A Hierarchical Metapopulation Model for Disease Dynamics Built on Population Movements of Both Patch-Coupling and Migration

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Abstract. In the past, frequent movements and migration were studied separately in metapopulation models. The levels that exist in a hierarchical metapopulation model were also limited to two and only recently increased to multiple levels. Moreover, a generalisable deterministic model was not available. Here we introduce a novel model incorporating both movement scenarios as well as a multiple-level structure. We describe the system in simple differential equation form. The simulations of different distributions of local contact rates for disease transmission suggest that local information is important for predicting disease dynamics. The comparison between the results from a solely migration-based multilevel model and the model discussed in this paper suggests that diseases with low transmission rates can spread rapidly and infect a large number of susceptible individuals in a short time if they appear in a population where frequent movements are dominant.
Keywords: multilevel system, patch-based system, disease modelling, epidemic, mathematical epidemiology

1 Introduction

It is now clear that understanding and prediction of the progress of disease requires consideration of the spatial arrangement of individuals and many recent computational models of disease spreading place a strong emphasis on the role of the spatial heterogeneity of human populations [1–3]. It is clear that the distribution of individuals within a population significantly affects the process of pathogen dispersal, but the exact operating functions linking transmission and spatial pattern are still unknown. However, even when using pathogen-related parameters that are invariant, e.g. a model where pathogen infectivity and virulence are kept constant throughout the simulations, the results generated by spatial models simulate the real epidemics very well [2, 4–6].

There are various ways to integrate spatial heterogeneity into models. One popular method is to build metapopulation models, where the population is divided into a network of smaller subpopulations on patches [2, 7, 8]. Individuals within each subpopulation are assumed to be well mixed. Metapopulation models allow explicit mathematical expressions and straightforward numerical solutions [9], and hence play an important role in mathematical epidemiology. Hierarchical metapopulation models are a special type of general metapopulation models [2, 10]. They consider

the hierarchy involved in human movements (i.e. that subpopulations have some non-random pattern of connections) [11, 12] and simulations from these models show that disease spreading is significantly influenced by multilevel movements [2, 6]. New studies based on real human mobility data also provide evidence to support the argument that individual movements occur at different levels [11, 12].

Another thing to consider is that the ways individuals interact with each other. Interactions between individuals involve both within-patch interactions and between-patch interactions. It is assumed that the contacts between individuals on each patch are frequent and hence random mixing applies within the subpopulation. Between-patch interactions are of more interest and usually modelled by two methods, depending on the frequency of movements. If the interaction between two patches is dominated by frequent movements (e.g. people commuting to and from work), the subpopulations are said to be interacting with each other in a way like particles randomly bumping into each other. In other words, any infection occurring on one patch has the force of infection on the susceptible individuals in the closely related patches [1, 13, 14]. The force of infection is defined as the per capita rate that the infected individuals transmit the disease to susceptible individuals [15]. Alternatively, if the movements between two patches mainly take the form of migration, it means that individuals migrate to the host population with the disease status they get from the home patch first and then take part in the disease transmission process in the host patch [1, 9]. These two scenarios were studied separately in the past [1, 13]. In real populations, it is obvious that both scenarios occur simultaneously.

Here we build a metapopulation model based on multilevel movements including both patch-coupling and migration. At the lowest level, where the population movements between the patches are most frequent, the patches are coupled by the force of infection; while patches with less frequent movements in between are linked by migration. In this paper, these two kinds of patch relationships are referred to as close-related patches and not close-related patches. Moreover, it does not necessarily mean that well-connected patches are geographically close, in contrast to previous work [2]. Human mobility tends to be more complex than animal migration or plant dispersal and is not necessarily related to geographic distances [16, 17].

2 The model

The hierarchical system we set up to describe the metapopulation model consists of L levels of movements. The number of patches at the same level is denoted by a fixed branching ratio B

for simple indexing. Therefore the total number of patches is B^L, denoted by n. We assume that the fewer the movements between two patches, the larger level difference (D) between them. The level difference is the number of levels to reach the common ancestor node in the hierarchy. The level difference between close-related patches, i.e. the first level when counting levels, is defined as 0. One possible system is illustrated in Figure 1 as an example. Individuals are assumed to be homogeneously mixed within each patch. All patches are assumed to have identical within-patch population dynamics and environmental conditions [8]. Person-to-person contact, which leads to disease transmission, takes place when individuals meet in one of the n patches. Patches interact with each other through either coupling or migration. Initially the total population is distributed evenly across all n patches and one infectious individual is introduced to the system. Individuals in each patch are classified in terms of their infection status: susceptible, infected or recovered, within which the numbers of individuals are denoted by S, I and R respectively. The susceptible class includes all healthy people with no immunity to the disease, the infected class includes people who have caught the disease. For simplicity, infected individuals are assumed to be infectious immediately. R denotes the recovered group, with lifelong immunity [13, 15]. We model a non-fatal, communicable disease, such as the common cold or influenza virus, spreading much faster than the natural demographic process. Therefore our basic framework is the simple *SIR* model (Equation 1) [15, 18]:

$$\begin{aligned} \frac{dS}{dt} &= -\beta IS \\ \frac{dI}{dt} &= \beta IS - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{1}$$

where disease is transmitted through person-to-person contact which is modelled by the adequate contact rate for disease transmission (β) and recovery rate (γ). This is the normalised form, i.e. all the variables are proportions. It models the pure disease transmission process within a homogeneously mixed population without considering other effects, such as demographic effects, spatiotemporal effects and so forth. In our system, where the interactions between patches take two different forms, we need to consider both effects. The force of infection for the simple *SIR* model is βI. To generalise it to the single patch in our model, we need to consider that the force of infection on patch i is affected by the sum of the infection situations on the patches close-related to it, i.e. $\sum_j \beta_{j,i} I_j$, where $j \in J$, J: the set of indices of the patches close-related to patch i. In this model, we allow the adequate contact rate to vary between close-related patches [13] and therefore

we can examine the effects of different contacting rules on disease dynamics. The range of values of β is consistent with that for influenza [15].

The other process that changes the number of infected individuals in patch is migration. Both emigration and immigration are assumed to operate between two patches which are not close-related. The per capita migration rates of infected, susceptible and recovered groups in the whole system are denoted by θ, ϕ and ξ respectively. The immigration rate of susceptible individuals from another patch to patch i is calculated by a function $\theta_{ki} = \theta C e^{-CD}$, where $k \in K$, K: the set of indices of the patches linked to patch i by migration. The subscript ki indicates immigration and ik emigration. Ce^{-CD} is a normalised general exponential function. It is the simplest form for representing the mechanism that the migration rate decays as the level difference between two patches increases. C is a constant, which scales the function and D is the level difference (described above). Similarly, $\phi_{ki} = \phi C e^{-CD}$ and $\xi_{ki} = \xi C e^{-CD}$ describe the rates of movement for infected and recovered individuals respectively. Finally, all the immigrants from different patches are summed and all the emigrants to different patches are subtracted for each disease group to get the total proportion of immigrants and emigrants.

As we described above, because all the patches are homogeneous in population distributions, disease spreading behaviours and patterns of between-patch movements, we are able to express the population dynamics on an arbitrary patch i into a differential equation form (Equation 2):

$$\begin{aligned}
\frac{dS_i}{dt} &= -(\sum_j \beta_{ji} I_i) S_i + \sum_k \theta_{ki} S_k - \sum_k \theta_{ik} S_i \\
\frac{dI_i}{dt} &= (\sum_j \beta_{ji} I_i) S_i - \gamma I_i + \sum_k \phi_{ki} I_k - \sum_k \phi_{ik} I_i \\
\frac{dR_i}{dt} &= \gamma I_i + \sum_k \xi_{ki} R_k - \sum_k \xi_{ik} R_i
\end{aligned} \tag{2}$$

All migration parameters have very small values in this paper and therefore have little effect on the variability of local population dynamics during the simulation. All simulations are run until equilibrium is reached. Here we use two systems with the same number of patches in total: 1) branching ratio of four ($B = 4$) and level of three ($L = 3$); 2) branching ratio of two ($B = 2$) and level of six ($L = 6$).

3 Results

Figure 2 shows the time series of proportion of infected individuals for β being uniformly distributed and normally distributed. More fluctuations and different sizes and durations of the infection changes are observed with the uniform distribution. It shows smaller but longer epidemics in the uniform distribution based model than those when β is drawn from a truncated normal distribution.

In previous work using hierarchical metapopulation models, contacts between patches are based solely on migration behaviours [2, 6] and comparison results with and without patch-coupling is shown in Figure 3. It clearly shows that patch-coupling accelerates the spread of the disease through the system and leads to more cases, even with a smaller contact rate for disease transmission.

4 Discussion

Combining close-related patches and migration-related patches, we obtain behaviours not observed in previous studies [2,6]. A uniform distribution of β means that we have little prior knowledge of the adequate contact rates on patches, so the random chosen value has equal opportunities to stay at any point within the lower and upper bounds. We have 64 uniformly distributed random numbers and we expect strong stochasticity. The results illustrated in Figure 2 confirm this. On the other hand, 64 normally distributed random numbers tend to surround the mean. Consequently we observe some randomness but still see the three-level pattern. In conclusion, estimating the adequate contact rates in real life is an important step towards choosing right models for predictions.

It was shown by previous studies that patch-coupling is a quick way for the disease dynamics on each patch to synchronise [13, 14], but observing the effects in a multilevel metapopulation model was not realised. We obtained a larger epidemic with even smaller contact rates for transmission in a model including patch-coupling. Therefore it demonstrates that epidemics are likely to be much worse in a large population where movements are frequent between sub-populations. We suggest that health authorities cannot ignore an infectious disease with low transmission rate that occurs in a large population where people have more frequent short-trips between subpopulations.

