

Temporal and Spatial Evolution of a SIR-type Epidemic – ARGESIM Comparison C17 - Definition

H. Hötzendorfer, N. Popper, F. Breitenecker, Vienna University of Technology hhoetz@osiris.tuwien.ac.at niki.popper@drahtwarenhandlung.at

The aim of this comparison is to numerically solve the classical Kermack McKendrick epidemic-model, given through a system of ordinary differential equations, and furthermore to develop a cellular automaton model whose properties represent the ones governed by the continuous model. Since for this purpose, lattice gas cellular automata (LGCA) seem to be well suited, different LGCA should be implemented, the results compared and arising differences interpreted and discussed.

The used software is not of primary interest as the concentration should be put on the comparison of completely different approaches to simulate the same process. Under certain assumptions analogy will have to be shown and advantages of one or the other approach emphasized.

General Description:

W. O. Kermack and A. G. McKendrick proposed in 1926 a simple SIR model for epidemic spread based upon a system of nonlinear ordinary equations. The abbreviation SIR stands for susceptible – infected – recovered and it deals with an epidemiological model to investigate the theoretical number of people infected with a contagious illness in a closed population over time.

As to simplify the model, several assumptions have been made. The first one considers the population size which has to be constant over the observed period of time.

This means that no in- or outflow (e.g. births or deaths) takes place. Besides that, incubation time of the infectious agent is zero and the duration of infectivity is the same as the length of the disease.

Taking into account all this information, the following system arises:

 $\frac{\partial S(t)}{\partial t} = -r \cdot S(t) \cdot I(t)$ $\frac{\partial I(t)}{\partial t} = r \cdot S(t) \cdot I(t) - a \cdot I(t)$ $\frac{\partial R(t)}{\partial t} = a \cdot I(t)$ (1)

Here *r* is the infection rate, *a* the recovery rate, S(t) the number of susceptible individuals, I(t) the number of infected individuals and R(t) the number of recovered individuals, at time *t* respectively.

Purpose of this comparison is not just solving this task with conventional methods but also to implement a cellular automaton model to obtain a solution for the problem. Therefore, a lattice gas cellular automaton (LGCA) should be considered to describe the epidemic. For those not being familiar with cellular automata in general and with LGCA in particular, a brief outline should explain the main properties.

Cellular automata are based upon a discretisation of space and time. Each cell can hold a finite number of states and the temporal evolution of the automaton is governed by transition rules which act locally and simultaneously on the cells. The transition rules can either be deterministic or probabilistic. Locality is introduced by a neighbourhood-function which defines the cells being determinant for updating the cell state (see Figure 1).



Figure 1: Graphical representation of different neighbourhood-functions in 2-dimensional cellular automata

Figure 2 shows a configuration of the probably best known example of a 2-dimensional cellular automaton, the Game of Life. Cells can hold two different states and Moore-neighbourhood (eight surrounding neighbours) is chosen as neighbourhoodfunction. The transition rules are purely deterministic but will not be presented here in detail.



Figure 2: Game of Life; 2-dimensional CA with two different states for each cell (black, white)

COMPARSIONS

SIMULATION NEWS EUROPE



We have to distinguish between the **HPP** (Hardy, de Pazzis, Pomeau - 1973) and the **FHP** (Frisch, Hasslacher, Pomeau - 1986) model. The first one is composed of a **square lattice** which contains no more than **four particles** per cell. Each particle is determinate by its lattice-vector which connects the cells to its four nearest neighbours and defines the direction the particle moves on. It is not possible that one cell contains two particles moving along the same direction. If and only if two particles collide when entering one cell from opposite directions, each particle changes direction by 90°. Usually the orientation of the deflection will be predefined but it may also be chosen randomly for each collision. Discussion of this feature will be content of a later task.



Figure 2: HPP and FHP lattice-gas cellular automaton; the lines represent the directions of the lattice-vectors connecting the cells with its neighbours; particles are not represented

The FHP model consists of **hexagonal** structure containing a maximum of **six particles** per cell again being defined by its lattice-vectors connecting the cell to its six nearest neighbours. Collision rules are more elaborated in that case; we chose the simplest ones, also called **FHP-I collision rules**.



A two-particle head-on collision redirects the particles by changing the direction of their lattice vector by 60° randomly clockwise or counter clockwise but equally for the two particles. A three-particle head-on collision again changes the direction equally by 60° either clockwise or counter clockwise but remaining the same for all collisions of this type. For further readings we recommend [2].

We now identify each particle of the automaton with one individual, which can either be susceptible, infected or recovered. Let *N* be the total number of nodes (cells) in the lattice and S_k the number of susceptible individuals in the entire lattice at time *k*. Then the probability of one susceptible individual to become infected in a single time step ($k \rightarrow k+1$) is

$$1 - (1 - r)^{\frac{l_k}{N}}$$
 (2)

and hence the expected number of susceptible individuals who become infected is

$$S_k \cdot \left(1 - (1 - r)^{\frac{I_k}{N}}\right) \cdot \tag{3}$$

The expected number of individuals who recover in a single time step is

$$a \cdot I_k \cdot$$
 (4)

