XXVI ENMC
ENCONTRO NACIONAL DE MODELAGEM COMPUTACIONAL
XIV ECTM
ENCONTRO DE CIÊNCIA E TECNOLOGIA DE MATERIAIS
2023

TWO MODELING APPROACHES FOR AN EPIDEMIC DISEASE SPREADING PROCESS

Douglas Ferraz Corrêa[1] - douglas.correa@iprj.uerj.br
Marcelo Cargnelutti Rossato[2] - m264880@dac.unicamp.br
David A. Pelta[3] - dpelta@ugr.es
Cláudio Fabiano M. Toledo[4] - claudio@icmc.usp.br
João Frederico C. A. Meyer[2] - joni@ime.unicamp.br
Antônio José da Silva Neto[1] - ajsneto@iprj.uerj.br
Luiz Bevilacqua[5] - bevilacqua@coc.ufrj.br
[1]Universidade do Estado do Rio de Janeiro, Instituto Politécnico - Nova Friburgo, RJ, Brazil
[2]Universidade Estadual de Campinas, Instituto de Matemática, Estatística e Computação Científica - Campinas, SP, Brazil
[3]Universidad de Granada - Dept. Computer Science & A.I, Granada, Spain
[4]Universidade de São Paulo - Instituto de Ciências Matemáticas e de Computação, SP, Brazil
[5]Universidade Federal do Rio de Janeiro, RJ, Brazil

Abstract. *The present work presents two different approaches for an epidemic disease spreading process based on the already established SIR Model, the first approach uses a cellular automata approach to simulate an epidemy, the second approach usea a biflux spatial diffusion equation, based on the model developed by Bevilacqua and Galeão to simulate the same phenomena. The results are shown in figures and briefly commented. The goal is to present those two approaches that might allow for a better representation of the phenomena.*

Keywords: *SIR Model. BG Model. Cellular Automata. Disease Spreading. Anomalous Diffusion.*

1. INTRODUCTION

Different models have been proposed to better understand the transmission mechanism of infections and in the most common models the population is divided into categories, usually: Susceptible, Infected and Recovered/Removed, this model is known as SIR model. There are several approaches that based on those categories tries to simulate an epidemic scenario. The regular SIR model formulation does not take in consideration the movement of the individuals in the population of study, some studies (Cai, Y. et al., 2019; Zhu, Ren and Zhu, 2018; Wang, Xie and Kuniya, 2020) have successfully applied the diffusion-reaction equation to model this phenomena in a SIR model.

In this work we present two different ways of modeling an epidemic disease spreading process based on the SIR model: a discrete process with the aid of a cellular automata and a continuum approach with the biflux anomalous diffusion equation.

It is stated in the literature that geographic features such as rivers and mountains have been implicated as physical barriers to a raccoon population movement, which means a potential impediments to epidemic rabies (Smith et al., 2002). Bevilacqua et al. (2011; 2013) have developed an analytical formulation for anomalous diffusion, the BG Model, in which temporary retention that may be caused by a physical barrier, is taken into consideration, resulting in a fourth-order partial differential equation, besides the retention phenomena a secondary flux of recovering can be represented by the subsidiary flux of this model (Bevilacqua, 2021).

Up to our knowledge, there are no previous implementation of the BG Model in modeling a disease spreading phenomena, due to the relevance of a retention phenomena in this context and the need for understanding the infection evolution we aim to propose a model based on biflux anomalous diffusion that we hope can be used to better represent this very broad and important subject.

2. CELLULAR AUTOMATA

A cellular automaton consists of a model in which space, time and states are discrete (Mistro, 1998). In one dimension, it can be understood as a vector with a finite set of possible values that can vary over time iterations, and in two dimensions it can be understood as a matrix with the same characteristics.

Therefore, we will define a SIR model through an $N \times M$ automaton where each cell represents an individual, who can be susceptible, infected or recovered. For the one-dimensional model, we consider $M = 1$. The possible state transitions are described on Figure 1.

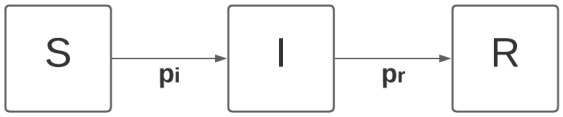

Figure 1: Diagram with the possible states and transitions.

For each iteration, all individuals interact with v other individuals who are up to d units away from them horizontally for the one-dimensional model and horizontally and vertically for the two-dimensional model. In each interaction between an infected and a susceptible individual, the latter can become infected with probability p_i. At the end of each iteration, infected individuals can recover with probability p_r.

