

# Spread of epidemic

Author: Marcin Jędrzejczyk

Supervisor: Dr hab. inż. Jarosław Wąs

## 1 Introduction

When disease spread quickly and affect many people we can call it epidemic. Some of the most unfamous epidemic thorough history are XIV century Black Death and Spanish Flu pandemic in 1918. Infected person is not significant, however when a great number of people is sick it creates serious health and economic threats. Goal of simulating spread of epidemic is to better understand how epidemic behave in time, how quickly it spreads depending on many factors.[6] It can also be used to picture how vaccination affect disease spreading and confirms that herd immunity is important.

## 2 Literature models

Main models for epidemic in cellular automata are as follows:

- SIR
- SEIR
- SIS
- SEIRS

where each letter stand for status types of persons:

- S - susceptible
- I - infected
- E - exposed
- R - recovered

The choosen model should represent a disease we want to model. SIR model assume that in recovery status individual get full immunity and on the other hand in SIS person can get sick even after successful treatment.[6]

The neighborhood we choose can greatly affect dynamics of disease spreading as stated in [3]. However more than type of neighborhood the type of connection between cells is important. There can be one, two three or none ways of transport. It affects equations and speed of spreading.[6]

During next step model calculate how many people will be in each cell in each of groups(susceptible,infected,exposed,recovered). Vaccinations, cell connections, neighbourhood and people spread are major factors in how disease will spread.

Each cell is not an individual person, but a representation of square portion of land. That said there can be a cell with only infected people in it. People can move from one cell to another.[6] Such approach have two major benefits: “ first it reduces the total number of cells and hence reduces computation time; secondly it provides generality.”[2]

How and when cell change their status? This questions are answered, by mathematical equations with proper assumption about model. When we assume: that population size is constant, there is no incubation period and cycle where one is contagious is the same as disease. We can use The Kermack-McKendrick model[1]

$$\frac{dS}{dt} = -\beta SI \quad (1)$$

$$\frac{dI}{dt} = \beta SI - \gamma I \quad (2)$$

$$\frac{dR}{dt} = \gamma I \quad (3)$$

- S - number of susceptible persons
- I - number of infected persons
- R - number of recovered persons
- $\beta$  - infection rate
- $\gamma$  - recovery rate

There is a lot of mathematical models in literature. The following equation focus on models ready to use in CA.

In [6] there are equations for SIR model. Population in each cell is constant  $S_{i,j}^t + I_{i,j}^t + R_{i,j}^t = 1$ , cell (i,j)state is identified, by 3-tuple( $DS_{i,j}^t, DI_{i,j}^t, DR_{i,j}^t$ ) Equations looks as follow:

$$I_{ij}^t = (1 - \epsilon) \cdot I_{ij}^{t-1} + v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} + S_{ij}^{t-1} \cdot \sum_{\alpha, \beta \in V^*} \frac{N_{i+\alpha, j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{i,j} \cdot I_{i+\alpha, j+\beta}^{t-1} \quad (4)$$

$$S_{ij}^t = S_{ij}^{t-1} - \omega \cdot S_{ij}^{t-1} - v \cdot S_{ij}^{t-1} \cdot I_{ij}^{t-1} - S_{ij}^{t-1} \cdot \sum_{\alpha, \beta \in V^*} \frac{N_{i+\alpha, j+\beta}}{N_{ij}} \cdot \mu_{\alpha\beta}^{i,j} \cdot I_{i+\alpha, j+\beta}^{t-1} \quad (5)$$

$$R_{ij}^t = R_{ij}^{t-1} + \epsilon \cdot I_{ij}^{t-1} + \omega \cdot S_{ij}^{t-1} \quad (6)$$

where:

$$\mu_{\alpha\beta}^{(i,j)} = c_{\alpha\beta}^{(i,j)} \cdot m_{\alpha\beta}^{(i,j)} \cdot v \quad (7)$$

- $(i, j)$  - cell with coordinates  $(i, j)$  in lattice
- $t$  - iteration
- $v$  - virulence of the epidemic  $v \in [0, 1]$
- $\omega$  - vaccination rate  $\omega \in [0, 1]$
- $c_{\alpha\beta}^{(i,j)}$  - connection factor  $cin\{0, 0.4, 0.6, 1\}$
- $m_{\alpha\beta}^{(i,j)}$  - movement factor  $m \in [0, 1]$
- $N_{i,j}$  - population size in cell  $(i, j)$
- $\epsilon$  - recovery rate  $\epsilon \in [0, 1]$
- $V$  -neighbourhood of size  $n$  is defined as the set of coordinates  $(\alpha, \beta) :$   
 $N = (\alpha_k, \beta_k), 1 \leq k \leq n$
- $V^*$  -  $V^* = V - (0, 0)$
- $S_{i,j}^t$  - number of susceptible individuals in cell  $(i, j)$  in time  $t$
- $I_{i,j}^t$  - number of infected individuals in cell  $(i, j)$  in time  $t$
- $R_{i,j}^t$  - number of recovered individuals in cell  $(i, j)$  in time  $t$

