

Disease Models as Cellular Automata

IDC 621 Modelling Complex Systems

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Introduction

Cellular Automata are mathematical models with the aim of simulating complex natural phenomenon, containing a large number of simple components which interact locally to collectively give rise to complex behaviour. They consist of discrete lattice sites, each with a finite set of possible integer values, which evolve in discrete time steps according to the “rules” of the cellular automata. The CA lattice can be N -dimensional, depending upon the requirements of the model.

We will particularly focus on the *SIRS model* for disease spread as our 2-D cellular automata here, with minor modifications made to it with an attempt to study how an epidemic spreads, and how different measures to reduce the spread can be implemented.

1 SIRS Model

The SIRS model is a 2-D cellular automata. The SIR in the name stand for the different states of the cells which represent individuals - Susceptible, Infected and Referactory, after which the cell returns back to the state Susceptible. The states of the CA take on finite values, and are updated according to the local update rule in discrete time.

1.1 Parameters and the Update rule

The length of the SIRS cycle is given by the parameter $\tau_0 = \tau_I + \tau_R$, where τ_I and τ_R are the lengths of the Infected and Referactory stages respectively. Each cell (i, j) is assigned a value $\tau_{ij}(t)$ which is updated over discrete time t . The variable $\tau_{ij}(t)$ can take integer values between 0 and τ_0 .

The different stages of the cells are described as follows-

- **Susceptible** $\tau_{ij} = 0$
- **Infected** $1 \leq \tau_{ij} \leq \tau_I$
- **Refractory** $\tau_{I+1} \leq \tau_{ij} \leq \tau_0$

The equations governing the dynamics at the nodes are-

$$\begin{aligned} \tau_{ij}(t+1) &= \begin{cases} 1 & \text{with probability } p \\ 0 & \text{with probability } 1-p \end{cases} & \text{if } \tau_{ij}(t) = 0 \\ \tau_{ij}(t+1) &= \tau_{ij}(t) + 1 & \text{if } 1 \leq \tau_{ij}(t) \leq \tau_0 - 1 \\ \tau_{ij}(t+1) &= 0 & \text{if } \tau_{ij}(t) = \tau_0 \end{aligned}$$

where $p = N_{\text{inf}}/N_{\text{total}}$, N being the number of neighbours. The infection rule, usually, is chosen to be stochastic, but can also be simplified so as to infect the cell even if a single neighbour is infected.

1.2 Modification to the SIRS Model

In this term paper, we will try to explore the spread of infection in an epidemic by introducing various parameters as control variables, and by adding another state to the model.

- **Quarantine:** We add a new Quarantine state ($\tau = -1$), where the cell permanently stays after it is isolated to prevent further spread. But since identifying each and every infection and sending people to quarantine is not a realistic possibility, we add a parameter \mathbf{P}_{quar} varying between 0 and 1 which is the probability of a particular infected cell to go into the Q state.
- **Identification Time:** Since identification of infectious cases and sending them into isolation requires time, we add another parameter \mathbf{M} which is the number of days after which people are sent into quarantine. This can be thought of as the testing time.
- **Lockdown efficiency:** We also add a lockdown bound of 2000 which is the limit of total infections (including quarantined) after which a 20 day lockdown is initiated. Since a perfect lockdown is an idealisation not possible in the real world, we add a parameter e as the lockdown efficiency. We allow the radius of the spread to decrease from $r = 3$ to $r = 1$ during the lockdown, with $e = 1$ implying a perfect lockdown (equivalent to $r = 0$), and $e = 0$ implying that the radius of spread is $r = 1$.
- For our present model, we will primarily consider fixed BCs and Moore neighbourhoods with weights attached to them such that if the cell of interest is at (i, j) , and the neighbour is at $(i \pm a, j \pm b)$ with $0 \leq a, b \leq r$, then the infection weight of that neighbour is $\omega_{ab} = r - \max(|a|, |b|) + 1$.

