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Density dependence, lifespan and the evolutionary dynamics of longevity

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ABSTRACT

Longevity is a life-history trait that is shaped by natural selection. Evolution will shape mortality trajectories and lifespans, but until now the evolutionary analysis of longevity is based principally on a density-independent (Euler–Lotka) framework. The effects of density dependence on the evolution of lifespan and mortality remain largely unexplored. We investigate the influence of different population demographies on the evolution of longevity, and show how these can be linked to adaptive radiations. We present a range of models to explore the intraspecific and interspecific density effects on longevity and, consequently, diversification. We show how the magnitude, type, and timing of mutation can also affect fitness, invasion and diversification. We argue that fitness of alternative strategies under a range of different demographic structures leads to flat, as opposed to rugged, landscapes and that these flat fitness surfaces are important in the evolution of lifespan and senescence.

1. Introduction

Our understanding of the evolution of many life-history traits is based on strategies that invade and then grow exponentially (Stearns, 1992; Caswell, 2001). These density-independent concepts of evolution are centred around the Euler–Lotka equation from which fitness (measured as the intrinsic rate of increase) can be derived as a function of birth and survival schedules (Fisher, 1958). However, populations cannot grow in an ever increasing manner. Resource competition, predation, mutualisms and other factors act to limit population growth. These density-dependent processes are well-known to affect the dynamical regulation of populations, but the effects of density-dependent processes on the evolution of life-histories is a contemporary issue in evolutionary demography (Boyce, 1984; Stearns, 1992; Reznick et al., 2002; Mueller et al., 2005).

One manifestation of these density-dependent processes among alternative strategies is lineage diversification. Adaptive radiations are the "evolution of ecological and phenotypic diversity within a rapidly multiplying lineage" (Schluter, 2000). These radiations rely on the simultaneous divergence of a single ancestor into multiple lines that differ in phenotypic, morphological, or physiological traits to exploit different niches (Simpson, 1953; Schluter, 2000). Thus, phenotypic diversity is generated and maintained through ecological interactions that drive divergent character displacement and allow novel niches to be realised. Evolutionary theory predicts that certain life-histories will be negatively associated with each other, control population growth and shift with alterations in population abundance (Cole, 1954).

Longevity is a life-history trait that is shaped by natural selection (Charlesworth, 2001; Mangel, 2001, 2002; Bonsall, 2006), and understanding how evolution has shaped mortality trajectories and lifespans is a central theme in life-history theory (Bidder, 1932; Medawar, 1952; Williams, 1957; Hamilton, 1966) and evolutionary ecology (Keller and Genoud, 1997; Bronikowski and Promislow, 2005; Reznick et al., 2004; Mangel and Bonsall, 2004; Mangel and Munch, 2005; Bonsall, 2006; Munch and Mangel, 2006; Mangel et al., 2007). However, the consequences of density dependence on the evolution of lifespan remain largely unexplored. Here, we explore how the evolutionary effects of density dependence, demographic structure and changes in age-specific characteristics affect the evolution of longevity and, consequently, species diversification.

Schedules of survival and reproduction tend to vary non-randomly and, that they do, is evidence for the multiplicity of solutions to optimise fitness. Trade-offs generate this non-random variation and are manifest through resource allocation decisions (Levins, 1968). The ecological effects of these life-history trade-offs were first identified by Levins and Culver (1971) as a mechanism of coexistence. Although coexistence is well-known to depend on differential resource utilisation (e.g. Hardin (1960), Miller

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(1967), Armstrong (1976) and Armstrong and McGhee (1980)), Levins and Culver (1971) demonstrated that the proportion of an ecological habitat occupied is a balance between (colonisation) immigration and extinction rates. Coexistence is achieved through differential competition between strategies: poor colonists are good competitors and vice versa (Horn and MacArthur, 1972; Tilman, 1994; Kinzig et al., 1999, 2002; Bonsall and Mangel, 2004).

Levins and Culver (1971) also showed how coexistence can arise through trade-offs between competitive ability and mortality rate. Coexistence is possible if the superior competitor tends to be short-lived while the inferior competitor is longer lived. Species coexist, as those that are longer lived have access to scarce resources that would otherwise be consumed by the superior but shorter lived competitor (Bonsall et al., 2002). We begin by presenting our different models to explore the effects of density dependence on the evolutionary dynamics of longevity. We show how the evolution of different strategies depends on both the dynamics associated with the trade-off and also on the underlying population structure and the mutation rate. We discuss these results with reference to recent developments in the evolution of longevity and mechanisms of species diversification.

2. Mathematical models

2.1. Population dynamics

To investigate how different demographic structures affect evolutionary trajectories we analyse a set of different structured population models. Each mathematical model is composed of a component describing (identical) individual mass gain (m) and a component describing the population size (N) (Bonsall and Mangel, 2004). We first consider how it applies to a single species with age structure (juvenile and adult classes), which is the common situation for the analysis of the evolution of senescence (Charlesworth, 1994; Partridge and Mangel, 1999). Second, using a multispecies version, we explore how density dependence and lifespan affect the nature of interspecific adaptive radiations.

Our mathematical model is constructed from three components: (1) changes in individual mass of a strategy or age-class within a strategy which follows established allometric scaling relations (West et al., 2001); (2) the population size of a strategy or age-class within a strategy which follows density-dependent dynamics, and (3) a function describing the shape of the trade-off (see below).

2.1.1. Model I — Juveniles and adults with density-dependence

To generalise the classic approach to longevity, we consider a within-species structured model with separate pre-reproductive or juveniles (denoted by subscript $_J$) and reproductive or adult (denoted by subscript $_A$) classes where density dependence operates through within-class competition. This two-state population model provides an approximation to a fully age-structured one (Gurney et al., 1983) and combines the fidelity offered by age-structured models (von Foerster, 1959) with the analytical tractability of simple time-delayed models. Individuals within each stage are identical with respect to life-history demographies, and we are able to explore the consequences of variability and differences between age-classes on the patterns of evolutionary diversification. Thus, we are able to link the dynamics of evolutionary diversification with classic theories of aging. The dynamics of the juvenile age-class are:

$$\frac{dm_J}{dt} = \frac{a_J \cdot m_J^{\beta}(t)}{1 + \gamma_{JJ} \cdot N_J(t) \cdot m_J^{\beta}(t)} - b_J \cdot m_J(t) \tag{1}$$

$$\frac{dN_J}{dt} = r \cdot N_A(t) - \left[\mu_J + \gamma_{JJ} \cdot N_J(t) \cdot m_J^{\beta}(t)\right] \cdot N_J(t) - \eta_J \cdot N_J(t) \tag{2}$$

Table 1
Parameter definitions for the intraspecific competition model (Model I).

