A performance-prediction model for spacecraft magnetometers

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6th of May, 2019

Abstract:
SIRS model was stochastically implemented together with a machine learning based mechanism in order to simulate the seasonal spreading of Influenza in a localised region and the effect of a vaccine on the number of infected people. In addition agents can be dynamic and move across the system. Susceptible, Infectious and Recovered densities of people in the system were calculated. Then many characteristic quantities of the system, such as propagation speed, maximal infectious area, maximal distance of the illness, were analysed when varying infectivity and size of the system. A phase transition was observed in the distance of the illness when varying the size of the system. a , b, and c were analysed in order to

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1 Introduction

The 11th of March 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.

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. Thus, each person is surrounded by eight neighbours with whom he can interact. Numerically, this grid of people is implemented as a 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.

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 individual 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 resumed 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 rates

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. However, in the following the plotted quantity will always be the vaccinator density.
The overall model, composed of the SIRS model and the reward-based vaccination system, can be summarised by the diagram in figure 1.
Numerically, as shown in figure 1, 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.

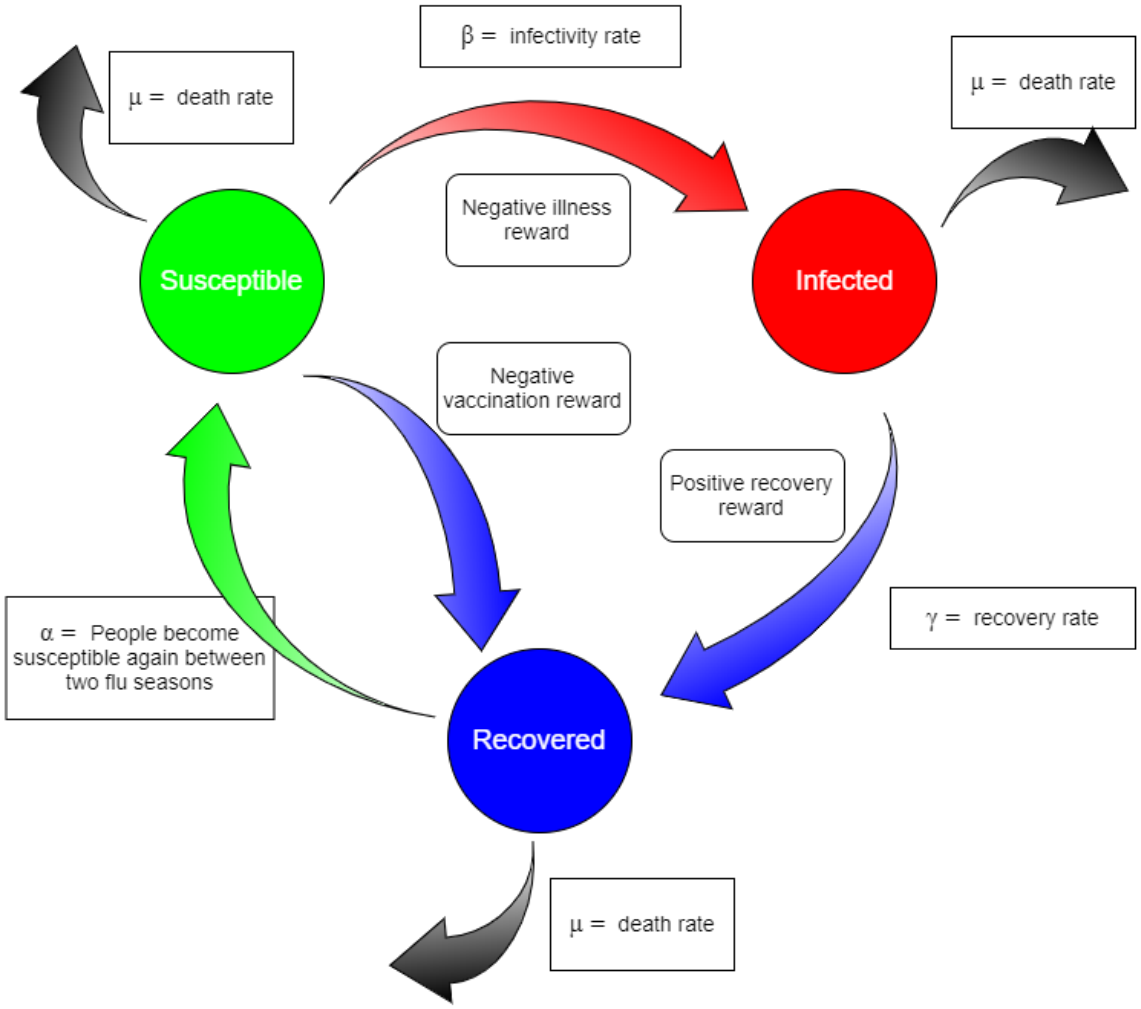

Figure 1: Diagram summarising all the interactions and the rewards in the system [6].

