Uppsala University

Modelling Complex Systems

Master's Programme in Engineering Physics

Lab 1: Automata

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Contents

1 Introduction

In the present report, the results from Lab 1: Automata are presented and discussed. The lab is centered around cellular automata, discrete computation models. A simple set of rules govern the behaviour of individual cells, which interactions give rise to complex system behaviours.

2 A firing brain

2.1

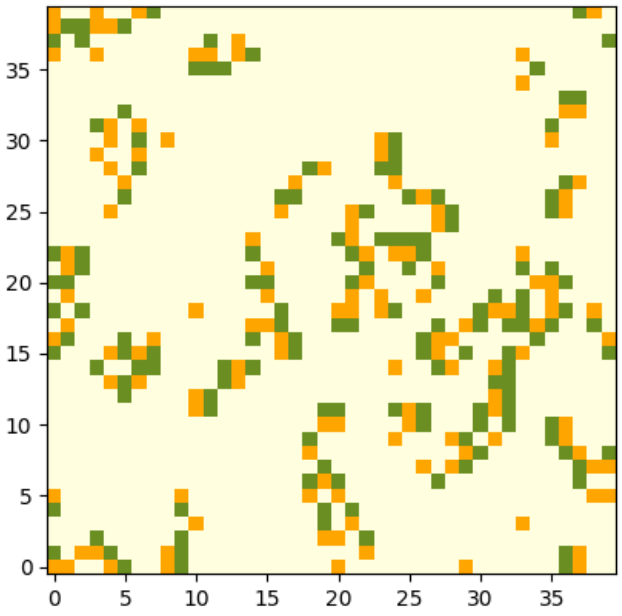

Figure 1: Plot of how the cells typically look after 10 time steps. Green indicates a firing cell, Yellow indicates a resting cell, and the ready cells are represented by the off-white background.

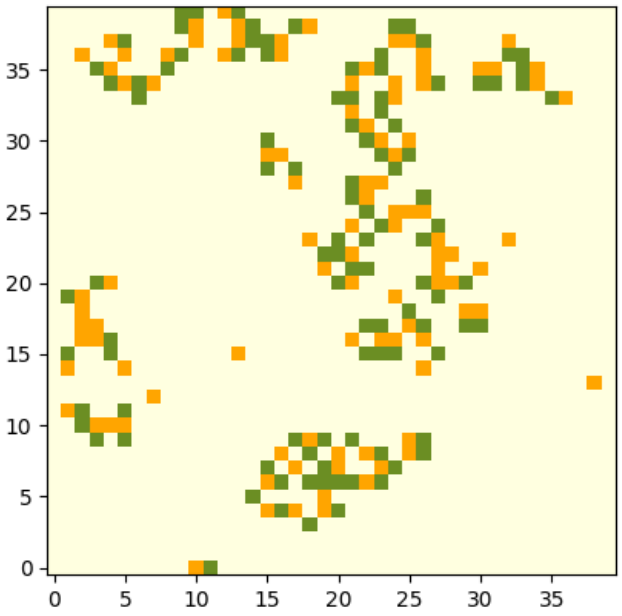

Figure 2: Plot of how the cells typically look after 20 time steps. Green indicates a firing cell, Yellow indicates a resting cell, and the ready cells are represented by the off-white background

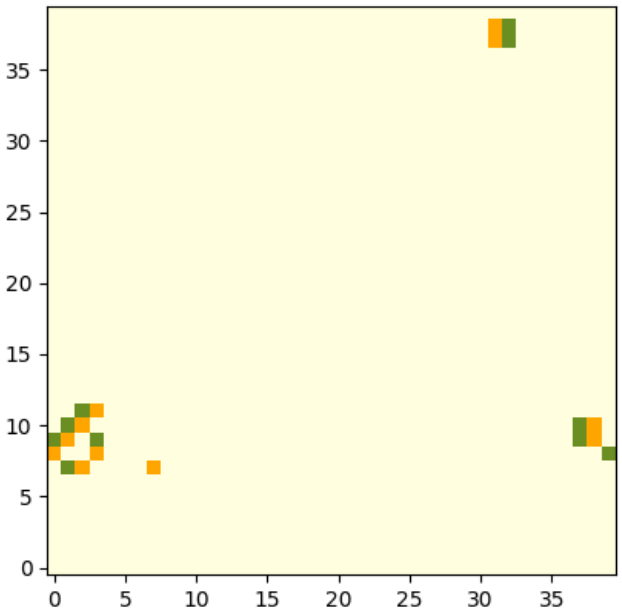

Figure 3: Plot of how the cells typically look after 100 time steps. Green indicates a firing cell, Yellow indicates a resting cell, and the ready cells are represented by the off-white background

