Alternative Approaches in Modelling of Continuous Systems

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Abstract

Due to high computational effort alternative approaches for simulating continuous systems have not been performable after their development several years ago. Nowadays computation power is high enough but as continuous models have been used for years and the solution of these is highly developed, alternative approaches still lack of acceptance. In this study we want to demonstrate the equality of 2 different modelling approaches (DE and CA) and also point out advantages of the alternative ones. As conclusion, the generality of these approaches is shown and a similar example is mentioned.

1 Introduction

Standard solutions for modelling continuous systems are differential equations. Depending on the problem ODE or PDE systems are implemented. As far as one can not solve the system analytically, implementations are solved by numerical algorithms, especially by discretization of the system. Evident for systems in physics or technical control theory, it is not so obvious to model all systems that way. Biology, Health Care or Social Sciences are only three disciplines where it is at least worth to analyse and to compare with other solutions. Because of faster computers also other approaches like Cellular Automata (near to the Agent based Simulation) can be used again (theories were often developed in the 50s and 60s of the 20^{th} Century but not computable in those days).

1.1 Motivation

A key question in developing alternative approaches for the modelling of real systems - and to obtain acceptance in academic and commercial fields - is to analyse differences and equivalencies and to show advantages and disadvantages of models. The idea is to develop a deeper understanding for a system and its model by analysing the modelling process. With a more or less simple example the equivalency of two different models shall be shown and

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furthermore the question where we can assume the end of the model and the begin of the implementation is given. In every case modelling a system and implementing such a model gives a structure to the model, which is not given in the system, also not for differential equations.
In the following example one can see different characteristics for CA models as well as for implementing differential equations. The latter ones encode the behavior in a "classic way" in spite the necessity of temporal discretization for solving them numerically. This temporal discretization is also necessary for CA but in addition, a spatial structuring of the system is also performed. Different approaches for this structuring cause different characteristics as can be observed for FHP and HPP models.
The idea is to describe the evolution of epidemic spread by means of a cellular automaton and to compare it with the classical Kermack McKendrick epidemic-model, given through a system of ordinary differential equations. The paper develops a cellular automaton model whose properties represent the ones governed by the continuous model. By showing (under certain assumptions) the equivalence of the models both in quality and quantity different properties of the systems can be described. It can be shown that the part of modelling an implementation can in a certain way be exchanged.

2 A Kermack McKendrick epidemic model

The simple SIR model for epidemic spread is based upon a system of non-linear ordinary equations [KM27]. 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 resulting system is:

$$\begin{aligned} dS(t)/dt &= -r \cdot S(t) \cdot I(t) \\ dI(t)/dt &= r \cdot S(t) \cdot I(t) - a \cdot I(t) \\ dR(t)/dt &= a \cdot I(t) \end{aligned} \tag{1}$$

where 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.

2.1 Cellular Automaton Modelling and Implementation

Cellular automata are based upon a discretization 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.
As we are studying epidemic spread, a LGCA has been chosen, allowing for the simulation of diffusion processes. LGCA are two-dimensional cellular automata with particles moving from cell to cell during each time-step of the automaton [WG01]. Therefore, the

definition of different states for the cells becomes obsolete, rather each particle can hold different states (in our case this will be susceptible, infected or recovered). Since LGCA descend from fluid dynamics, basic physical quantities like mass and momentum are conserved. Evolution (the motion of the particles) consists of propagation and collision. Concerning the structure of the chosen implementation we have to distinguish between the HPP [HPdP73] and the FHP [FHP86] 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 entering one cell from opposite directions each particle changes direction by $90°$.
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 clock-wise 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. According to [FL01] we have assigned each particle of the cell with one individual and furthermore that infection only occurs within individuals belonging to the same cell. Each particle can either be of state susceptible, infected or recovered and let S_k be the number of susceptible individuals in the entire lattice at time k. Assuming N to be the total number of nods (cells) in the lattice equality to qualitative behavior of the system of ODEs can be shown as follows.
The probability of one susceptible individual to become infected in one single time step ($k \to k+1$) is $1-(1-r)^{\frac{I_k}{N}}$ and hence the expected number of susceptible individuals who become infected is $S_k\left(1-(1-r)^{\frac{I_k}{N}}\right)$. The expected number of individuals who become recovered in a single time step is $a \cdot I_k$. For a well stirred population this yields to:

$$\begin{aligned} S_{k+1} &= S_k(1-r)^{\frac{I_k}{N}} \\ I_{k+1} &= I_k + S_k\left(1-(1-r)^{\frac{I_k}{N}}\right) - a \cdot I_k \\ R_{k+1} &= R_k + a \cdot I_k \end{aligned} \tag{2}$$