	Parameter	Definition
General parameters	m	Individual mass gain
	N	Population size
	$\delta\mu$	Magnitude of mutation
Mass gain	a	Resource uptake rate
	β	Allometric scaling constant
	b	Resource utilisation rate
Juvenile class	N_J	Juvenile class population size
	γ_{JJ}	Strength of juvenile competition
	μ_J	Juvenile class mortality rate
	η_J	Juvenile maturation rate
Adult class	N_A	Adult class population size
	r	Per capita recruitment rate
	γ_{AA}	Strength of adult competition
	η_A	Adult lifespan
	μ_A	Adult class mortality rate

where a_J and b_J are resource uptake and utilisation rates by the juveniles, γ_{JJ} is the strength of intraspecific competition, β is the allometric scaling constant, r is the per-capita adult reproductive rate, μ_J is the mortality rate of juveniles and η_J is the between-stage maturation term ($\frac{1}{\eta_J}$ is the mean length of the pre-reproductive stage). The juvenile population size increases through births (at rate r) and declines through natural mortality (at rate μ_J), within-class competition (at rate $\gamma_{JJ} \cdot N_J \cdot m_J^{\beta}$) and maturation (at rate η_J). The dynamics of the adult age-class are:

$$\frac{dm_A}{dt} = \frac{a_A \cdot m_A^{\beta}(t)}{1 + \gamma_{AA} \cdot N_A(t) \cdot m_A^{\beta}(t)} - b_A \cdot m_A(t) \tag{3}$$

$$\frac{dN_A}{dt} = \eta_J \cdot N_J(t) - \left[\mu_A + \gamma_{AA} \cdot N_A(t) \cdot m_A^{\beta}(t)\right] \cdot N_A(t) - \eta_A \cdot N_A(t) \tag{4}$$

where a_A and b_A are resource uptake and utilisation rates, γ_{AA} is the strength of intraspecific competition within the adult age-class and $\frac{1}{\eta_A}$ is the reproductive stage life expectancy (mean lifespan). The adult population size increases through maturation (at rate η_J) and declines due to natural mortality (at rate μ_A), the attainment of maximum lifespan (η_A) and within-class competition (at rate $\gamma_{AA} \cdot N_A \cdot m_A^{\beta}$). The parameter definitions for this model are given in Table 1.

This model (Eqs. (1)–(4)) has a single strategy with two age classes and a distributed lifespan. Our next set of models have multiple strategies, no explicit age structure and distributed or fixed lifespans. Into these models, we incorporate both intraspecific and interspecific competition. This allows us to explore the evolutionary dynamics of longevity in terms of trade-offs between mortality rate and density-mediated competition. Common to all these models is the dynamics of mass gain and the individual mass (m_i) dynamics of the ith strategy are:

$$\frac{dm_i}{dt} = \frac{a_i \cdot m_i^{\beta}(t)}{1 + \sum_j \gamma_{ij} \cdot m_j^{\beta}(t) \cdot N_j(t)} - b_i \cdot m_i(t) \tag{5}$$

where a_i is the resource uptake rate, b_i is the resource utilisation rate, γ_{ij} is the strength of density dependence (between the ith and jth strategies, assumed to be symmetric between strategies), β is an allometric scaling constant and N_j is the size of the jth population. All parameter definitions for this set of models are given in Table 2.

2.1.2. Model II — Fixed lifespan

A fixed lifespan model (Bonsall and Mangel, 2004) allows us to separate the rate of mortality (μ_i) from maximum lifespan (τ_i). The

Table 2
Parameter definitions for the density-mediated competition models (Models II & III).

	Parameter	Definition
General parameters	λ_i	Fitness of the ith strategy
	$f(\mu_i)$	Mutated mortality rate of the ith strategy
Mass gain	m_i	Individual mass gain of the ith strategy
	a_i	Resource uptake rate of the ith strategy
	b_i	Resource utilisation rate of the ith strategy
	β	Allometric scaling constant
Population-level	N_i	Population size of the ith strategy
	r_i	Per capita recruitment rate of the ith strategy
	μ_i	Mortality rate of the ith strategy
	γ_{ij}	Strength of competition between the ith and jth strategy
	τ_i	Maximum lifespan
	η	Common basal lifespan

population dynamics of the ith strategy are now:

$$\frac{dN_i}{dt} = r_i \cdot N_i(t) - \left(\mu_i + \sum_j \gamma_{ij} \cdot m_j^{\beta}(t) \cdot N_j(t)\right) \cdot N_i(t) - S_i(t, \tau_i) \quad (6)$$

where r_i is the per-capita recruitment rate of the ith strategy and $S_i(t, \tau_i)$ is the through-class survival term:

$$S_i(t, \tau_i) = r_i \cdot N_i(t - \tau_i) \times \exp - \left(\int_{t-\tau_i}^{t} \left[\mu_i(x) + \sum_j \gamma_{ij} \cdot m_j^{\beta}(x) \cdot N_j(x)\right] dx\right). \quad (7)$$

Population size now changes through increases due to recruitment, declines through extrinsic mortality, competition, and the attainment of a maximum lifespan.

2.1.3. Model III — Distributed lifespan

If we assume that lifespan is exponentially distributed (MacDonald, 1978, 1989) then the population dynamics of the ith strategy are:

$$\frac{dN_i}{dt} = r_i \cdot N_i(t) - \left(\mu_i + \sum_j \gamma_{ij} \cdot m_i^{\beta}(t) \cdot N_j(t)\right) \cdot N_i(t) - \eta \cdot N_i(t). \quad (8)$$

In the absence of competition, the mean lifespan of the ith strategy would be $\frac{1}{(\mu_i+\eta)}$. Thus, this is a model of an adaptive radiation occurring through the evolution of species-specific values of mortality rates (μ_i) under a common basal lifespan η. We refer to the case where $\eta = 0$ as the no-lifespan model.