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 function of the form:

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

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 2.

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 group (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.

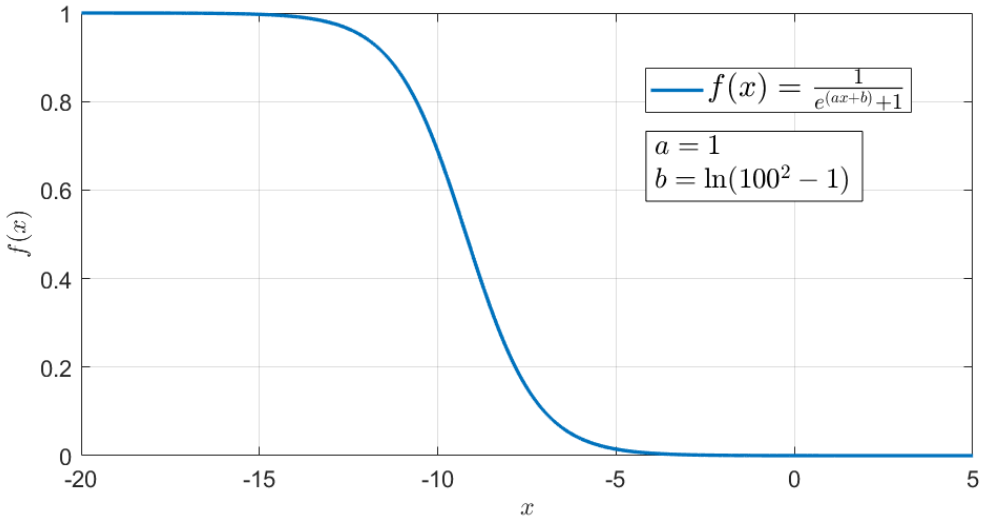

Figure 2: Probability distribution used in the vaccination process in the case of $N = 100$.

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. 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.

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 $\sim$ 300 weeks and the resulting densities of S, I and R in the system are given in figure 3 (*top*). The same simulation is then run but turning the vaccination mechanism on. The same quantities are given for this simulation in figure 3 (*bottom*).