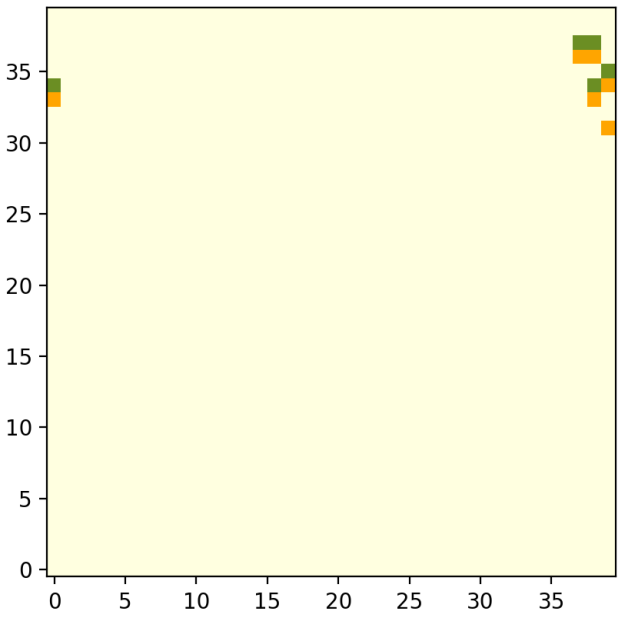

Figure 4: Plot of how the cells typically look after 1000 time steps. Green indicates a firing cell, Yellow indicates a resting cell, and the ready cells are represented by the off-white background

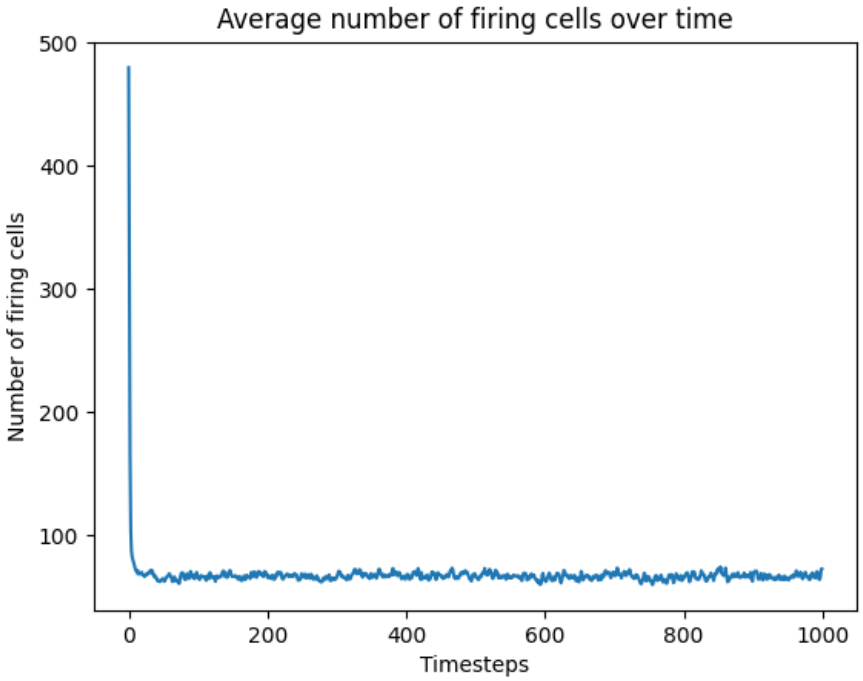

Figure 5: Average number of firing cells, calculated from 100 runs, as a function of the number of time steps.

