Selection for intermediate mortality and reproduction rates in a spatially structured population

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How local interactions influence both population and evolutionary dynamics is currently a key topic in theoretical ecology. We use a 'well-mixed' analytical model and spatially explicit individual-based models to investigate a system where a population is subject to rare disturbance events. The disturbance can only propagate through regions of the population where the density of individuals is sufficiently high and individuals affected by the disturbance die shortly after. We find that populations where individuals are sessile often exhibit very different dynamic behaviour when compared to populations where individuals are mobile and spatially well mixed. When mutations are allowed which affect either offspring birth rates or mortality rates, the well-mixed populations always evolve to a state where a single disturbance event leads to extinction. Populations often persist substantially longer if individuals are sessile and they disperse their offspring locally. We also find that for sessile populations selection may favour short-lived individuals with limited offspring production. Population dynamics are found to be strongly influenced by the host characters that are evolving and the rate at which host variation is introduced into the system.

Keywords: disturbance; spatially structured populations; evolution

1. INTRODUCTION

Many plant and animal populations experience disturbance events which kill or remove individuals and change resource availability (Pickett & White 1985). The spatial scale of a disturbance event may be strongly influenced by the spatial arrangement of individuals near the focus of the disturbance (Turner 1987). Many types of disturbance cannot propagate through a population where individuals are sufficiently spread apart (Knight 1987). Examples include the outbreak of an infectious disease (Satō *et al.* 1994; Rand *et al.* 1995; Keeling 1999) and the spread of wildfire (Knight 1987; Green 1989; Bradstock *et al.* 1998). Predation can also be an example of a propagating disturbance. Individuals that are densely clumped may be more susceptible to predation when compared to more isolated individuals if predators intensify their effort to find new prey in areas where they have recently encountered prey (Hassell & May 1974).

In this paper, we investigate models of systems where populations experience rare disturbance events which are rapidly lethal to individuals. We focus on disturbances that are influenced by, and in turn alter, the spatial structure of the population, and examine selection acting on an organism's birth rate and mortality rate. For the remainder of this paper we will use the language of a host–disease system (e.g. the rabbit–myxomatosis system), but the results of this paper are applicable to other systems where disturbance events (i) kill or remove

individuals, (ii) only propagate through densely populated regions, and (iii) propagate at a time-scale that is much faster than the time-scales of births and deaths.

We consider a habitat composed of a linear arrangement of identical patches, which may be either empty or occupied by a single host individual. Disease outbreaks occur on randomly chosen hosts at some fixed rate. The disease spreads quickly to neighbouring hosts and stops propagating when no uninfected host lies within some neighbouring region of all those infected. All infected hosts die immediately after they have infected their neighbours. Each individual host is characterized by a mortality rate and a birth rate. We compare a model which assumes individuals are randomly distributed in space at all times (i.e. spatially well mixed) with a model where individuals are sessile. For both models we examine population dynamics and evolutionary dynamics of the host birth rate and mortality rate.

The well-mixed model predicts that selection will always favour hosts with lower mortality rates and higher birth rates. Eventually, the host population evolves to a state where it is so uniformly dense that it then becomes highly susceptible to extinction by the next disease outbreak. The sessile model predicts that selection may favour hosts with intermediate birth rates and mortality rates if hosts disperse their offspring locally. Moreover, an evolving host population is more likely to persist in the sessile model when compared with the well-mixed model. In the sessile model individuals with low mortality rates and high birth rates are likely to be distributed within large dense clumps. Although in the short term these

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individuals have an advantage at spreading within the population, because they are clumped, they eventually become weeded out of the population through disease. However, long-term persistence of individuals with intermediate birth rates and mortality rates is possible in the sessile model because they are generally associated with regions of low host density and hence are less susceptible to disease. Selection can lead to a population which is generally in a state where it will not be driven to extinction by the next disease outbreak.

2. THE WELL-MIXED MODEL

Consider a habitat consisting of a large number of contiguous patches that may be either occupied or unoccupied by a single host individual. The first model we consider assumes hosts are mobile and at any time they are randomly distributed within the habitat. For simplicity we assume the habitat is spatially one-dimensional (1D) (we briefly address the case of multiple spatial dimensions in the discussion).