Thus, the total sum of all the weights for radius r is

$$\Omega(r) = \sum_{i=1}^r (8i)(r - (i - 1)) .$$

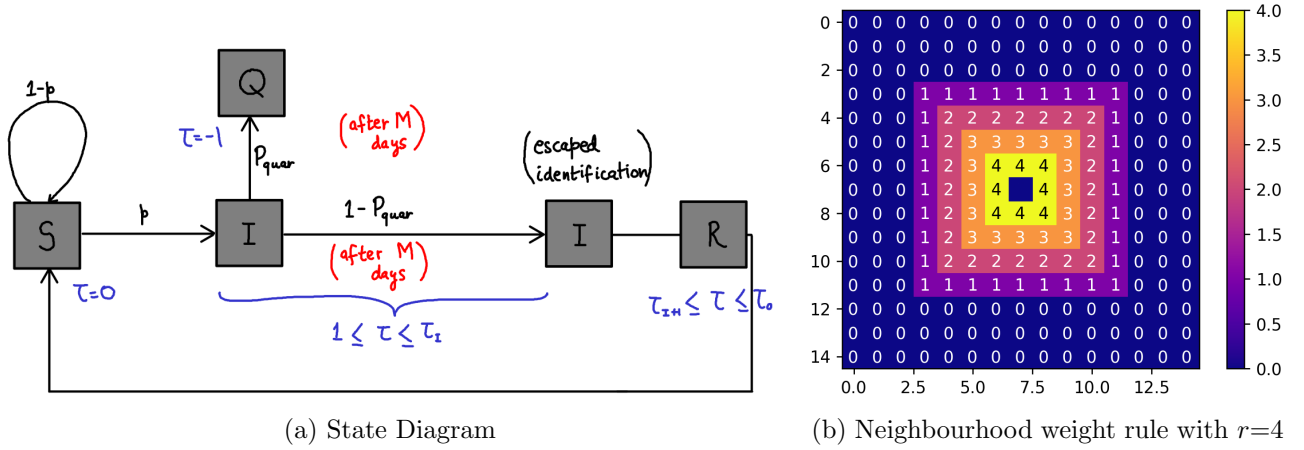


Figure 1: Modified SIRS model

2 Analysis and Results from the model

Although our model is by no means close to simulating real world epidemics, we will try to get an idea of what parameters could possibly affect the spread of infection the most, and what could be the possible ways if reducing the rate of spread of infection. Our primary aim is to analyze what could help us in reducing the *total number of infections* which is a good enough representative of the “spread of infection” in some given time for which our simulations are running.

The fixed parameters in our simulations would be the time of simulation $t = 100$, the grid size of 100×100 , $\tau_I = 4$, $\tau_R = 7$, the lockdown period of **20** days initiated once the total infections reach **2000**, and fixed boundaries at $\tau_I + 1$. The initial conditions also remain the same with a single infected seed ($\tau = 1$) in the middle and the rest of the cells as susceptible ($\tau = 0$).

We will use the following values of the parameters in our simulation runs-

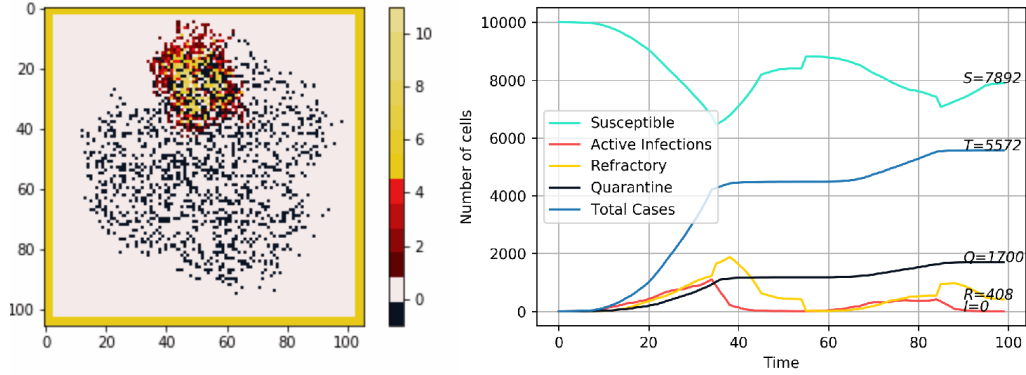
- P_{quar} (probability of quarantine) $\in \{0.25, 0.5, 0.75\}$
- M (number of days for isolation) $\in \{1, 3\}$
- e (efficiency of lockdown) $\in \{0.25, 0.5, 0.75\}$

We don't use the either extremes of P_{quar} and e because they lead to predictable results, which are sometimes overly idealistic. We will sort our results by holding e constant and varying the other parameters.