2.2

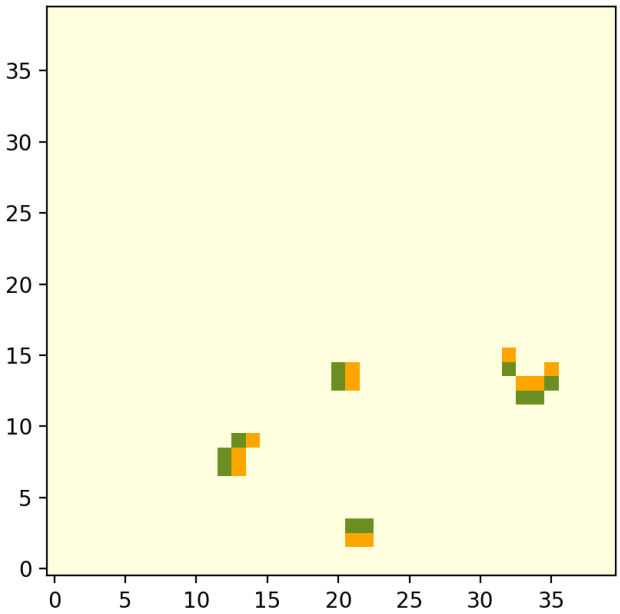

Figure 6: (a) Examples of shapes that move forward at rate of one cell per time step, while preserving the same shape.

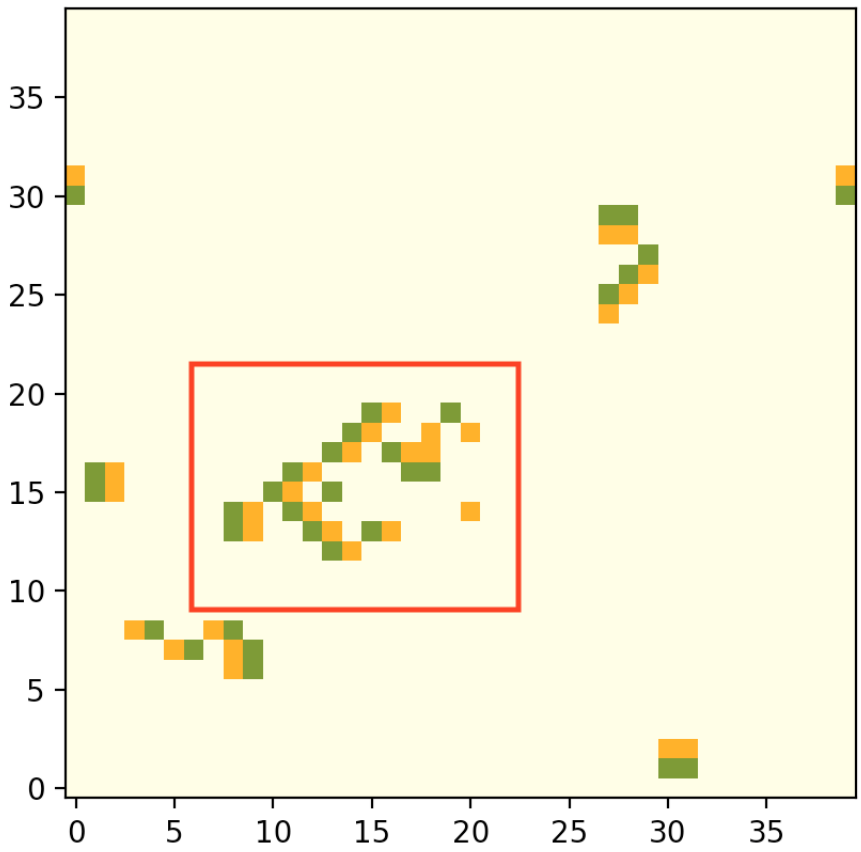

Figure 7: (b) Example of a shape that moves forward at a rate of one cell per time step, launching other shapes behind it. The shape in question is highlighted and is moving to the left.

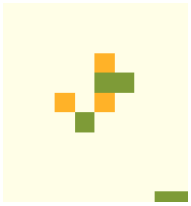

Figure 8: (c) Example of shape that moves forward at rate of less than one cell per time step, while returning to the same shape after some period.

(d) Despite a relatively large number of runs, no example of a shape that stays stationary but oscillates periodically was found. This might be simple unfavourable luck of the draw, as initial conditions were randomized. It is plausible that the reason for this lack is that the rules are very conducive to movement. This hypothesis might be bolstered by the fact that it is not entirely clear that a simulation of firing neurons should have these stationary oscillations.

