Modeling the Effects of Evolutionary History and Stochasticity on Population Responses to Rapid Environmental Change

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Abstract

Anthropogenic environmental changes have dramatically impacted natural populations. Phenotypic responses represent a key set of mechanisms by which organisms cope with these environmental changes. Some species have responded well and proliferated, while most have responded poorly and declined. Success depends both on the evolutionary pressures that shape a population's phenotypic strategies, as well as contingent events as population responds to a particular change. This dissertation develops theory to better understand the role of both past evolution and contingency on the ability of populations to cope with rapid environmental change. My first chapter models the influence of variability in seed fall on the evolution of caching behavior in scatterhoarding rodents, with parameters derived from the European beech (*Fagus sylvatica*) and yellow-necked mice (*Apodemus flavicollis*). We find that caching behavior is more prevalent when seed fall is more variable, illustrating the importance of environmental variability in driving phenotypic evolution. My second chapter models the evolution of within-generational plasticity, transgenerational plasticity, and bet-hedging as part of a unified "cue integration system" under a range of historical environmental conditions. We examine how different cue integration systems affect populations' ability to cope with multiple types of environmental change, finding that populations tend to cope poorly when environmental change makes previously reliable cues unreliable. This chapter ties together the effect of historical conditions on the evolution of phenotypic strategies and the ability of those evolved strategies to enable a population to respond to a changing environment. My third chapter models how different kinds of contingency, in the form of demographic stochasticity, sex-ratio stochasticity, and phenotypic stochasticity, affect the likelihood of extinction for a population that must rapidly adapt to a changing environment in order to persist. I find that phenotypic stochasticity contributes relatively more to the ability to predict extinction or recovery than demographic stochasticity or sex ratio stochasticity early during rescue but contributes less as time goes on. Taken together, these chapters tie together the effects of evolutionary history and stochastic contingency on populations' ability to respond

to environmental change and make important progress in understanding the complex ways in which anthropogenic change affects the natural world.

Introduction

Human activity has had a profound impact on natural populations, leading to widespread population declines and extinction (Ceballos et al. 2017; Román-Palacios and Wiens 2020), loss of habitat (Brooks et al. 2002; Mantyka-pringle et al. 2012), changing environments due to climate change (Bellard et al. 2012; Doney et al. 2012; Ummenhofer and Meehl 2017; Urban 2015), and the spread of invasive species (Bellard et al. 2012). Species many respond to these disturbances in many ways, such as by shifting their ranges to track changing conditions (Bates et al. 2014; Tingley et al. 2012) or altering their behavior, physiology, or other phenotype through either phenotypic plasticity (Charmantier et al. 2008; Wong and Candolin 2015) or rapid evolution (Hendry et al. 2017). There is substantial variation in the success of these responses: many species have coped poorly with environmental change and declined, while others have proliferated and spread (Sih 2013). This dissertation develops mathematical theory to better understand the variation in population responses to environmental change and its impact on population persistence.

The success or failure of a phenotypic response to changing conditions is a key factor in determining how well populations respond to environmental change (Charmantier et al. 2008; Merila and Hendry 2014; Nicotra et al. 2010). Whether organisms possess the capacity for such phenotypic responses strategies depends on whether the population's historical environment promoted the evolution of such capacity. In particular, populations that experienced a history of environment variability often have evolved strategies to deal with this variability (e.g. plasticity, dispersal, behavior), which in turn allow them to better cope with rapid environmental change (Candolin and Wong 2012; Donelan et al. 2020; Hendry et al. 2008; Sih 2013; Sih et al. 2011). Thus past environments, though evolutionary pressure, shape population responses to contemporary

changes.

My first chapter examines a case study in the effect of environmental conditions on the evolution of behavioral strategies. Many granivores exhibit scatterhoarding behavior, which is to say that they store seeds in numerous small caches over winter (Gómez et al. 2019; Lichti et al. 2017; Pesendorfer et al. 2016; Vander Wall 1990). These caches are undefended and at high risk of pilferage by conspecifics, making the evolution of this behavior difficult to explain. In many forests, seed production by trees is concentrated in what are referred to as mast years, creating patterns of pulsed resources (Kelly 1994; Kelly and Sork 2002). Using parameters derived from the European beech (*Fagus sylvatica*) and yellow-necked mice (*Apodemus flavicollis*), we model the evolution of caching in scatterhoarders to determine the conditions under which environmental variability (seed fall patterns) promotes seed caching. Our finding that caching behavior is more prevalent when mast years are more intense and less frequent illustrates the importance of environmental variability in driving phenotypic evolution.

My second chapter then examines the conditions under which the phenotypic strategies selected for by historical environmental variability allow a population to cope well with different changes to its historical environmental conditions. We consider the phenotypic strategies of within-generational plasticity (where the environmental experienced by an organism during its own lifetime affects its phenotypic expression), transgenerational plasticity (where the environments experienced by an organism's parents or grandparents affects its phenotypic expression), and diversified bet-hedging (where an organism produces offspring with a variety of phenotypes in order to hedge against uncertain future conditions) as part of unified "cue-integration system" for utilizing environmental information to cope with environmental variability (Botero et al. 2015; Dall et al. 2015; English et al. 2015; Kuijper and Hoyle 2015; Leimar and McNamara 2015; McNamara et al. 2016; Shea et al. 2011). This chapter links together the questions of how historical environments affect the evolution of phenotypic strategies and which strategies allow a population to best cope with different forms of environmental change. Using a linear reaction-norm model of plasticity (following McNamara et al. 2016), we solve for the optimal mix of WPG, TGP, and

bet-hedging for a given historical environment and then examine the change in long-term growth rate when a population with this historically optimal strategy experiences a rapid environmental change that causes the strategy to no longer be optimal. Our finding that populations tend to suffer the most when previously reliable environmental cues become unreliable in a changed environment illustrates the important role of historical environmental conditions in hindering or facilitating a population's ability to cope with rapid environmental change.

Populations do not only rely on pre-evolved phenotypic strategies to cope with environmental change. Many populations are capable of rapidly adapting to new environmental conditions that would otherwise cause extinction, a phenomenon known as evolutionary rescue (Bell 2017; Carlson et al. 2014; Vander Wal et al. 2013). However, even when rapid, evolution can be slow compared to phenotypic plasticity. Populations are predicted to experience a demographic decline prior to successful adaptation and may reach low population sizes during this process, making the population vulnerable to stochasticity in individual births and deaths (Bell and Gonzalez 2009; Engen et al. 1998, 2001; Lande 1988, 1998). This stochasticity may affect both the rate of evolution, through genetic drift, and the rate of demographic decline, through demographic stochasticity and sex-ratio stochasticity (Lande 1988). All three forms of stochasticity may contribute to persistence or extinction of populations undergoing evolutionary rescue, but they do so through different pathways.

My third chapter develops a method for separating the contribution of different forms of stochasticity, at different points in time, to ultimate population outcomes such as extinction or persistence. I then use this method decompose the effects of phenotypic stochasticity, demographic stochasticity, and sex-ratio stochasticity on persistence or extinction, time to extinction, and time to recovery for populations undergoing evolutionary rescue in response to an abrupt environmental change. My finding that phenotypic stochasticity contributes relatively more to the ability to predict extinction or recovery than demographic stochasticity or sex ratio stochasticity early during rescue, but not later, illustrates how the importance of different forms of stochasticity may change over the course of evolutionary rescue.

The three chapters give complementary insight into population responses to rapid environmental change. The focus in chapter 3 on rapid evolution and the timing of stochasticity complements the previous two chapters, which focus on the long-term evolution of phenotypic strategies and do not consider rapid evolution or how the time of specific stochastic events affects the population. Further, both environmental stochasticity and demographic stochasticity are important sources of variability for populations (Boettiger 2018; Engen et al. 1998). Chapters 1 and 2 focus on the effect of external, environmental variability on evolution and population growth, while chapter 3 focuses on demographic stochasticity and other forms of variability that are intrinsic to the population. Taken together, these chapters tie together the effects of evolutionary history and stochastic contingency on populations' ability to respond to environmental change and make important progress in understanding the complex ways in which anthropogenic change affects the natural world.

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Mast seeding promotes evolution of scatter-hoarding

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Abstract

Many plant species worldwide are dispersed by scatterhoarding granivores: animals that hide seeds in numerous, small caches for future consumption. Yet, the evolution of scatterhoarding is difficult to explain because undefended caches are at high risk of pilferage. Previous models have attempted to solve this problem by giving cache owners large advantages in cache recovery, by kin selection, or by introducing reciprocal pilferage of "shared" seed resources. However, the role of environmental variability has been so far overlooked in this context. One important form of such variability is masting, which is displayed by many plant species dispersed by scatterhoarders. We use a mathematical model to investigate the influence of masting on the evolution of scatter-hoarding. The model accounts for periodically varying annual seed fall, caching and pilfering behavior, and the demography of scatterhoarders. The parameter values are based mostly on research on European beech (*Fagus sylvatica*) and yellow-necked mice (*Apodemus flavicollis*). Starvation of scatterhoarders between mast years decreases the population density that enters masting events, which leads to reduced seed pilferage. Satiation of scatterhoarders during mast events lowers the reproductive cost of caching (i.e. the cost of caching for the future rather than using seeds for current reproduction). These reductions promote the evolution of scatter-hoarding behavior especially when interannual variation in seed fall and the period between masting events are large.

Introduction

Masting, or periodic, synchronized production of abundant seed crops, is a common reproductive strategy of many plants (Kelly 1994, Kelly and Sork 2002) and a classic example of a pulsed resource (Ostfeld and Keesing 2000, Yang et al. 2010). Masting provides foundational, yet unstable resource levels, creating cycles of feast and famine in food webs (Clark et al. 2019). These cycles strongly influence behavior and life-history strategies of consumers. For example, animals migrate to track populations of masting plants (Jenni 1987), enter diapause to survive lean periods between mast-events (Maeto and Ozaki 2003), and increase reproduction in anticipation of masting (Boutin et al. 2006, Bergeron et al. 2011). However, despite decades of research, many impacts of masting on consumers remain poorly understood (Clark et al. 2019). Here we use a mathematical model to show that masting can play an important but overlooked role in the evolution of a widespread animal behavior: scatter-hoarding.

Scatter-hoarding is defined as caching seeds for future consumption in many small, widely-dispersed caches (Vander Wall 1990). This caching strategy is used by numerous species of animals, most notably by rodents and corvids (Pesendorfer et al. 2016, Lichti et al. 2017, Gómez et al. 2019). Scatterhoarders provide essential seed dispersal services in many ecosystems throughout the world. According to a recent review, there are 1279 species of plants known to rely on this mode of seed dispersal, although this number is certainly underestimated (Gómez et al. 2019). However, even though scatter-hoarding is so widespread, the evolutionary advantage of this behavior is not obvious because the caches are undefended and often suffer very high rates of pilferage (Schmidt and Ostfeld 2008; Jansen et al. 2012; Zwolak et al. 2016; Dittel et al. 2017). Thus, scatter-hoarding appears vulnerable to cheating by non-caching pilferers (Andersson and Krebs 1978; Smulders 1998; Vander Wall and Jenkins 2003).

First attempts to solve this problem focused on the role of the owner's advantage. According to a model by Andersson and Krebs (1978), scatter-hoarding can evolve when cache owners are substantially more likely to recover caches than are naive foragers. Empirical estimates of the owner advantage vary widely but appear relatively high in

scatter-hoarding birds (particularly those that rely on specialized spatial memory: e.g. Brodin 2010) and quite low in mammals. In most studies on rodents, cache owners are only 2-4 times more likely to recover their caches when compared with naïve individuals (Jacobs and Liman 1991; Jacobs 1992; Briggs and Vander Wall 2004; Thayer and Vander Wall 2005; Vander Wall et al. 2006; 2008; Hirsch et al. 2013; Gu et al. 2017; see also Steele et al. 2011). In many systems, the owner's advantage appears to be insufficient to prevent substantial cache loss to pilferage (2-30% lost per day, according to a review by Vander Wall and Jenkins 2003, though this rate might vary depending on environmental characteristics like soil moisture: Vander Wall 2000).

A later model by Vander Wall and Jenkins (2003) suggested that caching can represent an adaptive, stable strategy when all caches are reciprocally pilfered by scatter-hoarding animals with overlapping home ranges (see also Smulders 1998). Under this scenario, caches represent a collective resource used by selfish individuals (Vander Wall and Jenkins 2003). The reciprocal pilferage hypothesis predicts that animals are unlikely to avoid pilferage, but can compensate for it by pilfering caches of other individuals. As a corollary, individuals should invest in their pilfering tactics rather than in theft-reducing strategies (but see e.g. Dally et al. 2006, Steele et al. 2008, Galvez et al. 2009, Shaw and Clayton 2013, Hirsch et al. 2012, Muñoz and Bonal 2011 for examples of potentially costly behaviors aimed to reduce pilferage).

Environmental variability represents an additional, potent mechanism for scatter-hoarding that has been largely overlooked in the existing models. Such variability is pervasive in ecosystems dominated by plants that produce scatterhoarder-dispersed fruits because such plants usually show pronounced masting (Herrera et al. 1998; Vander Wall 2001). Examples of scatterhoarder-dispersed masting plants can be found in the tropics (Norden et al. 2007; Mendoza et al. 2018), deserts (Meyer and Pendleton 2015; Auger et al. 2016), and in temperate zones (Koenig and Knops 2000; Schauber et al. 2002; Shibata et al. 2002). While studies of masting have often emphasized the benefit of masting to plants in terms of reduced per capita seed predation ("predator satiation": Kelly 1994), masting also has important effects on consumer population dynamics that can feedback to affect the

evolution of caching. In particular, the cycles of satiation and starvation induce striking fluctuations in consumer population size (Ostfeld and Keesing 2000; Yang et al. 2010; Bogdziewicz et al. 2016). Typically, masting triggers a temporary increase in consumer population size followed by a pronounced crash. Thus, when the next mast year comes, seed to consumer ratios are particularly high (Kelly 1994; Ostfeld and Keesing 2000).

We use a mathematical model to investigate the influence of mast-related fluctuations in scatterhoarder population size on the evolution of scatter-hoarding. The model mimics interactions between a masting tree and a scatterhoarding rodent. The scatterhoarders consume or cache harvested seeds and pilfer or recover their own caches over years that differ in both the magnitude of seed fall and the number of competing consumers. Previous models demonstrated that caching is influenced by the owner's advantage in cache recovery and the probability that scatterhoarders survive long enough to use the caches (Andersson and Krebs 1978; Smulders 1998; Vander Wall and Jenkins 2003). However, both the proportion of recovered seeds and scatterhoarder survival depend on the magnitude of seed fall and the resulting fluctuations in population size (Pucek et al. 1993, Theimer 2005, Zwolak et al. 2016). Thus, we expand on previous models by including the effects of environmental variability resulting from mast seeding on caching behavior. We do this by treating the proportion of seeds that are cached, rather than immediately consumed, as an evolving trait and examining how the evolutionarily stable strategy of this caching behavior varies with (1) masting intensity, (2) the frequency of mast years, (3) the owner's advantage in cache recovery, and (4) the survival of scatterhoarders. Our results demonstrate that mast-related fluctuations in scatterhoarder population size reduce both the risk of cache loss to pilferers and the reproductive cost of caching (i.e. the cost of caching seeds for future use rather than using seeds for current reproduction), thus promoting the evolution of scatter-hoarding.