3. BIFLUX ANOMALOUS DIFFUSION

For the sake of simplicity, in this section we shall assume the discrete and symmetric case of redistribution.

Let's consider a region in space that contains a high concentration of particles, represented by cell i in Fig. 2. In a particle spreading process with retention, a portion (α) of the particles is

retained, and the non-retained portion (β) is redistributed into neighboring cells. This process is represented in Fig. 2, where $\beta = 1 - \alpha$. When it comes to the diffusion of a infectious agent in a population β can also be related to the probability of a state transition.

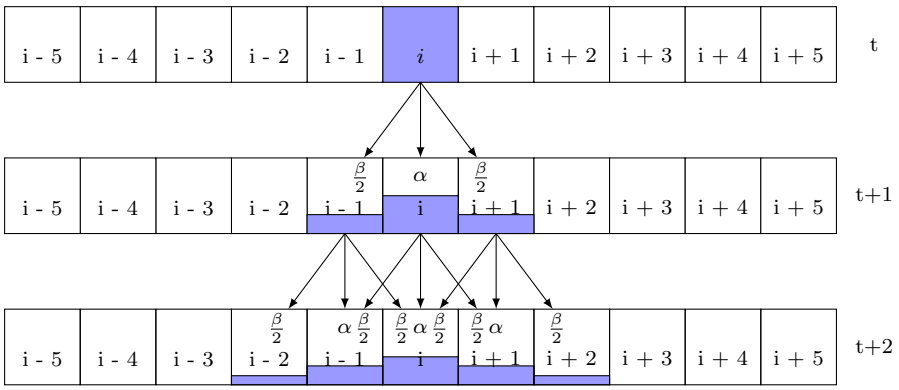

Figure 2: The discrete distribution as a function of time in the BG model for biflux anomalous diffusion with constant β.

This phenomenon in a continuous medium and with the possibility that the redistribution has a spatial dependence is governed by the following partial differential equation (Bevilacqua et al., 2011):

$$\frac{\partial p(x,t)}{\partial t} = K_2 \frac{\partial}{\partial x}\left[\beta(x)\frac{\partial p(x,t)}{\partial x}\right] - K_4 \frac{\partial}{\partial x}\left[\beta(x)(1-\beta(x))\frac{\partial^3 p(x,t)}{\partial x^3}\right] \tag{1}$$

where $p(x,t)$ represents the concentration, K_2 is the diffusion coefficient and K_4 is the reactivity coefficient.

Equation (1) is called the Biflux Anomalous Diffusion Equation, or BG Model for anomalous diffusion and it is the base of development of the more general equation. For a review of how Eq. (1) was obtained we recommend reading the analytical development of this model in Bevilacqua et al. (2011; 2013).

3.1 Biflux Theory in the Disease Spreading Context

Let's focus now on the phenomenological characteristics of the BG Model applied in an epidemic disease spread context, which is one of the approaches proposed in the present study.

According to Bevilacqua (2021) the subsidiary flux can represent a flux of state transition, that is for example, from the Infected state to the Recovered state. Based on that we start by stating the following hypothesis

(i) There's an evolution in space of the agent state;

(ii) There is a constant rate of recovering;

(iii) Rate of infected is also affected by a secondary flux of recovering;

(iv) A recovered agent never leaves this states, i.e. once recovered it becomes immune to the infection.

hypothesis i) means we are representing the phenomena with a spatial diffusion formulation, ii) means the main recovering mechanism is not the secondary flux, iii) sets the implementation of the secondary flux as a state transition flux and iv) simplifies the studied phenomena not accounting for deaths in the population with the exception for deaths due to the boundary conditions.

With the above hypothesis the proposed model is given by the following system of partial differential equations

$$\frac{\partial S}{dt} = \frac{\partial}{\partial x}\left(D_S \frac{\partial S}{\partial x}\right) - rSI \tag{2}$$

$$\frac{\partial I}{dt} = d_i \frac{\partial}{\partial x}\left(\beta(x)\frac{\partial I}{\partial x}\right) + rSI - \alpha I - K_4 \frac{\partial}{\partial x}\left[\beta(x)(1-\beta(x))\frac{\partial^3 I(x,t)}{\partial x^3}\right] \tag{3}$$

$$\frac{\partial R}{dt} = \frac{\partial}{\partial x}\left(D_R \frac{\partial S}{\partial x}\right) + aI + K_4 \frac{\partial}{\partial x}\left[\beta(x)(1-\beta(x))\frac{\partial^3 I(x,t)}{\partial x^3}\right] \tag{4}$$

here $S(x,t), I(x,t)$ and $R(x,t)$ denotes the densities of susceptible, infected and recovered individuals at location x and time t. D_S, d_I and D_R are positive diffusion coefficients, α is the rate of recovering and r is the rate of disease transmission.

