Stochastic, Meta-population and Lattice-based models for modeling infectious diseases

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Abstract

Stochastic models offer a realistic way of implementing uncertainty into modelling and simulation. Such models differ from their deterministic counterparts on five different levels, namely variability, negative (co-)variance, increased transients, stochastic resonance and extinction [1]. In this report these features were found through the implementation of the SIR model, an important model used to understand the dynamics of infectious diseases. Although stochastic models differ from deterministic models on these different features, one can still find the underlying deterministic dynamics through the application of stochastic SIR models. It was also found in meta-population and lattice-based models, that interaction between populations can have a big effect on the spreading of infectious diseases. This was shown by the important parameter ρ, the relative strength of transmission within populations.

1 Introduction

In real-world situations, often events will not follow a predetermined path. If we would want to model such a real-world situation, we can take into account a certain amount of uncertainty. For the modelling of infectious diseases, it can be very useful to do this by using a stochastic model in specific situations, for instance when there is a small population, or when we want to simulate a disease that just started to spread [1]. Compared to deterministic models, stochastic models can be distinguished by five features: variability, negative co-variances, increased transients, stochastic resonance and extinctions. These features can effect the dynamics of modeling epidemiology drastically. Also, when modeling infectious diseases, spatial features can be a large influence on the dynamics. If an infectious host travels through different populations this can greatly influence the spreading of the disease. With the increase of globalization since the 19^{th} century [2], it is important to take these spatial features into account as well. In this report, we will first look into the distinguishing features by doing experiments with the SIR model, an epidemiological model used for understanding the spreading of infectious diseases. We evaluate these features in a SIR discrete event model using Gillespie's direct algorithm. After these features have been explored, we will look further into spatial models (a Meta-population and a Lattice-based model) to study the spread of infectious diseases.

2 Method

To take uncertainty into account in the SIR model, we could use noise-based modifications to the differential equations that are normally used in deterministic models. But when dealing with low numbers, or in our case a lower population, it is required to use a discrete event model. In this report we use Gillespie's First Reaction Method, which is equivalent to Gillespie's Direct Algorithm, but can be seen as a more intuitive approach. Before going into the experiment, we will look into this Gillespie's Direct Algorithm for implementing a discrete event model in combination with the SIR model.

2.1 Gillespie's Direct Algorithm

Instead of using noise, in an event-driven approach the events will be randomized to a certain extent. In Gillespie's direct method, we determine the rates of all possible events and then choose the rate with the lowest δt according to the equation found in Algorithm 1. For the SIR model, this results in the formulas shown in the Equations 1 below. Without demography, β shows the rate of transmission and γ the rate of recovery. For demography we will use μ, which represents the rate at which individuals suffer from natural mortality. This μ will also be used as a birth rate, to keep the population constant. Depending on the population size, δ shows the rate at which an infected individual joins the population. ϵ corresponds to a commuter travelling through the population, which will add force to the infection in a certain population.

$$\begin{array}{rcr} \text{Transmission } \beta\dfrac{XY}{N} & & X-1, Y+1 \\ \text{Recovery } \gamma Y & & Y-1, Z+1 \\ \text{Birth } \mu N & & X+1 \\ \text{Death } \mu X, \mu Y, \mu Z & \rightarrow & X-1, Y-1, Z-1 \\ \text{Import } \delta N & & Y+1 \\ \text{External source } \epsilon X & & X-1, Y+1 \end{array} \tag{1}$$

In this experiment the model will be expanded with these events step by step which allow us to explore the features of stochastic models. We start with a population without demography (births and deaths). Then we will add demography and finally we will add simple imports to the model by using the Import and External Source events. These rates and events are implemented into Gillespie's Direct Method algorithm.

Data: Events E_1 ... E_n
Result: Spreading of Infectious disease for SIR over time
initialization of parameters (β γ μ δ ϵ);
while *time* < *total_time* **do**
  Calculate rates of every event E_n;
  Find the event rate with the lowest δt by computing for every event n: $\frac{-log(Rand_n)}{EventRate_n}$;
  Update time t with δt;
  Perform event E_n on X, Y and Z;
  Repeat this process;
end

Algorithm 1: Gillespie's First Reaction Method

The algorithm first computes the rates of every event as described in Equations 1. Then the rate with the lowest δt is computed, added as the new timestamp and then that event will be executed. This process is repeated until the simulation reaches the desired time. A further explanation of this algorithm can be found in Algorithm 1.

