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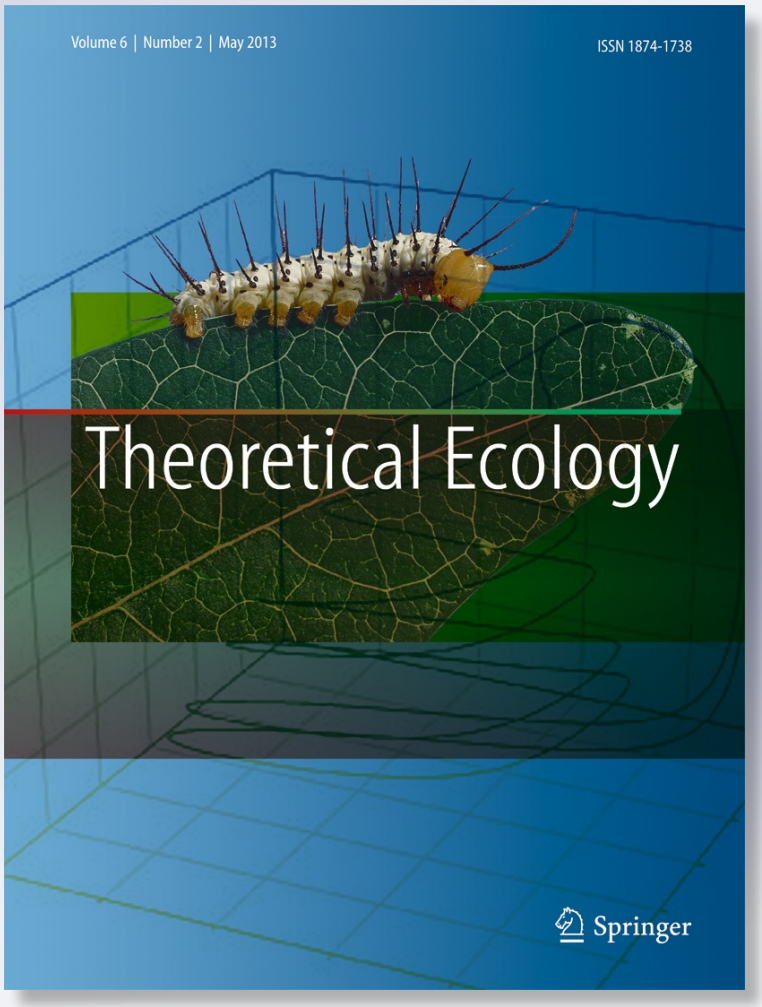
Theoretical Ecology

ISSN 1874-1738
Volume 6
Number 2

Theor Ecol (2013) 6:153-164
DOI 10.1007/s12080-012-0167-z



Springer

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ORIGINAL PAPER

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Received: 7 November 2011 / Accepted: 25 June 2012 / Published online: 15 July 2012

Abstract Understanding the factors that influence successful colonization can help inform ecological theory and aid in the management of invasive species. When founder populations are small, individual fitness may be negatively impacted by component Allee effects through positive density dependence (e.g., mate limitation). Reproductive and survival mechanisms that suffer due to a shortage of conspecifics may scale up to be manifest in a decreased per-capita population growth rate (i.e., a demographic Allee effect). Mean-field population level models are limited in representing how component Allee effects scale up to demographic Allee effects when heterogeneous spatial structure influences conspecific availability. Thus, such models may not adequately characterize the probability of establishment. In order to better assess how individual level processes influence population establishment and spread, we developed a spatially explicit individual-based stochastic simulation of a small founder population. We found that increased aggregation can affect individual fitness and subsequently impact population growth; however, relatively slow dispersal—in addition to initial spatial structure—is required for establishment, ultimately creating a tradeoff between probability of initial establishment and rate of subsequent spread. Since this result is sensitive to the scaling up of component Allee effects, details of individual dispersal and interaction kernels are key factors influencing population level processes. Overall, we demonstrate the importance of considering both spatial structure and individual level traits in assessing the consequences of Allee effects in biological invasions.

Keywords Biological invasion · Allee effect · Spatial structure · Individual-based simulation

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Introduction

The seemingly distinct interests of conservation biologists and invasion ecologists converge on a common thread—understanding the factors that influence the viability of small populations. The ecological literature abounds with the broad notion that there is a positive relationship between population size and successful establishment and persistence (Williamson 1996; Lockwood et al. 2005, 2007; Simberloff 2009). This relationship is due in part to demographic stochasticity, which has a stronger impact at smaller populations, but it can also reflect deterministic density dependence that increases the likelihood of extinction at low numbers. It has long been known that when a population is small, the reproduction and survival rates of individuals may decline with decreasing population density (Allee 1931), for a wide range of mechanistic reasons (listed in Holt et al. 2004). This principle has been empirically observed in numerous species ranging from bacteria to plants and animals, including asexual as well as sexual taxa. There is a growing recognition that such Allee effects can have profound consequences in natural populations and communities (Courchamp et al. 2008). With growing threats of species invasions, emerging infectious diseases and biotic homogenization, characterizing the factors that permit successful establishment at low numbers is challenging but essential.

In this paper, we use an individual-based model to examine how density dependence is experienced at the level of individuals, and how this translates into implications for population

establishment and spread over different spatial and temporal scales. For positive density dependence to occur in a population (a "demographic Allee effect", Stephens et al. 1999), it is necessary that a positive relationship exists between population size and at least one measurable component of individual fitness (a "component Allee effect" Stephens et al. 1999). Conversely, however, net individual fitness may not be significantly depressed at low densities, despite the action of component Allee effects (e.g., finding a mate), if there is compensation in other fitness components that are enhanced at low density (e.g., competition for resources might be reduced). The relative strengths of these processes may differ among otherwise identical individuals, due to their spatial position in the population and the spatial configuration of their neighbors, mediated through the spatial scaling of density-dependent processes. Conflicting demands on individuals can influence behavioral responses to such pressures. One strategy that might mitigate component Allee effects and thus affect population dynamics is limited dispersal leading to aggregation (Grindrod 1988; Padron and Trevisan 2000; Gascoigne et al. 2009). Essentially, the net effect of being in a small population may be different for each individual based on its traits and its neighborhood, producing a range of ecological consequences, which depend on how the population is spatially structured.

