How mutation shapes the rate of population spread in the presence of a mate-finding Allee effect

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Abstract

Although past work has considered how evolution and Allee effects each shape population spread, these factors have rarely been considered together. We develop an integrodifference equation model that tracks individuals of multiple dispersal types (i.e., short- and long-distance dispersers) of male and female individuals subject to a strong Allee effect due to mate-finding process. We use our model to explore how mutation between different dispersal types affects the rate of population spread, since this evolutionary mechanism has been shown to lead to both faster and slower spread in a previous individual-based model. We ask, under what conditions does mutation cause the population to spread faster (or slower) than it spreads without mutation (from the same initial conditions)? We find that mutation can both speed up and slow down invasions. Speeding up occurs in a relatively small range of parameter space near the Allee threshold of the population. Slowing down occurs across a broad range of parameters.

Introduction

It is increasingly recognized that the spatial spread of populations is shaped not only by ecology, but also by evolution (Phillips 2015; Miller et al. 2020). Here, "evolution," very generally, refers to a change of traits over time. Such a change can occur via a number of mechanisms. In spreading populations, these mechanisms include spatial sorting (individuals sort by dispersal ability), spatial selection (selection varies with population density which varies spatially), mutation (changing from one type to another), and gene surfing (stochasticity at the low-density population edge allows the persistence of types that are not otherwise favored) (Edmonds et al. 2004; Phillips et al. 2008; Shine et al. 2011). A key question is whether we can draw generalities about the effects of evolution on spread dynamics, and on spread speed in particular. A majority of modeling studies suggest that "evolution speeds up population spread," based on the observation that, over time, individuals with higher dispersal ability come to dominate the population edge (Travis and Dytham 2002; Travis et al. 2009; Burton et al. 2010; Bénichou et al. 2012; Ramanantoanina et al. 2014; Williams et al. 2016). The generality of the statement, however, sometimes eschews which mechanism(s) of evolution (see above) were present in the corresponding study or responsible for the outcome. At least two models have found the opposite effect that evolution can slow population spread (Korolev 2015; Shaw and Kokko 2015). In both of these studies, the population dynamics included an Allee effect. We use an integrodifference model to shed more light on how one particular evolutionary mechanism, mutation, interacts with population dynamics to result in faster or slower spread rate of populations.

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Arguably the most prominent example that evolution can increase population spread rate is the invasion of cane toads in Australia (Phillips et al. 2006), which has inspired more research and models than we can list here. Perkins et al. (2013) formulate and simulate a very complex model that combines population dynamics and dispersal in an integrodifference equation with quantitative genetics and reproduces the patterns observed in the data. Several reaction-diffusion models abstract much of the complex biological processes and study the question more generally. In these models, the diffusion coefficient is a continuously distributed trait that evolves neutrally, modeled by a diffusion operator in trait space (Bénichou et al. 2012; Bouin et al. 2012; Bouin and Calvez 2014). Those models show that at the leading edge of the population front, the distribution of diffusion coefficients is skewed towards higher values, that the trait-structured population spreads faster than an unstructured population with the average trait value, and that the population spread rate may even accelerate over time (Bouin et al. 2017). Even the addition of moderate mortality, penalizing long-distance dispersers, does not significantly affect front acceleration (Bouin et al. 2018). An even simpler integrodifference spread model of only finitely many dispersal types and clonal reproduction (without mutation or type switching) also found front acceleration and spatial sorting (Ramanantoanina et al. 2014). Even if only very few individuals with high dispersal ability were initially present, those would eventually end up at the invasion front and determine the (high) spreading speed.

A different mechanism of increased spread rates has been found in reaction-diffusion and integrodifference equations with two or finitely many types. If types cooperate then a multi-type population can spread faster than any of the component types ("anomalous spreading speed") (Weinberger et al. 2007). Since a mutation process often leads to cooperative dynamics between phenotypes (at least at low densities), the spread speed of populations with two or more dispersal types can be higher than the speed of each type on its own, even if the mutation rate between types is very small (Elliott and Cornell 2013; Keenan and Cornell 2021). This mechanism can only occur if there is a trade-off between dispersal ability and fecundity (Lutscher 2019).

To date, most of the theory on spread rates with multiple types (in particular all the references mentioned in the preceding paragraph) has assumed that the highest population growth rate occurs at low density, i.e., that there is no Allee effect. While this assumption is very convenient for mathematical analysis, it is often not realistic. Allee effects are common in biological systems, and occur when individual fitness (e.g., per capita growth rate) increases with population density (Stephens et al. 1999). Perhaps the best-studied class of examples is a mate-finding Allee effect, where the challenge of finding mates at low density can lead to lower growth (Gascoigne et al. 2009). Since population density

is, by definition, low at an invasion front, and since several aspects of evolution are closely related to mate-finding, we need to consider the evolution of spread rates in multi-phenotype populations under Allee effects.

How Allee effects impact population spread in the absence of evolution has been studied for a long time; e.g., Weinberger (1982). Similarly, the impact of mate-finding on spread rates has been studied with and without Allee effect (Miller et al. 2011; Shaw et al. 2018). To examine how an Allee effect interacts with spread rates of different types, Korolev (2015) studied a pair of reaction-diffusion equations, one for each dispersal phenotype, and with strong Allee effect, modeled by a cubic nonlinearity. By linearizing the equations at a single-type traveling wave and studying invasion conditions for the other type, he showed that when the Allee threshold is high, types with lower dispersal rate could invade, so that the population spread rate decreased. The underlying mechanism is that a high Allee threshold disadvantages a long-distance disperser as their density will necessarily be below that Allee threshold ahead of the front. Korolev (2015) considered mutations to occur on a much slower time scale than ecological processes and did not distinguish individuals by sex. Shaw and Kokko (2015) studied a similar question with very different means. They used an individual-based model that included dispersal, mate-finding, reproduction, mutation, and density dependence. The evolving attributes of individuals were related to dispersal behavior: how likely is an individual to settle in a certain location, given the number of individuals of the same or the opposite sex present at that location? They found that when resource competition among females was low, evolution acted to alleviate the mate-finding Allee effect and slow the rate of population spread. However, when resource competition among females was high the opposite occurred: evolution sped up the rate of population spread, exacerbating the Allee effect. While the model by Korolev (2015) abstracts much biological realism to obtain analytical results, the model by Shaw and Kokko (2015) includes too much detail to have such results. We present a model of intermediate complexity that explicitly includes mate-finding and evolution but assumes a much simpler dispersal process than the one in Shaw and Kokko (2015). While we do not have analytic solutions, our model is simple enough to draw general conclusions about how evolution can speed up or slow down population spread.

We develop an integrodifference equation model that tracks individuals of multiple dispersal types (i.e., short- and long-distance dispersers) of male and female individuals. We include mate-finding by using a submodel to describe the pair formation process (Veit and Lewis 1996; Shaw et al. 2018). This process induces a strong Allee effect. We use our model to explore how mutation as one specific mechanism of evolution affects the rate of population spread, in the presence of this mate-finding Allee effect. We focus on mutation since

this mechanism has been previously shown to lead to faster or slower population spread in a more complex individual-based model (Shaw and Kokko 2015). We consider the scenario of a population of organisms adapted to equilibrium conditions near their population capacity, and then explore what happens when a subset of individuals are introduced to a novel habitat, as was done by Shaw and Kokko (2015). We then ask: under what conditions do simulations with mutation in dispersal parameters spread faster (or slower) than simulations without mutation? We find that mutation can both speed up and slow down invasions. In fact, slowing down invasions occurs across a broad range of the considered parameter space.

