Modeling epidemics dynamics on heterogenous networks

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1. Introduction

One of the major threats for any living species is the outbreak of an infectious disease. Even after the development of modern medicine and the appearance of antibiotics, the human population is still at risk for the appearance of resistant strains of bacteria or lethal, infectious viral species. A series of epidemic and pandemic events in the last hundred years—from the Spanish flu to the spread of AIDS to the recent avian and swine flu—remind us that the danger still exists. The study of infection dynamics—its spread, the chance of an outbreak, the effects of pathogen infectivity, virulence and so on—is thus of extreme importance and constitutes a substantial part of current ecology literature (Anderson and May, 1992; Bailey, 1975; Murray, 1993).

The recent outbreaks of various human epidemics were characterized by very fast and nonlocal geographic spreads. The old scenario, known from the middle ages—like, say, the spread of the bubonic plague—when the disease propagated locally from town to the neighboring villages and then to the next towns—is replaced nowadays by almost instantaneous prevalence around the globe due to the intensive use of air-traffic. This poses an urgent need for a new type of infection dynamics models, and a new series of works by Colizza and Vespignani (2008, 2007), Colizza et al. (2007, 2006) have addressed this issue.

The classical mathematical models in epidemiology—SIR (susceptible-infected-recovered) and SIS (susceptible-infected-susceptible) have been presented originally for a well-mixed, uniform population (Kermack and McKendrick, 1927; Bailey, 1975; Weiss and Dishon, 1971). In the original form these models admitted no age nor spatial/social structure, and the analysis was based on deterministic differential equations. In the last decades, much work has been done in extending these models in order to include structured populations. Moreover, for small groups of individuals and for the first steps of the epidemics, the number of infected agents is small, hence the stochastic nature of the infection-recovery process must be taken into account. See a review by Vespignani (2008).

A spatially structured stochastic model has been recently analyzed for a metapopulation divided among habitat patches on a regular lattice. The intra-patch infectivity within a subpopulation is assumed to be larger than the inter-patch one. This system was subjected to extensive numerical studies (Getz et al., 2005) with a few analytic results presented (Kessler and Shnerb, 2008). To model actual human dynamics in the modern world, Colizza et al. (2007) have extended this type of model even further, taking into account the heterogenous structure of urban populations and airline connectivity. Their model describes a segregated population for which the connections between sub-communities obey a scale-free statistics, in agreement with recent observations regarding air-traffic networks (Guimerà et al., 2005).

Here we show that the heterogeneity of the network poses a new complication in the modeling procedure: on an irregular network the movement of individual agents interferes with the demography of the sub-populations. Allowing migration between nodes on an irregular network yields a nontrivial steady state size distribution for the nodes. This distribution almost surely differs...
from the initial conditions imposed in a simulation. As a result, the dynamics is affected heavily by the demographic changes as the population approaches the steady state. To overcome this difficulty we suggest a “travelers model”, within which individuals stay on their original location (node) while infecting others on neighboring sites. In this model the size of the population on a site is kept fixed so the modeler may study any realistic initial conditions.

The main aim of this work is to explain and discuss this travelers dynamics. En passant, a few interesting observations are made:

1. On heterogenous networks an increase in the movement of agents may decrease the size of the epidemic at the steady state, although it increases the chance of an outbreak.
2. These contradictory effects of movement make the estimation of $R_0$ using the steady state densities quite problematic.
3. Simple heterogenous networks, like a star structure, yield results that are very similar to a full scale-free network model (Baxter et al., 2008).
4. The effect of limited immunization of the hub population is much more pronounced, with respect to the prediction based on previous models.

Why is there an interference between demography and movement on heterogenous networks? On regular networks all “nodes” (locations, cities, habitat patches) have the same number of “links”, (i.e., routes) that agents may use for movement. Starting, say, from a uniform state where all local populations are equal, the influx of immigrants into a node is balanced by the outbound flow of emigrants, and in a steady state all nodes admit the same population (up to small fluctuations that may appear since the movement of individuals is stochastic). On heterogenous networks, on the other hand, the number of links varies among nodes. There are a few “hubs” with thousands of links (like big cities or airfields where headquarters of airline carriers are located), and many dead ends (end nodes) connected only by a few, or even a single, link. In such a case, density independent migration must introduce a drift, either towards the hubs (if the chance per agent to move is fixed) or from the hub (if the chance of migration per link is fixed).