Models of population dynamics can either implicitly or explicitly make assumptions about how component Allee effects can lead to a demographic Allee effect (Taylor and Hastings 2005). Deterministic models can easily describe population level behavior through the use of a bistable growth function representing demographic Allee effects. Such models typically assume generalized mean field dynamics, where each individual has an equal probability of interacting with every other individual. This assumption is often not justified in natural populations, where interactions are localized among individuals, and so this approach may miss unexpected outcomes. One step towards incorporating spatially delimited interactions is to use spatial reaction–diffusion models (Okubo 1980). Such models add a level of complexity to mean field approaches and can usefully address issues such as the asymptotic wave of advance of an invasion. However, reaction–diffusion models also leave out features of spatial organization, localized interactions among individuals, and stochasticity that may be crucial for population persistence.

Random effects and spatial and temporal fluctuations may lead to significant deviations from expectations from deterministic models of population growth and spread, particularly when numbers are low, as in the initial phases of an invasion. Specifically, Taylor and Hastings (2005) summarize prior work reporting that stochasticity tends to increase the probability of establishment for populations initially smaller than the Allee threshold. Furthermore, recognizing the importance of each individual being discrete and at a specific location, where it interacts with neighbors, as a general theme in population dynamics (Durrett and Levin 1994) ultimately implies that the spatial dimension of individual interactions should be explicitly addressed, and should tend to amplify the importance of stochasticity in initial establishment. Indeed, the inspiration for Allee effects originated from observations of interactions among individuals within animal aggregations (Allee 1931). We suggest that the detailed spatial patterning of individuals across a landscape can influence the magnitude of component Allee effects leading to an impact on the net demographic Allee effect, and thus can ultimately influence persistence and invasion dynamics. Individual behavior and demographic rates should be primarily impacted by the local environment in which an individual resides, including individuals with whom it interacts. Thus, characterizing the spatial structuring of local neighborhoods should be key in scaling up to population-wide density dependence in births and deaths. A detailed portrayal of intraspecific interactions and individual behaviors in a spatially explicit context is, we argue, essential to understanding emergent characteristics of successful establishment and spread.

To investigate if (and how) component Allee effects scale up to demographic Allee effects, and to disentangle the influences of spatial structure, dispersal, and local interactions on population establishment and spread, we developed a spatially explicit individual-based stochastic simulation of an invasion process for a small founder population with localized interactions. Our results help explain how even very small populations can sometimes establish, despite the existence of overall strong positive density dependence leading to Allee effects and the expectation of heightened extinction risks at low numbers (Courchamp et al. 2008; Gascoigne et al. 2009). Our primary aim is to develop a deeper understanding of the ecological consequences of Allee effects and how small founder populations could succeed—despite processes leading to (on average) strong positive density dependence that at low population sizes might be expected to doom them to extinction.

Individual-based model

Because invasions are inherently stochastic spatial processes, we constructed an individual-based model that incorporated randomness in birth, death, and movement events in continuous space and time. We formulated a stochastic version of a well-studied, reaction–diffusion model (Lewis and Kareiva 1993; Murray 1993; Keitt et al. 2001; Kot 2001; Drake et al. 2005; Drury et al. 2007; Kanarek and Webb 2010) to investigate the influence of individual interactions on the dynamics of population growth and spread, in this context. This deterministic model is

$$\frac{\partial N(x,t)}{\partial t} = rN(x,t)\left(\frac{N(x,t)}{a} - 1\right)\left(1 - \frac{N(x,t)}{C}\right) + D\nabla^2 N(x,t), \quad (1)$$

where $N(x,t)$ is population density, which is a function of position, x (a vector for two- or three-dimensional space), and time, t. The population growth rate depends on the intrinsic growth rate r (assumed positive), as well as the population density relative to both an Allee threshold, a, and an environmental carrying capacity, C, where $0<a<C$. If an isolated population is below the Allee threshold, it has negative population growth and faces certain extinction. Thus, the Allee threshold represents the minimal population density for population survival; the carrying capacity represents maximal population density if invasion succeeds. The diffusion coefficient, D, scales the rate of population spread across the habitat. Given diffusion with an Allee effect, the initial population for model (1) must be large enough over a sufficiently large initial area in order to survive (Lewis and Kareiva 1993; Murray 1993; Kot 2001; Drake et al. 2005; Drury et al. 2007; Kanarek and Webb 2010; Vercken et al. 2011). This leads to an expectation of a minimal patch area and initial population required for persistence and spread. Since these conditions are based on the population perspective, we explored how constraints at the individual level can influence these results about how initial conditions influence eventual establishment.

We used the population model as the basis for our stochastic individual-based simulation, so that we could compare individual results to the population level perspective from the well-known reaction–diffusion equation. Hence, we interpreted Eq. (1) from an individual perspective and defined birth, death, and movement events accordingly. The simulation is an event-driven Markov process based on Gillespie's direct algorithm with inter-event times exponentially distributed (Gillespie 1977; Renshaw 1991; Birch and Young 2006; Erban et al. 2007). This framework allows birth and death events, based on component Allee effects, to occur in continuous time, where individual i has a birth rate, b_i (the rate at which the individual gives birth) and death rate, d_i, each of which depends on its local population size, N_i. A neighbor-counting scheme is used to find N_i (the portion of the total population within individual i's neighborhood), given a particular local interaction kernel (i.e., the distance-dependent interaction between a given individual and its neighbors). For the top-hat interaction kernel (Fig. 1a), for instance, N_i is the number of individuals within a specified distance, S_T, from individual i; these interact with individual i to affect its reproduction and survival.

To generate an Allee effect at low density, and negative density dependence at higher density, the specific relations used for per individual birth and death rates are as follows:

$$b_i = \frac{N_i}{a} + \frac{N_i}{C} = \frac{N_i(a+C)}{aC} \text{ and } d_i = 1 + \frac{N_i^2}{aC} = \frac{aC + N_i^2}{aC} \tag{2}$$

(derived similarly to those in Ackleh et al. 2007). When the local population size N_i is either a or C, birth and death rates are equal and overall individual fitness (i.e., r_i, the difference between birth and death rates) is 0. For N_i between these values, the birth rate exceeds the death rate and overall individual fitness r_i is positive, while outside these values the death rate is higher than the birth rate. The quantity a defines a local Allee threshold density for each individual, whereas C is a measure of individual neighborhood carrying capacity. To preserve generality, our aim was to consider the effect of the density experienced by each individual in terms of a broad range of fitness-related traits. The mechanisms that produce component Allee effects in reproduction and affect individual birth rates may include fertilization efficiency and/or reproductive facilitation (Courchamp et al. 2008). Since we assume that individual death rates are primarily affected by high density, density dependence in death rates can be considered the result of various mechanisms giving rise to intraspecific competition.