Model derivation

We begin by formulating the simplest possible model that contains all the relevant processes. We assume a semelparous population whose life cycle is divided into a synchronized growth phase (during which spatial movement is negligible) and a synchronized dispersal phase (during which population dynamics are negligible, but see "Dispersal-induced mortality"). The order of life cycle events are the following: pair formation, offspring production, death of the parent generation, density-dependent offspring survival, dispersal. We census the population immediately after the dispersal phase. We track the continuous density of females (F_i) and males (M_i) of dispersal type $i = 1, 2$. We denote the total female and male population as $F = F_1 + F_2$ and $M = M_1 + M_2$. The total population is $F + M = 2N$. (We will show later that the density of female and male individuals are the same, except possibly at the initial condition. Hence, we can then write N instead of either F or M.)

Reproduction

Reproduction consists of pair formation, offspring production, and density-dependent survival. We use the same mechanistic approach to pair formation as Veit and Lewis (1996), and Shaw et al. (2018). Females and males, independent of dispersal type, search for monogamous mating partners locally according to a mass-action law. If there are initially F female and M male individuals, then the number of pairs formed after τ time units is given by

$$P(F, M) = \frac{FM(1 - \exp((M - F)\tau))}{F - M\exp((M - F)\tau)}, \quad \text{if} \quad M \neq F \quad (1)$$

and

$$P(F, M) = \frac{(F + M)^2}{F + M + 1/\tau} = \frac{N^2}{N + 1/\tau} = P(N), \quad \text{if} \quad M = F = N. \quad (2)$$

Each pair will produce b offspring with a 1:1 sex ratio. Hence, the number of female and male offspring is $bP/2$ each.

The parents die after offspring production. There is a local limiting density of n surviving offspring. Hence, the number of surviving female and male offspring from F female and M male parents is $\frac{1}{2}\min\{bP(F, M), n\}$ each. This process of searching for mates introduces a potential mate-finding Allee effect (Stephens et al. 1999; Shaw et al. 2018).

Inheritance

Individuals differ with respect to dispersal. We assume a haploid system with only two types ($i = 1, 2$). Mating then occurs in four possible pairs: (1,1), (1,2), (2,1), and (2,2), where the first (second) entry indicates the female (male) type. We introduce the proportions of females (males) of type 1 as $f = F_1/F$ ($m = M_1/M$). Hence, under random mating, the frequency of (1,1) pairs is given by fm and similarly for the other cases. Each offspring inherits its dispersal type from one of its two parents at random with equal probability. If there is mutation, then an offspring of any type may switch to the other type with a mutation probability that we denote by μ. The fraction of offspring of type 1, T_1, is then given by

$$\begin{aligned} T_1 = T_1(f, m) &= (1 - \mu)fm + f(1 - m)/2 \\ &\quad + (1 - f)m/2 + \mu(1 - f)(1 - m) \\ &= \frac{f + m}{2} + \mu(1 - f - m). \end{aligned} \quad (3)$$

Inheritance is assumed to be independent of sex, so that females and males are equally likely to be of dispersal type 1 or type 2.

Dispersal

We consider an infinite one-dimensional landscape and denote spatial location by x. Dispersal is modeled by a dispersal kernel, $\tilde{K}(x, y)$, so that $\tilde{K}(x, y)\Delta x$ is the probability that an individual moves from location y to $[x, x + \Delta x)$. Since we assume a homogeneous landscape, dispersal depends only on distance. We therefore write $K(x - y) = \tilde{K}(x, y)$. Many different dispersal kernels have been suggested in the literature (Nathan et al. 2012; Bullock et al. 2017). We shall not be concerned with the effects of different dispersal kernels, but we shall assume throughout that all kernels are exponentially bounded. In all our simulations, we consider Gaussian kernels. We shall characterize different types by different dispersal distances. Hence, we use

$$K_i(x) = \frac{1}{\sqrt{2\pi d_i}}\exp\left(\frac{-x^2}{2d_i}\right), \quad (4)$$

with variance $d_i > 0$, $i = 1, 2$. Without loss of generality, we may choose $d_1 < d_2$, so that type-1 individuals are

short-distance dispersers (hereafter "short type") and type-2 individuals are long-distance dispersers ("long type").

The full model

To project the densities of short type and long type females and males from one generation to the next, we put all the preceding steps together. We denote by $F_i^{(t)}(x)$ and $M_i^{(t)}(x)$ densities of female and male individuals of type i immediately after the dispersal phase in generation t. Then the densities of the two types of females in the next generation are

$$F_1^{(t+1)}(x) = \frac{1}{2} \int K_i(x - y) \min\{bP(y), n\} T_1(y) \mathrm{d}y \qquad (5)$$

$$F_2^{(t+1)}(x) = \frac{1}{2} \int K_i(x - y) \min\{bP(y), n\} (1 - T_1(y)) \mathrm{d}y, \quad (6)$$

with pair formation and type frequency given by

$$P(y) = P(F^t(y), M^t(y)), \qquad (7)$$

$$T_1(y) = T_1(f(y), m(y)), \qquad (8)$$

as in (1) or (2) and (3) and

$$F^t(y) = F_1^t(y) + F_2^t(y), \quad M^t(y) = M_1^t(y) + M_2^t(y), \qquad (9)$$

$$f(y) = \frac{F_1^t(y)}{F^t(y)}, \quad m(y) = \frac{M_1^t(y)}{M^t(y)}. \qquad (10)$$

Since we assumed a 1:1 offspring ratio and since sex and dispersal type are independent, the densities of males of each type in subsequent generations equals the densities of females in that type, even if this is not true in the initial generation. Hence, $M_i^{(t)} = F_i^{(t)}$ for $i = 1, 2$ and $t > 1$, and pair formation is given by the simpler formula $P(F, M) = P(N)$ in (2).

Nonspatial dynamics

Before we explain the setup that we use to determine how mutation affects population spread in our system, we consider a few model simplifications to understand the most basic properties of our model. In the absence of spatial structure, when there is no difference between dispersal types (i.e., $d_1 = d_2$), the model collapses to the very simple difference equation

$$N^{(t+1)} = \min\{bP(N^{(t)}), n\} \qquad (11)$$

with $P = P(N)$ as in (2); see Fig. 1, left plot. This model has a strong Allee effect: There are three steady states. The extinction state ($N = 0$) and the carrying capacity ($N = n$) are locally stable, the (intermediate) Allee threshold ($N = (\tau(b - 1))^{-1}$) is unstable. Since P is a monotone function of N, solutions of (11) are monotone. Populations decay to extinction if their initial density is below the Allee threshold and increase to carrying capacity if the initial condition is above.

Vice versa, when we set the total population to carrying capacity we can consider the trait dynamics independently.
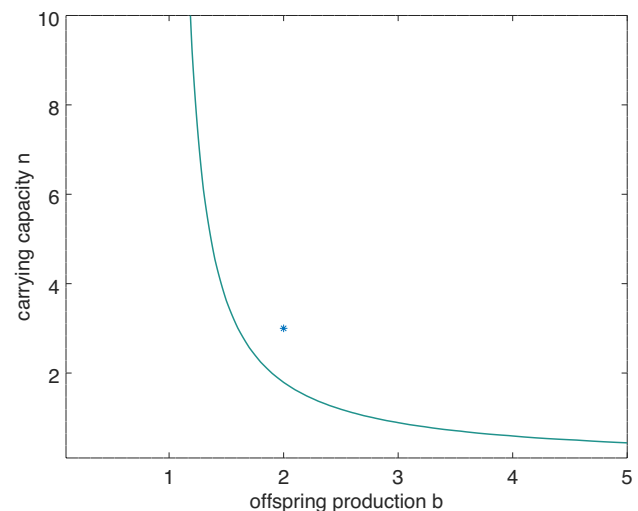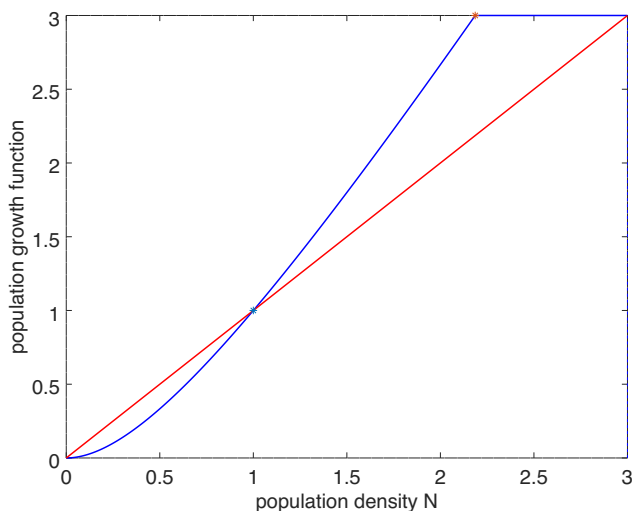

