Eidgenössische Technische Hochschule Zürich
Swiss Federal Institute of Technology Zurich

A study of the effect of vaccination on an agent-based stochastic SIRS model

Delmotte, Nicholas
ndelmotte@student.ethz.ch

Pedrelli, Loris
lpedrelli@student.ethz.ch

Rojkov, Ivan
irojkov@student.ethz.ch

6th of December, 2019

Supervisor: Dr. Antulov-Fantulin, Nino

Abstract:
An SIRS model was stochastically implemented together with an agent based mechanism in order to simulate the seasonal spreading of influenza in a localised region and the prophylactic effect of a vaccine on the number of infected people. Susceptible, Infectious and Recovered densities of people in the system were calculated. Firstly the herd immunity effect was observed in local systems, then many characteristic quantities of the system, such as propagation speed, maximal infectious area, maximal distance of the illness, were analysed when varying infection rate and size of the system. A phase transition was observed in those quantities, indicating a change in the geographical expansion of the disease, from local to global.

Contents

1 Introduction

On the 11th of March 2019, the World Health Organisation (WHO) launched the Global Influenza Strategy for 2019-2030 aimed at preventing seasonal influenza with vaccines, controlling the spread of influenza from animals to humans and preparing for the next influenza pandemics. Seasonal influenza (commonly called flu) is a viral disease which affects worldwide about 3 to 5 million people and causes about 290'000 to 650'000 respiratory deaths per year [1]. In this report the effect of vaccines and the importance of collective vaccination behaviour will be studied by implementing an SIRS model mixed with an agent based model which simulates the human interaction between neighbours. This localised interaction allows us to study the spreading of the disease with regards to the topology the system. To do so, some characteristic quantities of the system, and of its agents, will be defined and then their behaviour will be studied under the variation of different parameters of the model.

2 Theory and numerical implementation of the model

2.1 Grid-like disposition

To modelise the spread of a disease through a population, we imagine a grid-like disposition, where each square corresponds to one person (cf. Fig. 1). Thus, each person is surrounded by eight neighbours with whom he can interact. Numerically, this grid of people is implemented as an N by N matrix of structures (by default $N = 100$). These structures contain all the attributes of a person (*state*, *vaccinated*, *reward*, *age*). This disposition allows a disease to spread via contact from neighbour to neighbour.

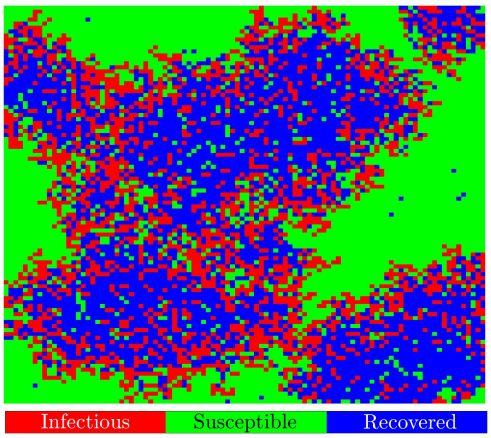


Figure 1: Example of a grid-like system showing the states of the agents.

2.2 SIRS model

A very common model used in epidemic simulation is the Susceptible-Infectious-Recovered-Susceptible model (SIRS): each individual belongs to one of the three classes (see attribute *state*): Susceptible (S), Infectious (I) or Recovered (R). The transfer from one class to another is regulated via some rates r: the infection rate β, the recovery rate γ, the rate at which the individual gets susceptible again α. In this model an individual can also die at a mortality rate μ. In order to keep a closed system (i.e. a constant total number of people), any dead individual is replaced by a susceptible newborn. In addition, a θ coefficient is added in order to consider the zero event, i.e. the case when nothing happens. The evolution of this model is implemented stochastically. During one step of the illness evolution, a grid cell (i, j) is randomly chosen using two uniformly distributed random variables $x, y \sim \mathcal{U}(0, 1)$ following:

$$i = \lfloor Nx + 1 \rfloor \qquad j = \lfloor Ny + 1 \rfloor \tag{1}$$

Then, depending on the cell's state A, the probability of changing from this to state B, $p_{A\to B}$, is computed as follows:

$$p_{A\to B} = \frac{Q_{A\to B}}{Q} \tag{2}$$

using

$$Q_{A\to B} = \sum r_{A\to B} \qquad\qquad Q = \sum_B Q_{A\to B} \tag{3}$$

where $Q_{A\to B}$ is the total rate of transition from A to B on the whole lattice and Q is the total rate of something happening. Note that in the case of a constant rate on the lattice we will have $Q_{A\to B} = N^2 r_{A\to B}$. The rates in equation (3) are specific to the couple of states A and B that we are considering and are summarised in table 1.

Transition	$r_{A\to A}$, $\forall A$	$r_{A\dagger\to S}$, $\forall A$	$r_{S\to I}$	$r_{I\to R}$	$r_{R\to S}$
Rate	θ	μ	β	γ	α

Table 1: Rates for all the possible transitions in the system. $r_{A\dagger\to B}$ means that going from state A to state B involves the death of the individual.

In order to choose which transition occurs, a uniformly distributed random variable $z \sim \mathcal{U}(0,1)$ is used. The condition for transition i to be chosen is then:

$$\sum_{k=0}^{i-1} \frac{Q_k}{Q} < z \leq \sum_{k=0}^{i} \frac{Q_k}{Q} \tag{4}$$

then the attributes, among which the *state*, of the individual in the cell are updated. Note that if $Q = 0$, then nothing happens.
Finally, time t is updated by a time interval Δt which is chosen exponentially in order to have memoryless intervals. To do so, a last uniformly distributed random value $w \sim \mathcal{U}(0,1)$ is taken; then the time interval is given by

$$\Delta t = -\frac{1}{Q}\ln(1-w) \tag{5}$$

and time is updated as $t \to t + \Delta t$ as well as the *age* of each person in the system.

2.3 Choice of the SIRS rates r

The rates in table 1 need to be defined in order to run a simulation. All the rates are defined in per-week units.

Rate β is defined on the basis of actual data of the infection rate of influenza in Switzerland during years 2017, 2018 and 2019 [2], which gives a time dependent contribution $\beta_{season}(t)$. In addition the rate at node (i,j) is also dependent on the local density of Infectious individuals $\rho_I(i,j)$ defined as the fraction of Infectious among the nearest neighbours:

$$\beta = \beta_{season}(t)\rho_I(i,j) \tag{6}$$

Numerically, in order to drive the disease, and to ensure it is never eradicated fully, a new 'Patient Zero' is chosen periodically. This just means that about once a week, a random person is given the disease. If the disease is already present, this person is statistically

insignificant, and if the disease is eradicated, this person allows the system to continue evolving by reintroducing it.

