability for stochastic resonance is linked to the birth (and death) rate.

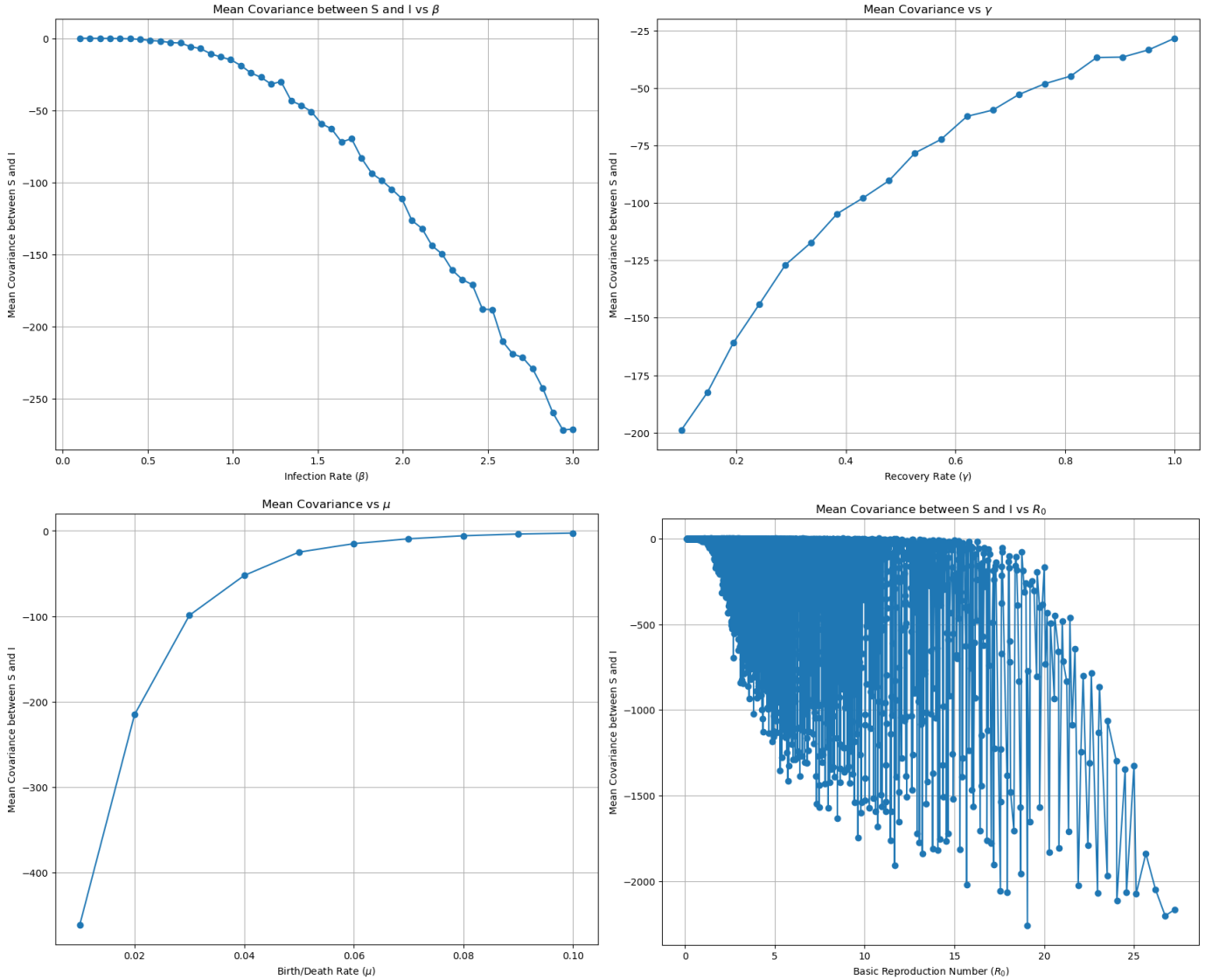

Figure 3: Mean covariance between the number of susceptible and infectious individuals obtained in simulations of the stochastic SIR spread over the population of the size $N = 1000$, as a function of i) infectious rate β in $[0, 1, 3]$, ii) recovery rate γ in $[0.1, 1]$ and iii) birth/death rate μ in $[0.01, 01]$, and the resulting basic reproductive number R_0 in $[0, 27.27]$. Ten simulations were ran for each parameter combination (β, γ, μ).