2.2. Evolutionary dynamics

We use approaches similar to adaptive dynamics (Metz et al., 1992, 1996; Geritz et al., 1998; Waxman and Gavrilets, 2005) to explore the evolutionary consequences of mutations in mortality rates. Adaptive dynamics is based on three main assumptions:

(i) Ecological and evolutionary timescales are separated
(ii) Mutations are infinitesimal
(iii) Mutant frequencies are rare as they invade a resident population.

In contrast, we consider the reciprocal invasion conditions when mutations are finite (and sometimes small) to determine when an alternative strategy will replace a resident strategy. We proceed by starting with an initial single strategy, forward iterating the ecological model and thus creating a background in which a mutation evolves. We then determine the probability of a mutation around an extant strategy and evaluate whether this new strategy (i) fails to invade, (ii) invades and replaces or (iii) invades and coexists with the group of extant strategies.

The appropriate measure of fitness of initially rare strategies invading existing resident strategies when density dependence operates is the per capita growth rate (Metz et al., 1992; P'asztor et al., 1996). By evaluating changes in fitness with respect to a specific life-history trait, invasion dynamics, evolutionary maxima and minima (i.e. when the change in fitness is zero), and Evolutionary Stable States (ESS) can be determined. The life-history trade-offs are explicitly linked to the population dynamics and the optimal ESS may change as these variables alter (P'asztor et al., 1996; Bonsall, 2006). The curvature of the function around the ESS points provides a measure of the strength of selection and an indication of the shape of the fitness surface.

We are able to couple theories of senescence (Medawar, 1952; Williams, 1957) and mechanisms of evolutionary diversification. Links between the mechanisms of aging (mutations in mortality rate) and the implications of trade-offs (optimality and life-histories) for the evolution of lifespan can be explored with Model I. Mutations can either be additive ($\mu \to \mu + \delta\mu$) or proportional ($\mu \to \mu(1+\delta\mu)$). An additive mutation in mortality rate acting on the juvenile stage (N_J') leads to the following population dynamics for the alternative strategy (N_J', N_A'):

$$\frac{dN_J'}{dt} = r \cdot N_A'(t) - \left[(\mu_J + \delta\mu_J) + \gamma_{JJ} \cdot N_J^* \cdot m_J^{*\beta}\right] \cdot N_J'(t) - \eta_J \cdot N_J'(t) \quad (9)$$

$$\frac{dN_A'}{dt} = \eta_J \cdot N_J'(t) - \left[\mu_A + \gamma_{AA} \cdot N_A^* \cdot m_A^{*\beta}\right] \cdot N_A'(t) - \eta_A \cdot N_A'(t) \quad (10)$$

where N_J' and N_A' are abundances of the juvenile and adult classes for an invading strategy, $\delta\mu$ is the change in mortality rate, and N_J^*, N_A^*, $m_J^{*\beta}$, and $m_A^{*\beta}$ are the equilibrial population sizes and mass gains for the resident strategy (determined from Eqs. (1)–(4)). Similarly, an additive mutation in mortality rate acting on the reproductive stage (N_A') leads to:

$$\frac{dN_J'}{dt} = r \cdot N_A'(t) - \left[\mu_J + \gamma_{JJ} \cdot N_J^* \cdot m_J^{*\beta}\right] \cdot N_J'(t) - \eta_J \cdot N_J'(t) \quad (11)$$

$$\frac{dN_A'}{dt} = \eta_J \cdot N_J'(t) - \left[(\mu_A + \delta_{\mu A}) + \gamma_{AA} \cdot N_A^* \cdot m_A^{*\beta}\right] \times N_A'(t) - \eta_A \cdot N_A'(t). \quad (12)$$

The fitness (λ) of an invading strategy when mutations act in the juvenile stage can be obtained from the determinant of (Pielou, 1977):

$$\begin{pmatrix} \lambda + \left[(\mu_J + \delta\mu_J) + \gamma_{JJ} \cdot N_J^* \cdot m_J^{*\beta}\right] + \eta_J & -r \\ -\eta_J & \lambda + \left[\mu_A + \gamma_{AA} \cdot N_A^* \cdot m_A^{*\beta}\right] + \eta_A \end{pmatrix} \quad (13)$$

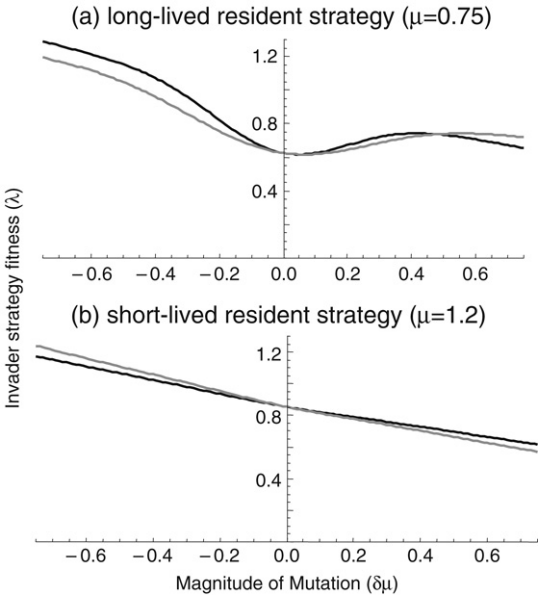


Fig. 1. Effects of the magnitude of mutation on invader fitness under additive (black line) and multiplicative mutations (grey line) under (a) long-lived resident strategy ($\mu = 0.75$) and (b) short-lived resident strategy ($\mu = 1.2$). Mortality rates similar to the resident ($\delta\mu = 0.0$) are costly in terms of fitness whereas large mutations that lead to distinct long-lived strategies are more likely to invade. In general, mutations that favour increased longevity ($\mu \to 0$) lead to higher fitness gains. However, the nature of the mutation (additive, multiplicative) on fitness depends on the background (resident) trait values. The absolute value is dependent on parameter values, and populations grow if $\lambda > 0$.