When a disease outbreak occurs on a patch that contains a host, the disease spreads rapidly to neighbouring hosts. A parameter B ($B=0,1,\dots$) specifies the maximum number of unoccupied patches a disease can cross and still infect a host. This parameter allows us to model disease propagation via some locally dispersing vector. The disease stops propagating when no uninfected hosts are separated from infected hosts by B or less unoccupied patches. All infected hosts are removed from the system immediately after the outbreak. The fraction of patches occupied by hosts at time t is denoted $n(t)$. If hosts are randomly distributed at the time of the outbreak, then the average number of hosts infected and subsequently killed by the disease is

$$s = \frac{2 - (1 - n)^{B+1}}{(1 - n)^{B+1}}. \quad (1)$$

Equation (1) is derived by considering clusters of occupied patches, having an average size $(1+n)/(1-n)$, connected in series with connection probability $1 - (1 - n)^B$.

We now consider a system that is infinite in size (i.e. there are an infinite number of patches). Suppose all hosts have the same instantaneous per capita birth-rate b , and disease-independent mortality-rate m . Host offspring are dispersed to a randomly chosen patch and die if the patch is occupied by a host. In the absence of disease, the host population dynamics in the infinite system are governed by the logistic equation

$$\frac{dn}{dt} = [b(1 - n) - m]n. \quad (2)$$

Equation (2) assumes a continuous and homogeneous mixing of the hosts, thereby removing all spatial correlations (Wilson *et al.* 1993; Durrett & Levin 1994; Rand *et al.* 1995; Nisbet *et al.* 1997).

Suppose patches now become the foci of disease outbreaks at rate d . Host dynamics in the presence of the disease are governed by (Henley 1993),

$$\frac{dn}{dt} = [b(1 - n) - m - ds]n. \quad (3)$$

Irrespective of B , if the host birth-rate b is greater than $m + d$, there exists a unique non-trivial equilibrium host population density. The host population will be driven to extinction if the birth rate is less than this critical value. An important point is that for any value of d the population will persist if b is sufficiently high and m is low.

We now introduce evolutionary dynamics into the model. We assume an offspring's birth rate and mortality rate may differ from its parents through mutation, and so the population average rates may evolve. There are assumed to be no trade-offs between these two rates. An invasible analysis (MacArthur & Levins 1967; Turelli 1981) of the analytical model (equation (3)), shows that if the host population is monomorphic and at equilibrium, then a mutation can invade the population if it leads to an increased birth rate or a decreased mortality rate. Hence the host population evolves towards ever increasing birth rates and ever decreasing mortality rates, which in turn increases the host density.

The previous analysis assumed an infinite system. To investigate evolutionary dynamics in a finite system we now simulate the system using an individual-based model (IBM), sometimes referred to as a probabilistic cellular automaton (Rand *et al.* 1995).

The habitat is represented by N patches linked in the form of a ring. Hosts reproduce asexually and each host is associated with a genotype, which is composed of a birth rate and a mortality rate. The simulation proceeds in time by identifying when the next 'event' occurs. There are three possible events: (i) a single host dies from a cause other than disease, (ii) a single host gives birth, and (iii) a disease outbreak occurs on a patch. Hosts that die are removed from the system, emptying their associated patch. When a host gives birth, it produces a single offspring which is then dispersed to one of its parent's two neighbouring patches. If the patch is already occupied the offspring is assumed to die. If the offspring survives, its genotype is determined by first setting it equal to its parent's genotype, but there are probabilities, p_b and p_m , that the offspring will exhibit a mutant birth rate or a mutant mortality rate. The birth rate of a mutant offspring is set to a random value uniformly distributed within α_b of its parent's birth rate. Similarly, a mutant's mortality rate is set to a random value within α_m of its parent's mortality rate. If a disease outbreak event occurs, the disease is propagated through the population and infected hosts are removed from the system. After every event all surviving hosts are randomly redistributed among patches. Note that this IBM is an example of an asynchronous updating model (Durrett & Levin 1994; Rand *et al.* 1995).