Methods

Modeling approach

We consider a population of scatterhoarders that experience three distinct periods of seed availability in each year: fall, winter/spring, and summer. During the fall, seeds become available, and scatterhoarders gather and either immediately consume or cache them. Energy from consumed seeds contributes to reproduction while cached seeds may be recovered for use during the subsequent winter/spring. During the winter/spring, scatterhoarders are sustained by seeds from their own caches or seeds they pilfer from the caches of other scatterhoarders. As caching behavior may not always be favored, we implicitly assume the availability of other winter resources that prevent population extirpation. During the summer, scatterhoarders survive and reproduce using resources other than seeds. Caching behavior for an individual is represented by the threshold T, such that the individual consumes up to T seeds during the fall and caches the rest. We determine the conditions under which caching behavior is favored by solving for the evolutionary stable strategy (ESS) of T. The central conundrum is that the strategy of scatter-hoarding appears vulnerable to cheating by non-caching pilferers, who may invade the population and outcompete caching individuals. If the population is monomorphic for a particular caching threshold, then this threshold value is an ESS if this population cannot be invaded (outcompeted) by individuals with any other threshold value.

Model description

During fall (period 1), there is seed fall of $S(t)$ from the primary seed source. Seeds are gathered at a rate proportional to the density $n_1(t)$ of scatterhoarders during this period. The proportionality constant a_1 corresponds to the per-capita (i.e., per scatterhoarder) seed harvest rate. Seeds are also lost to other sources (e.g. competitors, germination, decay, etc.) at a per-capita rate of L_1. If all seeds are gathered or lost to other sources by the end of the fall, then the amount of seed gathered per individual equals:

$$G(t) = \frac{a_1 S(t)}{L_1 + a_1 n_1(t)}$$

All seeds above a threshold, T, are cached by individuals for later in the year. The seeds which are not cached, $\min\{G(t),T\}$, are used for survival and reproduction. The number of

offspring produced by an individual, $R_1(t)$, at the end of the fall is a saturating function of min{$G(t),T$}, with a maximal number of offspring b and a half saturation constant h (i.e. h is the amount of resources required to produce $b/2$ offspring). The fraction of adults surviving from the first period (fall) to the second period of the year (winter/spring) equals s_1. Thus, the total density $n_2(t)$ of individuals entering winter/spring equals

$$n_2(t) = [R_1(t) + s_1]n_1(t) \text{ where } R_1(t) = \frac{b\, min\{G(t), T\}}{h + min\{G(t), T\}}$$

The main resource available to individuals during winter/spring is the total size of seed caches $\max\{G(t)\text{-}T,0\}n_1(t)$. Owners of the cached seed who survived gather their cached seed at a rate proportional to the size of their seed caches. This proportionality constant a_2 corresponds to the per-capita rediscovery and use rate of their caches. All other individuals are assumed to pilfer seed from others' caches at a per-capita rate a_{pil}. Seeds are lost to other sources at a per-capita rate of L_2. If all cached seeds are gathered or lost by the end of winter/spring, then the fraction of caches recovered by its owner given that the owner survived from fall to winter/spring is

$$M(t) = \frac{a_2}{L_2 + a_2 + a_{pil}(n_2(t) - 1)}$$

while the fraction of caches that was pilfered by each non-owner is

$$O(t) = \frac{a_{pil}}{L_2 + a_2 + a_{pil}(n_2(t) - 1)}$$

For seed caches whose owner died, the fraction that was recovered by a living non-owner is

$$D(t) = \frac{a_{pil}}{L_2 + a_{pil}n_2(t)}$$

The total amount of cached seed gathered by a surviving individual from fall is the amount of seed recovered from its own caches plus the amount of seeds pilfered from the caches of other surviving individuals and the caches of deceased individuals:

$$C_{survivor}(t) = \max\{G(t) - T, 0\}[M(t) + O(t)(s_1 n_1(t) - 1) + D(t)(1 - s_1)n_1(t)]$$

Since individuals that were born at the end of the fall had no opportunity to cache, the total amount of cached seed gathered by these individuals in the spring/winter is only the amount pilfered from the caches of either surviving or deceased individuals:

$$C_{new}(t) = \max\{G(t) - T, 0\}[O(t)s_1 n_1(t) + D(t)(1 - s_1)n_1(t)]$$

If s_2 is the fraction of individuals surviving to summer (period 3), then the density of individuals entering summer equals

$$n_3(t) = [R_2(t) + s_2]n_2(t)$$

where $R_2(t)$ is the population-level per-capita fecundity corresponding to the weighted combination of reproductive contributions of individuals surviving from fall and new individuals born at the end of fall (for simplicity, we assume the same value of h for surviving and new born individuals):

$$R_2(t) = \frac{b\, C_{survivor}(t)}{h + C_{survivor}(t)} \frac{s_1}{R_1(t) + s_1} + \frac{b\, C_{new}(t)}{h + C_{new}(t)} \frac{R_1(t)}{R_1(t) + s_1}$$

During this final period of the year (summer), individuals rely on other resources with availability A to reproduce and survive with probability s_3 (these resources represent alternative foods, such as fungi, invertebrates, or seeds of other plant species; adjusted to obtain population dynamics consistent with patterns observed in the field). Thus, the density of individuals entering the fall of the next year equals

$$n_1(t + 1) = [R_3(t) + s_3]n_3(t)$$

where $R_3(t)$ is the per-capita reproduction. We model this per-capita reproduction using a Beverton-Holt function

$$R_3(t) = \frac{\beta}{1 + \alpha n_3(t)}$$

where β is the maximal summer fecundity and α determines the strength of intraspecific competition. By composing the equations across the three periods of the year, the yearly update rule for population densities at the beginning of fall is

$$n_1(t + 1) = [R_3(t) + s_3][R_2(t) + s_2][R_1(t) + s_1]n_1(t)$$

We modeled seed fall S(t) in the fall as a periodic function of time where the period P corresponds to the time between masting years. In the masting years, $S(t)=S_{high}$, next year $S(t)=S_{min}$ (typically, seed crops produced after mast years are particularly scant: Pearse et al. 2016, Bogdziewicz et al. 2020), then $S(t) = S_{low}$ until another mast year. Our analysis assumes that the average seed output, $(S(1)+S(2)+\ldots+S(P))/P$, is fixed and what varies is the

proportion of total seed output in the masting year. Higher intensity of masting means more seeds during the masting year, but concomitantly fewer seeds in other years (as opposed to just increasing seed output in masting years with no effect on seed production in other years). Similarly, when we vary the number of years between masting events, the average seed output remains the same (i.e., longer intermast interval corresponds to higher seed production in mast years).

Figure 1-1 illustrates typical dynamics of the model for the baseline parameters described below. In this figure, seed masting occurs every four years (Fig. 1-1a) and leads to a stable, four-year population cycle (Fig. 1-1b). Population densities (Fig. 1-1b) exhibit seasonal as well as yearly variation. Highest densities are reached at the end of the masting year (year 1) and crash to low densities the ensuring years (years 2-4). For lower caching thresholds, caching occurs in all years except the year after a masting event (green bars in Fig. 1-1a). Table A1 in Appendix A lists all parameters for the model and their meaning.

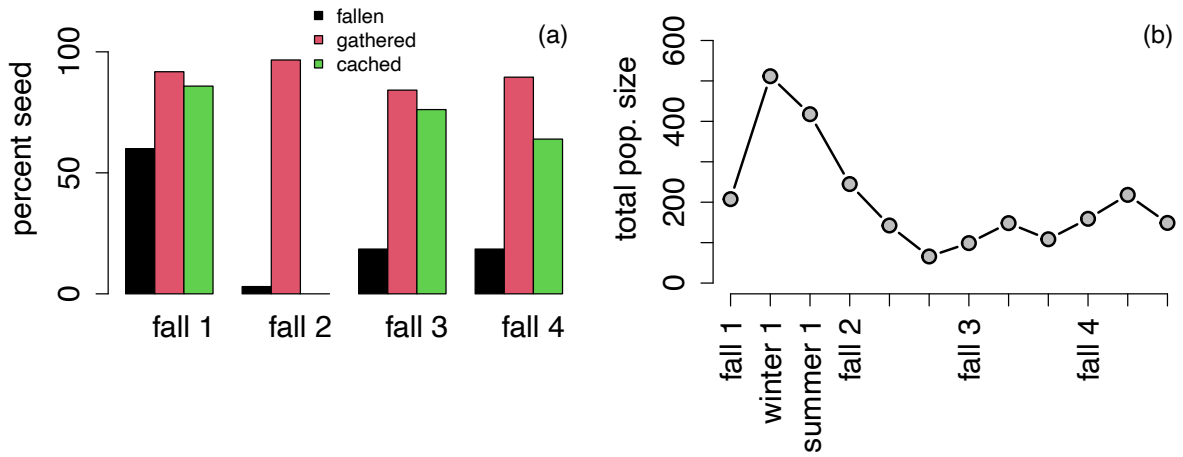

Fig. 1-1. *The annual dynamics of fall seeds (a) and seasonal population dynamics (b) when masting years occur every 4 years. Both (a) and (b) correspond to a stable, periodic solution of the model. In (a), the percent of seeds that fall each year from all of the seeds in 4 year masting interval year are plotted as black bars; the first year corresponds to the masting year that is followed by years of lower seed fall. The percent of fallen seed that are gathered each year correspond to the red bars, while the*

percent of gathered seed that is cached are the green bars. In (b), the total population densities vary intra- and inter-annually; the highest densities occur in the winter/spring period of the masting year after which the population densities crash to lower densities. Parameter values as described in the main text with a 6-fold owner's advantage in cache recovery.

Model Parameters

The parameter values are based mostly on research on European beech (*Fagus sylvatica*) and yellow-necked mice (*Apodemus flavicollis*). *Apodemus* mice are among the most important seed predators and scatterhoarders in Eurasia (e.g. Muñoz and Bonal 2011, Shimada et al. 2015, Yang et al. 2019, Wróbel and Zwolak 2019). While we found it useful to base our parameter estimates on a specific, reasonably well-studied system, we also performed a global sensitivity analysis for our main conclusions (Appendix B). Specifically, for each of main results, we reran the simulations 100 times with each parameter, call it x, chosen independently from a uniform distribution on the interval [x/1.5,1.5x].

The parameters a_1 and L_1 (per-capita harvest rate and per-capita seed loss) may be reduced to the single parameter L_1/a_1. Rearranging the equation for the amount of seeds gathered G yields $L_1/a_1 = \frac{(1-G/S_0)n_1}{G/S_0}$. The parameters n_1 and n_2 were taken to be the average species-wide density for A*podemus flavicollis*: 17.7 individuals/ha (Jones et al. 2009). Estimates of the proportion of seeds removed from the forest floor (G/S_0) tend to be variable with Zwolak et al. (2016) reporting 78% seed removal during mast years and 91% seed removal during non-mast years for *A. flavicollis*, and Le Louarn and Schmitt (1972) reporting 61% and 74% seed removal by *Apodemus sylvaticus* during two different years. We selected the average value of 76% as our estimate of seed removal. Thus, $L_1/a_1 = 5.59$. In all cases where L_1 and a_1 were treated as separate parameters, we used $a_1 = 1$ and set L_1 equal to our choice for L_1/a_1.

The parameters a_{pil} and L_2 may similarly be reduced to L_2/a_{pil}. Zwolak et al. (2016) estimated the recovery of seeds from artificial caches to be 54% during nonmast years and

5% during mast years, which we assumed to be roughly equivalent to the proportion of seeds recovered from an abandoned cache ($D/\max\{G(t)-T,0\}$). We then estimated L_2/a_{pil} to be between 15.1 and 336.3. In our analysis, we set this value to the upper end of this range (300) as this leads to more conservative estimates of when caching evolves. In all cases where $L_2$and a_{pil} were treated as separate parameters, we used $a_{pil}=1$ and set L_2 equal to our choice for L_2/a_{pil}.

We let $a_2=3$ (when $a_{pil}=1$). This value approximates the results of several studies on scatterhoarding rodents (Vander Wall et al. 2006, 2008; Thayer and Vander Wall 2005; Hirsch et al. 2013) that documented seed removal rates by cache owners and naïve foragers. However, we explored scenarios with both higher and lower cache owner's advantage (see Results).

We assumed a maximum litter size of 11 individuals (Macdonald and Tattersall 2001), with one breeding event per period (2-3 litters per year: Pucek 1984). Assuming that half the population are female and half of the individuals born are female, this yields $b=5.5$.

Half-saturation constant for mid-year reproduction (h) was set as 124 seeds/offspring multiplied by half the maximum number of female offspring (b). This value was calculated on the basis of energy contents of beech seeds (Grodziński and Sawicka-Kapusta 1970), energy requirements of yellow-necked mice (0.60 kcal/g/day: Jensen 1982; average body mass of yellow-necked mice is 28.3 g: AnAge), and typical costs of reproduction-related energy expenditure in small mammals (25% increase in energy expenditure during gestation and 200% increase during lactation: Millar 1978; 1979; Gittleman and Thompson 1988; Sikes 1995; Zhu et al. 2015), given the length of gestation and lactation in yellow-necked mice (26 and 22 days, respectively: AnAge). Note that the link between food availability and reproduction limits winter breeding to masting events (which are known to result in winter reproduction in our and related study systems: Jensen 1982, Pucek et al. 1993, Wolff 1996, Ostfeld et al. 1996).

We used 77.5% as the yearlong monthly survival rate (calculated from data on winter survival in Pucek et al. 1993: see also Jensen 1982 for similar values). We assumed that each period lasts four months, yielding 0.775^4=36.1% as the survival rate for each period ($s_1, s_2,$ and s_3). Winter survival rates in Pucek et al. (1993) are similar to monthly summer survival rates reported or calculated from other studies (e.g. Bujalska and Grüm 2008, Sozio and Mortelliti 2016), thus we assumed equal survival across all seasons in our initial scenario, but examined how relaxing this assumption affects caching rates (see Results).

In principle, food availability affects both reproduction and survival in a manner that depends on life history allocation. That is, an organism can allocate most of its energy budget to enhanced survival or enhanced reproduction, or a blend of the two. Predicting this life history allocation is complex (Roff 2002), thus rather than attempt to predict the optimal allocation (which should depend on optimal caching and vice versa), we draw on the natural history of the system to argue that as a first pass, it is more important to examine how food consumption affects fecundity as opposed to survival. An increase in food consumption clearly increases fecundity. In contrast, we assume that the consumer has alternative food sources (see above) that are sufficient to allow it to survive adequately even if it does not allocate any additional energy from the focal seed source towards survival. We further assume that allocating extra energy to increased survival is not very effective (in our system) because survival also depends heavily on predation, disease, etc. (Jędrzejewska & Jędrzejewski 1998). In this scenario, survival depends little on the amount of the focal seed source consumed. This is also in line with numerous empirical studies reporting that rodents allocate extra energy to increase reproductive output rather than survival (meta-analyzed by Prevedello et al. 2013). In addition, a critical point for relating food to demography is that in short-lived, fecund animals such as rodent scatterhoarders elasticity for survival is low whereas elasticity for reproduction is higher (Heppell et al. 2000), which means that even if survival does vary, this variation does not affect population growth as much as do changes in reproduction. Accordingly, we focus on effects of food on reproduction, and simplify the analysis by assuming that survival is a parameter that is constant across years. However, we do vary survival across all years (see Results).