Births and deaths are assumed to be independent Poisson processes. Once a simulation is initialized, the birth rates and death rates of all individuals are summed to give an overall event rate E for the population. Since the sum of Poisson processes is also a Poisson process, at any time, the time interval (Δt) until the next event (birth or death) is exponentially distributed with a mean of $1/E$. Once this interval is determined using a random number generator, the event is assigned to a specific individual and designated a birth or a death using another random number and probabilities based on the contribution of each rate to the total event rate (the probability that an event is individual i giving birth or dying is b_i/E or d_i/E, respectively). If the event is a death, the individual is deleted; if it is reproduction, a new individual is added to the population at the parental location. Following this event, all individuals disperse (see below), their birth and death rates are then calculated according to their new local population size, and the event rates summed to give the new E. The time until the next event (Δt) is generated, and this process is then repeated for the duration of the simulation (which is run long enough so that either extinction occurs, or persistence is essentially ensured).

The simulation is spatially explicit and follows a population of discrete organisms of a single species introduced into a two-dimensional landscape. The spatial framework consists of a physically homogeneous environment treated as a continuous region (as opposed to, for example, a discrete lattice), assumed large enough for edge effects to be negligible (i.e., in our simulations, the boundaries were never reached) but with periodic boundaries. An individual i is located at coordinates (x_i, y_i), and every individual, including newborns, moves following any birth or death event that occurs anywhere in the population, which gives nearly continuous movement. In order to closely approximate Brownian motion (i.e., the second term on the right in

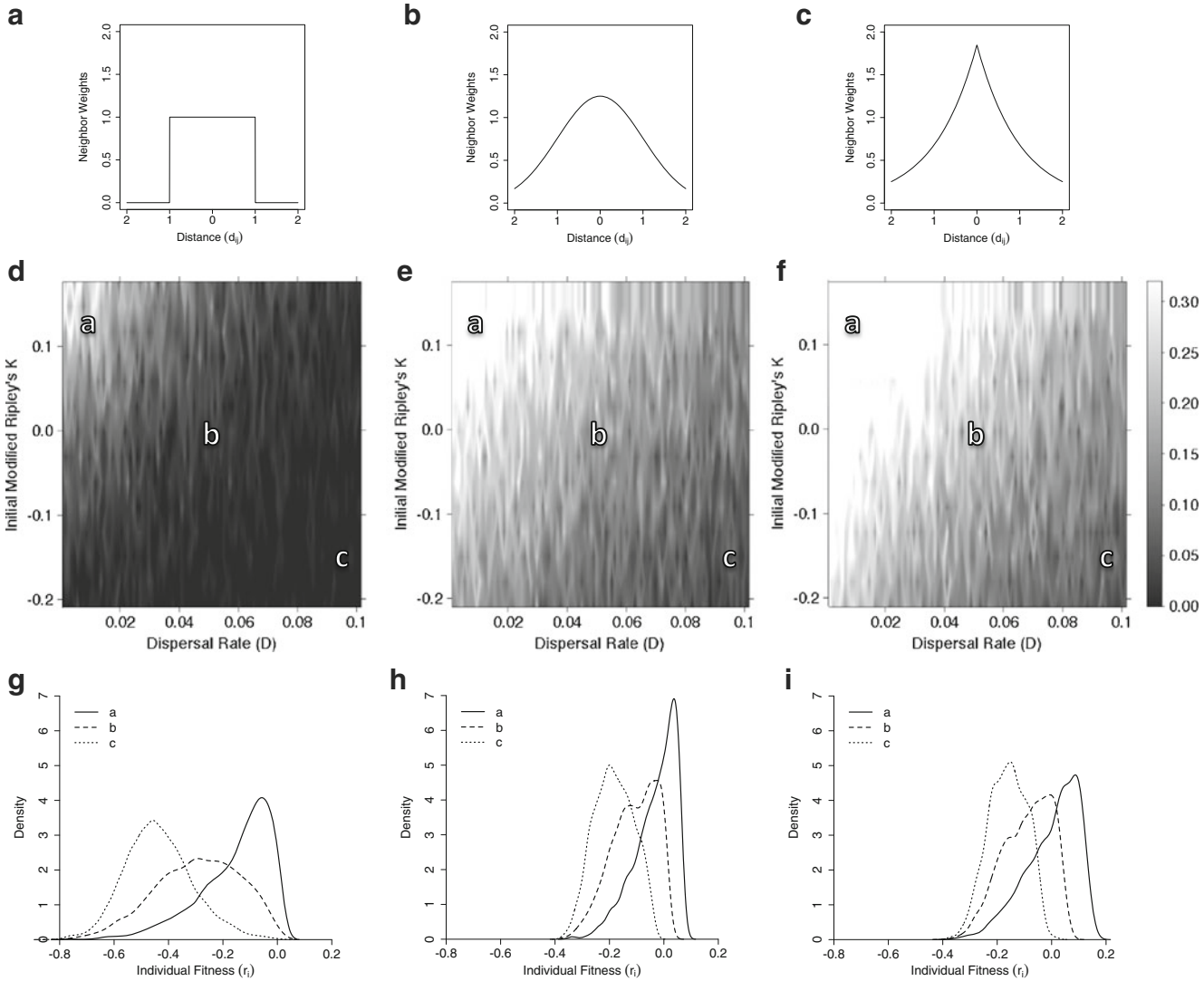

Fig. 1 The effect of initial spatial structure (modified Ripley's K for R=1) and dispersal rate (**d**) on individual fitness and overall invasion success of a population. Top-hat (**a**), Gaussian (**b**), or Laplace (**c**) indicate the local interaction kernel that was used to generate the results in the corresponding column. The probability of invasion success (**d**–**f**) (*white*, > 30 %) is calculated as the proportion of 100 realizations that show an increase in population size by the end of the simulation. The density subplots (**g**–**i**) correspond to the parameter combinations indicated by the letters **a**–**c** in the probability plot above and show the relative frequencies of individual fitness (r_i) at time of introduction (t= 0). The relative frequencies have been smoothed to be presented as continuous distributions of individual fitness generated using kernel density estimation with the "stats" package in R (v. 2.10.0, 2009)

Eq. (1), which is the diffusion component), the distance an individual moves in each coordinate direction is normally distributed with mean 0 and variance $2D\Delta t$ (Birch and Young 2006; Twomey 2007); Δt (the time since the last event) was generally sufficiently small to adequately approximate continuous movement. The direction moved is random, with all directions equally likely. Individual i's new coordinates after this bout of movement are given by:

$$x_i(t+\Delta t) = x_i(t) + \sqrt{2D\Delta t}\xi_x, \qquad (3)$$
$$y_i(t+\Delta t) = y_i(t) + \sqrt{2D\Delta t}\xi_y,$$

where ξ_x and ξ_y are independent zero-mean unit–variance Gaussian random deviates. (These are generated using $\xi_x = \sqrt{-2\ln U}\cos 2\pi\theta$, $\xi_y = \sqrt{-2\ln U}\sin 2\pi\theta$ where U and θ are uniform random variables in the interval [0,1)).