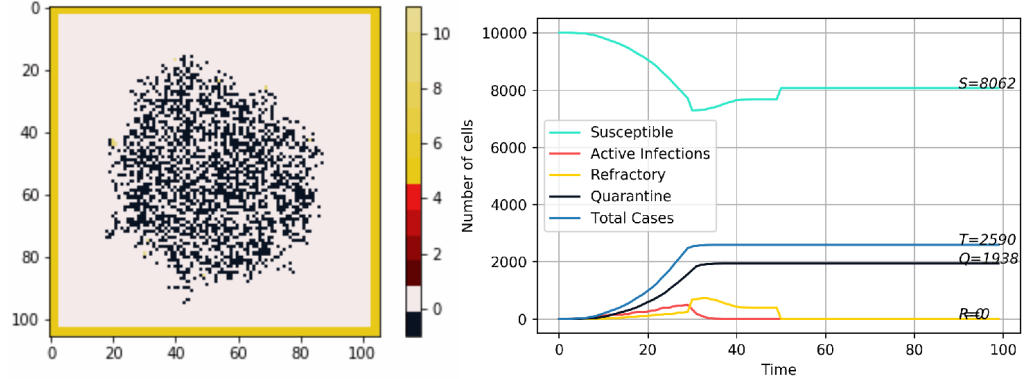
2.1 Results for $e = 0.75$

We will start with a relatively high efficiency of lockdown, which should mean that as soon as the lockdown is imposed, the infection should mostly die out. We will increase the fraction P_{quar} eventually and see how it affects the spread.

- $P_{\text{quar}}=0.25$: Even when only 25% of infections are quarantined, the high efficiency of the lockdown helps in almost clearing out the infections once the lockdown sets in, for both $M = 1$ and $M = 3$. The lockdown helps in slowing down the spread, but once in a few simulations, one or two infections remain which can act as a seed for a further wave, though it doesn't last long.
- $P_{\text{quar}}=0.5$ and 0.75 : The effects of a higher quarantine rates are, as expected, to almost always get rid of the infection wave once the lockdown sets in. The high value of P_{quar} helps the quarantine wave to catch up with the infection wave. The infection usually dies out faster when $M = 1$ compared to $M = 3$, but the difference is negligible.



(a) 2nd wave in $P_{\text{quar}}=0.25$, $M = 1$ ($t=65$)



(b) Elimination of infection $P_{\text{quar}}=0.5$, $M = 3$ ($t=60$)

Figure 2: Summary of results for $e = 0.75$

2.2 Results for $e = 0.5$

We now obtain the results with a lower value of lockdown efficiency. Intuitively, we expect that the lockdown would be “leaky”, and the infection spread would be higher.

- $P_{\text{quar}}=0.25$: When the lockdown efficiency is low at 50% and the $P_{\text{quar}}=0.25$, the effect of difference in $M = 1$ and 3 is negligible, and the leakiness of the lockdown is apparent since the

low quarantine rate cannot match with the infection wave, and the lockdown creates a lot of different seeds which proliferate the infection spread further unless imposed for a longer time.