Figure 1a shows examples of host population dynamics generated by the well-mixed model. One of the examples assumes all host genotypes are equal and fixed in time; the other allows host rates to mutate and hence evolve. The fixed genotype example shows host persistence in the presence of the disease. The analytical model predicts that in the long term approximately 58% of the patches will be occupied. In this example, the analytical model and the IBM generate similar population dynamics. With

Figure 1. Dynamics produced by the well-mixed individual-based model (IBM) when $N = 1000$ patches. (a) Two examples of population dynamics are presented: host genotypes are fixed (thin line); host genotypes may evolve (thick line). Both examples assume an initial host genotype: $(b, m) = (0.5, 0.2)$. Each patch becomes the focus of disease outbreaks at rate $d = 0.001$ per unit time and can jump single empty patches ($B = 1$). The dashed line indicates the equilibrium population size predicted by the analytical model. (b) Evolutionary dynamics of the evolving example presented in (a). The solid line is the mean birth rate of hosts; the dashed line is the mean mortality rate of hosts. The variation in these rates within the population is also shown by the dotted lines which indicate two standard errors from the population mean.

evolution, the host population density generally increases with time but the population suffers outbreaks of increasing severity. Eventually the host population experiences a disease event which causes extinction. The path of evolution for this example is presented in figure 1b. As time progresses the population tends to evolve higher birth rates and lower mortality rates. The parameter values we used to describe host mutations for all examples presented are: $p_b = p_m = 0.1$, $\alpha_b = 0.05$ and $\alpha_m = 0.01$, respectively. All the general patterns of evolutionary dynamics presented in this paper are found to be robust to variations in initial conditions and system size.

Extinction occurs in the evolving system because the system is finite in size and disease outbreaks are rare compared to the time-scale at which births and deaths occur. Hosts usually experience non-disease conditions

and evolve as if diseases were not present. This situation leads to an increase in the average population density which increases its susceptibility to extinction from subsequent disease outbreaks. Unlike the analytical model, the IBM predicts host extinction even when host birth rates are high and mortality rates are low.

When considering such a stochastic model one must be careful when referring to 'persistence' in a finite system. The empty state (i.e. no hosts) is an absorbing state, and there is always some probability that all hosts will die during some time interval before they give birth. Thus all systems will go to extinction after some finite time. However, the time until extinction rapidly increases as the size of the population increases. We say an evolving host population is persistent if the population size is generally attracted to some non-zero value and the probability of the population experiencing an extinction event does not increase with time. Formal definitions of persistence can be found in Durrett & Levin (1994) and Rand *et al.* (1995).

The dynamics presented in figure 1 are typical for a wide range of parameter values. Extinctions also occur when we fix either the birth rate or the mortality rate, and only allow the other rate to evolve. The average time to extinction increases as (i) the rate and magnitude of mutations decrease, (ii) the number of patches increases, and (iii) the number of empty patches a disease can jump (B), is reduced.

3. THE SESSILE MODEL

We now consider the case when hosts are sessile, yielding a population which exhibits spatial structure correlated through time. Population dynamics are examined by considering a sessile version of the IBM, which is simply the well-mixed IBM except hosts are not randomly redistributed after each event. Formulating an analytical model that incorporates spatial structure, which can then be used to make predictions about selection, is a difficult task (Rand *et al.* 1995); it will not be attempted here, but will be the focus of future work.

First, consider the case when all hosts have the same fixed genotype. Examples of population dynamics are presented in figure 2. Long-term persistence occurs for a wide range of parameter values. As with the well-mixed IBM, we observed the sessile IBM to generate host extinctions when the birth rate is very low and when it is high. Extinctions also occur when the rate of disease outbreak is high.

We now investigate the case when host rates evolve. Figure 3a shows evolutionary dynamics of the birth rate when all hosts have the same fixed mortality rate, and vice versa in figure 3b. Both examples show the evolving host character stabilizing and long-term persistence. Neither stabilization nor long-term persistence was ever observed with the well-mixed IBM when either character evolved alone or together.