2.2 Spatial models

Apart from stochastic models, another feature that is important in the spreading of infectious diseases is a spatial feature. In this report, we look at two possible models for implementing this feature, namely a meta-population model and a lattice-based model.

2.3 Meta-population model

If we use the SIR equations to implement a meta-population model, we map the rates and events as seen in Equations 2. Here ρ is the relative strength of transmission to sub-population i from j. We calculate the rate for every population i.

$$\begin{aligned} \text{Transmission}_i = \beta_i X_i \sum \rho_{ij} \frac{X_j}{N_i} \quad &\rightarrow \quad X_i - 1, Y_i + 1 \\ \text{Recovery}_i = \gamma_i Y_i \quad &\rightarrow \quad Y_i - 1, Z_i + 1 \end{aligned} \tag{2}$$

Here we apply the same algorithm as with the standard stochastic models. Initially we will use two populations without demography and later expand this to evaluate the effect of sub-population transmission factor ρ between 'neighbor' populations.

2.4 Lattice-based model

For the lattice-based model we implement a square grid (41x41) with initial disease populations to examine the possible spreading in spatial dynamics. For every population we use the differential equations from the deterministic SIR model. We only apply changes to a population if they are in contact with a population where there are infectious individuals ($Y > 0$), and every change is subject to a random number *Rand*. Where *Rand* is a random number between 0 and 1 and every group, Susceptibles (X), Infectious (Y), Recovered(Z) only changes when this number falls between certain intervals as shown in the used equations below.

$$\frac{dX_i}{dt} = \mu - \beta X_i \frac{(1 - \sum \rho_{ij})Y_i + \sum \rho_{ij} Y_j}{(1 - \sum \rho_{ij})N_i + \sum \rho_{ij} N_j} - \mu X \rightarrow textifRand < 0.33 \tag{3}$$

$$\frac{dY_i}{dt} = \beta X_i \frac{(1 - \sum \rho_{ij})Y_i + \sum \rho_{ij} Y_j}{(1 - \sum \rho_{ij})N_i + \sum \rho_{ij} N_j} - \mu Y_i - \gamma Y_i \rightarrow textifRand >= 0.33 \text{and} Rand <= 0.66 \tag{4}$$

$$\frac{dZ_i}{dt} = \gamma Y_i - \mu Z_i \rightarrow textifRand > 0.66 \tag{5}$$

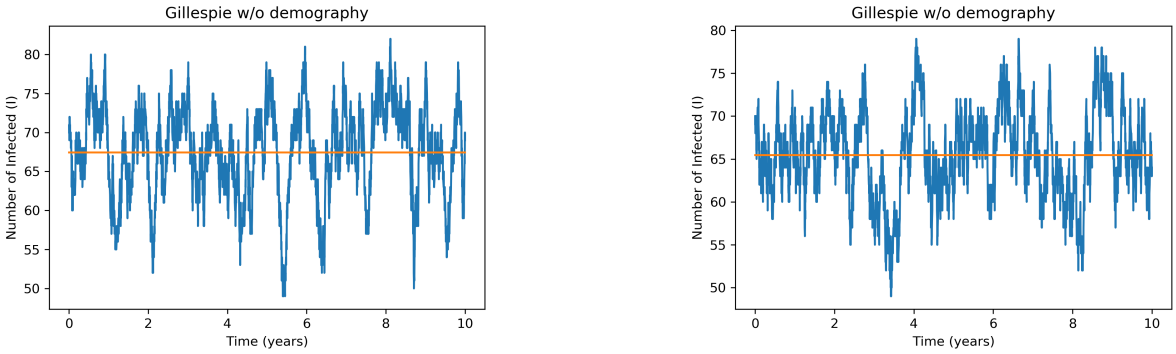
3 Results

Firstly, we will go through the SIR model step by step which will allow us to go over the distinguishing features. Afterwards we will look into experiments done with spatial models.

3.1 Stochastic Models

In this section, we will look at the five distinguishing features of stochastic models as compared to deterministic models, namely variability, negative co-variance, increased transients, stochastic resonance and extinctions. We will add events to the model as we go over these features.

- **Variability**
  Compared to deterministic models, stochastic models evidently have no precise outcome. The statistical features will be comparable to the deterministic model such as the mean and variance. But every simulation will have a "different" outcome. In Figure 1 we can see that different simulations with the same parameters can lead to different outcomes based on the same parameters, although the outcome is comparable to the deterministic model in terms of mean and variance.