In article [3] equations for SEIR model were presented( state of cell  $(i, j)$  is identified by 4-tuple  $(S_{ij}^t, I_{ij}^t, E_{ij}^t, R_{ij}^t)$ ):

$$S_{i,j}^{t+1} = S_{i,j}^t - v \cdot S_{i,j}^t \cdot I_{i,j}^t - v \cdot S_{i,j}^t \cdot \sum_{\alpha, \beta \in N^*} \frac{Q_{i+\alpha, j+\beta}}{Q_{i,j}} \cdot d_{i+\alpha, j+\beta}^{i,j} \cdot I_{i+\alpha, j+\beta}^t \quad (8)$$

$$E_{i,j}^{t+1} = (1 - \sigma) \cdot E_{i,j}^t + v \cdot S_{i,j}^t \cdot I_{i,j}^t + v \cdot S_{i,j}^t \cdot \sum_{(\alpha, \beta) \in N^*} \frac{Q_{i+\alpha, j+\beta}}{Q_{i,j}} \cdot d_{i+\alpha, j+\beta}^{i,j} \cdot I_{i+\alpha, j+\beta}^t \quad (9)$$

$$I_{i,j}^{t+1} = (1 - \epsilon) \cdot I_{i,j}^t + \sigma \cdot E_{i,j}^t \quad (10)$$

$$R_{i,j}^{t+1} = R_{i,j}^t + \epsilon \cdot I_{i,j}^t \quad (11)$$

- $(i, j)$  - cell with coordinates  $(i, j)$  in lattice
- $t$  - iteration
- $v$  - virulence of the disease
- $Q_{i,j}$  - population size in cell  $(i, j)$
- $\sigma$  - portion of exposed persons who become infected,  $\sigma \in [0, 1]$
- $\epsilon$  - recovery rate  $\epsilon \in [0, 1]$
- $N$  - neighbourhood of size  $n$  is defined as the set of coordinates  $(\alpha, \beta) :$   
 $N = (\alpha_k, \beta_k), 1 \leq k \leq n$
- $N^*$  -  $N^* = N - (0, 0)$
- $S_{i,j}^t$  - number of susceptible individuals in cell  $(i, j)$  in time  $t$
- $I_{i,j}^t$  - number of infected individuals in cell  $(i, j)$  in time  $t$
- $E_{i,j}^t$  - number of exposed individuals in cell  $(i, j)$  in time  $t$
- $R_{i,j}^t$  - number of recovered individuals in cell  $(i, j)$  in time  $t$

### 3 Project goals

The aim of this project is to implement SIR model of epidemic spread in cellular automata with JAVA. Main programming reference will be code from laboratory classes where cellular automata was used. The UI will be created with JavaFX and SceneBuilder. If possible interface will have many parameters to choose, like: vaccination value, people spread in lattice (random, uniform, constant), epidemic model (SIR, SIS, SEIR, SEIRS), neighbourhood size and type, if not only simple parameters for one model will be present.

## 4 Implemented models

### 4.1 Neighbourhood

Cell can have Von Newman or Moore neighbourhood, it is possible to change its size (from 1 to 4).

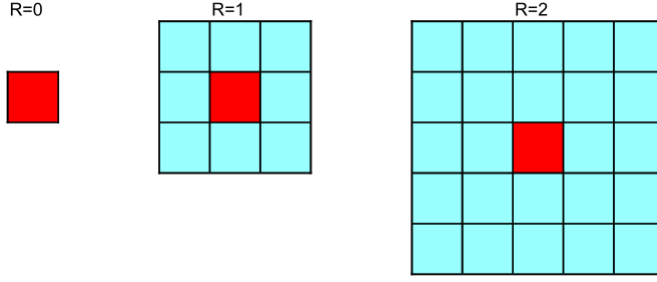


Figure 1: Moore neighbourhood

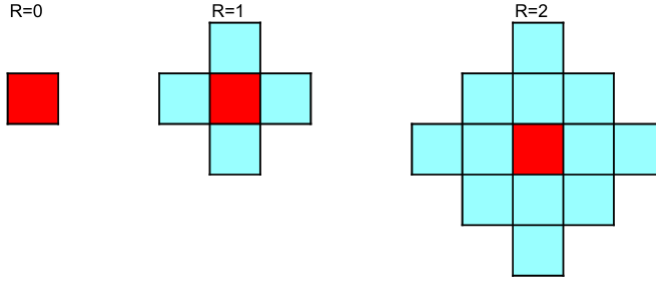


Figure 2: VonNewman neighbourhood

To not hardcode adding of neighbours to cell, following equations found in [4][5] were used:

$$N_{x_0,y_0}^M = \{(x, y) : |x - x_0| \leq r, |y - y_0| \leq r\} \quad (12)$$

$$N_{x_0,y_0}^V = \{(x, y) : |x - x_0| + |y - y_0| \leq r\} \quad (13)$$

- $x_0, y_0$  - target cell coordinates
- $x, y$  - all other cells coordinates
- $r$  - neighbourhood size
- $N_{x_0,y_0}^V$  - set of neighbours(VonNewman) cells for target cell
- $N_{x_0,y_0}^M$  - set of neighbours(Moore) cells for target cell

## 4.2 Epidemic models

SIR model was implemented with transition equations proposed by[6]. It was decided to use it, because :

- it takes vaccination into account
- parameters of environment are considered
- each cell has small population of individuals

SEIR model was implemented with transition equation proposed by[3].

SIS model was implemented, by changing SIR model equations.

### 4.3 Code

Program was written in Java. Simulation is created, by use of Timeline class, which object is used to execute doIteration() function. To avoid writing a lot of ifs and switches Factory pattern was used for creating Cell objects for chosen model.

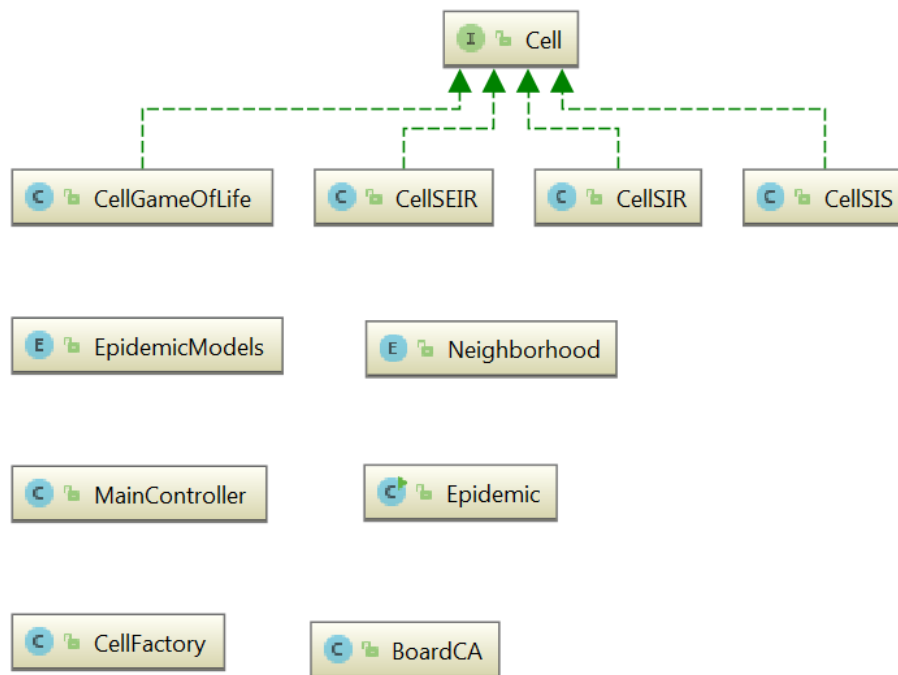


Figure 3: Class diagram

Cell.java

```

1 import javafx.scene.paint.Paint;
2 import java.util.HashMap;
3

```

```

4 public interface Cell {
5     void addNeighbour(Cell c);
6     Paint getColor();
7     void calculateNewState();
8     void changeState();
9     void clear();
10    int getType();
11    void setType(int i, HashMap<String, Double> par);
12    int getNumberOfTypes();
13 }

```

For testing how code works and if program design is correct game of life was also implemented with not hard coded parameters.

#### 4.4 Finished GUI

The user interface was created with JavaFX and SceneBuilder.