Colizza et al. (2007) have assumed the following movement model: any individual agent emigrates with a certain probability per unit time; upon emigration, the agent chooses its destination at random from all sites connected to its original location with equal chance. As explained, such a procedure induces a drift into the hubs. This effect may be easily recognized by looking at the star structure presented in Fig. 1. Any individual that leaves the end sites must choose the hub as its destination, while the one emigrating from the hub chooses one of eight end-sites. Accordingly, starting from an equal subpopulation on any node, the movement dynamics leads to a steady state in which the hub community is much larger. As the size of the sub-community is a major factor determining the chance of local outbreaks, the resulting epidemic dynamics will be correlated almost solely with the events on the hub, where the rest of the network remains more or less passive.

The correlation between the connectivity of a node and its population size, as occurs in the steady state of the “migration model” of Colizza et al. (2007), is not unrealistic. Air-traffic hubs are typically found in the proximity of big cities and megalopolises. However, it is clear that the logic beyond this phenomenon should be reversed: people have not accumulated in Chicago or in Houston because of their large airports. The large population of a city is the reason for the existence of an airport in its vicinity, not its result.

We suggest a modified movement model for which, even on heterogenous networks, agent migration does not interfere with the demographic properties of the sub-populations. The basic idea is simple: since the vast majority of the airline travelers purchase round-trip tickets, travelers will reach their destination, stay there for a relatively short time, and then fly back. In that sense the passengers act like mosquitos in the process of vector transition, and induce “long range infection” between sub-communities. In the next sections, this basic insight will be integrated into a formal model. The model will be analyzed from various aspects including its ability to predict the effect of imperfect immunization.

2. The travelers model

Following (Getz et al., 2005; Lloyd-Smith et al., 2005; Kessler and Shnerb, 2008), let us present a travelers model for the movement between human sub populations. The population is divided into $L$ local communities, each admits an integer number
of individuals \(N_i, i = 1 \ldots L\). The set of \(N_i\)'s determines the demographic structure of the metapopulation and is kept fixed in time; this reflects the assumption that the timescale for demographic shifts is much larger than the timescale associated with the disease outbreak. The topology of the network is determined by links connecting sub-populations (these links correspond to airlines). The degree of a node (number of bidirectional connections to other nodes) is \(k\). In Fig. 1, a concrete example is illustrated: for this star configuration, \(L = 9\), for the hub \(k = 8\) and all the end nodes have \(k = 1\).

Throughout this paper we keep using the star structure as a toy model for a full scale-free network. This setup is fairly simple to simulate. It is interesting to note that the results from this star arrangement are very similar to those obtained from a full scale-free topology (Colizza et al., 2007)—it may be the case that this toy model captures, at least for some range of parameters, the involved hierarchy of a real network.

Now let us specify the dynamics of the disease. We consider here an SIS infection model with frequency dependent transition [type II in the terminology of Anderson and May (Anderson and May, 1992; McCallum et al., 2001)]. The dynamics of the infectious pathogen is determined by three parameters: \(\beta\) measures the infectivity, \(\mu\) is the recovery rate, and \(\chi\) is a dimensionless parameter that gives the proportion of nonlocal infections. \(\chi\) is the relative chance that an individual travels and infects people somewhere else, or becomes infected on a different site, rather than in his own community. If \(S_i\) is the number of those susceptible individuals on the \(i\)-th site and \(I_i\) is the number of infected individuals on the \(j\)-site, the reaction kinetics is described schematically by,

\[
S_i + I_j^{(1-\chi)N_i}2I_i, \\
S_i + I_j^{\beta N_i/N_j}I_i + I_j, \\
I_j \rightarrow S_i.
\]

For a well-mixed population of size \(N\) the basic reproduction number for type II infection is \(R_0 = \beta/\mu\) (in contrast with \(R_0 = \beta N/\mu\) for the type I model, where transitions are frequency independent). While \(R_0\) is independent of \(N\), the chance of an outbreak is still \(N\) dependent as will be shown below. This effect is attributed to demographic stochasticity: for small \(N\) an outbreak may be avoided or a spontaneous fadeout may occur even if \(R_0 > 1\), as fluctuations drive the pathogen to extinction.

