Stochastic SIR models

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1 Introduction

The SIR model can be modelled by solving a system of ordinary differential equations (ODEs), which describe a specific model. This can be solved in an discrete way by integrating the systems over time. This will give the time as a continuous variable.

However, this system of ODEs can also be solved by adding stochasticity, which gives a more accurate representation of real world examples.

In this report we present our findings on stochastic implementations of the SIR model. This includes a stochastic single population model, a stochastic meta-population model and a stochastic lattice based model.

2 Theoretical background

In this report we solely examine the stochastic behaviour of a SIR model. We add stochasticity into the SIR model by making use of the event driven Gillespie algorithm. In this algorithm we model each event instead of computing the values of X, Y, Z for every time-step. This becomes more clear when we look into the actual algorithm:

1. Label all possible events $E_1, ..., E_n$.
2. For each event determine the rate at which it occurs $R_1, ..., R_n$.
3. For each event, m, calculate the time until it next occurs, $\delta t_m = \frac{-1}{R_m} log(RAND_m)$
4. Find the event, p, that happens first (has the smallest δt).
5. The time is now updated, $t \rightarrow t + \delta t_p$, and event p performed.
6. Return to Step 2.

2.1 Single population

The events of a model can be extracted from the corresponding system of ODEs. The system of ODEs for a SIR with demography, with equal birth- and death-rate is described by:

$$\frac{dX}{dt} = \mu N - \beta XY/N - \mu X$$
$$\frac{dY}{dt} = \beta XY/N - \gamma Y - \mu Y$$
$$\frac{dZ}{dt} = \gamma Y - \mu Z$$

X, Y and Z being the number of susceptible, infected and recovered people respectively. This system translates into the following events (with corresponding rates):

	Rate	Event
Infection	$\beta XY/N$	X = X - 1, Y = Y + 1
Recovery	γY	Y = Y - 1, Z = Z + 1
Birth	μN	X = X + 1
Death of X	μX	X = X - 1
Death of Y	μY	Y = Y - 1
Death of Z	μZ	Z = Z - 1

When for example the rate of *birth* happens to occurs first, it will triggers the event of increasing the amount of susceptible people with one new person, $X = X + 1$.

Because of the stochastic nature of our model we expect to see different behaviour in comparison to the deterministic approach. This behaviour can be categorised in five hallmarks:

1. Variability: since we make use of random numbers, different simulations (with the same parameters) will have slightly different outcomes.

2. Negative co-variances: because of the interaction of the non-linear dynamics with the stochasticity we expect to see a negative co-variance between the X and Y class.

3. Increased transients: the transient of the stochastic implementation is increased with respect to the deterministic implementation. This is due to the perturbations being stochastic. However these perturbations are countered by the endemic attractor of the underlying deterministic model.

4. Stochastic resonance: the stochastic perturbations can have a frequency close to the natural frequency. In this case the model will resonate, creating oscillations around the endemic state.

5. Extinctions: the events our integer based, therefore extinction may occur. This happens in the long-term when Y is becoming zero, therefore the disease will go extinct and cannot be revived. In the short term the same behaviour may happen when Y_0 is small and due to the stochastic behaviour the event of recovery might be picked in the early stage.

2.2 Meta-population model

To simulate multiple populations we use the Gillespie algorithm as well. The set of events changed due to the change of our system of ODEs (ignoring demography):

$$\frac{dX_i}{dt} = \lambda_i X_i -$$
$$\frac{dY_i}{dt} = \lambda_i X_i - \gamma_i Y_i$$

With X_i being the amount of susceptible people in population i and Y_i the amount of infected people in population i. We also see the introduction of the force of infection λ_i which is described as follows:

$$\lambda_i = \beta_i \sum_{j=1}^{n} \rho_{ij} \frac{Y_j}{N_i}$$

With ρ_{ij} being the strength of transmission to population i from population j.

λ_i tells us that the force of infection of a sub-population i is the sum of the prevalence of all the populations.

