Stochastic Compartment Model of Epidemic Spreading in Complex Networks with Mortality and Resetting

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***Abstract*—We propose an epidemic compartment model, which includes mortality caused by the disease, but excludes demographic birth and death processes. Individuals are represented by random walkers, which are in one of the following states (compartments) S (susceptible to infection), E (exposed: infected but not infectious corresponding to the latency period), I (infected and infectious), R (recovered, immune), D (dead). The disease is transmitted with a certain probability at contacts of I to S walkers. The compartmental sojourn times are independent random variables drawn from specific (here Gamma-) distributions. We implement this model into random walk simulations. Each walker performs an independent simple Markovian random walk on a graph, where we consider a Watts-Strogatz (WS) network. In order to mimic the effect of long-distance travelers, we subject the simple Markov walks to stochastic resetting, which means that the walkers in each time step are relocated to any node of the network with a certain probability. Only I walkers may die. For zero mortality, we prove the existence of an endemic equilibrium for basic reproduction number $\mathcal{R}_0 > 1$ and for which the disease free (globally healthy) state is unstable. We explore the effects of long-range-journeys (stochastic resetting) and mortality. Our model allows for various interpretations, such as certain chemical reactions, the propagation of wildfires, and in population dynamics.**

Keywords – Compartment model; mortality; random walks; complex graphs; resetting; population dynamics.

I. Introduction

Sudden outbreaks of epidemics are recurrently threatening humanity and represent major challenges for human societies and public health services. Since the breakout of the COVID-19 pandemic, epidemic models have attracted considerable attention. More than ever, there is a need of basic understanding of the underlying mechanisms of epidemic propagation. In many cases persistent oscillatory and quasi-periodic behavior or spontaneous outbursts, features, are observed. One of the first works tackling the issue of oscillatory dynamics is the one by Soper [1], which appeared a century ago in the literature. So-called compartmental models, where the individuals of a population are divided according to their states of health, have become popular in the field of epidemic modeling. The first model of this type was introduced a century ago in the seminal work of Kermack and McKendrick [2], where individuals are in one of the states (compartments) susceptible (to infection) - S, infected and infectious - I, recovered (immune) - R. While standard SIR models are able to capture essential features of some common infectious diseases such as mumps, measles, rubella and others, they have revealed to be unable to describe above-mentioned oscillatory and quasi-periodic behaviors. The classical SIR model has been generalized in many directions

[3]-[6] and consult [7] for a model related to the context of COVID-19 pandemic.

In the present paper, we explore the spreading of a disease by combining a microscopic multiple random walkers approach with a compartment model exhibiting random compartmental sojourn times. In this work we close a gap in existing models, and establish an exact stochastic system of evolution equations describing the transitions among the compartments (see (2) and (3)) from which explicit, in general non-Markovian convolutional evolution equations can be obtained, by averaging over the involved random variables. These equations are general and beyond existing Markovian models when non-exponentially distributed compartmental sojourn times are assumed. Our formulation allows for arbitrary compartmental sojourn time distributions including time-fractional ones, and also incorporate a stochastic notion of mortality into the dynamics. This novel stochastic approach opens a large field to tackle the spreading dynamics of a wide range of real-world diseases, with and without mortality. Moreover, our model allows for further generalizations, such as inclusion of demographic effects originating from natural birth and death processes. Such generalizations may be of interest for classes of diseases with a "slow" dynamics evolving on time-scales (such as decades) where changes in the population number become relevant. A prominent example is Hansen's disease (leprosy), which exhibits extremely long latency periods (around five years).

By conducting a linear stability analysis, we prove for zero mortality that the disease free state is stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$ ($\mathcal{R}_0$ denotes the basic reproduction number), where a globally stable endemic state emerges whenever the compartment sojourn times have finite means, for which we obtain explicit formulas (see relations (6)). These formulas generalize the well-known classical results of Kermack and McKendrick [2] to arbitrary distributions of compartmental sojourn times and multiple compartments.

