Longevity hinders evolutionary rescue through slower growth but not necessarily slower adaptation

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Abstract

"Evolutionary rescue" is the process by which a population experiencing severe environmental change avoids extinction through adaptation. Theory predicts when rescue is favored, but currently relies on assumptions of non-overlapping generations and therefore annual, semelparous life cycles. It has been hypothesized that longevity and iteroparity impede rescue by slowing rates of adaptation, although this hypothesis has rarely been tested. We develop a model with longevity determined by the balance of survival and reproduction and selection acting on survival through a quantitative trait. We analytically characterize the demography and evolution of cohorts, demonstrating that longevity allows repeated selection over the lifetime, with the surprising finding that this can decouple mean genotype and phenotype within a cohort. We then simulate populations at three longevities responding to environmental change. Under perfect trait heritability, longevity produces negligible differences in adaptation rates, but under low heritability, high-longevity populations experience a transient phase of rapid phenotypic change accompanied by slower genotypic change. More importantly, longevity is associated with slower intrinsic population growth rates regardless of trait heritability, ultimately hindering rescue. We connect these results to prior demographic and evolutionary theory and recent efforts to incorporate longevity into models of adaptation.

Introduction

Evolutionary rescue, the phenomenon of populations avoiding extinction following environmental change by adapting to novel conditions, has emerged as an important concept in recent decades due to climate change and widespread habitat alteration and degradation. This includes both theoretical (Carlson et al. (2014), Kopp & Matuszewski (2014)) and empirical work (e.g., Bell & Gonzalez (2009), Agashe et al. (2011), Hufbauer et al. (2015)), with considerable progress in generating and testing predictions about which factors influence population persistence following severe environmental change. At present, models of evolutionary rescue assume non-overlapping generations and an absence of age or stage structure (e.g., Bürger & Lynch (1995), Gomulkiewicz & Holt (1995), Barfield & Holt (2016)). While these assumptions increase analytical tractability and allow for validation with model systems where life history can be controlled in the laboratory (e.g., algae [Bell & Gonzalez (2009), Bell (2012)], flour beetles [Agashe et al. (2011), Hufbauer et al. (2015)], and *Daphnia* [Loria et al. (2022)]), they do not reflect the life history of many organisms, particularly organisms with large body size or long life cycle likely to be of conservation concern (Purvis et al. (2000), Dirzo et al. (2014)).

Models with non-overlapping generations assume a discrete life cycle where individuals' lifespans last at most one breeding period, typically cycling annually (Young (1981), Morris (2009)), as well as a semelparous reproductive strategy where individuals only have one reproductive bout during the lifespan (Cole (1954), Young (1981), Morris (2009)). In contrast, overlapping generations can arise due to perenniality (lifespan potentially spanning multiple years or breeding periods, Young (1981), Morris (2009)) or iteroparity (multiple reproductive episodes over the lifespan, Cole (1954)). Both of these life-history strategies rely on longevity, or the ability to survive between years or breeding periods, a feature which is mostly absent from contemporary models of rescue (although see Draghi et al. (2024), discussed below). High longevity and associated life-history traits are hypothesized to hinder evolutionary rescue and environmental tracking by slowing rates of phenotypic change, henceforth referred to as the "slow adaptation

hypothesis", which posits that increasing longevity increases generation time and thus slows rates of phenotypic change through reduced generational turnover (Chapin et al. (1993), Vander Wal et al. (2013)) or life history stages buffered from selection (Shoemaker & Lennon (2018)).

Although models examining the effects of longevity on populations adapting to changing environments are rare, there have been advances supporting the slow adaptation hypothesis. However, these results may generalize poorly to long-lived organisms. Kuparinen et al. (2010) and Cotto et al. (2017) found that rates of phenotypic change slowed with increasing adult survival in response to environmental change in models of boreal trees and alpine plants, respectively. Both models assumed selection operated only on seedling establishment; maladapted adults therefore could be removed from the population and thus would continue to contribute maladaptive alleles to the gene pool. While realistic in several settings, this assumption is often violated in nature, for example when traits influencing adult survival such as body size are subject to selection (Milner et al. (1999), Ozgul et al. (2010)). Selection on adult survival is an example of "viability selection," i.e., differential survival among phenotypes, which differs from "fertility selection," differential number of surviving offspring among phenotypes. Both of these types of selection can change population phenotypic distributions (Coulson & Tuljapurkar (2008)), but the theory of rescue primarily focuses on fertility selection. Schmid et al. (2022) likewise found support for the slowing adaptation hypothesis with a model of a multi-stage life cycle where selection acted upon the transition from juveniles to adults. They noted that longevity allowed for life cycle complexity and "storage" of individuals in life stages that are shielded from selection. However, this is not a necessary feature or consequence of longevity. Dormancy illustrates this: dormant stages are often shielded from challenging or stressful environments, but dormancy appears in many organisms considered "short-lived" in their non-dormant forms, such as annual plants *via* seed banks (Templeton & Levin (1979)), freshwater copepods (Hairston & de Stasio (1988)), and even microbes (Shoemaker & Lennon (2018)). Thus while life-cycle buffering can slow rates of adaptation, it does not necessarily follow that all long-lived organisms will face slower adaptation following environmental change.

Draghi et al. (2024) created the first model to our knowledge to directly examine the effects of overlapping generations on rescue. Their analysis demonstrated that when maladaptation had stronger effects on survival than reproduction, then increasing maladaptation increased generation times. Increasingly long generation times as populations evolved were associated with reduced capacity to adapt to quickly-changing environments. Crucially, their analysis only focused on changes in generation time during rescue and did not explore variation in baseline (equilibrium) rates of longevity. This still leaves open the question of how life history variation influences evolutionary rescue.

Whereas the contemporary rescue literature focuses primarily on longevity's effects on rates of adaptation, demographic and life-history theories demonstrate that longevity has a trade-off with population- or lineage-level rates of increase. The r-vs-K selection hypothesis posits that certain species are "r-selected" and invest more in reproduction that allows them to achieve rapid growth at low density (Pianka (1970)). In contrast, "K-selected" species invest more in survival and competitive ability, stabilizing growth rates in harsh environments but slowing intrinsic population growth rates (Pianka (1970)). This trade-off is similarly apparent in the "fast-slow" pace-of-life continuum, where species closer to the "fast" end of the continuum have higher low-density population growth rates than species on the "slow" end (Oli (2004), Salguero-Gómez et al. (2016)). Thus, while high survival that produces longevity can be advantageous for stabilizing population size in harsh or fluctuating environments or when populations are at low densities, a population comprising these longer-lived individuals will take longer to return to original size following a dramatic reduction in size due to its lower growth rate (Pimm et al. (1988)). Life history theory also holds that population persistence over time depends on the geometric (multiplicative) mean of population growth rates over time: holding expected (arithmetic mean) rates of annual population growth constant between two populations with differing variances in annual growth rates, persistence of the population with less variable growth is favored because its geometric mean growth rate will be higher (Lewontin & Cohen (1969)). Annual and semelparous strategies typically have higher variance in population growth rates owing to large variance in

realized reproductive output. The synthesized result is that low-longevity, faster pace-of-life strategies (associated with semelparity, low annual survival, and non-overlapping generations) are typically classified as "boom and bust" due to higher expected rates of population growth but with higher variance among years, while high-longevity, slower pace-of-life strategies (associated with iteroparity, higher annual survival, and overlapping generations) have population growth rates that are constrained but more temporally stable.

Models of rescue with only non-overlapping generations and annual, semelparous life history strategies thus characterize one extreme of the fast-slow continuum. This is despite many threatened species and populations having "slow" pace-of-life characteristics, such as longer gestation time (Purvis et al. (2000)), large body size (Dirzo et al. (2014)), or smaller litter size per reproductive bout (Cardillo (2003)). As such, current predictions about rates of adaptation or recovery to initial size might poorly generalize to many conservation targets. As suggested above, rates of adaptation could be slower under longevity and overlapping generations, and delayed reproduction and smaller litters could constrain population growth if the environmental change drastically reduces population size. In this case, current theory of rescue, which does not account for longevity and overlapping generations, might underestimate extinction risk for many conservation targets. Alternatively, the stabilizing influences of longevity on population growth rates either during initial phases of decline or at low density, or viability selection acting through adult survival, might lower or negate extinction risks in high-longevity populations relative to low-longevity populations. This is a critical gap in theory that limits our ability to predict population fates under severe environmental change, particularly in long-lived organisms likely to be actively managed or of conservation concern.

Here, we seek to address this issue with a simple demographic model with only two vital rates (fecundity and survival), with no stage structure and simple age structure. Our model applies selection on all individuals in each time step; as such, unlike other models modifying longevity mentioned above, individuals are subject to successive rounds of viability selection. We examine the effects of longevity by manipulating survival and fecundity such that expected

lifetime fitness remains constant among strategies even as longevity varies. We address how pace of life influences population size, rates of phenotypic and genotypic change, and age structure over time. We also demonstrate the effects of phenotypic variance and trait heritability (i.e., the degree to which offspring trait values reflect parental trait values) on rates of phenotypic and genotypic change and incorporate them into our analysis. Finally, we examine the net effect of these processes on the timing and overall probabilities of extinction.

Methods

Analytical model

Consider a population where demography is determined by two vital rates: survival and reproduction. The population exists in an environment with discrete time steps; each time step, individuals experience mortality influenced by a quantitative trait as described below. After mortality, surviving individuals reproduce with a mean fecundity per individual of r, which is identical across individuals. The expected population growth rate in time step t is $\mathbb{E}[\lambda_t] = \bar{s}_t + \bar{s}_t r = \bar{s}_t(1+r)$, where $\bar{s}_t$ is the mean survival in the population at time t.

An individual's trait takes on a phenotypic value ζ_i, which measures the difference between the individual's trait value and that favored by the environment. Under this construction, fitness is maximized for an individual with $\zeta_i = 0$. We assume Gaussian selection with a selection surface of width ω such that $s(\zeta_i) \propto \exp\left(-\zeta_i^2/2\omega^2\right)$ (Lande (1976)). For simplicity, we assume that selection operates with the same strength across the lifespan (i.e., ω is independent of age). This allows us to scale phenotypes and their respective components by the width of the selection surface; define $z_i = \zeta_i/\omega$. If σ^2 denotes the variance of unscaled phenotypes, then the variance of scaled phenotypes is $\gamma^2 = \sigma^2/\omega^2$. Large γ^2 corresponds to high phenotypic variance subject to strong selection, while low γ^2 corresponds to low phenotypic variance subject to relatively weak selection. We will focus our analysis on scaled phenotypes (z) rather than un-scaled phenotypes (ζ) for ease of notation. The phenotype of individual i can be decomposed into the sum of a

genetic contribution (henceforth, "breeding value"), b_i, and a non-inherited contribution, (henceforth, "environmental component"), e_i (Falconer & Mackay (1996)). We denote the variance in breeding values within a population as γ_a^2 and the variance of environmental components as γ_e^2. We assume random mating in the population, such that γ_a^2 is also the additive genetic variance for the trait in the population (Lynch & Walsh (1998)). For an individual with phenotype z_i, survival in one time step is $s(z_i) = \widehat{s}\exp\left(-z_i^2/2\right)$, where $\widehat{s}$ is the annual maximum rate of survival, i.e., the annual survival probability of an individual perfectly adapted to the environment. In an environment that is constant, the intrinsic fitness (expected lifetime number of offspring) of an individual with survival s_i is $W_i = s_i r/(1 - s_i)$, since the number of time steps individual i lives for is geometrically distributed with rate s_i, and expected reproductive output in each time step is r.