We have studied this model using Monte-Carlo individual-based simulation and an analytic mean-field approach. For the simulations, each node was occupied by \(N\) inhabitants (here we considered only the case where \(N\) is the same for all sub-populations), each of which may be either susceptible or infected. The update form \(t\) to \(t + \Delta t\) has been carried out as follows: the nodes are visited sequentially. If the number of infected persons on a certain node (say, the \(i\)-th patch) is an integer \(I_i\), \(p\) of them recover (become susceptible) where \(p\) is taken from the binomial distribution \(B(\mu \Delta t, I_i)\). To precede one calculates the parameter

\[
\tilde{I}_i = (1-\chi)I_i + \sum_j \chi_{ij}/k_j,
\]

where the sum over \(j\) runs on all sites connected to the \(i\)-th one. \(\tilde{I}_i\) is a weighted measure for the number of sick persons that may infect a susceptible on \(i\), and the division of \(I_j\) by \(k_j\) reflects the fact that a traveler from the \(j\)-site chooses its destination with equal chance among all \(k_j\) patches connected to \(j\). Given \(\tilde{I}_i\), \(q\) susceptibles change their status to infected, where \(q\) is taken again from the binomial distribution

\[
B\left(1 - \left[1 - \frac{\beta \Delta t \tilde{I}_i}{N_i}\right], S_i\right).
\]

Note that \(1 - \beta \Delta t /N_i\) is the chance of a susceptible person to escape infection from a single sick person.

When global quantities are measured we define \(\rho_i\) as the overall fraction of infected individuals (total number of infected in the whole sample divided by the size of the global population).

3. Results

We have simulated the travelers IBM described above on the star network of Fig. 1 (with 10 end nodes), and compared our results with those obtained from the model of Colizza et al. (2007). The only difference between the two approaches is the spatial dynamics of agents: in our model the demography is kept fixed, while the former model allows for migration of individuals from site to site. As explained above, since any emigrant chooses its destination at random from all possible sites connected to its original location, a drift towards the hub appears. Demography changes in the migration model, and kept fixed in the travelers model. All simulations start with the same number of individuals on each site.

This difference between models manifests itself in Fig. 2. A single-infected individual has been placed on one end node, an outbreak occurs, and the number of susceptible and sick persons is plotted until the system reaches its steady state. Indeed this is a metastable state as any finite epidemic should disappear at the end due to large fluctuation (fadeout). However the timescale related to this rare event is exponentially large compared to the total number of sick people and is way beyond the times considered here. (Kessler and Shnerb, 2007.) A typical time evolution is plotted separately for the hub and for the average over all end nodes. The results of the interference between agents dynamics and local demography are clearly seen in the lower panel; evidently the drift towards the hub causes a demographic steady state that involves a strong depletion of the population on the end nodes along time. As a result almost all the activity takes place on the hub, and the overall time evolution summed over all sites (inset) is very similar to that of the hub alone. This should be compared with the fixed demography travelers model suggested here (upper panel), where a substantial part of the activity takes place on the end nodes.

Another moral that one can gather from Fig. 2 is that even when the demography is kept fixed (travelers model, upper panel), the size of the epidemic on the end nodes is substantially smaller than its size on the hub. This happens because any susceptible on the hub may be infected by all sick persons in the system, while the end sites inhabitants could be infected only from the hub. In terms of the ecology of the pathogen, the end sites act almost like “sinks” (spatial regions where the reproductive number is negative) as it is relatively hard for the pathogen to find there a new host. Indeed, the mean-field deterministic approximation discussed below predicts that the total number of infected individuals in the system decreases when the level of motion (travel) described by the parameter \(\chi\) increases. This surprising effect is demonstrated in Fig. 3.

Fig. 4 summarizes the results for the chance of an outbreak and the steady state epidemic size for both models. It shows again that, while an outbreak is more likely for strong dispersal, the corresponding steady state size of the epidemic \(\rho_i\) is (in the travelers model) lowered.