Numerical Methods

To identify the evolutionary stable caching strategies, we examined whether a small mutant subpopulation using the caching threshold T_m can invade a resident population using the caching threshold T. When the mutant subpopulation densities $m_i(t)$ in each of the periods $i=1,2,3$ are sufficiently small, the effect of the mutant population on the resident population and itself is negligible. Hence, the dynamics of the mutant in the initial phase of invasion can be approximated by the mutant's growth rate when the population is composed entirely of residents. We now describe these dynamics.

As the mutant and resident individuals only differ in their caching strategy, the amount of seeds gathered in year t by a mutant individual equals the amount of seeds gathered $G(t)$ by a resident individual. As for the resident dynamics, yearly update of the mutant's fall density is of the form

$$m_1(t+1) = [Q_3(t) + s_3][Q_2(t) + s_2][Q_1(t) + s_1]m_1(t)$$

$Q_1(t)$ corresponds to the number of offspring produced by a mutant individual during the fall and only differs from the resident in its threshold T_m

$$Q_1(t) = \frac{b\ min\{G(t), T_m\}}{h + min\{G(t), T_m\}}$$

$Q_2(t)$ corresponds to the number of offspring produced by a mutant individual during the winter/spring given by a weighted combination due to the fraction of individuals that survived from the fall and individuals born in the fall:

$$Q_2(t) = \frac{b\ C_{m,survivor}(t)}{h + C_{m,survivor}(t)} \frac{s_1}{Q_1(t) + s_1} + \frac{b\ C_{new}(t)}{h + C_{new}(t)} \frac{Q_1(t)}{Q_1(t) + s_1}$$

where $Q_2(t)$ differs from $R_2(t)$ only in its first term due to surviving individuals with the mutant caching strategy: $C_{m,survivor}(t) = \max\{G(t) - T_m, 0\}M(t) + \max\{G(t) - T, 0\}[O(t)(s_1 n_1(t) - 1) + D(t)(1 - s_1)n_1(t)]$. Finally, the number of offspring produced by a mutant over the summer is the same as the resident i.e. $Q_3(t) = R_3(t)$.

Whether the mutants playing strategy T_m are able to invade the residents playing the strategy T or not depends on their long-term per-capita growth rate

$$s(T, T_m) = \lim_{t\to\infty} \frac{1}{t} \log[Q_3(t) + s_3][Q_2(t) + s_2][Q_1(t) + s_1]$$

provided the limit exists. Over the parameter space (see previous section) that we simulated, the population dynamics always converged to a periodic solution whose period kP is a multiple k of the seed masting period P. Typically, this multiple was 1 or 2 or 4, the latter two corresponding to period-doubling bifurcations. We developed R code to efficiently approximate these periodic solutions. For these periodic solutions of the resident dynamics, the long-term per-capita growth rate of mutant strategy T_m against resident strategy T equals

$$s(T, T_m) = \frac{1}{kP} \sum_{t=1}^{kP} \log[Q_3(t) + s_3][Q_2(t) + s_2][Q_1(t) + s_1]$$

A strategy T is an evolutionarily stable strategy (ESS) for caching if $s(T,T_m)<0$ for all strategies $T_m \neq$T. To find ESSs for caching, we derive in Appendix C an explicit expression for the fitness gradient $\frac{\partial s}{\partial T_m}(T, T)$ when the resident population is playing threshold strategy T. When the fitness gradient is positive, mutants with a higher threshold strategy than the residents can invade while mutants with a lower threshold strategy fail (Geritz et al. 1997). When the fitness gradient is negative, the opposite occurs. As mutants with larger or smaller thresholds fail when invading a resident population playing the ESS, the fitness gradient equals zero at an ESS. Hence, we identified ESSs by iteratively solving for thresholds T at which the fitness gradient $\frac{\partial s}{\partial T_m}(T, T)$ is zero (Fig. C1 in Appendix C).

Our results focus on the fraction of seeds cached (F) rather than the caching threshold (T), as this quantity is easier to interpret. The relationship between these two measures of caching is given by $F = \max\{0, \frac{G(t)-T}{G(t)}\}$. As the amount of seeds gathered G(t) varies from year to year, the percentage of seeds cached when playing the ESS also varies from year to year. We also examine pilferage risk and the marginal reproductive cost of caching. Pilferage risk is the probability (expressed as a percent) that a seed is pilfered from a surviving individual's cache during winter/spring and equals $100(n_2(t) - 1)O(t)$. If F_m denotes the percentage of seeds cached by mutant individuals, then the marginal reproductive cost of caching equals the infinitesimal reduction in reproductive output for a mutant individual

caching an infinitesimal amount of seeds rather than consuming them i.e., $-\frac{dQ_1}{dF_m}\Big|_{F_m=0} = \frac{bhG(t)}{(G+h)^2}$.

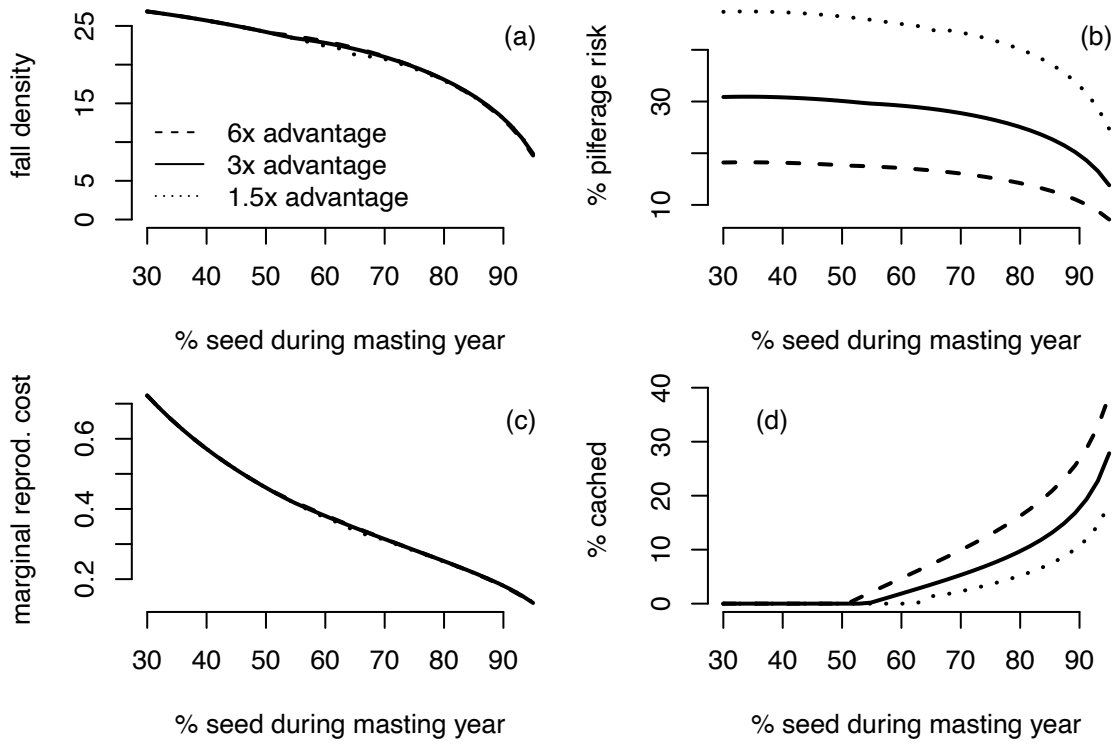

Fig. 1-2. *Mast year fall population density in individuals/ha (a), pilferage risk, defined as the probability that cached seed would be pilfered (b), marginal reproductive costs of caching (c), and proportion of seeds cached rather than eaten (d) as a function of masting intensity (expressed as the percentage of total seed production that occurs during mast years), with mast occurring every 4th year. Dashed, solid, and dotted lines represent the magnitude of owner's advantage in cache recovery (owners 6, 3, and 1.5 times more likely to discover their own caches relatively to naïve foragers). All dependent variables are given at the evolutionary stable caching strategy and its associated periodic population dynamics.*

Results

Increasing intensity of masting results in decreased fall scatterhoarder population density (i.e., the density that enters masting events; Fig. 1-2a). This occurs because reproduction is a saturating function of seeds gathered and the reproductive gains of higher seed availability during masting years are outweighed by the reproductive losses due to lower seed availability during non-mast years.

Increasing masting intensity also reduces the risk that a cached seed would be pilfered (Fig. 1-2b), particularly when a high proportion of seeds are produced during mast years. The responses of pilferage risk and fall density are correlated (see, also, Figs. 1-3 and 1-4) because lower population density means fewer pilferers.

Furthermore, increasing masting intensity is associated with a decline in marginal reproductive costs of caching – the cost of caching seeds for future use rather than using seeds for current reproduction (Fig. 1-2c). Lower population densities and higher seed abundance during mast years mean more seeds per individual. Because reproduction is a saturating function of seeds consumed, the marginal reproductive cost of caching declines as seed abundance increases.

As a result of reduced pilferage risk and marginal costs, increasing masting intensity causes an accelerating increase in the ESS proportion of seeds cached rather than eaten (Fig. 1-2d).

Higher recovery advantage by cache owners reduces pilferage risk (dashed vs. solid vs. dotted lines Fig. 1-2a), but has little effect on population densities and marginal reproductive costs in the fall of masting years. Consequently, higher owner advantage selects for greater caching.

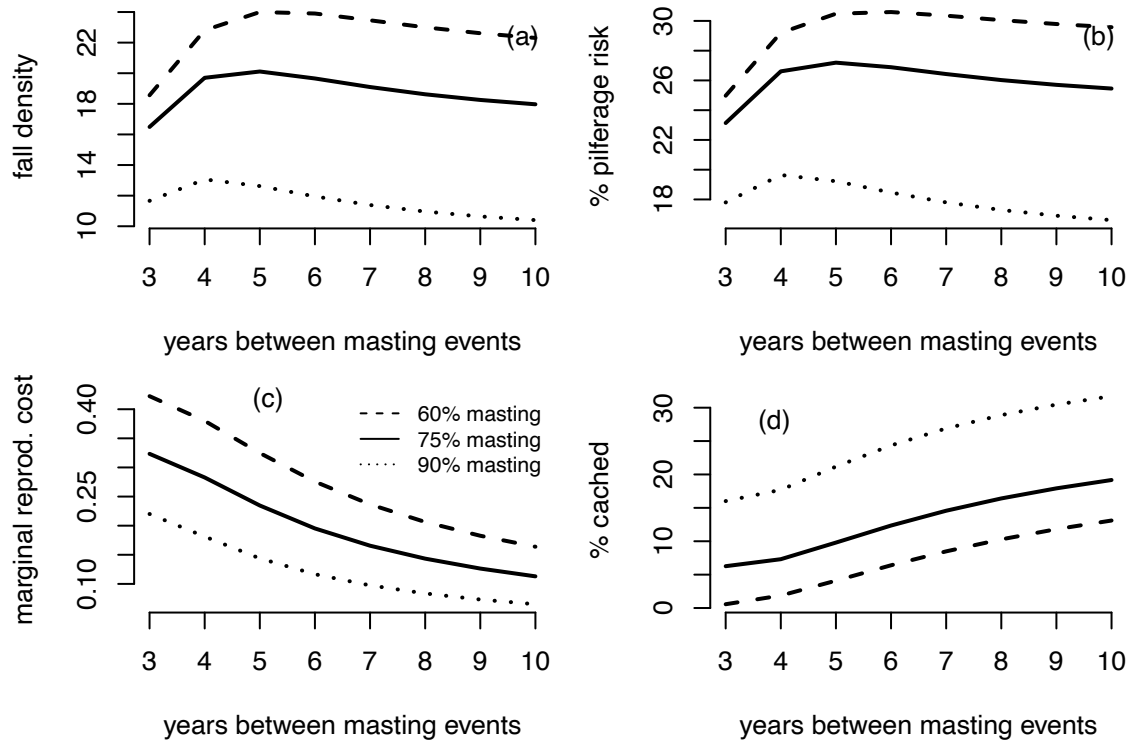

Fig. 1-3. *Mast year fall population density (a), pilferage risk (b), marginal reproductive costs of caching (c), and proportion of seeds cached rather than eaten (d) as a function of masting interval. Dashed, solid, and dotted lines represent masting intensity (60, 75 or 90% of total seed production occurring during mast years). All dependent variables are given at the evolutionary stable caching strategy and its associated periodic population dynamics.*

More years with poor seed crops between masting events lowers marginal reproductive costs (because there are more seeds per individual) (Fig. 1-3c) but can increase or lower densities of individuals entering the fall of a masting year (Fig. 1-3a) which increases or lowers the risk of seed pilferage (because more or fewer individuals enter winter) (Fig. 1-3b). Collectively, the lower reproductive costs outweigh the effects of pilferage risk and select for more caching (Fig. 1-3d). Varying masting intensity (60, 75 or 90% of seeds produced during mast years: dotted, solid or dashed line on Fig. 1-3) affects the magnitude of these changes in the manner consistent with Fig. 1-1., with only minor effects on the shape of responses to the masting interval.