Life history strategies

We denote the maximum absolute intrinsic fitness, i.e., the expected number of offspring per individual perfectly adapted to its environment, as $\widehat{W} = \widehat{s}r/(1 - \widehat{s})$. Henceforth, we will hold $\widehat{W}$ constant among life histories, modifying $\widehat{s}$ and r to define different strategies (e.g., large $\widehat{s}$ and small r corresponds to a high-longevity, slow pace-of-life strategy). Thus, for a given life history with fixed survival $\widehat{s}$, the corresponding fecundity is $r = \widehat{W}(1 - \widehat{s})/\widehat{s}$.

For a given $\widehat{s}$ and r, the maximum expected population growth rate per time step is $\widehat{\lambda} = \widehat{s}(1 + r)$. Because declining $\widehat{s}$ is associated with increasing r as described above, a life history with fixed $\widehat{W}$ and $\widehat{s}$ will have maximum growth rate $\widehat{\lambda} = \widehat{s} + \widehat{W}(1 - \widehat{s})$. The maximum growth rate has a monotonically decreasing relationship with $\widehat{s}$. As $\widehat{s}$ approaches zero (the case of semelparity with non-overlapping generations), $\widehat{W}$ approaches $\widehat{\lambda}$ (although when $\widehat{s}$ is exactly zero, $\widehat{\lambda} = 0$). As $\widehat{s}$ approaches one, $\widehat{\lambda}$ also approaches one. This agrees with observed patterns that populations of longer-lived organisms have slower population growth rates (Pimm et al. (1988), Oli (2004), Salguero-Gómez et al. (2016)).

Equilibrium dynamics

Age distribution and population growth. For this section, we assume the population is at stable age distribution and mutation-selection balance. Furthermore, we assume that the population has mean phenotype $\bar{z} = 0$, i.e., is adapted to its environment.

Upon birth, a cohort of newly-born individuals will have phenotypes that are normally distributed with variance γ_0^2. After k rounds of Gaussian selection under a constant environment, the phenotypic distribution will remain normally distributed with variance $\gamma_k^2 = \gamma_0^2/(1 + k\gamma_0^2)$ (see Appendix S1 for proof). As such, the expected probability that an individual at age k will survive to age $k + 1$ is

$$\bar{s}_k = \widehat{s}\sqrt{\frac{1}{1+\gamma_k^2}} = \widehat{s}\sqrt{\frac{1+k\gamma_0^2}{1+(k+1)\gamma_0^2}}. \tag{1}$$

This equation demonstrates that expected survival within the cohort increases with age (k), converging on $\widehat{s}$ at a rate proportional to γ_0^2. This is an example of the "frailty effect," wherein survival within a cohort increases over time due to the removal of less-fit individuals (Vaupel et al. (1979)). We demonstrate in Appendix S2 that defining the frailty of an individual with phenotype z_i as $y_i = z_i^2$ produces a gamma distribution of frailties within a cohort, in which case the analysis and predictions of Vaupel et al. (1979) will hold.

Assume that at equilibrium, the population has intrinsic growth rate λ^* such that $1 \leq \lambda^* \leq \widehat{\lambda}$. Then, the population has stable age distribution defined by p_k, where

$$p_k = \frac{r}{1+r}\left(\frac{\widehat{s}}{\lambda^*}\right)^k \frac{1}{\sqrt{1+k\gamma_0^2}}. \tag{2}$$

See Appendix S3 for proof. An alternative parameterization can be shown by noting that $p_0 = r/(1+r)$, in which case $\widehat{s} = (1-p_0)\widehat{\lambda}$ and the age distribution can be expressed as

$$p_k = p_0\left(\frac{1-p_0}{\lambda^*/\widehat{\lambda}}\right)^k \frac{1}{\sqrt{1+k\gamma_0^2}}. \tag{3}$$

Setting $\gamma_0^2 = 0$, in which case there is zero phenotypic variation and all individuals have survival $\widehat{s}$ (and thus $\lambda^* = \widehat{\lambda}$) produces a geometric distribution of ages in the population. Increasing γ_0^2 will increase the mass of older cohorts in the distribution due to the frailty effect (see Appendix Figure S1). Larger p_0, corresponding to smaller $\widehat{s}$, implies a relatively large newborn cohort and therefore a faster pace of life.

Phenotypic components and their variances. Because selection acts on each cohort independently, the phenotypic variance γ^2 is the age-weighted mean of each cohort variance and can be expressed as

$$\gamma^2 = \sum_{k=0}^{\infty} p_k \gamma_k^2 = \sum_{k=0}^{\infty} p_0 \left(\frac{1 - p_0}{\lambda^* / \widehat{\lambda}} \right)^k \frac{1}{\sqrt{1 + k\gamma_0^2}} \frac{\gamma_0^2}{1 + k\gamma_0^2}. \tag{4}$$

The equilibrium population growth rate can be written as $\lambda^* = \widehat{\lambda} / \sqrt{1 + \gamma^2}$ where γ^2 is defined as in equation 4. The divisor $\sqrt{1 + \gamma^2}$ is the population-wide "variance load" (Lande & Shannon (1996), Ashander et al. (2016)), which reduces the population growth rate by increasing the mean phenotypic distance of individuals from the environmental optimum.

Upon birth, individual breeding values are normally distributed with mean $\bar{b} = \bar{z}$, and variance $\gamma_{a,0}^2$ and the environmental components are normally distributed with mean zero and variance $\gamma_{e,0}^2$. In the newborn age class, breeding values and non-genetic components are uncorrelated such that $\gamma_{a,0}^2 + \gamma_{e,0}^2 = \gamma_0^2$. Individuals in age class k, having experienced k rounds of selection, will have phenotypic variance $\gamma_k^2 = \gamma_0^2 / (1 + k\gamma_0^2)$ as noted above. However, each round of selection produces negative correlations between breeding values and environmental components within the cohort. For example, the joint distribution of b and e after one round of selection is

$$
\begin{aligned}
p(b,e)W(b+e) &\propto \exp\left[-\frac{1}{2}\left(\frac{b^2}{\gamma_{a,0}^2}+(b+e)^2+\frac{e^2}{\gamma_{e,0}^2}\right)\right] \\
&= \exp\left[-\frac{1}{2}\left(\frac{b^2}{\frac{\gamma_{a,0}^2}{1+\gamma_{a,0}^2}}+2be+\frac{e^2}{\frac{\gamma_{e,0}^2}{1+\gamma_{e,0}^2}}\right)\right],
\end{aligned}
$$

which is a bivariate normal distribution with variances $\gamma_{a,1}^2 = \gamma_{a,0}^2(1+\gamma_{e,0}^2)/(1+\gamma_{a,0}^2+\gamma_{e,0}^2)$ and $\gamma_{e,1}^2 = \gamma_{e,0}^2(1+\gamma_{a,0}^2)/(1+\gamma_{a,0}^2+\gamma_{e,0}^2)$ and correlation $\rho_1 = -\sqrt{\gamma_{a,0}^2\gamma_{e,0}^2/((1+\gamma_{a,0}^2)(1+\gamma_{e,0}^2))}$. The correlation is induced by the Gaussian selection function's non-linearity with respect to the phenotype, z, which is the sum of components b and e. Correlation will be induced for any selection surface that is a non-linear function of the phenotype. The correlation is negative because the same optimum phenotype can be reached by multiple combinations of b and e, with an increase in one necessitating a decrease in the other to maintain the phenotype. In Appendix S4, we show via induction that, for age class k,

$$
\gamma_{a,k}^2 = \gamma_{a,0}^2\left(\frac{1+k\gamma_{e,0}^2}{1+k(\gamma_{a,0}^2+\gamma_{e,0}^2)}\right) \tag{5}
$$

$$
\gamma_{e,k}^2 = \gamma_{e,0}^2\left(\frac{1+k\gamma_{a,0}^2}{1+k(\gamma_{a,0}^2+\gamma_{e,0}^2)}\right) \tag{6}
$$

$$
\rho_k = -k\sqrt{\frac{\gamma_{a,0}^2\gamma_{e,0}^2}{(1+k\gamma_{a,0}^2)(1+k\gamma_{e,0}^2)}}. \tag{7}
$$

Although the phenotypic variance approaches zero as cohorts age, variances in breeding values and environmental components asymptotically approach non-zero quantities. As the phenotypic variance approaches zero, b and e approach perfect negative correlation (ρ_k approaches -1 as k grows large). Surprisingly, this demonstrates that older age classes may contain considerable genetic variation even as they might contain very little phenotypic variation. This correlation will not be present in a strictly semelparous, annual life history strategy as correlations induced by selection will not be transmitted to offspring because offspring do not, by definition, inherit environmental components of the parental phenotype.

Response to environmental change

We now consider an environmental change such that the mean population phenotype $\bar{z}$ is $\theta > 0$ units away from the environmental optimum. Say that the environmental shift occurs in time step 0 such that $t \geq 0$ is the number of time steps since the environmental change. As with other classic models of rescue (Gomulkiewicz & Holt (1995), Kopp & Matuszewski (2014)), the environment remains constant after the environmental shift.

Demographic effects of pace of life. The expected intrinsic population growth rate, $\mathbb{E}[\lambda]$, per time step can be decomposed into multiplicative effects as follows:

$$\mathbb{E}[\lambda] = \widehat{\lambda}\left(\exp -\frac{\bar{z}^2}{2}\right)\left(\frac{1}{\sqrt{1+\gamma^2}}\right). \tag{8}$$

The first term in the product, $\widehat{\lambda}$, is the maximum attainable growth rate in the population and the two subsequent terms (both bounded above by 1) represent maladaptation load (i.e., reduction in growth rate due to mean population phenotype diverging from the environmental optimum) and variance load (i.e., reduction in growth rate due to mean phenotypic distance from the optimum, Lande & Shannon (1996)), respectively. Setting $\bar{z} = 0$ produces $\lambda = \lambda^*$ as described in the previous section. The above decomposition demonstrates that the same phenotypic shift (i.e., setting $\bar{z} = \theta \neq 0$) will reduce population growth rates by the same multiplicative factor for all life history strategies. However, by holding $\widehat{W}$ and γ^2 constant across life history strategies and letting $\widehat{\lambda}$ vary, the same degree of environmental change (θ) will produce different intrinsic population growth rates among different life history strategies (Fig. 1a).