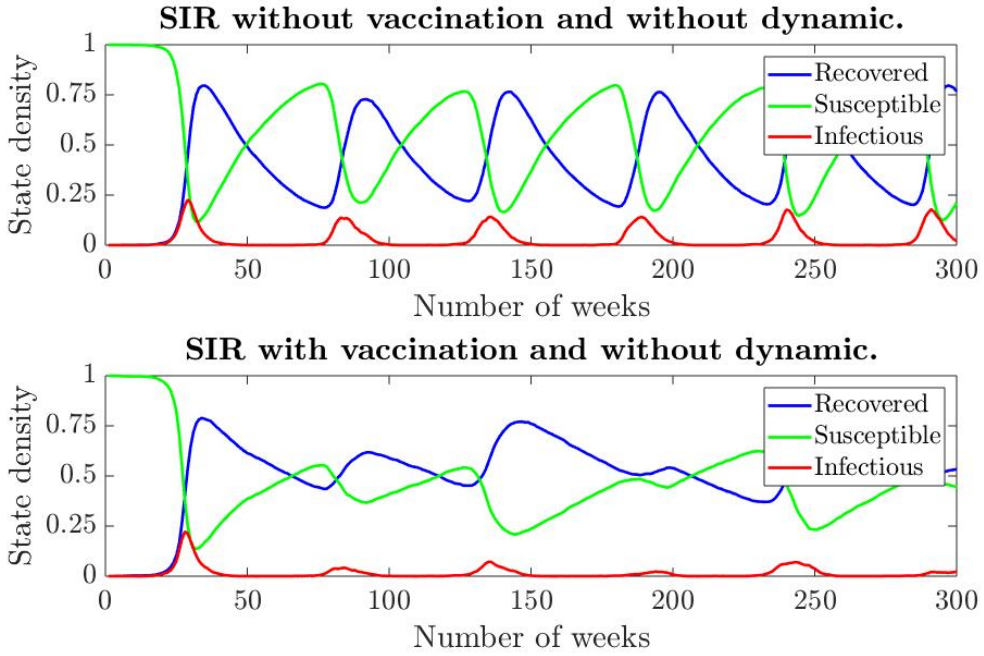

Figure 3: Two SIRS models: (*top*) without vaccination and with static agents; (*bottom*) with vaccination and with static agents. Periodic trend is caused by the use of a seasonal β rate. In the case with vaccination (*bottom*), the spreading of the illness is strongly damped.

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 Dynamic and static SIRS system

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

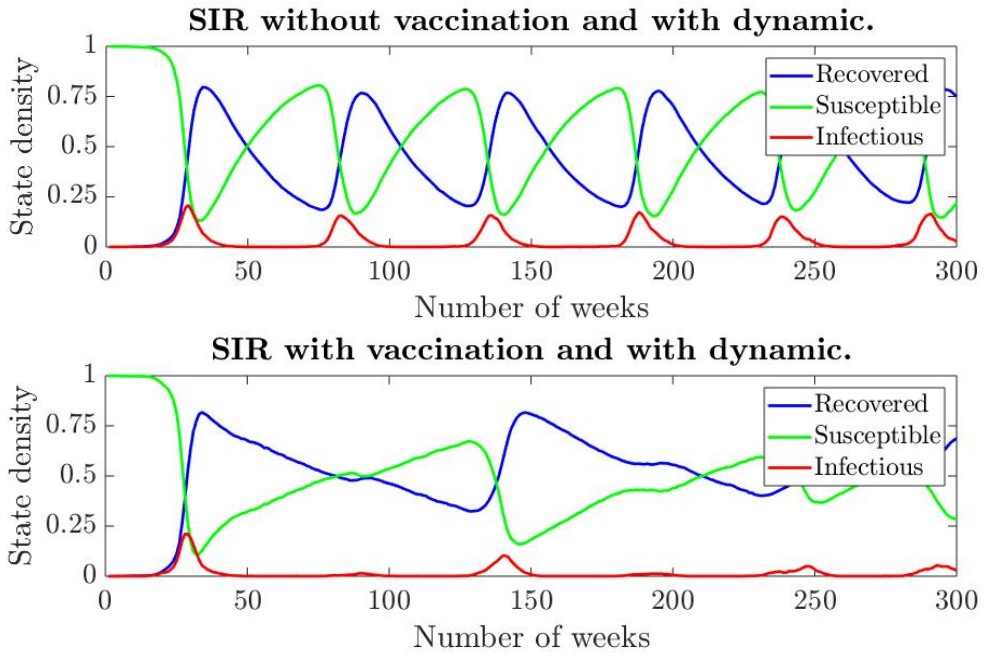

Figure 4: Two SIRS model: (*top*) without vaccination and with dynamic agents; (*bottom*) with vaccination and dynamic agents. Dynamic agents do not seem to perturb the evolution of the disease.