Two boundary conditions are used to generate results with the proposed model, the first is the Dirichlet boundary condition given by

$$S(x,t) = S_0 \tag{5}$$

$$I(x,t) = I_0 \tag{6}$$

$$R(x,t) = R_0 \tag{7}$$

at $x = 0$ or $x = L$. With $S_0 = I_0 = R_0 = 0$ an environment where the boundary of the region is hostile for the survival of the population is represented. Due to the characteristics of the phenomena $S(x,t)$, $I(x,t)$ and $R(x,t)$ are all ≥ 0 for $\{x \in \mathrm{R} | 0 < x < L\}$ and with the divergence theorem it is trivial to show that the population decays to 0 when $t \to \infty$.

The second is the Neumann boundary condition, given by

$$\left.\frac{\partial S(x,t)}{\partial x}\right|_{x=0,L} = 0 \tag{8}$$

$$\left.\frac{\partial I(x,t)}{\partial x}\right|_{x=0,L} = 0 \tag{9}$$

$$\left.\frac{\partial R(x,t)}{\partial x}\right|_{x=0,L} = 0 \tag{10}$$

in this case the population number remains constant since there's no flux on the boundaries. The second boundary condition for Infected is defined as no subsidiary flux at the boundary.

4. RESULTS

4.1 Cellular Automata

A summary of all parameters used in the simulations is shown in Table 1.

Table 1: Parameters of the SIR model via automaton.

Parameter	Meaning
N	Number of automaton lines
M	Number of automaton columns
v	Number of interactions per individual
d	Maximum interaction distance
p_i	Probability of infection
p_r	Probability of recovery

To simulate the one-dimensional model, we consider $N = 1000$ and $M = 1$. As for the two-dimensional model, we take $N = M = 100$. In both cases, we let the infection and recovery probabilities $p_i = 0.03$ and $p_r = 0.06$ fixed, changing only the number of interactions with neighbors v and the maximum interaction distance d.

In Figure 3, the result of 300 iterations of the one-dimensional model with $v = 3$ and $d = 50$ is presented.

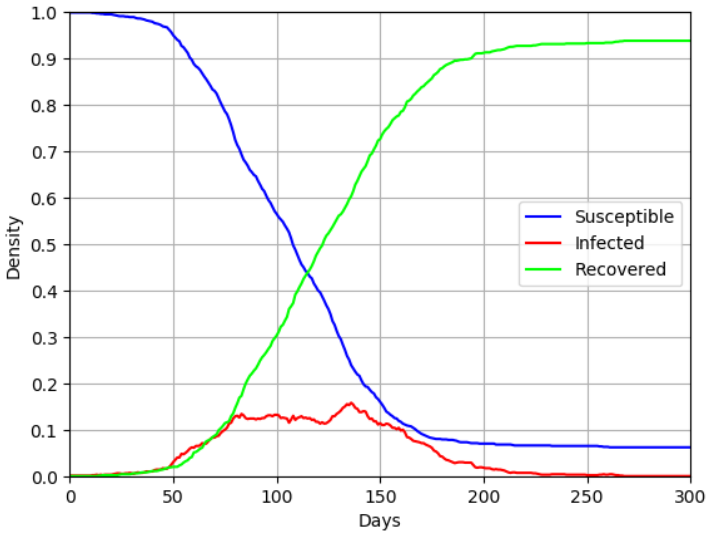

Figure 3: One-dimensional simulation results with $v = 3$ and $d = 50$.

It should be noted that the value of d must be much smaller in the two-dimensional model than in the one-dimensional model. For individuals far enough from the frontier, while in the one-dimensional model an individual has $2d$ neighbors, in the two-dimensional model this number grows to $(2d + 1)^2 - 1$.

Therefore, while in the one-dimensional model for $d = 50$ each individual had 100 neighbors, for $d = 3$ and $d = 5$ in the two-dimensional model, each individual has 48 and 120 neighbors, respectively. The result of the simulations of 400 iterations of the two-dimensional model with $d = 3$ and $d = 5$ for $v = 3$ are presented, respectively, in Figures 4 and 5.