We can find the phenotype needed to produce stable population growth by setting the left hand side of Equation 8 equal to 1, substituting $\widehat{\lambda} = \widehat{W}(1-\widehat{s}) + \widehat{s}$, and solving for $\bar{z}$:

$$\widetilde{z} = \sqrt{2\log\left(\widehat{W}(1-\widehat{s}) + \widehat{s}\right) - \log\left(1+\gamma^2\right)}. \tag{9}$$

$\bar{z} < \widetilde{z}$ will produce population growth rates that are above replacement. For $\theta > \widetilde{z}$, in which case adaptation is necessary for persistence, larger $\widetilde{z}$ implies smaller $\theta - \widetilde{z}$, i.e., a smaller amount of phenotypic change required to recover positive population growth. Figure 1a and Equation 9 demonstrate that following a large environmental shift, slower life history paces (with larger $\widehat{s}$) will require greater amounts of phenotypic change to recover positive growth rates (i.e., have smaller $\widetilde{z}$.) This means that if the rate of phenotypic adaptation does not vary according to longevity (survival), then for the same degree of phenotypic variance, higher longevity will require more time to recover positive growth rates. Figure 1b demonstrates this by substituting in $\widehat{\lambda} = \widehat{W}(1 - \widehat{s}) + \widehat{s}$ evaluated at different values of $\widehat{s}$ for the maximum population growth rate in the original model of rescue by Gomulkiewicz & Holt (1995); this analytic expression assumes that genetic diversity and therefore rate of adaptation are constant over time (see Appendix S4 for more detail). Under the hypothesis that longevity slows down the rate of adaptation, population trajectories for higher-survival groups in Figure 1b would show even more rapid decline and slower recovery to original size than the lowest-survival group trajectory.

Trait evolution within cohorts through viability selection. Consider the cohort of individuals age k at the time of the environmental shift. Denote the mean phenotype, breeding value, and environmental phenotypic components in age class k at time step t as $\bar{z}_{k+t,t}, \bar{b}_{k+t,t}$ and $\bar{e}_{k+t,t}$, respectively. For all k at the time of the environmental shift $(t = 0)$, $\bar{z}_{k,0} = \bar{b}_{k,0} = \theta$ and $\bar{e}_{k,0} = 0$ in expectation.

After one round of viability selection, the mean phenotype of the cohort age k at the time of environmental change is $\bar{z}_{k+1,1} = \theta/(1 + \gamma_k^2) = \theta(1 + k\gamma_0^2)/(1 + (k+1)\gamma_0^2)$. After t rounds of selection, $\bar{z}_{k+t,t} = \theta(1 + k\gamma_0^2)/(1 + (k+t)\gamma_0^2)$, demonstrating that in expectation the mean phenotype for each cohort approaches zero (the optimum), but this rate is slower for older cohorts due to their reduced phenotypic variance at the time of the environmental change. In fact, for any given phenotypic value $0 < z' < \theta$, the expected time until the mean cohort phenotype is equal to z' scales linearly with age:

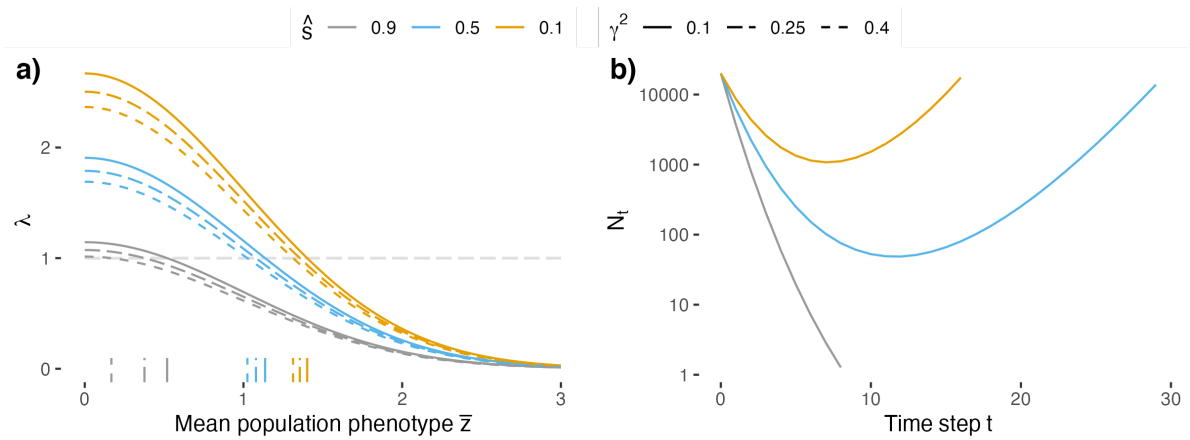

Figure 1: (a) λ as a function of mean phenotypic distance from the environmental optimum, $\bar{z}$, for different life history strategies, $\widehat{s}$, and population phenotypic variance, γ^2. Horizontal gray dashed line is $\lambda = 1$. Vertical ticks on the horizontal axis are $\widetilde{z}$, the value of $\bar{z}$ at which each curve intersects with the gray dashed line (see Equation 9). (b) Analytical solutions for population size over time for three life history strategies according to the model of Gomulkiewicz & Holt (1995), with $\theta = 2$, $N_0 = 20000$, and $h^2 = 0.5$ for each life history (see Appendix S4 for more detail).

$$t_z(k, z') = \frac{\theta - z'}{z'} \left(\frac{1}{\gamma_0^2} + k \right). \tag{10}$$

In Appendix S5, we demonstrate that the cohort of age k at the time of the environmental shift will have the following mean breeding value and environmental components after t rounds of selection:

$$\bar{b}_{k+t,t} = \theta \left(1 - \frac{t\gamma_{a,0}^2}{1 + (k+t)(\gamma_{a,0}^2 + \gamma_{e,0}^2)} \right) \tag{11}$$

$$\bar{e}_{k+t,t} = -\theta \left(\frac{t\gamma_{e,0}^2}{1 + (k+t)(\gamma_{a,0}^2 + \gamma_{e,0}^2)} \right). \tag{12}$$

Variances and correlation between the components are the same as defined in Equations 5-7. Equations 11 and 12 show that the mean cohort breeding value is never closer to the environmental optimum than the mean cohort phenotype, i.e., $\bar{z}_{k+t,t} \leq \bar{b}_{k+t,t}$,. Equality is only achieved for $t > k$ when $\gamma_{e,0}^2 = 0$, i.e., the trait is perfectly heritable. For $\gamma_{e,0}^2 > 0, \bar{e}_{k+t,t}$ falls below 0, becoming more negative as t increases.

For fixed $\gamma_{a,0}^2$, increasing $\gamma_{e,0}^2$ will increase the rate of change in the mean cohort phenotype (by increasing γ_0^2; see Equation 10) but Equation 11 demonstrates that it will also slow down the rate of genotypic change (producing a more positive $\bar{b}_{k+t,t}$, i.e., a mean breeding value further from the novel optimum.) Setting Equation 11 to zero and solving for t shows that the time until the mean breeding value of cohort k reaches a phenotypic value z' is

$$t_b(k, z') = \frac{\theta - z'}{z'} \left(\frac{1 + k(\gamma_{a,0}^2 + \gamma_{e,0}^2)}{\gamma_{a,0}^2 - \frac{\theta - z'}{z'}\gamma_{e,0}^2} \right). \tag{13}$$

Equations 10 and 13 show the following. First, $t_b(k, z') \geq t_z(k, z')$, with equality only if $\gamma_{e,0}^2 = 0$, i.e., if the trait is perfectly heritable. This means that it will take at least as long for the mean cohort breeding values to reach a specified phenotypic value than it will for the mean cohort phenotypes themselves. This follows from the fact, noted above, that within a cohort the mean phenotype is always as close or closer to the environmental optimum than the mean breeding

value is. Consequently, there could be a period for adult cohorts subject to strong selection where the mean cohort phenotype has reached $\widetilde{z}$ but the mean cohort breeding value has not; during this period, the cohorts will have an intrinsic replacement rate at or above 1 but will also contribute alleles to the gene pool with fitnesses that are below 1. Second, because $t_z(k, z')$ increases with k, older cohorts at the time of the environmental change will in expectation take longer to reach $\widetilde{z}$ through viability selection than younger cohorts; this is due to their reduced phenotypic variation after having experienced k rounds of selection before the environmental shift. From this, one may predict that longevity slows down the contributions of viability selection to phenotypic change in the population at large, as higher longevity implies an older-skewed age distribution and thus a greater proportion of individuals in cohorts with slow phenotypic change. Third, unlike $t_b(k, z')$, $t_z(k, z')$ is independent of the relative contributions of $\gamma^2_{a,0}$ and $\gamma^2_{e,0}$ to γ^2_0, i.e., the heritability of the trait does not affect the rate of cohort phenotypic change through viability selection. This is because response to selection in surviving individuals is through the phenotype, whereas the response to selection through differences in fertility or offspring viability is only through the heritable variation in the phenotype transmitted to offspring (Lande (1976)), i.e., the breeding value. Put another way, viability selection acts on z and therefore on both b and e, while fertility selection has only b able to respond to selection because parental e is not transmitted to offspring. From this final point, one may predict that, at least within the cohorts that are alive at the time of the environmental change, longevity will *increase* rates of phenotypic change in the population by increasing the rate of viability selection in the population relative to fertility selection, with stronger effects for less heritable traits.

The analysis thus far has focused on the dynamics of cohorts alive at the time of the environmental shift (for whom the expected phenotype at the time of the environmental shift is θ), but these results also hold for cohorts born after the environmental shift if θ is replaced by $\bar{z}_{0,t}$ for $t > 0$. However, because we assume that mating is random and does not depend on parental age, mean phenotypes of each new-born cohort depend on the distribution of breeding values within the population as a whole. We are unable to make analytical progress in deriving tractable

expressions for the mean breeding value in the population at large following the environmental change, even if we make the assumption that the population remains at stable age distribution following the environmental change. In any case, this assumption is very unlikely to be met, as the reduced phenotypic variance in older cohorts means that fewer individuals in older cohorts will be close to the novel environmental optimum, in which case mortality will be higher in older cohorts. Deviation from the stable age distribution influences not only mean breeding values in successive cohorts, but also variance in breeding values, i.e., $\gamma^2_{a,0}$. This means that the predictions made in this section will hold at the level of individual cohorts but might not hold at the level of the population. We thus conclude this section and turn to simulation methods to assess effects of longevity on growth and adaptation at the population level.