Let us give a brief sketch of the state of the art and some related works, where we confine the discussion to recent developments with focus on epidemic spreading models in various kinds of random networks. In order to relate macroscopic compartment models to microscopic dynamics, epidemic spreading has been studied in random graphs with emphasis on the complex interplay of the network topology and spreading features [8]-[11]. Further works consider stochastic compartmental models combined with random walk approaches [12]-[19] including non-exponentially distributed compartmental sojourn times leading to non-Markovian models [20]-[24]. An increasing number of works consider epidemic propagation on networks. In reference [19], involving generalized Laplacian operators, spreading features are thoroughly analyzed, where an upper bound for the epidemic SIS threshold for any graph topology is obtained. Related works to our model can be found in references [17], [21]-[24] and [34].

The remainder of our paper is organized as follows. In section II we introduce a mean field picture of our compartment model with the transition pathways among the compartments, where we establish novel stochastic compartment evolution equations with mortality. Special attention is given to the analysis of the case of zero mortality, for which we derive explicit formulas of the endemic state as well as the condition of its existence. Section III is devoted to the outline of the multiple random walkers approach. Inclusion of stochastic resetting into the random walks enables us to study the effect of long-distance travelers. In Section IV we summarize the main results of the present stage of this project as far presented in this paper. Finally, we conclude our ongoing project in section V and discuss future directions together with some possible generalizations of our model.

II. Mean field compartment model

Here, we study the large class of infectious diseases with direct transmission among individuals, which also exhibit mortality. The large list of these diseases includes Influenza, COVID-19, Chickenpox, Hepatitis A, Ebola, and many others. We propose a compartment model, in which individuals ("random walkers") are in one of the following states (compartments) S (susceptible to infection), E (exposed: infected but not infectious corresponding to the latency period), I (infected and infectious), R (recovered, immune), and D (dead). We assume random waiting times t_E, t_I, t_R in compartments E, I, R. The delay time t_E is the latency period, i.e., the time between the moment of infection (transition S to E) and outbreak of the disease (transition E to I). t_I is the duration of the disease (infected and infectious state) during which the walker can infect S walkers and die. We introduce a random survival time t_M measured from the moment of transition into compartment I (outbreak of the disease). The walker survives if $t_M > t_I$ and dies otherwise (when $t_M < t_I$). A surviving walker passes through the SEIRS pathway

$$\mathrm{S} \to \mathrm{E} \to \mathrm{I} \to \mathrm{R} \to \mathrm{S}.$$

A walker which dies from the disease (i.e., $t_M < t_I$) runs through the SEID pathway

$$\mathrm{S} \to \mathrm{E} \to \mathrm{I} \to \mathrm{D}.$$

For the infection rate, we assume a simple bilinear function inspired from the mass-action law

$$\mathcal{A}(t) = \beta S(t) J(t), \tag{1}$$

where $\beta > 0$ is a constant, which contains the information on the probability of infection in a contact of an S and I walker and features of the random walks. The stochastic formulation of the evolution equations of the compartmental population

fractions reads

$$\begin{aligned}
\frac{dS(t)}{dt} &= -\mathcal{A}(t) + \big\langle \mathcal{A}(t-t_E-t_I-t_R)\Theta(t_M-t_I)\big\rangle \\
&\quad +J_0\big\langle \delta(t-t_I-t_R)\Theta(t_M-t_I)\big\rangle \\
&\quad +R_0\big\langle \delta(t-t_R)\big\rangle \\
\frac{dE(t)}{dt} &= \mathcal{A}(t) - \big\langle \mathcal{A}(t-t_E)\big\rangle \\
\frac{dJ(t)}{dt} &= \big\langle \mathcal{A}(t-t_E)\big\rangle - \big\langle \mathcal{A}(t-t_E-t_I)\Theta(t_M-t_I)\big\rangle \\
&\quad -J_0\big\langle \delta(t-t_I)\Theta(t_M-t_I)\big\rangle - \frac{dD(t)}{dt} \\
\frac{dR(t)}{dt} &= \big\langle \mathcal{A}(t-t_E-t_I)\Theta(t_M-t_I)\big\rangle \\
&\quad +J_0\big\langle \delta(t-t_I)\Theta(t_M-t_I)\big\rangle \\
&\quad -J_0\big\langle \delta(t-t_I-t_R)\Theta(t_M-t_I)\big\rangle \\
&\quad -\big\langle \mathcal{A}(t-t_E-t_I-t_R)\Theta(t_M-t_I)\big\rangle \\
&\quad -R_0\big\langle \delta(t-t_R)\big\rangle
\end{aligned} \tag{2}$$

and the mortality rate

$$\frac{dD(t)}{dt} = J_0\big\langle \delta(t-t_M)\Theta(t_I-t_M)\big\rangle + \big\langle \mathcal{A}(t-t_E-t_M)\Theta(t_I-t_M)\big\rangle. \tag{3}$$