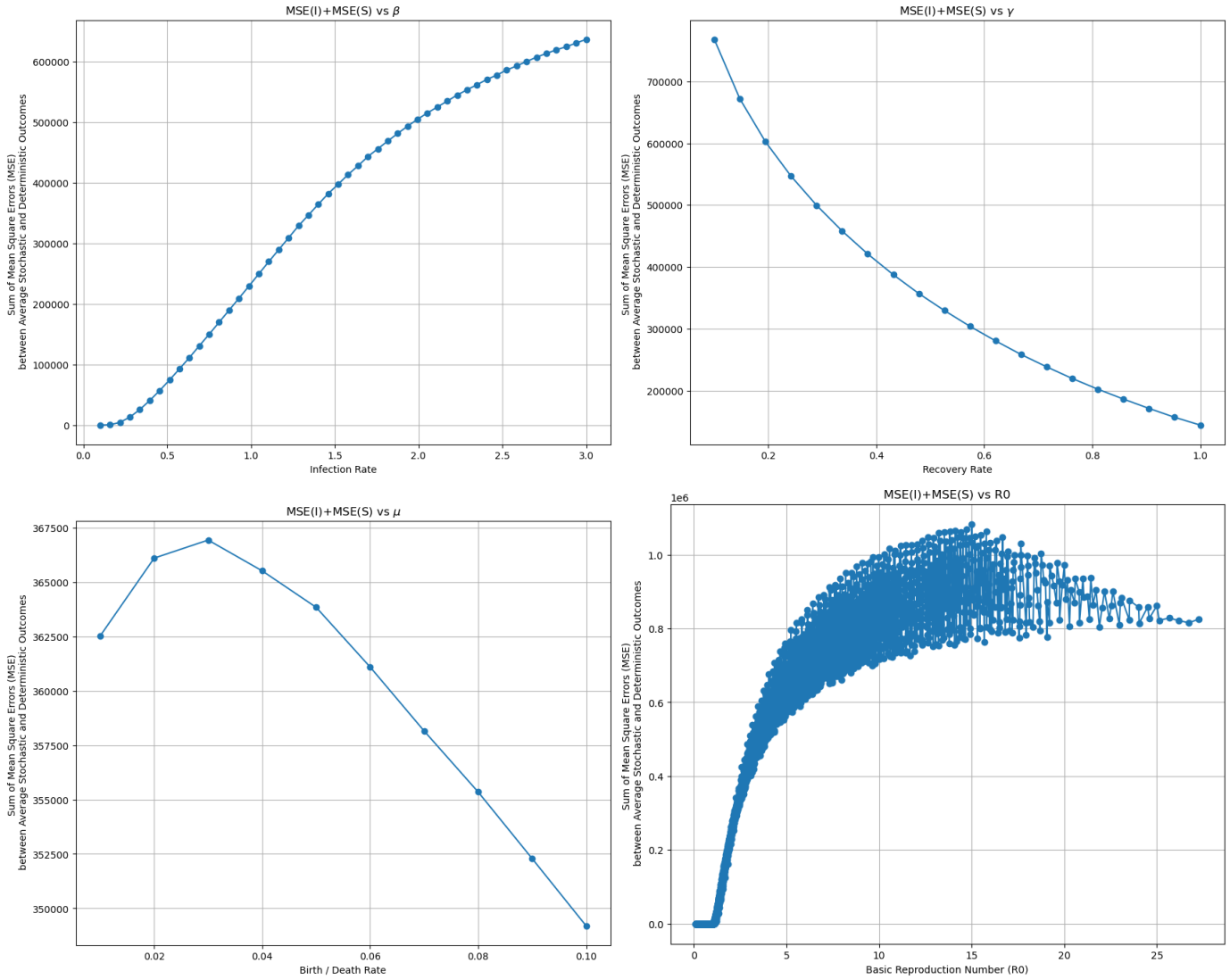

Figure 4: The sum of the mean square error difference between the mean stochastic outcome and the deterministic outcome using the same parameter sets (β, γ, μ) plotted against the values of individual parameters. The sum of zero implies identical outcomes for number of susceptible and number of infectious individuals across the observation time both from the mean stochastic and the deterministic modelling. Consequently, high values of the sum imply higher differentiation in the results. The population size is $N = 1000$, as a function of i) infectious rate β in $[0, 1, 3]$, recovery rate γ in $[0.1, 1]$ and birth/death