Simulation model

We implement a stochastic, individual based simulation model following the dynamics described above in R, version 4.1.2 (R Core Team (2020)). Populations are initialized by drawing a sample from the stable age distribution at the specified initial population size, N_0. Then, for each individual, breeding values and environmental components are drawn according to a bivariate normal distribution with mean θ for breeding values, mean 0 for environmental components, and variances defined by the individual's age (see Equations 5-7). Individuals are also randomly assigned a sex, male or female with equal probability, upon initialization.

Each time step begins with selection acting through survival, followed by reproduction, and is concluded by a population census. Thus, the census population in time step t is the number of individuals recorded after reproduction in time step t. Survival of each adult is determined by a Bernoulli draw, where for individual i the probability of survival was $s_i \exp\left(-\alpha N_{t-1}\right)$, where N_{t-1} is the population size in the previous time step (according to the census taken at the end of time step $t-1$, immediately preceding selection on survival). The Ricker term causes negative density dependence (Ricker (1954)), which is important for accurately assessing population growth and extinction risk under intraspecific competition (Scholl et al. (2022), Nordstrom et al.

(2023)) and also reduces computational load of simulations. Because the Ricker term is applied to survival (i.e., all individuals are subject to identical strength of density dependence in each time step), and reproduction is density independent, density dependence does not perturb populations from their stable age distribution. We assume this form of density dependence because other forms of density dependence that perturb populations from their stable age distribution (e.g., density dependence limiting only recruitment of offspring) obscure our ability to identify effects of longevity, as differences between life histories may be due to density dependence rather than longevity *per se*. To simulate reproduction, each time step every surviving female is paired with one randomly-selected surviving male and produces a number of offspring determined by a draw from a Poisson distribution with mean $2r$. Extinction occurs if the population has no females or no males. For each offspring, the breeding value is determined by drawing a normally distributed random variable with mean equal to the individual's mid-parent value and variance equal to $\tilde{\gamma}_a^2/2$, where $\tilde{\gamma}_a^2$ is the remaining additive genetic variance in the population after selection on surviving adults (Lynch & Walsh (1998)). A mutation is added to each offspring's breeding value using a normal distribution with mean 0 and variance chosen to produce mutation-selection balance at equilibrium (see Appendix S6 for expression and derivation). Phenotypes are then produced by adding an environmental component drawn from a normal distribution with mean 0 and variance $\gamma_{e,0}^2$ to each individual's breeding value.

Simulation experiment

We analyze the effects of longevity on population persistence under environmental change as follows. Our simulation design is a three-way factorial experiment with variables longevity ($\widehat{s}$), phenotypic diversity (γ^2), and heritability of the trait (h^2). We set $\widehat{W} = 3$ for all life histories. For a fixed $\widehat{W}$, longevity is determined by the balance of the variables r and $\widehat{s}$. We designate three life-history treatments: high longevity ($\widehat{s} = 0.9$), medium longevity ($\widehat{s} = 0.5$), and low longevity ($\widehat{s} = 0.1$), with r at appropriate levels. Because the phenotypic variance in the population is important for determining rates of phenotypic change (Lande (1976), Houle (1992)), we include

treatments for high ($\gamma^2 = 0.4$), medium ($\gamma^2 = 0.25$), and low ($\gamma^2 = 0.1$) phenotypic diversity. Finally, we include heritability of the trait, as the analysis above demonstrates that non-heritable phenotypic variance (γ_e^2) decouples mean genotypes and phenotypes within a cohort as individuals go through multiple rounds of selection. We define trait heritability as the degree of trait variance in each new-born cohort that is inherited from parents, i.e., $h^2 = \gamma_{a,0}^2/\gamma_0^2$. We thus determine the initial degree of additive genetic variance and environmental component variance in each life-history treatment by fixing h^2 and setting $\gamma_{a,0}^2 = h^2\gamma_0^2$ and $\gamma_{e,0}^2 = (1-h^2)\gamma_0^2$. We use heritability values of $h^2 = 0.25$, $h^2 = 0.5$, and $h^2 = 1$. The first two values are realistic for life-history traits in natural populations (Wood et al. (2016)) and the final value, for which $\gamma_e^2 = 0$, serves as a "control" for examining the decoupling of genotypes and phenotypes. Density dependence was set *via* the Ricker α parameter to produce identical carrying capacities (20000) across treatments. Table 1 gives $\widehat{\lambda}$ and λ^* for each life-history treatment.

Analysis of simulations

For each life-history treatment, we ran 1000 simulations of up to 100 time steps. Preliminary analysis suggested that standard errors were sufficiently small to precisely estimate mean population size over time with this number of simulations. Within each treatment, for each time step, we estimated mean population size, $\bar{N}_t$. These means included extinct populations as size zero, thus incorporating extinctions into the expectations. To assess probabilities and rates of extinction, we also recorded the number of surviving populations within each time step for each treatment.

We analyzed rates of adaptation and changes to the age distribution within the population by running a separate, smaller batch of 200 simulations lasting up to 50 time steps for all three levels of longevity and heritability but only the highest level of phenotypic variance ($\gamma^2 = 0.4$). Because phenotypic components and age distributions are undefined for extinct populations, extinctions make unbiased estimation of rates of change in phenotypic components and age distributions impossible. As such, we averaged state variables in all time steps until the first

$\widehat{s}$	γ^2	r	$\widehat{\lambda}$	λ^*
0.1	0.40	27	2.8	2.67
0.1	0.25	27	2.8	2.56
0.1	0.10	27	2.8	2.37
0.5	0.40	3	1.8	1.91
0.5	0.25	3	1.8	1.83
0.5	0.10	3	1.8	1.68
0.9	0.40	0.33	1.2	1.14
0.9	0.25	0.33	1.2	1.10
0.9	0.10	0.33	1.2	1.01

Table 1: Longevity ($\widehat{s}$) and phenotypic variance (γ^2) treatments in simulation experiments, and their corresponding fecundity per mating bout (r), maximum population growth rate $\widehat{\lambda}$, and equilibrium density-independent population growth rate (λ^*) for each treatment. Heritability treatments are not included in this table as they do not directly influence equilibrium population growth rates.

extinction was recorded; we ran a smaller number of simulations per treatment to reduce the occurrences of early extinctions and thus maximize the number of time steps in our analysis. Likewise, we ran simulations only for the highest level of phenotypic variance, as that level had the fewest extinctions in preliminary analysis. In these simulations, for each age class in each time step we estimated the mean size (number of individuals), the mean value and mean variance for each phenotypic component, and the mean correlation between breeding value and environmental phenotypic components. Results were qualitatively similar for lower phenotypic variance treatments.

Results

Population size, growth, and extinction

In all time steps until reaching carrying capacity, mean population size was largest for low-longevity strategies and smallest for high-longevity strategies (Fig. 2). Low-longevity treatments were the fastest to return to their original size; low equilibrium growth rates (λ^*) for the high-longevity treatments considerably slowed the rate of population growth as populations of longer-lived organisms took considerably more time to return to their original size. Population sizes and growth rates increased with heritability and typically increased with increasing phenotypic variance; the one exception was high-longevity strategies, for which mean population size was highest at intermediate phenotypic variance rather than high phenotypic variance. This is explained by low phenotypic variance increasing extinction rates for all treatments (as demonstrated below), while high phenotypic variance decreases λ^* through increased variance load. For the high-longevity strategies, low $\widehat{\lambda}$ constrained λ^* to be very close to 1 such that population growth was constrained even though high phenotypic variance allowed for faster phenotypic change.

Among longevity treatments with the same heritability and phenotypic variance, lower longevity was associated with earlier onset of extinctions (Fig. 3). However, because these lower-longevity populations were able to recover positive growth rates quickly (Fig. 2), they were only at risk of extinction for a short period of time (Fig. 3). In contrast, because high-longevity populations took longer to recover population growth rates and spent more time at low density (Fig. 2), they were at risk of extinction for a longer period of time (Fig. 3). The cumulative result was that high-longevity treatments had higher rates of extinction after 100 time steps, although extinctions typically occurred well before this time. In treatments where extinctions were common, medium-longevity strategies had higher rates of extinction than low-longevity strategies. However, in treatments where extinctions were uncommon (less than 25%, e.g., high heritability and low variance or low heritability and high variance), extinction was slightly more frequent for