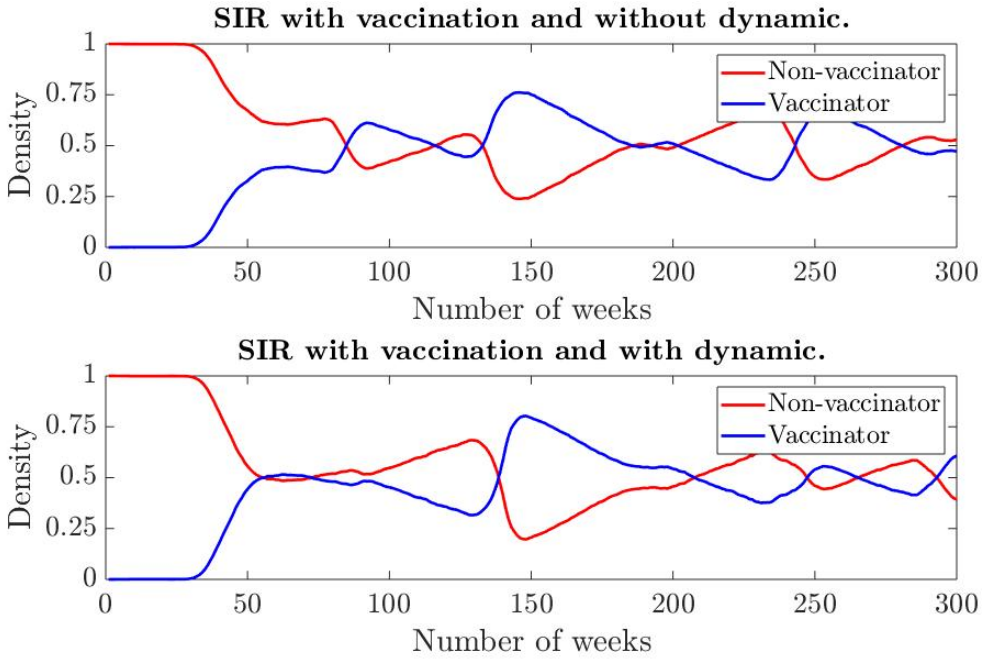

Figure 5: Densities of vaccinators and non-vaccinators in an SIRS model with vaccination and (*top*) without dynamic agents / (*bottom*) with dynamic agents. Dynamic agents increase the gap between the densities of these two groups.

Why does it seem that with displacement vaccination is not such a good idea ? Is it because we have a competition between the displacement and the vaccination?

However the data are not sufficiently clear to give a clear conclusion.
Indeed the data is not very useful in answering that question. We would need either different graphs or we can just cut out the vaccination density part and leave only the state density graphs.

3.3 Difference between fixed and varying beta

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 6 and 7 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.

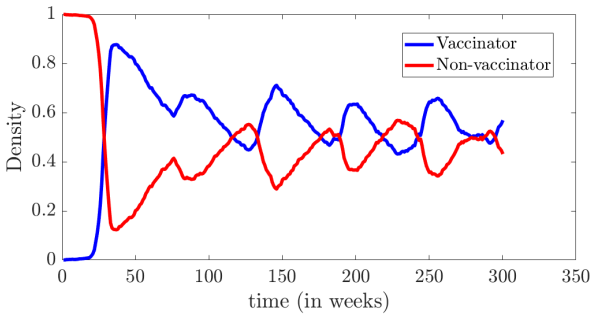

Figure 6: Vaccinator density for seasonal β

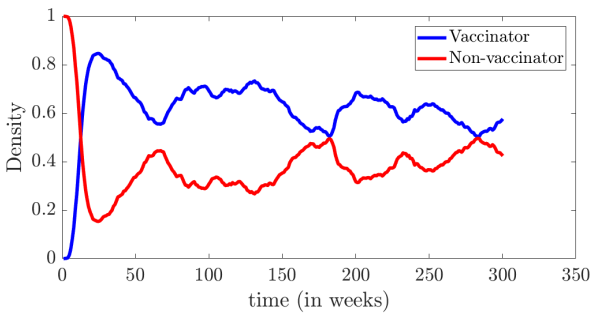

Figure 7: Vaccinator density for β fixed (approx.) at the peak of seasonal β

3.4 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 8 and 9 show the normal case of a randomly distributed patient zero. Figure 8 shows the yearly periodic variation of the disease, as expected, with each outbreak being more or less the same intensity. In figure 9, 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.

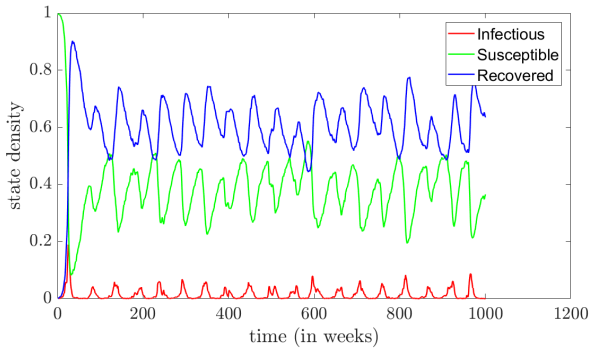

Figure 8: State density for normal random patient zero distribution.