For a model with two populations, the interaction matrix would be:

$$\rho = \begin{bmatrix} \rho_{ii} & \rho_{ij} \\ \rho_{ji} & \rho_{jj} \end{bmatrix}$$

We expect a delay in the spread of the epidemics between the population in which the disease was introduced with respect to the initially healthy populations. This is evident since initially all people are susceptible in the other populations and none are infectious, until a infectious person travels to this population. The rate of commuting is typically a low number, which explains the delay.

2.3 Lattice model

In the meta-population model space is not taken into account. This means that people from population 1 can commute to for example population 9 without passing any intermediate populations. However we can adjust the interaction

matrix ρ in such a way that ρ_{ij} only has a value if i and j are defined as neighbours.

We create an algorithm in such a way that a NxM grid would turn into the appropriate interaction matrix ρ with only a value for ρ_{ij} if i and j are said to be neighbours on NxM grid.

Taking the spatial component into account, the SIR model including demography (with N being constant) can be modelled using the following system of ODEs:

$$\frac{dX_i}{dt} = \mu N - \beta \frac{X_i}{N}((1 - \sum_j \rho_{ji})Y_i + \sum_j \rho_{ij} Y_j) - \mu_{X\,i}$$
$$\frac{dY_i}{dt} = \beta \frac{X_i}{N}((1 - \sum_j \rho_{ji})Y_i + \sum_j \rho_{ij} Y_j) - \gamma Y_i - \mu Y_i$$

According to Keeling and Rohani [1] the wave of the spreading of the disease throughout the grid is linear.

3 Experimental methods

3.1 Stochastic SIR model

3.1.1 Variability

Running the same model 10 times will result in the plot displayed in figure 1.

Even though we are running the model with the same parameters we get different outputs due to the random behaviour. Therefore a correct way to model situations is by running multiple repetitions and extracting the mean. In the rest of our experiments we choose to work with a random seed, which will give us the same random number for every instance we run. Therefore we can sustain the integrity of our plots.

3.1.2 Negative co-variance

The co-variance between X and Y in the stochastic model is expected to be increasingly large. This is due to the fact that we model each event singly, meaning that no event can happen at the same time-step. In our model specifically this means that an event which increases X will always decrease Y and vice versa, when not taking demography into account.

In order to explore the negative co-variance we create a simulations with different R_0, each containing 100 repetitions. We then extract the mean of the multiple co-variances between X and Y for each R_0. The results in figure 2 shows us that indeed the co-variance is always negative.