This is a somewhat surprising result. It is known that the movement of individuals facilitates the spread of an epidemic, and in particular that the critical infection rate \(R_c\) for a spatially segregated population decreases as \(\chi\) grows (see, e.g., Getz et al., 2005; Lloyd-Smith et al., 2005; Kessler and Shnerb, 2008). One may expect, thus, that the number of healthy people will be
Fig. 2. Upper panel: typical time development of the disease, using the fixed population dynamics suggested here, on a star network. The results of a fully stochastic simulation is presented, with 1000 individuals on each site. $\chi = 0.1$ and $R_0 = 2$. The number of susceptible and infected on the center ($I_c$ and $S_c$) and the average number of the end sites ($I_e$ and $S_e$) are indicated separately. The graphs follow the development of the outbreak from the introduction of a single sick subject to the steady state. In the inset, the global dynamics of the disease (total numbers of infected and susceptible individuals vs. time) is presented. Lower panel: the same outcomes are graphed for the movement model considered by Colizza et al., where migration acts to increase the total population, as the majority of dynamics happen there.

smaller if the migration rate is larger. What we observe here is that, on heterogenous networks, increased mobility facilitates the outbreak but reduces the size of the epidemic at the steady state.

This effect may, or may not, appear in the model used by Colizza et al., where migration acts to increase the total population on the hub. Concentration of the total population acts in favor of the disease and this (at least for some topologies) may invert the sign of the response function $d\rho_r/d\chi$. As seen in Fig. 4, for the set of parameters used for our simulation the steady state size of the infection is independent of $\chi$ in the migration model.

The contradictory roles played by migration in nonuniform spatial models—lowering $R_c$ while decreasing $\rho_1$, may pose a serious problem if one is trying to use the steady-state size of the epidemic $\rho_1$ in order to retrieve $R_c$. The standard method (Anderson and May, 1992), based on the assumption of a well-mixed large population, obtains $R_c$ from the relation $R_c = 1/(S/N)$, where $S/N$ is the steady state fraction of the susceptibles. This method has already been criticized (Keeling and Rohani, 1995), but now we see that it may fail on a heterogenous network just because of the topology: a glance at Fig. 3 convinces one that a totally different estimation may be obtained for $R_c$, although the threshold for an outbreak is the same, $R_0 = 1$, for all cases. Steady state estimations not only fail to give the right value, they even fail to give the right order: a pathogen with a smaller $\rho_1$ may be more dangerous, in terms of the chance for an outbreak, than one that has a larger $\rho_1$.

3.1. Vaccination strategy

Many recent studies were focused on the case of limited immunization (Pastor-Satorras and Vespignani, 2002; Cohen et al., 2000a, b, 2003; Chen et al., 2008), trying to explain how to immunize a population with a minimal number of immunization supplies. This question has become very important since the last pandemic spread very fast while the immunization was under preparation, limited and/or very expensive.

These studies showed that the random uniform immunization is a highly inefficient strategy on scale-free networks. These networks provides an ideal environment for the spreading of infective agents through the hubs, and random immunization can not avoid percolation through the network. On the other hand, immunization based on the nodes’ connectivity hierarchy (the vaccination of hubs takes place before the vaccination of the end sites) should be used in order to avoid outbreak (Pastor-Satorras and Vespignani, 2002).

We examine now a vaccination strategy with a fixed, limited number of immunization supplies on the star network. It is already known that one should deposit all the immunizations on the hub, trying to disconnect different parts of the network. How does the immunization affect the outbreak if only a fraction of the hub population is immunized? Let us check the results and compare between the travelers scenario and the migration model.

Fig. 5 presents the results from our IBM Monte-Carlo. First, we initialized the network with $N$ susceptible individuals per site. One sick individual was introduced into the system and the process continued according to the algorithm described above. Once the system reached the point where 10% of individuals were infected, we immunized $f \cdot N$ susceptible individuals on the central node where $f$ stands for the fraction of immunity (between 0 and 1). Only healthy subjects were immunized, so the effect of immunization is to decrease, effectively, the population on the hub.
Fig. 4. Some characteristics of the stochastic simulations, all obtained for the Travelers model (upper panel) and the migration model (lower panel) with a star geometry. Each panel shows 4 sub-figures. (a) presents the probability of extinction, i.e., the chance that there is no outbreak when a single sick individual is introduced. This chance is 100% below the critical $R_0$ and is finite above criticality as a result of demographic stochasticity. Note that as $\chi$ grows, the chance of an outbreak increases, as expected. The steady state value of $r_I$ is plotted in (b) as a function of $R_0$, emphasizing that in the round-trip travelers model the density of infected individuals decreases when there are more travelers, as opposed to the chance of an outbreak depicted in (a). In the migration model the differences are negligible. (c) and (d) illustrate the dynamics on the center node: the total number of infected (c) and the total number of susceptible (d) are shown in absolute numbers. Note that the total population in this simulation is 11,000, so the lower panels reflect the strong drift towards the hub in the migration model.
which represents the total difference of the infected population for a small time interval $\Delta t$. $I_k^{in}$ and $I_k^{out}$ are the number (per unit time) of susceptibles who become sick or recover, correspondingly, within $\Delta t$. Clearly

