Numerical simulation of agent-based modeling of spatially inhomogeneous disease dynamics

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Wolfgang Bock, Torben Fattler, Isti Rodiah, and Oliver Tse

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Introduction

In recent years much about the modeling and understanding of various types of disease spreading and epidemic behavior have been studied. In principle one can distinguish two types of models for disease spread. On the one hand there is the classical SIR-model from Kermack and McKendrick [1] which describes the time evolution of the number of susceptible (S), infected (I) and recovered (R) individuals by a system of ordinary differential equations. This model has been developed and extended exhaustively in the last 90 years. Among those extensions are the introduction of new compartments to model vector-borne diseases, see e.g. [2], delay equations to model incubation time, e.g. [3], models considering the age and wealth structure etc. Recently, models with fractional derivatives have also been considered [4]. Unfortunately, we are unable to provide a detailed account concerning this subject and refer the interested reader to [5]. A main drawback of the models described above is that they do not provide any information about the spatial spread of a disease. Nevertheless, there have been various approaches to link many different SIR-areas to obtain spatial behavior. In the SIR-model case, an advection-diffusion equation has been identified as the limiting equation, see e.g. [6]. Another approach in incorporating spatial information for the SIR-model may also be found in [7].

Although the SIR-model and all its extensions are very flexible in describing the different aspects of disease dynamics, the modeling assumptions of the disease spread is purely on the macroscopic level. However, for many different diseases the infection mechanism is only known on the microscopic, i.e., particle-to-particle or individuum-to-individuum level.

One way to consider both microscopical modeling and spatial resolution is to describe of the disease dynamics by means of an interacting particle system with suitable interaction potentials. Fundamental in this area are dynamics in so-called marked configuration spaces [8]. These techniques together with a proper scaling of the microscopic system, the so-called Vlasov scaling, have been recently used to model the dynamics of cancer cells [9]. In our approach the components of particle configurations consist of susceptible and infected/infective particles that interact with one another. One may also easily incorporate other types of particles to model recovery or short time immunity. The microscopic dynamics then results from suitable "spin-flip"-processes (particle changes the type).

This article shows first numerical results for the SIS-system without movement. The SIS-system allows infective/infected particles to recover and become susceptible again. The numerical methods are based on the analysis provided in [10].
MODEL AND SIMULATION

MICROSCOPIC MODEL

In [10] the disease spread is modeled via marked configuration spaces. The idea is to identify an individual with its position in \( \mathbb{R}^2 \) and moreover to describe an individual’s health state via marks which correspond to a susceptible (+) and an infective/infected (-) state. The evolution of the two-component system in the state space is done in such a way that at each random moment of time, a mark (+) may flip to (-), while keeping the site:

\[
(y^+, y^-) \mapsto (y^+ \setminus \{x\}, y^- \cup \{x\}), \quad x \in \gamma^+,
\]

where \( \gamma^+ \) and \( \gamma^- \) are the configurations w.r.t. the corresponding marks (+) and (-).

The Markov pre-generator \( L_{\text{flip}}^- \) for the evolution of observables \( F \in \mathcal{FP}(\Gamma^2) \) is described by

\[
(L_{\text{flip}}^- F)(y^+, y^-) := \sum_{x \in y^-} c^-(x, y^-) \left( F(y^+ \setminus \{x\}, y^- \cup \{x\}) - F(y^+, y^-) \right),
\]

where \( c^-(x, y^-) \geq 0 \) is the rate at which a (+) particle at \( x \in \gamma^+ \) flips to a (-) particle in dependence on the surrounding (-) particles. Here \( \Gamma^2 \) denotes the two-component configuration space over \( \mathbb{R}^2 \) and \( \mathcal{FP}(\Gamma^2) \) is a space of suitable test functions (cf. [10]).