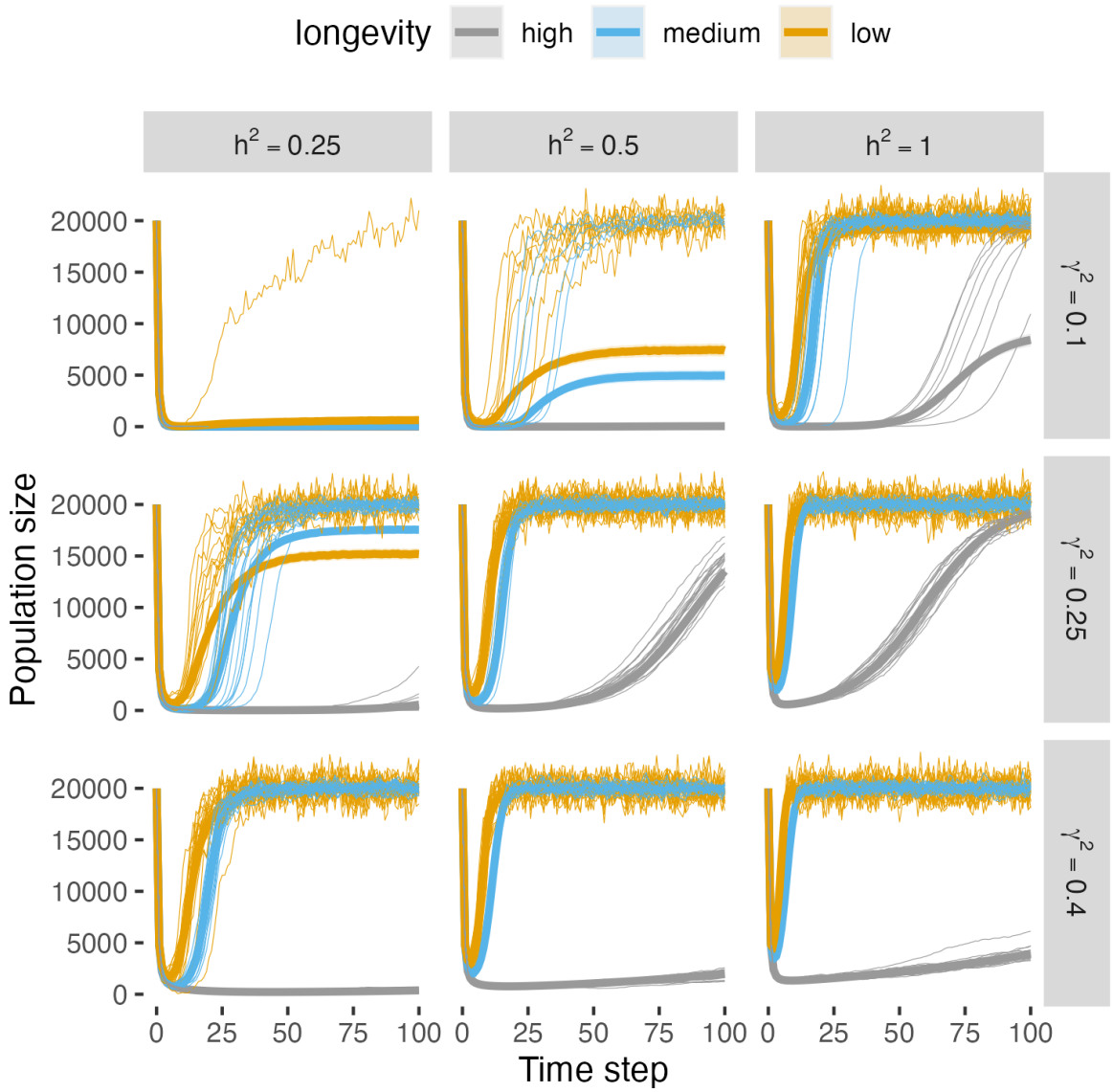

Figure 2: Mean population size over time for the 27 life-history treatments. Thick lines are the mean population size of 1000 trials per treatment, including extinct populations as size zero. Shaded regions are twice the standard error on each side of the mean (errors are mostly too small to be visible on this plot). Twenty randomly-selected realizations of population size over time per treatment are plotted with thin lines.

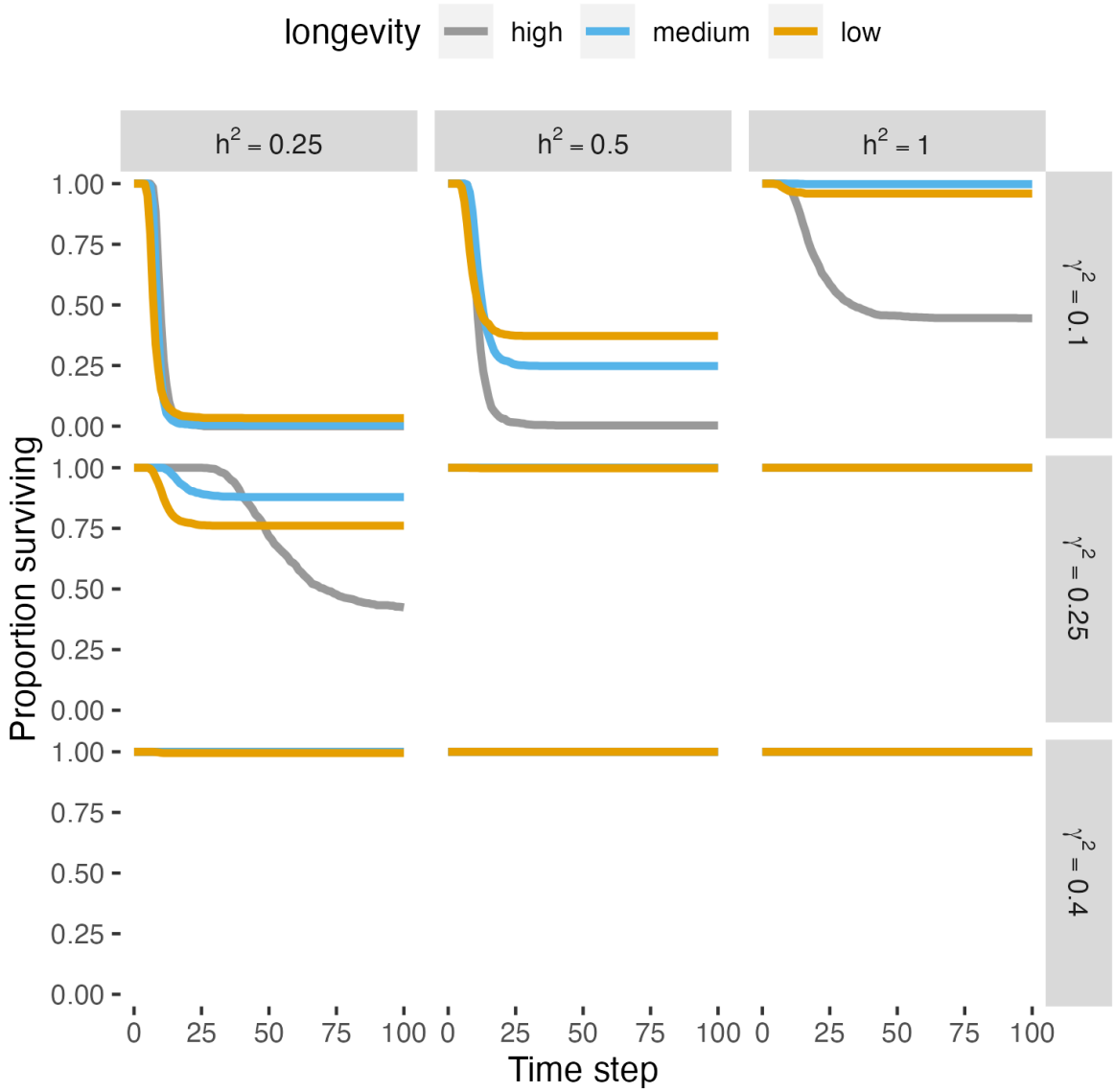

Figure 3: Proportion of surviving populations (out of 1000 per treatment) over 100 time steps.

low longevity than medium longevity. This is due to the higher variation in population growth rates among low-longevity populations (see Fig. 2, thin lines for individual populations); a consequence of this higher variation was that populations in the lower tail of the distribution of growth rates for low-longevity populations were particularly vulnerable to extinction. Extinction risk increased as phenotypic variance or heritability decreased, as expected (Gomulkiewicz & Holt (1995)).

Rates of phenotypic and genotypic change

For imperfect heritabilities (i.e., $h^2 < 1$) and high and medium longevity, we observed the predicted decoupling of mean genotype and mean phenotype at the population-level (Fig. 4), although it only occurred transiently. During this transient period, mean phenotypes rapidly approached, but did not reach, the environmental optimum. This period can best be marked by two distinct phases in the dynamics of $\bar{e}$, the environmental component of the phenotype: a rapid (5-10 time step) period of increase in magnitude of $\bar{e}$ (negative vertical direction in Fig. 4), followed by a longer period of relaxation as $\bar{e}$ returned to zero. The durations of both phases increased with increasing longevity and decreasing heritability. During the first phase, there was a rapid rate of phenotypic change as populations approached, but did not necessarily reach, the environmental optimum. In high-longevity populations with larger changes in $\bar{e}$, the approach of $\bar{e}$ to zero caused the rate of phenotypic change to slow, eventually falling behind rates of phenotypic change in lower-longevity populations. Thus, although there was a short phase of rapid phenotypic change, on longer timescales populations with higher longevity had a slower approach to the novel phenotypic optimum. During both of these phases, rates of genotypic change lagged behind rates of phenotypic change. Importantly, this decoupling of phenotype and genotype disappeared under perfect heritability, under which there was minimal difference in rates of phenotypic change among longevity treatments. One low longevity population with heritability 0.25 went extinct in time step 13; to avoid survivor bias, analysis of phenotypic components at this heritability level only includes time steps zero through 13. Differences in rates of

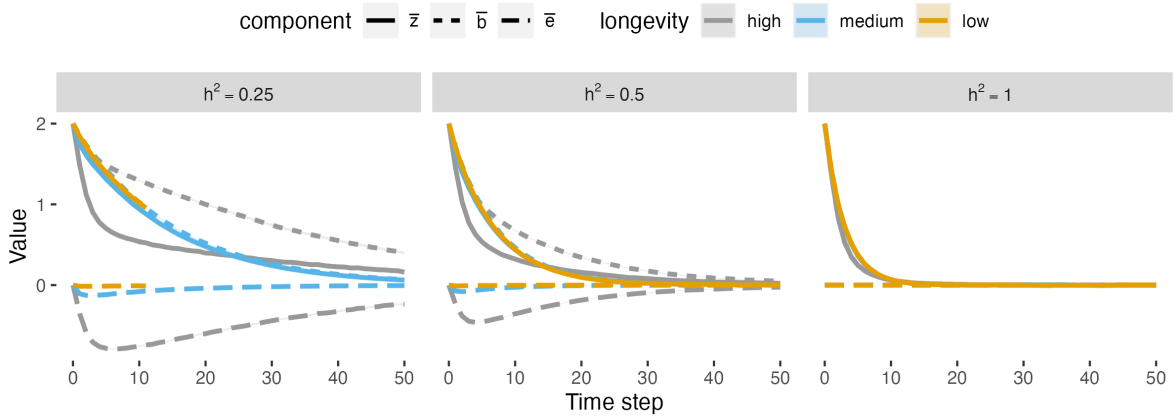

Figure 4: Mean phenotypic components ($\bar{z}$, $\bar{b}$, $\bar{e}$) for each longevity treatment over time for each of three levels of heritability. For all time steps, the phenotypic optimum is zero. Component means for the low-longevity in the left panel are only estimated for time steps when all populations are extant. The component means of the medium-longevity group in the right panel are obscured by the low-longevity means.

phenotypic and genotypic change between medium and low longevity treatments were slight.

Age- and age-variance structure

Environmental change perturbed high-longevity populations from the stable age distribution by disproportionately removing older individuals from the population, reducing the mean population age (Fig. 5). These populations returned to their pre-change steady state within 50 time steps; return to this steady state was faster with higher heritability. At low heritability, after the initial reduction in size for older age classes, the size of the older age classes "overshot" the stable age distribution such that they temporarily comprised a larger proportion of the age distribution compared to the steady state distribution. Age structure in medium- and low-longevity populations was minimally changed following the environmental change, due to these life histories having more mass in the age-zero class (Appendix S3, Fig. S1).