Fig. 1 Left: The blue curve represents the single-population growth function $\min\{bP(N), n\}$. The red line is the 45 degree line. The traveling wave speed is positive if the signed area between the two curves is positive. Right: The threshold in b-n-space that separates popula-tion advance (above the curve) from population retreat (below the curve). The star indicates the parameters on the left: $b = 2$ and $n = 3$. We fixed $\tau = 1$

Since the fraction of females and males of the two types are the same after the initial generation, we consider only the fraction of short type females, denoted by f. According to (3), this fraction changes according to

$$T_1(f) = f + \mu(1 - 2f)$$

from one generation to the next. The function T_1 has $f = 1/2$ as the only fixed point when $\mu \in (0, 1)$. This point is stable. Hence, if there is some mutation, then the two types will approach equal densities. If there is no mutation, the frequencies will not change.

When we combine the two preceding observations, we find that if $\mu \in (0, 1)$, our nonspatial model of two types and two sexes will either converge to the extinction state or to the state where each of the four densities (female/male, type 1/2) is exactly $n/4$. We will use this result later to set initial conditions for the simulations of spreading populations.

Spread of a single type

When we include space and dispersal into the simple model (11), we obtain

$$N^{(t+1)}(x) = \int K(x - y) \min\{bP(N^t), n\} dy, \quad (12)$$

with $P(N) = N^2/(N + 1/\tau)$ as in (2). The spatial spread behavior of this model is known (Weinberger 1982; Wang et al. 2002). If the initial population density is too low or the initial extent of the population is too small, the population will go extinct. If the initial density is high enough on a large enough spatial extent, the population will spread in space. The equation has constant-speed traveling wave solutions that connect the carrying capacity with the extinction state. If

$$\int_0^n \min\{bP(N), n\} dN > n^2/2, \quad (13)$$

then these waves have positive speed, i.e., the population advances. If the reverse inequality holds, the speed is negative and the population retreats. The value of the integral is increasing in b and n. The threshold, in b–n parameter space, between spread and retreat is plotted in Fig. 1, right plot. By rescaling b and n, we can achieve $\tau = 1$, which we shall assume from hereon.

Setup of the simulations

In the case without Allee effect, the asymptotic speed of spread is typically linearly determined and given by a relatively simple explicit formula (Weinberger 1982; Girardin 2017). Unfortunately, there is no such formula in the case with Allee effect (for a rare exception, see Kot et al. (1996)).

We will therefore rely on numerical simulations. We use the convolution approach, combined with fast Fourier transform to simulate the model until a constant-speed traveling wave is established and calculate its speed; see Lutscher (2019), chapter 8.

The important question is how to choose initial conditions. Since our model has an Allee effect, these initial conditions can affect the final simulation outcome. We choose our initial conditions following the work by Shaw and Kokko (2015) with our specific question in mind (see "Introduction"). We assume that the population is well adapted locally and then introduced into novel territory, and we test whether the spread speed in the novel territory is higher with or without mutations. Hence, we initialize population densities at the stable positive state of the nonspatial model in the presence of mutation. This is also the state that we expect to arise in the wake of a spreading population with mutation.

We consider a localized initial condition, where the densities are initially positive only on a small interval, and a wave-like initial condition, where the densities are positive on a half line. In both cases, the initial densities are constant over the interval where they are positive. In the simplest case, as described above, each of the four types has density $n/4$. We then run two simulations: one with mutation ($\mu > 0$) and one without ($\mu = 0$), and we compare the two speeds.

Results

Spreading speed(s), with and without mutation

In all our simulations, we observed two possible behaviors. With mutations ($\mu > 0$), the entire population developed into a single traveling wave, i.e., all four components (short and long type females and males) eventually traveled at the same speed. This observation is not too surprising. Biologically speaking, since each type generates the other type by mutation, it seems obvious that neither can evade the other. Mathematically, results of this type have been proven for cooperative systems (Lui 1989), and a positive mutation probability gives rise to cooperative elements in our model, although the overall model is not cooperative. Without mutation, however, we never observed the two types spreading together. Eventually, there was always one type that raced ahead while the other stalled. While we cannot prove it, our many simulations lead us to conjecture that the joint speed of propagation of two types with mutation is bounded between the speeds of either type in isolation.

Which type dominates the front?

Which of the types managed to outrun the other in the absence of mutation depended on parameters. By way of
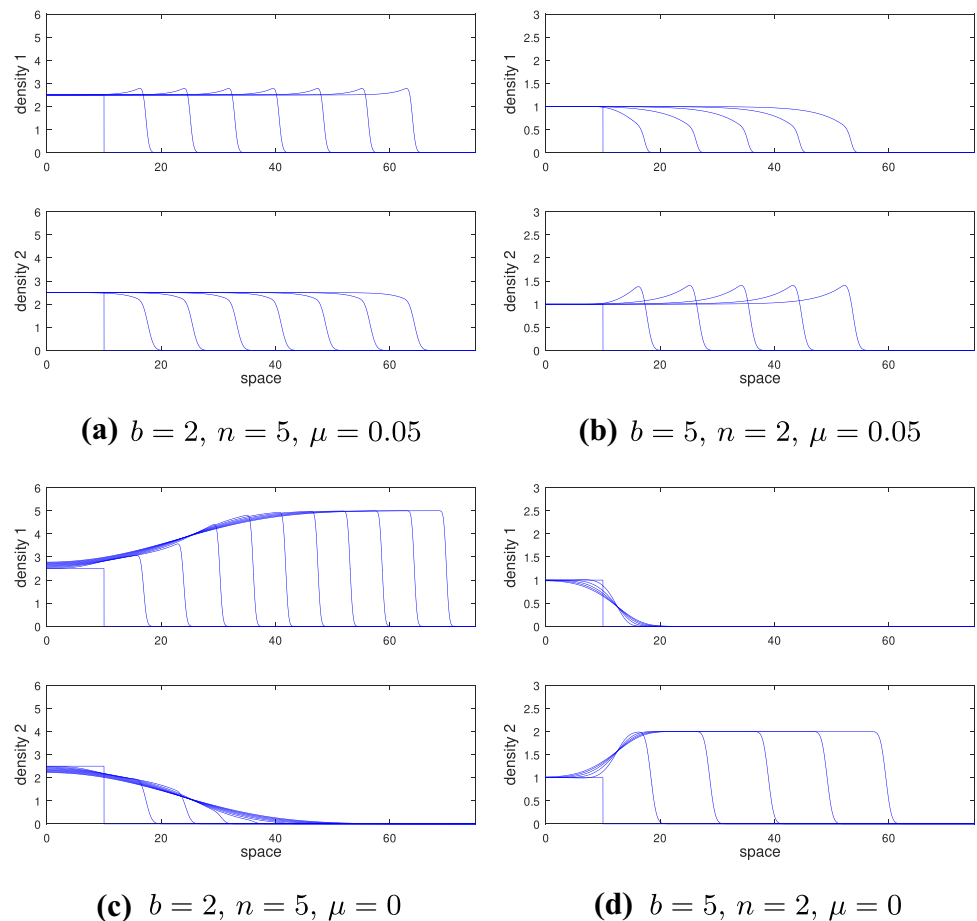
example, when reproduction (*b*) was low and carrying capacity (*n*) was high (left column in Fig. 2), the short type outran the long type without mutation (bottom row) and settled at its single-type speed. With mutation, the two types spread together at a higher joint speed (top left). When reproduction was high and carrying capacity was low (right column), the long type outran the short type without mutation (bottom plot) and settled at its single-type speed. With mutation, both types spread together at a lower joint speed (top right). Hence, mutations increased invasion speed in the first scenario but decreased it in the second. Overall, when the two types spread jointly (with mutation) the population spread faster when reproduction was high and carrying capacity was low than when reproduction was low and carrying capacity was high.