$S(t), E(t), J(t), R(t), D(t)$ denote, the fractions of the susceptible, exposed, infected, recovered (immune), and dead walkers populations, where $S(t)+E(t)+J(t)+R(t)+D(t)=1$. We consider initial conditions $S(0)=S_0$, $J(0)=J_0$, $E(0)=0$, $R(0)=R_0$, $D(0)=0$ and assume that the disease occurs at $t=0$ for the first time with a few infected walkers J_0, no exposed and dead walkers, and possibly some immune (vaccinated) walkers R_0, allowing to explore effects of vaccination. $\Theta(..)$ indicates the Heaviside unit step function, $\delta(..)$ the Dirac's δ-distribution, and $\big\langle \dots \big\rangle$ stands for averaging with respect to the contained (independent) random variables $t_E, t_I, t_R, t_M > 0$ drawn from probability density functions (PDFs)

$$Prob(t_{E,I,R,M} \in [\tau, \tau+\mathrm{d}\tau]) = K_{E,I,R,M}(\tau)\mathrm{d}\tau$$

indicating the probabilities that $t_{E,I,R,M} \in [\tau, \tau+\mathrm{d}\tau]$. The following averaging rule applies

$$\big\langle f(t_{E,I,R,M})\big\rangle = \int_0^\infty f(\tau)K_{E,I,R,M}(\tau)\mathrm{d}\tau. \tag{4}$$

For causal functions as in (2) this yields convolutions

$$\big\langle A(t-t_{E,I,R,M})\big\rangle = \int_0^t A(t-\tau)K_{E,I,R,M}(\tau)\mathrm{d}\tau.$$

With these relations, the evolution equations (2) and (3) can be averaged taking convolution forms (see [22, 23] for related details).

a) Zero mortality – endemic equilibrium: The limit of immortality of the walkers is retrieved from (2) for $t_M=\infty$ thus $\Theta(t_M-t_I)=1$ and $\Theta(t_I-t_M)=0$ and therefore $\frac{d}{dt}D(t)=0$. Then equations (2) read

$$\begin{aligned}
\frac{dS(t)}{dt} &= -\mathcal{A}(t) + \big\langle \mathcal{A}(t-t_E-t_I-t_R)\big\rangle \\
&\quad +J_0\big\langle \delta(t-t_I-t_R)\big\rangle + R_0\big\langle \delta(t-t_R)\big\rangle \\
\frac{dE(t)}{dt} &= \mathcal{A}(t) - \big\langle \mathcal{A}(t-t_E)\big\rangle \\
\frac{dJ(t)}{dt} &= \big\langle \mathcal{A}(t-t_E)\big\rangle - \big\langle \mathcal{A}(t-t_E-t_I)\big\rangle - J_0\big\langle \delta(t-t_I)\big\rangle \\
\frac{dR(t)}{dt} &= \big\langle \mathcal{A}(t-t_E-t_I)\big\rangle + J_0\big\langle \delta(t-t_I)\big\rangle \\
&\quad -J_0\big\langle \delta(t-t_I-t_R)\big\rangle \\
&\quad -R_0\big\langle \delta(t-t_R)\big\rangle - \big\langle \mathcal{A}(t-t_E-t_I-t_R)\big\rangle
\end{aligned} \tag{5}$$

with $S(t)+E(t)+J(t)+R(t)=1$. In order to derive the endemic equilibrium, it is convenient to work with Laplace transformed (5), where $\hat{f}(\lambda)=\int_0^\infty f(t)e^{-\lambda t}\mathrm{d}t$ is the LT of $f(t)$. We use the limit value theorem $f(\infty)=\lim_{\lambda\to 0}\lambda\hat{f}(\lambda)$ to obtain the constant asymptotic values of the endemic equilibrium as [22]