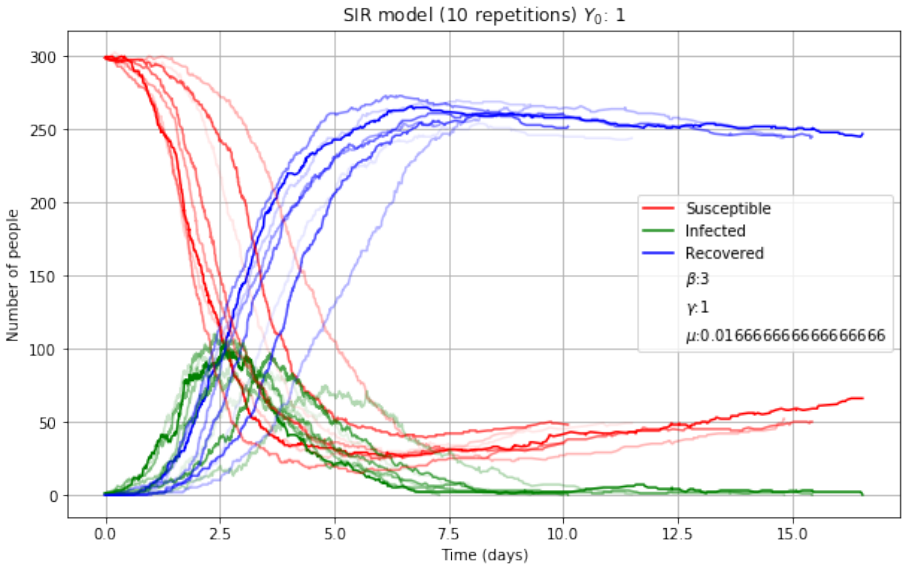

Figure 1: Variability in stochastic SIR

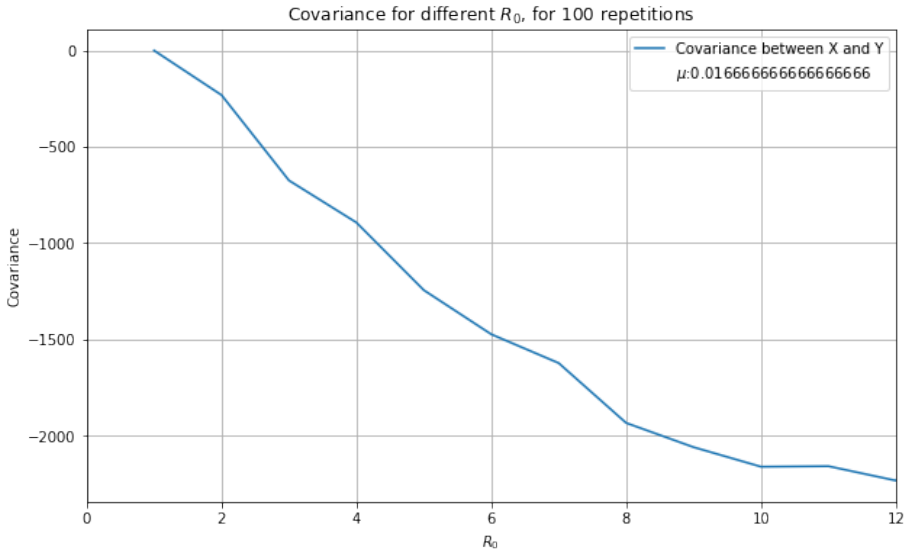

Figure 2: Co-variance between X and Y

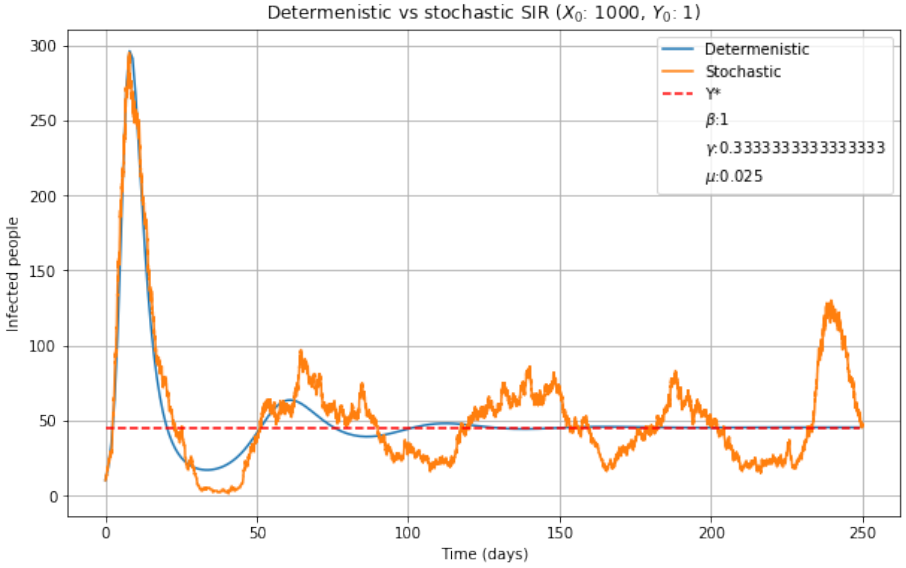

Figure 3: Transients of deterministic and stochastic model

3.1.3 Increased transients

The transient of our model is the part before it reaches the endemic equilibrium. The endemic equilibrium $Y*$ is a stable state in which $Y > 0$. We can compute $Y*$ by using the formula derived by Keeling and Rohani [1]:

$$Y* = \frac{\mu}{\beta}(R_0 - 1)$$

We know that our stochastic model will not be stable because of its stochastic nature. However, due to the underlying deterministic model the perturbations get attracted to the stable state, thus creating fluctuations around this stable state. In figure 3 we can see a demonstration of such behaviour.

