Diffusion Approximation in a Stochastic Cellular Automaton Model for Epidemics

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Abstract. Although microscopic models are nowadays getting more and more popular among, still the modelling approach lacks of appropriate mathematical theory to confidentally rely on the outputs of the derived models. Especially unexpected chaotic group behaviour and the inability to validate and parametrise the model often leads to unusable simulations. The investigated testcase, a simple cellular automaton (CA) simulating the temporal development of a SIR (Susceptible-Infected-Recovered) type epidemic, shows a field of application for so called complexity theory. In order to explain and analyse the aggregated simulation results of the CA, certain methods usually used in Markov theory for quantum mechanics, basically extensions of so called diffusion approximation [1], are applied. Finally, already suspected, correlations to the solutions of the famous SIR differential equations, formerly derived by Kermack and McKendrick [2], can be proven with analytical methods and extended by convergence results and qualitative error estimations.

Introduction

Due to tough limited resources usage of modelling and simulation to support strategic planning has nowadays become an indispensable part of management. Especially the increasing number of simulations for emerging problems within so called soft-sciences like medicine, biology or sociology can be observed. Main reason for this development is the exponentially increase of computational resources (compare Moore's law [3]) making it possible to simulate very complex, individual-based models, which, correctly validated, produce reliable results. Nevertheless the validation process for these models is very difficult, requires lots of data and heuristic parameter-sweeps for sensitivity analysis. The usage of microscopic models always involves the danger, that maybe unpredictable chaotic group behaviour distorts the results. Most of the microscopic modelling methods, like agent-based models and cellular automata, somehow lack of necessary mathematical basis.

Nevertheless compared to classic macroscopic modelling methods some of the advantages and disadvantages of so called microscopic or individual-based models can be summarized in Table 1.

Advantages	Disadvantages
Lower abstraction level compared to reality	Difficult to validate
Easy to understand for non-experts	High computational efforts
Very flexible regarding system changes	Difficult to document regarding reproducibility
More suitable for eye-catching visualisations	Sometimes unpredictable and chaotic results

Table 1: Some advantages and disadvantages of microscopic models.

Within epidemiology so called SIR (Susceptible – Infected - Recovered) strategy already poses the base for lots of flexible microscopic models for diseases and vaccine strategies (see e.g. [4],[5]). Hereby the spread of one single serotype in a certain environment among certain individuals is studied, wherein the individuals are divided into the aformenetioned three subclasses. Infected individuals forward the spread of the disease among the susceptible individuals. Recovered individuals are meant to stay immune against the serotype for the further progress of the disease (died individuals are included here).

Several years ago a team of the AMSDM group (Applied Modelling, Simulation and Decision Making), a cooperation of the "dwh GmbH" and the group of Prof. Felix Breitenecker at Vienna University of Technology), created lots of epidemics-related teaching material for modelling lectures. This material was developed in the context of a huge project with the Federation of Austrian Social Insurance analysing the effect of different vaccine strategies against influenza viruses on the Austrian social system (population, financial aspects, etc.) supported by theoretical models ([6], [7]). Due to interesting results especially one of them attracted special attention. The emphasis is laid on a stochastic cellular automaton (short CA), in detail described in chapter 1, which can be used to simulate simple epidemics with the aforementioned SIR strategy. For more information the reader is referred to [18]. In addition the influence of vaccinations before the breakout of the disease can be investigated confirming the flexibility of agent-based models compared to most macroscopic models.

1 Comparison of two Modelling Approaches

Some resulting curves of the mentioned cellular automaton model can be seen in Figure 1. The three graphs located at the lower part of the figure are calculated by the number of cells sharing the same state. The green line shows the number of "susceptible" cells, the red and blue line illustrate the number of "infected" and "immune" cells. The upper part of the figure shows the state of the cellular automaton at a certain time during the simulation run.

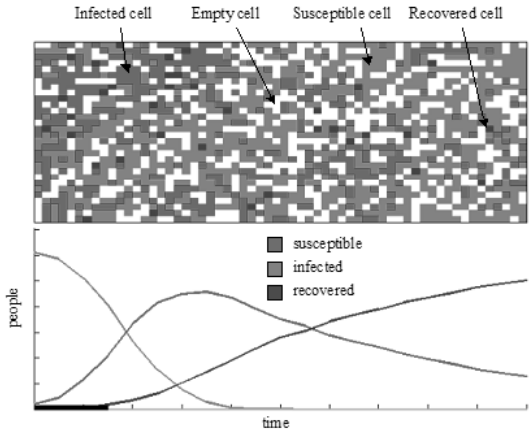

Figure 1: Example Result of the Cellular Automaton.