The top-hat interaction kernel, with finite boundaries, represents, in a sense, a restricted spatial scale within which individuals perceive their local population size, and outside of which they do not (i.e., they are short-sighted in sensing or experiencing effects of conspecific neighbors). We also implemented two other local interaction kernels (for calculating the effective number of neighbors, N_i, causing the density dependence experienced by each individual). These were a bivariate

Gaussian function, $N_i = \sum_j M \exp\left(-|d_{ij}|^2/2S_M^2\right)$, and a Laplace or back-to-back exponential function, $N_i = \sum_j W \exp\left(-S_W|d_{ij}|\right)$ (Fig. 1b and c, respectively). In these cases, S_M *and* S_W determine the width of the kernels and M and W are scalars that influence the weight that each individual j places on individual i as a determinant of its fitness components, given their Euclidean distance apart, d_{ij}. These parameter values (Table 1) were assigned in order to maintain consistency and to generate results broadly comparable across kernels, given similar initial conditions. Because the impact of the spatial scales of density dependence and dispersal have to be assessed relative to each other (Murrell 2006), we present our results with fixed widths for the local interaction kernels, while varying dispersal rates over the range that was found to produce any possibility of successful establishment.

The distance, d_{ij}, between individuals influences component Allee effects and individual fitness because reproduction and survival are based on local population size, where what counts as "local" for an individual is determined by the interaction kernel. In order to track distances between individuals and better gauge how their spatial distributions influence individual and population level behavior, we use a summary statistic—Ripley's K statistic, a second-order spatial point pattern metric that gauges deviations from spatial homogeneity (Ripley 1976). The traditional use of Ripley's K is to describe characteristics of mapped positions of points (in our case individuals) at different spatial scales. The concept is based on the density of individuals per unit area and the expected number of individuals (according to a Poisson distribution, for instance) in a circle of radius R centered on a randomly chosen individual. Depending on the actual spatial pattern of individuals, for a given radius R around an individual, the deviation from the expected number of individuals indicates the average local distribution of abundance experienced by each individual—and the scale at which overdispersion and clumping are observed. In our simulations, we fix R at a distance matching the interaction distance (and therefore characterize distributions only at this scale) and compare the expected and observed spatial distributions (as a standardized or modified Ripley's K) to interpret the spatial pattern of each population upon introduction and then over time.

Table 1 Parameters, their descriptions, and values used

Parameters	Definition	Value
a	Allee threshold	25
C	Carrying capacity	100
D	Dispersal rate	0.001 to 0.100
S_T	Width of top-hat kernel	1.0
M	Scalar for Gaussian kernel	1.25
S_M	Width of Gaussian kernel	1.0
W	Scalar for Laplace kernel	1.85
S_W	Width of Laplace kernel	1.0
R	Distance for Ripley's K statistic	1.0
$\widehat{K}_0(R)$	Initial Modified Ripley's K	−0.2 to 0.2

We measure Ripley's K as follows:

$$K(R) = \lambda^{-1} \sum_i \sum_{j \neq i} I\left(d_{ij} < R\right) h_i(R)/N \tag{4}$$

where N is the total number of individuals, λ their density (number of individuals divided by the total occupied area or 'study plot' in the simulation), and d_{ij} the Euclidean distance between individuals i and j. I is an indicator function that equals 1 if the distance between individuals is less than R and 0, otherwise. The total occupied area is the area of a circle encompassing the entire population, with the radius the distance from the center of the plot to the farthest individual. We correct for edge effects that arise because individuals located near the boundary of the study plot may have local neighborhoods that include areas within an area beyond the defined edge of the study plot. To account for this, following Fortin and Dale (2005), we incorporate a term $h_i(R)$, which is an edge correction weight, the reciprocal of the proportion of the area of the circle centered on i with radius R that is within the study plot (1 if the circle is completely in the study area).

In effect, $K(R)$ is proportional to the fraction of all individuals separated by distances less than R (Fortin and Dale 2005), which is directly related to the average local density for the top-hat interaction kernel. If individuals were distributed with complete spatial randomness (CSR), the expected value of $K(R)$ is πR^2 for a homogeneous Poisson process (Dixon 2002). For ease of interpretation, we focus on departures from CSR and use the modified Ripley's K (Fortin and Dale 2005), $\widehat{K}(R) = \sqrt{K(R)/\pi} - R$. Overall, $\widehat{K}(R)$ will take on negative values if individuals are overdispersed and will conversely become more positive with more spatial structure and clustering, with a zero value indicating CSR. We note that identical values of Ripley's K do not indicate identical spatial point patterns for different populations, but does indicate similar levels of aggregation.

For direct comparisons of the effects of spatial structure, dispersal rate (D), and the local interaction kernel on population dynamics, certain parameter values and initial conditions were maintained across all simulations (i.e., initial population size, maximum area of initial introductions, Allee threshold a, and carrying capacity C; Table 1). These values were chosen such that each introduced population was at the "tipping point" (Allee threshold) of critical size and area based on the analytically derived conditions of the deterministic demographic Allee effect model (Kanarek and Webb 2010). The other parameters and initial conditions (i.e., dispersal rate, initial modified Ripley's K) were varied

(Table 1). To group results by initial modified Ripley's K, we divided the range −0.2 to 0.2 into 20 equal intervals. For each interval, we initialized simulations with 25 individuals randomly placed in the unit circle centered at the origin and calculated the modified Ripley's K. If this was in the desired interval, the simulation was run, while if not, the points were discarded and the simulation re-initialized. Due to the stochastic nature of this model, this was repeated until 100 realizations were obtained for each combination of initial modified Ripley's K and dispersal rate for each local interaction kernel. Simulations were run until the population either went extinct, or a minimum of 100 time units had elapsed.