Rate γ is defined as the inverse of the recovery time. For children under 15, the recovery time is taken to be from 5 to 10 days and for adults from 3 to 8 days [3], thus

$$\gamma^{-1} = \begin{cases} (5+5x)/7, & \text{if age} < 15 \\ (3+5x)/7, & \text{if age} \geq 15 \end{cases} \tag{7}$$

where $x \sim \mathcal{U}(0,1)$ is a uniformly distributed random variable.

Rate α is defined as the inverse of the time of validity of a vaccine (around 6 months for influenza [4]), then this rate is given by:

$$\alpha = \frac{1}{6 \cdot 4} \tag{8}$$

Rate μ is defined on the basis of actual data of mortality in Switzerland during years 2016, 2017 and 2018 [5]. Thus the mortality rate will be age dependent.

Rate θ was chosen to be of the same order of magnitude as β and γ and set to 1 in order to stabilise the simulation.

2.4 Vaccination based on a reward system

Whilst the SIRS model is useful to simulate the spread of diseases, the main interest of this project is to study vaccination trends. For this, it is necessary to introduce an additional parameter to the existing SIRS model: the choice of vaccination. Moreover, the decision to vaccinate oneself should be based on the perceived risk of getting vaccinated versus the risk of getting ill. As such, it is also necessary to introduce a reward system that encapsulates the costs and benefits of passing from one state of the SIRS model to another. This reward can be seen as the memory a person has of his past experiences with the illness.
Overall, our model thus characterises a person using four key variables:

1. The person's *state* (S, I or R)
2. The person's *vaccination* choice (vaccinator or not)
3. The person's *reward* based on past experiences (represented by a scalar)
4. The person's *age*. This variable is mainly used to determine the rate of death and has little bearing on the simulations.

Note that the vaccination choice and the state of vaccination (a person is or is not vaccinated) are not always the same. In the following results section the plotted quantity will always be the vaccinator density, and not the vaccination density.
The overall model, composed of the SIRS model and the reward-based vaccination system, can be summarised by the diagram in figure 2.
Numerically, as shown in figure 2, three rewards rw are implemented:

1. The (negative) reward for falling ill, which is set to -10.
2. The (positive) reward for recovering from the illness, which is set to +2.
3. The (negative) reward for vaccinating oneself, which is set to -4.

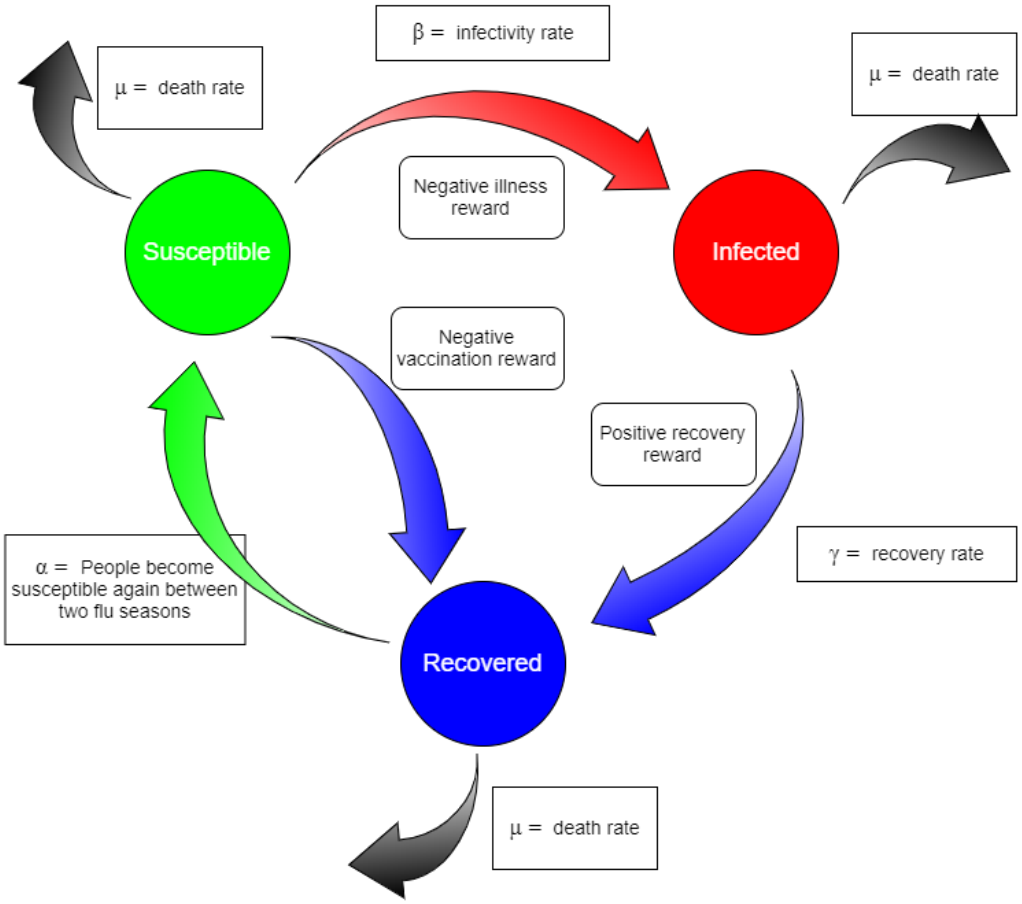


Figure 2: Diagram summarising all the interactions and the rewards in the system [6].

The reward for recovering is important since it helps to differentiate the case where someone died before recovering, and since the reward of a dead person is transmitted to the new person born in its place (we consider that in a family the experience is transmitted directly to the new generation).
The total reward of a person is used to compute the likelihood of them changing their vaccination strategy. This total reward is actually split into two parts: the total reward accrued whilst the person was a vaccinator, and the total reward accrued whilst the person was a non-vaccinator. Thus, when looking at one's neighbours, one will only look at the rewards accrued during their current choice.
Numerically, each person (or element of the grid) calculates the average reward rw_V of his vaccinator neighbours (including himself if he is a vaccinator), and the average reward rw_{NV} of his non-vaccinator neighbours (including himself if he is a non-vaccinator). He then computes the difference Δrw between the average reward of his 'faction' and the average reward of the opposite 'faction'. This Δrw is then plugged into a Fermi-Dirac distribution of the form [7]:

$$f(x) = \frac{1}{\exp(ax + b) + 1} \tag{9}$$

where we set $a = 1$ and $b = \ln(1/\epsilon - 1)$, $\epsilon = 1/N^2$, N^2 the size of the system. A visualisation of this function can be found in figure 3.
This function returns 0 for $x \gg 0$, 1 for $x \ll 0$ and ϵ for $x = 0$. Thus, if the average reward of a person's 'faction' is much larger than the average reward of the other 'faction's', then $\Delta rw > 0$ and $f(\Delta rw) \approx 0$, and for the opposite case $f(\Delta rw) \approx 1$.
This function f is therefore the probability to change vaccination choice.
In the special case where the person and all his neighbours share the same 'faction' (as is the case at the beginning of the simulation when everyone is set by default to 'non-vaccinator'), the value of Δrw is set to 0. The decision whether to vaccinate or not is made every week [8].