Studies (see [8]) have shown, that a comparison between the aggregated CA-results and the famous SIR differential equation model (short ODE model) by Kermack and McKendrick, 1927 [2], seen in (1) is justified.

$$\begin{aligned} \frac{dS}{dt} &= -\alpha IS \\ \frac{dI}{dt} &= \alpha IS - \beta I \\ \frac{dR}{dt} &= \beta I \end{aligned} \tag{1}$$

This nonlinear system of differential equations can only be solved using numerical integration algorithms, as it does not have any nontrivial analytical solutions. A MATLAB generated plot of the Runge-Kutta-Fehlberg (4^{th} order with step-size control) approximation of the solution is shown in Figure 2.

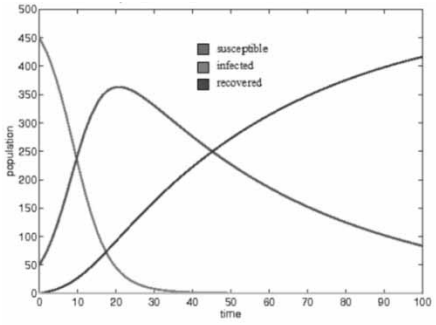

Figure 2: Example Result of Classic SIR Differential Equations.

It is undeniable that the solution curves of both models look very similar. Further comparative studies (also [9]) showed that there is a correlation between the parameters of the ODE model (Infection rate α, Regeneration rate β) and the stochastic CA model (movement rules, regeneration probability, neighbourhood, population-density and infection-probability) in form of closed equations. Unfortunately these were mostly derived via basic stochastics and experiments, without any statements regarding convergence and errors between those two approaches.

A possible way to compare these two completely different modelling ideas with mathematical techniques is presented in the following chapters. One must not forget that a stochastic, time and space discrete model is hereby compared with a deterministic, completely continuous model posing a big challenge. Starting to analyse the CA model by a series of transformations finally the ODE formulation will be derived. Convergence results and error estimations are going to appear as by-products of these.

2 Cellular Automaton – Definition

Although usually presented and implemented as a cellular automaton the most comprehensible description of this model is claimed in form of an agent-based model. The transition rules for the cellular automaton can be derived analogously. Although the underlying modelling concept is completely different in this simple case both modelling approaches (agent-based and CA) end up with the same model.

2.1 Space

Let Ω be a discrete two-dimensional rectangular grid with $M = M_x \cdot M_y$ aligned cells $c_{i,j}, i \in \{0, \dots, M_x\},\ j \in \{0, \dots, M_y\}$. Each cell itself is partitioned into four cell-fractions: $\{c_{i,j,1,1}, c_{i,j,1,2}, c_{i,j,2,1}, c_{i,j,2,2}\}$.

2.2 Agents (non-empty cells)

A number $N \leq 4M$ of agents $a_n(t), n \in \{0, \dots, N\}$ are placed onto the grid. Each agent is assigned exactly one cell-fraction: $a_n(t)_1 \in \{i, j, k, l\}$. Furthermore each agent has one of three states ('susceptible', 'infected' or 'recovered'"): $a_n(t)_2 \in \{1,2,3\}$. To simplify the speech and to support a pictorial representation, the state of the agent is usually called as a prefix of the word agent: e.g. 'infected agent'.

2.3 Simulation

The simulation is performed time discrete with equidistant steps $\{1, \dots, t_{end}\}$. Each time step is split into two phases. First of all the so called infection-phase is performed wherein new-infections and regenerations are calculated. During this phase each agent is addressed once and basically two cases can lead to a state-change:

- If the agent is susceptible and shares a cell with an infected agent, there is a certain probability $P(1,2)$ that the agent gets infected too.
- If the agent is already infected, there is a certain probability $P(2,3)$ that the agent recovers and becomes immune against the disease.

All state changes are done simultaneously.

After this a series of movement rules are applied during so called movement-phase. Hereby all agents are shifted corresponding to certain laws to achieve new arrangements. Basically they are inspired by movement rules of so called lattice gas cellular automata (compare FHP model [10]) and will not be explained here in detail.

A summary of all these ideas is found in Figure 3. It can be seen that even this rather simple model (it is usually very well understood if a picture similar to Figure 3 is given) is very difficult to be described in a formal language especially regarding mathematical formulas, functions and equations.

3 Analysis and Transformations

In order to achieve convergence results and error esitimation several stochastic methods known from Markov theory can be used. Concrete a three-dimensional version of the so called diffusionapproximation can be applied. Therefore it is, first of all, necessary to convert spatial influences like neighbourhood and transition conditions into probabilities.