The question of whether mutation will speed up or slow down the invasion of a population of two types can then be answered seemingly easily: if the short type outruns the long type in the absence of mutation, then mutation will increase the speed but if the long type will outrun the short type in the absence of mutation, then mutation will decrease the speed.

But how can the short type outrun the long type? It is the combination of a population-level Allee effect, introduced by mate-finding, and the inheritance scheme (Fig. 3). Initially, both types are equally present. After the first dispersal event, the short type density (solid) is higher than that of the long type (dashed) inside the original extent but lower outside (top panel). The total population increases where the total population density exceeds the Allee threshold (bottom panel). The Allee threshold is high (horizontal dashed line) when *b* is low and low (solid) when *b* is high. In the absence of mutations, the dominant dispersal type of the offspring will be that of the dominant parent type. Hence, inside the original population extent, the short type will dominate and outside the long type. But if the Allee threshold is high the region in space where the long type reproduces dominantly is very small. If, at the same time, the carrying capacity is low, the density of long type offspring is low. In subsequent dispersal events, the long type then spreads its density so thinly that it is likely to be below the Allee threshold, which continues the decline of the long type. Alternatively, if the Allee threshold is low and the carrying capacity is high, the long type can take advantage of its higher dispersal abilities and establish a high density ahead of the short type. Once this dominance is established, the type dominant at the population edge spreads unperturbed by the other type.

Fig. 2 Four scenarios of spread. Each scenario consists of two plots: density 1 denotes the short type and density 2 the long type. Plots (**a**) and (**b**) are with mutation, whereas (**c**) and (**d**) are without. Plots (**a**) and (**c**) have low reproduction and high carrying capacity, whereas (**b**) and (**d**) have the reverse case. Other parameters are $d_1 = 0.1 < d_2 = 0.5$ and $\tau = 1$. Initial conditions are uniform on $[-10, 10]$. Spread is symmetric in both directions but only the positive direction is shown. Densities are plotted every 40 generations (left column) and every 10 generations (right column). The resulting speeds are 0.19 (top left), 0.14 (bottom left), 0.45 (top right) and 0.51 (bottom right)
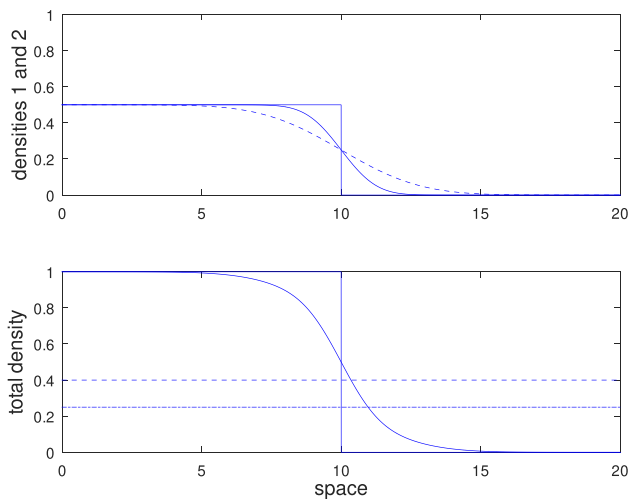

(a) $b = 2$, $n = 5$, $\mu = 0.05$

(b) $b = 5$, $n = 2$, $\mu = 0.05$

(c) $b = 2$, $n = 5$, $\mu = 0$

(d) $b = 5$, $n = 2$, $\mu = 0$

Fig. 3 Schematic illustration for how a short type can dominate a long type. Top panel: The (identical) initial conditions for both types (step function) and the resulting densities of the short (solid) and long (dashed) type after the first dispersal event. Bottom panel: The total population density initially (step function) and after the first dispersal event (solid curve), together with the Allee threshold (horizontal line) in the case of low b (dashed) and high b (solid)

Fig. 4 Difference in the asymptotic speed of invasion with mutation and without mutation. Positive values indicate that the scenario with mutation spreads faster. The value of zero for b, n small indicates that the population is not viable in this regions of parameter space. Dispersal parameters are $d_1 = 1 < d_2 = 5$

How frequently and under which conditions does it happen that the short type outruns the long type in the absence of mutation so that mutation will speed up the invasion? And when does the reverse effect occur? Increasing b favors the long type, all else being equal, and increasing n does the same (Fig. 4). The short type only outruns the long type near the extinction threshold in b–n space below which the overall population fails to spread; see Fig. 1. The likelihood that the short type outruns the long type increases as the difference in the variance of the corresponding dispersal kernels increases and as the spatial extent of the initial introduction decreases.

The role of initial conditions

The importance of initial conditions is also evident in the following numerical experiment where initial conditions are allowed to differ slightly between the two types (Fig. 5). We consider the region of parameter space where the long type dominates the invasion front and sets the speed, while the short type appears at the front through mutation (top panel), whenever the initial conditions are identical for both types (as we generally assume). However, when the short type has a small headstart, it continues to dominate the invasion front and it sets the speed, while the long type cannot break through the dominance of the other type at the front (bottom panel). Since all model parameters are identical in the two simulations, we are led to conjecture that there may not be a unique spreading speed in this system and not a unique traveling wave
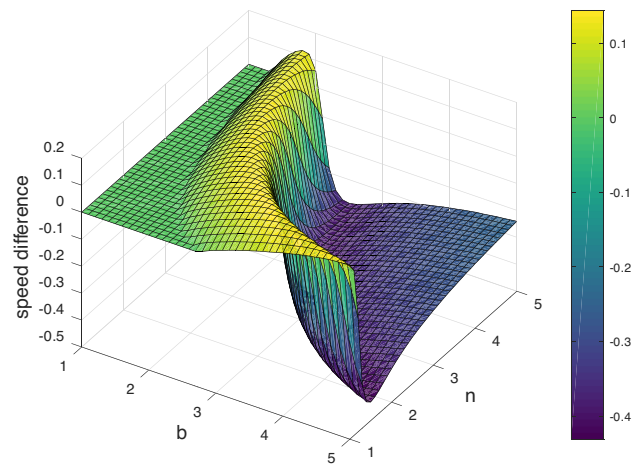
profile, even when the mutation probability is positive. A mathematical proof of uniqueness or nonuniqueness is beyond the scope of this paper. Instead, we eliminate the question of initial conditions by choosing identical initial spatial extent for all types, as motivated by the biological question of interest (see "Introduction").
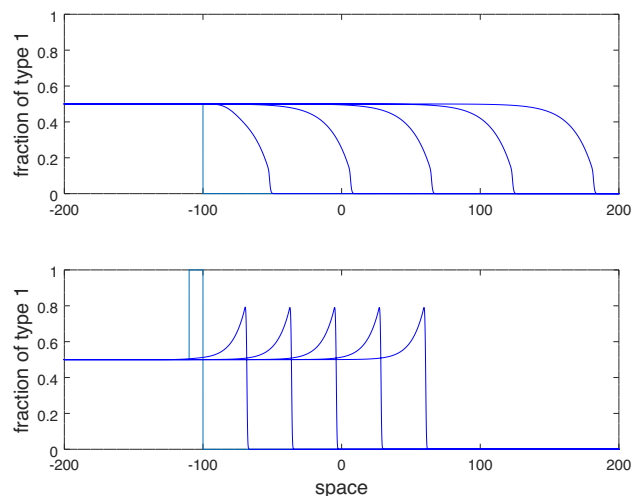