For a well stirred population equations (2), (3) and (4) yield to:

$$S_{k+1} = S_k \cdot (1-r)^{\frac{I_k}{N}}$$
$$I_{k+1} = I_k + S_k \cdot \left(1 - (1-r)^{\frac{I_k}{N}}\right) - a \cdot I_k$$
$$R_{k+1} = R_k + a \cdot I_k$$

The following considerations may give a relation to a system of difference equations. Taylor expansion for small $\ensuremath{\mathsf{r}}$

$$(1-r)^{\frac{I_k}{N}} = 1 - \frac{r \cdot I_k}{N} + \frac{I_k \cdot (I_k - N) \cdot r^2}{2 \cdot N^2} + \cdots$$

keeping only the first two terms and defining

$$\mathcal{O}_{S}(k) = \frac{S_{k}}{N}, \dots$$
 yields to

$$\rho_{S}(k+1) = \rho_{S}(k) - r \cdot \rho_{S}(k) \cdot \rho_{I}(k)$$

$$\rho_{I}(k+1) = \rho_{I}(k) + r \cdot \rho_{S}(k) \cdot \rho_{I}(k) - a \cdot \rho_{I}(k)$$

$$\rho_{R}(k+1) = \rho_{R}(k) + a \cdot \rho_{I}(k)$$
(5)

ssue 41/42



This system of difference equations (5) is of equal structure as the previously given system of differential equations (1).

Task a – CA and ODE Simulations

Find the solution for the problem by solving the system of ODEs (1) using the initial values and parameters given in Table 1.

| $S(t=0) = S_0$ | 16000 |
|------------------|---------------------|
| $I(t=0) = I_0$ | 100 |
| $R(t=0) = R_0$ | 0 |
| Infection rate r | 0.6·10 ⁴ |
| Recovery rate a | 0.2 |

Table 1: initial values and parameters for task a

Following this data, implement a FHP LGCA with a domain size of 100×100 (and therefore 10^4 hexagons), an infection rate of r = 0.6 (in accordance to the parameter value for the previous task divided by the number of hexagons) and periodic boundary conditions to remodel the system and compare the obtained results. To prescribe an initial configuration, uniformly distribute the individuals of type S and I.

Oppose the results for the FHP model to a HPP model and discuss the differences. Furthermore change the properties of the HPP model regarding the direction of the deflection of particles to obtain random motion (not only depending on the initial configuration) and compare the results to the former ones.

Task b - Vaccination Strategies in CAs

Use the properties of the FHP model of the previous task and implement different strategies for vaccination of susceptible individuals in LGCA. A group of 4000 susceptible individuals should be vaccinated. Therefore, assume having the infected individuals grouped together in one half of the domain and vaccinate a part of the rest of the population. Experiment with different policies for vaccination and oppose the results. In particular, implement vaccination in the whole domain, vaccination in the part of the domain containing the infected individuals (epidemic area) and vaccination of individuals being located at the borders of the epidemic area. Once again solve the continuous model, wherein a vaccination process can easily be described by setting R_0 = 4000 but note that spatial inhomogenities can not be represented in this approach.

Task c – ODE vs. CA Solutions

Until now, spatial grouping of infected individuals can be observed which will consequently change the results of the simulation in comparison with the continuous approach. Change the FHP LGCA to avoid spatial inhomogenities of different groups of individuals. For these purpose, ensure perfectly uniform distributions for all three groups of populations (S(t), I(t), R(t)) by randomly rearranging all individuals in every time step of the automaton. The fact that this assumption destroys basic principles of LGCA is not decisive for our studies.

Use parameter values given in Table 2 for these simulations.

| $S(t=0) = S_0$ | 40000 |
|------------------|---------------------|
| $I(t=0) = I_0$ | 1000 |
| $R(t=0) = R_0$ | 0 |
| Infection rate r | 0.3·10 ⁴ |
| Recovery rate a | 0.2 |

Table 2: initial values and parameters for task c

Show that for these parameter values the obtained results for the continuous and the LGCA approach are not only of equal qualitative behaviour but also lead to fairly similar quantitative values. Explain the slight differences concerning the speed of epidemic spread by comparing the data with the solution of the difference equation (5).

Experiments with lower values of S_0 and interpret the growing discrepancies.

References

- Henryk Fukś, Anna T. Lawniczak: Individualbased lattice model for spatial spread of epidemics, Discrete Dynamics in Nature and Society 6 (2001), 191-200
- [2] Dieter A. Wolf Gladrow: Lattice gas cellular automata and lattice Boltzmann models: an introduction, Springer (2001)
- [3] G. Rousseau, B. Giorgini, R. Livi, H. Chaté : Dynamical phases in a cellular automaton model for epidemic propagation, Physica D 103 (1997), 554-563
- [4] B. Schönfisch: Propagation of fronts in celluar automata, Physica D 80 (1995), 433-450
- [5] Shih Ching Fu: Modelling Epidemic Spread using Cellular Automata, Thesis (2002)
- [6] Hokky Situngkir: Epidemiology through Cellular Automata – Case of study: Avian Influenza in Indonesia, Dept. Computational Sociology; Bandung Fe Institute
- [7] Bruno di Stefano, Henryk Fukś, Anna T. Lawniczak: Object Oriented Implementation of CA/LGCA Modelling Applied to the Spread of Epidemics
- [8] L. Hufnagel, D. Brockmann, T. Geisel: Forecast and control of epidemics in a globalized world, *PNAS* 101 (2004), 15124-15129