We note in figure 3 that the endemic equilibrium is reached at around 140 days for the deterministic model. This equilibrium is reached by the stochastic model at 150 days, making the transient of the stochastic model to be longer.

3.1.4 Stochastic resonance

If we zoom in on figure 3 we get a more clear view of the stochastic resonance in our model, which is pictured in figure 3. In this figure we see that the stochastic model has a lot of perturbations. Sometimes the perturbations have the same frequency as the natural frequency of the underlying deterministic model. This cause the model to resonate, creating the drastic changes. We can see this for

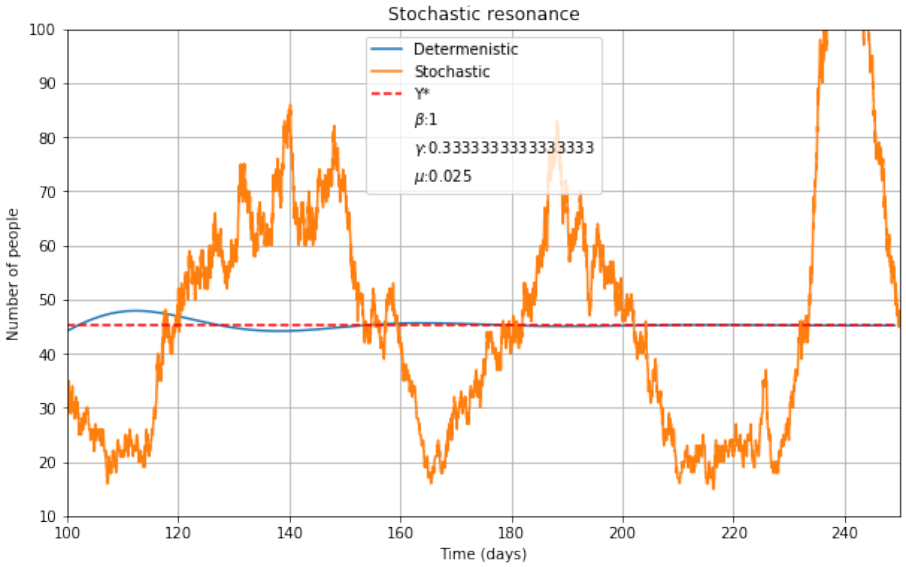

Figure 4: Stochastic resonance

example at $T = 150$ where the amount of infected people changes from 70 to 50 in under 10 days.

3.1.5 Extinction

To counteract the extinction of our Y class we add imports to our model. These imports are infectious immigrants and lead to the event $Y = Y + 1$, with rate δ. The result can be found in figure 5.

In this figure we see that the disease dies out at around ten days. However when the imports are introduced we see an increase in the amount of infected people. At around 18 days the disease gets reintroduced by an import, however it dies out quickly. At around the 20 days the disease gets reintroduced again, this time to more effect starting a small epidemic.

3.2 Meta-populations

For our meta-population experiments we choose to start with two stochastic meta-populations, both containing 300 people. We define our interaction matrix as:

$$\rho = \begin{bmatrix} 1 & \frac{1}{1-0} \\ \frac{1}{100} & 1 \end{bmatrix}$$

We introduce the disease in population 1, by adding one infected person, of which the result is shown in figure 6.

