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The impact of human mobility on spatial disease dynamics

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Abstract—Understanding human mobility is crucial for modeling the spatial spread of human infectious diseases. The quantitative description of spatial epidemics is based on two prominent theoretical approaches, diffusive dispersal and direct coupling or effective force of infection. The first ansatz assumes random-walk movement of the host between different locations whereas the second employs an effective force of infection between distinct populations. Both models are inconsistent with important aspects of human mobility, most importantly the bidirectional movements between individuals' homes and distant location. We introduce and investigate a novel epidemiological model that explicitely takes into account this bidirectional nature of human movements. In various topologies (networks and lattices) we find significant differences as well as similarities among all three models, depending on the parameters. On a lattice we obtain an analytical expression for the velocity of the propagating epidemic front. In contrast to the diffusion approach, our model predicts a saturation of the velocity with increasing traveling rate. Our analysis is supported by numerical simulations on both lattices and networks and provides a framework for incorporating the abundance of pervasive data on individual human mobility into disease dynamics modeling.

I. Introduction

Infectious diseases remain a pressing challenge for mankind [1]. Investigations on epidemics in human and animal populations require accurate assessments of their spatiotemporal dynamics [2] as infectious diseases spread among different locations due to movements of their host. It is frequently assumed that hosts move chaotically or perform random walks (Fig.1(a)) in space and therefore host mobility has been described within reaction-diffusion dynamics [3, 4, 5, 6, 7]. However, humans spend most of their time in particular places (at home, work, etc.) to which they always return, rarely performing long trips and multi legged trips among distant places[8, 9]. To account for this, heuristic models were introduced in which an effective force of infection between spatially separated populations was introduced to mimic the effect of underlying mobility implicitely (Fig.1(b)). Typically, the force of infection exerted by one population onto a distant one is assumed to be proportional to prevalence of the disease in the distant location [10]. However this approach lacks explicit incorporation of host movements as well as a systematic derivation and analysis of the applicability of this approach. In particular, it remains unclear how coupling depends on the individual movement rates.

Here we propose a method for incorporating bidirectional mobility as random movements on overlapping individual topologies. In particular we consider star-shaped network topologies corresponding to commuting movements of individuals between their home location (center node) and accessible destinations (distant nodes) and investigate properties of the resulting epidemics on regular lattices and random networks. Invididuals moving on these star-shaped networks are required to return to their home location from a visit to a distant location before they visit another distant location. We find that in this model movements at low travel rates yield similar results as reaction-diffusion models. In contrast, when travel rates are high, our model corresponds to the effective force of infection approach. For lattice topologies we treat our model analytically and obtain an expression for the velocity of the epidemic wave front. Contrary to the widely accepted reaction-diffusion model leading to the unbounded increase of the front velocity with increasing travel rate, in our model the velocity has an upper bound.

II. The model

Consider M locations that are connected in some way and accessible to one another. Each location is populated by N_n individuals living there permanently. At every moment an individual belonging to the n-th location can travel to some other location m. Generally, at a given point in time and in every location n individuals from other location m reside (Fig.1(d)). Without loss of generality we choose an SIS epidemiological model (generalization towards other models like SIR, SIRS, SEIR are straightforward) [1], in which susceptible and infected individuals interact in each node. We denote by I_n^m and by S_n^m the number of infecteds and susceptibles, respectively, belonging to the n-th location but currently located in the m-th location. In a given location infecteds and susceptibles originating in every other location may interact. Additionally, individuals belonging to n travel, they can leave location m and travel to some other location k with a rate ω_{km}^n. Travel dynamics and infection events can be described by the following set of reactions

$$\begin{aligned} I_m^n + S_k^n &\xrightarrow{\alpha} I_m^n + I_k^n \\ I_n^m &\xrightarrow{\beta} S_n^m \\ X_n^m &\underset{\omega_{mk}^n}{\overset{\omega_{km}^n}{\rightleftharpoons}} X_n^k, \end{aligned}$$

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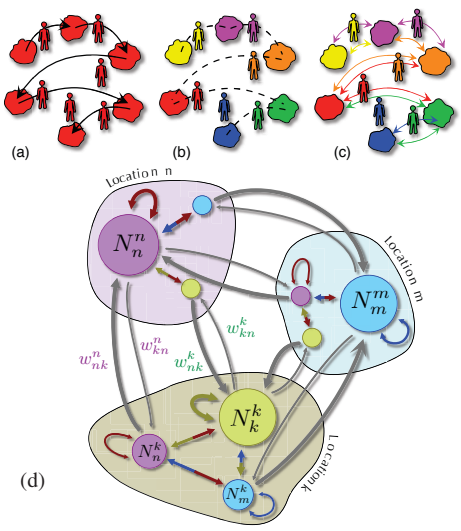