Evolution does not always lead to long-term host persistence in this system. If the probability of a mutation or the magnitude of mutations is sufficiently high, the host population will crash. In these cases the population evolves too rapidly in comparison with the rate of disease outbreaks, producing a single large dense clump which is then wiped out by the next disease outbreak. When

Figure 2. Two examples of host population dynamics for the sessile individual-based model (IBM) when all hosts have a common genotype. Genotypes are as follows: (a) $(b, m) = (0.7, 0.2)$; (b) $(b, m) = (1.0, 0.2)$. Each patch becomes the focus of disease outbreaks at rate $d = 0.0002$ per unit time and can jump single empty patches ($B = 1$). The habitat contains 1000 patches. Patches occupied by a host are represented by a black square. Space is depicted in the horizontal, time in the vertical. Each simulation is run for 1000 time units (≈ 200 generations). Disease outbreaks are identified by the sudden gaps appearing in the populations.

mutation parameters are not too high, dense clumps of individuals with either high birth rates or low mortality rates often become weeded out of the population. The result can be a long-term persistent population containing individuals with intermediate mortality and birth rates. Mutation rates have been shown to be important in determining population dynamics in other spatially explicit host–disease systems (Keeling & Rand 1995).

Figure 3. Evolutionary dynamics generated by the sessile individual-based model (IBM) when disease occurs at rate $d = 0.0002$ patch $^{-1}$ t $^{-1}$ and can jump single empty patches ($B = 1$). The habitat contains 1000 patches. (a) Host mortality rate is fixed ($m = 0.2$) and the host birth rate is allowed to evolve. (b) Host birth rate is fixed ($b = 1.0$) and the host mortality rate is allowed to evolve. The variation in these rates within the population for both examples is also shown by the dotted lines which indicate two standard errors from the population mean.

Regions containing individuals with intermediate mortality and birth rates may resist invasions from more productive individuals if host dispersal is local. Gaps within such regions open up frequently through host death. If neighbouring hosts have a low birth rate then these gaps may persist for some time, inhibiting disease propagation. We did not observe long-term persistence when we modified the sessile IBM, so that offspring were dispersed globally. In this case offspring may be dispersed over long distances, allowing highly fecund or long-lived individuals to establish anywhere within the system; this quickly closes any gaps. Thus, if hosts disperse offspring over large areas, then isolated regions of less productive individuals may not form and the system behaves much like the well-mixed model (Durrett & Levin 1994).

When the birth rate and the mortality rate evolve at the same time a different pattern of evolutionary dynamics is observed (figure 4). In this case neither rate stabilizes; on the contrary, both rates evolve towards ever higher values. The explanation is that it is those parents which neighbour

Figure 4. Host population dynamics for the spatial IBM when both host birth rate and host mortality rate can evolve. Initially all hosts have a common genotype: $(b, m) = (1.0, 0.2)$. Each patch becomes the focus of disease outbreaks at rate $d = 0.0002$ per unit time, and can jump single empty patches ($B = 1$). The habitat contains 1000 patches. (a) Host population dynamics. (b) Evolutionary dynamics of the host birth rate and the host mortality rate. The variation in these rates within the population is also shown by the dotted lines which indicate two standard errors from the population mean.

a gap and have the higher birth rates that are most likely to succeed in getting their offspring into the gap; therefore there is selection for higher birth rates. However, there is also selection for these offspring to have higher mortality rates as well, otherwise they will quickly form dense clumps and be susceptible to disease. The result is selection for individuals which produce offspring at a faster rate and are shorter lived. In a real system other factors are expected to place an upper limit on a mutant's birth rate. As the birth rate and mortality rate increase, the number of births (and hence the number of mutation events) increases between disease outbreak events. This increase in the rate of mutation events can allow the population to evolve to a state where it is susceptible to extinction by an outbreak. Like the previous examples when only a single rate evolved, we observed long-term persistence to be less likely as the rate and magnitude of mutations are increased.

4. DISCUSSION

There is currently a great deal of interest in trying to understand how spatial interactions may influence

evolutionary dynamics, particularly with regard to host–disease systems (Claessen & de Roos 1995; Rand *et al.* 1995; Keeling 1999). Here we have presented a preliminary investigation which shows that spatially localized interactions can have pronounced effects on evolutionary dynamics. These effects have been identified by carefully comparing the dynamics generated by two IBMs, which only differ in how they assume individuals distribute themselves within their habitat. There have been a number of other studies where IBMs have been used to investigate the effect of local interactions on population and evolutionary dynamics. Many of these studies have focused on either plant dynamics or host–disease dynamics. We now briefly compare the results of two previous studies with those produced by our study.