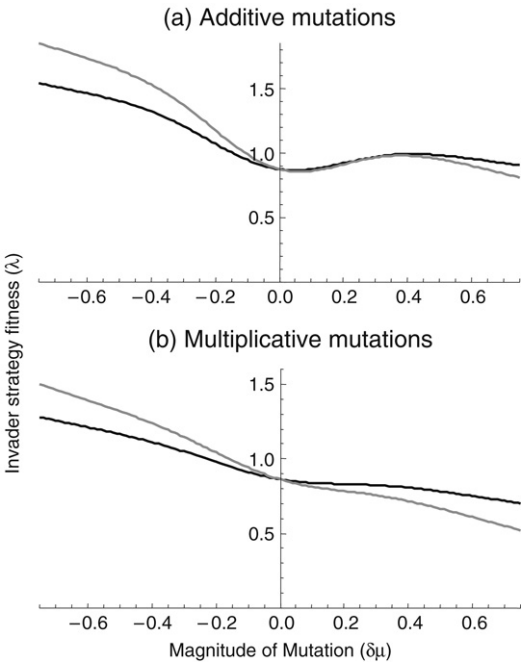


Fig. 2. Effects of early (black line) versus late (grey line) mutations on invader fitness under (a) additive and (b) multiplicative mutations. Late acting mutations have a more costly effect on the evolution of short-lived strategies ($\delta\mu > 0$) but may favour the evolution of effects that increase longevity ($\mu \to 0$) as the mutations lead to higher fitness gains.

where the entries in this matrix are the partial derivatives (i.e., $\frac{\partial \frac{dN_J'}{dt}}{\partial N_J'}$, $\frac{\partial \frac{dN_J'}{dt}}{\partial N_A'}$, $\frac{\partial \frac{dN_A'}{dt}}{\partial N_J'}$ and $\frac{\partial \frac{dN_A'}{dt}}{\partial N_A'}$) associated with the invader strategy in the presence of the resident strategy (which is at equilibrium, $N_J^*, N_A^*, m_J^{*\beta}, m_A^{*\beta}$).

2.3. Trade offs

Throughout, to describe the form of the competition-longevity trade-off we use a standard Gaussian function (Bonsall and Mangel, 2004; Mangel et al., 2007):

$$\gamma_{ij} \cdot \exp\left(-\frac{1}{2}\left[\frac{f(\mu_i) - \mu_j}{\sigma}\right]^2\right) \tag{14}$$

where $f(\mu_i)$ is the invader's trait value or function, μ_j is the resident's trait value and σ is a measure of the intensity of competition operating between the different strategies. This is the fundamental competition (γ_{ij}) – longevity trade-off: a large of value of γ_{ij} can be mitigated if $f(\mu_i)$ is quite different from μ_j. While there are other ways of describing trade-offs (Bonsall and Mangel, 2004; Mangel et al., 2007) this functional form has been extensively used in quantitative genetics and provides a biologically plausible way to describe life-history trade-offs (e.g. Kisdi (1999)).

3. Results

3.1. Magnitude and direction of mutation

Fitness critically depends on the nature of mutation and the trait background into which it invades (Fig. 1). The effects of mutations in mortality rate (longevity $\sim$ 1/mortality) on fitness are weaker when the mutations are small and the trait values are similar to the resident ($\delta\mu \to 0$). In the limit as $\delta\mu \to 0$ any fitness benefits are manifest through the population dynamics and demographic effects of differences in the strengths of competition. In contrast, mutations that are of relatively large magnitude and decrease mortality rate (increase longevity) tend, in general, to have a positive effect on fitness. Mutations of large magnitude that increase mortality rate (shorter-lived strategies) have a weaker effect on fitness (Fig. 1). If the extant strategy is long-lived (Fig. 1(a)), then additive and proportional mutations favouring long-lived strategies have different fitness consequences, but if the resident strategy is short-lived, the fitness benefits are similar (Fig. 1(b)). Even though the fitness of short-lived strategies under this background trait value is weaker for both mutational models, proportional mutations leading to short-lived strategies have lower fitness than additive mutations leading to short-lived strategies (Fig. 1(b)).

The timing of mutations also has important fitness consequences (Fig. 2). Late acting mutations are always more detrimental to fitness than mutations acting early on the evolution of short-lived strategies. Late acting mutations (either multiplicative or additive) are more likely to promote the evolution of long-lived strategies. More specifically, these late acting mutations have limited effect, as the processes of competition outweigh any benefits that the small changes in mortality might confer. However, large additive and positive changes in mortality through mutations that act late are more costly, as they have a detrimental effect on reproductive capacity (Fig. 2(a)). Similarly, large multiplicative mutations acting late, have costly effects on the evolution of short-lived strategies (Fig. 2(b)). Moreover, if the magnitude of mutation is large and of similar size to the mortality rate, then the effects on fitness may be prohibitively costly on the evolution of shorter-lived strategies. In contrast, large negative changes in mortality (mediated by additive or multiplicative mutations) that act late, allow the evolution of long-lived strategies. This change occurs as alterations in mortality rate outweigh both the effects of competition and the positive effect on the reproductive capacity (Fig. 2(a) and (b)).

The evolutionary response to selection on mortality depends on both the type (additive, multiplicative) and the timing

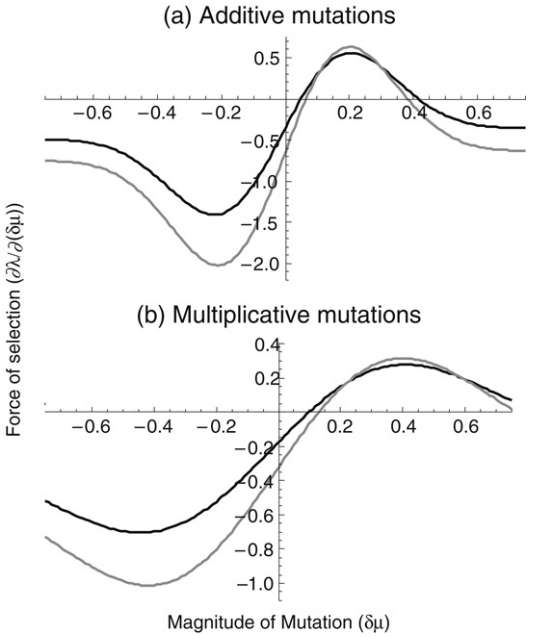


Fig. 3. The magnitude of early and late (a) additive and (b) multiplicative mutations on the force of selection. Fitness values that are close to existing strategies ($\delta\mu = 0$) are much more likely to be under disruptive selection. This effect is more pronounced under early acting mutations (black line) than late-acting mutations (grey line).