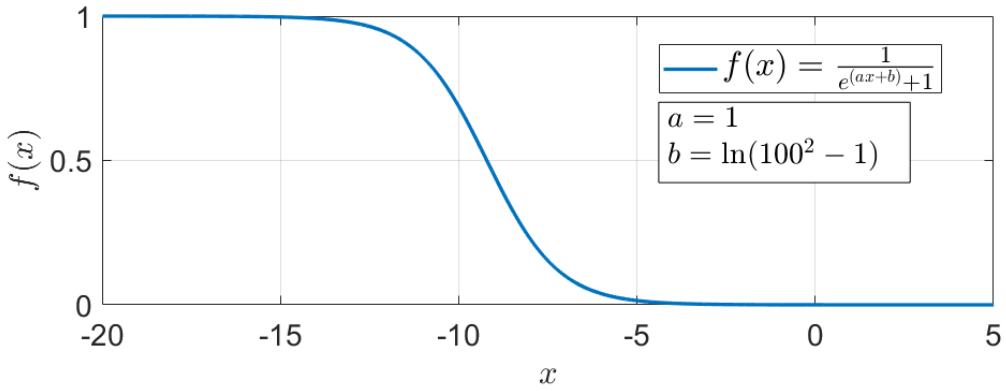


Figure 3: Probability distribution used in the vaccination process in the case of $N = 100$.

2.5 Displacement

Imagine now that the system represents a crowded room (for example a waiting room in a hospital or a public square during Christmas markets), then it would make sense to give the possibility to the agents to move across the grid looking for the best position far away from Infectious people. To do so, after each step in the evolution of the illness, the agent in cell (k, l) decides whether to remain where it is (with probability p_r) or move to cell (i, j) (with probability $p_{(k,l)\to(i,j)}$). These probabilities depend on the current state of the chosen agent:

State	$\rho_{\mathbf{I}}(k,l)$	$\mathbf{p_r}$	$\mathbf{p_{(k,l)\to(i,j)}}$
S	$= 0$	1	0
S	> 0	$(1-\rho_I(k,l))/(1-\rho_{I,tot})$	$(1-\rho_I(i,j))/(1-\rho_{I,tot})$
I or R	≥ 0	1/2	$1/(2N_{nn})$

Table 2: Table summarising the probabilities of displacement for each state.

where N_{nn} is the number of nearest neighbours of cell (k, l) and $\rho_{I,tot}$ is the sum of the densities of infectious over all the nearest neighbours.

3 Results

In this whole chapter, the parameters were chosen as described in section 2 unless specified. Note as well that at $t = 0$ the system is initialised such that all rewards are 0, ages are

chosen following a probability distribution based on ages in Switzerland, all people are Susceptible to start, and all people are also non-vaccinators to start.

3.1 Static SIRS system with and without vaccination

First of all the SIRS model is tested to verify that it works in its fundamental setup, i.e. without any vaccine and with static agents. The β rate is kept to be seasonally variant. The model is run during ~ 300 weeks and the resulting densities of S, I and R in the system are given in figure 4 (*top*). The same simulation is then run but turning the vaccination mechanism on. The same quantities are given for this simulation in figure 4 (*bottom*).

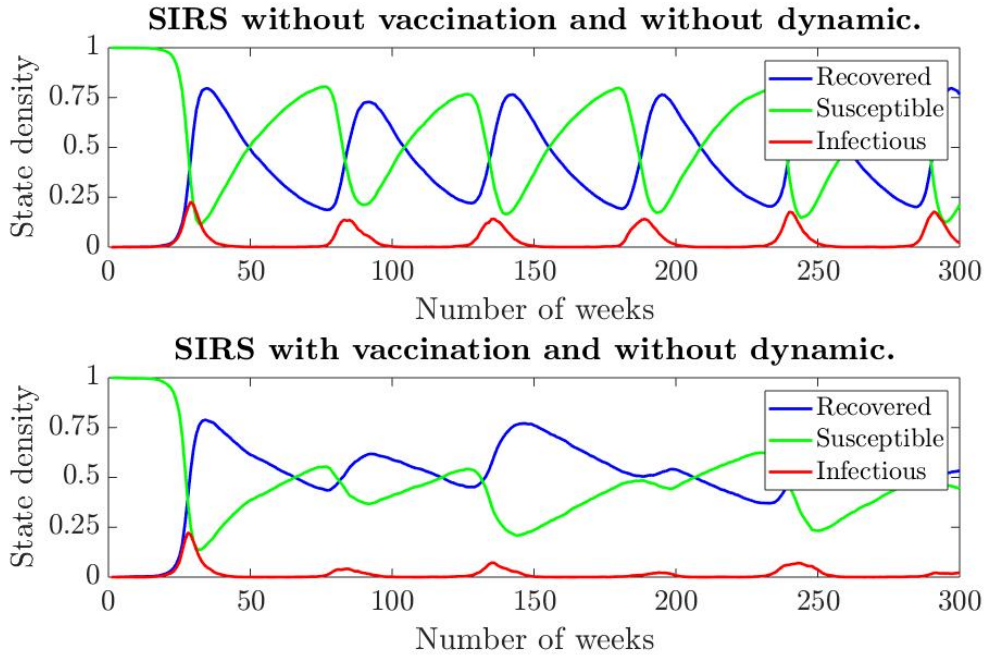


Figure 4: Two SIRS models: (*top*) without vaccination and with static agents; (*bottom*) with vaccination and with static agents. The periodic trend is caused by the use of a seasonal β rate. In the case with vaccination (*bottom*), the spreading of the illness is strongly damped.

From comparing these figures it appears obvious that the effect of vaccination is to diminish the spreading of the illness in the population. Indeed the peaks in the infectious density are all more or less of the same height when vaccination in absent but become sensibly smaller after the first peak in the case of a vaccinating system.

3.2 Difference between fixed and varying β

In this section, the system size is set to $40 \times 40 = 1600$ *cells.*

Until this point, the model has been characterised by a flu-like infection rate β, that peaks during 'flu season' and ebbs during the rest of the year. It can be interesting, however, to study an illness with a fixed infection rate throughout the year.
Figures 5 and 6 show the difference in vaccination rate for these two different forms of the β function. For a seasonal infection rate, the vaccination rate follows a sharp, yearly, up and down trend, reflecting the fact that people can only be infected at a specific point in the year. For a fixed infection rate, the average vaccination density is slightly higher

since more people will be infected over a year. Additionally, the trend is less sharp since people are more likely to be immediately punished for not vaccinating, as opposed to the slower yearly punishment of the seasonal infection rate.