This disruption to the age structure in high-longevity strategies coincided with an increase

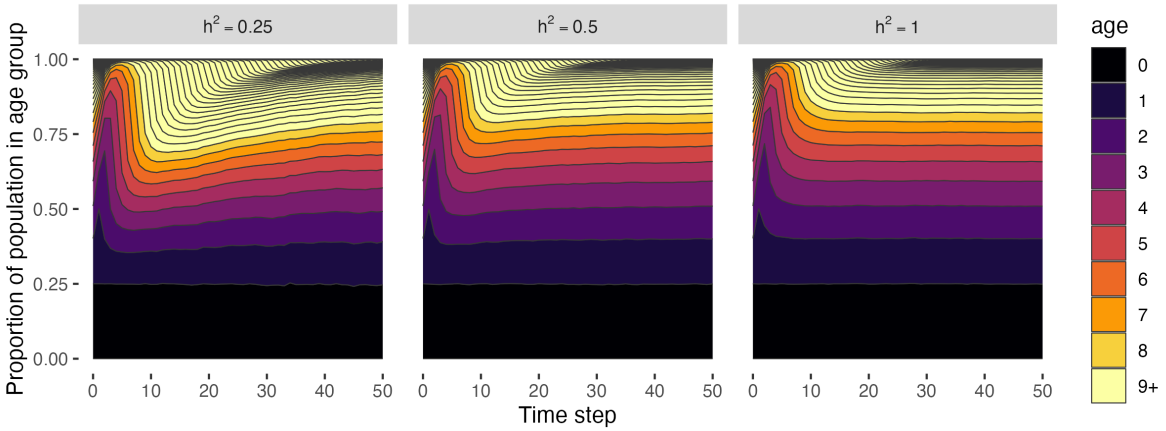

Figure 5: Mean age distribution over time for high-longevity populations at different heritability levels. Classes for ages nine and older are combined into one color code for visual clarity.

in phenotypic variance (γ^2) in the population at large, followed by a gradual return to equilibrium levels (Fig. 6). Specifically, phenotypic variance increased because the environmental change shifted the population's age structure towards younger age classes (Fig. 5) with higher phenotypic variance. Medium-longevity strategies also experienced an increase in phenotypic variance, but the increase was much smaller in magnitude and shorter in duration. At perfect heritability, phenotypic variance (and therefore additive genetic variance, γ_a^2) increased but then very quickly returned to equilibrium values, demonstrating that environmental change and disrupted age structure affects additive genetic variance in the population at large regardless of heritability. With lower heritability, the increase in phenotypic variance became slightly larger in magnitude and took longer to subside to equilibrium values; this coincided with a large increase in environmental phenotypic variance (γ_e^2), and the increase in additive genetic variance became considerably smaller. These results suggest that there was a transient disruption to population age structure, producing a transient increase in phenotypic variance available for selection to act on, but that with lower heritability most of this available phenotypic variance was in environmental components of the phenotype and thus response to selection was lost when producing new offspring.

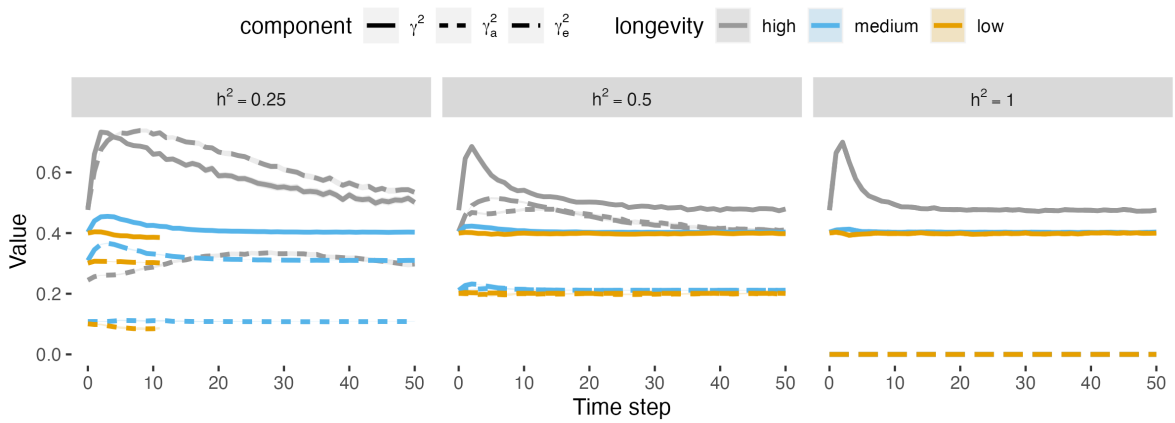

Figure 6: Phenotypic component variances over time. Component variances for the low-longevity strategies in the left panel are only estimated for time steps when all populations are extant.

Discussion

We demonstrate that longevity, defined here as increased survival and associated reduced fecundity per reproductive bout, does impede evolutionary rescue following a single environmental shift as is often posited, but not necessarily due to slowing rates of phenotypic change. In our model where viability selection acted on survival, all members of a population experienced selection in each time step; among populations with the same phenotypic variation but differing life history paces, we found that longevity can be associated with a transient increase in the rate of phenotypic adaptation, followed by a slowing rate of approach to the novel phenotypic optimum compared to populations with lower longevity. Crucially, this phenomenon relied not only on high longevity but also low heritability, as repeated selection on individual survival produced different responses to selection between surviving adults and their offspring. When the trait under selection was perfectly heritable, there was only a slight difference in the rate of phenotypic change among life-history strategies, suggesting that previous findings supporting the slowing-adaptation hypothesis (Kuparinen et al. (2010), Cotto et al. (2017), Schmid et al. (2022)) do not hold in all cases. We find that the primary mechanism through which longevity hinders evolu-

tionary rescue is demographic rather than evolutionary, as longevity slows population growth (Oli (2004), Gaillard et al. (2005), Gamelon et al. (2014), Salguero-Gómez et al. (2016)).

Our results show that longevity and its associated slow intrinsic growth rates are disadvantageous for populations adapting to sudden and challenging environmental changes. Constrained intrinsic population growth rates often correlate with longevity, as trade-offs between survival or growth and reproduction preclude populations' abilities to grow rapidly (Pianka (1970), Pimm et al. (1988)). While longevity often has the advantage of slowing expected rates of decline, in our simulations the mean population size for high-longevity strategies was much smaller throughout time than for lower-longevity strategies. On a short timescale, high survival associated with longevity can buffer populations of long-lived organisms that reach low densities, delaying extinction or slowing further declines (Pimm et al. (1988)). However, on longer timescales (such as those required for adaptation to very novel conditions), low recruitment per reproductive bout associated with longer-lived species will slow the time it takes for these populations to escape from low densities (Pimm et al. (1988)) where populations are susceptible to genetic or demographic stochasticity. Although not explicitly incorporated into our model, this increased time spent at low density can have long-term genetic consequences by forcing populations through a bottleneck (Whitlock (2000)), possibly slowing rates of phenotypic change (Frankham et al. (1999)) and making populations vulnerable to entering an extinction vortex (Gilpin & Soulé (1986), Nordstrom et al. (2023)). Although prior rescue literature has predicted longevity would adversely affect rescue through reduced rates of adaptation, it does not appear to address this important demographic consequence of slower paces-of-life. There are likely other results from life history and demographic theory that could be incorporated into the theory of evolutionary rescue to predict the fates of conservation targets in changing environments.

One unexpected consequence of the lowered intrinsic growth rates is that longevity implies a smaller range of environmental shifts or perturbations (i.e., values of θ) that a population is able to withstand without incurring population decline. This is best illustrated by Fig. 1a: higher-longevity populations have a narrower range of population-mean phenotypic values $\bar{z}$ for which

$\lambda \geq 1$. We defined the critical value of mean population phenotype, $\widetilde{z}$, required to produce stable population densities; an environmental change of magnitude $\theta > \widetilde{z}$ will necessitate rescue to guarantee persistence. Because $\widetilde{z}$ decreases with increasing $\widehat{s}$ and decreasing r (see Equation 9), populations with slower life histories will require evolutionary rescue for a wider range of environmental changes. The pattern in Fig. 1a resembles that reported by Oli (2004), who placed wild mammal populations on the fast-slow pace-of-life continuum and found that "fast" pace-of-life populations typically had mean growth rates well in excess of 1 while populations on the slow end had mean growth rates much closer to 1. We scaled phenotypes and the degree of environmental change, θ, by the selection strength, ω (see Methods), but on the scale of the environmental driver that determines fitness, it is possible that species on the "slow" end of the spectrum face weaker selection for the same magnitude of change in the environmental driver. Expressed another way, it is possible ω (the width of the selection surface, inversely proportional to strength of selection) may increase with longevity, in which case curves in Fig. 1a would decline more slowly, allowing longer-lived populations to withstand greater shifts in the environment while maintaining positive intrinsic growth rates. That higher longevity could be associated with a narrower range of tolerable disturbances is a surprising finding given that longevity and high baseline survival provide "buffering" against increasing environmental variation or magnitudes of fluctuations (Morris et al. (2008), Dalgleish et al. (2010)), including buffered response to single environmental and climatic anomalies (Compagnoni et al. (2021), Jackson et al. (2022)). However, our result is possibly distinct in that it deals with single, sudden, and large change in the mean condition of an environment rather than fluctuations or background variance.

We also find that longevity does not necessarily slow down rates of phenotypic change as predicted (e.g., Chapin et al. (1993), Vander Wal et al. (2013), Shoemaker & Lennon (2018)). Vander Wal et al. (2013) hypothesized that rescue occurs slowly in mammals, reasoning that the generations until rescue found in studies with model organisms in laboratory settings (e.g., algae, Bell & Gonzalez (2009)) multiplied by longer generation times of mammals produced longer times until rescue. Our analysis demonstrates that this reasoning ignores phenotypic

change that may happen within a generation. In our model where individuals face selection in every time step, we found that for perfectly heritable traits, longevity had near-negligible effects on rates of adaptation, and at lower trait heritabilities the rate of phenotypic change was initially faster with increased longevity (Fig. 4). In fact, phenotypic change occurred more rapidly in longer-lived populations at all heritability levels when estimated on a per-generation basis rather than a per-time step basis (see Supporting Information S9 and Figure S19). Prior models found that longevity slowed phenotypic change through combinations of reduced generational turnover (Kuparinen et al. (2010), Cotto et al. (2017)) and life stages shielded from selection (Schmid et al. (2022)), but likewise assumed that selection acted only once in an individual's lifetime. Removing this assumption and allowing individuals to face multiple rounds of selection clarifies how these two mechanisms relate to each other: reduced generational turnover slows rates of adaptation if maladapted individuals in the longer-lived stages (or oldest individuals) are not removed, but we demonstrate this effect can be negated if these individuals face selection. Draghi et al. (2024) also found that changes in generation time that occur as populations are adapting to novel conditions coincided with differing rates of phenotypic change, with changes that increased generation time slowing adaptation. This consistent with our results: we observed disruptions to the stable age distribution primarily through increased mortality among older individuals, reducing the mean age in the population (Fig. 5), and increasing the degree of phenotypic variation in the population (Fig. 6). This was associated with more rapid phenotypic change, evident even in the case of perfect heritability, where adaptation proceeds slightly more quickly for high-longevity strategies than low-longevity ones (compare solid gray and orange lines, Fig. 4, right panel). Synthesizing these results requires distinguishing between phenotypic change that can occur between generations with those that can occur within generations; the latter dynamic is missing from the majority of the rescue literature.