$$I_k^{out} = \mu I_k,$$

where $\mu$ stands for the recovery rate, which is the only means of exiting the $I_k$ class. The influx is

$$I_k^{in} = (1-\chi) \frac{\beta}{N} S_k I_k + \frac{\beta}{N} S_k \sum_{k'} P(k'|k) \frac{\gamma}{k'} I_{k'},$$

where the first term stands for infection on the same node, and the second term describes the chance of being infected by travelers at/from connected nodes. The summation is over $k'$, the degree of the linked sites, and $P(k'|k)$ stands for the probability that a site with $k$ links will be connected to a site with $k'$ links.

Assuming uncorrelated network, $P(k'|k) = k P(k')/\langle k \rangle$ (Pastor-Satorras et al., 2001; Dorogovtsev and Mendes, 2003),

$$\frac{\text{d} I_k}{\text{d} t} = -(1-\chi) \frac{\beta}{N} S_k I_k - \frac{\beta}{N} S_k \sum_{k'} P(k'|k) \frac{\gamma}{k'} I_{k'} - \mu I_k.$$

In the same manner, the equation for $S_k$ takes the form,

$$\frac{\text{d} S_k}{\text{d} t} = \mu I_k - (1-\chi) \frac{\beta}{N} S_k I_k - \frac{\beta}{N} S_k \sum_{k'} P(k'|k) \frac{\gamma}{k'} I_{k'}.$$

One can easily verify that these equations conserve the local population, $\text{d}(S_k + I_k)/\text{d} t = 0$. This is a result of the assumption at the basis of the analysis that $N$ is time independent.

### 4.1. The outbreak

The basic reproductive number $R_0$ plays a major role in the early stages of the pandemic. $R_0$ is essentially the average number of successful offsprings that a parasite is intrinsically capable of producing (Anderson and May, 1992). Introducing a single-infected individual is equivalent, under the deterministic approximation, to initial conditions for which $I$ is slightly larger than zero, such that $I/S \to 0$ so $S \to N$, $S/N \approx 1$. Using the relation $\sum_k P(k) I_k = \langle I \rangle$. Eq. (5) takes the form:

$$\frac{\text{d} I_k}{\text{d} t} = -(1-\chi) \frac{\beta}{N} S_k I_k + \frac{\beta}{N} S_k \sum_{k'} P(k'|k) \frac{\gamma}{k'} I_{k'} - \mu I_k.$$

Let $\bar{I}$ denote $\langle I \rangle$. When multiplying the above expression by $\sum_k P(k)$, one obtains a simple equation for $\bar{I}$

$$\frac{\text{d} \bar{I}}{\text{d} t} = \frac{\text{d} I_k}{\text{d} t} = (\beta - \mu) \bar{I},$$

and using

$$I_0 = \langle I_{k|t=0} \rangle = \sum_k P(k) I_{k|t=0},$$

the solution for this equation is

$$\bar{I} = I_0 e^{(\beta - \mu) t}.$$}

We have obtained the classical result that an outbreak may occur when $R_0 = \beta/\mu > 1$.

The deterministic models predict a critical value $R_0=1$. However, it is known (Getz et al., 2005; Lloyd-Smith et al., 2005; Kessler and Shnerb, 2008) that stochastic fluctuations may cause a spontaneous fadeout even above this critical value. For example, if $\beta = 2$ and $\mu = 1$ then $R_0=2$, which means that on average two persons are infected by a sick individual before he recovers. Yet there is a finite chance for the recovery of the single-infected introduced (the founder) before the first infection. As shown by the figures above, below the critical $R_0$ outbreaks never appear, but above this value the chance for an outbreak is not 1.