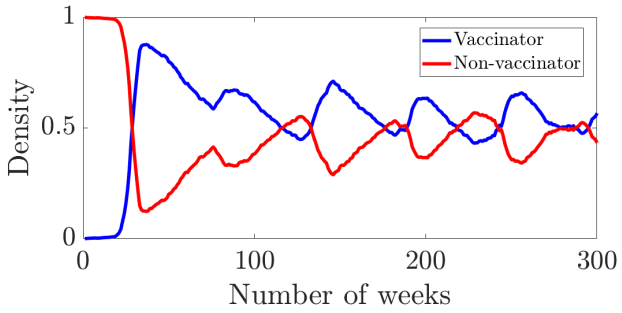


Figure 5: Vaccinator density for seasonal β

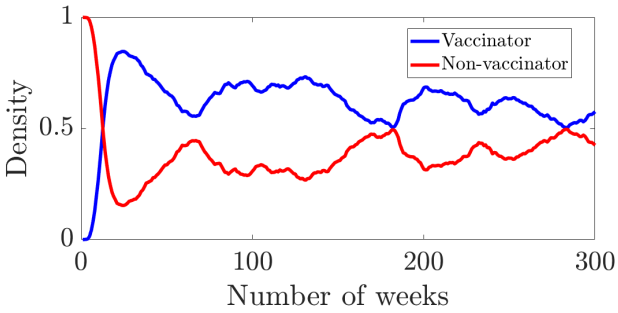


Figure 6: Vaccinator density for β fixed (approx.) at the peak of seasonal β

3.3 Static Patient Zero

In this section, the system size is set to $40 \times 40 = 1600$ *cells.*

In all the simulations until this point, a new 'Patient Zero' of the disease was chosen each week in a random location. It can however be interesting to study how the vaccination density can vary locally in the system if the Patient Zero is fixed in a certain position. Figures 7 and 8 show the normal case of a randomly distributed Patient Zero. Figure 7 shows the yearly periodic variation of the disease, as expected, with each outbreak being more or less the same intensity. In figure 8, the square system has been divided into four smaller squares (of size $\lfloor N/2 \rfloor \times \lfloor N/2 \rfloor$), and then the local vaccination density has been computed for each smaller square. Unsurprisingly, since the Patient Zero's location is chosen randomly, all four squares follow the same trend.

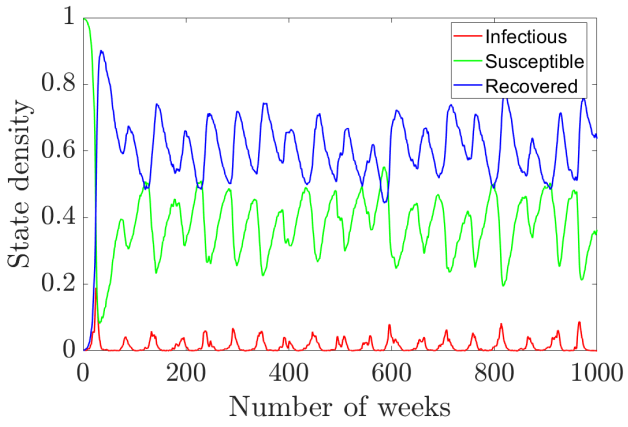


Figure 7: State density for normal random Patient Zero distribution.

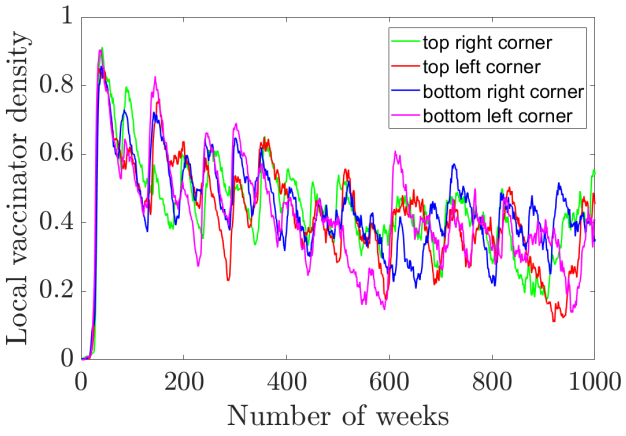


Figure 8: Local vaccination density for normal random Patient Zero distribution.

In figures 9 and 10, the Patient Zero's location has been restrained to the cells in the upper left corner (x and y positions from 1 to 10). Figure 9 shows more spread out disease outbreaks, of varying intensity. It seems intuitive that when a disease's origin is known

(at least approximately), it is more easily contained. Figure 10 shows the same local vaccination density as before. The top left square now has a noticeably higher vaccination rate than the other squares. The bottom right square also has the lowest vaccination rate, since it is located furthest from the origin of the disease.

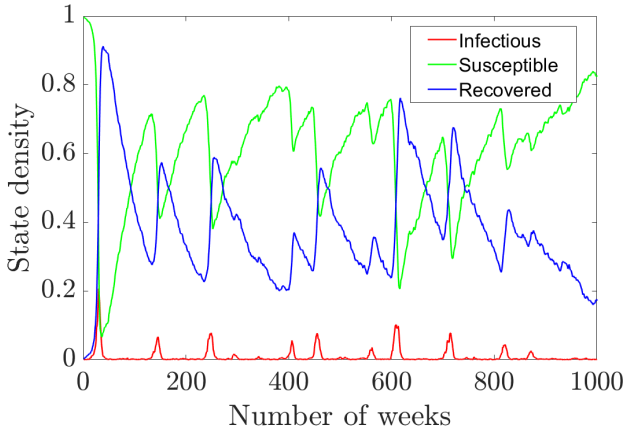


Figure 9: State density when Patient Zero is in [1:10,1:10] range.

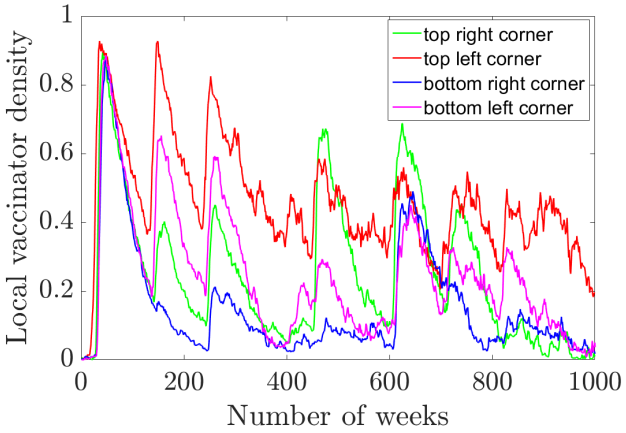


Figure 10: Local vaccination density when Patient Zero is in [1:10,1:10] range.