$$\begin{aligned}
S_e &= \frac{1}{\mathcal{R}_0}, \qquad\qquad \mathcal{R}_0 = \beta\langle t_I\rangle, \\
E_e &= \frac{\mathcal{R}_0-1}{\mathcal{R}_0}\frac{\langle t_E\rangle}{\langle T\rangle} \\
J_e &= \frac{\mathcal{R}_0-1}{\mathcal{R}_0}\frac{\langle t_I\rangle}{\langle T\rangle} \\
R_e &= \frac{\mathcal{R}_0-1}{\mathcal{R}_0}\frac{\langle t_R\rangle}{\langle T\rangle}.
\end{aligned} \tag{6}$$

The endemic equilibrium is independent of the initial conditions, where $\langle T\rangle = \langle t_E+t_I+t_R\rangle$ and $\mathcal{A}_e = \frac{\mathcal{R}_0-1}{\mathcal{R}_0}\frac{1}{\langle T\rangle}$. (6) exists for $\mathcal{R}_0=\beta\langle t_I\rangle > 1$, which also is the spreading condition of the disease when $S_0=1$ is considered. $\mathcal{R}_0$ indeed is the basic reproduction number. In (6) $\big\langle t_{E,I,R}\big\rangle = \int_0^\infty \tau K_{E,I,R}(\tau)\mathrm{d}\tau$ stand for the mean compartmental sojourn times, assuming here their finiteness. Relations (6) generalize the classical result [2] to arbitrary waiting time distributions and multiple compartmen Here we consider Gamma distributed waiting times due to the high flexibility of Gamma distributions to adopt the behaviors of a wide range of real world diseases (see e.g., [22], [23] for details).

III. Random walk simulations with resetting

We assume that each walker navigates for discrete times independently on an ergodic network [25], [26]. In order to describe the random walk of each walker, we denote with $i=1,\dots N$ the nodes of the network and introduce the symmetric $N\times N$ adjacency matrix (A_{ij}), where $A_{ij}=1$ if the pair of nodes i,j is connected by an edge, and $A_{ij}=0$ if the pair is disconnected. Further, we assume $A_{ii}=0$ to avoid self-connections of nodes. We restrict our analysis to undirected networks, where edges have no predefined direction and the adjacency matrix is symmetric. The degree k_i of a node i counts the number of its neighbor nodes (connected with i by edges). Each walker performs independent Markovian steps