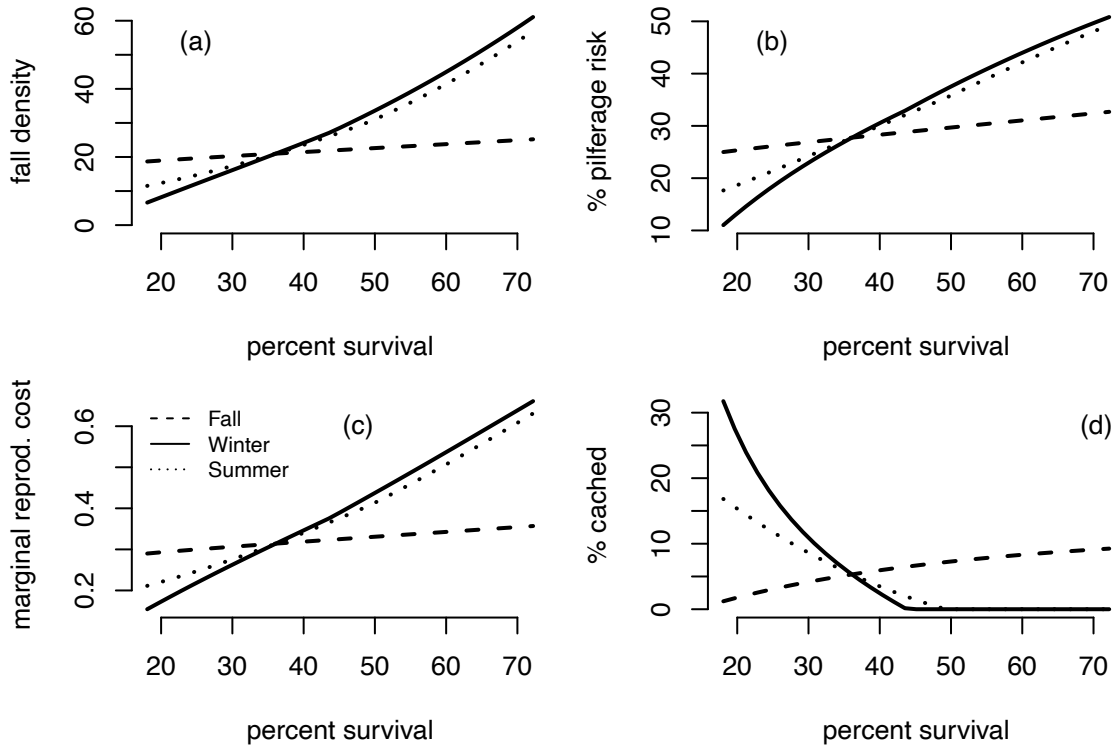

Fig. 1-4. *Mast year fall population density (a), pilferage risk (b), marginal reproductive costs of caching (c), and proportion of seeds cached rather than eaten (d) as a function of scatterhoarder survival. Dashed, solid, and dotted lines denote responses to changes in fall, winter/spring, and summer survival, respectively. All dependent variables are given at the evolutionary stable caching strategy and its associated periodic population dynamics.*

Increasing the survival of scatterhoarders leads to increases in fall population density, pilferage risk, and marginal reproductive costs of caching (Fig. 1-4). These effects are the strongest due to increases in winter survival, intermediate due to summer survival, and the weakest due to fall survival. This is likely because after masting the greatest concentration of births occurs in the fall and winter, resulting in the winter population having a higher percentage of new individuals (who are not subject to mortality during the previous period) than the summer and fall populations. Thus, an increase in mortality in the fall affects a smaller proportion of the population than an increase in mortality in the winter or summer. Despite increasing marginal reproductive costs and pilferage risk, increasing fall survival,

unlike winter/spring or summer survival, selects for more caching. This occurs because, unlike summer or spring/winter survival, fall survival increases the likelihood that an individual caching in the fall will survive to the winter/spring to make use of their cache.

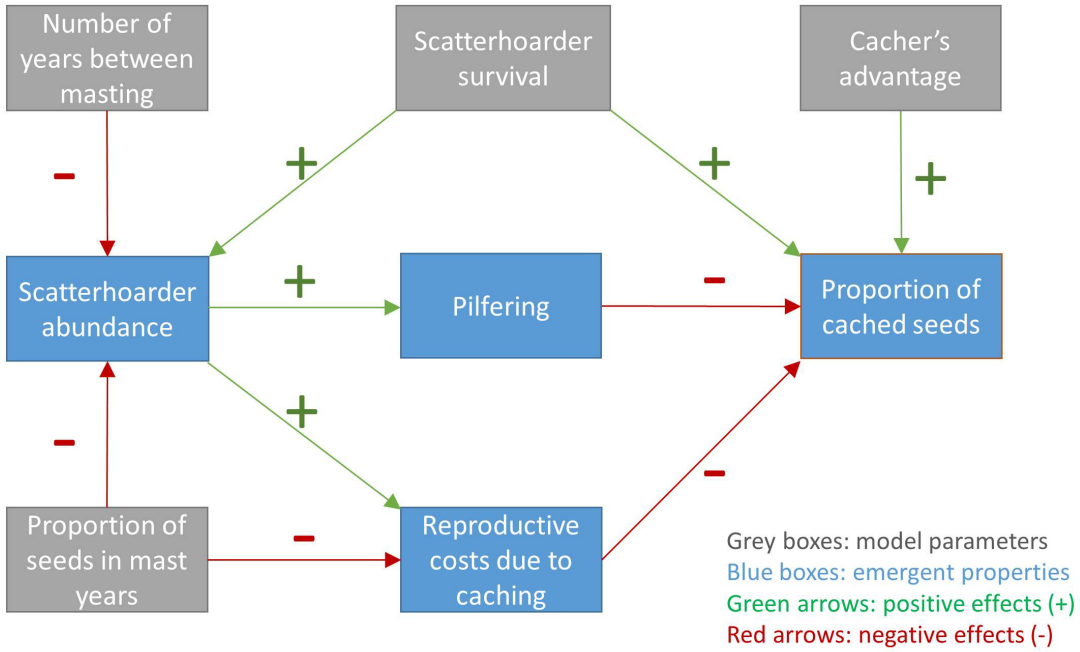

Fig. 1-5. *Relationships between input parameters (masting interval and intensity, expressed as the proportion of seeds produced during mast years, scatterhoarder survival, and cacher's advantage in cache recovery) and emergent properties of the model (scatterhoarder population density, the proportion of cached seeds that are pilfered, reproductive costs of caching, and the quantity of interest: the proportion of seeds cached by scatterhoarders).*

Discussion

The fact that masting causes strong fluctuations in populations of seed-eating animals has been well-known for a long time (Curran and Leighton 2000; Ostfeld and Keesing 2000; Bogdziewicz et al. 2016), yet the traditional research focus has been on how the satiation-starvation cycle reduces seed losses to pre- and post-dispersal seed predators. More recently, researchers suggested that seed masting is one of the means by which plants manipulate behavior of their dispersers (Vander Wall 2010). According to this reasoning, satiation of current energy needs induces granivores to cache seeds for future use (Vander Wall 2010).

Here we show that the effects of masting on population dynamics and caching behavior are mutually dependent. By decreasing the degree of pilfering, the satiation-starvation cycle due to more extreme seed masting events may promote the evolution and maintenance of seed caching behavior. Thus, the decrease in seed predation, increase in per capita scatterhorder satiation, and reduction in pilfering pressure may each represent an important pathway by which the scatterhorder satiation-starvation cycle induced by masting may improve plant recruitment (Fig. 1-5). These nuanced interactions between plant and seed predator emphasize the importance of studying the feedbacks between population dynamics and behavioral evolution.

Our results suggest that when seed production is highly variable, seed caching can evolve even when cache owners have little advantage over naive foragers in seed recovery (compare with Andersson and Krebs 1978). However, the mechanism that we describe is not mutually exclusive with other evolutionary explanations of scatter-hoarding. It can promote this behavior in synergy with the cache owner's advantage (Andersson and Krebs 1978) and reciprocal pilferage (Vander Wall and Jenkins 2003).

The costs of cache loss to pilferers are reduced in our model because periods of intense seed production coincide with low densities of scatterhoarders – and thus few potential pilferers (see Dittel and Vander Wall 2018 for experimental data demonstrating that the magnitude of cache pilferage is determined by the abundance of scatterhoarders). When there is pronounced masting with relatively long intervals between masting events, densities of scatterhoarders entering the start of the next large masting event are low (Figs. 1-2 & 1-3). Consequently, individuals are able to collect enough seeds to satiate their reproductive needs. As the yearly fitness is determined by the geometric mean of their fitness across the seasons and this geometric mean decreases with variation (Lewontin & Cohen 1969; Gillespie 1977; Schreiber 2015), the benefits of reducing seasonal variation in fitness by increasing winter/spring reproduction (fueled by cached seeds) outweigh the diminishing returns of increasing reproduction in the fall (fueled by immediate seed consumption).

Our results make a prediction that plants dispersed by scatterhoarders should have high interannual variation of seed production (typically measured with coefficient of variation, CV) relatively to plants dispersed by other means. This appears to be the case, at least when plants dispersed by scatterhoarders (synzoochorously) are compared to plants dispersed by frugivores (endozoochorously) (Herrera et al. 1998, Kelly and Sork 2002, Pearse et al. 2020). When explaining this pattern, researchers emphasized contrasting selective pressures acting on these groups of plants. Avoiding the risk of satiating frugivores was suggested as a factor that stabilizes seed production in plants dispersed endozoochorously. On the other hand, variable seed production in synzoochorous plants was interpreted as an adaptation that enabled reducing seed mortality caused by animals that act as seed predators and only incidentally disperse seeds (Herrera et al. 1998). However, we suggest that the high CV of plants dispersed by scatterhoarders can also be linked to the caching behavior of scatterhoarders (see also Lichti et al. 2020 for a model exploring the connection between caching behavior and seed trait evolution).

If, as our simulations suggest, masting intensity and mast interval are important for seed caching, then changes in plant masting patterns might affect the dynamics of seed caching, and therefore also the recruitment in plant populations. Our model is loosely based on the European beech – *Apodemus* mice system (Jensen 1982; Zwolak et al. 2016). Several studies have suggested that the European beech shows more frequent masting in recent years, probably due to global warming (Kantorowicz 2000; Schmidt 2006; Övergaard et al. 2007; Bogdziewicz et al. 2020). This could shift the beech-rodent interactions towards antagonism, with higher rodent abundances (predicted also by Imholt et al. 2013), more seed consumed and fewer cached (recall that caching declines with more frequent masting: Fig. 1-3). On the other hand, a recent meta-analysis of global data suggests that masting has become more pronounced (Pearse et al. 2017). Such a change could make seed caching more profitable for granivores (higher intensity of masting promotes caching: Fig. 1-2). However, extreme interannual variation in seed crops might lead to a decline and even extinction in granivore populations, due to the difficulty in tracking resource levels.

Moreover, any environmental change that affects scatterhoarder population dynamics could alter caching behavior and, thereby, impact seed mortality. For example, we found that increased scatterhoarder survivorship during the winter or summer may select against caching behavior by increasing population densities entering the masting years (Fig. 1-4). Thus, changes in winter or summer conditions that are favorable for mice could harm seedling recruitment both directly by increasing seed predation and indirectly by discouraging seed caching. In contrast, autumn conditions that are favorable for mice are likely to improve seedling recruitment because increased fall survivorship of scatterhoarders selects for more caching.

Just like every model, the one presented here simplifies reality. For example, in many ecosystems different masting species co-occur. Such species often mast synchronously due to shared climatic drivers (Curran et al. 1999; Kelly and Sork 2002; Schauber et al. 2002; Shibata et al. 2002; Bogdziewicz et al. 2018). If seed production is synchronous, the consequences for scatterhoarders will be similar to masting by one tree species. However, if masting is asynchronous, its outcome might be similar to reducing masting interval (Fig. 1-3d), i.e. the selective pressure to cache seeds will be weaker.

Furthermore, populations of scatterhoarders that have relatively high survival and low reproduction (e.g. corvids) might not fluctuate in response to masting as strongly as do populations of more productive scatterhoarders, such as chipmunks (Bergeron et al. 2011), squirrels (McShea 2000; Selonen et al. 2015), or mice (Pucek et al. 1993, Wolff 1996; Falls et al. 2007). However, even in species such as corvids, masting can affect the benefits of caching through similar mechanisms, i.e. reduced risk of interspecific seed pilferage (due to decreased abundance of sympatric rodents: e.g. Thayer and Vander Wall 2005) and lower marginal reproductive costs of caching.

Examining ultimate causes and ecological determinants of caching behavior will help to understand former selective pressures on synzoochorous plants, current dynamics of seed dispersal, and future alterations in seed dispersal patterns caused by global changes. Our study provides a step in this direction and suggests several promising avenues for prospective research. For example, future work should address the evolution of caching

reaction norms instead of the simple threshold for caching considered here. Additionally, evolution of caching strategies could be different when individual variation in personalities or, more generally, phenotypes of seed dispersing animals (Zwolak 2018, Zwolak and Sih 2020) is taken into account. Finally, future studies could examine these interaction from the plant perspective, for example by determining masting patterns that maximize seedling recruitment.

Data accessibility: The code for the main functions of the model, model results, and sensitivity analyses is included as Supplementary Data.

Authors' contributions. RZ and SS conceived the study. SS and DC developed and analyzed the model with feedback from RZ and AS. RZ wrote the first draft of the manuscript. All authors critically revised the draft and approved the final version of the article.

Competing interests. We declare we have no competing interests.

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Evolutionary history mediates population response to rapid environmental change through within-generational and transgenerational plasticity

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Abstract

Rapid environmental change is affecting many organisms; some are coping well but many species are in decline. A key mechanism for facilitating success following environmental change is phenotypic plasticity. Organisms use cues to respond phenotypically to environmental conditions; many incorporate recent information (within-generation plasticity) and information from previous generations (transgenerational plasticity). We extend an existing evolutionary model where organisms utilize within-generational plasticity, transgenerational plasticity, rapid evolution, and bet-hedging. We show how, when rapid evolution of plasticity is not possible, the effect of environmental change (altering the environment mean, variance, or autocorrelation, or cue reliability) on population growth rate depends on selection for within-generation plasticity and transgenerational plasticity under historical environmental conditions. We then evaluate the predictions that populations adapted to highly variable environments, or with greater within-generational plasticity, are more likely to successfully respond to environmental change. We identify when these predictions fail, and show environmental change is most detrimental when previously reliable cues become unreliable. When multiple cues become unreliable, environmental change can cause deleterious effects regardless of the population's evolutionary history. Overall, this work provides a general framework for understanding the role of plasticity in population responses to rapid environmental change.

Introduction

Most populations are facing rapid environmental change (e.g., habitat loss/fragmentation, exotic species, harvesting, pollutants, climate change; Wong and Candolin 2015). Some cope poorly and decline, while others fare so well that they become invasive or urbanized pests (O'Brien and Leichenko 2003; Sih 2013; Wilson et al. 2020). A major issue is understanding this variation in ability to cope with environmental change. How well organisms respond often depends on how well they adjust their phenotypes (e.g., behavioral type, morphology, physiology, and life history; Charmantier et al. 2008; Merila and Hendry 2014; Nicotra et al. 2010) to novel environmental situations. Because phenotypic strategies for living in historically variable environments may also allow species to persist after rapid environmental change (Candolin and Wong 2012; Donelan et al. 2020; Hendry et al. 2008; Sih 2013; Sih et al. 2011), the environment in which a population evolves will directly impact its response to change. Adaptive responses to variable environments include adaptive tracking (rapid genetic evolution), phenotypic plasticity, and bet hedging (Botero et al. 2015; Crowley et al. 2016). These adaptations clearly differ in their speed of response to change. Evolution, even when "rapid", is still substantially slower than pre-existing phenotypic plasticity. Often, plasticity is necessary to provide a cushion against extinction, allowing populations time to adapt to their new environment (Chevin et al. 2013; Chevin and Lande 2015).

Although phenotypic plasticity has traditionally been studied as the effect of an individual's own experiences on its phenotype (its within-generational plasticity, WGP), a recent area of high interest is transgenerational plasticity (TGP) – the effects of previous generations' experiences (in particular, parents') on the phenotypes expressed by focal individuals (offspring; Bonduriansky and Day 2009; Salinas et al. 2013). While it has long been known that experiences can alter parental behaviors that influence offspring phenotypes, such as parental investment (including parental care; Trivers 1972) and parental effects on offspring environments (e.g., via oviposition or other habitat choice, niche construction; Odling-Smee et al. 1996), more recent work has focused on less "visible" genetic and physiological mechanisms (e.g., epigenetic carryovers, yolk hormone, or

microbiota effects; Bale 2015; Bell and Hellmann 2019; Herman and Sultan 2016).