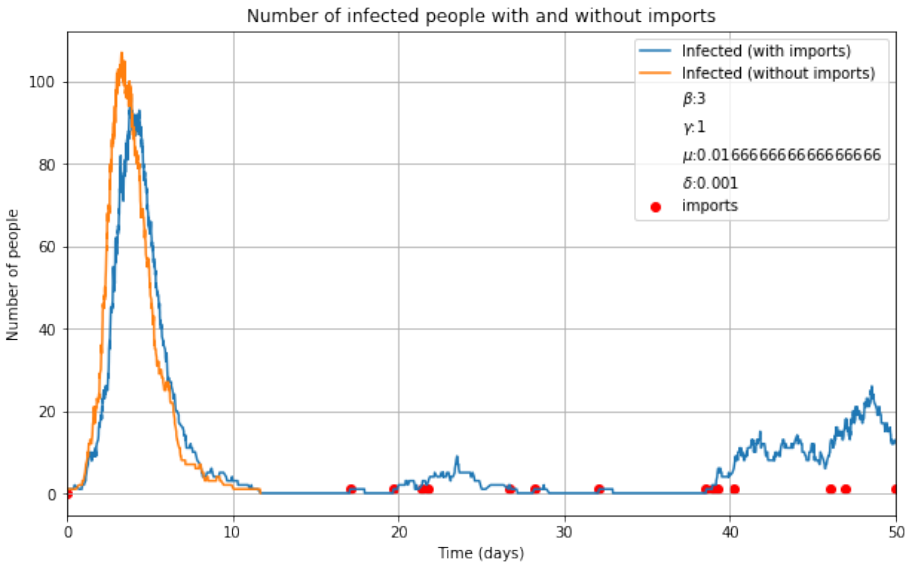

Figure 5: Countering extinction with imports

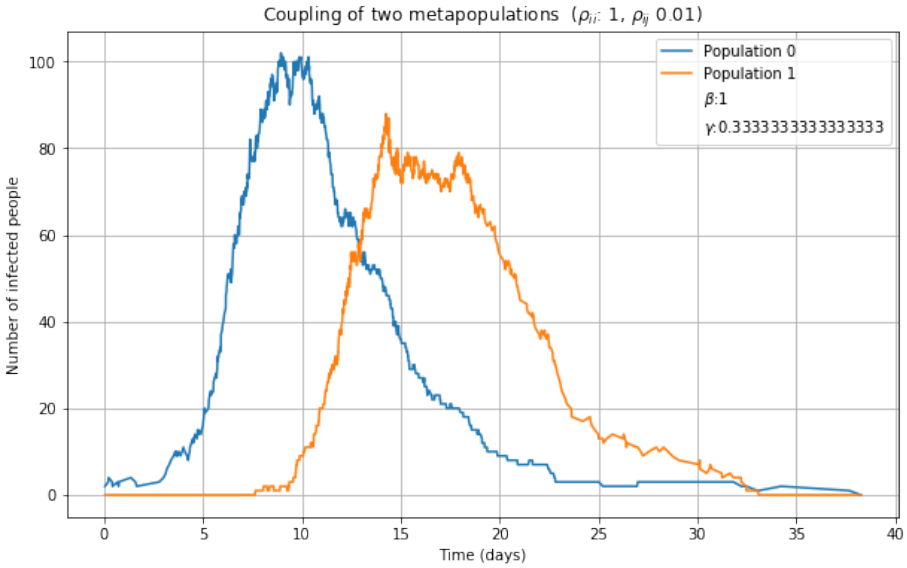

Figure 6: Delay of epidemics in two meta-populations

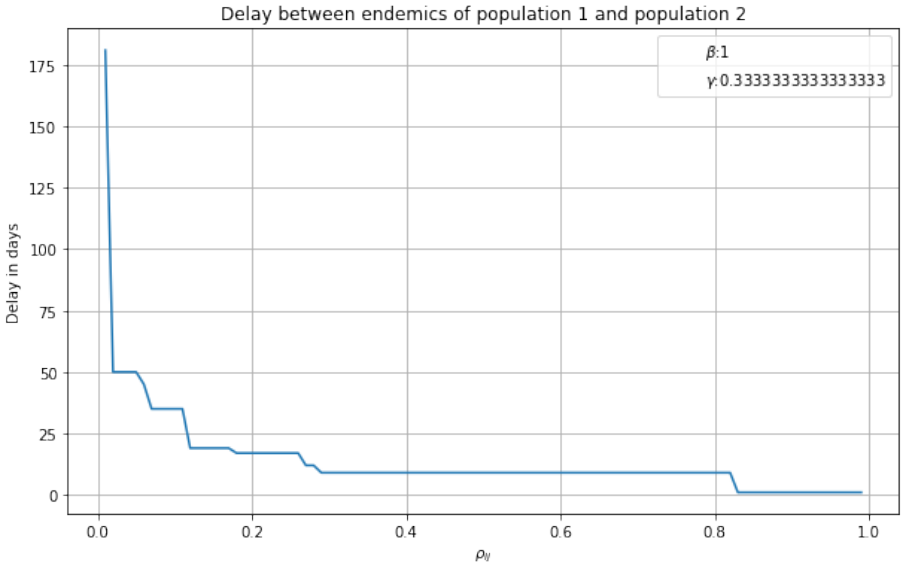

Figure 7: Delay for different ρij

We see a delay in the spread of the epidemic in population two. We further examine the delay between epidemics by setting out different values for ρ_{ij} and measure the delay in days, which is shown in figure 7. In this figure we can see that the relationship between the delay and the ρ_{ij} seems to be exponentially.