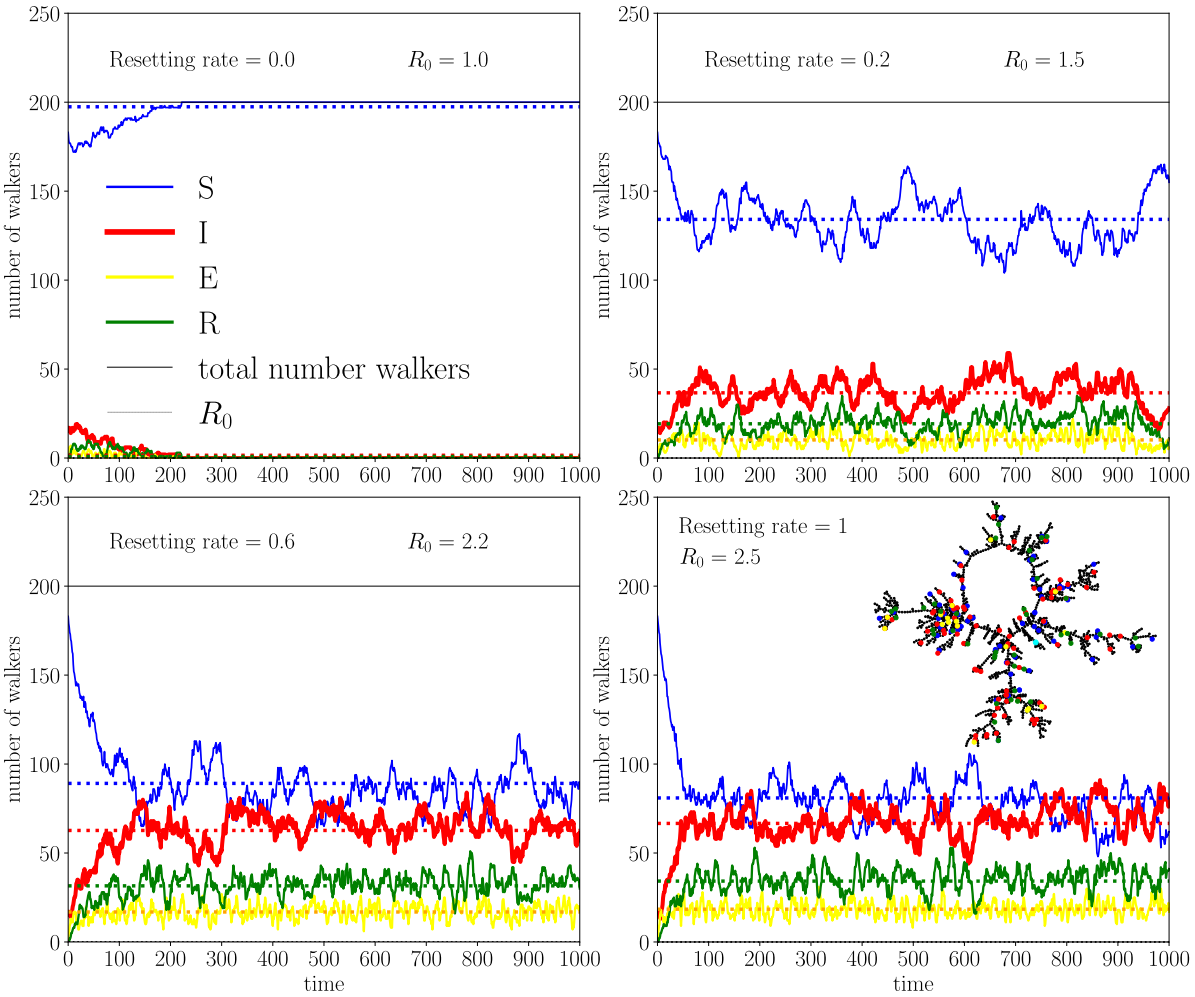

Figure 1. Effect of resetting on the spreading for zero mortality with emergence of endemic states in a large world Watts-Strogatz (WS) network (generated by the PYTHON NetworkX library) of 1500 nodes with 200 walkers. Colors indicate the compartments of walkers. Compartmental sojourn times are Gamma distributed with $\langle t_I \rangle : \langle t_R \rangle : \langle t_E \rangle = 4 : 2 : 1$, which can be identified in the plots, corroborating (6) for all considered resetting rates p. The infection state of the graph at runtime 1000 is exhibited by the inset. The basic reproduction number $\mathcal{R}_0$ is monotonously increasing with p.

