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Appendix1

Model description

We developed a simple model to illustrate how disease dynamics may change when explicitly considering, and varying, the phenology and synchrony of migration. To this end, we combined a dynamic network model with an epidemiological and a migration model such that infection status and location of individuals ('nodes' in the network) could change as a result of infection dynamics and migration, respectively. We followed disease prevalence over time and location.

Network dynamics

In the model, individuals are defined as nodes in the network and individuals can be connected via links ('edges'). We used a dynamic network, in which links between individuals can form and dissolve over time; yet, the average number of edges per node in the network (i.e. the mean degree) is preserved. Thus, the model incorporates time-varying contact structures. We assumed a relatively sparse, undirected network with a mean degree of 0.5; links lasted on average 5 time-steps. We used a temporal exponential-family random graph model that provides a method of constructing networks with a given set of properties. Markov chain Monte Carlo can then be used to create a range of plausible networks that agree with a wide variety of information collected on network structures even if the complete network is unknown. We used the statnet suite for constructing our network models (Handcock et al. 2003), for details see (Goodreau et al. 2008) and (Handcock et al. 2008), and its sub-package 'EpiModel' (Jenness et al. 2014) for the epidemiological network model and R ver. 3.1.1. for all further analyses and scenarios (<www.r-project.org>).

We defined a network consisting of 200 individuals (nodes). In addition to changing links, individuals could migrate and get infected or recover – as a consequences of infection dynamics and migration.

Infection dynamics

We used a SIS (susceptible–infectious–susceptible) model (Keeling and Rohani 2008), in which susceptible individuals can be infected with probability, τ, if connected to an infected individual.

Infected individuals recover with probability γ, and re-enter the pool of susceptibles. Although we had no particular parasite or disease in mind, we used parameter values of $\tau = 0.2$ and $\gamma = 0.07$, i.e. infections lasted on average 14 days.

Migration

We consider a simple type of migration from a starting to a destination site. Migration is instantaneous and depending on specific scenarios, individuals migrated at or around a mean migration date, $t_{mig} = 100$. As we assume the two sites to be distant, no links (and thus, no parasite transmissions) were allowed between individuals at disparate sites.

For simplicity, we assume a time-step of one day; and we followed disease dynamics over 200 days. At the start of each simulation, all individuals were located at the starting site and 10% of the individuals were randomly infected.

As the model included various stochastic components, e.g. formation and dissolution of links, parasite transmission and recovery from infection, we ran 100 repetitions of each scenario and analysed the average prevalence per site as well as its 25% and 75% quantiles (not shown).

Scenarios

We explored the role of migration synchrony and phenology on disease dynamics in two sets of scenarios: First, we investigated the gradient from fully synchronous to asynchronous migration by increasing the standard deviation around the mean migration date, $t_{mig} = 100$, from $\sigma = 0$, 1, 2, 5 to 10. The reasons for such variations in migration dates are not further specified here but could be due to, e.g. variation in fuelling rates (Seewagen et al. 2013), sex-specific constraints and selection pressures (Saino et al. 2010), differential use of environmental factors that trigger departure (Gordo et al. 2013, Otero et al. 2014), or a delayed departure of infected individuals. The latter might result from disease symptoms such as fatigue, lowered activity, slower fuelling (Bradley and Altizer 2005, van Gils et al. 2007).

Secondly, we explored changes in the phenology of parasites relative to migration phenology. To this end, we introduced a period of increased (environmental) parasite pressure (or increased susceptibility to infection) at one site and varied the onset of this period relative to migration. We used a period of 20 days of increased parasite pressure; and this period started at day $t = 60$, 90, or 120 at the starting site such that it was either distinctly before or distinctly after migration, or individuals migrated in the middle of this period. During this period, individuals at the starting site were additionally infected with a probability of 0.5.

Increased parasite pressure or susceptibility may result from various processes: Individuals using parasite-rich habitats within a site (Hoye et al. 2012), or the aggregations of immense

numbers of migrants at key refuelling locations en route enhance parasite transmission and thus, increase prevalence (Krauss et al. 2010). Some studies have also suggested periods of increased susceptibility during the annual cycle in migratory animals resulting from, e.g. seasonal variations in immune function (Buehler et al. 2008) or the intense physiological demands of migration that may trade off with immune responses (Gylfe et al. 2000).

Results

For scenarios on changes in synchrony, we found prevalence to remain at the same level on both starting and destination site in an entirely synchronously migrating population. However, if individuals migrated asynchronously, i.e. spread out over time, prevalence gradually decreased at the starting site and gradually increased at the destination and thus, differed considerably for a long period (Fig. 3a–b).

Changing the onset of elevated parasite pressure also affected prevalence. While prevalence obviously was unaffected when parasite pressure was increased *after* all individuals had migrated to the destination site, prevalence in the population at the starting site was elevated when parasite pressure was elevated before and even more so, if it coincided with migration. These variations in prevalence at the starting location then spilled over to, and influenced, prevalence at the destination (Fig. 3c–d). At the destination site, prevalence subsequently decreased towards a low level (which is determined by epidemiological parameters and average number of links).

Thus, both migration synchrony and phenology importantly shaped (local) disease dynamics (Fig. 3) and prevalence varied widely with alterations in the phenology or synchrony of migration even though the underlying epidemiology was kept constant.

Although the model presented here is on purpose kept simple, its findings have implications for the spread of diseases – if we want to estimate in how far migratory animals contribute to the long-distance spread of parasites and pathogens, we need to be specific about the timing of migration.

Obviously, there are many more issues that could be explored with such epidemiological model, including applying it to specific pathogens and parasites, to a variety of migratory animals with more complex migrations and thus, testing particular assumptions on (changes in) contact structures, and interactions between synchrony and phenology in combination with demographic processes.

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