3 Spatial Epidemics

3.1

A video of the 1D epidemic for different recovery rates γ can be seen via the link below:
LINK TO YOUTUBE VIDEO.

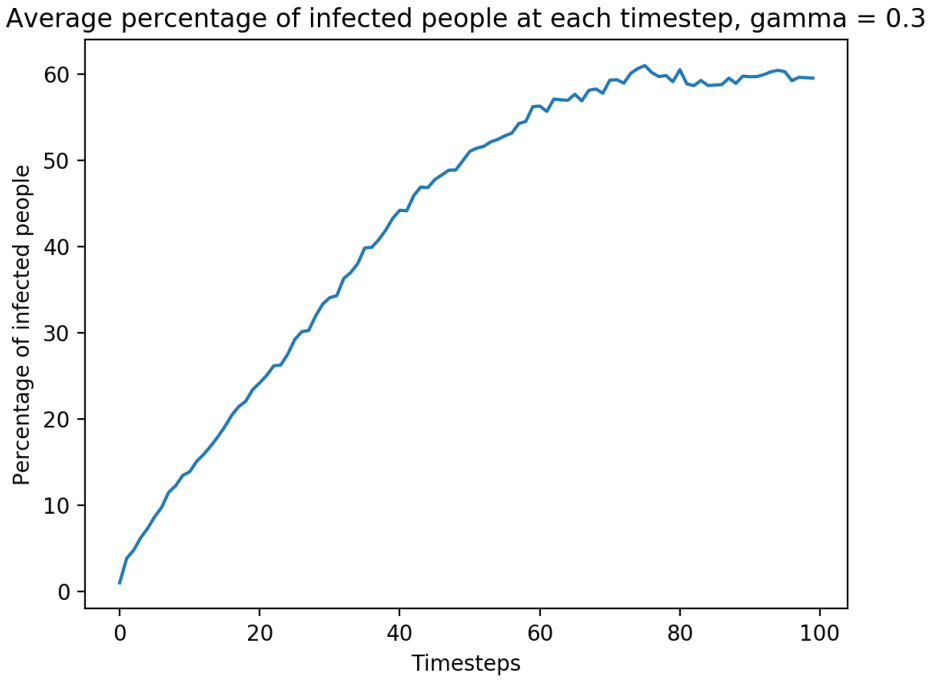

Figure 9: The percentage of infected people at each time step for $\gamma = 0.3$. The value at each time step is an average of 100 runs.