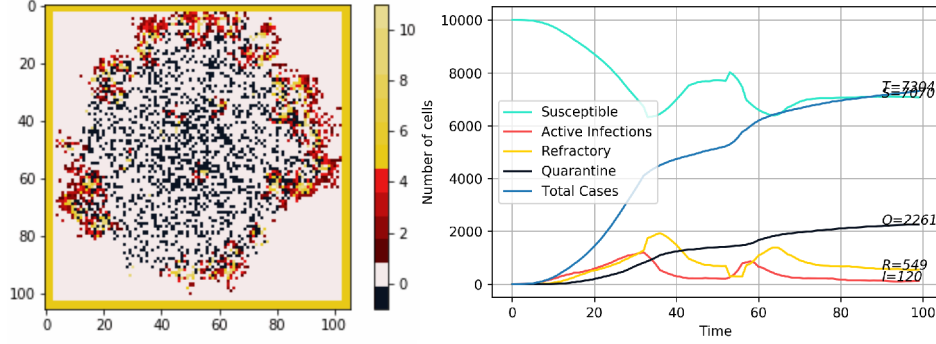
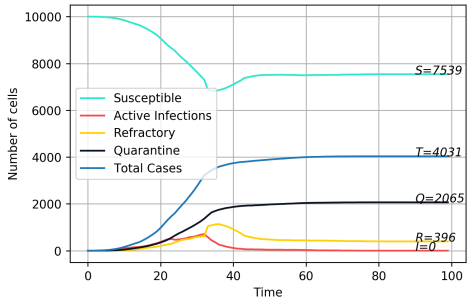
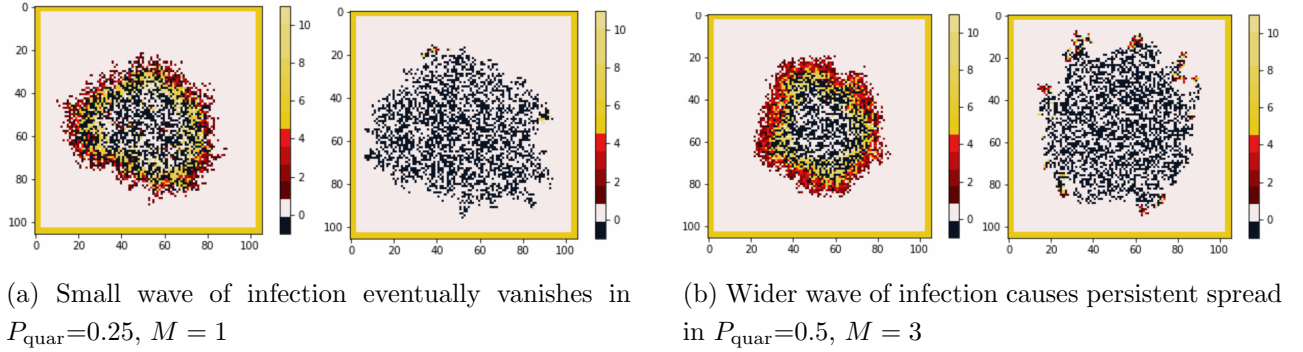
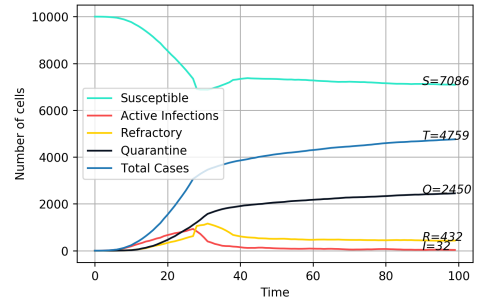


Figure 3: Bursts of infection after lockdown for $P_{\text{quar}}=0.25$ ($t=55$)

- $P_{\text{quar}}=0.50$: In this case, the infection wave is eventually caught by the quarantine wave once the lockdown sets in when $M = 1$. For $M = 3$, the lag due to the days between infection and quarantine doesn't completely halt the infection spread, and albeit slow, it does continue. The $M = 3$ simulation had a much wider infection wave compared to $M = 1$ which wasn't suppressed by quarantine, but the spread was considerably slow.



(c) Plot for $P_{\text{quar}}=0.5$, $M = 1$



(d) Plot for $P_{\text{quar}}=0.5$, $M = 3$

Figure 4: Results for $P_{\text{quar}}=0.5$

- $P_{\text{quar}}=0.75$: Very similar to the $P_{\text{quar}} = 0.5$ case, the infection wave is wider in $M = 3$ compared to $M = 1$, and although the number of infected people are very low eventually, the infection does not die. A major takeaway is that the infection does not completely die away even if the quarantine rate P_{quar} is high, and the number of days M required for isolation plays a very important role in the spread.