In order to focus on the relationship between an individual's extrinsic environment (local population size) and the intrinsic density-dependent birth–death process, we fixed a and C to be the same for each individual (since including further individual variability leads to evolutionary consequences, which is beyond the scope of this investigation). Given that every population is introduced into a similar context with individuals that are not intrinsically different, we can determine how and why emergent spatial heterogeneity in demographic rates due to stochastic variation in local densities becomes an important factor in the probability of establishment and spread.

Results

We observed the emergence of departures from population level, mean field behavior (especially with non-spatial models) when the strength of component Allee effects interacts with spatial structure and affects individual fitness. The outcome of this interaction is well illustrated by comparing Fig. 2a and b, which show two populations of identical invaders (D=0.05) with differing initial spatial structure. Initially, from the global perspective, overall density is the same (λ=7.96, 25 individuals in a unit circle). From the individual perspective, however, local density differs, as indicated by the modified Ripley's K statistic (at t=0, $\widehat{K}_0(R) = -0.07$ for Fig. 2a and $\widehat{K}_0(R) = 0.14$ for Fig. 2b). The subsequent snapshots through time reveal that when the initial population is small, the population with the more clumped initial distribution (Fig. 2b) succeeds in establishing. Population growth occurs because higher average local densities have more births than deaths (at least in some regions). The population with the less clumped initial distribution (Fig. 2a), with lower local densities, by contrast declines due to the Allee effect. Aggregation thus can minimize the negative impacts of Allee effects.

Figure 3a and b, on the other hand, show populations introduced with equivalent spatial structures (i.e., $\widehat{K}_0(R) = 0.04$), but different dispersal abilities (D=0.001 for Fig. 3a and D=0.1 for Fig. 3b). Increased dispersal rates destroy the spatial structure (i.e., $\widehat{K}(R)$ decreases over time), and the population inexorably goes extinct. Where individuals remain in close proximity to each other, because they are sluggish dispersers, they can mitigate component Allee effects and thus successfully establish, but they can spread only very slowly. The clumped distribution that influences this result emerges from constrained (slow) dispersal and is enhanced as more offspring are produced and remain within clusters. So given an Allee effect, the most likely invasive species may not be those with high inherent dispersal rates.

To further demonstrate this point, Fig. 4 illustrates the effect of dispersal rate on spatial structure over time. These simulations were all initialized with a similar spatial structure value describing their initial spatial distribution (i.e., $\widehat{K}_0(R) = 0.04$) and dispersal rate was varied. Out of 200 realizations for each dispersal rate, each trajectory represents the average modified Ripley's K over time, grouped by whether or not the population went extinct. It should be noted that the proportion of extinctions increased from approximately 60–90 % as the dispersal rate increased from 0.001 to 0.10. Although increased dispersal reduces the spatial structure and aggravates, the population can nonetheless at times succeed by chance, but not as readily as one that maintains a higher degree of clustering. Likewise, even though reduced dispersal helps retain spatial structure, establishment is certainly not guaranteed; the population (due to other sources of stochasticity) can easily fail to establish, even if individuals are sufficiently clustered to alleviate the Allee effect.

These plots (Figs. 2 and 4) not only demonstrate that initial spatial structure and dispersal rate can influence establishment, but that there can be additional effects on persistence and spread. Even though the spatial distribution can vary for different instantiations of populations with identical initial Ripley's K values, when a particular realization maintains a relatively constant Ripley's K value with increasing population size, the population is essentially pinned and will undergo little growth and spread. In the cases of slow dispersal (Figs. 3a and 4), the preserved or emergent (depending on initial conditions) spatial structure often leads to a greater chance for successful establishment; however, once a cluster reaches carrying capacity, spread is generally halted or greatly slowed (Ripley's K stays constant, Figs. 3a and 4). At this point, there is a chance that rare dispersal events may eventually give rise to the formation of other clusters, although it is difficult for individuals to move far enough away from dense local interaction neighborhoods to both reduce mortality by limiting competitive interactions and still successfully reproduce (due to component Allee effects). Figures 2 and 4 show that with intermediate dispersal (D=0.05), there is less probability for