We now introduce a third population, changing the interaction matrix to:

$$\rho = \begin{bmatrix} 1 & \frac{1}{100} & \frac{1}{1000} \\ \frac{1}{100} & 1 & \frac{1}{1000} \\ \frac{1}{1000} & \frac{1}{1000} & 1 \end{bmatrix}$$

The interaction rates from i to 3 and vice-versa are set to be much lower than the interaction rates between population 1 and 2. Therefore 3 can resembles a distant population.

The result is shown in figure 8. We see a delay of the epidemic in the three different populations. However, the delay between the epidemics of population 2 and population 3 is much lower than the delay between the epidemic of 1 and 2. This is because of 3 being affected by 1 and 2, while 2 is initially only affected by 1.

3.3 Lattice based model

Since the spatial component is taken into account, the lattice based model is a more accurate representation of the spreading of a disease for example in a country.

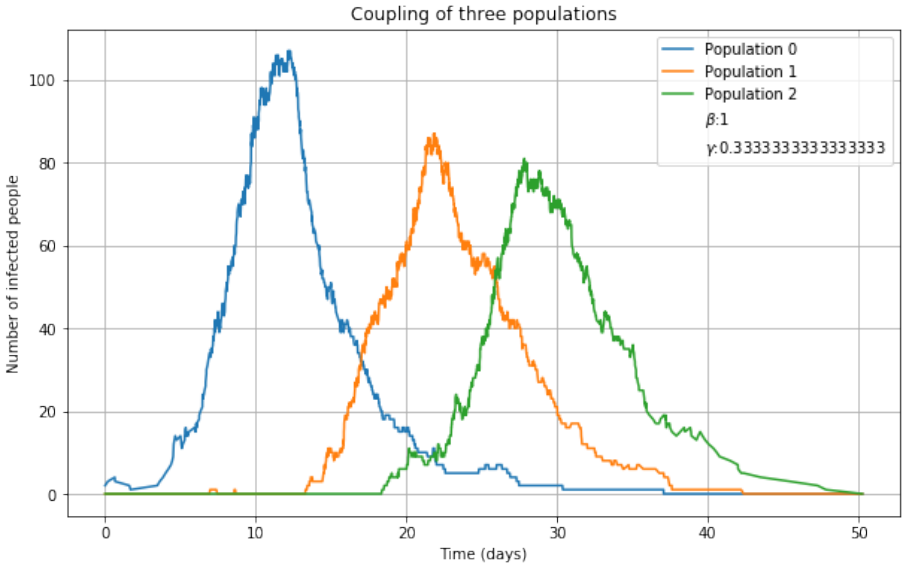

Figure 8: Three meta-populations

We choose to model 100 populations on a 10x10 grid, each containing 100 persons. We thus have a total population of 10.000 people. We start by introducing 10 infected people into the class P_{start}. In our visualisations of this lattice (shown in figure 9) the colour represents the amount of infected people for each box, yellow being the highest value and purple the lowest.

In figure 9 we see the disease spreading outwards, to the neighbours of P_{start}. We know that speed of this spreading is determined by R_0 and ρ. However we did not manage to determine the speed and thus could not find the relation of them to the speed. Since we did not manage to determine the speed we could not ascertain whether the speed was linear.

4 Conclusion

For the stochastic single population model we explored the five hallmarks of stochastic behaviour. We verified the variability, the negative co-variance between X and Y. Also We witnessed the increased transients and the stochastic resonance. Finally, we countered the extinction through imports.