Taylor expansion for small r

$$(1-r)^{\frac{I_k}{N}} = 1 - \frac{r \cdot I_k}{N} + \frac{I_k(I_k-N)r^2}{2N^2} + \dots \tag{3}$$

keeping only the first two terms and defining $\rho_\tau(k) = \frac{\tau_k}{N}$ yields to

$$\begin{aligned} \rho_S(k+1) &= \rho_S(k) - r\rho_S(k)\rho_I(k) \\ \rho_I(k+1) &= \rho_I(k) + r\rho_S(k)\rho_I(k) - a\rho_I(k) \\ \rho_R(k+1) &= \rho_R(k) + a\rho_I(k) \end{aligned} \tag{4}$$

The LGCA has been implemented in MATLAB and the results were opposed to that solving the system of ODEs. The HPP-model is in so far not very good suited, as it comprises spurious invariants (not decisive for our simulation) and furthermore decouples individuals in two parts which will never meet (chessboard instability). Thus, if one assigns infectious particles in only one of the two populations, the other half will remain in susceptible state forever. This also influences the local building of areas with fast and severe epidemic spread and other areas which may remain untouched of epidemic spread initially.

2.2 Scenarios and Results

Figure 1 shows results for the system of ODEs and for the implemented FHP-I model. Qualitative consistency may easily be observed and hence we focus on the reasons for the quantitative differences and their implications.

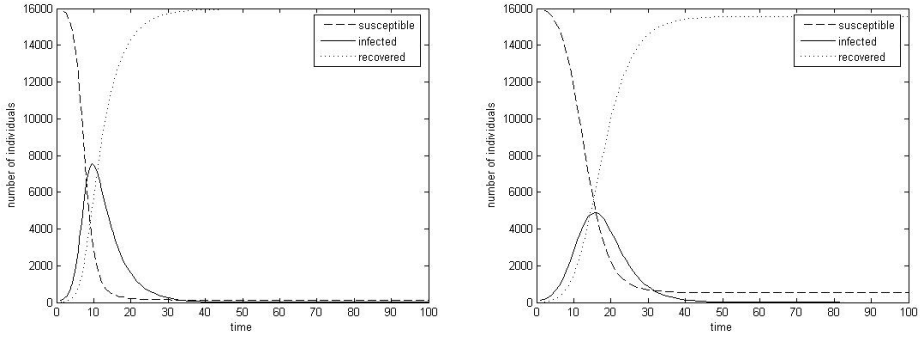

Figure 1: The left figure shows the results for the solution of the system of ODEs applying an explicit Runge-Kutta of order $(4, 5)$. The right figure shows results for the FHP-LGCA for a domain-size of 100×100, periodic boundary conditions and averaging over 10 simulation runs. Used parameters and initial conditions: $a = 0.2$, $r = 0.6$, $S_0 = 16000$, $I_0 = 100$, $R_0 = 0$

Considering the results one can see that epidemic spread seems to be slower in the CA implementation compared to the solution of the continuous model. One reason for this behaviour is the spatial inhomogeneity of the CA model. The epidemic does not spread uniformly over the domain but spatial groupings of infected individuals arise. This definitely slows down the epidemic spread. To avoid this behavior, homogeneity can be introduced by rearranging all individuals after every time step of the automaton. Thus, the LGCA model loses of its generality but the results converge to that of the continuous model which is by definition a homogeneous one.
Another reason for emerging differences must be searched in the population density (number of particles per cell) of the cellular automaton. The derivation of the equality of the approaches is only valid, if every cell holds at least one susceptible individual what means

that the domain has to be pretty packed. Otherwise, infected individuals being located in cells without susceptible individuals would not have the chance to infect any other individual what again slows down the infection process. Figure 2 shows the fairly similar results for the solution of the continuous system, the according difference equations and the FHP-LGCA with a redistribution of individuals after every time step to assure homogeneous distribution of infected individuals.