In figures 11 and 12, the Patient Zero's position has now been fixed, again in the upper left corner (in cell [3,3] exactly). Figure 11 shows an even sparser distribution of outbreaks. Figure 12 shows the local vaccination density. As expected, the upper left square has the highest vaccination density.

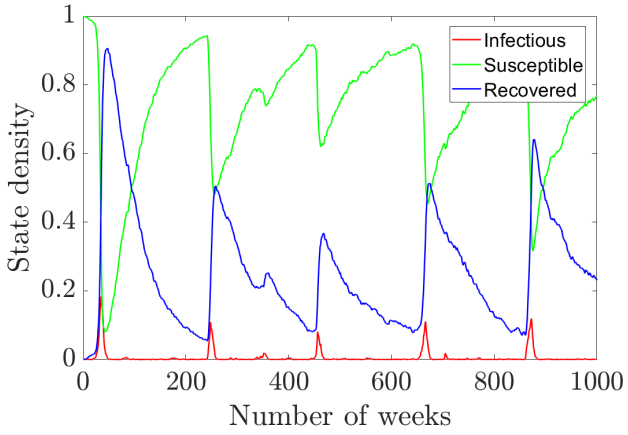


Figure 11: State density for Patient Zero at fixed [3,3] position.

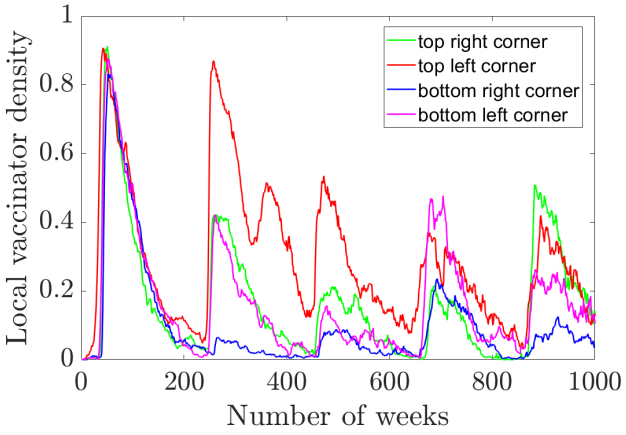


Figure 12: Local vaccination density for Patient Zero at fixed [3,3] position.

Several interesting remarks can be made. Firstly, as mentioned previously, as the position of the Patient Zero is focused, the number of disease outbreaks diminishes. This on its own is not surprising, but it can also be noticed that, despite the closest neighbours keeping a high vaccination density, the overall vaccination density in the system appears to diminish. This is not entirely intuitive since one might assume that for a lower overall vaccination density, the disease would be more likely to spread. However, figures 11 and 12 show that, for a fixed Patient Zero, outbreaks only depend on the local vaccination density of the

upper left square. Because these cells have such a high incentive to vaccinate, they shield the other cells of the system, allowing the population overall to greatly benefit.
A final point of interest is the fact that in figures 10 and 12, the vaccination peak of the upper left square is sometimes lower than the peak of one of its neighbouring squares. If one looks closely, one can see that these cases correspond to the worst outbreaks of the disease, that is to say when it fully breaks out of the upper left square and spreads to the whole system. Such an outbreak affects the whole system equally. However, before the outbreak, the upper left square naturally had more vaccinators, and was thus less negatively impacted by the outbreak than its neighbouring squares. This explains why the reaction of the neighbouring squares was bigger in these cases.

3.4 Variation of infection rate β

In this section the Patient Zero is always considered to be fixed in the center of the system.

The next simulation consists in varying β from 0.1 to 10 (note that β is fixed at these values; it is not seasonal) and, for each such value, letting the system evolve during 200 weeks. The infectious agent ratio is then plotted for each value of β in figure 13 (*left*). From this graph it seems clear that as β increases, the peak of maximal infection moves to the left. The same behaviour is observed for the peak of maximal recovery as can be seen in figure 13 (*right*) where the number of recovered agents is plotted for different values of β. This seems to be logical since if the infection rate β is larger, then at the beginning the disease will spread much faster arriving earlier at its maximum and consequently people will recover at earlier times.

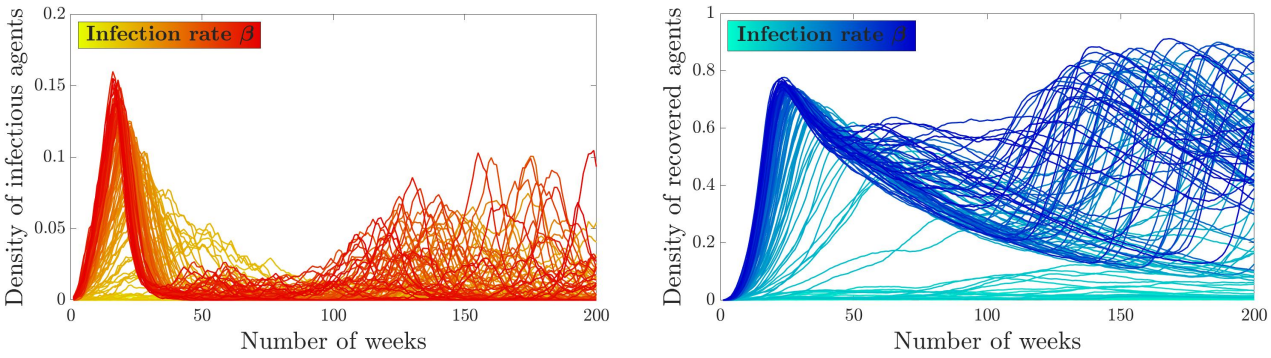


Figure 13: (*left*) Infectious density and (*right*) Recovered density plotted for each value of rate β. Both peaks move to earlier times when β increases.

Another quantity which describes how the infection spreads is the *maximal infectious area* defined as the surface of the largest connected cluster of infected people in the system. This was computed for each beta and the result is given in figure 14. The last quantity computed under the variation of β is the *propagation speed of the illness in the system* defined as the height of the first infection peak divided by the time elapsed from the beginning of the simulation. The result is given in figure 15. Both these quantities go through a phase transition as β approaches 1, meaning there is a sharp change in these quantities. The existence of such a phenomenon can be explained by the following: there exists a certain critical value of the infection rate $\beta_{crit} \approx 1$ at which the disease transitions from local, i.e. the infectious people are contained in a part of the system, to global, i.e. the infection is diffused in the whole system. After a critical infection rate β_{crit}, the

maximal infectious area and the propagation speed increase following a power law with exponents 0.58 and 0.42 respectively.