Transgenerational plasticity is particularly interesting in the context of adaptation to environmental variability because it is a response of "intermediate" speed, occurring faster than rapid evolution but more slowly than within-generational plasticity. While numerous empirical studies have revealed substantial variation in the strength of WGP and TGP (Auge et al. 2017; Luquet and Tariel 2016; Sultan et al. 2009; Wadgymar et al. 2018; Walsh et al. 2015), few have explicitly tested predictions on why we might expect WGP or TGP to be strong or weak in any given system, and those that have paint the role of TGP as weak or highly context dependent (Donelson et al. 2018; Uller et al. 2013). Though some papers have quantified the relative importance of adaptive tracking, plasticity and bet-hedging in specific systems (Colicchio and Herman 2020; Furness et al. 2015; Simons 2014), a key relatively unmet need is for a conceptual framework to more generally predict and explain when each of these mechanisms is expected to contribute to a population's response to rapid environmental change (Donelan et al. 2020).

We contend that this conceptual framework can be found at the intersection of two existing bodies of theory. The first consists of approaches that predict the relative effects of environmental change on individuals or species depending on the evolutionary mismatch between their previously adaptive traits and the novel, post-change conditions (Sih 2013; Sih et al. 2011, 2016). Organisms will perform poorly if the new conditions are mismatched with responses that were adaptive in their evolutionary past. A key assumption is that the species' initial plastic response to environmental change, before they have time to evolve, is critically important. This line of thinking predicts that organisms that evolved in variable, unpredictable conditions or generalists that are more flexible (plastic) should respond better to environmental change (Blois et al. 2013; Sol et al. 2016). Several recent models have explicitly applied this approach to explain variation in responses to: novel environmental variation per se (Botero et al. 2015; Crowley et al. 2016), exotic predators (Ehlman et al. 2019), novel stimuli that are safe but appear dangerous (e.g., ecotourists; Trimmer et al. 2017), habitat fragmentation (Crowley et al. 2019), and to explain life history shifts in phenology in response to climate change (McNamara et al. 2011).

The second body of relevant theory explains rapid evolution, WGP, TGP and bet hedging as components of an adaptive system for coping with environmental variability. This system uses direct and indirect environmental information to predict the optimal phenotype as it varies with the environment through time. Several recent models have addressed how patterns of environmental variation shape which combinations of components yield the best predictions and therefore represent evolutionary optima (Botero et al. 2015; Dall et al. 2015; English et al. 2015; Kuijper and Hoyle 2015; Leimar and McNamara 2015; McNamara et al. 2016; Shea et al. 2011). In particular, McNamara et al. (2016) examine the full range of components- rapid evolution, WGP, multiple kinds of TGP, and bet hedging- within a single modeling framework.

Using an extension of the McNamara et al. (2016) model, we identify how environmental changes can cause a previously adaptive strategy to become maladaptive and quantify how much this maladaptation can reduce population growth rate. Because the McNamara et al. (2016) model was not formulated with environmental change in mind, we extend it to allow for changes in the mean environment (e.g., change in average temperature, or predation risk) and for systematic biases in an organism's perception of the environment (e.g., population-wide tendencies for prey to misgauge predation risk), thereby accounting for two common types of environmental change. We examine how key parameters before environmental change determine the evolution of a population's reliance on rapid evolution, TGP, WGP, or bet hedging. We then explore how different forms of change affect population growth. The goal of these analyses is to generate concrete predictions about how the environment in which a population evolved influences its response to environmental change.

Conceptual Framework

The fundamental challenge of living in a variable environment is that an organism is uncertain about future environmental conditions and therefore does not know which phenotypes will yield the greatest reproductive success. Organisms can improve their success by utilizing cues that

convey information about the current and past environments. The pathways by which the cues can influence traits are numerous and include sensory and cognitive mechanisms (Dukas and Ratcliffe 2009; Shettleworth 2010), and biochemical, hormonal, and neurological mechanisms including DNA methylation or demethylation and histone modifications (Bale 2015; Fusco and Minelli 2010; Norouzitallab et al. 2019). We refer to these pathways, in aggregate, as the cue integration system of an organism. Following McNamara et al. (2016) we divide the information pathways into two broad classes: detection-based pathways and selection-based pathways. Detection-based pathways convey information gathered by directly experiencing an environment. Selection-based pathways convey environmental information through the transmission from generation to generation of heritable elements (either genetic or non-genetic) that influence phenotype. Due to selection, the relative frequency of an element in one generation contains information about the fitness of individuals with that element in previous generations' environments (Dall et al. 2015).

Environmental cues are not perfectly accurate; we consider three potential sources of error. First, cues may have fixed biases (cue bias) when environmental indicators are unreliable or are utilized incorrectly by the cue integration system. Prey that do not recognize a novel predator would have a fixed bias in their perception of predation risk. Second, cues may experience random error that affects the entire population (population-level cue error) when environmental indicators vary from generation to generation in their ability to predict the actual environmental conditions. Early snowmelt during one winter may predict a warm spring favorable to early flowering while during another it may be followed by a lethal cold snap. Third, individuals may randomly experience different cues in the same environment (individual-level cue error) due to local differences in environment indicators, individual differences in perception, or inaccuracy in the information pathways coordinating phenotypic development. McNamara et al. (2016) examine only individual-level cue error. Because the selection-based cues are elements inherited from an individual's parents we assume only individual-level error in transmission, while detection-based cues are subject to individual-level error, population-level error, and cue bias because they convey information gathered directly from the environment.

Following McNamara et al. (2016), we model an organism's cue integration system as a set of weights assigned to each of four information channels (figure 2-1), two detection-based and two selection-based respectively: (i) a within-generational channel that transmits environmental cues experienced by an organism during development or as a juvenile (juvenile cue), (ii) a transgenerational channel that transmits environmental information experienced by a parent to its offspring (maternal environment cue), (iii) a second transgenerational channel that transmits the parent's phenotype (including plastic trait expression) to the offspring through non-genetic inheritance (maternal phenotype cue), and (iv) genetic inheritance, which transmits alleles of fixed phenotypic effect from parent to offspring (genetic cue). Although an organism's fitness is determined by many traits, for simplicity, we assume that one trait (e.g., germination time) or one correlated suite of traits (e.g., antipredator behavior) has a disproportionately large effect on fitness. In addition to the four information channels, the cue integration system also places weight on a randomization component (diversified bet-hedging) and a "reference phenotype" representing the phenotype that would result if an individual ignored all cues. An effective cue integration system is one that weights these components such that organisms, on average, can match their phenotypes to a randomly fluctuating phenotypic optimum.

We assume that a population's phenotypic distribution changes over three distinct time scales. The shortest time scale occurs over one generation: the environment changes, adults reproduce, cues are transmitted to or detected by the offspring, and the offspring determine their phenotype based on these cues. Over the intermediate time scale, enough generations have passed that we assume the environmental regime and the population's phenotypic dynamics have reached a stationary state. We then consider the long-run per-capita growth rate of the population over that joint environmental-genotypic-phenotypic distribution. While the genetic cue exhibits rapid evolution (provided the phenotype is not genetically canalized), we assume that the cue integration system itself evolves sufficiently slowly that it can be considered fixed and monomorphic over this timescale. The longest time scale is that over which the cue integration system evolves. We do not model the evolutionary dynamics of the cue integration itself and merely assume that

the cue weights and reference phenotype evolve to maximize long-run per-capita growth rate within a stable environmental regime. We also assume that the cue integration system evolves to compensate for persistent cue biases (i.e., these biases are set to zero).

The framework for studying environmental change assumes that before environmental change occurs, the environmental regime is stationary and the population evolved the optimal cue integration system for that regime (i.e., we look at the system over the longest timescale). This environmental regime is characterized by its long-term mean, its long-term variance, its autocorrelation between years, and the reliability of the information channels used to determine phenotype. We consider an abrupt change in any combination of these parameters. We then look at the new growth rate of the population over the intermediate time scale; the environment and phenotypic distribution reaches its new stationary state, but the population's dynamics are still mediated by a cue integration system adapted to the old environment (Sih et al. 2016). The separation of timescales between environmental change and the evolution of plasticity, along with our focus on the intermediate timescale, separates our approach from models that assume rapid evolution of plasticity and focus on the shortest timescale (e.g. Kuijper and Hoyle 2015; Lande 2009).

Examples to which our conceptual framework applies include responses of prey to shifts in predation risk and plant germination to shifts in climate. First consider the illustrative scenario where the environmental variable in question is predation risk and an organism's morphology (Agrawal et al. 1999; Donelan and Trussell 2018; Luquet and Tariel 2016; Walsh et al. 2015) or personality (boldness/cautiousness; Reale et al. 2010; Sih et al. 2004; Sih and Del Giudice 2012) are traits that influence the organism's fitness (Moiron et al. 2020; Sih et al. 2003). The introduction of a novel predator increases the mean predation risk and causes persistent inaccuracies in the organism's perception of predation risk (the new predator may not be perceived as dangerous; e.g., lionfish Diller et al. 2014; Medina et al. 2014). The fate of the population will then be impacted by the historical accuracy of detection-based cues and whether the historical environment was safe or dangerous.

A second illustrative scenario concerns seed germination, which often has within-generational and transgenerational components associated with temperature or precipitation cues (Galloway and Etterson 2007; Herman and Sultan 2016). These cues impact the timing of germination and, consequently, can substantially impact fitness. Therefore, historical patterns of temperature and rainfall, and the reliability of past climatic cues, should play substantial roles in a population's phenological response to climate change (Cuello et al. 2019; Herman and Sultan 2011; Whittle et al. 2009). Optimal germination time may be affected by changes in the mean temperature or rainfall (Milbau et al. 2009; Petru and Tielboerger 2008), increased climatic variation (Gremer and Venable 2014), or a decoupling of environmental cues from future conditions (increasing the population-level cue error; Donohue et al. 2010).

Model and Methods

The Model

Following McNamara et al. (2016), we consider a population of asexual organisms with discrete, non-overlapping generations. We assume that in each environment there is a single phenotype with the highest fitness, the optimal phenotype θ. Environmental fluctuations from generation to generation result in fluctuations in the optimal phenotype. Organisms use the environmental cues to adjust their phenotypes toward the optimal phenotype.

Environmental Dynamics.

We model the dynamics of the environment $\theta(t)$ as a first-order autoregressive process with autocorrelation $\lambda = Corr(\theta(t), \theta(t+1))$ and mean μ_θ. Fluctuations $\phi_\theta(t)$ in year t are normally distributed with a zero mean and variance σ_θ^2 and are independent of the past. We call σ_θ^2 the year to year environmental variance. The environment process is then written as:

$$\theta(t) = \mu_\theta + \lambda(\theta(t-1) - \mu_\theta) + \varphi_\theta(t). \tag{2-1}$$

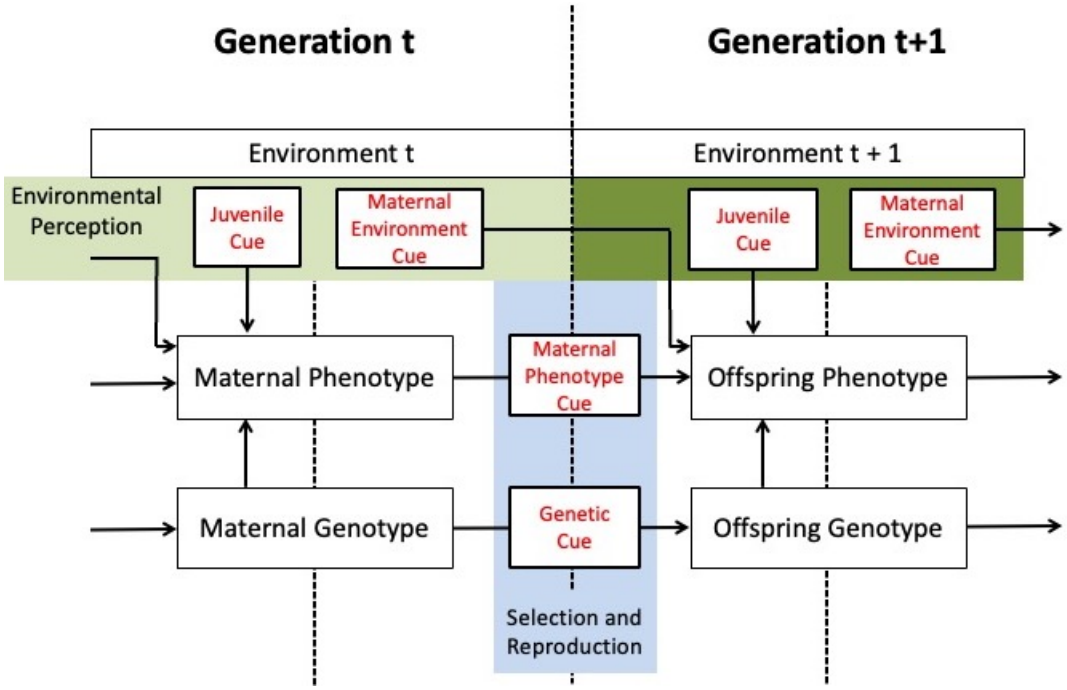

Figure 2-1: An individual's phenotype is determined by a combination of four environmental cues, in red, and the weights assigned to them. The environment is constant within a given generation, but can change between generations. Information about the environment perceived by an individual early in life influences its own phenotype (juvenile cue); this is within-generation plasticity (WGP). Information perceived later as an adult can be passed on through reproduction to influence its offspring's phenotype (maternal environment cue). An individual's genes (genetic cue) are passed on and influence offspring phenotypes. An individual's phenotype may also have a non-genetic direct influence on offspring phenotypes (maternal phenotype cue). Effects of maternal environment and phenotype cues on offspring are forms of transgenerational plasticity (TGP). Phenotypes and genotypes within the population are subject to selection each generation.

At stationarity, the variance is $V_\theta = \frac{\sigma_\theta^2}{1-\lambda^2}$.

Detection-based Cues

The cue integration system makes use of two detection-based cues, the juvenile and maternal environment cues, which occur through direct observations of the environment θ. The juvenile cue, c_J, represents information about the current environment, $\theta(t)$, that an individual gathers during early development. The maternal environment cue $c_{ME}(t)$ (the adult cue in McNamara et al. 2016) is derived from the environment $\theta(t-1)$ experienced by the mother as a mature adult. It does not affect her phenotype m', which is set during development. This maternal environment may influence the mother's reproductive choices that in turn affect offspring development. For example, resource availability or predation risk during the mother's adult life (regardless of her early-life conditions) may influence brood size and location, resource provisioning, or parental care (Creighton 2005; Gibbs and Van Dyck 2009; Westneat et al. 2015). This cue may not necessarily interact with her genotype, z' or phenotype m' and could in theory vary between broods of offspring as a form of contextual plasticity.