Figure 1. Mobility in spatial epidemics models. (a) Reaction-diffusion: unbounded random movements; (b) effective force of infection between populations (c) explicit incorporation of bidirectional host movements. Color code symbolizes bidirectional links due to hosts from different nodes. Panel (d) illustrates theoretical model for bidirectional movements.

where X is a place holder for I and S. α and β denote infection and recovery rates respectively. The corresponding set of mean-field equations for infecteds read (analogously for susceptibles)

$$\frac{d}{dt}I_n^m = \frac{\alpha}{N^m}S_n^m \sum_k I_k^m - \beta I_n^m + \sum_k \left(\omega_{mk}^n I_n^k - \omega_{km}^n I_n^m\right), \tag{1}$$

where $m = 1, ..., M$ and $N^m = \sum_k (I_k^m + S_k^m)$ is the number of individuals in the m-th location. In the case of all-to-all coupling we have $2M^2$ equations (1). One can reduce this number to $2M^2 - M$ taking into account conservation of the population of the location n: $\sum_m (I_n^m + S_n^m) = \text{const}$. The number of equations reduces further if locations are not all-to-all coupled. Note that the global endemic state or stationary solution $I_{total}^\star/N_{total} = 1 - \beta/\alpha$ is identical to a single well-mixed population. The travel rates $\{\omega_{km}^n\}$ quantify the travel behavior of individuals belonging to population n. We can think of them as overlapping weighted directed subgraphs. If $\{\omega_{km}^n\}$ are n-independent, we recover the reaction-diffusion case. In the following we consider the case of overlapping star-shaped networks corresponding to commuting between home and destination locations with $\omega_{km}^n = \omega_{nm}^n \delta_{kn} + \omega_{kn}^n \delta_{mn}$.

We assume that in equilibrium detailed balance is fulfilled and thus with $N_n^{m\star} = (I_n^m + S_n^m)^\star$ we have $N_n^{n\star}/N_n^{m\star} = \omega_{nm}^n/\omega_{mn}^n$. Thus the rates ω_{mn}^n and ω_{nm}^n can be defined operationally as fractions of individuals remaining in their home location and those that are traveling. The stationary population size of n is then given by:

$$N^{n\star} = \frac{N_n}{1+\varepsilon_n} + \sum_{m\neq n} \frac{\omega_{mn}^n}{\omega_{nm}^n}\frac{A_{mn}N_m}{1+\varepsilon_m}, \tag{2}$$

where $\varepsilon_n = \sum_k A_{kn}\omega_{kn}^n/\omega_{nk}^n = \sum_k A_{kn}N_n^{k\star}/N_n^{n\star}$ is the fraction of the population belonging to n that are located outside of n and A_{kn} denotes an adjacency matrix. Its elements are 1 if travelling is possible between k and n and 0 otherwise.

If commuting is very frequent as compared with the infection rate $\omega_{mn}^n \sim \omega_{nm}^n \gg \alpha, \beta$, detailed balance is fulfilled for infecteds and susceptibles separately and the last term in Eq. (1) vanishes. Realistically we have $\varepsilon_n \ll 1$ which means individuals belonging to n remain there for most of the time. For the number of infecteds from location n we can then reduce to an effective force of infection model:

$$\frac{d}{dt}I_n = \alpha S_n \sum_k \epsilon_{nk} I_k - \beta I_n,$$

where the coupling strengths $\epsilon_{nk} = \sum_m p_n^m p_k^m / N^{m\star}$ are explicitly related to travel rates and $p_n^m = N_n^{m\star}/N_n$ is an occupation probability. Hence direct coupling represents a special case of our general model.

III. Dynamics on Lattices

First we consider a homogeneous one-dimensional lattice of locations of size N separated by a distance d. Furthermore we assume next-neighbor coupling and that only infecteds can travel (this assumption can be relaxed and yields similar results). For simplicity we further assume $\beta = 0$, i.e. no recovery from the disease. We denote the number of infecteds being at home by I_n^n and the number of infecteds being in the neighborhoods $(n-1)$ and $(n+1)$ by I_n^- or I_n^+, respectively. This yields

$$\begin{aligned} I_n^n + S_n &\xrightarrow{\alpha} 2I_n^n \\ I_{n\mp1}^\pm + S_n &\xrightarrow{\alpha} I_{n\mp1}^\pm + I_n^h \\ I_n^n &\underset{\omega_2}{\overset{\omega_1}{\rightleftarrows}} I_n^\pm \end{aligned}$$