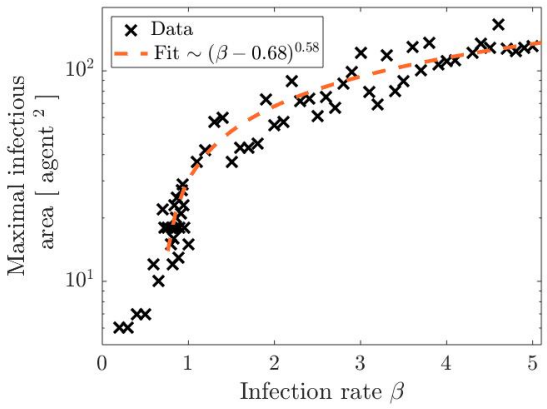


Figure 14: The maximal infectious area increases after $\beta_{crit} \approx 0.68$ with a power of 0.58.

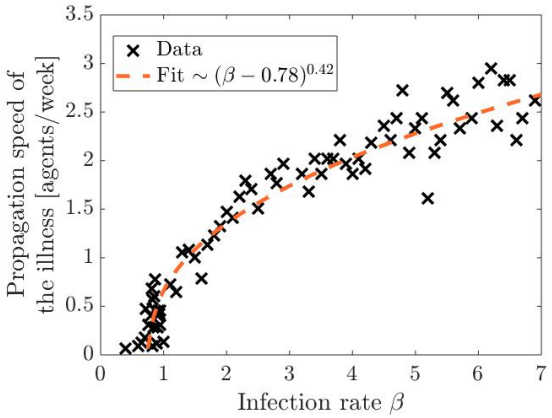


Figure 15: The propagation speed of the illness increases after $\beta_{crit} \approx 0.78$ with a power of 0.42 .

3.5 Variation of system size N

Here the size of the system N is varied between $N = 6$ and $N = 150$ and for each value of N the system evolves during 200 weeks. The value of β is fixed at the previous critical value 1, and for each simulation the 'Patient Zero' is considered fixed in the center of the system.

As before, infectious and recovered agent densities are plotted for each value of N and are presented in figure 16 (*left*) respectively (*right*). Unlike before, the peak of maximal infection and maximal recovery move to the right as the dimension of the system increases. This can be explained by the fact that, as the N increases, the illness will arrive more slowly at the boundary of the system. In addition people will begin to recover around Patient Zero before the outbreak has reached the limits of the system and thus the peak of infection will arrive later and will be smaller, with respect to the total number of people in the system.

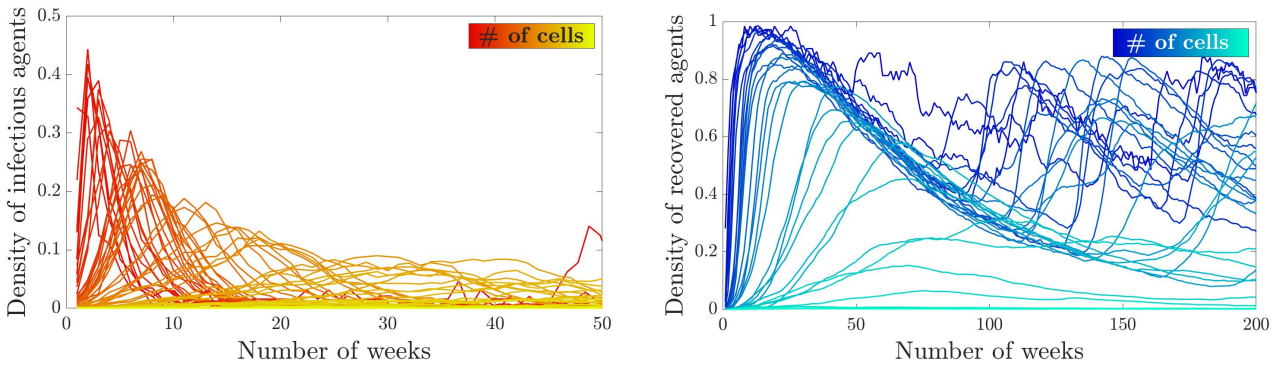


Figure 16: (*left*) Infectious density and (*right*) Recovered density plotted for each value of N. Both peaks decrease and move to later times when β increases.

This time the maximal area of infection needs to be normalised in order to compare the different obtained values which are presented in figure 17. The graph has a peak when

N approaches 18. This is actually more likely to be a limit of the numerical model than the manifestation of a physical phenomenon. Indeed for very small systems the stochastic method is much less reliable, and can give very different results for two runs of the same simulation. Thus the points for small systems should be discounted. The remaining points (after $N = 20$) follow the expected trend. That is to say that, as the system size grows, the size of the maximal area of infection remains relatively constant, but the normalisation factor does not. Thus a slow decline can be observed.
Another interesting calculated quantity is the maximal distance of an infectious agent from Patient Zero. This is computed when varying N and is presented in figure 18. For small systems sizes, the disease spans the entire system and reaches the corners. The maximal distance is then the distance from the center (where the Patient Zero is) to one corner: $d_{max} = \sqrt{2}N/2$. This distance normalised is thus $\sqrt{2}/2 \approx 0.7$, which is what is observed in the graph. For larger systems, however, the disease does not have the time or is not infectious enough to reach the borders. Thus a phase transition from a global expansion regime to a local expansion regime is observed around $N = 100$. This value represents the distance which a disease of infectiousness $\beta = 1$ struggles to reach in 200 weeks (the time of each simulation).

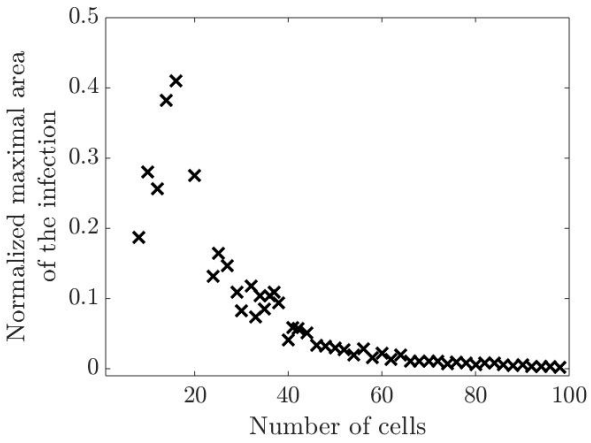


Figure 17: The normalised maximal infectious area has a peak for a size of $N \approx 18$ when $\beta = 1$.

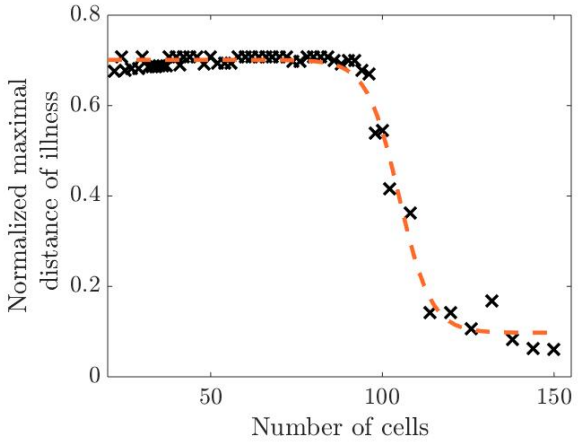


Figure 18: In the normalised maximal distance of the illness there is a phase transition at $N \approx 100$ when $\beta = 1$.