Our most interesting results emerged when looking at lower trait heritabilities that more resemble those found in natural populations (Wood et al. (2016)). In our simulation results, the combination of low heritability and high longevity was associated with a transient increase in

the rate of phenotypic change but a decrease in the rate of genotypic change (Fig. 4). We demonstrate analytically that, at the cohort level, repeated episodes of directional selection will decouple mean cohort phenotype and genotype, as non-heritable variance in the trait is exposed to selection (Equation 11). In age-structured populations, an individual potentially provides multiple contributions to changes in the population's trait distribution over time, including a contribution through offspring ("fertility selection") and a contribution through its own survival ("viability selection", Coulson & Tuljapurkar (2008)). Under viability selection, the whole of the phenotype will respond to selection, while the response to fertility selection will only be through the heritable portion of the phenotype (Lande (1976)), i.e., the breeding value (Falconer & Mackay (1996)). Increasing longevity increases the weight of the contributions of viability selection relative to the contributions of fertility selection, possibly increasing the rate of phenotypic change in the population. This effect is absent from prior models of rescue and environmental tracking with non-overlapping generations and semelparity, in which case the entirety of the contribution to the next generation is through fertility selection, which will have a smaller response in magnitude when heritability is low (Lande (1976)). However, this increasing rate of phenotypic change associated with decoupling is transient and comes at a cost of slowing the rate of genotypic change (Fig. 4). Directional selection for, say, a large trait value will select for large z by simultaneously selecting for large b and large e. Equations 11 and 12 and Figure 4 demonstrate that also allowing selection on the non-heritable phenotypic component slows down genotypic change, as the selection for large z will maintain individuals that might not have large b but do have large e; these genetically-maladapted individuals will remain in the gene pool and contribute their alleles to successive cohorts. This decoupling of mean phenotypes and genotypes might explain the result of Coulson & Tuljapurkar (2008) that birth weights of red deer offspring consistently were smaller than the birth weights of their mothers. They found that viability selection produced a pattern of increased birth weight as cohorts aged, but also that the birth weights of the offspring were often lower than the birth weights of mothers. We observed a similar phenomenon, where repeated selection on survival produced adults with phenotypes closer to a novel environmental

optimum, but because this viability selection acted on non-heritable phenotypic variance, the breeding values (and thus offspring phenotypes) of these surviving parents were further from the phenotypic optimum. However, this phenomenon was transient in our simulations; while we were not able to mathematically characterize this transient process, it is similar qualitatively and in effect to transient buffering due to phenotypic plasticity (Lande (2009), Chevin & Lande (2010)), wherein there is a rapid phase of accelerated phenotypic change due to plastic effects (providing key buffering during phases of population decline, Chevin & Lande (2010)) and a longer period as the phenotypic change is "assimilated" into change in the underlying genes.

This repeated selection on survival-related traits provides an evolutionary mechanism for a "frailty effect" of Vaupel et al. (1979), wherein mean survival or fitness within a cohort increases as it ages. Increasing survival with age is a common (although not universal, Jones et al. (2014)) life history pattern; this mechanism through repeated selection can occur without plasticity or change in underlying traits or ontogeny. This is relevant to perennial organisms in particular for which selection on birth or body weight (or proxies thereof) influences survival probabilities (Milner et al. (1999), Ozgul et al. (2010)). Frailty effects can also produce "cohort selection" (Kendall et al. (2011)), which increases population growth rates relative to populations with homogeneity in survival. The demographic consequences of this cohort selection merit future research in the context of evolutionary rescue; it is possible that cohort selection and the associated reduction of phenotypic variance among older individuals had a negative effect on rescue in our simulations, as the reduced phenotypic variance combined with a sudden change in the environmental optimum produced disproportionate mortality (Fig. 5). Regardless, if cohort selection buffered long-lived populations in our simulations, it had small effects in comparison to other factors that increased extinction risk for high-longevity populations.

From these results we can predict the following. For a single, drastic, and sustained change in the optimal phenotype favored by the environment, longer-lived populations will be the most likely to go extinct, although these extinctions will occur on longer time horizons than extinctions in shorter-lived populations. The first species to go extinct are most likely to be short-lived;

this is due to increased stochasticity in population growth rates for fast life histories. The higher variance in population growth trajectories could create a heavy "tail" of especially-unlucky or poor populations which face extinction risk even if the "typical" population is unlikely to go extinct. This is consistent with recent work in plants (Compagnoni et al. (2021)) and mammals (Jackson et al. (2022)) showing stronger responses to climatic anomalies in shorter-lived species. However, as time since the environmental change increases, our model predicts that extinctions among surviving short-lived species should cease as populations sufficiently adapt and return to a large size, while extinctions will continue for longer-lived species. These longer-lived species will adapt, but their constrained population growth rates will keep them at smaller densities for longer, leaving them vulnerable to extinction. These predictions are similar to those of Pimm et al. (1988), who suggested that large body size (which typically correlates with high survival and longevity) can buffer populations at small density and forestall extinction on short timescales, but will increase extinction risk on longer timescales by constraining growth and keeping populations at smaller densities where they will be more susceptible to stochasticity or catastrophe. The net effect, particularly for populations with a high carrying capacity, is that long-lived populations are less likely to be successfully rescued than short-lived ones. We can also generate a prediction about rates of adaptation: namely, that long-lived adults subject to multiple rounds of directional selection under the novel environment will produce offspring that are, on average, less adapted to the environment than they are. Indeed, one would expect that with increasing age of parents, the phenotypic difference between parents and offspring will grow larger. Coulson & Tuljapurkar (2008) found a relationship where the phenotypic difference between red deer parents and their offspring was smallest at intermediate ages. That the phenotypic difference grew with advanced parental age is consistent with our prediction, although the large difference at young ages suggests that at young ages other important factors also influence the phenotypic-genotypic relationships in between parents and offspring.

Of course, to fully integrate the theories of life history and evolutionary rescue will require a more complicated model and analysis than we present here. Our model assumes single, age-

invariant parameters for survival and growth, but life histories often have varying investment in growth, survival, and reproduction over the course of the life cycle (Gadgil & Bossert (1970), Jones et al. (2014)). We intentionally modeled populations with simple demography, isolating effects of longevity while removing other life-history features (such as life stages buffered from selection) that are not necessary for longevity, even if they sometimes appear in the life histories of long-lived organisms. Our model and results can serve as a null model for comparison if comparing influences of other features that may coincide with longevity, e.g., senescence. We suspect it is also possible to extend the model to include varying reproduction and survival over the course of the lifetime without extreme complexity by, e.g., modeling survival using the discrete Weibull distribution, adding only one additional model parameter (Nakagawa & Osaki (1975)). We also assumed a simple form of density dependence, acting on survival independently of age or trait value. We chose this because it did not perturb populations from the stable age distribution. However, the life-cycle stage or vital rate influenced by density dependence can influence evolutionary rescue and rates of adaptation. For example, density dependence acting primarily on reproduction rather than survival can increase generation times, slowing rates of adaptation (Draghi et al. (2024)). The model of Cotto et al. (2017) assumed density dependence acted only on recruitment. This, combined with selection acting only on recruitment rates, likely contributed to their result of longer-lived species having slower adaptation, as maladapted adults were not subject to competition. Finally, future analysis must also generalize to other regimes of environmental change. As with other classic models of evolutionary rescue (Gomulkiewicz & Holt (1995), Chevin & Lande (2010), Kopp & Matuszewski (2014)), we modeled a single environmental shift, but with constant conditions after the shift has occurred. However, longevity and high survival are advantageous in variable environments (Orzack & Tuljapurkar (1989), Morris et al. (2008)). We speculate that our results under a constant environment will also hold for sudden environmental shifts in noisy environments, so long as the magnitude of the sudden shift is large in comparison to the magnitude of the fluctuations. Increasing the magnitude of the fluctuations relative to the magnitude of the sudden change should produce increasingly favorable results in

longer-lived populations. In the case of gradually-changing environments, relative rates of directional trend and variance around the trend are also studied in the context of evolutionary rescue and persistence under changing environments (Bürger & Lynch (1995)), although these models also typically assume non-overlapping generations. Following life-history theory (Orzack & Tuljapurkar (1989), Morris et al. (2008), Schmid et al. (2022)), we similarly expect that increasing the magnitude of fluctuations relative to the degree of environmental shifts will generally produce outcomes more favorable to higher-longevity populations, although these effects will depend on the heritability of the trait (Lyberger et al. (2021)).

Data archiving statement

We have attached an anonymized version of code to reproduce our simulated experiment and analysis, as well as code to validate analytical expressions and raw data from simulation output, with our manuscript submission. A non-anonymized version of our repository was previously uploaded publicly to GitHub but is currently set to private. We pledge to upload all code and output data to Dryad upon acceptance of the manuscript.

Literature Cited

Agashe, D.; Falk, J. J. & Bolnick, D. I. 2011: Effects of founding genetic variation on adaptation to a novel resource. Evolution 65(9):2481–2491.

Ashander, J.; Chevin, L.-M. & Baskett, M. L. 2016: Predicting evolutionary rescue via evolving plasticity in stochastic environments. Proceedings of the Royal Society of London, Series B: Biological Sciences 283:20161690.

Barfield, M. & Holt, R. D. 2016: Evolutionary rescue in novel environments: towards improving predictability. Evolutionary Ecology Research 17:771–786.

Bell, G. 2012: Evolutionary rescue of a green alga kept in the dark. Biology Letters 9:20120823.

Bell, G. & Gonzalez, A. 2009: Evolutionary rescue can prevent extinction following environmental change. Ecology Letters 12(9):942–948.

Bürger, R. & Lynch, M. 1995: Evolution and extinction in a changing environment: a quantitative-genetic analysis. Evolution 49(1):151–163.

Cardillo, M. 2003: Biological determinants of extinction: why are smaller species less vulnerable? Animal Conservation 6(1):63–69.

Carlson, S. M.; Cunningham, C. J. & Westley, P. A. 2014: Evolutionary rescue in a changing world. Trends in Ecology & Evolution 29(9):521–530.