where S_n denotes the number of susceptibles in n. In the corresponding mean-field equations we can approximate $S_n, I_n^\pm$ by their continuous counterparts and perform a Taylor expansion: $I_{n\pm1}^\pm \to I^\pm(x\pm d) \approx I^\pm \pm d\nabla I^\pm + \frac{d^2}{2}\Delta I^\pm$. In a homogeneous chain the size of one location remains constant during an epidemic and equals N. Change of variables $u_n = I_n/N$, $v_n = (I_n^+ + I_n^-)/N$ and $w_n = (I_n^+ - I_n^-)/N$ leads to the following mean-field equations for the densities of infecteds

$$\begin{aligned} \partial_t u &= \alpha(1-u-v)(u+v+D\Delta v + d\nabla w) + \omega_2 v - 2\omega_1 u \\ \partial_t v &= 2\omega_1 u - \omega_2 v \\ \partial_t w &= -\omega_2 w, \end{aligned} \tag{3}$$

where $D = d^2/2$ and where we used the condition $N = S_n + I_n^+ + I_n^- = \text{const}$. The third equation has the solution $w \sim e^{-\omega_2 t}$ and for $t \gg \omega_2^{-1}$ we have $w \approx 0$ and can thus discard w leaving only first two equations (3). Steady states are $u^\star = 0$, $v^\star = 0$ and $u^\star = \omega_2/(2\omega_1 + \omega_2)$, $v^\star = 2\omega_1/(2\omega_1 + \omega_2)$. In the second steady state the density of infecteds in one city is $u + v = 1$, i.e. remains the same as in an isolated population. The system of equations (3) exhibits traveling wave solution

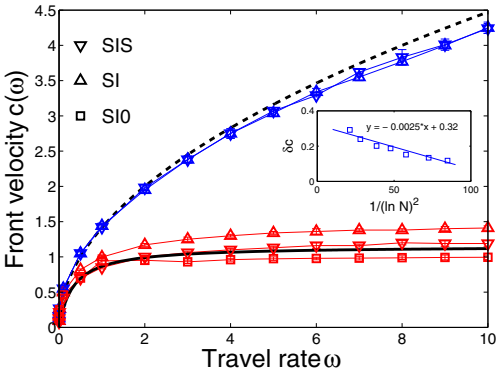

Figure 2. The front velocity c as a function of rate ω. The dashed line corresponds to hosts performing random walks as given by eq.(6). The solid line corresponds to hosts performing bidirectional movements as given by (5). Symbols represent results of numerical simulations. Number of agents per site is $N = 10^4$. The inset represents a scaling of the velocity deviation δc from the analytical prediction (5) with N.

the front velocity of which are given by

$$c = \frac{2\alpha\omega_1\sqrt{D\left(2+\frac{\omega_2}{\omega_1}\right)}}{\alpha+\omega_2+2\omega_1}. \quad (4)$$

If the forward and backward rates are significantly different from each other, two extreme cases can be considered. If the forward rate ω_1 is small, i.e. Eq. (4) holds, there is no disease spread. If the backward rate ω_2 is small, the system is determined by forward rate ω_1.

For equal forward and backward rates ($\omega_1 = \omega_2$), the velocity is given by

$$c = \frac{2\sqrt{6D}\alpha\omega}{\alpha+3\omega}. \quad (5)$$

The dependence of the velocity on α and travel rate ω as well as results of stochastic numerical simulations is depicted in Fig. 2. For comparison, the front velocity dependence of the reaction-diffusion scenario is also depicted. The velocity is in this case reads [3, 4]

$$c = 2\sqrt{D\omega\alpha}, \quad (6)$$

and we see that it does not saturate with increasing travel rate. Surprisingly, we observe this saturation in the bidirectional model. This is due to the fact that for large times only the probability to meet an infected person impacts on the spread of the infection. From (5) it follows that the asymptotic values of the velocity is proportional to the reaction rate $\lim_{\omega\to\infty} c = \frac{2\sqrt{6D}}{3}\alpha$. Fig. 2 shows a slight deviation of numerical results from the mean-field analytical prediction (5). This is due to fluctuations of the stochastic system that are consistent with inverse square logarithmic scaling of the velocity deviation δc as a function of the number of particles N (Inset in Fig. 2)., a scaling that is known to be typical for this type of reaction-diffusion system [11].