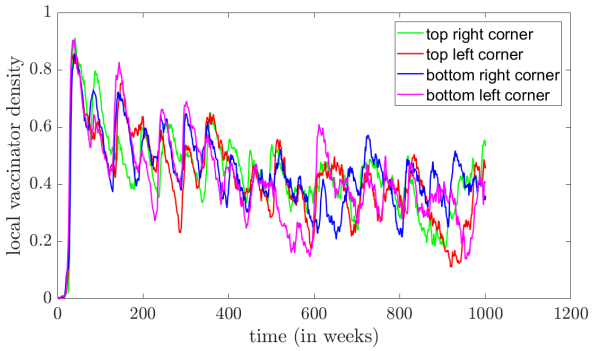

Figure 9: Local vaccination density for normal random patient zero distribution.

In figures 10 and 11, 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 10 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 11 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.

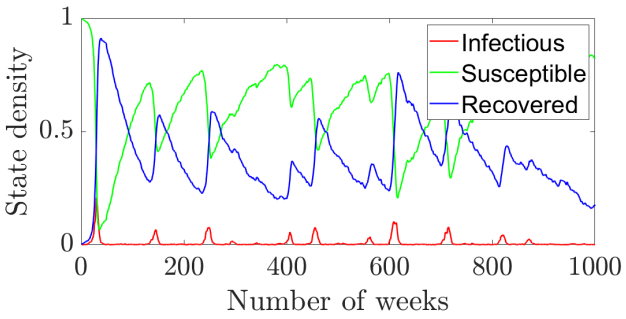

Figure 10: State density for patient zero in [1:10,1:10] range.

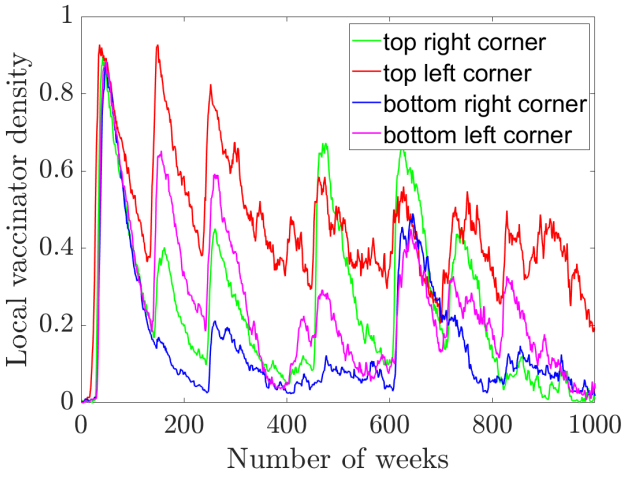

Figure 11: Local vaccination density for patient zero in [1:10,1:10] range.

In figures 12 and 13, the patient zero's position has now been fixed, again in the upper left corner (in cell [3,3] exactly). Figure 12 shows an even sparser distribution of outbreaks. Figure 13 shows the local vaccination density, with an added line representing the close neighbours of the patient zero (cells with x and y positions from 1 to 5 are considered close neighbours here). As expected, the close neighbours have the highest vaccination probability followed by the complete upper left square.

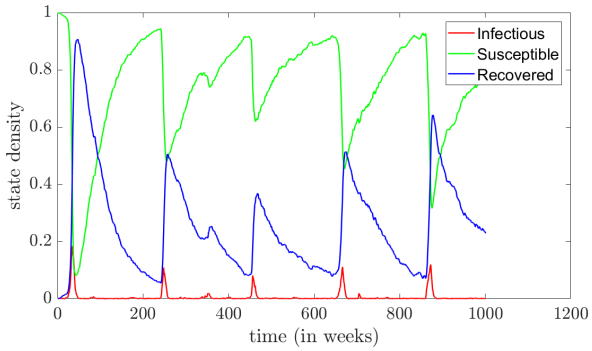

Figure 12: State density for patient zero at fixed [3,3] position.

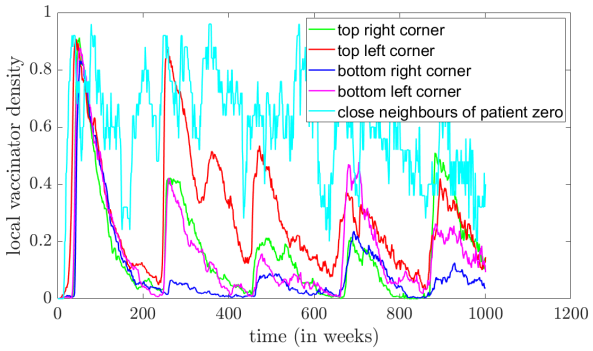

Figure 13: Local vaccination density for patient zero at fixed [3,3] position.