The results are depicted in Fig. 5. In the travelers model the effect of immunization is evident and an increase of $f$ leads to an appreciable reduction of the chance of an outbreak. In the immigration model the effect is much less pronounced, since there is a flow of agents to the hub.

### 4. General mean-field solution

In this section we present an analytic solution to the deterministic equations describing the dynamic of a spatial infection process. We consider the travelers model when the effect of stochasticity may be neglected. As in the simulations we assume here the same number of individuals $N$ on each site; $I_k$ and $S_k$ stand for the average infectious population and the susceptible population, respectively, with the same connectivity $k$. Following the standard procedure for networks we assume that all nodes of degree $k$ admit the same dynamics.

Using the method presented by Colizza and Vespignani (2008), the rate equation for the infected population is defined by

$$\frac{\Delta I_k}{\Delta t} = I_k^{in} - I_k^{out},$$

where $I_k^{out} = \mu I_k$. The influx is

$$I_k^{in} = (1-\chi) \frac{\beta}{N} S_k I_k + \frac{\beta}{N} S_k \sum_{k'} P(k'|k) \frac{\gamma}{k'} I_{k'},$$

which represents the total difference of the infected population for a small time interval $\Delta t$. $I_k^{in}$ and $I_k^{out}$ are the number (per unit time) of susceptibles who become sick or recover, correspondingly, within $\Delta t$. Clearly

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One can easily verify that these equations conserve the local population, $\text{d}(S_k + I_k)/\text{d} t = 0$. This is a result of the assumption at the basis of the analysis that $N$ is time independent.

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Let $\bar{I}$ denote $\langle I \rangle$. When multiplying the above expression by $\sum_k P(k)$, one obtains a simple equation for $\bar{I}$

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and using

$$I_0 = \langle I_{k|t=0} \rangle = \sum_k P(k) I_{k|t=0},$$

the solution for this equation is

$$\bar{I} = I_0 e^{(\beta - \mu) t}.$$
This effect of stochasticity can not be seen within this mean-field framework.

4.2. Steady state of a pandemic

In the steady state,
\[
\frac{\partial k}{\partial t} = \frac{k_{\text{in}} - p_w k}{k} = 0, \tag{11}
\]
using Eq. (5) and that \(S_k = N - I_k\), we get
\[
(1 - \chi) \beta \langle N-I_k \rangle k + \chi \beta \langle N-I_k \rangle k \langle I_k \rangle - \mu k = 0. \tag{12}
\]
We define \(z_k\) as the fraction of the infected individuals in the group of sites with connectivity \(k\), i.e., \(I_k = z_kN\). Substituting this into the above equation, one may easily get a general description of the steady state:
\[
(1 - \chi) R_0(1 - z_k) + \chi \langle z_k \rangle -(1 - z_k) = 0. \tag{13}
\]
For simplicity, we will focus from now on the star structure with \(L\) sites (see Fig. 1). In this case we can divide the nodes into two classes: the central node that holds \(L - 1\) links and “end nodes” holding only one connection. We define \(I_c, S_c, I_e, S_e\) as the relative densities of susceptibles and infected on the center and on the end nodes, respectively. The steady state equations for the infected population on the central node and on the end nodes reads as follows:
\[
(1 - \chi) R_0 z_c(1 - z_c) + R_0(1 - z_c)(L - 1) \langle z_e \rangle - z_c = 0.
\]
\[
(1 - \chi) R_0 z_e(1 - z_e) + R_0(1 - z_e) \langle z_c \rangle - z_e = 0. \tag{14}
\]
This set of equations may be solved numerically, and the results are depicted in Fig. 3. While the migration rules in our model keep the overall size of any subpopulation fixed and for the case considered each site admits the same population, the steady state shows a different density of infected individuals. As \(\chi\) grows, the infected fraction on the hub grows, but on the end site, the epidemic size decreases. The overall density per site of the infected population, \(\rho_i = (z_c + (n - 1) z_e) / n\), decreases with \(\chi\). It is easy to verify that this effect is weak if the movement is rare (the first order correction to \(\rho_i\) evaluated perturbatively for small \(\chi\) vanishes) but is pronounced when the travel parameter \(\chi\) is large. In particular for \(\chi = 1\) (infection only to neighboring sites) \(z_c = 1 - 1/R_0\) (growth with respect to the value \(1 = 1/R_0\) for zero migration) but \(z_e\) scales like \(1/n\), so all the \(n - 1\) nodes yield a non-extensive contribution to \(\rho_i\).