The last quantity of interest is the *prominence* of the normalised maximal distance of the illness, shown in figure 19. Prominence is defined as the height of the peak's summit above the lowest contour line encircling it but containing no higher summit. It is a measure of the independence of a summit [9]. This quantity presents the same phase transition as in figure 18, around $N = 100$. However, the difference between the two graphs is seen for smaller system sizes. Indeed, where figure 18 shows that all the systems with $N < 100$ have the same (normalised) maximal distance, figure 19 shows that the prominence of the maximal distance of infection decreases with the system size. This means that it is a much more common event for the disease to reach the corners of the system for very small systems than for slightly larger systems, which struggle to reach it even once. The prominence thus gives us much more information than the simple maximal distance graph.

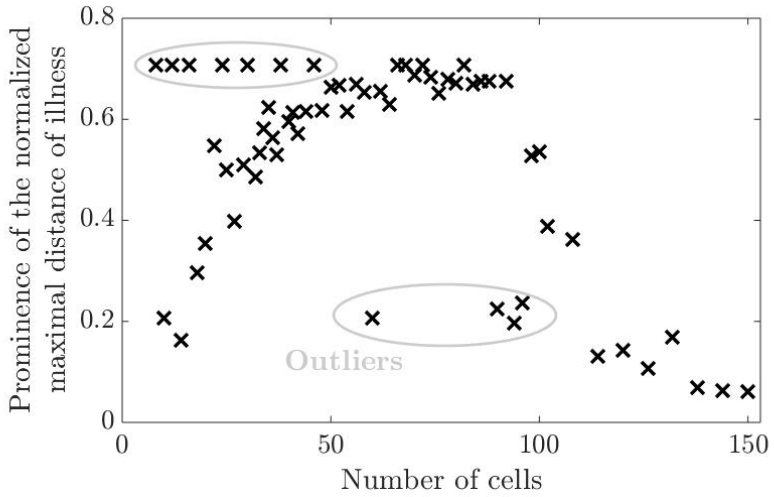


Figure 19: The prominence of the normalised distance of the illness presents too a phase transition around $N \approx 100$.

3.6 Average vaccination density for varying rw_{ill}

In this section, the system size is set to $40 \times 40 = 1600$ *cells.*

Figure 20 shows the evolution of the average vaccination density as a function of the rw_{ill} parameter. This graph was obtained by simulating a 2000 week (≈ 40 years) period for each rw_{ill} value, with a seasonal β. The average vaccination density is then computed on the last 1000 weeks to allow it to stabilise after the first, most impactful, outbreak.

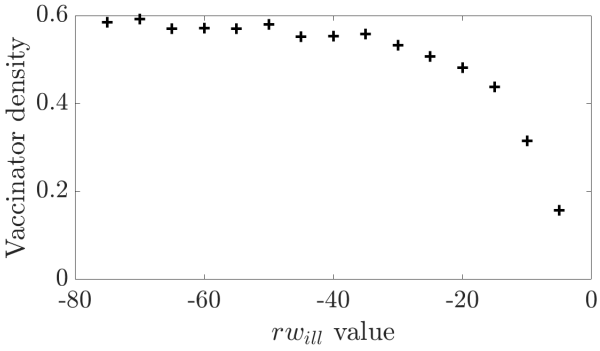


Figure 20: Average over last 1000 weeks of the simulation of the vaccinator density for varying rw_{ill} parameter

As expected, the figure shows that the vaccination density increases as the reward for falling sick decreases. This increase is sharp at first but slows down as rw_{ill} gets more and more negative. One possible explanation for this phenomenon is that, as the vaccination density increases, a point is reached where the disease can no longer spread effectively before the 'flu season' ends. The outbreaks are then contained to a small region around the Patient Zero, meaning that the general population no longer feels the need to vaccinate, despite there being a very negative reward for falling sick.

4 Possible improvements or elements of further research

The overall algorithm could be improved in many different ways. First of all the age dependency of the system could be improved, for example by adding a different β for different classes of age. Another improvement could be to add another type of mortality which is disease dependent simulating the weakness of an ill person. Also it could be interesting to have a time delay between the moment at which the person gets the infection and the moment he becomes infectious, i.e. an incubation time, as it is done in an SEIRS model with an additional state Exposed. One more interesting improvement could be the implementation of geographic obstacles into the system, for example a mountain or a lake through which the disease can not spread, or to change the form of the system itself (rectangles, circles, ...) in order to study the dependence of the disease on the topology.

5 Conclusion

This paper presents a model for simulating vaccination trends in an SIRS system. The first basic simulations confirm expected results, such as vaccination being an effective way to prevent the spreading of future diseases, which implies that the model works, at least on this basic level. Several more interesting results were then explicited.
Firstly, for a very localised disease origin, the number of vaccinators is needed to be very high to restrain the area of effect of a disease. However, if the disease manages to break out from the vaccinated population one year, it can spread very quickly to the whole system, in which case the non-vaccinated population suffers greatly compared to the vaccinated one (this explains the so called *herd immunity*).
Secondly, when varying various parameters, certain phase transitions were observed. Notably, when varying the infection rate β of the disease for a fixed system size, a phase transition is observed for $\beta = 1$, corresponding to the transition from a local disease to a global one (within the scope of the system size). When varying the system size N for a fixed β, the opposite phase transition was observed for $N = 100$, corresponding to a transition from global to local disease (once again within the scope of the system size).
Finally, we underlined the usefulness of prominence in extracting additional information from the maximal distance of the disease, which appeared constant when it was first plotted.
Overall, the model appears to work well and presents many interesting remarks, but could be improved upon, or complexified, in order to highlight more subtle social dynamics that are perhaps not present in the simulations as they stand now.

References

[1] *Ask the expert: Influenza Q&A*, World Health Organisation (WHO), 06.11.2018, Last consulted: 03.12.2019,
https://www.who.int/en/news-room/fact-sheets/detail/influenza-(seasonal)

[2] *Pyramide des Âges*, Federal Statistical Office (FSO), 30.08.2017,
Last consulted: 15.11.2019
https://www.bfs.admin.ch/bfs/en/home/statistics/catalogues-databases/graphs.assetdetail.3302625.html

[3] *Grippe saisonnière: foire aux questions*, Federal Public Health Office, 22.10.2018, Last consulted 13.11.2019,
https://www.infovac.ch/docs/public/influenza/faq-grippe-aw-faq-fach-fr.pdf.