Inghe (1989) modelled the growth of a herbaceous perennial population which was composed of a number of genets. Sites arranged on a square lattice could be occupied by a single ramet and each ramet was associated with a genet. Genets expanded by clonal growth to empty adjacent sites, and all genets were assumed to have the same competitive ability. This study investigated genet survival when the population experienced localized disturbances of fixed size. Square-shaped disturbances were randomly placed within the habitat, killing all ramets within their bounds. Disturbance size had a dramatic effect on genet survival; as the disturbance size increased, the number of genets which persisted in the population decreased. Inghe (1989) showed that genets could often persist if they were associated with clusters which were larger than the size of each disturbance event. In contrast, our model shows genets having a better chance of survival if they are distributed among many small isolated clusters. The reason our results differ is that we do not fix disturbance size; it varies depending on the spatial distribution of individuals.

Rand *et al.* (1995) investigated the effect of spatial interactions on selection in a spatially two-dimensional host–pathogen system. Hosts could be either infected by the pathogen, or healthy and susceptible to infection. All hosts reproduced by sending propagules to neighbouring sites. Infected hosts passed the disease to uninfected neighbours at some finite rate, and infected hosts were removed from the system at a fixed rate. They investigated selection acting on the rate at which the pathogen was transmitted between hosts. This system contrasts with ours because we assume the propagation of the disturbance is effectively instantaneous, and we investigate selection acting on the hosts rather than the disturbance. Despite these differences both studies show similar phenomena. One common feature is that the rate of evolution is much slower in the spatially structured model compared to the well-mixed model (compare figure 1 with figures 3 and 4). Rand *et al.* (1995) observed that, unlike their well-mixed analytical model, their spatial model exhibited a threshold pathogen transmission rate above which the pathogen tended to die out. In their spatial model, if pathogen transmissibility was allowed to mutate it evolved towards the threshold value (i.e. the pathogen evolved itself to the boundary where it could exist). We too observe threshold values in our spatial model which determine if a host population is

quickly driven to extinction. However, if the rate at which mutations introduced into our system is not too great, then birth rates and mortality rates do not always evolve towards their threshold values, and populations often persist for long periods (figure 3).

Our model shows that the rarity of disturbance events can have an impact on evolutionary dynamics. If the disturbance is very rare, the population may evolve as if the disturbance never occurs, making it vulnerable to extinction by the next disturbance event. However, if the disturbance events are frequent they may also drive the population to extinction. In a multi-species system this relationship between host persistence and d is consistent with the intermediate disturbance hypothesis on diversity (Connell 1978).

Levin & Pimental (1981) showed that selection could favour intermediate parasite growth rates. Like our model, it was shown that although genotypes with higher growth rates had a higher fitness over short time-scales within isolated colonies, these genotypes had a lower fitness within the whole population over longer time-scales.

Although many systems may be effectively 1D in space (e.g. systems where populations tend to aggregate along the borders of multiple habitat types), most systems are arranged in two or three dimensions. Preliminary investigations of a two-dimensional version of each model show similar behaviour to the 1D versions. The most notable difference is that, as hosts have more neighbours in two dimensions, disturbances tend to propagate through larger regions of the population. This tendency results in larger fluctuations in the population size. The sessile version also shows that evolution can lead to selection for intermediate birth and mortality rates.

The disturbance we have modelled here is extreme in its characteristics; it always transmits to adjacent neighbours and kills individuals quickly. Many disturbances are not this virulent. For example, diseases may not always be passed on from an infected individual to a neighbouring susceptible individual, and often they do not kill their hosts. An interesting next phase of this study would be to investigate the selection pressures placed on a population to resist the effects of the disturbance (i.e. to stop or slow down its propagation). Future work could also investigate the benefits and costs associated with dispersing offspring over larger distances, and how selection could influence rates of mutation.

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