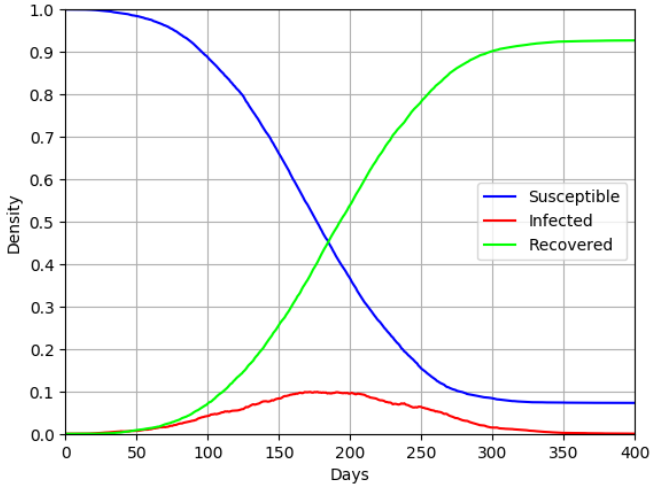

Figure 4: Two-dimensional simulation results with $v = 3$ and $d = 3$.

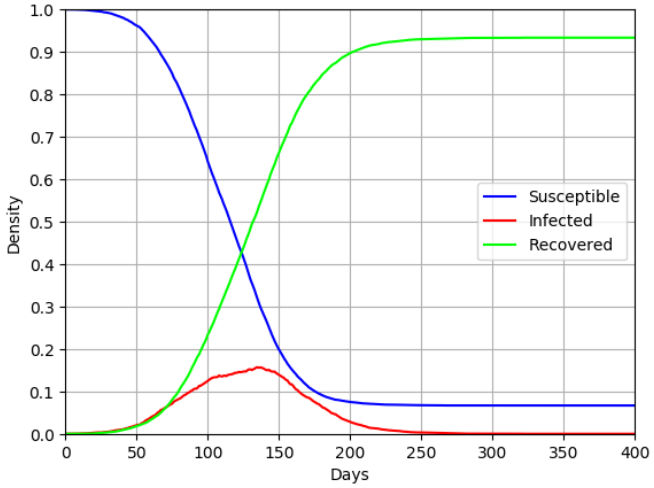

Figure 5: Two-dimensional simulation results with $v = 3$ and $d = 5$.

4.2 BG Model

The results were generated with the following parameters

Table 2: Parameters of the BG Model.

Parameter	Meaning	Value
$D_r = D_s = d_i$	Diffusivity Coefficient	0.02
r	Rate of infection transmission	0.03
α	Rate of recovery	0.06
K_4	–	10^{-4}

and the initial condition is given by (Wang, Xie and Kuniya, 2020)

$$I(x, 0) = 0.01e^{-(x-10)^2} \tag{11}$$

$$S(x,0) = 1 - I(x,0) \tag{12}$$

$$R(x,0) = 0 \tag{13}$$

Figure 6 shows the numerical solution of Eqs (3), (4) and (2), with the parameters presented in Table 2 and the no flux at the boundaries condition.

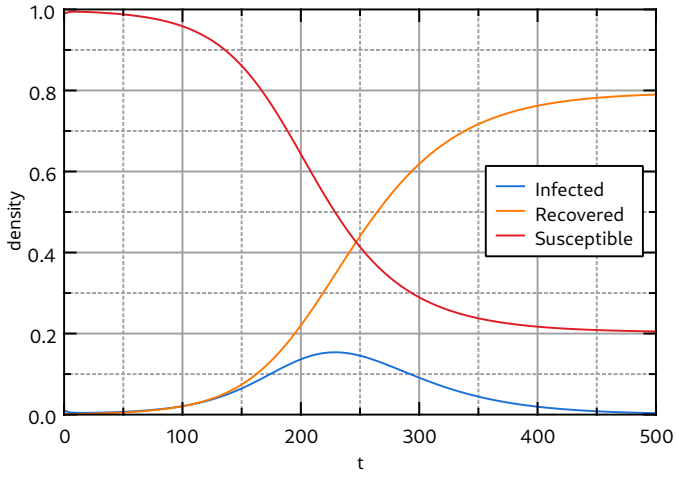

Figure 6: Solution of the proposed BG Model for a disease spreading process with $\beta = 0.5$ and constant population.