For our research to meta-populations we started of with coupling two populations. We then introducing ten infected people in population 1 and zero in population 2. We have seen that the epidemic happens in both the populations, with population 2 having a delay. The delay in our model has an exponential relationship to the ρ_{ij} for two populations with $\beta = 1$ and $\gamma = \frac{1}{3}$.

After introducing a new distant population into our model we witnessed that

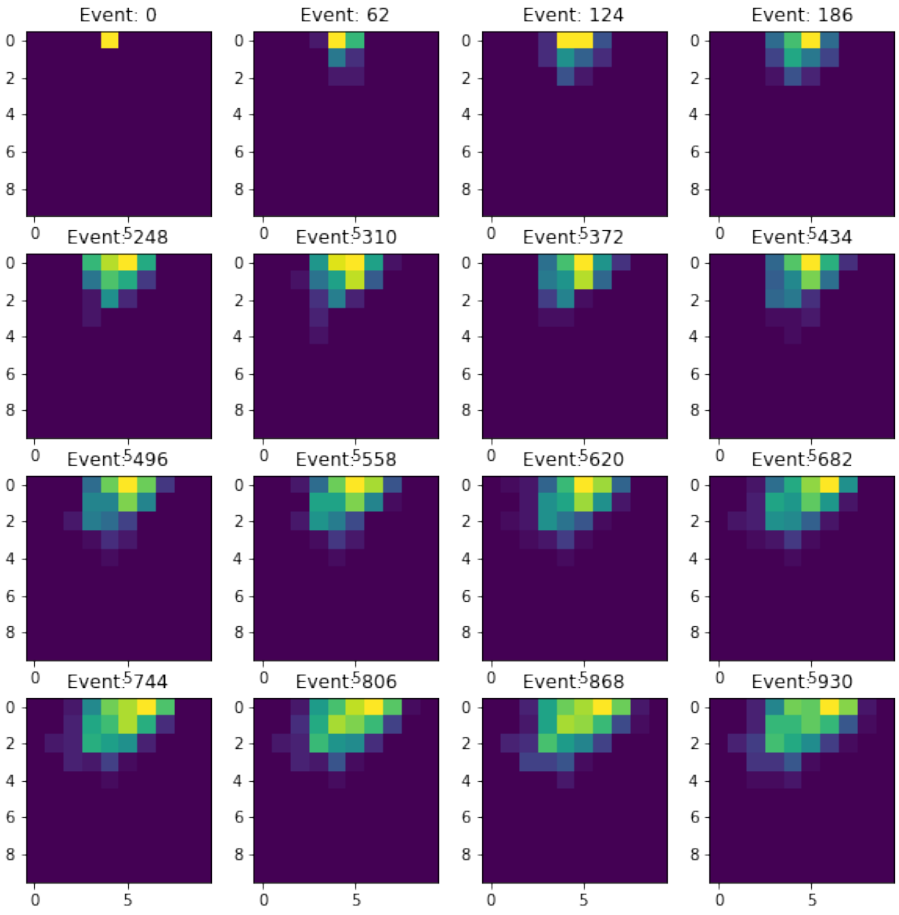

Figure 9: Visualisation of spreading of disease through lattice

the epidemic in this population also has a delay. However the delay between 2 and 3 was smaller than the delay between 1 and 2, even though the interaction rates to and from 3 were much lower. This was caused by the fact that population 3 is affected initially by two infected populations with infected people, while 2 is only infected by 1.

The lattice based model showed us the spread of a disease in a two-dimensional-space of 100 populations containing 100 people each.

5 Discussion

The plot showing the increased transient could have been fitted to other parameters to get a more clear image of where the transient of the stochastic model ended.

The perceived delay in the meta-populations could have been compared to the delay in the non-stochastic implementations because we know that this delay is supposed to be smaller. Also, in future work we verify whether the relationship between the delay and ρ_{ij} is exponentially for all β and γ.

Finally, in order to make a more comprehensive review of the lattice based model we should have determined the speed of the wave in order to make more statements. This could be done by computing the derivatives of the different time-steps. This would show us the change over time, which should have a linear relation. Due to the lack of time we could not apply this.

References

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