Fig. 5 Different wave profiles and speeds arise as the initial extent of one type changes. The curves show the short type fraction of the total density (where positive). Top panel: both types are initially present for $x < -100$. The short type has lower density at the front that moves at speed 0.29. Bottom panel: the short type has a headstart over the long type who is initially present only for $x < -110$. The short type establishes the higher density at the front that moves at speed 0.16. Parameters are $b = 2$, $n = 5.1$, $d_1 = 0.1$, $d_2 = 0.5$, and $\mu = 0.01$. Simulations ran for 1000 generations; profiles are plotted every 100 generations

Model extensions

Dispersal-induced mortality

Model modifications

Dispersal is risky and costs energy (Bonte et al. 2012). Most models for evolution and range expansion assume that individuals experience mortality during dispersal (Phillips et al. 2008; Travis et al. 2009; Burton et al. 2010; Shaw and Kokko 2015). We include dispersal-induced mortality into our model from the previous section and study the effects. More specifically, we assume that there is a constant per unit time probability of mortality during the dispersal process (see Lutscher (2019), chapter 12), and we assume that mortality increases with expected dispersal distance. We model these assumptions by multiplying the Gaussian dispersal kernel with the survival probability $e^{-d_i m} < 1$, where d_i is the dispersal parameter (variance) of type i and m is a scaling factor. The resulting kernel,

$$K_i(x) = \frac{e^{-d_i m}}{\sqrt{2\pi d_i}} \exp\left(-\frac{x^2}{2d_i}\right), \tag{14}$$

does not integrate to unity.

However, we can include the survival probability into the growth function and continue to operate with dispersal kernels that integrate to unity. This view allows us to adapt some of the preliminary observations from "Spread of a single type" to this new situation. The curve in b–n space that separates population spread from retreat (Fig. 1) now depends on the dispersal type. A type with high d_i has a lower survival probability and therefore requires larger values of b and/or n to persist and spread, compared to a type with low d_i. The speed at which a single type in isolation spreads decreases as the dispersal mortality factor (m) increases to the point that at large enough m the single type retreats rather than advances.

We also need to adapt our initial conditions for spread simulations with two types. When dispersal behavior has no impact on demography, the equal distribution of both types is a steady state of the nonspatial equations. When we include dispersal-induced mortality, this is no longer true. Since higher dispersal implies larger mortality, the steady-state density of the type with higher dispersal is lower than that with lower dispersal. We were not able to find an analytic expression of the coexistence state for two types, but we can determine the densities that we use for initial conditions numerically very easily.

Comparison to results with no dispersal mortality

When simulating the two types spreading from their initial conditions as described in the setup, there are some similarities and some differences between the case with and without dispersal-induced mortality. In the absence of mutation ($\mu = 0$), there is still eventually only one type present at the invasion front and this type sets the speed. With mutation ($\mu > 0$), both types are present at the invasion front and spread at the same speed. If the long type dominates the population edge without mutation, then including mutation will slow down the overall spread of the population, as it did in the previous section. However, because of the additional dispersal-induced mortality, it is harder for the long type to dominate the population edge in the absence of mutation. Even if the long type dominates the population edge without mutation, the short type is not stalled but will slowly invade and replace the long type in a second wave; see Fig. 6. This phenomenon is related to stacked waves that have been observed in other contexts in reaction-diffusion equations (Iida et al. 2011) and in integrodifference equations; see Marculis and Lui (2016) and references therein.

As in the case without dispersal mortality, it appears that also with dispersal mortality, the spread rate with mutation ($\mu > 0$) is bounded between the single-type spread rates without mutation. What is different now is that the spread rate of the long type can be slower than that of the short type because of the additional mortality. In particular, as explained above, each type has its own extinction boundary in b–n space and there is a range in that space where the long type retreats (i.e., has a negative spread rate) and the short type advances (i.e., has a positive spread rate); see condition (13). In this case, the short type will outrun the long type without mutation, and adding mutation will slow down the total population because the increase in dispersal distance is less than the decrease due to the higher mortality (Fig. 6).

Effect of mutation probability

So far, we only compared spread rates with and without mutation. Now, we investigate how spread rates depend on mutation probabilities. Since mutation probabilities are typically small, we limit our simulations to $0 \leq \mu \leq 0.2$. We find four distinct patterns (Fig. 7). When the long type dominates the population edge in the absence of mutation, then the population spread rate decreases monotonically with increasing mutation probability (green curve at the top). Because of the increased mutation probability, a larger number of long types turns into short types, which slows the advance of the front. When the short type dominates the population edge in the absence of mutation while the long type is faster in isolation, then the population spread rate increases monotonically with mutation probability (red curve in the middle). The underlying mechanism is the same as above: an increase in mutation probability this time leads to an increase in the faster
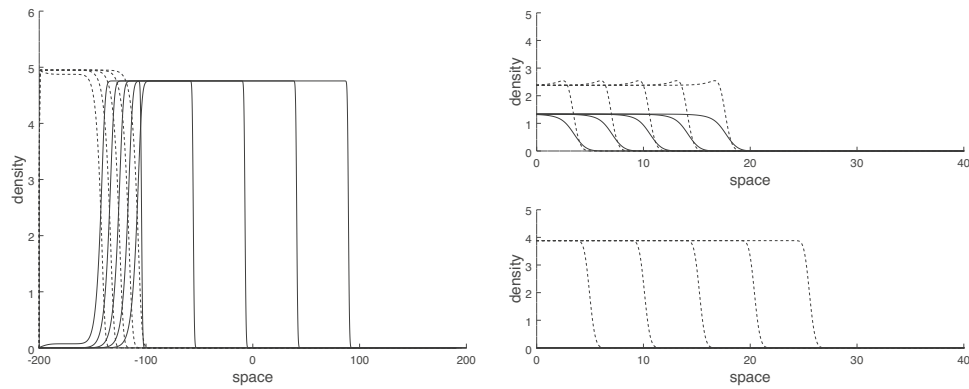
Fig. 6 Left: The long type (solid) spreads quickly to the right but is slowly replaced by the short type (dashed). There is dispersal-induced mortality ($m = 0.1$) but no mutation ($\mu = 0$). Initially, both types are present for $x < 150$; densities are plotted every 100 generations. Other parameters are $b = 3$, $n = 5$, $d_1 = 0.1$, and $d_2 = 0.5$. Right: The joint (rightward) spread of both types with mutation ($\mu = 0.1$,
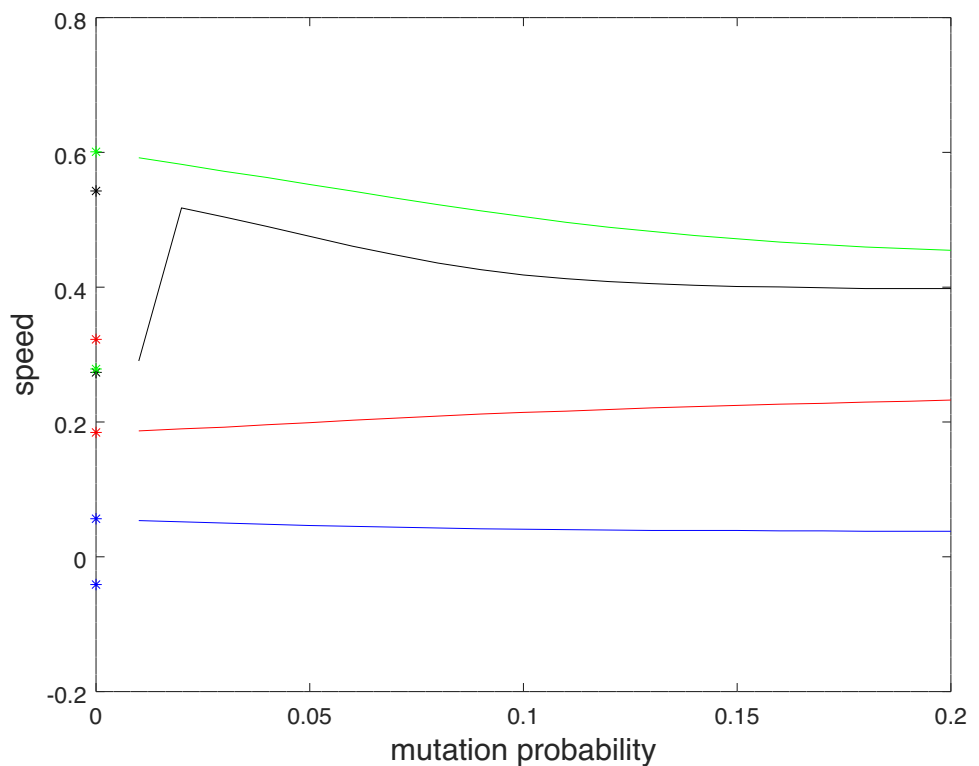
top panel) can be slower than the spread of the short type (dashed) in isolation ($\mu = 0$, bottom panel) when dispersal-induced mortality ($m = 0.3$) has a strong impact on the long type. Initially, both types are present for $x < 0$; densities are plotted every 100 generations. Other parameters are $b = 1.7$, $n = 4$, $d_1 = 0.1$, and $d_2 = 0.5$