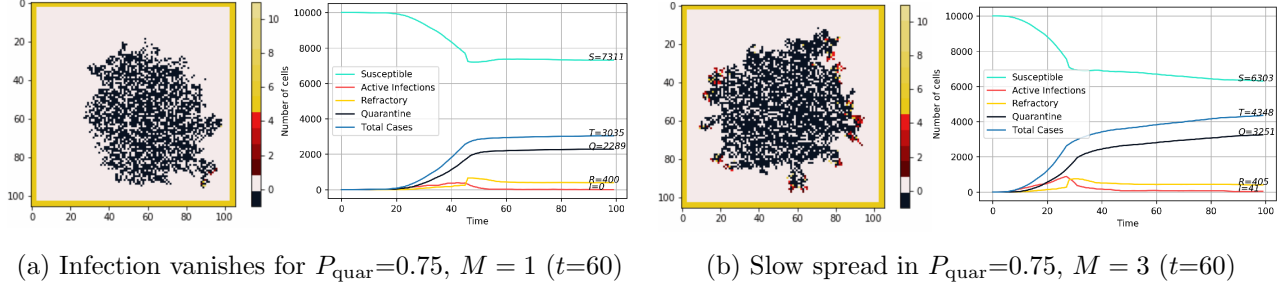


Figure 5: Infection still persisting despite high value of P_{quar}

2.3 Results for $e=0.25$

Now we'll discuss the results for a population where the people are not at all stringent about the lockdown rules, and we'll see if it really does make a difference if even strict quarantine measures imposed do any good.

- $P_{\text{quar}} = 0.25$: This is the weakest possible lockdown plus quarantine case, where the quarantine percentage as well as lockdown efficiency are particularly low, both of which compound on top of each other to create a devastating infection spread. The difference in $M = 1$ and $M = 3$ is negligible, but $M = 3$ does infect a higher percentage of the population.

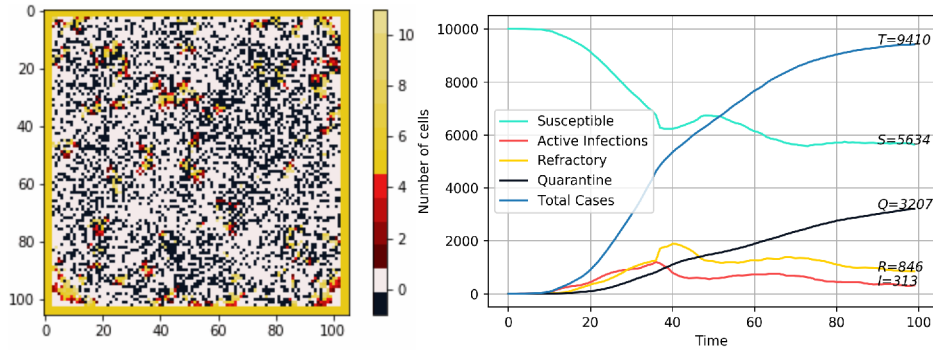


Figure 6: High spread of infection due to low P_{quar} and low e for $M = 1$ ($t=50$)

- $P_{\text{quar}} = 0.5$: Increasing the quarantine rate does decrease the infection rate by some amount, but the lockdown imposition isn't enough to contain it, both at $M = 1$ and $M = 3$. The images below show the spread of the infection.