**Specification of the flip rate.** In [10] the prescribed flip from a (+) particle to a (-) particle shall be interpreted as a healthy individual getting infected by the surrounding infectious individuals with a certain rate of infection \( c^+ \), which we describe in the following. With \( R \in (0, \infty) \) we denote the maximal distance of possible infection and set \( i_0 \in (0, 1] \) to be the risk of infection for a single healthy individual at direct contact with an infected one. Via the function

\[
[0, \infty) \ni r \mapsto \phi^+_0(r) := \phi^+_0(r) := \mathbb{I}_{[0, i_0]}(r) i_0 \phi_0 \in [0, \infty),
\]

we describe the risk of infection for a healthy individual depending on distance to a single infected individual, where \( \phi_0 \) is e.g. of the form given in Figure 1. For fixed \( x \in \mathbb{R}^2 \) the rate of infection for a single healthy individual at location \( x \) in the surrounding \( \gamma^- \in \Gamma^2 \) of infective individuals is given by

\[
c^-(x, y^-) := \sum_{y \in y^-} \phi(|x - y|), \quad x \in \mathbb{R}^2, \quad y^- \in \Gamma^2.
\]

We want (2) to serve as a flip rate in the Markov pre-generator (1).

**MODELING RECOVERY OF PARTICLES**

We consider a second type of evolution in our two-component system in the state space \( \Gamma^2 \). Namely, at each random moment of time, a mark (-) may flip to (+), while keeping the site:

\[
(y^+, y^-) \mapsto (y^+ \cup \{y\}, y^- \setminus \{y\}), \quad y \in \gamma^-.
\]

The corresponding Markov pre-generator \( L_{\text{flip}}^+ \) is described by

\[
(L_{\text{flip}}^+ F)(y^+, y^-) := \alpha \sum_{y \in y^-} \left( F(y^+ \cup \{y\}, y^- \setminus \{y\}) - F(y^+, y^-) \right),
\]

where \( \alpha \in [0, 1] \) is the constant rate at which a (-) particle at \( y \in \gamma^- \) flips to a (+) particle.

**MODELING THE DISEASE**

Combining to above defined Markov pre-generators we obtain a Markov pre-generator

\[
(L_{\text{dis}} F)(y^+, y^-) := (L_{\text{flip}}^+ F)(y^+, y^-) + (L_{\text{flip}}^- F)(y^+, y^-)
\]

\[
= \sum_{x \in y^-} c^-(x, y^-) \left( F(y^+ \setminus \{x\}, y^- \cup \{x\}) - F(y^+, y^-) \right)
\]

\[
+ \alpha \sum_{y \in y^-} \left( F(y^+ \cup \{y\}, y^- \setminus \{y\}) - F(y^+, y^-) \right),
\]

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which describes the evolution of a two-component system in the state space $\Gamma^2$ modeling the spread of an infectious disease.

**FIGURE 1**: The potential of infection with maximal distance of possible infection $R = 0.05$.

**NUMERICAL SIMULATION OF THE MACROSCOPIC MODEL**

In this section, we will give a brief introduction into the numerical method which was used for simulation. As a basis we have the particle system for disease spread formulated in Section 1. Therefore we consider particles in a subset of $\mathbb{R}^2$ without movement. The spread of the disease is modeled via a flip from susceptible to infected states. The flip is performed according to an infection rate. Since the infected particles influence the infection rate at a certain point in the plane, the computation of these rates is the main task during the numerical evaluation. After the infection rates are computed for every susceptible particle a uniform distributed random variable is chosen and compared to the infection rate in order to transmit the particle from the susceptible to the infested state.

Briefly the procedure of numerical implementation is as follows:

(i) Generate the state of particles and distribute the particles uniformly in space.
(ii) Calculate the infection rate or possibility of infection for each particle.
(iii) Generate random variable, then compare it with the infection rate. If random variable is smaller than infection rate, the state of particle is changed.

In the particle simulation, we consider the physical space $[0, 1] \times [0, 1] \subset \mathbb{R}^2$. We choose the risk of infection $i_0 = 0.02$ and the recovery rate $\alpha = 0.1$. The potential of infection is $\phi_R$ as given in Figure 1. The spread of the disease among a population of 3600 particles is shown in Figure 2. Susceptible individuals are depicted as white spots while infective/infected ones as black spots. In the simulation, we fix an initial region for infective individuals to be the circle with center $(0.3, 0.5)$ and radius 0.1. Figure 2 shows an equidistant spatial distribution of particles. In this particular case, we have the initial number of infected $I(0) = 112$ and the initial number of susceptible $S(0) = 3488$.