[4] *Ask the Experts - Influenza*, Immunisation Action Coalition, 07.11.2019, Last consulted 03.12.2019, https://www.immunize.org/askexperts/experts_inf.asp

[5] *Décès selon le sexe, la nationalité, l'état civil et l'âge*, Federal Statistical Office, 12.09.2019, Last consulted 18.12.2019,
https://www.bfs.admin.ch/bfs/fr/home/statistiques/population/naissances-deces/deces.assetdetail.8966351.html

[6] Diagram drawn on the web site draw.io.

[7] Sheryl L. Chang, Mahendra Piraveenan, Philippa Pattison and Mikhail Prokopenko, *Game theoretic modelling of infectious disease dynamics and intervention methods: a mini-review*, 14.01.2019, https://arxiv.org/abs/1901.04143

[8] Fu Feng, Rosenbloom Daniel I., Wang Long and Nowak Martin A, *Imitation dynamics of vaccination behaviour on social networks*, 278 Proc. R. Soc. B,
http://doi.org/10.1098/rspb.2010.1107

[9] *Topographic prominence*, Wikipedia, 30.11.2019, Last consulted 02.12.2019,
https://en.wikipedia.org/wiki/Topographic_prominence

A Dynamic and static SIRS system

It can be interesting to investigate the effect of dynamic agents on the infectious density. Thus, the same simulations as in the first section are run switching displacement of agents on.

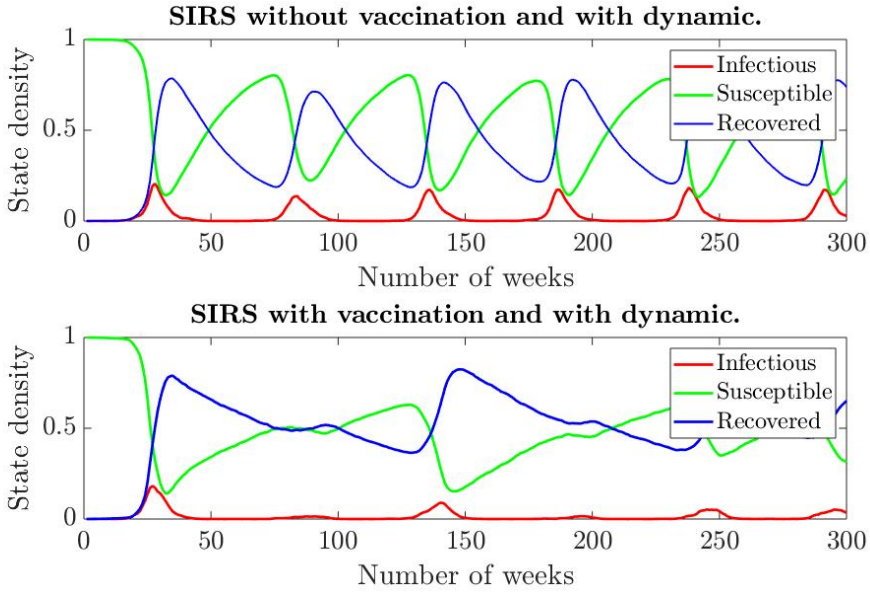


Figure 21: Two SIRS model: (*top*) without vaccination and with dynamic agents; (*bottom*) with vaccination and dynamic agents. Dynamic agents do not seem to perturb the evolution of the disease.

From figure 21 we see that the behaviour with dynamic is essentially the same as in the static case. The number of vaccinators (see figure 22) seems to decrease quicker in the case of dynamic agents, however the data are not sufficiently clear to give a final conclusion.

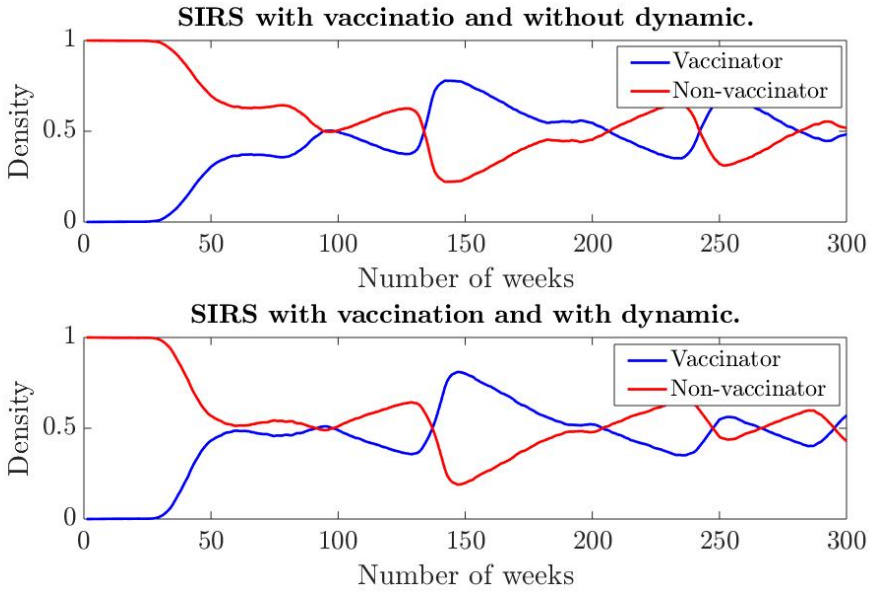


Figure 22: Densities of vaccinators and non-vaccinators in an SIRS model with vaccination and (*top*) without dynamic agents / (*bottom*) with dynamic agents. Dynamic agents seem to increase the gap between the densities of these two 'factions'.

ETH

Eidgenössische Technische Hochschule Zürich
Swiss Federal Institute of Technology Zurich

Declaration of originality

The signed declaration of originality is a component of every semester paper, Bachelor's thesis, Master's thesis and any other degree paper undertaken during the course of studies, including the respective electronic versions.

Lecturers may also require a declaration of originality for other written papers compiled for their courses.

I hereby confirm that I am the sole author of the written work here enclosed and that I have compiled it in my own words. Parts excepted are corrections of form and content by the supervisor.

Title of work (in block letters):

A STUDY ON THE EFFECT OF VACCINATION ON AN AGENT-BASED STOCHASTIC SIRS MODEL

Authored by (in block letters):

For papers written by groups the names of all authors are required.

Name(s):	**First name(s):**
DELMOTTE	NICHOLAS
PEDRELLI	LORIS
ROSKOV	IVAN

With my signature I confirm that

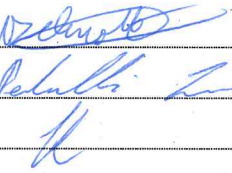
- I have committed none of the forms of plagiarism described in the 'Citation etiquette' information sheet.
- I have documented all methods, data and processes truthfully.
- I have not manipulated any data.
- I have mentioned all persons who were significant facilitators of the work.

I am aware that the work may be screened electronically for plagiarism.

Place, date

Zürich 06.12.2019

Signature(s)



For papers written by groups the names of all authors are required. Their signatures collectively guarantee the entire content of the written paper.