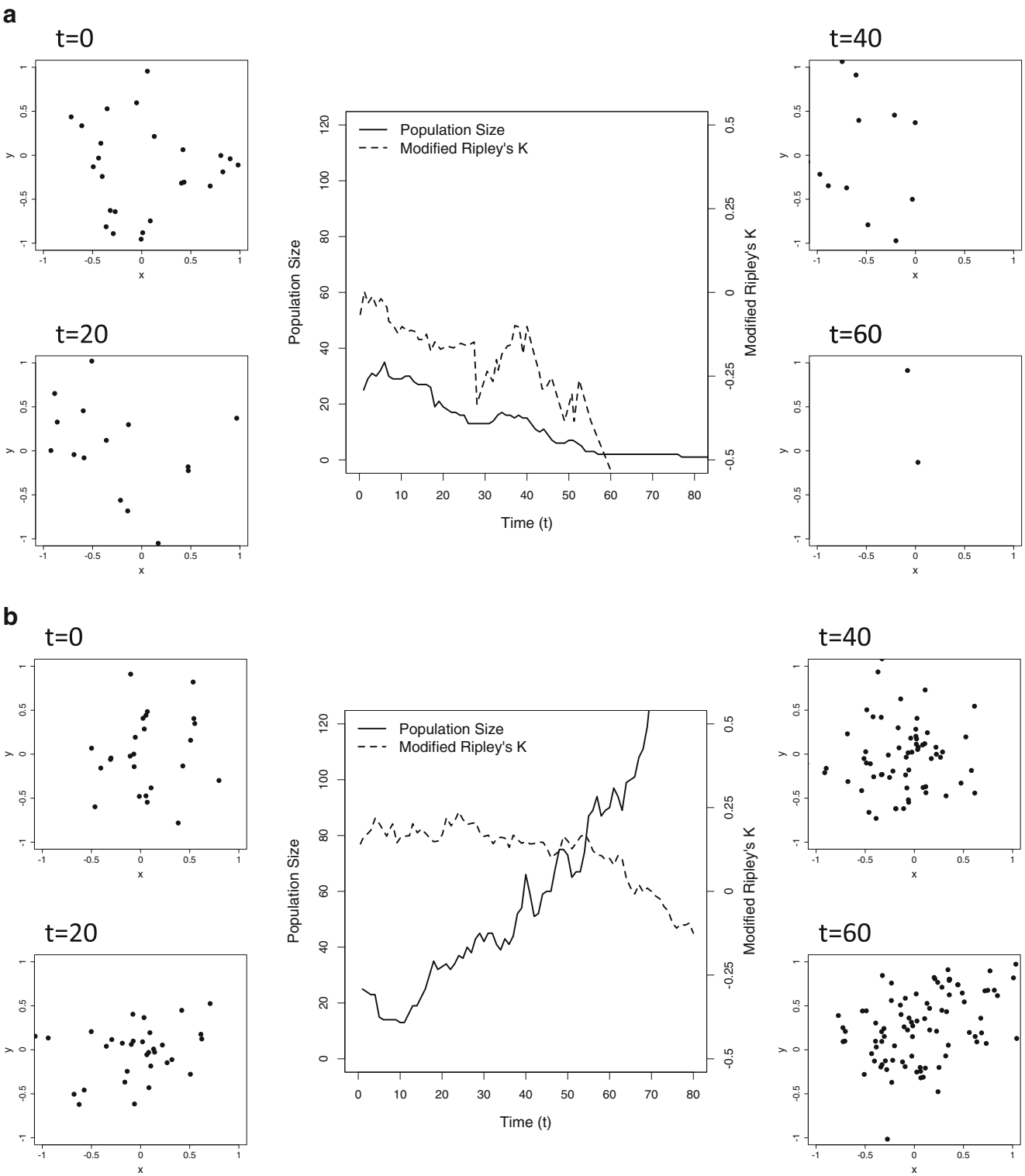

Fig. 2 Realizations of simulations of populations where individuals are identical except for initial spatial structure. In each case, temporal snapshots show how individuals are distributed across space following the introduction of 25 individuals within a unit circle. The corresponding graphs show the temporal dynamics describing how population size and spatial structure (modified Ripley's K) change. In **a**, the dispersal rate is the same as in **b**, where D=0.05; however, individuals are initially overdispersed in **a** with $\widehat{K}_0(R) = -0.07$, and this loosely structured population declines steadily to extinction. Individuals in **b**, on the other hand, are tightly clumped initially, with $\widehat{K}_0(R) = 0.14$, and maintain this level of aggregation until the population successfully grows. The top-hat local interaction kernel was used and other parameter values are given in Table 1

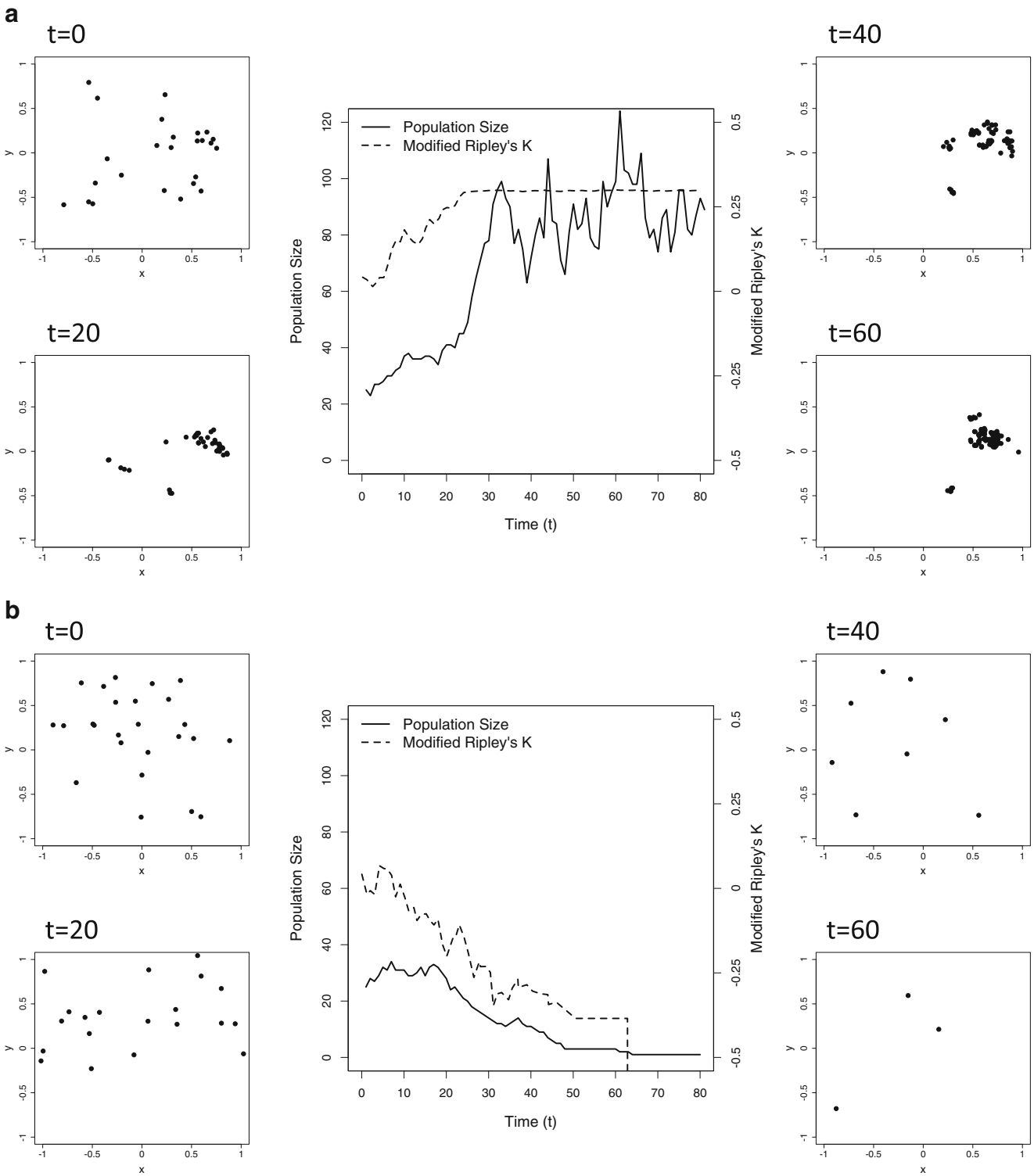

Fig. 3 Realizations of simulations of populations where individuals are identical except for dispersal rate. In each case, temporal snapshots show how individuals are distributed across space following the introduction of 25 individuals within a unit circle. The corresponding graphs show the temporal dynamics describing how population size and spatial structure (modified Ripley's K) change. Individuals in **a** and **b** have similar initial spatial structure [the same $\widehat{K}_0(R) = 0.04$], but differ in dispersal ability with D=0.001 and 0.1, respectively. In these cases, slow dispersal in **a** leads to spatial aggregation and population growth, whereas fast dispersal in **b** destroys spatial structure and leads to population decline. The top-hat local interaction kernel was used; other parameter values are given in Table 1