rate μ in $[0.01, 01]$, and the resulting basic reproductive number R_0 in $[0, 27.27]$. Ten simulations were ran for each parameter combination (β, γ, μ).

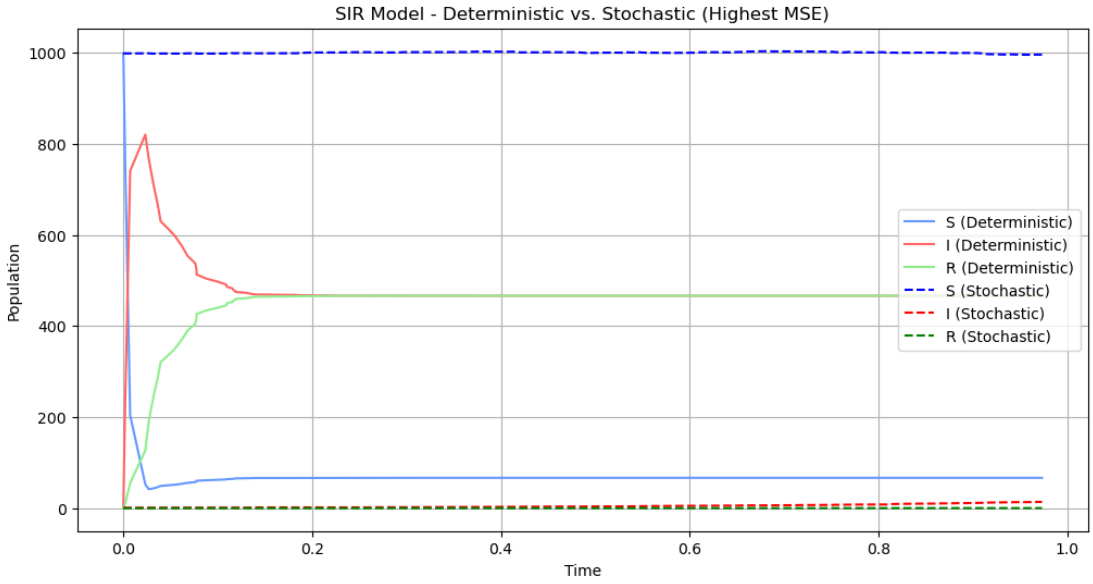

Figure 5: Mean stochastic and deterministic outcomes of the SIR model with basic adjustments for demography for the values of parameters $(\beta, \gamma, \mu) = (3.0,\ 0.1,\ 0.1)$ which result in the highest mean square error diffence between the two outcomes across all considered parameter values: infectious rate β in $[0, 1, 3]$, recovery rate γ in $[0.1, 1]$ and birth/death rate μ in $[0.01, 01]$, with the resulting basic reproductive number R_0 in $[0, 27.27]$