type, so that the entire population speeds up. Because of dispersal-related mortality, it is possible that the long type in isolation has a slower spread rate than the short type in isolation (see above). If this is the case and if the short type dominates the population edge in the absence of mutation, then increasing mutation will decrease the overall speed (blue curve at the bottom).

The behavior of the black curve (second from the top) is the most surprising. Here, the short type dominates the

population edge without mutation. A very small mutation probability leads to a slightly higher spread rate since the long type is now present at the front but at relatively small density. But a small increase in mutation probability (here from $\mu = 0.01$ to $\mu = 0.02$) leads to a jump of overall spread rate. Then as the mutation probability increases further, the spread rate decreases again. It turns out that the small increase in mutation probability enables the long type to dominate the population edge and thereby increase

Fig. 7 Four patterns of population spread rate as a function of mutation probability μ. For each pattern, the two stars on the vertical axis indicate the spread rates of the two types in isolation and the curve in the corresponding color shows the joint spread rate with mutation. In blue, the short type spreads faster in isolation; in all other cases, the long type spreads faster in isolation. Green: $b = 4$, $m = 0.1$. Black: $b = 4$, $m = 0.3$. Red: $b = 2.5$, $m = 0.3$. Blue: $b = 1.6$, $m = 0.3$. Other parameters are: $n = 5$, $d_1 = 0.1$, and $d_2 = 0.5$

the speed significantly. A further increase in mutation probability only leads to an increase in the short type at the edge, and hence a slowing down. The abrupt increase in spread rate with mutation probability could be another indicator of bistability in the system (see above). To test this possibility, we ran simulations with mutation probability high enough to give the long type an advantage (here $\mu = 0.02$) until the wave profile was established. Then we reduced the mutation probability to where the above simulations indicate a slow overall spread rate (here $\mu \leq 0.01$). Instead of the wave slowing down, we observed that it sped up and approached the speed of the long type in isolation as $\mu \to 0$ (plots not shown). Hence, we have a second numerical indicator that we can have two different stable traveling wave solutions with different speeds for the same parameter set.

Effect of dispersal parameters

As the final aspect in this section, before we consider three and more different types, we investigate how the spread of two types depends on the dispersal parameters $d_{1,2}$. For all simulations so far, we had a constant ratio $d_2/d_1 = 5$. We now choose dispersal parameters $d_i = 0.05, 0.1, 0.2, \ldots, 0.6$ and run simulations for each pair to compare the speed with and without mutation (Fig. 8). Positive values indicate that the speed with mutation is higher, i.e., that mutation increases the spread rate; negative values indicate that mutation decreases speed. When the difference in dispersal parameter between the two types is large, then the shorter type outcompetes the other in the absence of mutation, so that including mutation increases the spread rate. When the difference in dispersal parameters is small, it depends. When both d_i are small, the long type has an advantage and dominates the population edge in the absence of mutation, so that mutation slows the joint spread. When both d_i are large, the shorter type has the advantage and mutation speeds up the joint spread. The critical value between the two cases seems to be around $d_i = 0.4$ for the parameters chosen here.

Korolev ([2015]) obtains a related but somewhat different result. In his work, the fitness of a mutant whose dispersal ability differs only slightly from the dominant type changes in a nonmonotone way and changes sign. When the Allee threshold is high, slower (but not too slow) mutants can invade, whereas when the Allee threshold is low, faster (but not too fast) mutants can invade. As mentioned in the introduction, Korolev ([2015]) uses adaptive dynamics and linearizes around a single-type traveling wave to calculate invasion fitness, whereas we consider both types to be initially present at the two-type steady state.
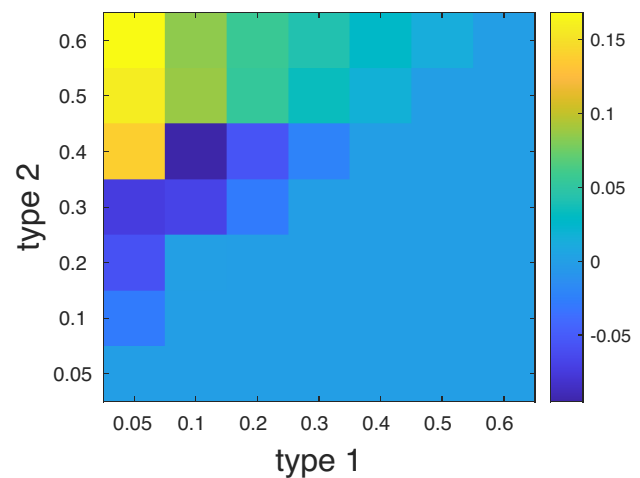

Fig. 8 Results of pairwise simulation runs for two dispersal types with different dispersal parameters d_i. The heatmap shows the difference of the speed with mutation and the speed without mutation. Positive values indicate that the speed with mutation is higher. Parameters are $b = 2.5$, $n = 5$, $\tau = 1$, $\mu = 0.05$, $m = 0.1$. The simulations ran for 1000 generations, except the case $d_1 = 0.4$ and $d_2 = 0.5$ where more generations were necessary for the short type to exclude the long type and establish the spread rate. Only the values for $d_2 \geq d_1$ are shown; the situation is symmetric

Multiple dispersal types

Model modifications

In this section, we generalize the model to more than two dispersal types while keeping all other assumptions in place. We pick dispersal parameters

$$d_1 < d_2 < \cdots < d_k$$

and denote the frequency of females (males) with dispersal parameter d_i by f_i (m_i). The total number of female (male) individuals with dispersal parameter d_i is $F_i = F f_i$ ($M_i = M m_i$), where the total number of female (male) individuals is F (M), as before. Also as before, since there is no assortative mating with respect to dispersal distance, the pair formation process depends only on the total number of females and males. Hence, $P = P(F, M)$ is the total number of pairs formed according to (1) and (2), depending on whether $F = M$ (locally) or not. The total number of offspring produced is $b P(F, M)$ and the sex ratio is $1/2$. The maximum number of offspring supported by the environment is n. Hence the number of female offspring equals the number of male offspring and is again given by $\frac{1}{2} \min\{b P(F, M), n\}$.