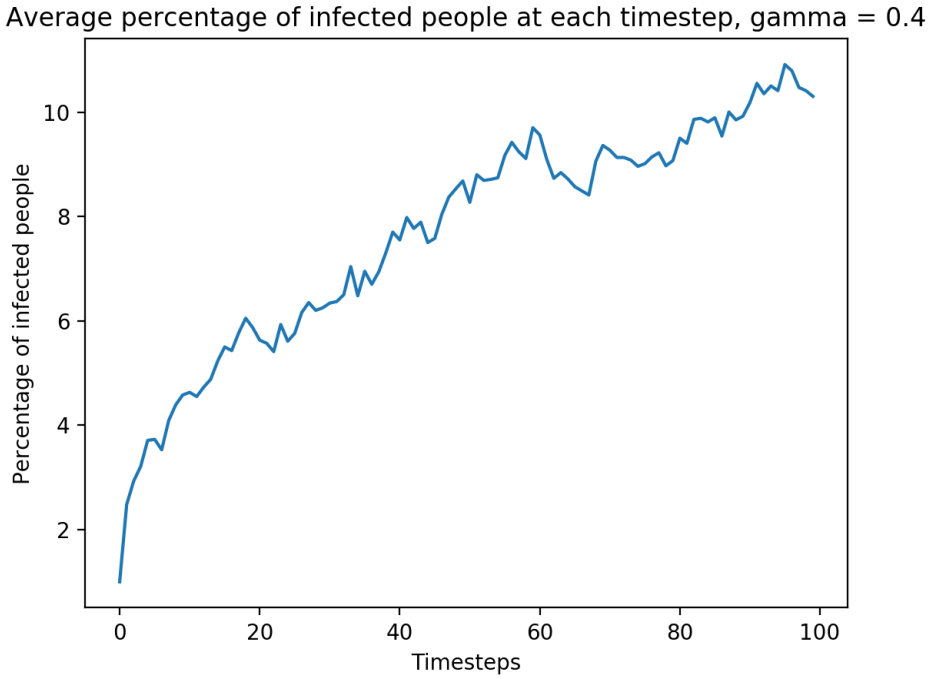

Figure 10: The percentage of infected people at each time step for $\gamma = 0.4$. The value at each time step is an average of 100 runs.

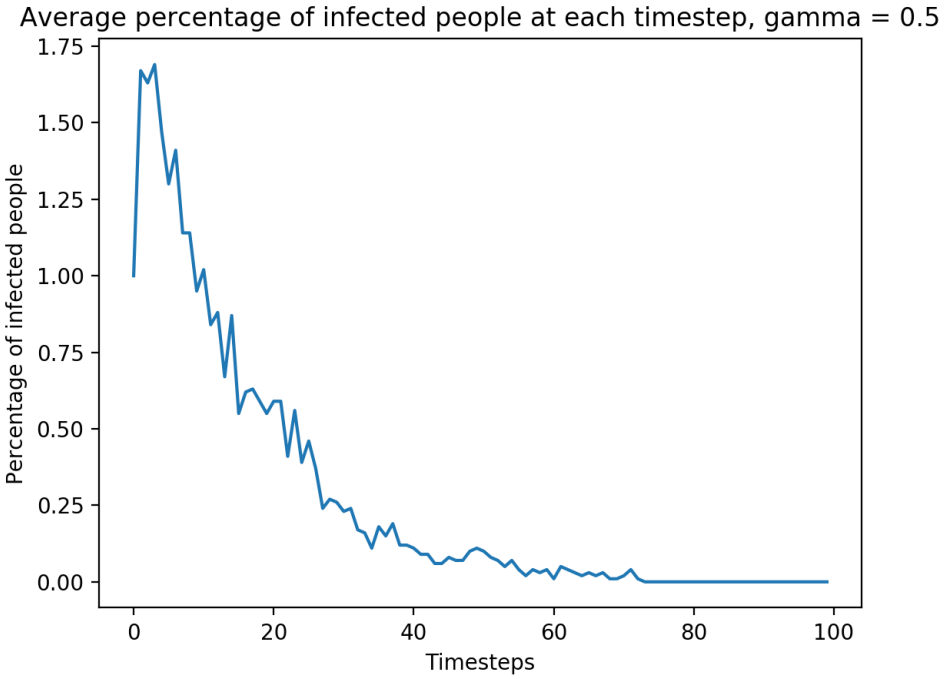

Figure 11: The percentage of infected people at each time step for $\gamma = 0.5$. The value at each time step is an average of 100 runs.