We promote a model based on both frequent movements and long-lived travels and it shows that differentiated contact rates make the disease dynamics more complicated but still tractable. Since local information of contact rates are not usually collected [14], we expect that investigations

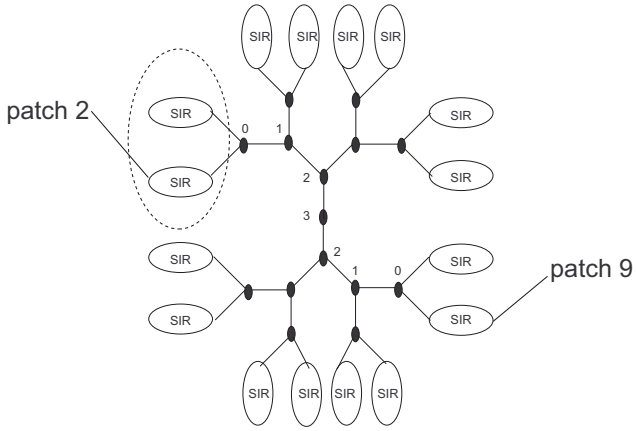

Fig. 1. One example of the multilevel system ($B = 2$, $L = 4$). The small ellipses represent the patches, the black nodes illustrate the different levels and the dashed ellipse represent one example of close-related patches. *SIR* means that the simple Susceptible-Infected-Recovered process applies within each patch. It also shows an example of calculating the value of D, the level difference between any two patches (see method for description). D = 3 for patch 2 and patch 9 (anticlockwise numbering) according to the levels they belong to (i.e. you have to move up three levels before these two patches share a common node).

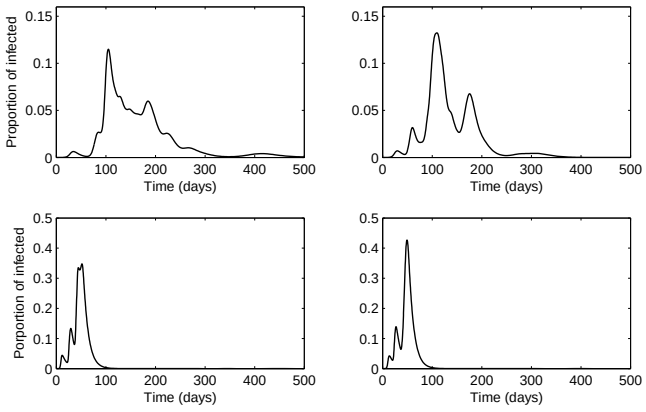

Fig. 2. The effects of two different distributions of β on disease spreading. $B = 4, L = 3$. (Top): two simulations based on a uniform distribution of β ($0.0 < \beta < 0.6$); (Bottom): two simulations based on a truncated normal distribution of β (mean = 0.3, standard deviation = 0.1, $0.0 < \beta < 0.6$). It shows that we get smaller but longer epidemic from the uniform distribution based model then those from the normal distribution. Moreover, the results from the uniform distribution are more stochastic.

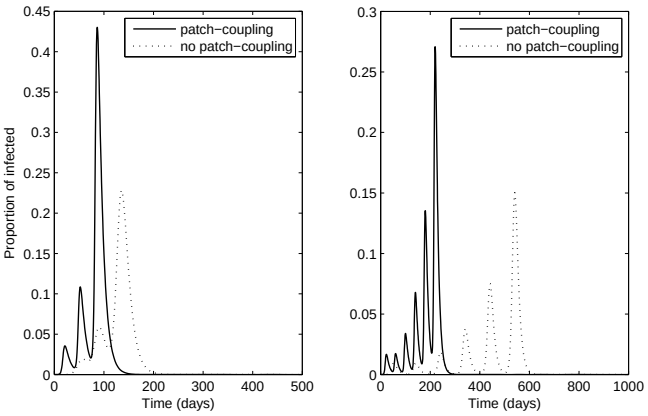

Fig. 3. Comparison between migration-based model and patch-coupling-migration-based model for two systems: $B = 4, L = 3$ (Left); $B = 2, L = 6$ (Right). For migration-based simulations (dashed line), $\beta = 0.3$ is used; whereas $0.1 < \beta_{ki} < 0.3$ is applied for patch-coupling-migration-based simulations (solid line), which means the adequate contact rate on average is smaller than three. It shows that even with a smaller contact rate, the size of epidemic is not reduced for patch-coupling-migration-based model. It also shows that the synchronisation of the system is more rapid in the patch-coupling-migration-based model.

on such data will be helpful both for validating the model and for facilitating better prediction of the spread of diseases through human populations.

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