In the absence of mutation, offspring inherit their trait value from either parent with equal probability. Hence, the probability that an offspring has trait value i is simply

$\frac{1}{2}(f_i + m_i)$. We assume that this value changes with mutation probability $\mu \in (0, 1)$. We consider simple nearest neighbor mutations only. Hence, an offspring that has inherited trait value i will switch to trait value $i \pm 1$ with probability $\mu/2$, as long as $i \neq 1, k$. For offspring who inherit the smallest or largest trait value, there are several options. We pick the simple case that all mutations from the lowest (highest) trait go to the next higher (lower) trait. If we write the vector of trait distributions $\mathbf{f} = (f_1, \ldots f_k)$, then the next-generation distribution of traits is obtained from the current distribution via matrix multiplication $\mathbf{f}^{\text{next}} = A\mathbf{f}$, where

$$A = \begin{bmatrix} 1-\mu & \mu/2 & 0 & \ldots & \ldots \\ \mu & 1-\mu & \mu/2 & 0 & \ldots \\ 0 & \mu/2 & 1-\mu & \mu/2 & \ddots \\ \vdots & \ddots & \ddots & \ddots & \mu \\ 0 & 0 & 0 & \mu/2 & 1-\mu \end{bmatrix}.$$

With this choice, the stationary distribution is proportional to $[1/2, 1, \ldots, 1, 1/2]$.

We observe that just like in the case of two trait values, the numbers of female and male individuals after a reproduction event are the same (i.e., $F = M$), as are their distributions with respect to trait value (i.e., $f_i = m_i$ for all i). Hence, it is again sufficient to track females in our model.

Comparison to results with two types

Our model now has too many parameters to explore every possible behavior by simulations. Instead, we show that many of the mechanisms that led to the qualitative results with two types carry over to multiple types. First, we note that when there is no mutation ($\mu = 0$), then all our simulations showed that there is eventually only one type leading the front. If the difference in dispersal parameter are small, two or more types can coexist in long transients (see above), but eventually one of them dominates the population edge. That type then determines the asymptotic speed of spread, namely the speed of this type in isolation. If there is no dispersal-induced mortality, all other types will eventually stop spreading. If there is dispersal-induced mortality, we observe again secondary invasions of types with lower dispersal parameter but higher effective reproduction. A type with lower dispersal parameter replaces the one with higher parameter but is itself replaced by another one with even lower parameter. On the other hand, if there is mutation ($\mu > 0$), then all types are present at the leading edge and their densities converge to the nonspatial coexistence state behind the wave front. Hence, as was the case for two types, whether mutation speeds up or slows down spread comes down to comparing the speed set by a single type when $\mu = 0$ with the speed determined by all types when $\mu > 0$. We illustrate all these three behaviors in Fig. 9.

Which of the types dominates the population edge in the absence of mutation depends on all parameters and on the number of types present. Higher values of b (number of offspring) and n (carrying capacity) favor types with higher dispersal parameter; higher values of m (dispersal-induced mortality) favor types with lower dispersal parameter. In fact, by varying any one of these parameters, one can select which of the types will dominate the population edge in the absence of mutation.

When a lower (higher) dispersal type dominates the population edge in the absence of mutation, including mutation speeds up (slows down) spread. One example with varying mortality is shown in Table 1. For large dispersal mortality, types 1 or 2 dominate the population edged, and mutation speeds up spread. When there is no mortality, type 4 dominates the population edge and mutation slows the spread. Surprisingly, at intermediate values, type 3 dominates the population edge without mutation, and mutation can either increase or decrease the speed. In both cases, mutation will allow lower and higher dispersal types to persist, but since higher dispersal also carries higher mortality, the outcome depends on a trade-off and is difficult to predict.

Similar results arise when varying parameters b or n independently. Increasing the number of types within a given range tends to produce longer transients where multiple types are present at the leading edge in the absence of mutation. The overall qualitative results, however, do not change.

Discussion

Predicting the speed of invasion or range expansion is of fundamental importance for ecosystem management, yet the interplay between concurrent ecological and evolutionary processes makes the prediction of invasion speeds difficult (Phillips 2015). We studied how mutation (one mechanism for evolutionary change) affects the rate of population spread. We studied the case of an Allee effect in part because Allee effects are ubiquitous in nature and have their greatest effect at low population densities that arise at an invasion front, and in part because the two (very different) studies that had previously challenged the conventional wisdom that "evolution speeds up invasions" both include an Allee effect. For a concrete mechanism, we chose a mate-finding Allee effect. We developed an integrodifference equation model with two sexes and two types (short- and long-distance dispersers, later extended to multiple types). We found that, in the absence of mutation, one type comes to dominate the population edge: the long type dominates when fecundity (b) and/or local carrying capacity (n) are high and the short type dominates when b and/or n are small. Mutation ensures that all types are present at the invasion front. Consequently, mutation tends
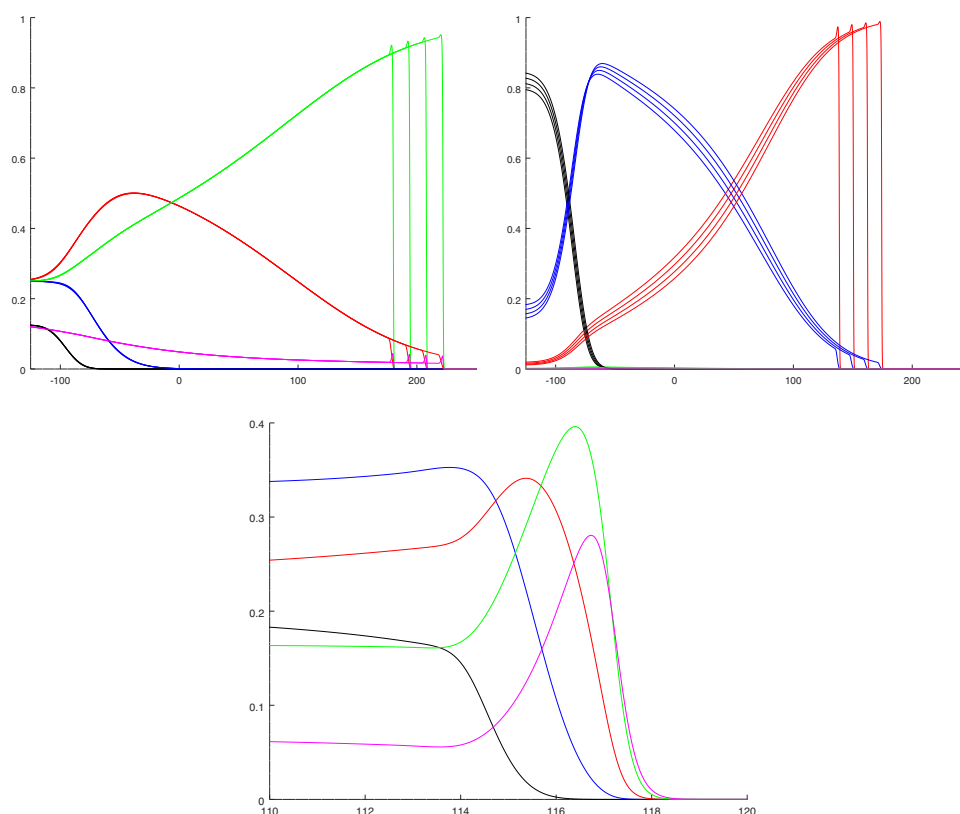
Fig. 9 Simulation outcomes for five types. Top left: without mutation ($\mu = 0$) and in the absence of mortality ($m = 0$), an intermediate dispersal type takes over the front edge ($d = 0.4$, green). Shorter types are stalled ($d = 0.1$, black, and $d = 0.2$, blue) or declining ($d = 0.3$, red), just as the longer type ($d = 0.5$, magenta). They will eventually stall as well. Top right: Without mutation but with dispersal-related mortality ($m = 0.02$), an intermediate type controls the edge ($d = 0.3$, red) but a slower type ($d = 0.2$, blue) replaces it, only to be replaced by an even slower type ($d = 0.1$, black). The longer types are eliminated. Bottom: With mutation ($\mu = 0.05$), all five types are present at the front and spread with a joint speed (colors and dispersal parameters as in the other two plots). In the top row, densities are plotted every 50 generations from 1050 to 1200. The bottom shows the profile at generation 1050. Parameters are: $b = 2$, $n = 5$, $\tau = 1$

to slow spread when the long type dominates the edge in absence of mutation, and mutation speeds up spread when the short type dominates the edge in absence of mutation. Furthermore, adding dispersal mortality induces a trade-off and increases the region of b-n parameter space where the short type is favored at edge (and thus where mutation speeds up spread).