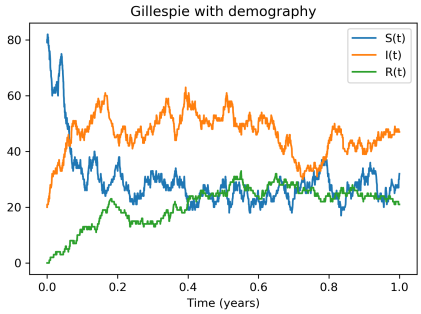
(a) Simulation 1

(b) Simulation 2

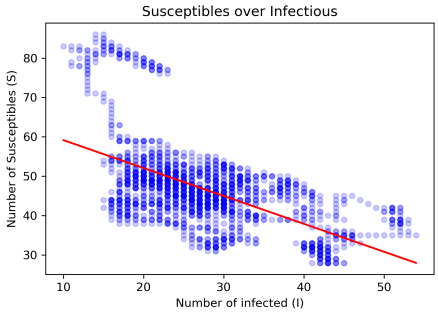
Figure 1: Stochastic SIR model without demography, ran with $\beta = 0.03$ $\gamma = 0.01$ over 10 years. The model was initialized with a total population of 100 and 70 infectious hosts. The orange line demonstrates the mean number of infectious over 10 years.

- **Negative co-variance**
  The stochastic nature of these models causes variation between the infectious and susceptible groups within the model. We see this when we add demography to the SIR model applied to the Gillespie algorithm. In Figure 2a we simulate a population over the course of ten years using the Gillespie algorithm. In Figure 2b we plot the number of infectious individuals over the number of susceptibles, which shows a negative co-variance.

- **Increased Transients**
  The rates of events of the applied SIR model in this report are still based on deterministic equations



(a) Gillespie with demography



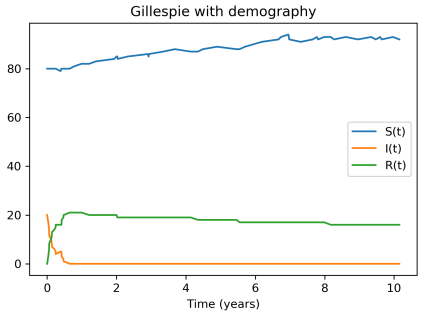
(b) Co-variance graph

Figure 2: Stochastic SIR model with demography (a), ran with $\beta = 0.1$, $\gamma = 0.01$ and $\mu = 0.02$ over 1 year. The co-variance graph (b) shows the Infectious group over the Susceptibles group. The found co variance is -50.45

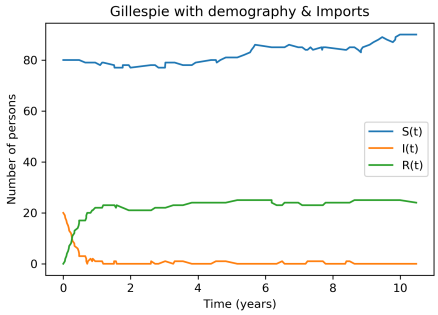
which can be derived from the dynamics of the model. Even though the stochastic model will diverge from the deterministic result, the underlying dynamics will converge to this equilibrium point. In Figure 2a we see that all three groups move away from and back to the deterministic attractor.

- **Stochastic Resonance** Eventually the dynamics will resonate around the natural frequency as shown in the oscillations in Figure 2a, same as seen in deterministic models due to the underlying deterministic dynamics of the model.

- **Extinctions** We can see in Figure 3a, that without any imports, the disease will rapidly extinct over a longer period. A disease that would not go extinct in such a situation, requires an external source such as an import. This is shown in Figure 3b. The infectious disease does not go extinct, since an import will reintroduce a disease back into the population. We will see similar behavior when we look at the meta-population without demography in the next section.



(a) Stochastic model with demography, ran with $\beta = 0.1$ $\gamma = 0.01$ over 10 years.



(b) Stochastic model with demography, ran with $\beta = 0.1$ $\gamma = 0.01$ over 10 years.