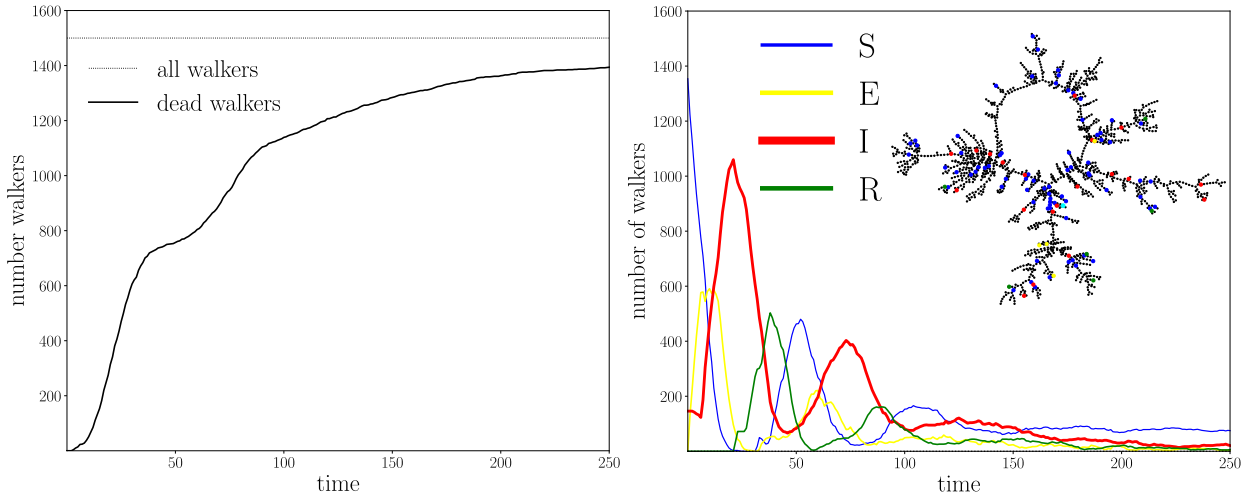

Figure 2. Spreading with high mortality and resetting in the WS graph of Figure 1 for resetting probability $p = 0.6$. The inset shows the infection state of the graph at runtime $t = 250$ (D walkers are invisible) with eventually only about 100 survived walkers out of 1500. We use the same color code as in Figure 1. The right frame depicts the epidemic wave and left frame the evolution of the cases of death.

between connected nodes. The steps from a node i to one of its $k_i = \sum_{j=1}^{N} A_{ij}$ neighbor nodes are chosen with probability $1/k_i$, leading for all Z walkers to the same transition matrix, namely [26]-[28]

$$\Pi(i \to j) = \frac{A_{ij}}{k_i}, \qquad z = 1, \ldots, Z, \qquad i, j = 1, \ldots, N, \quad (7)$$

which is by construction row-normalized $\sum_{j=1}^{N} \Pi(i \to j) = 1$. In addition, we relocate ('reset') the walkers at each time instant to randomly chosen nodes with a certain probability p. This modifies the transition matrix of the steps for each walker to

$$W_{i \to j} = q\Pi(i \to j) + pR_j, \qquad p + q = 1, \qquad (8)$$

where in our simulations we have uniform resetting probabilities $R_j = \frac{1}{N}$ to each node of the network. (8) introduces long-range journeys into the random walks, and the spreading behavior is modified compared to local walks (7). Stochastic resetting (SR) is a fundamental process in nature where dynamical systems are reset to the initial or randomly chosen states. SR occurred only a decade ago in the literature [29] and has meanwhile launched a myriad of models and opened a wide interdisciplinary field, e.g., [30]-[33] (and many others).

IV. Results and discussion

In Figure 1, we depict the simulated time evolution of compartmental populations (absolute numbers of walkers) under the influence of resetting for some values of relocation probability p and zero mortality. The independent motion of each walker is governed by (8). The parameters are such that no spreading occurs without resetting with $\mathcal{R}_0 = 1$ where the disease is eventually extinct (left upper frame). Increasing p introduces more long-range displacements where the number of contacts of S and I walkers and hence infection rates with basic reproduction numbers $\mathcal{R}_0$ increase. The disease is spreading from $p = 0.2$ with monotonously increasing endemic values E_e, J_e, R_e and $\mathcal{R}_0$ with p. Our simulations corroborate (6), i.e., the ratios of the observed endemic values correspond to the ratios of mean compartmental sojourn times. We determined $\mathcal{R}_0$ in the simulations from the first equation of (6).

We assumed in our mean field model, a simple mass-action law for the infection rates (1), leading with (5) to the endemic states (6). These endemic values are in excellent agreement with the large-time asymptotics obtained from the random walk simulations (see Figure 1). This remains true when the random walks of the individuals are subjected to resetting, which in the large time limit affects only the macroscopic transmission coefficient β. These observations suggest that random walks indeed offer suitable microscopic pictures of the corresponding spreading dynamics.

Animated simulation-videos on Watts-Strogatz graphs can be launched online by clicking on the slanted text for a case *without mortality and no resetting* (see (5)). A further animation video of the spreading under resetting ($p = 0.6$) on the graph of Figure 1 and similar parameters *includes mortality* (see (2), (3)). Simulation (Python) codes with parameters and further details can be obtained upon request or consult our website *supplementary materials*.

The present model can be generalized in several directions, for instance, to vector-borne transmission pathways [23] or assuming non-monotonous infection rates (different from simple mass-action-laws) for which under certain conditions the endemic equilibrium exhibits bifurcations, allowing for emergence of chaotic attractors [34].

