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Critical regimes driven by recurrent mobility patterns of reaction–diffusion processes in networks

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Reaction–diffusion processes[1] have been widely used to study dynamical processes in epidemics[2–4] and ecology[5] in networked metapopulations. In the context of epidemics[6], reaction processes are understood as contagions within each subpopulation (patch), while diffusion represents the mobility of individuals between patches. Recently, the characteristics of human mobility[7], such as its recurrent nature, have been proven crucial to understand the phase transition to endemic epidemic states[8,9]. Here, by developing a framework able to cope with the elementary epidemic processes, the spatial distribution of populations and the commuting mobility patterns, we discover three different critical regimes of the epidemic incidence as a function of these parameters. Interestingly, we reveal a regime of the reaction–diffussion process in which, counter-intuitively, mobility is detrimental to the spread of disease. We analytically determine the precise conditions for the emergence of any of the three possible critical regimes in real and synthetic networks.

Epidemic processes in complex networks have attracted the attention of physicists during the last two decades[6]. Several outstanding results have been the consequence of a mathematical analysis that borrows ideas from other physical processes. In particular, epidemic spread in networks can be thought of as reaction–diffusion processes, referring to the change of the concentration of two or more types of element: local reactions in which the elements are transformed into each other, and diffusion that causes the substances to spread out over the available space. In epidemiology, the elements in play are the subjects (humans or animals), characterized by their states in the evolution of the sickness (for example, susceptible, infected, recovered and so on). In complex networks, the reaction phase corresponds to the infections produced by the local interaction of subjects within a subpopulation (node), and the diffusion phase corresponds to their mobility through the network according to the connections (links) between nodes.

This approach to epidemic spread using reaction–diffusion processes, usually referred to as metapopulation models, has been largely studied in network science[10–14]; however, several challenges remain open[15,16]. The most representative of these challenges, from a physicist's perspective, is to complement large-scale agent-based simulations[17–19], by deriving models amenable to mathematical analysis[20] that capture the influence of human behaviour[21] and the existence of complex social structures.

Our proposal to fill this gap is to formulate a general 'microscopic' Markovian model describing the metapopulation reaction–diffusion dynamics. We start by analysing, at the individual level, the probabilities of infection in the scope of the susceptible–infected–susceptible (SIS) epidemic model[12]. We denote as λ and μ the infection and recovery probabilities respectively. This way, a susceptible (S) individual is infected with probability λ when interacting with an infected (I) subject. In turn, infected (I) individuals become susceptible (S) again with probability μ. Note that if there is no recovery (that is, $\mu=0$), the phenomenology we will report does not hold.

Motivated by the recurrent (commuting) nature of most urban and regional movements reported and modelled[8,9], let us explain two key assumptions of our model. First, we assume that each individual is associated with a certain subpopulation (her residence). Second, to incorporate the recurrence of human mobility patterns, we force all of the agents who have decided to move from their residence to return to them after each time step. This way, given N subpopulations (nodes), the variable $\rho_i(t)$ ($i=1,\ldots,N$) denotes the fraction of infected individuals associated with node i at time t. The time evolution of $\rho_i(t)$ is as follows:

$$\rho_i(t+1)=(1-\mu)\rho_i(t)+(1-\rho_i(t))\Pi_i(t) \quad (1)$$

where the first term in the right-hand side denotes the fraction of infected individuals associated with i that do not recover and the second term accounts for the fraction of healthy individuals associated with i that pass to infected at time $t+1$. In this second term, $\Pi_i(t)$ is the probability that a healthy individual associated with node i becomes infected at time t. This probability reads:

$$\Pi_i(t)=\left(1-p_{\mathrm{d}}\right)P_i(t)+p_{\mathrm{d}}\sum_{j=1}^{N}\frac{W_{ij}}{\sum_{l=1}^{N}W_{il}}P_j(t) \quad (2)$$

where p_{d} denotes the probability of moving and W_{ij} denotes the weight of the connection between nodes i and j. The first term in the right-hand side denotes the probability that a susceptible individual associated with node i becomes infected when remaining at node i, and the second one accounts for the probability that this individual catches the disease when moving to any neighbour of i. In addition, $P_i(t)$ denotes the probability that a healthy individual in (but not

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Competing interests

The authors declare no competing financial interests.

Additional information

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