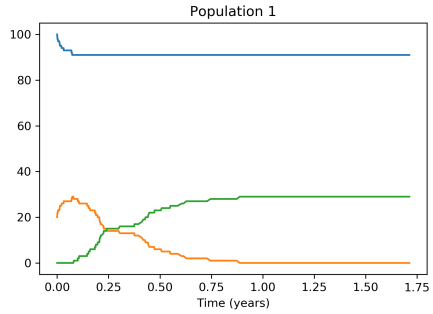
Figure 3: Stochastic SIR simulations where the disease goes extinct (a) and where an external source allows for the persistence of the disease (b)

3.2 Spatial Models

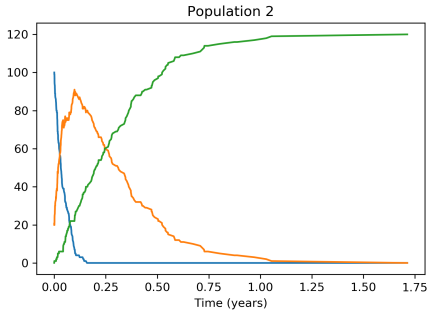
In the real-world, a disease will often not remain in just one population. That is why it is important to introduce a spatial feature to the SIR model to evaluate the spreading of infectious diseases. We first experiment with a meta-population model with two populations. Then we shortly look at multiple population and we finally look at lattice-based models to evaluate the spreading of an infectious disease.

3.3 Meta-population model

In Figure 4 we see the effect of stronger coupling from population 1 to population 2. The bigger the effect of other populations, the stronger the delay in the model will be. In this model, a very strong coupling factor for population 1 to population 2 was used, namely $\rho_{21} = 0.9$. The coupling from population 2 to 1 was very small ($\rho_{12} = 0.1$) and as we can see in the graph this has a very small effect on the dynamics of the disease. The disease goes extinct after about one year and reached only a small amount of the population whereas the disease reaches a lot of people in the other population and takes somewhat longer to go extinct.



(a) Stochastic model with demography, ran with $\beta = 0.1$, $\gamma = 0.01$ and $\rho_{12} = 0.1$ over 2 years. We see here that the disease already extincts within approximately 1 year.



(b) Stochastic model with demography, ran with $\beta = 0.1$, $\gamma = 0.01$ and $\rho_{21} = 0.9$ over 2 years. Here the disease suffers frome extinction later than in population 1

Figure 4: A meta-population model where the coupling effects between two populations are shown.

When we simulate a meta-population with multiple populations, we see this effect even more clear. In Figure 5 we see that for eight populations with increasing relative strength of transmission ρ, the delay in the extinction of disease gets bigger. Here all populations are coupled to their neighbor(s). Note that population 1 and 8 are only coupled with their neighbors, respectively population 2 and population 7, which makes the effect somewhat smaller.

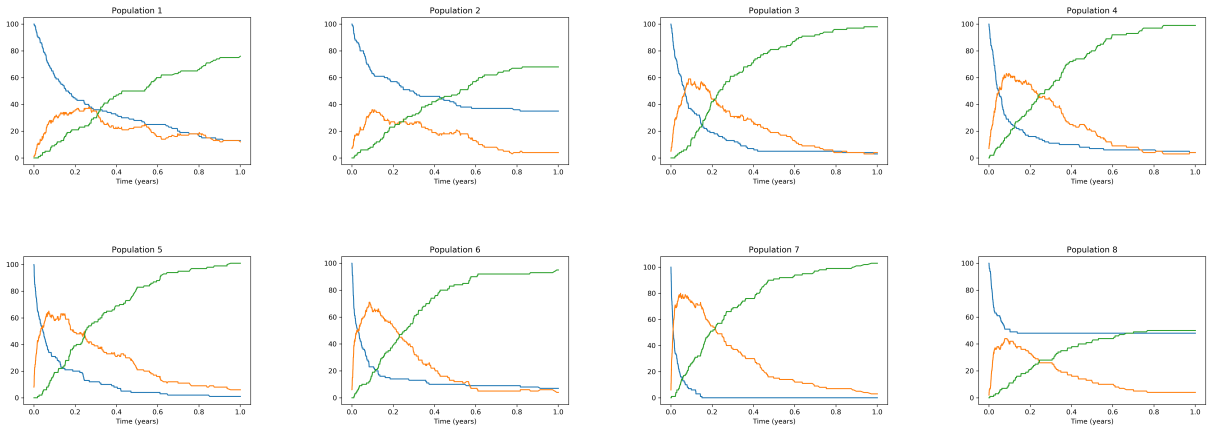
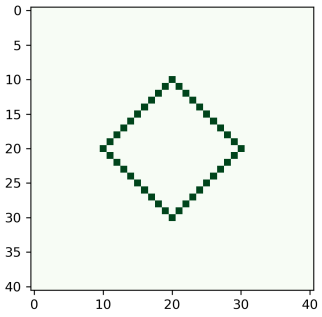