Detection-based cues are subject to individual-level errors, population-level errors, and fixed cue bias. The individual-level errors, ϵ_J^{ind} and ϵ_{ME}^{ind}, vary between individuals within a given generation and are normally distributed with zero mean and standard deviations σ_J^{ind} and σ_{ME}^{ind}, respectively. McNamara et al. (2016) only accounted for this source of error. The population-level errors, $\epsilon_J^{pop}(t)$ and $\epsilon_{ME}^{pop}(t)$, are shared by all individuals, but vary from generation to generation. They are normally distributed with zero mean and standard deviations σ_J^{pop} and σ_{ME}^{pop}, respectively. The cue biases (μ_J and μ_{ME}) are shared by all individuals and fixed across generations. The cue integration system is assumed to compensate for cue biases (i.e., they evolve to zero over the longest timescale). Relaxing this assumption results in discontinuities in the fitness surface (Supplement 2-1). The juvenile and maternal environment cues then equal

$$c_J = \theta(t) + \mu_J + \epsilon_J^{pop}(t) + \epsilon_J^{ind}$$

$$c_{ME} = \theta(t-1) + \mu_{ME} + \epsilon^{pop}_{ME}(t) + \epsilon^{ind}_{ME}.$$

Selection-based cues and randomization

In addition to the detection-based cues, individuals can make use of maternal phenotype and genetic cues. The maternal phenotype m' may be thought of as a genotype by environment interaction determined in early life based on the mother's juvenile environment $\theta(t-1)$ (e.g., irreversible plasticity). Phenotypic information can be passed to offspring, with individual-level transmission error ϵ^{ind}_{MP}, through epigenetic modifications, hormones in the womb or eggs, maternal provisioning, etc. For example, predation risk or access to resources during a mother's early life may alter her gene expression that in turn affects her morphology (higher defenses, altered body type) or her persistent behavioral response (personality) in ways that follow her throughout her lifetime (Bourdeau and Johansson 2012; Chapman et al. 2010; Relyea 2001) and induces similar phenotypic changes in her offspring (Agrawal et al. 1999; Donelan and Trussell 2015; Reddon 2012; Sobral et al. 2021). This maternal phenotype cue is distinguished from the maternal environment cue by its interaction with an individual's genotype (selection vs. detection), its susceptibility to only individual-level error, the life stage the mother experiences her inducing environment (early life vs adult life), and the reversibility of this induction (irreversible vs reversible or contextual).

The genetic cue z represents the transmission of environmental information through heritable genetic variation in the reaction norm elevation, with error due to mutation ϵ_{mut}. If recent past environments correctly 'predict' the current environment (i.e., if autocorrelation is high), recent selection-driven genetic change should produce phenotypes that are better matched to the current environment than the long-term mean phenotype, while if environments fluctuate randomly (low autocorrelation), the recent past is 'noise' that does not predict the current environment. If z' is the mother's genetic trait value and m' is the mother's phenotype, then the maternal phenotype cue c_{MP} (the maternal cue in McNamara et al. 2016) and the genetic cue z of an individual are

$$c_{MP} = m' + \epsilon^{ind}_{MP} \text{ and } z = z' + \epsilon_{mut}$$

where ϵ_{MP}^{ind} and ϵ_{mut} are normally distributed with zero mean and standard deviations of σ_{MP}^{ind} and σ_{mut} respectively.

The reaction norm elevation is modeled by a fixed component μ representing the population's long-term mean phenotype plus a variable component consisting of the genetic cue z scaled by the weight w_z. $w_z z$ represents the deviation of individual genotypes from the long-term mean μ. A large value of w_z allows allelic variation to strongly influence phenotype (i.e., relying heavily on genetic cues) thereby increasing the genetic heritability of the trait and promoting strong genetic responses to selection. A small value of w_z decouples genetic evolution from phenotypic determination by making the phenotype robust to variation in genetic background, a process known as genetic canalization (Waddington 1957; Wagner et al. 1997). Thus, w_z represents the degree to which the phenotype is genetically canalized or decanalized and the reference phenotype μ represents the phenotype that results if the trait is both environmentally canalized (i.e., no plasticity) and genetically canalized.

In population genetic terms, genetic canalization is the evolution of reduced mutational effects (mutations result in smaller deviations from the ancestral phenotypic state; Flatt 2005; Hansen 2006). Genetic canalization occurs through mechanisms such as modifier alleles or other epistatic interactions, functional redundancy among genes, or dominance (De Visser et al. 2003; Flatt 2005; Takahashi 2019). Differences in genetic canalization can be empirically quantified by comparing the mutational variances among different experimental lines or by measuring the change in the among-genotype component of phenotypic variance in response to a genetic perturbation (e.g., mutation introgression, or gene knockout or inhibition; Flatt 2005; Takahashi 2019; Takahashi 2017). Many populations exhibit genetic variation in fitness-related traits, so it is natural to question how often developmental processes allow complete genetic canalization. In Supplement 2-6 we examine our model under the assumption that complete genetic canalization is not possible.

The final factor governing an individual's phenotype is not a cue, but a normally-distributed randomization term ϵ_R with mean 0 and standard deviation σ_R. When individuals use randomization to determine their phenotype, the population exhibits diversified bet-hedging (Bull

1987).

The cue integration system

The phenotype x of an individual is given by a linear combination of the cues with the cue integration system consisting of the reference phenotype μ (the phenotype which would result if the individual ignored all cues) and a set of weights w_Z, w_J, w_{ME}, w_{MP}, w_R on the cues:

$$x = \mu + w_Z z + w_J(c_J - \mu) + w_{ME}(c_{ME} - \mu) + w_{MP}(c_{MP} - \mu) + w_R \epsilon_R. \tag{2-2}$$

A larger value of w_J means that organisms exhibit stronger within-generational plasticity, and larger values of w_{ME} and w_{MP} result in stronger transgenerational plasticity. $\mu + w_Z z$ is the elevation of the reaction norm when measured in the reference environment with the optimal phenotype μ. Over the longest timescale, μ is assumed to evolve so that it is equal to the μ_θ.

Individual Fitness and Population Growth Rate

Following McNamara et al. (2016), we assume that the optimal phenotype has a maximal log fitness r_max and that log fitness decreases quadratically with the distance of the phenotype x from the optimal phenotype θ:

$$R(x, \theta) = \exp\left(r_{max} - \frac{1}{2\omega}(x - \theta)^2\right)$$

where ω, the width of the fitness function, is always set to 1 (following McNamara et al. (2016)). For a given cue integration system, the distribution of phenotypes, genotypes, and environment approaches a stationary distribution. For a population and environment following this stationary distribution, let X be the phenotype of a randomly chosen individual and Θ a randomly chosen environmental state. Then the long-term per-capita population growth rate equals

$$r = \mathbb{E}[\log R(X, \Theta)].$$

Given sufficient time, the cue integration system $(\mu, w_Z, w_J, w_{ME}, w_{MP}, w_R)$ evolves to maximize r.

Methods

We explore evolution of cue weights prior to environmental change and the impacts of change on population growth rates using both analytical and numerical methods. We analytically derived the expression for per-capita population growth rate (Equations 2-3, 2-4, 2-6, and 2-7; derivation included in Supplement 2-1), which was used both for the pre- and post-change scenarios. We computed the pre-change cue weights that maximized population growth rate at stationarity by using the L-BFGS-B algorithm for bounded optimization, as implemented in the optim function of the base package of the R statistical programming language (Version 4.1.0; R Core Team 2021). We chose bounded optimization because convergence of the phenotypic variance to a unique equilibrium value is not guaranteed when $w_Z < 0$ or $w_{MP} <$ -1 (Supplement 2-1). w_Z was constrained to values between 0 and 10, w_J and w_{ME} between -10 and 10, w_{MP} between -1 and 10, and w_R between 0 and 10. In practice, optimal cue weights very rarely exceeded one and, with the exception of w_{MP}, were always positive. Ten randomly-chosen initial conditions were tested to ensure the absence of multiple local maxima; when different initial conditions led to different maxima, the global maximum was taken (this only occurred in a narrow range of parameter space where w_R transitioned from zero to positive).

We examined optimal cue weights across a range of pre-change environmental parameters. Environmental variance and population-level cue error are in the same units as the width of the fitness function (ω), which was set to one, so their ranges were selected to span from four-fold less than the ω (0.25) to four-fold more than ω (4). Because the optimal phenotypic variance is equal to the variance of the mismatch between the mean phenotype and the optimal phenotype minus ω^2 (Tufto 2015), this parameter range should be sufficient for bet-hedging to be observed. Autocorrelation was allowed to vary between 0 and 0.9. In order to keep the parameters in a range that allows for juvenile, maternal environment, and maternal phenotype cues, when otherwise unspecified $\lambda = 0.7$ and $\sigma_\theta^2 = 1$. When varying autocorrelation, we also allowed the environmental variance to vary in order to hold constant the stationary variance of the

environmental process ($Var(\theta)$). This is equivalent to parameterizing the environmental process in terms of autocorrelation and stationary variance. The default values for individual-level cue error, population-level cue error, and mutation were chosen to be small (with respect to $\omega = 1$): $\mu_\theta = 0$, $\sigma^2_{mut} = 0.25$, $(\sigma^{ind}_J)^2 = 0.25$, $(\sigma^{ind}_{ME})^2 = 0.25$, $(\sigma^{ind}_{MP})^2 = 0.25$, $(\sigma^{pop}_J)^2 = 0.25$, $(\sigma^{pop}_{ME})^2 = 0.25$.

For post-change results, we examined the effects of four environmental changes on the population's long-run per-capita growth rate. The changes were (i) the mean environment increases by 1 (i.e., a change in the mean environment results in 1 unit change in the optimal phenotype, not to be confused with an increase in environment quality), (ii) the juvenile cue bias increases by 1 (i.e., juveniles get worse at evaluating the environment), (iii) the environmental variance increases by 0.4, and (iv) the autocorrelation decreases by 0.1. The changes in the mean environment and juvenile cue bias were chosen to equal the width of the fitness function ω. For environmental variance and autocorrelation, these changes are 10% of the total parameter range we examined. The directions of the environmental changes were chosen to make the environment less predictable. When a change (such as an increase in autocorrelation) makes the environment more predictable, per-capita growth rate always increases. We chose to represent the environmental changes as absolute changes rather than proportional changes because the growth rate is a linear function of many of the environmental parameters.

In order to determine the robustness of our main results to parameter choice, we conducted global sensitivity analyses using Latin hypercube sampling to randomly sample parameter values in the ranges $\lambda \in (0,1)$, $\sigma^2 \in (0,4)$, $\sigma^2_{mut} \in (0,4)$, $(\sigma^{ind}_J)^2 \in (0,4)$, $(\sigma^{ind}_{ME})^2 \in (0,4)$, $(\sigma^{ind}_{MP})^2 \in (0,4)$, $(\sigma^{pop}_J)^2 \in (0,4)$, and $(\sigma^{pop}_{ME})^2 \in (0,4)$. For each parameter combination, we calculated the set of optimal cue weights, as well as the reduction in population growth rate (fitness loss) associated with the environmental changes discussed in the main paper. The specifics of these analyses, as well as their results, are contained in Supplements 2-4 and 2-5.

Results

Long-term population growth

In Supplement 2-1, we show that the long-term per-capita growth rate, r, of the population can be decomposed into five terms

$$\mathbb{E}[r(w_Z, w_J, w_{ME}, w_{MP}, w_R)] = r_{max} - \mathcal{P} - \mathcal{F} - \mathcal{E} - \mathcal{S}. \tag{2-3}$$

The first of these terms is the maximum per-capita growth rate, and the remaining four we call the phenotypic variance load $\mathcal{P}$ (sensu Lynch and Lande 1993; Burger and Lynch 1995), the environmental fluctuation load $\mathcal{F}$ (sensu Ezard et al. 2014), the population-level error load $\mathcal{E}$, and the environmental shift load $\mathcal{S}$, respectively. The maximum growth rate r_{max} is the per-capita growth rate attained if all individuals perfectly match the optimal phenotype. The remaining terms (referred to as fitness loads) represent factors that reduce the growth rate below its maximal value. $\mathcal{F}$, $\mathcal{E}$, and $\mathcal{S}$ constitute a decomposition of the evolutionary lag load, which results when the mean phenotype of the population differs from the changing phenotypic optimum (Maynard Smith 1976). $\mathcal{P}$ and $\mathcal{F}$ were described for this model in McNamara et al. (2016), while $\mathcal{E}$ and $\mathcal{S}$ are new. We summarize the first-order (linear) relationships between environment, cue weights, and fitness loads in figure 2-2, which provides a useful conceptual framework for interpreting our results.

The phenotypic variance load $\mathcal{P}$ is the reduction in growth rate (fitness loss) due to deviation of individuals from the mean phenotype and is given by $\frac{1}{2}\ln(1+\sigma_X^2)$. In general, the phenotypic variance σ_X^2 must be solved numerically, but when $w_Z = 0$, the phenotypic variance is given by $\sigma_X^2 = \frac{1}{2}(\eta^2 + w_{MP}^2 - 1 + \sqrt{(\eta^2 + w_{MP}^2 + 1)^2 - 4w_{MP}^2})$, where $\eta^2 = w_J^2(\sigma_J^{ind})^2 + w_{ME}^2(\sigma_{ME}^{ind})^2 + w_{MP}^2(\sigma_{MP}^{ind})^2 + w_R^2$ (see Supplements 2-1 and 2-3). Phenotypic variance increases with all cue weights, with the magnitude of the increase for each cue scaled by its individual-level error. This load, therefore, selects for lower weights of cues with high individual-level error and for lower values of randomization.

The environmental fluctuation load $\mathcal{F}$ (or the environmental stochasticity load sensu Lynch and

Lande 1993) represents the reduction in growth rate due to imperfect tracking of environmental variation at the population level, discounting the effect of cue error. This term is given by

$$\mathcal{F} = V_\theta \frac{(1+G\lambda)(1-w_{_J})^2 + (1+G\lambda)(w_{_{ME}}+w_{_{MP}})^2 - (\lambda + G(1+\lambda^2)1 - \lambda B)(1-w_{_J})(w_{_{ME}}+w_{_{MP}})}{2(1-\frac{L}{2}(1-B)-B^2)(1+\sigma_X^2)} \tag{2-4}$$

where $G = \frac{-L+2B(1-\lambda)}{(1-\lambda)(1-\lambda B)+\lambda L}$, $B = \frac{w_{_{MP}}}{1+\sigma_X^2}$, and $L = \frac{w_{_Z}\sigma_{mut}}{\sqrt{1+\sigma_X^2}}$. The parameters determining the magnitude of this load are the environmental variance and autocorrelation, with the load increasing linearly with environmental variance.

This term equals zero when $w_{_J}$ equals 1 and all other cue weights are 0, reflecting the fact that in the absence of cue error the juvenile cue allows perfect prediction of the environment, and that relying on other cues is a compromise strategy to account for imperfect information. McNamara et al. (2016) prove that when $w_{_Z} = w_{_{MP}} = 0$, the optimal values of $w_{_{ME}}$ and $w_{_J}$ obey the inequality $w_{_{ME}} < \lambda(1-w_{_J})$. Indeed, when $w_{_Z}$ and $w_{_{MP}}$ are small, we can write

$$\mathcal{F} \approx V_\theta \frac{(1-w_{_J})^2 + [(w_{_{ME}}+w_{_{MP}}) - \lambda(1-w_{_J})](w_{_{ME}}+w_{_{MP}})}{2(1+\sigma_X^2)}. \tag{2-5}$$

Thus, if σ_X^2 is approximately constant, the environmental fluctuation load selects for higher maternal cue weights when autocorrelation is high and the juvenile cue weight is low. Note, however, that the maternal phenotype cue affects the fitness function through B while the maternal environment cue does not. We examine the additional complexities induced by the genetic cue, the maternal phenotype cue, and phenotypic variance in Supplement 2-3.