(early or late) of mutations (Fig. 3). When additive mutations act on mortality, selection has the strongest effect on invader fitness when the magnitude of these mutations is positive and relatively small (Fig. 3(a)). Mutations of increasing magnitude disproportionally affect invader fitness. Selection is stronger against mutations on mortality that act late than those acting early. When mutations have proportional effects on mortality, fitness tends to decline as the magnitude of the mutation effect on mortality rate increases. Depending on the strength of these mutations, selection acts strongest against late as opposed to early-onset mutations (Fig. 3(b)). Positive selection is strongest on multiplicative mutations of small effect that act late and favour shorter-lived strategies. If multiplicative mutations are of large effect, then selection is also stronger against late acting mutations that lead to extremely short or long-lived strategies.

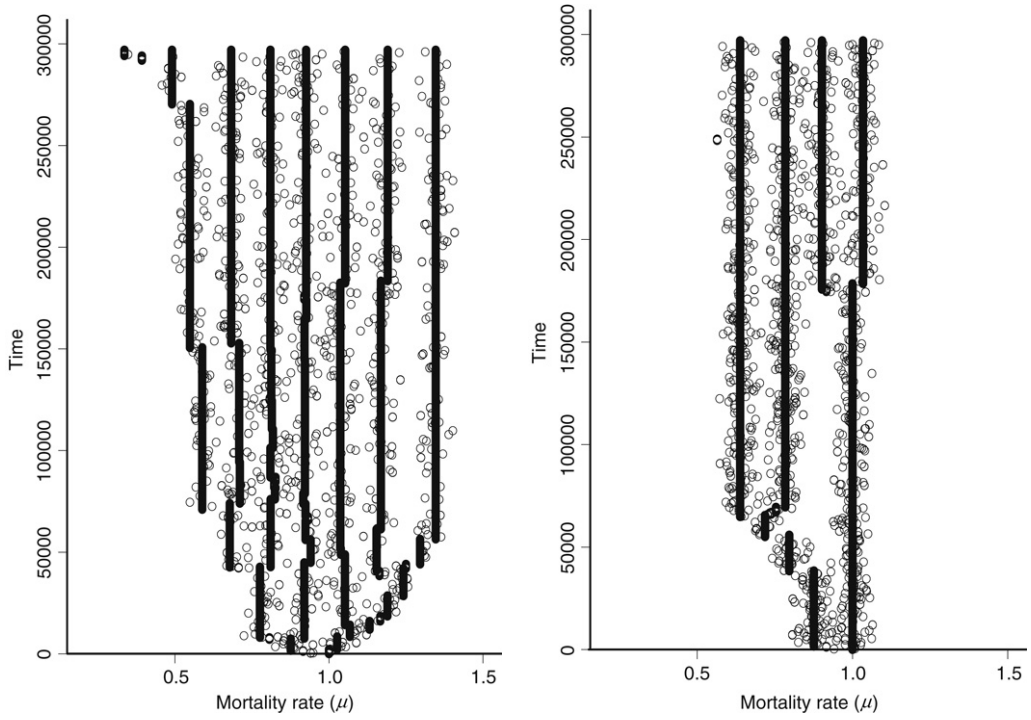
3.1.1. Invasion dynamics

Invasion trajectories illustrate the importance of the timing of mutations in determining evolutionary outcomes (Fig. 4). Mutations acting at the time of reproduction, reduce density and promote the evolution of short-lived strategies. Such mutations also promote the evolution towards increased longevity ($\mu \to 0$). In contrast, mutations acting early in life are more likely to contribute to the evolution of many different coexisting strategies, in particular both long and short-lived strategies (Fig. 4); this is similar to the classical, density independent result.

3.2. The importance of flat fitness surfaces

The fitness functions, first derivative, ESS conditions and curvature around each evolutionary stable point for the fixed and distributed lifespan models are given in Table 3. The fitness surfaces for changes in mortality rate (μ_i) of the invader with one resident strategy (μ_j) for these models are shown in Fig. 5. For each model, there is always at least one ESS at maximum longevity ($\mu_i \to 0$). In this limit, there is no density-independent mortality but there are still density-dependent effects. Moreover, the underlying age-structure determines whether alternative evolutionary stable points exist. For each model (Table 3) $\frac{d^2\lambda_i}{d\mu_i^2} < 0$ holds if:

$$\frac{\exp(-(\frac{\mu_i-\mu_j}{2\cdot\sigma})^2)}{\sigma^2} \to 0 \tag{15}$$



(a) Early acting mutation. (b) Late acting mutation.

Fig. 4. Numerical simulations of the evolution of diversification under (a) early-acting and (b) late-acting additive mutations. Diversification is much more likely under early-acting mutations where the effects of directional selection are stronger.

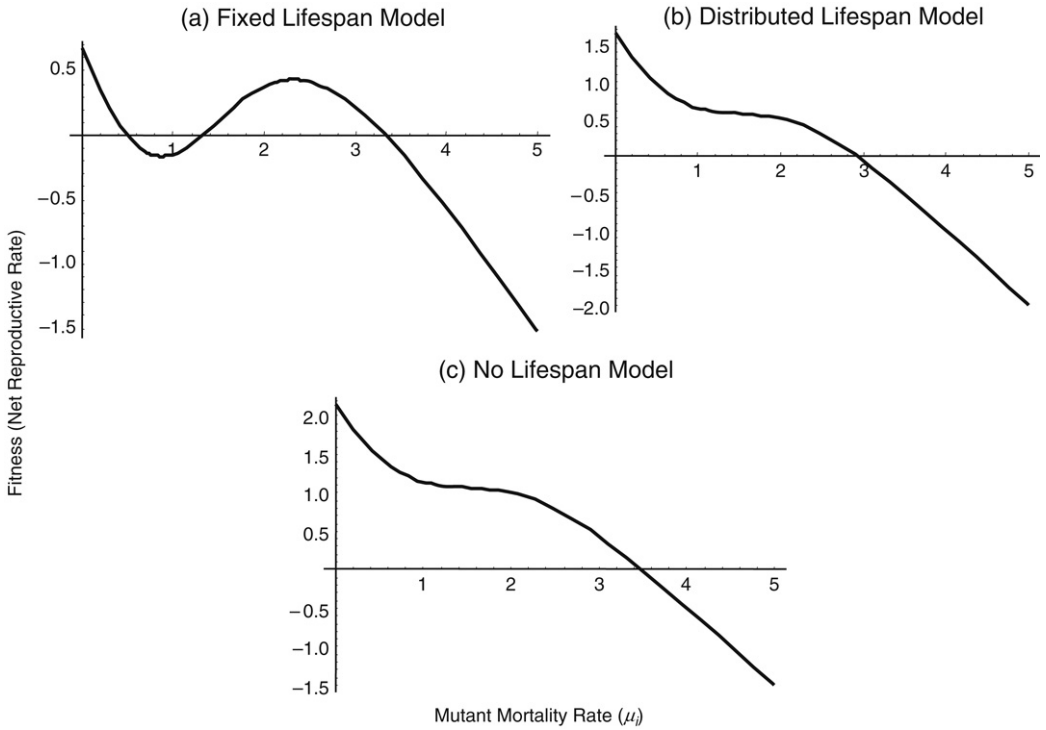
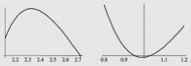