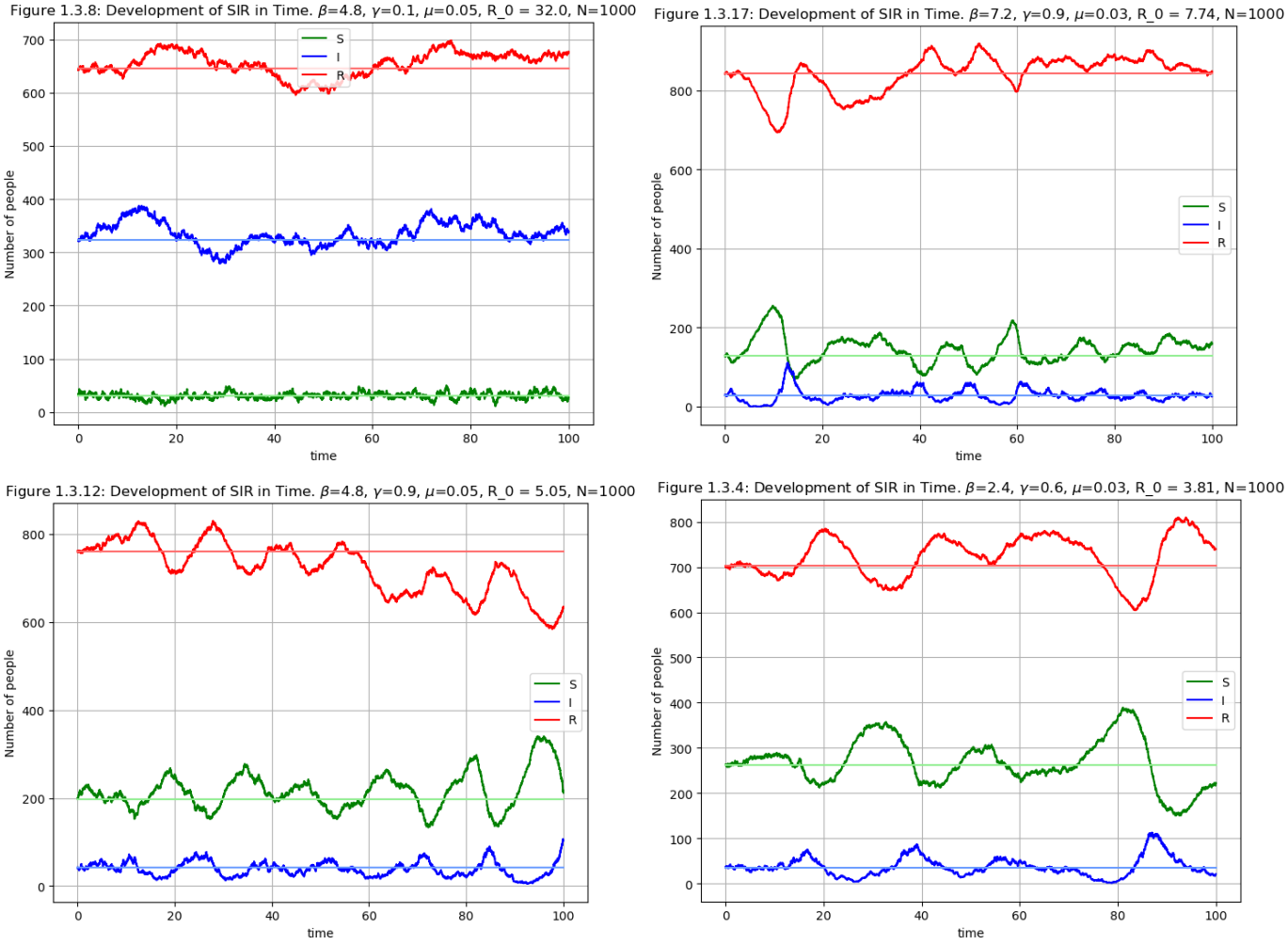

Figure 6: Add text

1.5 Experiment 3: Extinction Process and Critical Community Size

In order to find the relationship between R_0 and N with probability of extinction, we did 10 runs of 1000 days with different values of R_0 and N. We then determined in how many of these runs extinction was met and at what point in time.