The population-level error load, $\mathcal{E}$, is the fitness cost of utilizing cues with high population-level errors, $(\sigma_{_J}^{pop})^2$ and $(\sigma_{_{ME}}^{pop})^2$, and equals

$$\mathcal{E} = \frac{w_{_J}^2(\sigma_{_J}^{pop})^2 + w_{_{ME}}^2(\sigma_{_{ME}}^{pop})^2}{2(1-\frac{L}{2}(1-B)-B^2)(1+\sigma_X^2)}. \tag{2-6}$$

Equation 2-6 shows that unlike individual-level cue error, which affects growth rate through phenotypic variance, population-level cue error has a clear linear effect on growth rate. This load selects for lower weights of detection-based cues with high population-level error.

The environmental shift load, $\mathcal{S}$, is the fitness load due to the average mismatch between the mean and optimal phenotype (as opposed to $\mathcal{F}$ and $\mathcal{E}$, which are due to the mismatch variance).

This mismatch may arise due to biased cues or a shift in the mean environment. When the genetic cue is present ($w_z > 0$), rapid evolution in the elevation of the reaction norm (the genetic cue z) can compensate for this mismatch so long as the mismatch does not change over time. The load term equals

$$\mathcal{S} = \frac{(1 - w_J - w_{ME} - w_{MP})(\mu_\theta - \mu) - w_J\mu_J - w_{ME}\mu_{ME}}{2(1-B)^2(1+\sigma_X^2)} \tag{2-7}$$

when $w_Z = 0$ and $\mathcal{S} = 0$ otherwise. This term selects for decreased cue bias and for concordance between the reference phenotype μ and the average optimal phenotype μ_θ. This term does not affect selection on the cue weights per se because it is equal to zero, independent of the cue weights, when μ, μ_J, or μ_A evolve to their optimal values of $\mu = \mu_\theta$ and $\mu_J = \mu_A = 0$. We treat μ, μ_J, or μ_A as under control of the cue integration system because when they are treated as fixed parameters the long-term growth rate becomes discontinuous and may lack a global maximum (Supplement 2-1). $\mathcal{F}$, $\mathcal{E}$, and $\mathcal{S}$ are all decreasing functions of phenotypic variance and therefore select for greater randomization.

Effects of Past Conditions on the Cue Integration System

The equations 2-3, 2-2, 2-4, and 2-6 show that the juvenile cue weight will depend on the balance between the phenotypic variance load $\mathcal{P}$ and the error load $\mathcal{E}$, which select for reduced cue weight, and the environmental stochasticity load $\mathcal{F}$, which selects for increased cue weight (up to $w_J = 1$). Biologically, this means that relying on the juvenile cue allows organisms to track environmental variation more closely, but makes them more vulnerable to cue error. This balance depends, in part, on the juvenile cue errors and the environmental variance, respectively. When the juvenile cue has been historically less accurate (e.g., higher population-level cue errors) our model predicts that organisms will rely less upon this cue (figures 2-3*A*,2-4*A*). When the environment has historically had high variance, there is a higher signal to noise ratio for the juvenile cue and organisms rely more heavily upon it (figures 2-3*B*,2-3*D*).

As with the juvenile cue, the phenotypic variance load $\mathcal{P}$ and the error load $\mathcal{E}$ select for lower

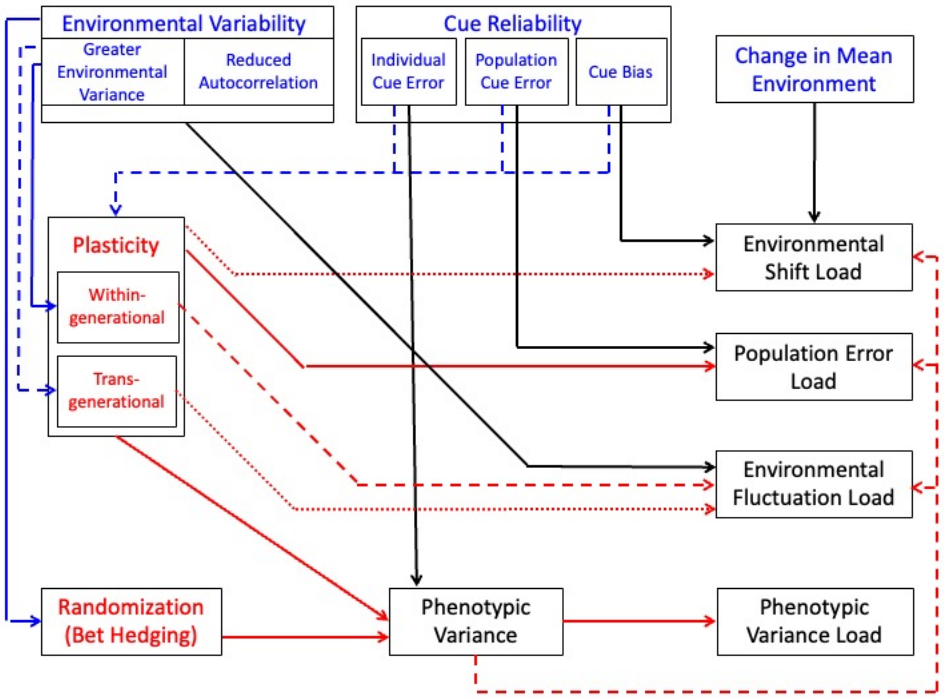

Figure 2-2: A qualitative schematic of interactions between environmental conditions, optimal cue weights, and fitness loads. The blue boxes at the top of the figure are environmental parameters. The red boxes to the left are the optimal cue weights. The black boxes to the right are the fitness loads. Solid arrows depict positive effects, dashed arrows depict negative effects, and the dotted arrows depict context-dependent effects. Blue arrows denote the effect of environmental parameters on cue weights, red arrows denote the effect of cue weights on fitness loads, and black arrows denote the effect of environmental parameters on fitness loads. The schematic does not show second order interactions, such as how the environmental shift load changes with simultaneous changes in mean environment and cue bias. The genetic cue is not shown because it is not utilized over the parameter range examined in our main results.

maternal environment cue weights while only the phenotypic variance load selects for lower maternal phenotype cue weights. If w_Z and w_{MP} are small, the environmental stochasticity load $\mathcal{F}$ selects for higher transgenerational cue weights only when autocorrelation is high and juvenile cue weight is low, and selects against the transgenerational cues otherwise (see equation 2-5). Our results support this approximation, showing that the transgenerational cue weights are higher when the juvenile cue is unreliable and the environment is stable (high autocorrelation, low environmental variance; compare 2-3*D* to 2-3*B* and 2-4*D* to 2-4*B*). The conditional relationship between the transgenerational cue weights and the environmental stochasticity load creates a negative association between within-generational and transgenerational plasticity (figure S2-6). Biologically, this implies that the transgenerational cues act as substitutes for the juvenile cue when they are reliable but the juvenile cue is not.

When both WGP and TGP are ineffective (population-level cue error is high and environment variance is high or autocorrelation is low), the population error $\mathcal{E}$, environment shift $\mathcal{S}$, and environmental stochasticity loads $\mathcal{F}$ are all high. Because greater phenotypic variation reduces these loads, greater weight on randomization (i.e., diversified bet hedging) is then evolutionarily favored (figures 2-3*B*, 2-4*B*; See Tufto 2015 for the formal mathematical conditions under which bet hedging is favored when plasticity is present).

Strikingly, the genetic cue is not utilized ($w_Z = 0$) over the entire parameter range considered in the main text. We show in Supplement 2-3 that the genetic cue and the maternal phenotype cue have opposing effects on the environmental fluctuation load (e.g., G tends to increase with w_{MP}, but decreases with w_Z), making it sub-optimal to simultaneously utilize both cues. We further show that utilizing the genetic cue decreases the optimal values of TGP via the fluctuation load $\mathcal{F}$. We also find that genotype-environment covariance decreases with plastic cue weights (McNamara et al. 2016), extending the results of Tufto (2015) to include transgenerational plasticity (though the results for genotype-environment correlation are more complicated). As a result, the genetic cue is only utilized when autocorrelation is high, environmental variance is low, and both WGP and TGP are inaccurate (figure S2-5*B*; McNamara et al. 2016). See figure S2-4 for optimal cue

weights patterns under these conditions.

Total plasticity (i.e., the sum of the juvenile, maternal environment, and maternal phenotype cue weights) decreases slightly with increased juvenile cue error (compare figure 2-3*B* to 2-4*D*), but increases substantially with autocorrelation (figure 2-4*B*,*D*). This is likely because when autocorrelation is low, TGP and rapid evolution are not favored, leaving WGP as the only response to environmental variation. When environmental variance is low, the sum of cue weights decreases dramatically because the environmental stochasticity load is small relative to the error loads; plasticity is less necessary because the environment is less variable. Thus, the total plasticity can be low either when individuals do not need to respond to their environment or when both past and current environmental cues are unreliable.

As expected, the long-term per capita growth rate is lowest when none of the cues are reliable (figures 2-3*C*, 2-4C) because all of the fitness loads are high. However, when only one cue is reliable (e.g., either TGP when population-level juvenile cue error is high and environmental variation is low, or WGP in the opposite conditions), growth rates remain relatively high because individuals evolve to rely primarily on the reliable cue. The optimal growth rate for a given historical environment also serves as the maximal attainable growth rate for a population that finds itself in that environment post-change.

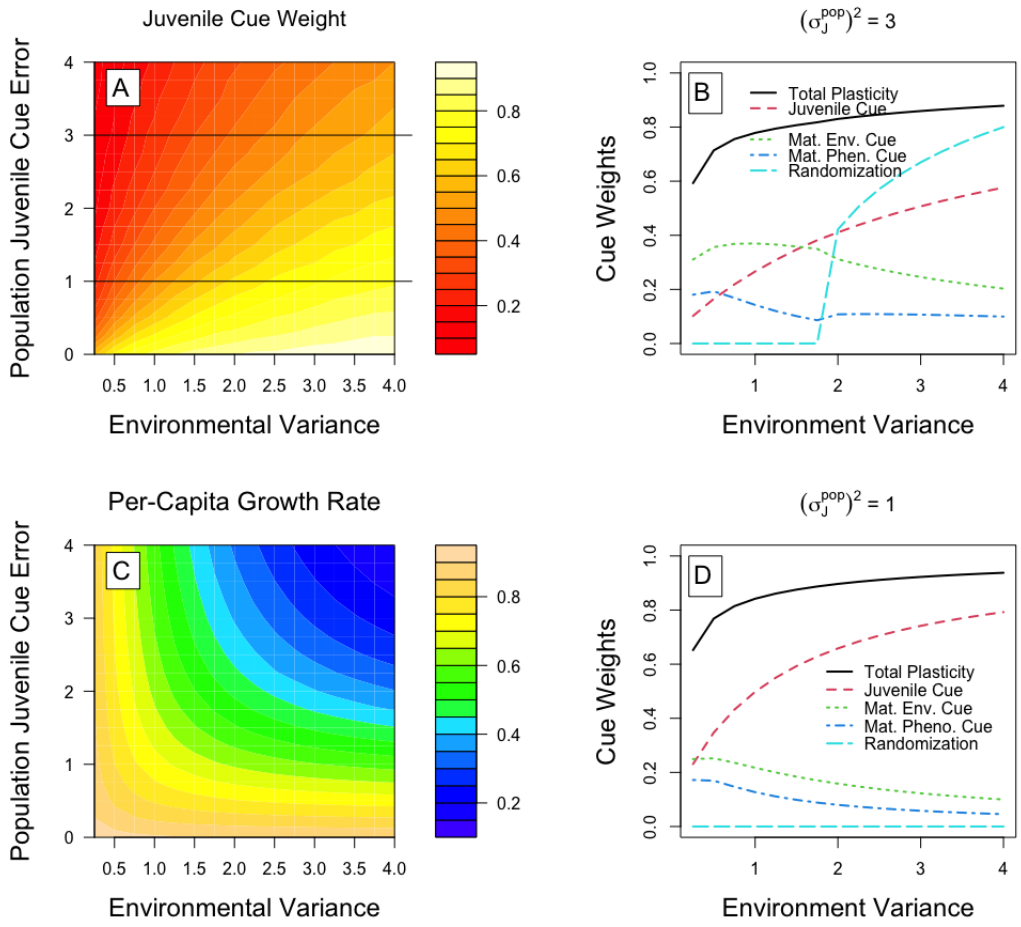

Figure 2-3: The effect of environmental variance and population-level juvenile cue error on optimal cue weights and per-capita growth rate. A) The optimal weight for the juvenile cue. The two horizontal lines denote cross-sections of parameter space corresponding to panels B and D. B) Optimal cue weights as a function of environmental variance when the population-level juvenile cue error $(\sigma_j^{pop})^2$ is equal to 3. C) Per-capita growth rate of a population with optimized cue weights as a function of environmental variance and population-level juvenile cue error. D) Optimal cue weights as a function of environmental variance when the population-level juvenile cue error is equal to 1. Remaining parameters: $r = 1$, $\lambda = 0.7$, $\sigma_{mut}^2 = 0.25$, $(\sigma_{ME}^{ind})^2 = (\sigma_{MP}^{ind})^2 = (\sigma_j^{ind})^2 = (\sigma_{ME}^{pop})^2 = 0.25$.