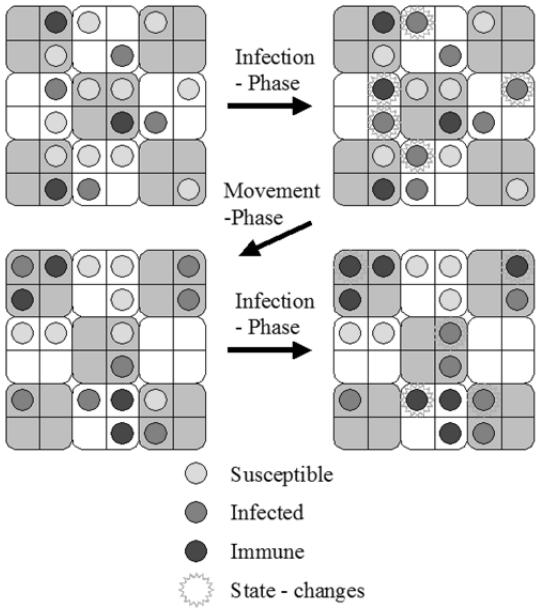

Figure 3: Visualisation of the rules for the SIR Cellular Automaton.

3.1 Extracting probabilities

Defining movement rules in general is basically related to two main ideas: First the motion needs to guarantee a good mixture among the individuals. A bad systematic motion could lead to (usually) unwanted loops or clustering. From this point of view, the ideal movement rule to guarantee a perfect mixture would be a complete stochastic re-arrangement of all agents at the end of each time-step:

$$a_n(t)_1 = (i, j, k, l) \Rightarrow a_n(t+1)_1 = (X, Y, U, V).$$

Though this is not compatible with the second basic aspect of movement, namely that agents cannot cover an infinite range (usually only 1-2 cells) during one time step, a closer look at this aspect is taken. Defining the agitation of all agents completely randomly, basically two independent random processes are responsible for the state of each agent, hence the model is cleanly stochastic and can be described by transition probabilities then. For a fixed index k the probability of agent k changing its state from susceptible to infected can be calculated by multiplying the probability of being placed next to an infected agent during movement-phase and the probability of getting infected by this agent. By simple uniform distribution argument this probability can be calculated to:

$$P(a_k(t+1)_2 = 2 | a_k(t)_2 = 1) = \\ = \frac{3\rho \#\{a_k(t)_2 = 2\}}{N} P(1,2).$$

Hereby ρ denotes the population-density $\rho = \frac{N}{4M}$ and model parameter $P(1,2)$ is a fixed infection probability (as described earlier). Analysis of the original model using the original, lattice gas automaton inspired movement rules, shows that a random initial placement of the agents is enough to ensure at least (2) holds.

$$\omega_{1,2} \coloneqq P(a_k(t+1)_2 = 2 | a_k(t)_2 = 1) = \\ = \frac{3\rho \#\{a_k(t)_2 = 2\}}{N} P(1,2) + O(N^{-1}) \quad (2)$$

The Landau-symbol O indicates the asymptotical order of the expression. In this case the asymptotical expression is a result of accidentally clustering of infected or immune agents and strange distributions close to the borders which can lead to other contact probabilities. In both cases they can asymptotically be neglected regarding big numbers of agents (with constant density).

Obviousely the result of (2) can, in case of high densities and high numbers of infected agents, end up with a value higher than 1, which in terms of probability theory cannot be correct. Reason for this observation is a basically wrong ansatz wherein the contact probability is sloppily calculated via a simple multiplication of the number of neighboured cells (3) times the probability of observing an infected agent ($p_i \coloneqq \frac{\rho \#\{a_k(t)_2 = 2\}}{N}$). Nevertheless the correlation between the correct calculation, seen on the left hand side in (3) and the used calculation on the right hand side is very small and vanishs second order for small densities.

$$1 - (1 - p_i)^3 = 3p_i + O(\rho^2) \quad (3)$$

Surely the probability of regeneration is completely independent of the agents' position:

$$\omega_{2,3} \coloneqq P(a_k(t+1)_2 = 3 | a_k(t)_2 = 2) = P(2,3).$$

Thus, eliminating the influence of the spatial grid, the agent-vector can be described by an N-dimensional Markov-chain with transition tensor (4).

$$\Omega_i \coloneqq \begin{pmatrix} 1-\omega_{1,2} & \omega_{1,2} & 0 \\ 0 & 1-\omega_{2,3}(\vec{a}) & \omega_{2,3}(\vec{a}) \\ 0 & 0 & 0 \end{pmatrix} \\ i \in \{0, \dots, N\}. \quad (4)$$

As the transition probabilities depend on the sum of all agents sharing the same state the agents itself cannot be described by single Markov-chains which poses the main difference to classic microscopic Markov-models.