To start simulation:

- Choose model
- Choose neighbourhood type and size
- Set model parameters
- Click INIT button
- Change cell types, by mouse clicking(or dragging) on lattice (it is possible to choose new type of cell and cell parameters, depends on chosen model)
- Change speed of simulation on slider(optional, default 1s)
- To start click START button
- To pause click STOP button(you can resume with START button)
- Use CLEAR button to terminate simulation and be able to set up a new one

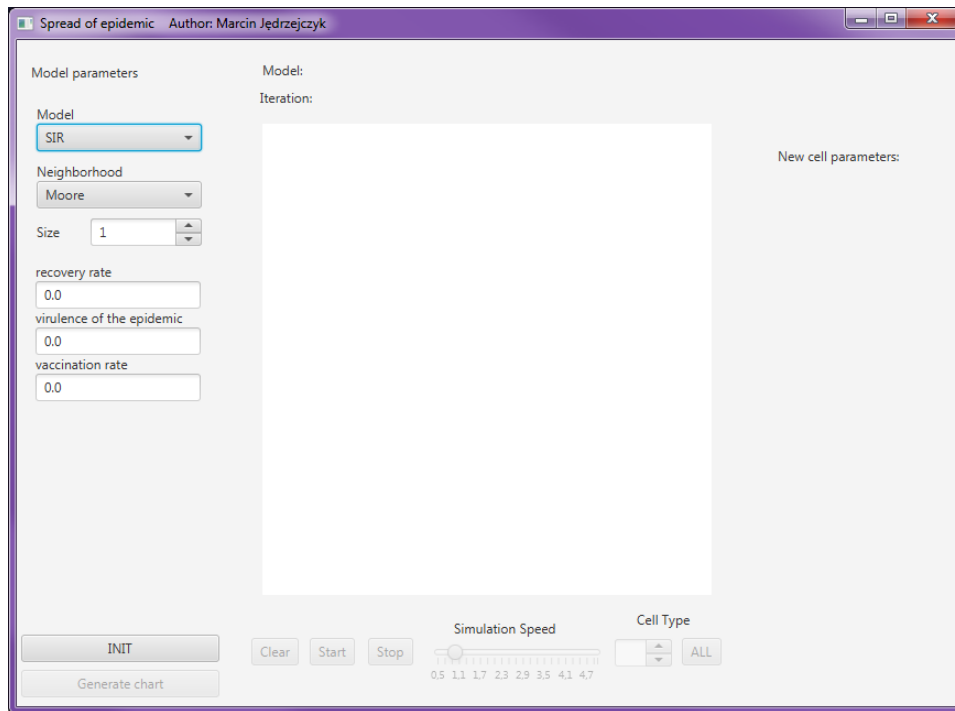


Figure 4: GUI - program start

## 5 Summary

Testing implementation of SIR model showed how great is impact of vaccination in preventing disease spreading. Also it possible to see quarantine, by using empty spaces or cell of recovery type. One observer anomaly is when all cells have connection factor=1, instead of covering whole lattice with 1 type of cells program create symmetric symbols.

SEIR model is less precise when it comes to spreading disease from neighbours, because it only take into account Euclidean distance between cell and its neighbours and not like SIR model connection factor, movement factor and virulence of the epidemic.

It is advised to check more published papers to find better mathematical model for SIS model.

It is possible to add more models. Program was build with idea of adding new models without big problems. It is proven, because implementation of SEIR model took less time than SIR model, actually copy paste and adjust. What is more Adding more parameters should not be a problem, because program stores them in hashmaps.



## Bibliography

- [1] Sharon Chang. “Cellular Automata Model for Epidemics”. W: (2008). URL: <http://csc.ucdavis.edu/~chaos/courses/nlp/Projects2008/SharonChang/Report.pdf>.
- [2] Shih Ching Fu i George Milne. “Epidemic Modelling Using Cellular Automata”. W: (sty. 2003).
- [3] B. Cissé, S. El Yacoubi i A. Tridane. “Impact of Neighborhood Structure on Epidemic Spreading by Means of Cellular Automata Approach”. W: *Procedia Computer Science* 18.Supplement C (2013). 2013 International Conference on Computational Science, s. 2603 –2606. ISSN: 1877-0509. DOI: <https://doi.org/10.1016/j.procs.2013.05.450>. URL: <http://www.sciencedirect.com/science/article/pii/S1877050913005930>.
- [4] Eric W. Weisstein. *Moore Neighborhood*. URL: <http://mathworld.wolfram.com/MooreNeighborhood.html>.
- [5] Eric W. Weisstein. *von Neumann Neighborhood*. URL: <http://mathworld.wolfram.com/vonNeumannNeighborhood.html>.
- [6] S. Hoya White, A. Martín del Rey i G. Rodríguez Sánchez. “Modeling epidemics using cellular automata”. W: *Applied Mathematics and Computation* 186.1 (2007), s. 193 –202. ISSN: 0096-3003. DOI: <https://doi.org/10.1016/j.amc.2006.06.126>. URL: <http://www.sciencedirect.com/science/article/pii/S0096300306009295>.