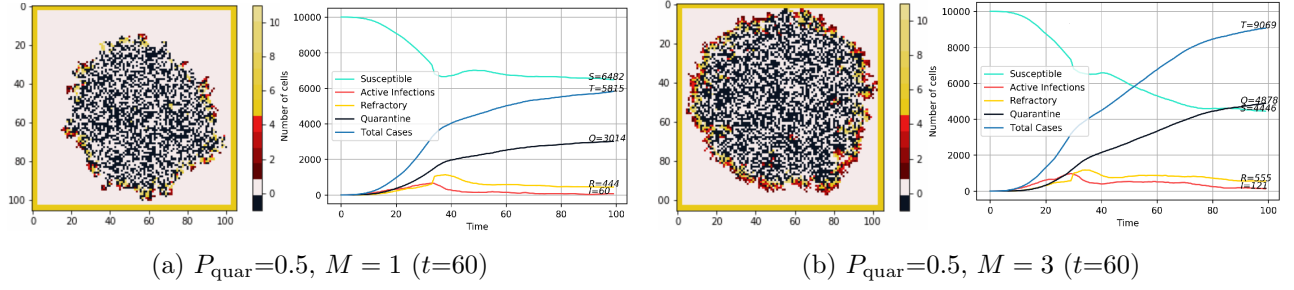


Figure 7: Infection persists for $P_{\text{quar}} = 0.5$

- $P_{\text{quar}} = 0.75$: Just like we noted in the case when $e = 0.5$, the infection barely ends for the $M = 1$ case (owing to the boundaries), whereas the increase in number of days to isolate i.e. for $M = 3$ allows the infection to spread rapidly, and infects most of the population despite the accurate identification. This leads us to another important conclusion. The percentage of quarantine/identification of cases can not always counter act the spread of infection when lockdown rules are not stringent. In addition, as the infection spread increases, the parameter M becomes more and more important.

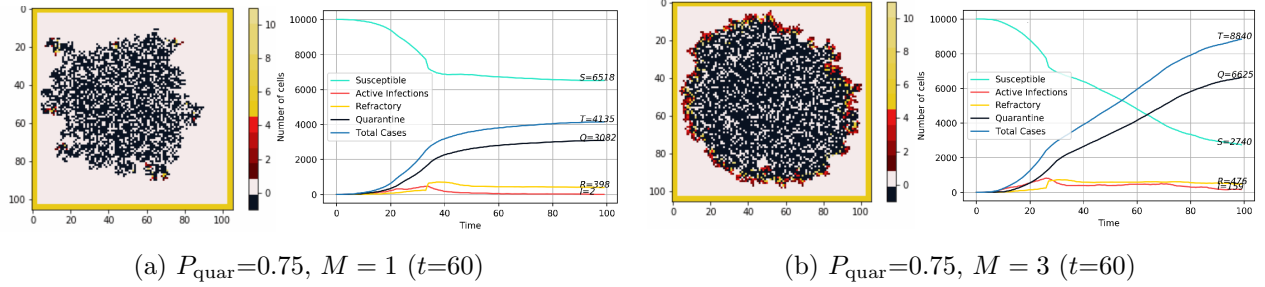


Figure 8: Despite high $P_{\text{quar}} = 0.75$, the infection spread is still high

3 Conclusions

As mentioned earlier, this is by no means a rigorous analysis of an epidemic spread. Through this short project, we aimed to generate a rough idea of what are the major contributing factors in an epidemic spread, considering the different parameters we introduced.

From our analysis, we were able to draw the following conclusions-

1. At low values of e , when the lockdown rules are being adhered to, even small and slow identification of cases (low values of M and P_{quar}) could help slow down the spread of the infection, although at really low levels of quarantine, as seen for $P_{\text{quar}} = 0.25$, some remaining infections after the lockdown can act as seeds for proliferating further infections.

2. As the population becomes less stringent about the lockdown rules, the role of parameter M , i.e. early identification of cases and sending them to quarantine plays a big role in ending the epidemic sooner.
3. Surprisingly, having a higher value of P_{quar} i.e. the percentage of infected people that are quarantined (can be thought of as true positives detected during testing) doesn't guarantee a faster end to the epidemic if the population isn't stringent about the lockdown rules. Again, as mentioned in the above point, the difference caused by different M values is significant.

Thus, from our rough analysis, we can understand that in case a large fraction of the population are essential workers, the lockdown efficiency would remain moderately low, and in such a scenario, accurate and reliable tests are indeed important, but identifying true positives sooner is also a high priority, because higher accuracy and slower identification can still lead to high spread of infection, hence an optimization between reliability and speed of testing is necessary.

Code

The code for this project was written in Python 3.7.6 and can be found on the following GitHub repository: <https://github.com/kaizokugarizoro/ComplexSystems>.