Table 1 Outcomes of spread simulations with 5 types ($d_k = 0.1 * k$) with varying dispersal-induced mortality m. The second row indicates which type wins (dominates the population edge) in the absence of mutation. The third row has the corresponding speed. The last row has the speed with mutation ($\mu = 0.05$). Other parameters are $b = 2$, $n = 5$, and $\tau = 1$. Simulations were run for 1200 generations to capture possibly long transients

m	0.1	0.05	0.02	0.01	0
winning type	1	2	3	3	4
speed without mutation	0.137	0.194	0.239	0.241	0.280
speed with mutation	0.181	0.204	0.234	0.243	0.250

Our model is closely related to the work by Shaw and Kokko (2015), who used an individual-based model and a complicated dispersal process where the probability of an individual to move and to stop moving depends on the density of other individuals of the same and opposite sex. They had shown that mutation can slow down invasions, contrary to previous theoretical findings. Our result shows that this slowing down does not require a complicated dispersal process but can happen with simple density-independent dispersal as well. Korolev (2015) had also concluded that "evolution can slow down an invasion" in the presence of an Allee effect. His work is based on separation of ecological and evolutionary time scales in that he shows that under certain conditions, a mutation to a slower disperser can successfully invade at a traveling wave of a single type. In contrast to this work, our work does not assume a separation of time scales and an initially low density of a mutant population. Instead, we have two or more types present at equilibrium densities in a confined region in space and observe how their interaction is organized as they all begin to spread in space.

This brings us to an important point. The statement that "evolution speeds up invasions" is not only too general to be true (because there are so many evolutionary mechanisms that could be at work), it also depends on how changes in spread rate are measured. The results arising from reaction-diffusion equations state that the population structured by dispersal rate spreads faster than an unstructured population whose dispersal rate is the average trait value (Bénichou et al. 2012; Bouin et al. 2012). But Fig. 3a in Bénichou et al. (2012) also shows that the asymptotic speed of the structured population is a decreasing function of mutation rate (at least for the range of values shown). The mechanism that they identify for this decrease is the same that we found: as the mutation rate increases, the trait distribution at the leading edge goes from being heavily to only moderately skewed towards high dispersal rates, so that the overall spread rate slows. In fact, as the mutation rate becomes very high, the trait distribution flattens to a uniform distribution and the overall speed approaches that of the unstructured population with mean trait value (Bénichou et al. 2012). From this point of view, an Allee effect does not seem necessary for "evolution to slow invasions".

Spatial sorting is a fundamental phenomenon when a spreading population can evolve (Miller et al. 2020). It can happen in the presence (Bénichou et al. 2012) or absence (Ramanantoanina et al. 2014) of mutations. In the absence of an Allee effect, faster dispersers tend to be found at the leading edge of a population, where they tend to mate with other fast dispersers, so that the offspring is, again, likely to be a fast disperser. As mutations occur, some of the fast-disperser parents will have slower disperser offspring, which then slows the overall invasion. Spatial sorting occurs in our model as well, but it is not necessarily the fastest disperser that accumulates at the leading edge. Because of the Allee effect, the density of the long type might be too far below the Allee threshold to produce enough offspring, and a short type might take over the leading edge; see Fig. 3. Once the short type dominates the leading edge, the mate-finding Allee effect prohibits the long type to break through and take the lead. In that sense, and in a curious turn of events, one could say that an Allee effect is necessary for mutation to speed up an invasion. But as mentioned above, this statement is also too general to be true and depends on how one measures changes in speeds. It does not apply for the approach by Korolev (2015).

Our model makes a number of assumptions that could be changed in future work to explore their effects. First we assume haploidy and a single locus; future models could explore the effects of diploidy and/or multiple loci with recombination. We expect that these changes would perhaps dampen the effect of mutation (since both diploidy and recombination serve to shuffle heritability, like mutation does), but would not qualitatively change our results.

Second, our model assumes that competition happens at birth (pre-dispersal); future extensions could explore the effect of competition after dispersal. This change could potentially change which dispersal type dominates the population edge. Finally, our model abstracts much of the dispersal process of the individual-based model by Shaw and Kokko (2015). It is a future challenge to include increasingly more details of their model (e.g., density-dependent and conditional dispersal) into the integrodifference model and compare, at each stage, how similar or different the two models behave. We expect our core results (that mutation can lead to faster or slower spread) will hold, but that where these outcomes occur in parameter space will depend on the details of dispersal; thus potentially explaining the discrepancies between our results and those of Shaw and Kokko (2015).

We made two intriguing mathematical observations that warrant more detailed investigation in future work. First, the joint speed of two types with mutation was always bounded between the speeds of either type in isolation. In other words, we did not see an anomalous speed in the sense of Weinberger et al. (2007) or Keenan and Cornell (2021). Since such an anomalous speed requires some trade-off between long-distance dispersal and reproduction success (Lutscher 2019), and since long-distance dispersal does not confer an advantage when there is a mate-finding Allee effect, it seems plausible that anomalous speeds would not appear here. Future work should prove this analytically. Second, we have strong numerical evidence for nonuniqueness of a traveling wave profile and speed, but no mathematical proof. Given that each type in isolation can spread at some given speed (which is unique because of the Allee effect), it is tempting to think of a perturbation argument that a small amount of mutation would lead to a joint spread with a speed close to the individual speed from which the perturbation occurred. Clearly, an Allee effect would be necessary for nonunique speeds since cooperative (Lui 1989) and mixed systems that are cooperative near zero (Girardin 2017) have a unique speed of propagation.

What implications do our findings have for biological applications? First, and probably foremost, is the insight that general statements on evolution speeding up or slowing down spread are to be taken cautiously. There are many evolutionary processes that act in different ways. Mutation is one of them and it acts simultaneously to change traits in the population over time, while also disrupting the effects of other evolutionary processes (e.g., selection). Another concept that relates mutation with slowing the speed of spread is that of mutational load, i.e., the accumulation of deleterious mutations over time; see Miller et al. (2020) and references therein. Our model does not keep track of mutation accumulation, and our results show that mutation can decrease or increase speed of spread. It will be interesting to combine models for mutation accumulation with our ideas

and consider the joint effect on the speed of spread. We also found that initial conditions are important in shaping which type dominates at the edge. This finding provides one potential explanation for why invasions into new regions are so hard to predict. Our study of the multiple-trait model concentrated on which of the features from the two-trait model extend to multiple traits, but was by no means a complete investigation of that case. It would be interesting to eventually consider a continuum of traits, similar to the reaction-diffusion models.

Availability of data and material Not applicable.

Code Availability Code is available from https://github.com/FrithjofL/LPS_Theoretical_Ecology.

Declarations

Ethics approval Not applicable.

Consent to participate Not applicable.

Consent for publication All persons entitled to authorship have been so named. The authors have approved the submission of this manuscript for publication.

Competing interests The authors declare no competing interests.

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