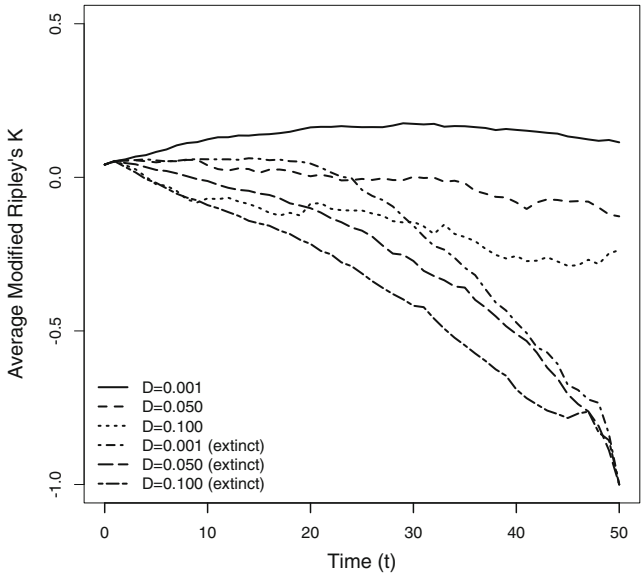

Fig. 4 The effect of dispersal rate (D) on spatial structure (modified Ripley's K) over time. Each simulation was initialized with similar spatial structure [$\widehat{K}_0(R) = 0.04$], while dispersal rate was varied from slow (D=0.001) to intermediate (D=0.05) to fast (D=0.1). Average modified Ripley's K values over 200 realizations for each dispersal rate are grouped by whether the population went extinct or succeeded. 40 % succeeded for D=0.001, 20 % succeeded for D=0.05, and 10 % succeeded for D=0.1

survival, but in this case, following initial establishment, the population can continue to grow and spread as the spatial structure gradually decreases. With rapid dispersal, establishment only really occurs if there is sufficient initial spatial structure (Fig. 1) and growth can occur rapidly enough to keep up with overdispersion (Fig. 4 where D=0.10). In any event, there is a clear trade-off that emerges between growth, establishment, and spread.

Overall, in drawing the connection between individual fitness and demographic Allee effects, we have presented the impact of spatial structure on individual fitness as a modulator of the probability of population success. Figure 1 demonstrates this by showing that spatial structure influenced by dispersal rate interacts with component Allee effects and scales up to impact population persistence. The probability of invasion success (denoted by the color bar on the side of Fig. 1f) is calculated as the proportion of the 100 realizations for each parameter combination (modified Ripley's K and dispersal rate) that show an increase in population size after a total time of 100 time units [i.e., $N(t=100)>N(t=0)$]. Only 25 individuals (the Allee threshold) are initially introduced, so few or none of these have fitness greater than 0, and extinction is likely. The probability of invasion success is highest with high spatial clustering and short-range dispersal (top left corner) and lowest with overdispersion and a high dispersal rate (bottom right corner). These dynamical outcomes are further evident with probability density plots of initial individual fitness r_i (Fig. 1g, h, and i) across 100 realizations for each parameter combination, indicated by the placement of the letters a–c in the corresponding plots for the probability of invasion success (Fig. 1d–f). It is clear that the probability of establishment decreases from top left to bottom right, primarily because initial individual fitness is generally too low (i.e., death rates outweigh birth rates) unless there is sufficient spatial structure. As the initial spatial structure increases, mean individual fitness increases, while the skewness of the distribution shifts from positive to negative, with many individuals more likely to reproduce than die. New individuals are born at the location of their parents, which increases clustering, while dispersal tends to decrease clustering and therefore depresses average local fitness.

Durrett and Levin (1994) claimed that "one should not worry too much about what neighborhood to choose [since] in most cases, qualitative behavior of the model does not depend on the neighborhood used." Our results in Fig. 1d–f are consistent with this perspective. We found that spatial structure becomes less important as the local interaction kernel is varied from top-hat to normal to exponential, but the population dynamics are qualitatively similar for all the different kernels. This is shown across Fig. 1, as the contour plot shifts with the particular kernels opening up a larger range of parameter space that lead to higher probabilities of success. As individuals with fat-tailed interaction kernels detect conspecifics beyond the restricted spatial scale of the top-hat perspective, there is less effect of local spatial structure on population growth, the component Allee effects merge into demographic Allee effects, and departures from mean-field predictions are not as striking.

Discussion

To investigate how spatial structure interacts with component Allee effects and scales up to impact demographic rates and population establishment, we have taken a bottom-up approach to a population model by summing over the spatially shifting effects of density dependence experienced by individuals in limited neighborhoods. The extent to which a population was clustered proved to be of key importance in relaxing the potentially negative impacts of component Allee effects on population establishment and persistence. We observed notable departures from mean-field models, since individuals did not encounter each other in proportion to the average density across the population (Law et al. 2003). With spatial structure and limited dispersal, individuals were sometimes able to mitigate component Allee effects based on chance vicissitudes in the density of their locally experienced, as opposed to globally measured, neighborhoods, and the population could avoid extinction. Future extensions of our studies should examine a range of alternative scenarios, such as movement

rates that depend on local density, different functional forms for birth and death rates, and different spatial scalings for the localized density dependencies in births and deaths. One particular mechanism leading to Allee effects, which might be particularly valuable to consider, are two-sex models, where isolated individuals are unlikely to mate.

We observed that spatially localized interactions play a significant role in the successful establishment of a population. Because the number of conspecifics within an individual's interaction neighborhood, based on the spatial structure of the population, impacts individual fitness (i.e., birth and death rates), initial spatial distribution upon introduction is a key determinant of successful establishment. We further demonstrated that dispersal rate strongly influences the spatial structure over time and hence becomes an additional important factor affecting not only establishment but future invasion dynamics as well. High dispersal tends to move individuals away from temporary clusters (and offspring away from parents), aggravating the demographic costs represented in component Allee effects. Low dispersal can mitigate the Allee effect, but hamper movement of an invasive species beyond its initial beachhead. Furthermore, we observed that populations declining towards extinction undergo distinct, systematic shifts in their spatial structure (Fig. 4). Overall, our examples illustrate a range of scenarios for which component Allee effects may either be suppressed (where the population succeeds in establishment) or result in depressed demographic growth rates leading to extinction. There is thus a crucial feedback between the spatial pattern of individuals across the landscape, and the emergent dynamics and ultimate fate of the population (Durrett and Levin 1994; Bolker and Pacala 1997).