The present paper reflects a snapshot of our work in progress. In the next steps, we analyze the evolution equations (2), (3) with mortality in order to derive the effective reproduction number R_M with mortality. Performing a linear stability analysis around the healthy initial state S_0, R_0, which consists of a fraction of susceptible walkers $S(0) = S_0 = 1 - R_0$, and some immune (vaccinated) walkers $R(0) = R_0$ leads to the spreading condition (instability of the initial state) for $\mathcal{R}_M > 1$. As a preliminary result of this follow-up analysis, we report here that the 'effective reproduction number' of the disease with mortality and presence of some immune walkers yields

$$\begin{aligned} \mathcal{R}_M = \quad & \beta(1 - R_0) \int_0^\infty \Phi_M(t)\Phi_I(t)\mathrm{d}t \\ & = \beta(1 - R_0)\big\langle min(t_M, t_I)\big\rangle \\ & < \beta \int_0^\infty \Phi_I(t)\mathrm{d}t = \beta\big\langle t_I\big\rangle = \mathcal{R}_0, \end{aligned}$$

where $\mathcal{R}_0$ is the basic reproduction number without mortality and no immune walkers at $t = 0$. In the immortal limit ($t_M \to \infty$, $\Phi_M(t) \to 1$) one has $\mathcal{R}_M \to \mathcal{R}_0$ (in absence of immune walkers $R_0 = 0$). This relation contains the mean of the "true" sojourn time $min(t_M, t_I)$ in compartment I and the persistent probabilities $\Phi_{M,I}(t) = \big\langle \Theta(t_{M,I} - t)\big\rangle = 1 - \int_0^t K_{M,I}(\tau)\mathrm{d}\tau$. Moreover, it contains the probability that a walker is in compartment I (infected and infectious and alive) $\Phi_M(t)\Phi_I(t) = \big\langle \Theta(t_M - t)\Theta(t_I - t)\big\rangle = \big\langle \Theta(min(t_M, t_I) - t)\big\rangle$. The next steps in this analysis will include the investigation of the large time asymptotics of the spreading dynamics with mortality, among other directions, which we will briefly outline subsequently.

V. Conclusion and future work

We proposed a multiple random walkers epidemic compartment model, which accounts for mortality: An infected walker may die during the period of its infection. We excluded demographic birth and death processes. The compartmental sojourn times were considered to be independent random variables drawn from specific (here Gamma-) distributions. By including stochastic resetting into the random walks, in which walkers are relocated to random positions, we are able to mimic the effects of long-range voyages on the spread of the disease. By considering zero mortality, we observed that the macroscopic compartment model (endemic states (6)) remains true for any resetting rate p, where the macroscopic transmission coefficient β is monotonously increasing with the resetting rate. Increasing numbers of long-range journeys

may drive the basic reproduction number to values above one, which launches the spreading of the disease. It follows that measures reducing long-range voyages can be an effective way to block the propagation of an epidemic. The results of the simulations suggest that in all cases, above equations (6) for the endemic states remain valid and capture well the large time asymptotics.

Finally, we conclude that our approach of multiple random walkers navigating independently in a complex network is a powerful tool to capture the microscopic dynamics of epidemic spreading. We included stochastic resetting into the random walks mimicking long-range voyages of the walkers and found that the basic reproduction number increases monotonously with the resetting rate p. The message of this result clearly is that prohibiting to a certain extend traveling in epidemic contexts can be effective to prevent spreading of the disease.

As mentioned, the next steps will include an asymptotic analysis of the spreading dynamics with mortality. To that end, we will investigate the evolution equations (2), (3) in the Laplace space and use the limit value theorem to determine the large time asymptotic state. This infinite time limit is supposed to be a disease free state, containing only susceptible walkers (walkers that survived the epidemic wave) and dead walkers. Also, the effect of resetting on the mortality of walkers (infinite time limit of the fraction of dead walkers) will be explored analytically and numerically in details. For a related analysis of a mortal vector borne disease, we refer to a recent model [23].

A further promising direction is to account for infection rates beyond the present mass-action law (1) by including information of the network topology and the random walk. Introduction of individual navigation rules for specific walkers can be of interest as well.

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