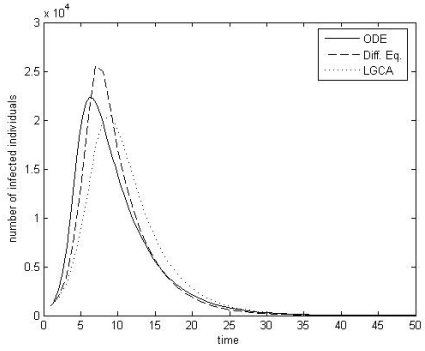

Figure 2: Comparison of results for homogeneous models. Shown is the number of infected individuals for solving the system of ODEs, the difference equations and the homogeneous FHP-LGCA again for a domain size of 100×100 and averaging over 10 simulation runs. Used parameters and initial conditions: $a = 0.2$, $r = 0.3$, $S_0 = 40000$, $I_0 = 1000$, $R_0 = 0$

The slight differences in the epidemic progression arise due to the big step size of 1 for the discrete approaches. Dividing the parameters a and r by a factor (e.g. 10) basically changes step size of the according explicit Euler method and thus leads to even better concordance. The number of infected individuals remains lower in the LGCA because the solution of the difference equations serves as upper bound for the automaton. The reason for this property is an increasing number of cells which do not contain any susceptible individuals in the course of the simulation.
Assuring homogeneous epidemic spread one must be aware to loose basic properties of the LGCA. In spite of the simple update rules of the CA the model is in a way more extensive than the system of ODEs and allows for analysing spatial epidemic spread behaviour. If one wants to gain similar results for a continuous model, systems of partial differential equations (PDEs) have to be considered. Solving systems of this form usually demands for a high computational effort.
Due to the simple evolution rules of the LGCA the computational effort for this system is quite manageable with modern computers. It only depends on the size of the considered domain. In order to obtain reasonable results, domain sizes should not be too small. The limiting factor for domain sizes in two dimensions seems to be the generation of random numbers as the update of particle states is probabilistic.

3 Discussion

With this study it is possible to show the adequacy of using alternative simulation approaches instead of classical continuous models. One has to be aware of properties, similarities but also differences of different approaches at the very beginning of the simulation cycle, the choice of a model to solve the system. So we have seen quite similar behaviour of our discrete LGCA model compared to the classical approach for a limited range of parameters. But analysing the reasons for differences beyond this ranges one can exploit the model to obtain results being more detailed and even closer to really observed behaviour. Latest studies use similar probabilistic models to study SARS spread incorporating the civilian aviation network [HBG04]. This model incorporates the benefits of different approaches and could be seen as "in-between" solution. Therefore, the population of the domain is split up in cities being comparable to our cells. In difference to the here presented model, individuals now are able to move to distal locations by plane. Once again, the relation between the probabilistic and the classical SIR model can be shown for large population sizes.
To achieve reliability and credibility for all these models in the specific science community (medical care, epidemic science...) formal and principle considerations like above have to be made. Modelling and Simulation has to be positioned as a stand alone discipline and not as a specific part of solving differential equations. Defining structures, verification, validation and knowing about doing so are main characteristics of this discipline.

References

[FHP86] U. Frisch, B. Hasslacher, and Y. Pomeau. Lattice-gas automata for the navier-stokes equation. *Phys. Rev. Lett.*, 56, 1986.

[FL01] Henryk Fuks and Anna T. Lawniczak. Individual-based lattice model for spatial spread of epidemics. *Discrete Dynamics in Nature and Society*, 6, 2001.

[HBG04] L. Hufnagel, D. Brockmann, and T. Geisel. Forecast and control of epidemics in a globalized world. *PNAS*, 101, 2004.

[HPdP73] J. Hardy, Y. Pomeau, and O. de Pazzis. Time evolution of two-dimensional model system: I. invariant states and time correlation functions. *J. Math. Phys*, 14, 1973.

[KM27] W. O. Kermack and A. G. McKendrick. A contribution to the mathematical theory of epidemics. In *Proceedings of the Royal Society of London. Series A, Vol. 115*, 1927.

[WG01] Dieter A. Wolf Gladrow. *Lattice gas cellular automata and lattice Boltzmann models: an introduction*. Springer, 2001.