4.3. Stochastic vs. deterministic models

It should be stressed that movement does facilitate the outbreak of an epidemic, but this effect is invisible in the deterministic model solved above. As seen in Fig. 4 b, the chance of an outbreak is larger when movement is larger. This, however, comes from the effect of demographic fluctuations. Dispersal decreases the effect of “kin competition” between pathogens: as the number of individuals on each site is fixed, larger migration avoids an effective decrease in the infection rate resulting from the depletion of the susceptible population (Keeling and Rohani, 1995). One of the approximations that lead to the deterministic theory is the replacement of numbers by densities, which implicitly assumes an infinite number of individuals on each site such that the effect of kin competition vanishes. Kin competition manifests itself only in stochastic models of discrete individuals, like the Hamilton-May model for dispersal (Hamilton and May, 1977; Comins et al., 1980).

Hamilton and May have considered two species which are identical in all aspects except for their migration rate. On a regular lattice or any other type of homogeneous environment the fast must win (we neglect for the moment the migration cost introduced by Hamilton and May, and assume that each emigrant reaches its destination). The reason is that agents do not have to compete with their offspring on the local resources, as the offspring leave the habitat patch occupied by the parent. On a heterogenous substrate, on the other hand, there is an advantage to the slow species that may “stick” to the oases and will suffer less demographic losses due to migration into bad spatial domains. While the second effect is deterministic, the first one is stochastic and disappears in the continuum limit, since in that limit the number of individuals allowed on a patch is infinite. For that reason the non-stochastic model of Hastings and coworkers (Hastings, 1983)—which is a spatial version of Hamilton-May with no migration cost—failed to retrieve Hamilton-May results and suggested that the best strategy is to decrease the migration rate to zero. A recent discussion of the relation between stochastic and deterministic models in the context of the evolution of dispersal rates may be found in (Kessler and Sander, 2009).

The same considerations are applicable to the rate equations, based on the continuum approximation, used here to analyze the deterministic dynamics. Such a model captures only the suppressive effect of migration: the increase of \(\chi\) induces a drift of infected individuals towards the hubs, that acts like a “trap”. Actually, on heterogeneous system like this there is a natural “dispersal cost” as some of the propagules end up in unfavorable sites; this effect leads to a maximal epidemic size at \(\chi = 0\), as obtained by Hastings (1983), and has been proven recently by Dockery et al. (2007). As shown above, although in a fully stochastic model a larger dispersal facilitates the outbreak of the disease, the effect considered here does not disappear and in the steady state. For the range of \(N\) considered above, \(\rho_i\) is smaller when \(\chi\) is larger.

5. Conclusions

The basic motivation of this work comes from the pioneering studies of Colizza and coworkers, who first suggested a realistic extension of the traditional epidemiological models to real human populations. These authors correctly assumed that the large hubs of the air traffic network correspond to sites with a large population. Still their movement model mixes the local demography with the migration dynamics such that the modeler can not determine the steady state demography. Here we have presented an improved model in which round-trip travelers are described as a vector for transition; using our scheme one may plug the actual demographic numbers and the steady state is not affected by the law of motion of the agents.

We did not try to model a realistic airline network in all its glory as did Colizza et al. (2007). Instead, we have discussed in detail the simple case of a star geometry using our technique. The results show a very interesting difference between the outbreak dynamics and the steady state behavior of the epidemic. The main take-home message is that birth–death processes on heterogeneous networks may differ strongly from their counterparts in a well-mixed population or on regular networks, and in particular that the steady state size of the epidemic may not reflect the right \(R_0\). Further extensions of this work to socially structured populations are also possible.

Finally, we have addressed another issue: the impact of a vaccination campaign. It turns out that the immigration model underestimates the impact of vaccination on the hub as it allows for immigration of susceptible individuals into the hub during the
outbreak. To say it another way, the spatial structure is not so important in the migration model since virtually all the activity happens on the hub. Conversely, in the travelers model, a vaccination campaign is much more effective as it allows for isolation of different parts of the network, and thus the infection can not “percolate” in space.

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