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 12 and 13 show that, for a fixed patient zero, outbreaks only depend on the 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 11 and 13, 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.

3.5 Variation of infectivity rate β

The next simulation consists in varying β from ?? to ?? (note that β is fixed at these values; it is not seasonal) and, for each value, letting the system evolve during 200 weeks. The infectious agent ratio is then plotted for each value of β in figure 14. 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 15 where the number of recovered agents is plotted for different values of β.

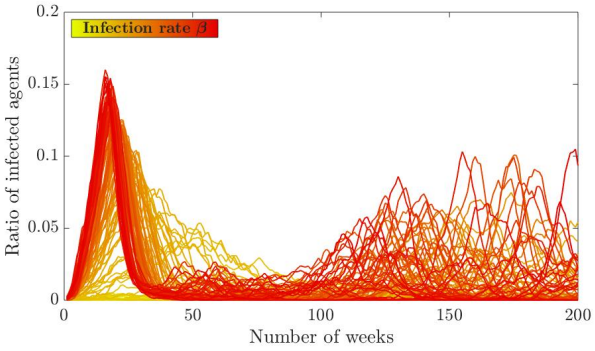

Figure 14: Infectious density plotted for each value of rate β. The peak of maximal infection moves to earlier times when β increases.

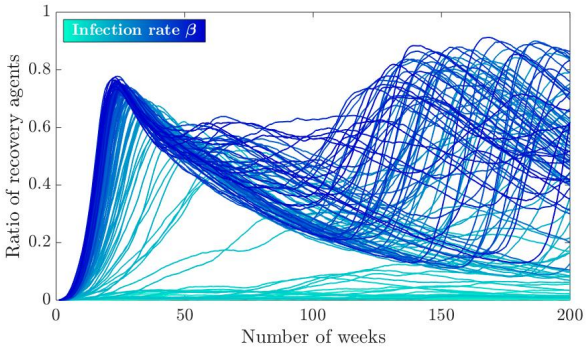

Figure 15: Recovered density plotted for each value of rate β. The peak of maximal recovery also moves to earlier times when β increases.

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 16. This quantity increases with β and seems to go through a phase transition around the value of 1. The last quantity computed under the variation of β is the propagation speed of the illness in the system defined as ???. The result is given in figure 17. This quantity increases as $\beta^{0.0135}$. The trend of these two last quantities allows us to conclude that, as expected, the peak of infection (and consequently of recovery) occurs earlier when the infectivity rate is larger, i.e. the spread of the illness is faster and. In addition the illness diffuses on a larger surface when β is larger.

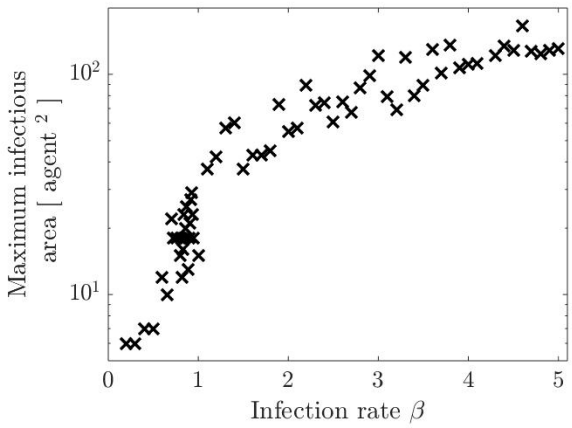

Figure 16: The maximum infectious area increases with β.

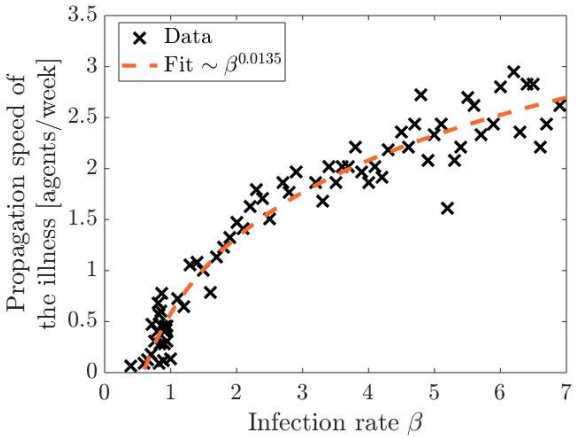

Figure 17: The propagation speed of the illness increases as $\beta^{0.0135}$.