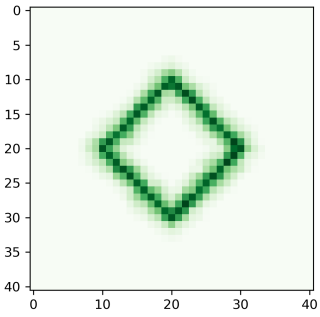
Figure 5: The above row shows population 1 through 4, whereas the bottom row shows population 5 through 8. Every population has the same parameters ($\beta = 0.1$, $\gamma = 0.01$) except for the relative strength of transmission ρ which increases per population. In population 8 we see a smaller delay than in population 7, but only because populations 1 and 8 are only coupled to one neighbor.

3.4 Lattice-based model

For the lattice-based model, we use a 41x41 grid to model a simple stochastic version of the SIR model. We can see the spread of the disease happen in a wave-like matter and the disease will eventually spread across the whole grid. Even though we added simple stochastic features to the model, we still see a linear basis throughout these dynamics. This is most likely caused by the underlying deterministic dynamics. In Figure 6 we can see that this happens very gradually for a low ρ. When we increase this parameter, we can see that the disease spreads more drastically. In Figure 7 we increase this strength within populations ρ and the disease spreads more within 400 steps. Here we can also observe the underlying deterministic and linear dynamics. The spreading is greatly influenced by the chosen ρ and changes in the other parameters will have more impact on the population themselves than the interaction between them.

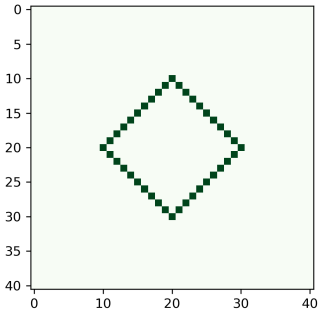


(a) The initial SIR model.

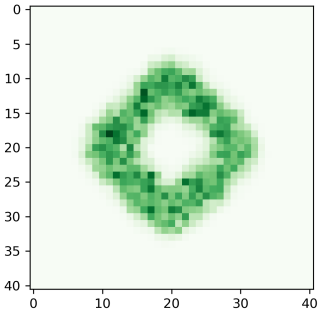


(b) Simulation after 100 steps

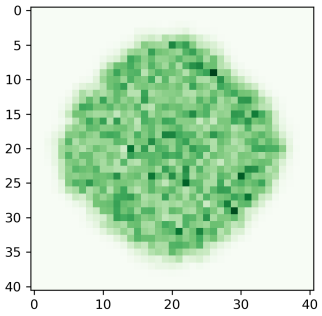
Figure 6: Simulation of a simple stochastic lattice model on a lattice of 41x41. Parameters that were used are $\beta = 0.1$, $\gamma = 0.01$, $\mu = 0.0005$ and $\rho = 0.1$ for every population.



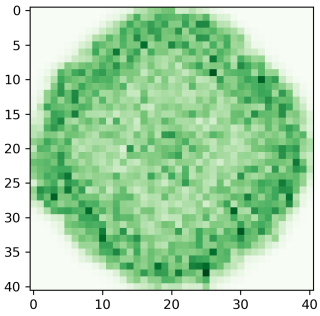
(a) The initial SIR model.



(b) Simulation after 100 steps



(c) Simulation after 200 steps



(d) Simulation after 300 steps

Figure 7: Simulation of a simple stochastic lattice model on a lattice of 41x41. Parameters that were used are $\beta = 0.1$, $\gamma = 0.01$, $\mu = 0.0005$ and $\rho = 0.5$ for every population.

4 Conclusion

In this report, stochastic models were evaluated to explore the dynamics of infectious diseases. Five features of stochastic models showed how they differ from their deterministic counterparts. We found that variability, negative variance, increased transients, stochastic resonance and extinctions were all features of stochastic models when implemented through the SIR model. These show differences from their deterministic counterparts, but also still maintain their underlying dynamics as they are based on deterministic differential equations. Furthermore, spatial SIR models were explored through the use of meta-population and lattice-based models. The relative strength of transmission within sub-populations ρ is an important parameter for the interaction within populations. A higher ρ will allow for the disease to spread easier and reaches a bigger amount of people than for smaller rates of ρ.

References

[1] Matt J Keeling and Pejman Rohani. *Modeling infectious diseases in humans and animals*. Princeton University Press, 2011.

[2] Peter N Stearns. *Globalization in world history*. Routledge, 2016.