Fig. 5. Fitness surfaces for the density-mediated interspecific competition models [(a) fixed lifespan model, (b) distributed lifespan model, (c) no-lifespan model] with respect to a single fixed resident natural mortality rate ($\mu_j = 1.0$). In all cases there is an important (density-independent) ESS as $\mu_i \to 0$. Other evolutionary maxima and minima result from the effects of competition-longevity trade-off and demographic structure.

Table 3

Fitness function (λ_i), partial selection differentials ($\frac{d\lambda_i}{d\mu_i}$), second derivatives ($\frac{d^2\lambda_i}{d\mu_i}$) and patterns of curvature around ESS points for the density-mediated interspecific competition models.

	Fitness function (λ_i)	Partial selectiondifferential - ESS critiera: $\frac{d\lambda_i}{d\mu_i} = 0$
Fixed lifespan	$r - \mu_i - \gamma_{ij} \cdot \exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) \cdot m_j^{*\beta} \cdot N_j^* - r_i \cdot \exp(-\left[\mu_i + \gamma_{ij} \cdot \exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) \cdot m_j^{*\beta} \cdot N_j^*\right] \cdot \tau_i)$	$-1 + \frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)}{\sigma^2} - \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) - \exp(-\left[\mu_i+\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*\cdot\tau_i\right])\cdot m_j^{*\beta}\cdot N_j^* r_i\cdot(\mu_i-\mu_j)\cdot\gamma_{ij}\cdot\tau_i}{\sigma^2}$
Distributed lifespan	$r_i - \mu_i - \gamma_{ij} \cdot \exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) \cdot m_j^{*\beta} \cdot N_j^* - \eta$	$-1 + \frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)}{\sigma^2}$
No lifespan	$r_i - \mu_i - \gamma_{ij} \cdot \exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) \cdot m_j^{*\beta} \cdot N_j^*$	$-1 + \frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)}{\sigma^2}$
	Second derivative - ESS criteria: $\frac{d^2\lambda_i}{d\mu_i^2} < 0$	
Fixed lifespan	$-\frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)^2}{\sigma^4} + \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*}{\sigma^2} - \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2) - \exp(-\left[\mu_i+\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*\cdot\tau_i\right])\cdot m_j^{*\beta}\cdot N_j^* r_i\cdot\gamma_{ij}\cdot\tau_i}{\sigma^2} - \frac{\exp(-\left[\mu_i+\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*\cdot\tau_i\right])\cdot m_j^{*\beta}\cdot N_j^* r_i\cdot(\mu_i-\mu_j)\cdot\gamma_{ij}\cdot\tau_i\cdot\left[-\frac{\mu_i-\mu_j}{\sigma^2} + \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)\cdot\gamma_{ij}\cdot\tau_i}{\sigma^2}\right]}{\sigma^2}$
Distributed lifespan	$-\frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)^2}{\sigma^4} + \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*}{\sigma^2}$	
No lifespan	$-\frac{\gamma_{ij}\cdot\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*(\mu_i-\mu_j)^2}{\sigma^4} + \frac{\exp(-(\frac{\mu_i-\mu_j}{2\sigma})^2)\cdot m_j^{*\beta}\cdot N_j^*}{\sigma^2}$	
	Curvatures - Second order expansion of fitness function	
Fixed lifespan		
Distributed lifespan		
No lifespan		

which is true if $|\mu_i - \mu_j| \gg \sigma^2$. From the partial selection differentials (Table 3) $\frac{d\lambda_i}{d\mu_i} = 0$ if μ_i and μ_j are either equal or the magnitude of their difference is large. Under the former condition, this an unstable evolutionary minima as $\frac{d^2\lambda_i}{d\mu_i^2} > 0$ while, in contrast, under the latter condition, this is evolutionary stable as $\frac{d^2\lambda_i}{d\mu_i^2} < 0$. In general, life-history traits that are similar to existing strategies lead to unstable, divergent selection, while traits that are substantially different from extant strategies lead