3.6 Variation of system size N

Here the size of the system N (which is understood as the number of cells of one side of the system; the total number of cells in the system is thus N^2) is varied between ?? and ?? and for each value of N the system evolves during 200 weeks. Then, as before, infectious and recovered agent ratios are plotted for each value of N and are presented

in figures 18 and 19. Unlike before, the peak of maximal infection and maximal recovery move to the right as the dimension of the system increases.

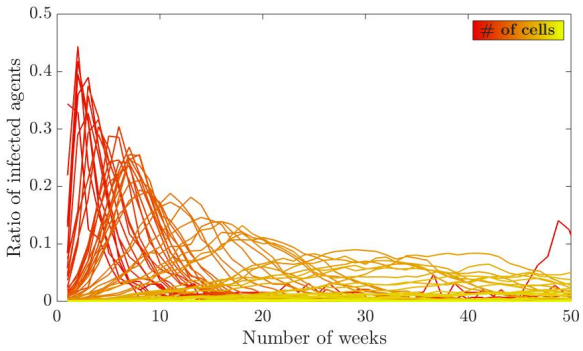

Figure 18: Infectious density plotted for many values of N. The peak of maximal infection moves to the right when N increases.

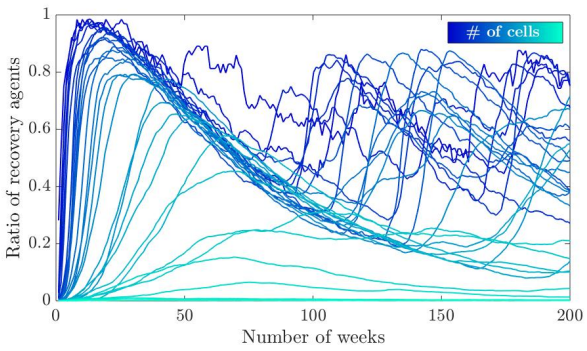

Figure 19: Recovered density plotted for many values of N. The peak of maximal recovery decreases and moves to the right when N increases.

This time the maximal area of infection needs to be normalised in order to compare the different obtained values which are presented in figure 20. The graph has a peak when N approaches 10, this is a resonance in the system.
Another interesting calculated quantity is the maximal distance of an infectious agent from Patient Zero (i.e. the person from which the spreading of the illness begun). This is calculated under variation of N and is presented in figure 21. It is clear from this graph that the system goes through a phase transition as N approaches the value of $N = 100$.
The last quantity we compute is the prominence of the normalised maximal distance of the illness. This quantity too seems to present a phase transition for a value of $N \approx 100$.

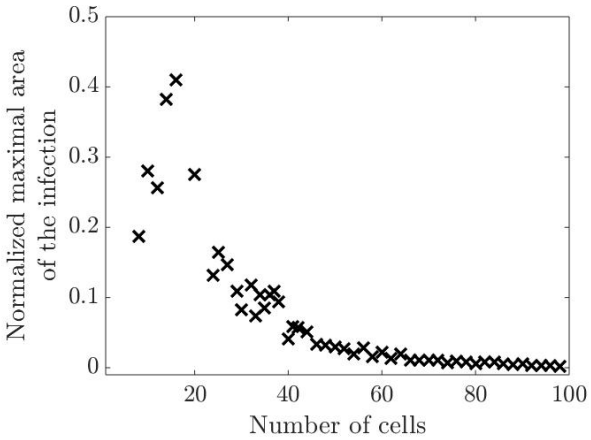

Figure 20: The normalised maximal infectious area has a resonance for a size of $N \approx 18$ when $\beta =???$.

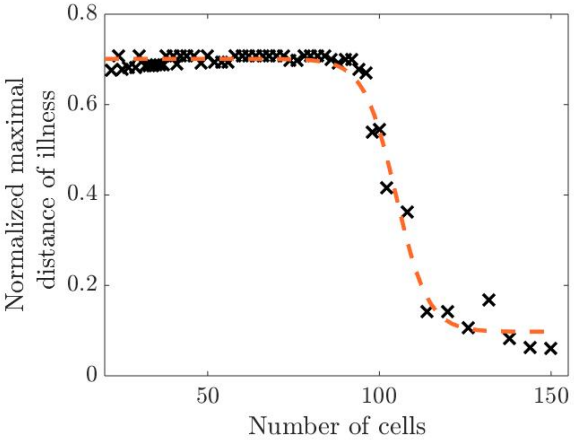

Figure 21: In the normalised maximal distance of the illness there is a phase transition at $N \approx 100$ when $\beta =???$.