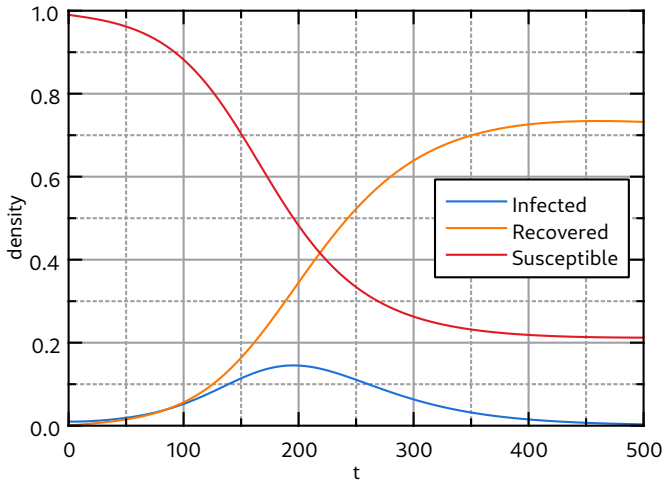

Figure 7: Solution of the proposed BG Model for a disease spreading process with $\beta = 0.5$ and decaying population.

Figure 7 shows the result with the deadly boundary condition, this implies the population number decays with time. Interestingly, in this case the Infected peak happens faster. The actual reasoning for this still needs more research. The computational cost of the BG Model implementation on a i7-12700H with NVIDIA RTX 3060 was around $16ms$, which is a reasonable time considering the relevance of the topic.

Comparing Figures 3 and 6 we can see two main differences, the first one is the density of people in the recovered and susceptibles categories are not the same, this happens because no correlation between the models parameters were used and both were chosen based on previous published work in the literature for each model. The second difference is the fact that the

Cellular Automata method was able to predict some degree of variations, almost like random category transfer happening during the simulation.

5. CONCLUSIONS

The present work shows two different approaches to modeling a disease spreading process, the first approach uses a SIR model with cellular automata while the second approach includes a subsidiary flux to represent a transition between agent state in a novel approach that we hope will allow for a better refinement of the phenomena, both approaches were able to represent the disease spreading process. When faster results are needed the BG Model proved itself less computational costly and with the capacity of representing a secondary flux of state transition, while the celular automata showed that it is a method capable of capturing small and random variations in the state transition that makes a difference when the population number is small, the difference becomes unnoticeable in the 2D scenario due to the bigger number of agents involved.

For future works we aim to introduce the retention effect on the cellular automata, while also evolving the theory behind the application of the BG Model in an epidemic process and, hopefully, find a correlation of parameters between both methods.

Acknowledgements

This study was financed in part by the Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - Brasil (CAPES) - Finance Code 001, Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) and Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (FAPERJ). D. Corrêa acknowledges CAPES also for the scholarship for his stay in the University of Granada (Grant CAPES/PrInt No. 88887.717186/2022-00). D. Pelta acknowledges support from projects PID2020-112754GB-I00, MCIN /AEI/ 10.13039 /501100011033 and FEDER/Junta de Andalucía-Consejería de Transformación Económica, Industria, Conocimiento y Universidades / Proyecto (BTIC-640-UGR20)

REFERENCES

Bevilacqua, L., Galeão, A. C. N. R., Costa, F.P., A new analytical formulation of retention effects on article diffusion process. An Acad Bras Cienc. 83 (2011) 1443-1464.

Bevilacqua L., Galeão, A. C. N. R., Simas, J. G., Doce, A. P. R., A new theory for anomalous diffusion with a bimodal flux distribution, J. Brazilian Soc. Mech Sci Eng. 35 n.4 (2013) 1-10.

Bevilacqua L., Processos de difusão de partículas da mesma natureza em dois estados de energia diferentes, Revista Brasileira de Ensino de Física, vol 43 (2021).

Mistro, D. C. *Modelos para dispersão de abelhas africanizadas: um zoom matemático*. 1998. 182 f. Tese (Doutorado em Matemática Aplicada) – Unicamp, Campinas, 1998.

Cai, Y. Lian, X., Peng, Z., Wang W., Spatiotemporal transmission dynamics for influenza disease in a heterogenous environment. Nonlinear Anal. Real World Appl. 46, 178–194 (2019)

Wang, J., Xie, F., Kuniya, T.: Analysis of a reaction–diffusion cholera epidemic model in a spatially heterogeneous environment. Commun. Nonlinear Sci. Numer. Simul. 80, 104951 (2020)

Zhu, D., Ren, J., Zhu, H.: Spatial-temporal basic reproduction number and dynamics for a dengue disease diffusion model. Math. Methods Appl. Sci. 41(14), 5388–5403 (2018)

David L. Smith and Brendan Lucey and Lance A. Waller and James E. Childs and Leslie A. Real, Predicting the spatial dynamics of rabies epidemics on heterogeneous landscapes, Proceedings of the National Academy of Sciences, v. 99, 3668–3672 (2002).