Chapin, F. S.; Autumn, K. & Pugnaire, F. 1993: Evolution of suites of traits in response to environmental stress. American Naturalist 142:78–92.

Chevin, L.-M. & Lande, R. 2010: When do adaptive plasticity and genetic evolution prevent extinction of a density-regulated population? Evolution 64(4):1143–1150.

Cole, L. C. 1954: The population consequences of life history phenomena. Quarterly Review of Biology 29(2):103–137.

Compagnoni, A.; Levin, S.; Childs, D. Z.; Harpole, S.; Paniw, M.; Römer, G.; Burns, J. H.; Che-Castaldo, J.; Rüger, N.; Kunstler, G.; Bennett, J. M.; Archer, C. R.; Jones, O. R.; Salguero-Gómez, R. & Knight, T. M. 2021: Herbaceous perennial plants with short generation time have stronger responses to climate anomalies than those with longer generation time. Nature Communications 12(1):1824.

Cotto, O.; Wessely, J.; Georges, D.; Klonner, G.; Schmid, M.; Dullinger, S.; Thuiller, W. & Guillaume, F. 2017: A dynamic eco-evolutionary model predicts slow response of alpine plants to climate warming. Nature Communications 8(1):15399.

Coulson, T. & Tuljapurkar, S. 2008: The dynamics of a quantitative trait in an age-structured population living in a variable environment. American Naturalist 172(5):599–612.

Dalgleish, H. J.; Koons, D. N. & Adler, P. B. 2010: Can life-history traits predict the response of forb populations to changes in climate variability? Journal of Ecology 98(1):209–217.

Dirzo, R.; Young, H. S.; Galetti, M.; Ceballos, G.; Isaac, N. J. & Collen, B. 2014: Defaunation in the anthropocene. Science 345(6195):401–406.

Draghi, J. A.; McGlothlin, J. W. & Kindsvater, H. K. 2024: Demographic feedbacks during evolutionary rescue can slow or speed adaptive evolution. Proceedings of the Royal Society of London, Series B: Biological Sciences (291):20231553.

Falconer, D. S. & Mackay, T. F. 1996: Introduction to Quantitative Genetics. Longman, London, UK, fourth edition.

Frankham, R.; Lees, K.; Montgomery, M. E.; England, P. R.; Lowe, E. H. & Briscoe, D. A. 1999: Do population size bottlenecks reduce evolutionary potential? Animal Conservation 2:255–260.

Gadgil, M. & Bossert, W. H. 1970: Life historical consequences of natural selection. American Naturalist 104(935):1–24.

Gaillard, J.-M.; Yoccoz, N. G.; Lebreton, J.-D.; Bonenfant, C.; Devillard, S.; Loison, A.; Pontier, D. & Allaine, D. 2005: Generation time: a reliable metric to measure life-history variation among mammalian populations. American Naturalist 166(1):119–123.

Gamelon, M.; Gimenez, O.; Baubet, E.; Coulson, T.; Tuljapurkar, S. & Gaillard, J.-M. 2014: Influence of life-history tactics on transient population dynamics: a comparative approach across mammalian populations. American Naturalist 184(5):673–683.

Gilpin, M. E. & Soulé, M. E. 1986: Minimum viable populations: processes of extinction. Conservation Biology: The Science of Scarcity and Diversity, (edited by M. E. Soulé). Sinauer Associates, Sunderland, Massachusetts, USA.

Gomulkiewicz, R. & Holt, R. D. 1995: When does evolution by natural selection prevent extinction? Evolution 49(1):201–207.

Hairston, N. G. & de Stasio, B. T. 1988: Rate of evolution slowed by a dormant propagule pool. Nature 366:239–242.

Houle, D. 1992: Comparing evolvability and variability of quantitative traits. Genetics 130:195–204.

Hufbauer, R. A.; Szücs, M.; Kayson, E.; Youngberg, C.; Koontz, M. J.; Richards, C.; Tuff, T. & Melbourne, B. A. 2015: Three types of rescue can avert extinction in a changing environment. Proceedings of the National Academy of Sciences, USA 112(33):10557–19562.

Jackson, J.; Le Coeur, C. & Jones, O. 2022: Life history predicts global population responses to the weather in terrestrial mammals. eLife 11:e74161.

Jones, O. R.; Scheurlein, A.; Salguero-Gómez, R.; Camarda, C. G.; Schaible, R.; Casper, B. B.; Dahlgren, J. P.; Ehrlén, J.; Garcia, M. B.; Menges, E. S.; Quintana-Ascencio, P. F.; Caswell, H.; Baudisch, A. & Vaupel, J. W. 2014: Diversity of ageing across the tree of life. Nature 505:169–173.

Kendall, B. E.; Fox, G.; Fujiwara, M. & Nogeire, T. 2011: Demographic heterogeneity, cohort selection and population growth. Ecology 92(10):1985–1993.

Kopp, M. & Matuszewski, S. 2014: Rapid evolution of quantitative traits: theory and theoretical perspectives. Evolutionary Applications 7:169–191.

Kuparinen, A.; Savolainen, O. & Schurr, F. M. 2010: Increased mortality can promote evolutionary adaptation of forest trees to climate change. Forest Ecology and Management 259(5):1003–1008.

Lande, R. 1976: Natural selection and random genetic drift in phenotypic evolution. Evolution 30:314–334.

Lande, R. 2009: Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. Journal of Evolutionary Biology 22(7):1435–1446.

Lande, R. & Shannon, S. 1996: The role of genetic variation in adaptation and population persistence in a changing environment. Evolution 50(1):434–437.

Lewontin, R. & Cohen, D. 1969: On population growth in a randomly varying environment. Proceedings of the National Academy of Sciences, USA 62(4):1056–1060.

Loria, A.; Cristecu, M. & Gonzalez, A. 2022: Genotype diversity promotes the persistence of *Daphnia* populations exposed to severe copper stress. Journal of Evolutionary Biology 35(2):265–277.

Lyberger, K. P.; Osmond, M. M. & Schreiber, S. J. 2021: Is evolution in response to extreme events good for population persistence? American Naturalist 198(1):44–52.

Lynch, M. & Walsh, B. 1998: Genetics and the Analysis of Quantitative Traits. Sinauer Associates, Sunderland, Massachusetts, USA.

Milner, J. M.; Albion, S. D.; Illius, A. W.; Pemberton, J. M. & Clutton-Brock, T. H. 1999: Repeated selection of morphometric traits in the Soay sheep on St. Kilda. Journal of Animal Ecology 68(3):472–488.

Morris, W. F. 2009: Life history. The Princeton Guide to Ecology, (edited by S. A. Levin et al.). Princeton University Press, Princeton, New Jersey, USA.

Morris, W. F.; Pfister, C. A.; Tuljapurkar, S.; Haridas, C. V.; Boggs, C. L.; Boyce, M. S.; Bruna, E. M.; Church, D. R.; Coulson, T.; Doak, D. F.; Forsyth, S.; Gaillard, J.-M.; Horvitz, C. C.; Kalisz, S.; Kendall, B. E.; Knight, T. M.; Lee, C. T. & Menges, E. S. 2008: Longevity can buffer plant and animal populations against changing climate variability. Ecology 89(1):19–25.

Nakagawa, T. & Osaki, S. 1975: The discrete Weibull distribution. IEEE Transactions on Reliability R-24(5):300–301.

Nordstrom, S. W.; Hufbauer, R. A.; Olazcuaga, L.; Durkee, L. F. & Melbourne, B. A. 2023: How density dependence, genetic erosion, and the extinction vortex impact evolutionary res-

cue. Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences 290:20231228.

Oli, M. K. 2004: The fast-slow continuum and mammalian life-history patterns: an empirical evaluation. Basic and Applied Ecology 5(5):449–463.

Orzack, S. H. & Tuljapurkar, S. 1989: Population dynamics in a variable environment. VII. The evolution of iteroparity. American Naturalist 133(6):901–923.

Ozgul, A.; Childs, D. Z.; Oli, M. K.; Armitage, K. B.; Blumstein, D. T.; Olson, L. E.; Tuljapurkar, S. & Coulson, T. 2010: Coupled dynamics of body mass and population growth in response to environmental change. Nature 466:482–485.

Pianka, E. R. 1970: On r- and K-selection. American Naturalist 104(940):592–597.

Pimm, S. L.; Jones, H. L. & Diamond, J. 1988: On the risk of extinction. American Naturalist 132(6):757–785.

Purvis, A.; Gittleman, J. L.; Cowlishaw, G. & Mace, G. M. 2000: Predicting extinction risk in declining species. Proceedings of the Royal Society of London, Series B: Biological Sciences 267(1456):1947–1952.

R Core Team. 2020: R: A language environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.

Ricker, W. E. 1954: Stock and recruitment. Canadian Journal of Fisheries and Aquatic Sciences 11:559–623.

Salguero-Gómez, R.; Jones, O. R.; Jongejans, E.; Blomberg, S. P.; Hodgson, D. J.; Mbeau-Ache, C.; Zuidema, P. A.; de Kroon, H. & Buckley, Y. M. 2016: Fast-slow continuum and reproductive strategies structure plant life-history variation worldwide. Proceedings of the National Academy of Sciences, USA 113(1):230–235.

Schmid, M.; Paniw, M.; Postuma, M.; Ozgul, A. & Guillaume, F. 2022: A trade-off between robustness to environment fluctuations and speed of evolution. American Naturalist 200(1):E16–E35.

Scholl, J. P.; Urbina-Casanova, R. & Iler, A. M. 2022: The importance of negative density dependence for rare species persistence. Biological Conservation 274:109729.

Shoemaker, W. R. & Lennon, J. T. 2018: Evolution with a seed bank: the population genetic consequences of microbial dormancy. Evolutionary Applications 11(1):60–75.

Templeton, A. R. & Levin, D. A. 1979: Evolutionary consequences of seed banks. American Naturalist 114(2):232–249.

Vander Wal, E.; Garant, D.; Festa-Bianchet, M. & Pelletier, F. 2013: Evolutionary rescue in vertebrates: evidence, applications and uncertainty. Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences 368:20120090.

Vaupel, J. W.; Monton, K. G. & Stallard, E. 1979: The impact of heterogeneity in individual frailty on the dynamics of mortality. Demography 16(3):439–454.

Whitlock, M. C. 2000: Fixation of new alleles and the extinction of small populations: drift load, beneficial alleles, and sexual selection. Evolution 54(6):1855–1861.

Wood, J. L. A.; Yates, M. C. & Fraser, D. J. 2016: Are heritability and selection related to population size in nature? Meta-analysis and conservation implications. Evolutionary Applications 9(5):640–647.

Young, T. P. 1981: A general model of comparative fecundity for semelparous and iteroparous life histories. American Naturalist 118(1):27–36.