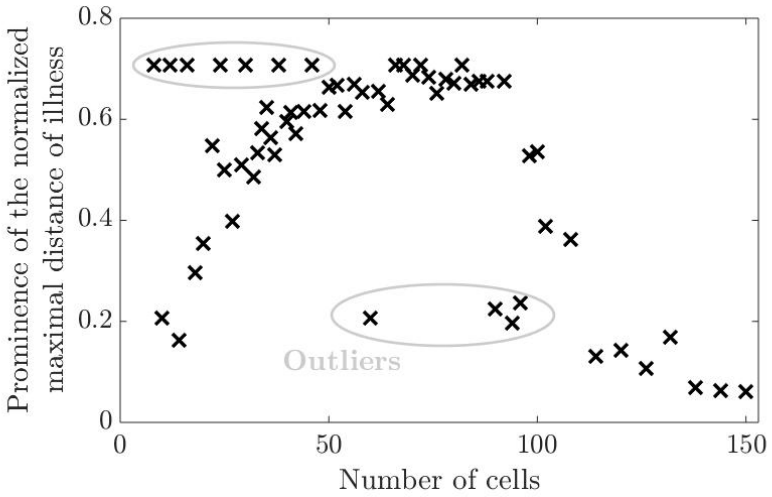

Figure 22: The prominence of the normalised distance of the illness presents too a phase transition around $N \approx 100$.

3.7 Average vaccination density for varying rw_{ill}

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

Figure 23 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.

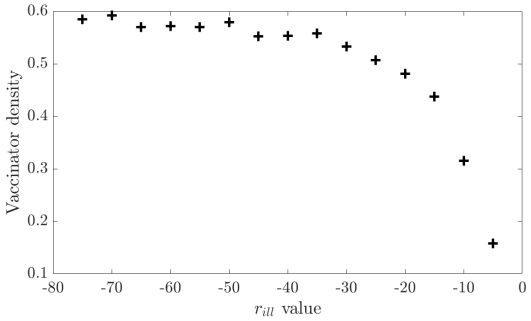

Figure 23: Average over last 1000 weeks of the simulation of the vaccinator density for varying rw_{ill} parameter

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 Conclusion

-several basic simulations confirm expected results, which implies our model works (at least on a basic level)
-vaccine is effectively a good way to prevent a future spreading of an illness
-for a very localised disease origin, the number of vaccinators is not needed to be very

high, but if the disease is particularly bad one year and breaks out from the vaccinated population, the non-vaccinated population suffers greatly compared with the vaccinated one

-

5 Possible improvements

different algorithm for the evolution of the grid: -count all rates on the whole grid (depending on each state) -make probabilities for each evenement -let the evenement happen and finally choose randomly the case (depending on the state) in which happens -increase and improve the age dependancy -alpha which depends on the time -delay dans la maladie (temps de incubation)

Different topology + obstacles

Acknowledgements

References

[1] Information taken from the official site of the *World Health Organisation (WHO)*: https://www.who.int/en/news-room/fact-sheets/detail/influenza-(seasonal) Last consulted 03.12.2019

[2] Pyramide des Âges (30.08.2017 version). Data taken from the Federal Statistical Office (FSO) website: https://www.bfs.admin.ch/bfs/en/home/statistics/catalogues-databases/graphs.assetdetail.3302625.html. Last consulted 03.12.2019

[3] Main questions about the seasonal influenza (22.10.2018 version). Data taken from the Federal Statistical Office (FSO) website: https://www.infovac.ch/docs/public/influenza/faq-grippe-aw-faq-fach-fr.pdf. Last consulted 03.12.2019

[4] Vaccine validity duration. Data taken from Immunisation Action Coalition website: https://www.immunize.org/askexperts/experts_inf.asp. Last consulted 03.12.2019

[5] Number of death (12.09.2019 version). Data taken from the Federal Statistical Office (FSO) website: https://www.bfs.admin.ch/bfs/fr/home/statistiques/population/naissances-deces/deces.assetdetail.8966351.html. Last consulted 03.12.2019

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