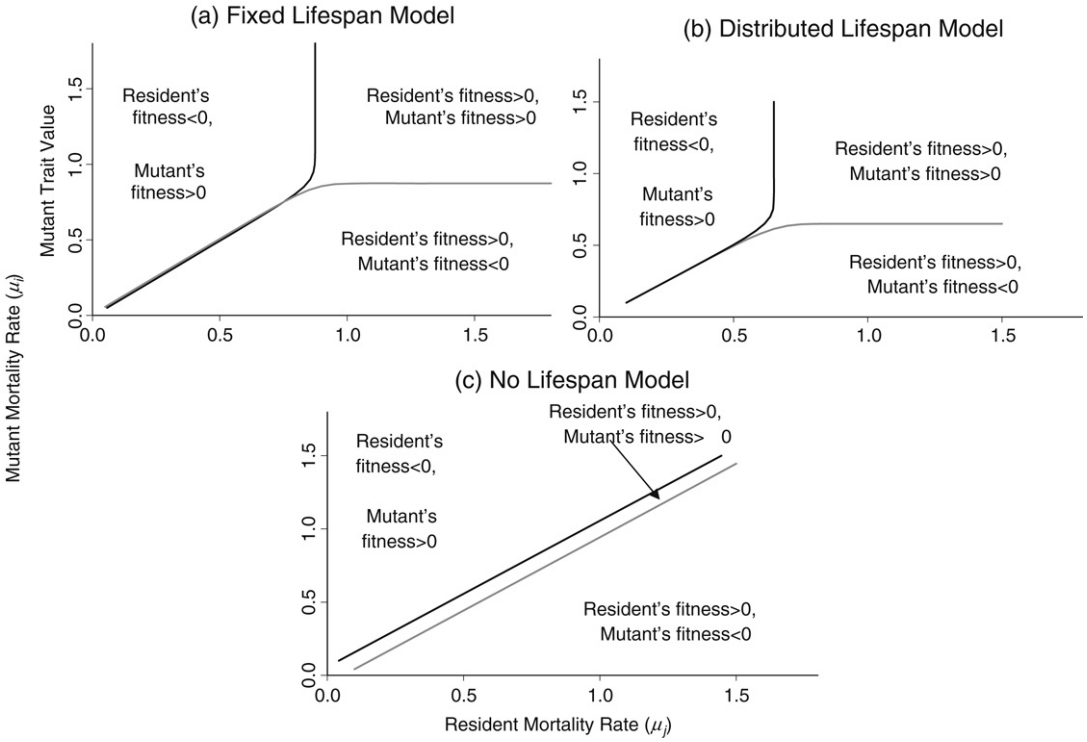


Fig. 6. Invasion boundaries for the density-mediated interspecific competition models with a single resident show the putative regions of pairwise co-occurrence of strategies with different natural mortality rates [(a) fixed lifespan model, (b) distributed lifespan model, (c) no-lifespan model]. Under the no-lifespan model, the possibility of coexistence of short-lived ($\mu_i \to \infty$) and long-lived ($\mu_i \to 0$) strategies is feasible. This is extended in the distributed and fixed models with the effects of demographic structure leading to similar invasion conditions.

to the coexistence of stable strategies. Substantial differences in traits can be quantified in terms of the curvature of the fitness surface; changes in demographic parameters (such as r_i, η) do not qualitatively change the shape of the fitness surface. In contrast, the shape of the fitness surface is affected by the population equilibrium values of the existing strategies (e.g. $N_j^*, m_j^{*\beta}$) and the intensity of competition (σ).

Flat fitness surfaces are predicted with the range of population demographic structures. Under the no-lifespan and distributed lifespan models, the fitness surfaces are similar due to the nested structure of these two models (Fig. 5(b) and (c)). More importantly, the fitness surface is relatively flat for invading strategies, with intermediate mortality rates and declines for mutant strategies that are extremely short-lived. The fixed lifespan model predicts fitness surfaces with multiple ESS points. Analysis of the curvature (Table 3) around each of the fitness maxima and minima reveals that functionally equivalent strategies are more likely to co-occur at fitness minima (where disruptive selection is predicted to be strongest) than at ESS points where strategies are short-lived.

The pairwise invasibility and putative boundaries of coexistence for the different between-strategy models are shown in Fig. 6. In each case, only mortality rate (μ_i) evolves. Other parameters can affect the boundaries of coexistence; for example, as $\eta \to 0$ the boundaries in Fig. 6(c) tend towards those in Fig. 6(a) reflecting the nested structure of these two models. In contrast, to the fitness surfaces, the fixed and distributed lifespan models have very similar invasion boundaries, suggesting that the initial trajectories of adaptive evolution under these demographies may be very similar.

3.3. Generation of diversity

Numerical solutions are required to consider more than two competing strategies and illustrate how strategies with different life-history traits may radiate and how patterns of coexistence can occur. Multiple strategies are predicted to co-occur (Fig. 7) and this follows an adaptive radiation: rapid phenotypic diversification as each strategy occupies a different part of niche space. Different phenotypic strategies may co-occur for long periods of time, and the number of extant strategies at any point may exceed the number present in the terminal assemblage. Comparing the evolutionary trajectories from the different models demonstrates that different population demographic dynamics feed back to affect diversification. The evolution of mortality rate under the fixed and distributed delay models promote adaptive radiations (Fig. 7). However, under the fixed lifespan model, there is greater variability with the evolution of both short- and long-lived strategies; in addition the distributed lifespan model shows directional evolution towards strategies with shorter lifespans (higher mortality rates). Coupling trait evolution and demographic structures allows fewer strategies to coexist than might be expected (not all possible niches are filled) and this introduces a form of demographically-driven and demographic-specific limiting similarity to the adaptive radiation. This phenotypic divergence results from asymmetry in competition and the development of included niches; more similar strategies experience greater effects of density-mediated interspecific competition and more distinct strategies with longer lived strategies exploit the scarce resources not consumed by the superior competitors.

4. Discussion

We have explored how density dependence and demography influence the patterns and processes associated with the evolution of lifespan and longevity. In particular, the competition-longevity trade-off operating within different structured populations affects both the patterns of species diversification and the mechanisms