**COMPARISON WITH THE DETERMINISTIC MODEL AND THE MEAN FIELD LIMIT**

**COMPARISON OF PARTICLE AND DETERMINISTIC SIS-MODEL**

In the particle model, we can consider specific infection rate in dependence of the surrounding, which overcomes the main drawback of classical SIR-models, e.g. [1]. However just considering the number of incidents should lead to an ODE system such as in [1] for a high number of particles. On the other hand the standard ODE model assumes a uniform distribution of all particles from the beginning and neglects all behaviour coming from spatial effects. In this
FIGURE 2: System of equidistant uniformly spatial distributed particles

For this purpose we choose the risk of infection $i_0 = 0.02$, the recovery rate $\alpha = 0.1$ and the maximal distance of possible infection $R = 0.05$. First, we consider a constant potential of infection $\phi = 1$. It means a susceptible particle contacts with all infected particles that have the same rate of infection. Figure 3 shows that the particle model is in good agreement to the classical SIS-model given by the ODE system

$$\frac{d}{dt} S(t) = -\beta S(t) I(t) + \gamma I(t), \quad \frac{d}{dt} I(t) = \beta S(t) I(t) - \gamma I(t),$$

where in this case $\beta = 0.02$ and $\gamma = 0.1$.

Then, we consider the particle model with a potential of infection $\phi_R$, i.e., infections are possible only if susceptible and infectious individuals are sufficiently close to each other. We consider again an equidistant spatial distribution of particles that have the initial number of infectious $I(0) = 112$ and of susceptible $S(0) = 3488$ individuals. Figure 4 shows that the dynamics of the particle model is “slower” than the classical SIS-model. This occurs since individuals only interact locally in the particle model, while they are assumed to interact globally for the classical SIS-model. In both cases, the results show that we can compare averaged descriptions of the particle dynamics, albeit having different infection radius leads to a different infection rates.

**COMPARISON OF PARTICLE AND DETERMINISTIC SIS-MODEL AND KINETIC EQUATION**

In [10], a kinetic equation is derived via a Vlasov scaling of the particle system. This kinetic equation is a partial differential equation which describes the space-time evolution of the densities corresponding to the infected or susceptible...
FIGURE 3: Deterministic model and empirical mean of the particle model with equidistant uniform spatial distribution of particle and $\phi = 1$

FIGURE 4: Deterministic model and empirical mean of particle model with equidistant uniform spatial distribution of particle and $\phi_R$
individuums, respectively. The resulting system of equations reads

\[
\begin{align*}
\frac{d}{dt} \rho_i^+(x) &= - \left( \phi * \rho_i^-(x) \right)(x) \rho_i^+(x) + \alpha \rho_i^+(x), \\
\frac{d}{dt} \rho_i^-(x) &= \left( \phi * \rho_i^+(x) \right)(x) \rho_i^-(x) - \alpha \rho_i^-(x), \\
\rho_0^+(x) &= \rho^+(x), \quad \rho_0^-(x) = \rho^-(x), \quad x \in \mathbb{R}^2, \quad t \geq 0,
\end{align*}
\]

(4)

To validate the Vlasov-scaling we compare the kinetic equation with averaged runs of the particle simulation. For this purpose, in the particle simulation, we choose the risk of infection \( i_0 = 0.02 \), the recovery rate \( \alpha = 0.1 \) and the maximal distance of possible infection \( R = 0.05 \). We consider the number of particles \( N = 8100 \). For the kinetic system, we consider an equidistant spatial distribution of particles. We simulate a hundred runs for the particle model with random uniform spatial distribution of particles. In order to tackle the problem numerically, we partition the domain \([0, 1] \times [0, 1]\) in 100 sub-domains. The kinetic equation is solved via a standard finite differences method with \( \Delta x = \frac{1}{100} \) and \( \Delta t = 0.01 \). Figure 8 shows the difference of the empirical mean in the particle model and the mean field in \( \| \cdot \|_2 \).

FIGURE 5: Average of a hundred runs of the particle model for the infected state

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We wish Chris Bernido all the best for his 60th birthday.
FIGURE 6: Numerical solution of the kinetic equation for the infected state

FIGURE 7: Difference between the average of particle model and the mean field for the infected state in each sub-domains
FIGURE 8: Difference between the average of the particle model and the kinetic system for the infected state in the $L_2$-norm

REFERENCES