Furthermore Markov-theory is going to pave the way to overcome the obstacles between discrete (CA model) and continuous (ODE model).

3.2 Time discrete to continuous

Suppose a given regular, homogeneous, time continuous but space discrete Markov-process $X(t), t \in [0, t_{end}]$ with transition matrix R and three possible states the Kolmogorov equation (5) holds which is in case of sufficient regularity solved by (6).

$$P(X(t) = i | X(0) = j)_{i \in \{1,2,3\}}{}' \\ = R \cdot P(X(t) = i | X(0) = j)_{i \in \{1,2,3\}} \quad (5)$$

$$P(X(t) = i | X(0) = j) = \exp(\mathrm{Rt}) \cdot \vec{\mathrm{e}}_\mathrm{j}. \quad (6)$$

Therefore a time continuous Markov-process according to $\Omega = \exp(R \cdot 1) \Rightarrow R \coloneqq \log(\Omega)$ seems to be an appropriate choice to approximate the time discrete one. Surprisingly the first order Taylor approximation: $R := R_i \coloneqq \Omega_\mathrm{i} - \mathrm{Id}, i \in \{0, \dots, N\}$ turns out to be the better choice in our case conserving mean and variance. Errors regarding this approximation can e.g. be calculated using the Taylor series remainder.

3.3 Spatial discrete to continuous

Key observation for the transformations is definitely that the so called observable vector defined by (7) 'counting' all Markov-processes sharing the same state is also a time continuous Markov-process with transition rates ϕ of which only two do not vanish.

$$\vec{o}(t) := \frac{1}{N}\begin{pmatrix} \sum_{i=1}^{N} \delta_{X_i(t),1}(t) \\ \sum_{i=1}^{N} \delta_{X_i(t),2}(t) \\ \sum_{i=1}^{N} \delta_{X_i(t),3}(t) \end{pmatrix} \tag{7}$$

The two non-zero rates are given in (8) and (9).

$$\phi_{N^{-1}\binom{i}{j}_k, N^{-1}\left(\binom{i}{j}_k + \binom{-1}{1}_0\right)} = iR_{1,2} = i\omega_{1,2} \tag{8}$$

$$\phi_{N^{-1}\binom{i}{j}_k, N^{-1}\left(\binom{i}{j}_k + \binom{0}{-1}_1\right)} = jR_{2,3} = j\omega_{2,3}. \tag{9}$$

These two transition rates, also called jump rates denote the rate for jumps of single agents from state 1 to 2 respectively from state 2 to 3. As the process is continuous and regular no more than one agent can change its state during an infinitesimal time-interval. Therefore all other rates vanish.

Summarizing the performed transformations Figure 4 is given.

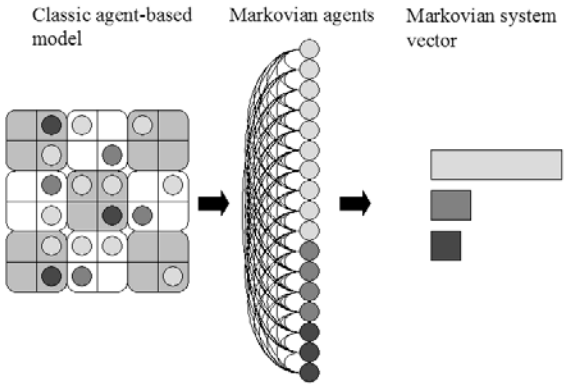

Figure 4: Transformation from CA to Markov Process.

For big numbers of N the observable can take any value between 0 and 1 and thus the probability function $p(t, \vec{o}|\vec{o}_0)$ can be approximated with a continuous and differentiable function taking account interpolation errors. As the observable vector is still markovian the so called diffusion approximation can be performed.