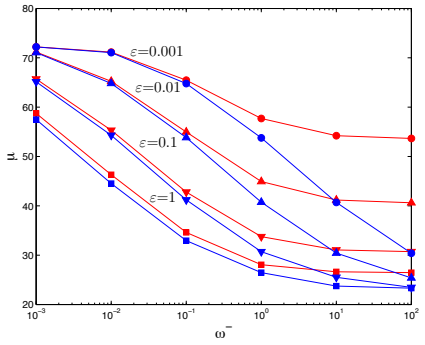

Figure 3. Mean peak time $\mu(\omega^-)$ of the epidemics on a random network defined as $\mu = \int dt I_{\text{tot}}(t)t / \int dt$ for random walk (blue lines) and bidirectional (red lines) travel patterns as a function of the backward travel rate ω^- for different commuter ratios: $\varepsilon = 1$; 0.1; 0.01 and 0.001. Maximal possible number of agents per site $N = 1000$. The network is a connected component of an Erdős-Rényi network with 400 nodes and average degree $\langle k\rangle = 2$.

IV. Network topology

A one-dimensional lattice constitutes the simplest possible case. In order to investigate the impact of complex network topologies we analyzed a random Erdős-Rényi network of coupled populations in which nodes are populated randomly according to a uniform distribution. We fixed the backward travel rate $\omega^- = \text{const}$ and chose forward travel rates as $\omega_{mn} = \omega^+ A_{mn}N_m / \sum_k A_{kn}N_k$ reflecting the distribution of individuals over the neighborhood according to the size of neighbor locations. Here ω^+ denotes an additional parameter measuring the forward travel rate $\omega^+ = \varepsilon\omega^-$. In order to compare our model with a reaction diffusion model, we chose inter-location flows of individuals to be equal in both models. Fig.3 illustrates the mean peak time of the epidemic $\mu = \int dt I_{\text{tot}}(t)t / \int dt$ obtained from numerical solutions of corresponding mean-field equations of an SIR model as a function of the backward travel rate and for different values of commuter ratio ε. At lower backward travel rates and at low commuter ratios the difference between epidemics in both models is small. The models' predictions deviate for high travel rates and for small commuter ratios. This result is in agreement with our predictions for a regular lattices. At small travel rates and small commuter ratio, travel events occur rarely compared to the outbreak time of an epidemic and it does not matter if a traveling individual returns home or not. At high travel rates, however, it is very probable that a random walker carries a disease further then just to neighboring nodes.

In a more realistic context we constructed a weighted network between counties in Germany according to a gravity model and compared a spatial SIR model based on bidirectional movments to a reaction-diffusion model. Results are shown in Fig. 4. An epidemic driven by bidirectional mobility is significantly attenuated in comparison to the epidemic driven by random walk travel pattern.

V. Conclusion

We introduced and analyzed an epidemic model that explicitly accounts for bidirectional host movements. In the limit

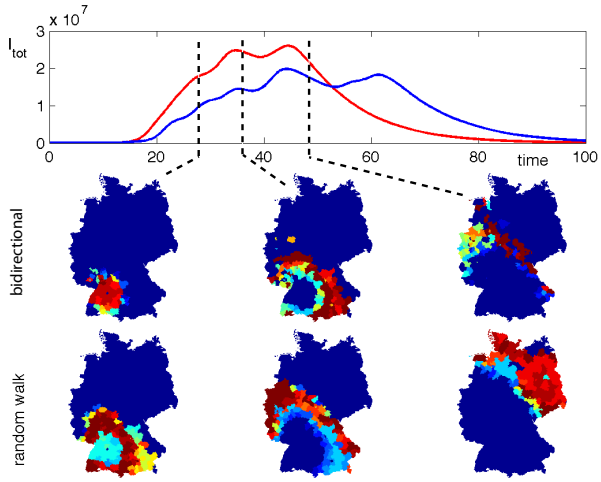

Figure 4. Evolution of an epidemic (SIR model) in a realistic context (Germany) driven by random walk (red line) and bidirectional movements (blue line) of the hosts. Upper panel: total number of infecteds as a function of time. Bottom panels: snapshots of maps of infecteds at $t = 27$; 35 and 49. One observes a clear difference between epidemics due to different movements patterns. Parameters: $\alpha = 1$, $\beta = 0.1$, $\omega^{-} = 10$, $\varepsilon = 0.001$.

of low travel rates both reaction-diffusion and bidirectional models behave similarly. An epidemic due to bidirectional movements on a regular lattice exhibits structurally different behavior as compared to widely accepted reaction-diffusion models. We showed that the epidemic wave front velocity saturates with increasing travel rate, an effect not observed in ordinary random walk models. As more data on human mobility becomes available we believe that our model presents a useful theoretical framework for incorporating this data as a first step in the development of mobility driven more quantitative models for spatial disease dynamics.

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