This feedback has previously been recognized in terms of trade-offs between growth and spread for survival of a population in a patchy habitat (Skellam 1951; Kierstead and Slobodkin 1953; Okubo 1980; Murray 1993). The idea was originally couched in terms of phytoplankton blooms that arise when a critical area is occupied such that the population avoids extinction by overcoming the dilution effect (i.e., where sufficient reproduction compensates for the loss due to diffusion into unfavorable habitat; Skellam 1951; Kierstead and Slobodkin 1953; Okubo 1980). By shifting the focus from the exogenous environment (and discrete patches of differing intrinsic quality), Allee effects in effect generate this same dynamic, constraining further range expansion at the periphery when populations are too small, are too short lived, or produce too few propagules because of reduced net reproduction (Hengeveld and Hemerik 2002), and at times precluding invasion in a continuous, homogeneous landscape (as shown here) or even into seemingly favorable regions. This behavior is a fundamental ecological and spatial consequence of Allee effects (i.e., an invasion front that is a result of being "pushed" from the inside out, as opposed to being "pulled" by the leading edge; Lewis and Kareiva 1993; Keitt et al. 2001; Kanarek and Webb 2010). Our simulations are consistent with this basic concept where, due the apparent breakdown of spatial structure by long distance dispersers, individual fitness is depressed, scaling up to generate population decline and eventual extinction.

Our investigation has focused on the early stages of invasion (i.e., introduction and establishment; Williamson 1996) and subsequent tradeoffs when spatially constrained dispersal and interactions also influence long-term dynamics. As limited dispersal allows individuals to overcome component Allee effects, a trade-off between positive and negative density dependence emerges when the population grows close to carrying capacity for a given cluster of individuals. We have observed patterns shaped by competition where clusters at carrying capacity are separated far enough to reduce interaction. This scenario likely emerges due to our assumption that the interaction kernel is the same for both reproduction and survival. When we implemented different interaction kernels, we found that this decoupling of the spatial kernel describing these different components of fitness generally influenced the long-term spatial patterns, rather than establishment, since the individual birth rates are primarily affected by low density and death rates at high density (cf. Stewart-Cox et al. 2005). Furthermore, we have shown that constraints on dispersal rate contribute to successful invasion by weakening Allee effects, and subsequently result in slower overall population growth and spread following the initial transient phase of establishment. In this sense, our model is complementary to other theoretical work (reviewed by Courchamp et al. 2008) that suggests that spatial consequences of Allee effects can include critical spatial thresholds, slower spread rates, accelerating and patchy invasion, range pinning, and pulsed range expansion. None of these previous models, however, have investigated both establishment and spread while emphasizing the emergent trade-offs that result from the interaction between component Allee effects and spatial structure.

The existence of Allee effects has been recognized empirically in a number of different taxa (Courchamp et al. 2008), and specifically the role of Allee effects in biological invasions has been noted (Taylor and Hastings 2005). For example, the invasion of the gypsy moth across the USA proceeded in a series of temporal pulses, due to strong mate-finding Allee effects requiring high density prior to spread (Johnson et al. 2006). Interestingly, the Allee threshold has been shown to differ depending on the environment, due to the effectiveness of pheromone transmission, and hence Allee effects have slowed invasion speed or even reversed the invasion, forcing range contraction in certain locations (Tobin et al. 2007). Furthermore, Robinet and Liebhold (2009) suggest that dispersal capabilities interact with Allee effects and consequently affect the establishment of gypsy moths. They found that the

growth rate and probability of establishment of populations of gypsy moths with flightless females is considerably higher than in populations with females fully capable of flight (Robinet and Liebhold 2009). Generally, in species where density is critical to persistence, aggregations can enhance reproductive success through mate finding and fertilization efficiency, and increase survival through environmental conditioning, predator dilution, antipredator behavior, and increased foraging efficiency (Stephens and Sutherland 1999; Berec et al. 2007; Courchamp et al. 2008; and references therein). It is likely that component Allee effects influence individual fitness, but whether or not demographic Allee effects are thereby present and population dynamics are affected depends on how local intraspecific interactions are modulated by dispersal and the spatial scale of the interactions (see, e.g., Berec et al. 2001 for an explicit mechanistic approach that demonstrates shifting extinction boundaries).

In conclusion, we have shown that because the strength of component Allee effects vary in time and space, the potential for spatial clustering and spatially dependent interactions can mitigate a significant reduction in the overall mean individual fitness and thus inhibit the emergence of strong demographic Allee effects and population decline. This qualitative result, however, suggests that even though a small founder population has an opportunity to successfully establish, further assumptions are required to better understand whether a successful invasion ultimately occurs. Too little or too much dispersal can lead to invasion failure because either a population becomes trapped or goes extinct. Between these two extremes is the possibility that an introduced population is comprised of individuals that interact and disperse on relative spatial scales, striking a balance between the formation of local aggregations and spread. In the event that a population establishes but fails to spread, the pronounced time lag that ensues may provide the opportunity for adaptive evolution to contribute to the long-term success of an invasive species (Kanarek and Webb 2010). Recognizing constraints on dispersal and the subsequent limitations on spread not only contributes to our understanding of how individual behavior affects population level dynamics, but provides valuable insight for invasion risk analysis.

Acknowledgments We would like to thank Michael Buhnerkempe and the rest of the Webb Lab, Dan Ryan, Ben Bolker, Greg Dwyer, and three anonymous reviewers for helpful comments and advice. ARK acknowledges funding from NSF-IGERT Grant DGE-#0221595 administered by PRIMES at CSU, travel grants from the Global Invasions Network NSF-RCN DEB-#0541673 for facilitating this collaboration, and by the National Institute for Mathematical and Biological Synthesis, an Institute sponsored by NSF, the U.S. Department of Homeland Security, and the US Department of Agriculture through NSF Award #EF-0832858, with additional support from The University of Tennessee, Knoxville. RDH and MB thank the University of Florida Foundation for support.

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