4 Diffusion Approximation

The Kolmogorov equation respectively the closely related master-equation poses the base for a lot of analytical transformations known within physicists under the name "diffusion approximation". This technique, first time published 1983 by N.G. Van Kampen ([1], [11]), is based on Taylor-series expansion (named after Kramers and Moyal [12]) and the variable substitution $\vec{o} =: \langle\vec{o}\rangle(t) + N^{-1/2}\vec{\xi}(t)$ (compare Itô [13]). This technique is commonly used to describe the temporal development of particle-probabilities. Here only the results of these technique are presented witch are valid with respect to asymptotic errors of order $O(\sqrt{N^{-1}})$. Key observation of these estimations is the derivation of an ODE for the mean value of the observable vector:

$$\left(\langle\vec{o}\rangle(t)\right)' = \begin{pmatrix} -1 \\ 1 \\ 0 \end{pmatrix}\langle\vec{o}\rangle_1(t)\omega_{1,2} + \begin{pmatrix} 0 \\ -1 \\ 1 \end{pmatrix}\langle\vec{o}\rangle_2(t)\omega_{2,3},$$

which, resubstituted, leads to

$$\left(\langle\vec{o}\rangle(t)\right)' = \begin{pmatrix} -\langle\vec{o}\rangle_1(t)\langle\vec{o}\rangle_2(t)\frac{3\rho}{N}P(1,2) \\ \langle\vec{o}\rangle_1(t)\langle\vec{o}\rangle_2(t)\frac{3\rho}{N}P(1,2) - \langle\vec{o}\rangle_2(t)P(2,3) \\ \langle\vec{o}\rangle_2(t)P(2,3) \end{pmatrix}. \tag{10}$$

Equation (10) can be determined to match the SIR ODE by Kermack and McKendrick. Variance of the observable can be calculated to vanish for $N \to \infty$ with square-root order. Applied time-scaling $\tau = t/c$ in addition to inverse scaling of the rates $\theta := c\omega$ finally leads to convergence of the aggregated results of the stochastic CA towards the solution of the ODE system when $c \to 0$ and $\rho \to 0$. Although the sloppily calculated probability for a state change from susceptible to infected is slightly wrong the error does not disturb the results. Nevertheless the better fitting ODE curves would be (compare with (1)):

$$\begin{pmatrix} S \\ I \\ R \end{pmatrix}' = \begin{pmatrix} -\alpha S(1-(1-I)^3) \\ \alpha S(1-(1-I)^3) - \beta I \\ \beta I \end{pmatrix}. \tag{11}$$

5 Conclusion

The presented strategy can without loss of generality be extended to other microscopic models and can help to improve understanding of unexpected group behavior in general. The diffusion approximation introduced by Van Kampen [1] was extended and used to show convergence between a classic ODE model and a time discrete Cellular Automaton. Hereby a similar strategy as introduced on the example on economic models in Aoki [14] and on the example of queuing processes in Dohse [15] was used to achieve the transformation from discrete to continuous.

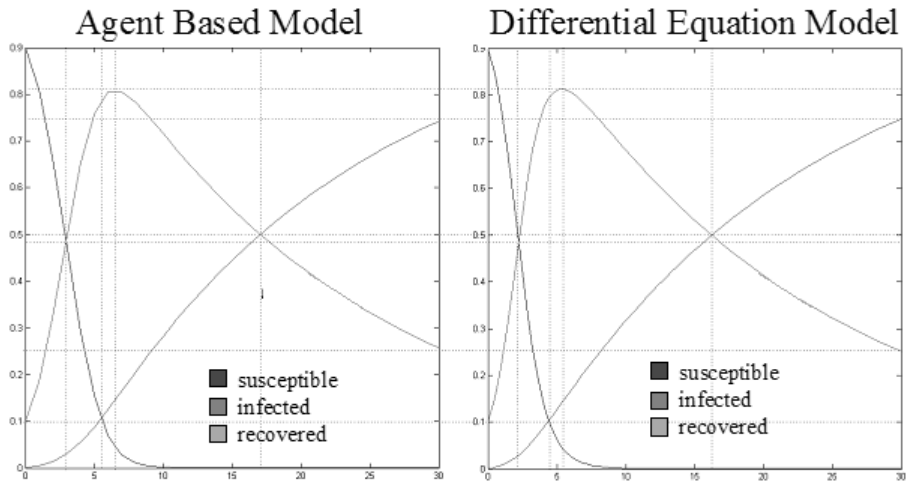

Figure 5: Direct comparison between ODE and CA model.

Figure 5 affirms the success of the explained technique with respect to small errors.

Though the restrictions for analysis of microscopic models using this strategy are very sharp, the validity of the central limit theorem for somehow weak dependent random variables/processes (see e.g. strong mixing [16],[17]), which in general poses the basis for the analysis of aggregated numbers, makes hope for the success of further analysis of aggregated observable vectors of individual-based models. Hopefully further theoretical research can help developing and validating new models on the one hand benefiting from the great flexibility of time discrete microscopic models and on the other hand profiting from fast computation properties and good validation methods of macroscopic models.

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