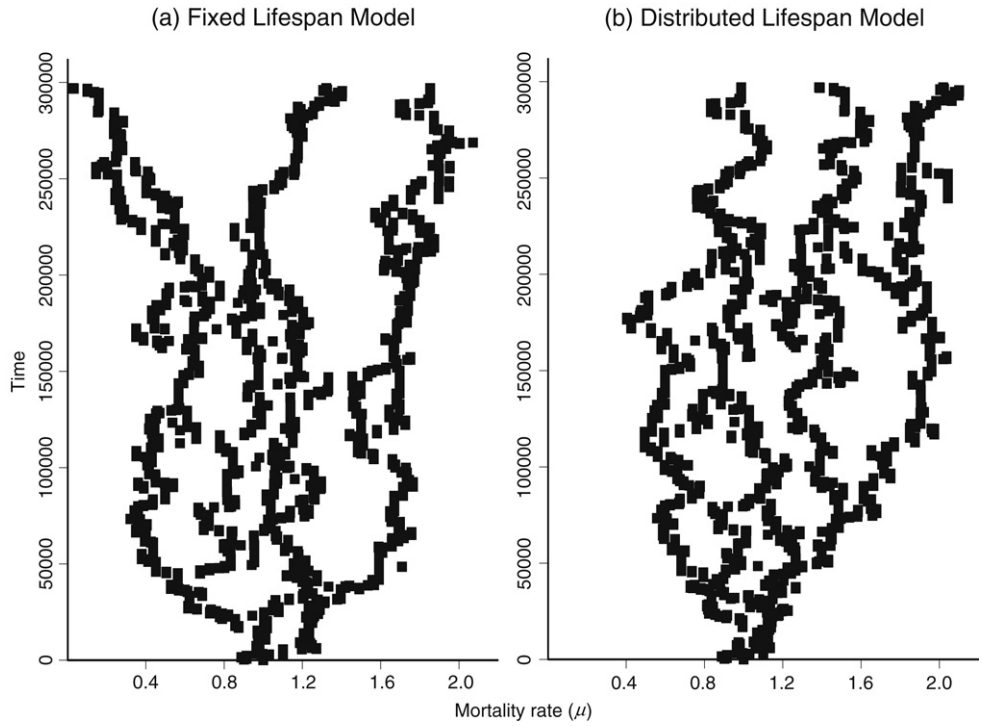


Fig. 7. Numerical simulations of the evolution of diversification under the (a) fixed lifespan and (b) distributed lifespan model. Initial strategies were started with the same conditions (Parameter value $\sigma = 0.05$).

maintaining diversity. A competition-longevity trade-off (see Eq. (15)) alleviates the intense effects of interspecific competition and favours the coexistence of both short- and long-lived strategies.

Understanding speciation and diversification necessitate determining how both divergence and coexistence co-occur. In the past, mechanisms associated with divergence have focussed on the modes of speciation (e.g. allopatric, sympatric, parapatric) and whether there is sufficient phenotypic and/or genetic heterogeneity to allow reproductive isolation to occur and be maintained (Dieckmann and Doebeli, 1999; Dieckmann et al., 2004). Mechanisms associated with coexistence following divergence, have received somewhat less attention; they require niche partitioning such as a competition-life history trait trade-off. Coexistence may be maintained through a variety of mechanisms such as fluctuating environments (e.g. Chesson and Warner (1981)) or spatial heterogeneity (e.g. Snyder et al. (2005)). However, the role of demographic structure coupled to life-history trade-offs, the heterogeneity that this introduces and its effects on coexistence following speciation, is a novel approach to this problem. We have shown that the effects of competition decline with the differences in lifespans, giving rise to disruptive selection, and lead to differing patterns of coexistence depending on the precise, underlying demographic dynamics and mutation rates.

One obvious question is how genetics act to constrain the asymptotic dynamics. These constraints may be manifest through deleterious mutations producing mutation-selection balance, or through the maintenance of genetic variation due to balancing selection. For instance, if mutations have finite (and small) effect then, when mutations balance selection, the segregation of alleles will lead to a distribution of effects (Kimura, 1965). While this distribution of effects may be symmetrical, it is entirely possible that some mutations of large effect may arise. While this might pose difficulties for assessing infinitesimal change under adaptive dynamics, it is entirely feasible to determine the outcome of large mutations on patterns of strategy co-occurrence under the evolutionary invasion approach we have developed here. Similarly, the maintenance of variation through balancing selection (via a frequency-dependent selection mechanism) is manifest through the selection-replacement dynamics. The numerical approach illustrated in Figs. 4 and 7 shows how differences in phenotypic traits affect both the patterns of diversification (directional selection) and the maintenance of diversity (balancing selection).

4.1. Density-dependence and the evolution of diversity

In density-independent theory, patterns in life-history evolution depend strongly on the age classes that are affected by selection (Charlesworth, 1994). Here, we have shown that density-dependent processes interact with demographic structure to affect the optimal mortality rate. Incorporating more explicit demographic details allows qualitatively new results on the evolution of optimal life histories (Gadgil and Bossert, 1970; Schaffer, 1974). For example, strategies are organised into distinct guilds, groups or clades (niches) and between guilds there is a degree of limiting similarity (Bonsall and Mangel, 2004; Bonsall et al., 2004), with the fitness amongst strategies often equivalent. Similarly, it is the density-dependent life-history trade-offs that lead to the evolution of strategies with increased lifespan.

The structure of many ecological assemblages and the maintenance of diversity in speciose groups (e.g., cichlids, rockfishes, finches) is likely to be driven by trade-offs, such as those between competitive ability and life-history traits. As illustrated in this study, this gives rise to flat fitness surfaces (different routes to equivalent fitness), and contrasts markedly with the ideas that fitness landscapes are dominated by a single optimum or that they are rugged (Gavrilets, 2004). The ultimate mechanisms underpinning flat fitness surfaces obviously require much more detailed theoretical and empirical attention, but one mechanism is that the costs of different age-structures (expressed through differences in development and/or physiology) lead to the evolution of different phenotypes (Mangel et al., 2007).

4.2. Senescence and the role of mutation

Senescence leads to a decrease in fertility and/or increase in mortality with age. The general theory of senescence argues that natural selection acts differentially with age. In age-structured populations, genes acting early in life to affect reproduction or survival have a greater impact on fitness than genes expressed later in life and the accumulation of mutations will have greater effect later in life (Medawar, 1952; Williams, 1957; Hamilton, 1966). While different specific theories on the mechanisms of the evolution of senescence have been proposed (mutation accumulation, antagonistic pleiotropy, disposable soma) and have been experimentally verified (Rose and Charlesworth, 1980; Service, 1987; Hughes and Charlesworth, 1994; Hughes and Reynolds, 2005; Charmantier et al., 2006; Reznick et al., 2004, 2006), the effects of mutation on the evolution of diversity (mediated by life history trade-offs in longevity) have remained relatively unexplored. We have shown that fitness is affected by the magnitude and direction of mutation. Those mutations of moderate effect allow alternative strategies to invade and co-occur (by reducing the effects of competition). Consistent with density independent theory, selection is stronger on mutations that affect mortality at the time of reproduction, and this mutational background has consequences not only the evolution of longevity but also for the processes of speciation and diversification.

In conclusion, the importance of ecological trade-offs, such as those between investing in longevity and investing in competitive ability, affect the likelihood of co-occurrence of different strategies. Here, we have shown that these trade-offs can lead to the coexistence of multiple strategies with differing life-history characteristics typically observed in many speciose assemblages (e.g., cichlids, rochfishes, finches). Both short- and long-lived strategies can evolve and co-occur. Our results emphasise the importance of both population structure (e.g., density-dependence, age-structure) and mutation rate, as processes affecting the evolutionary patterns of longevity.

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