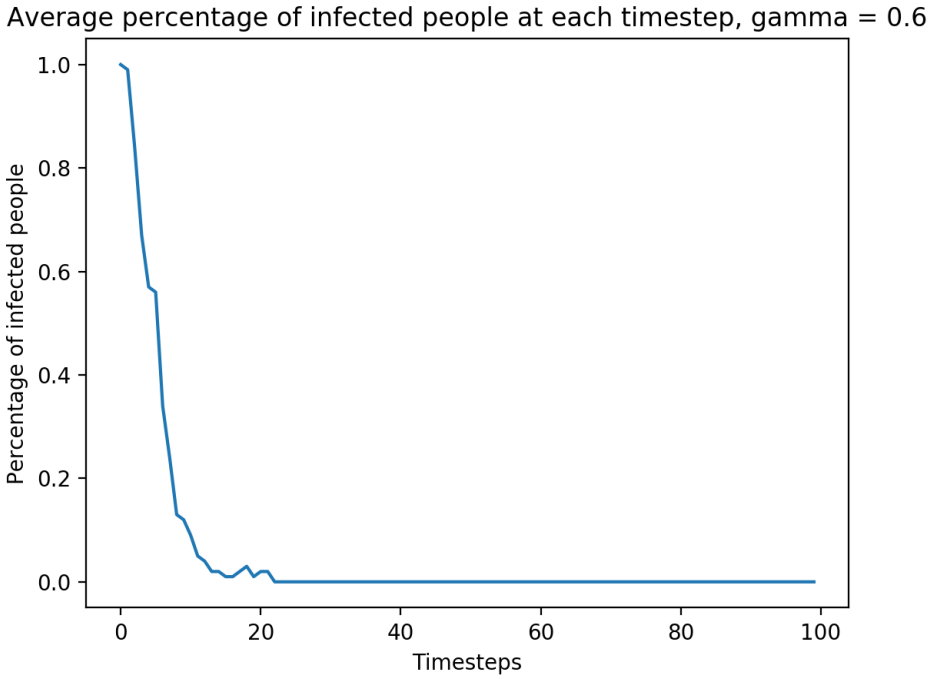

Figure 12: The percentage of infected people at each time step for $\gamma = 0.6$. The value at each time step is an average of 100 runs.

3.2

Table 1: Tabulated values of the virus extinction rate and average proportion of infected individuals at time t = T for all combinations of a selected set of values for the parameters γ & p. γ denotes the recovery rate and p denotes likelihood of an individual being infected at t = 0

γ	p	extinction at t = T [%]	avg. infected at t = T [%]
0.3	0.1	0	65.71
0.3	0.5	0	64.13
0.3	0.9	0	65.08
0.4	0.1	3	26.79
0.4	0.5	0	34.20
0.4	0.9	0	32.71
0.5	0.1	100	0
0.5	0.5	100	0
0.5	0.9	99	0
0.6	0.1	100	0
0.6	0.5	100	0
0.6	0.9	100	0

Note the symmetry in the table. There is probably a balancing point in which something resembling an equilibrium can be achieved. Such a point is however not present in the data. Instead, we observe a volatile quality, where the parameters either tend to render the virus very potent, or very transient.

3.3

Rules:

1. An infected individual recovers with a probability of γ, and continues to be infected with probability 1 - γ.

2. Those individuals who do not recover can infect others. If a susceptible individual has n infected neighbours at a chessboard distance of 1, then with probability $\alpha \cdot n$ it becomes infected.

3. Already infected neighbours can not be further infected.

4. A recovered individual becomes susceptible with a probability of $1 - \beta$, and can thereafter be infected again.

A video of the 2D epidemic for different recovery rates γ can be seen via the link below:
LINK TO YOUTUBE VIDEO.

The following cases use $\alpha = \frac{1}{8}$:

$\gamma = 0.3$ and $\gamma = 0.1$ give a very potent virus that dominates the domain convincingly even for a strong immunity parameter such as $\beta = 0.95$

For $\gamma = 0.45$ the virus remains on the verge of dying out and just barely manages to infect enough people to keep the spread going.

$\gamma = 0.6$ causes the individuals recover too quickly to spread the virus to their neighbors, resulting in the virus dying out.

If recovered individuals have a high probability of becoming susceptible, e.g. for $\beta = 0.1$. Even a relatively high recovery rate such as $\gamma = 0.6$ results in a disease that spreads over the entire domain. This is equivalent to a disease with a high rate of mutation, such as the common cold, for which immunity gained during recovery will not hinder new infections even in the near future. The majority of the population is healthy at any given time, the average infection does not last for long, yet the epidemic is wide spread with significant lasting power.

If the recovery rate is set relatively low, e.g. $\gamma = 0.2$, and the immunity lasting power is set exceptionally high, e.g. $\beta = 0.99$, we observe the phenomenon of herd immunity.

Using $\alpha = \frac{1}{20}$ gives significantly worse spread, but some interesting phenomenon can be observed when lowering γ and β to say 0.3 and 0.25 respectively. This can yield a simulation in which the disease very nearly goes extinct, slowly spreading and surviving best locally. This can be likened to a new disease for which new clusters can be thought of as mutations. At first, the disease only has high fitness for its local environment. As time goes by, it adapts to its surroundings and spreads far and wide. This adaptation is of course not expressed as rules, but in the beginning, the small cluster size leads to such a pattern. In this metaphor, an individual having eight infected neighbours (which gives 8/20 probability of infection) is likened to the disease being well adapted to that local neighbourhood.