	$R_0 = 1.1$	$R_0 = 1.2$	$R_0 = 1.3$	$R_0 = 1.4$	$R_0 = 1.5$	$R_0 = 1.6$	$R_0 = 1.7$
$N = 1000$	9	9	7	2	0	0	0
$N = 2000$	9	4	0	0	0	0	0
$N = 3000$	4	2	0	0	0	0	0
$N = 4000$	5	1	0	0	0	0	0
$N = 5000$	4	0	0	0	0	0	0
$N = 7000$	1	0	0	0	0	0	0
$N = 7000$	1	0	0	0	0	0	0
$N = 8000$	0	0	0	0	0	0	0

Table 1: Extinction rate R_0 against N. The values in the table represent the number of runs where extinction was met in 1000 days.

As you can see in table 1. When R_0 increases, the probability of extinctions occuring decrease. Additionally, when the population size N increases the probability of an extinction occuring also decreases. This is in agreement with Keeling and Rohani (2011). On the basis of this table, we are not able to determine the critical community size with certainty, because we 'only' did 10 runs. It is plausible that there would be at least 1 extinction if we did more runs. But it is very plausible that the critical community size for this specific system is somewhere around $N = 8000$.

1.6 Conclusion

In conclusion, we can implement Gillespie's Direct Algorithm to make a stochastic simulation model based on epidemiology. With this stochastic simulation model we can experiment in an effort to expose the hallmarks of stochastic simulations. In the first place, stochastic models show varience between runs which is what we saw in subsection 1.3. The standard deviation of the stochastic model was plotted in the figures. If there was no varience between runs, then the standard deviation would have been 0.
In the second place stochastic simulations show negative co-variences. We have seen that stochastic simulations could show negative co-variences. But it might also show positive co-variences. This is not related to the degree where the stochastic models are in agreement with the deterministic ones, as we have seen in section 1.3.
In the third and fourth place stochastic models show transients and stochastic resonance as discussed in section 1.4.
Finally, stochastic simulations show extinctions. These extinction depend on the value of R_0 and N. For both of these variables, the higher the value, the lower the probability of extinctions.

2 Spacial Models

2.1 Introduction

In the following section of the assignment, the focus of the exploration is on evaluating SIR spreads on various types of network models and on developing and evaluating vaccination strategies for those models. The SIR spreads are assessed across three standard theoretical network models (Erdos-Reyni, Barabasi-Albert and Watts-Strogatz) as well as on static (non-temporal) form of a real-life contact network provided by sociopatterns (Stehlé et. al 2011). The main theoretical reference for the assignment is the Albert-Lázló Barabási and Márton Pósfai book - Network Science (2016) and in particular the Charter 10 of Keeling and Rohani (2011).
We start the experiments by implementing SIR disease spread on a static version of the real-life sociopatterns contact network (2.2). In continuation, we generate multiple model networks with similar characteristics and network parameters and discuss how these statistics differ between network types and for different parameter settings (2.3). Next, we simulate and compare SIR spreads on the generated model networks (2.4). Finally, we conduct vaccination experiments on the sociopatterns the static version of the real-life contact network (2.5).

2.2 SIR Spreads on Socio-Patterns Network

Taking the static version of the real-life Socio-Patterns network as given we (Stehlé et. al 2011), we evaluate the realisations

2.3 Methods

2.4 Results

2.5 Discussion

2.6 Conclusion

References

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- Keeling, M. J., & Rohani, P. (2011). Modeling Infectious Diseases in Humans and Animals. United States: Princeton University Press.
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