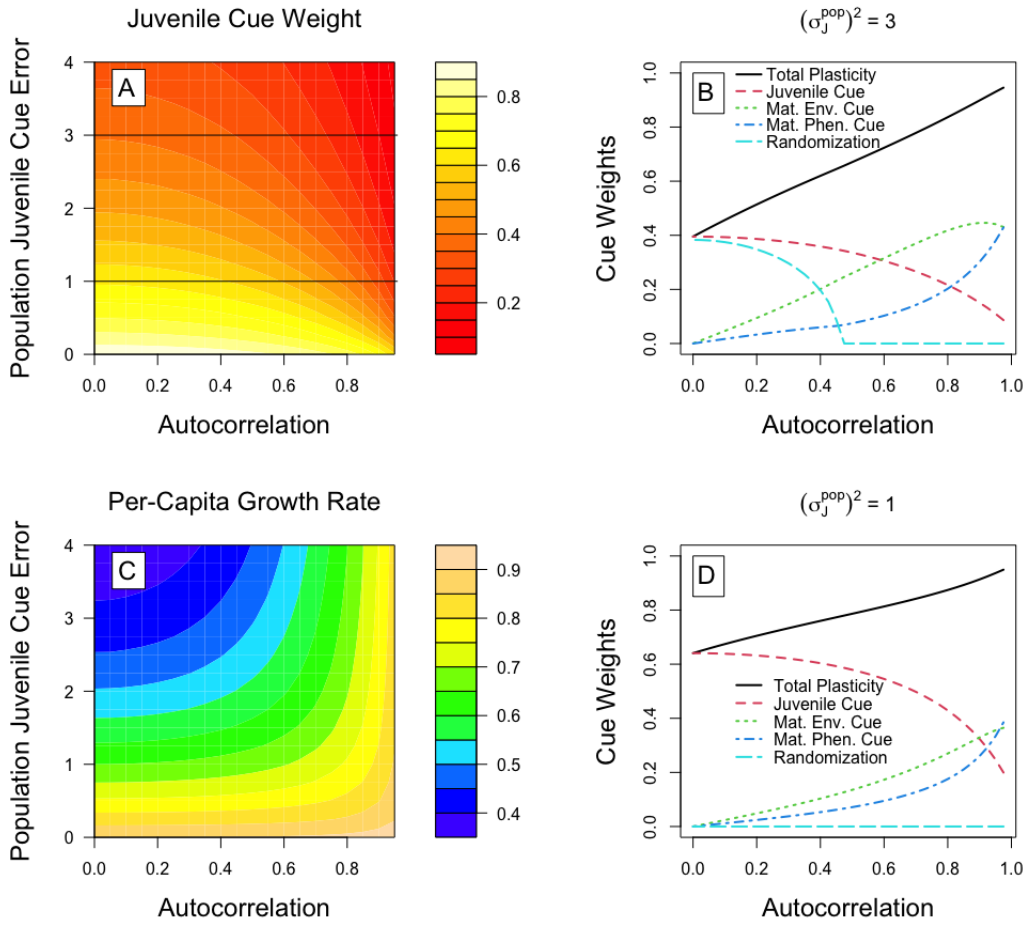

Figure 2-4: The effect of autocorrelation and population-level juvenile cue error on optimal cue weights and per-capita growth rate. A) The optimal weight for the juvenile cue. The two horizontal lines denote cross-sections of parameter space corresponding to panels B and D. B) Optimal cue weights as a function of autocorrelation when the population-level juvenile cue error $(\sigma_J^{pop})^2$ is equal to 3. C) Per-capita growth rate of a population with optimized cue weights as a function of autocorrelation and population-level juvenile cue error. D) Optimal cue weights as a function of autocorrelation when the population-level juvenile cue error is equal to 1. Remaining parameters: $r = 1$, $\sigma_\theta^2 = \frac{1-\lambda^2}{1-0.7^2}$, $\sigma_{mut}^2 = 0.25$, $(\sigma_{ME}^{ind})^2 = (\sigma_M^{ind})^2 = (\sigma_J^{ind})^2 = (\sigma_{ME}^{pop})^2 = 0.25$.

Effects of the Cue Integration System on Responses to Environmental Change

A main advantage of our partition of the growth rate is that most environmental parameters are found in only one fitness load and so the effect of many environmental changes can be determined by examining the relevant load term. We first consider a change in the mean environment (e.g., mean temperature or rainfall), which is located in the shift load $\mathcal{S}$. Equation 2-7 shows that maintaining a positive per-capita growth rate even after a change in mean environment depends not just on WGP, but on the population's total plasticity (i.e., the sum of the juvenile and maternal cue weights). The reduction in growth rate (fitness loss) is greatest when the pre-change environment favors low levels of plasticity. This occurs when both the environmental variance is low and population-level juvenile cue error is high (compare the left-most portions of figure 2-3*B*,*D* to that of figure 2-5*A*). Similarly, when the environmental autocorrelation is low (making TGP unreliable) and juvenile cue error is high (making WGP unreliable), total plasticity is low and the fitness loss due to a change in mean environment is high (compare the upper-left quadrant of figure 2-6*A* to the left-hand side of figure 2-4*B*). Note, however, that when population-level juvenile cue error and environmental variance are both large, total plasticity remains relatively high (figure 2-3*B*), and the reduction in growth rate from a change in mean environment remains low (figure 2-5*A*).

For changes in environmental variance, autocorrelation or cue error, figures 2-5*B*,*C*,*D* and 2-6*B*,*C*,*D* show that these changes are generally most harmful when they cause previously reliable cues to become unreliable. For increases in juvenile cue bias, the fitness loss is largest when, in the past, the juvenile cues were reliable and environmental variation was high or autocorrelation was low (i.e., when past conditions favored greater reliance on WGP; compare figures 2-5*B* and 2-6*B* with figures 2-3*A* and 2-4*A*). Under the opposite conditions that favor higher reliance on TGP and reduced WGP, changes that increase in juvenile cue bias are less costly. Equation 2-6 and figure S2-2 show that the results are qualitatively similar if environmental change results in an increase in the population-level juvenile cue error.

The environmental fluctuation load $\mathcal{F}$ increases linearly with environmental variance, so changes that increase environmental variation are most harmful when $\frac{\mathcal{F}}{V_\theta}$ is large. This occurs when both past environmental variation was low and population-level juvenile cue error was high (figure 2-5*C*). These past conditions favored weak WGP and relatively strong TGP (figure 2-3*B*, *D*), which becomes less reliable when greater environmental variation increases the average difference between adult and offspring environments. In contrast, if organisms evolved to rely heavily on WGP, then $\frac{\mathcal{F}}{V_\theta}$ is small and even if the organisms evolved with low to moderate environmental variation they continue matching well with their environment when the environmental variance increases.

When environmental autocorrelation decreases, the parent's environment is less predictive, so TGP is less effective. If the maternal phenotype and genetic cue weights (w_{MP} and w_Z) are small, then we can approximate the fitness loss from this reduction as $-\frac{\partial \mathcal{F}}{\partial \lambda} \approx V_\theta \frac{(1-w_J)(w_{ME}+w_{MP})}{(1+\sigma_X^2)}$. Thus, fitness loss from reduced autocorrelation is higher when TGP is relied on more heavily, which tends to be when past environmental variance was lower (figure 2-3*B*,*D*) or past autocorrelation was higher (figure 2-4*B*,*D*). Figure 2-6*D* shows the fitness loss associated with reduced autocorrelation is greater when past autocorrelation is higher. Figure 2-5*D* shows the fitness loss associated with reduced autocorrelation is lower under high environment variance than moderate environmental variance, particularly when population-level juvenile cue error is high and TGP is favored. In contrast, even though TGP is greater when past environmental variation is low, the fitness loss from reduced autocorrelation is actually higher under moderate environmental variation. This is because when environmental variation is low, the difference between past and current environments is small even when autocorrelation is reduced, leading to a smaller increase in environmental fluctuation load.

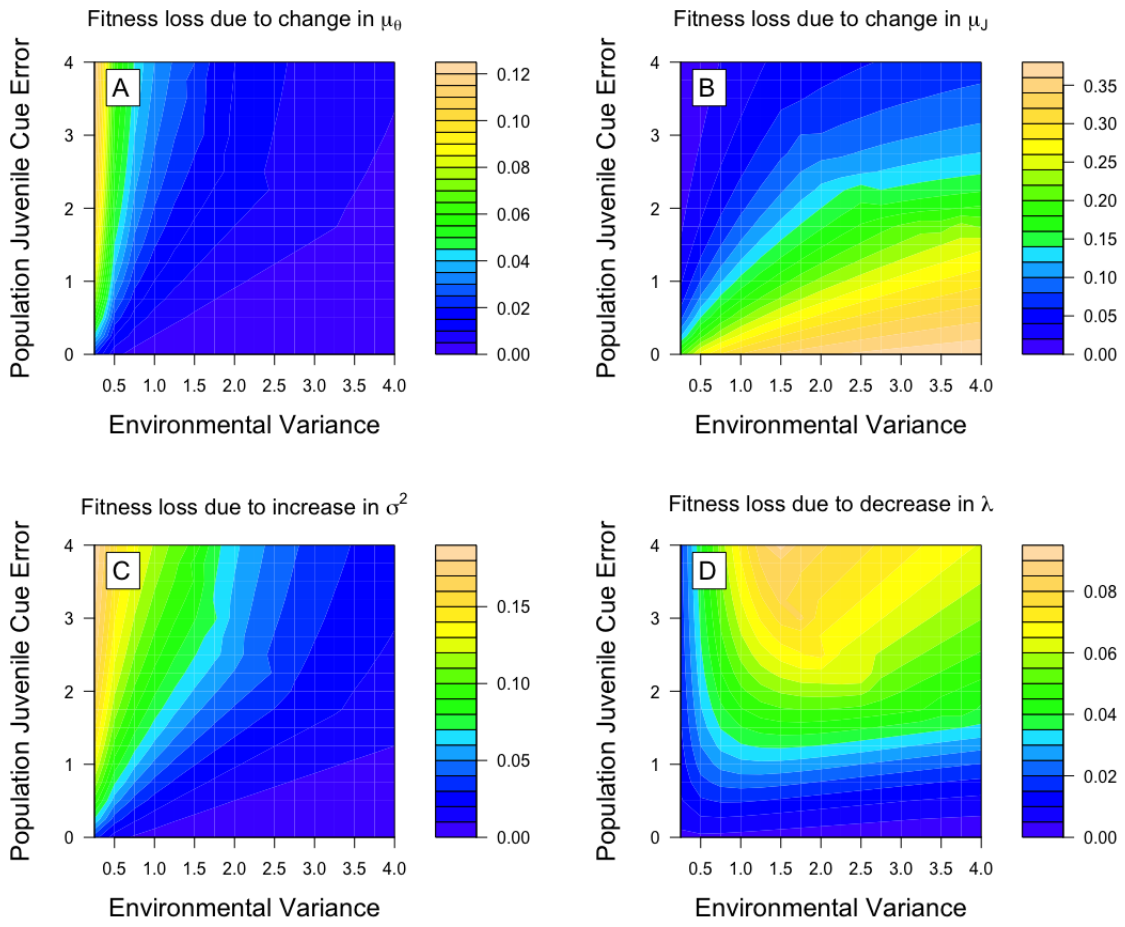

Figure 2-5: The effect of historical environmental variance and population-level cue error on the reduction in long-term per-capita growth rate (fitness loss) due to A) a change in the mean environment, B) a change in juvenile cue bias, C) a change in environmental variance, and D) a change in autocorrelation. For A and B the change is given by a 1 unit increase in mean environment and a 1 unit increase in juvenile cue bias. For C the change was an increase in environmental variance by 0.4, while for D the change was decrease in autocorrelation by 0.1. The other parameters were given as $r = 1$, $\lambda = 0.7$, $\mu_\theta = 0$, $\sigma^2_{mut} = 0.25$, $(\sigma^{ind}_{ME})^2 = (\sigma^{ind}_{M})^2 = (\sigma^{ind}_{J})^2 = (\sigma^{pop}_{ME})^2 = 0.25$.

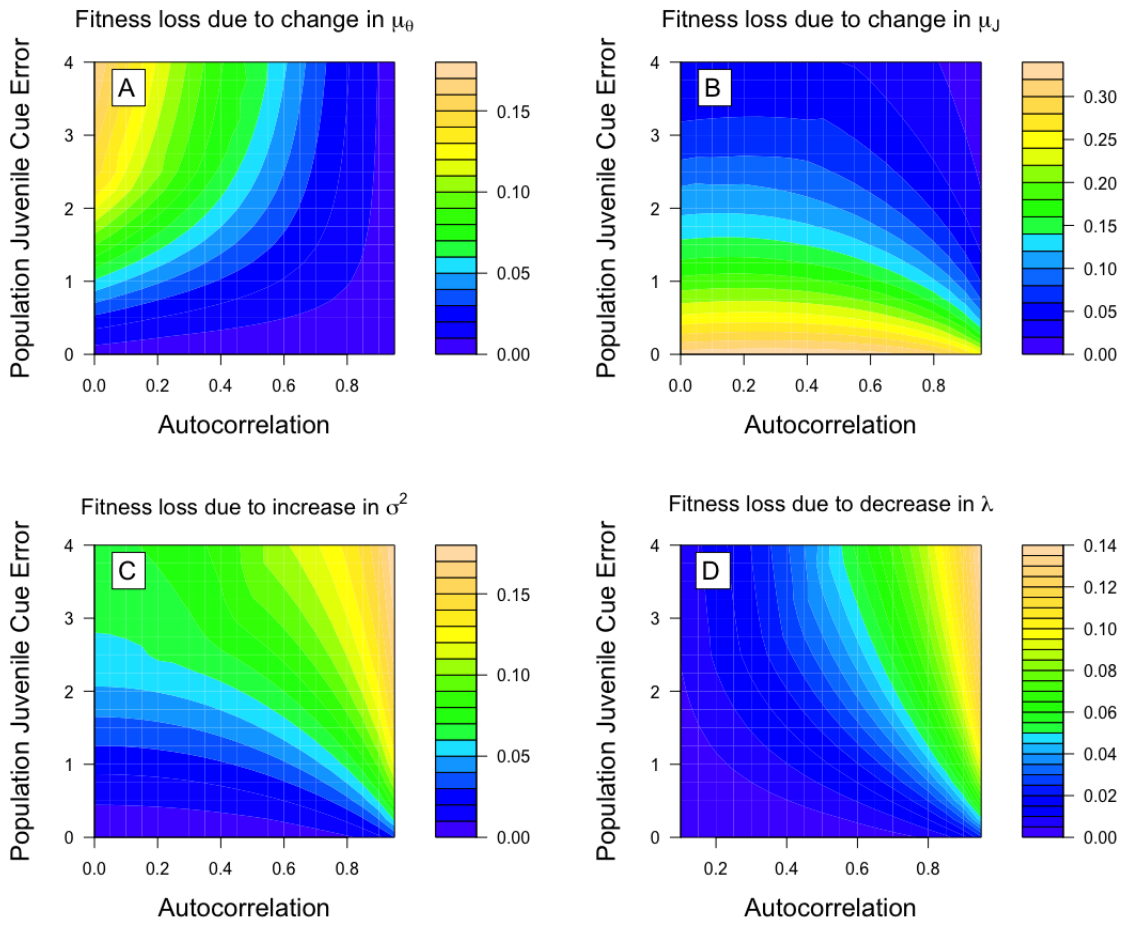

Figure 2-6: The effect of historical autocorrelation and population-level cue error on the reduction in long-term per-capita growth rate (fitness loss) due to A) a change in the mean environment, B) a change in juvenile cue bias, C) a change in environmental variance, and D) a change in autocorrelation. For A and B the change is given by a 1 unit increase in mean environment and juvenile cue bias, respectively. For C the change was an increase in environmental variance by 0.4, while for D the change was decrease in autocorrelation by 0.1. The other parameters were given as $r_{max} = 1$, $\sigma^2_\theta = \frac{1-\lambda^2}{1-0.7^2}$, $\mu_\theta = 0$, $\sigma^2_{mut} = 0.25$, $(\sigma^{ind}_{ME})^2 = (\sigma^{ind}_{M})^2 = (\sigma^{ind}_{J})^2 = (\sigma^{pop}_{ME})^2 = 0.25$.

Because the loads terms are additive, when changes in two environmental parameters affect different components of the per-capita growth, the total reduction in the growth rate is simply the sum of the reductions in growth rate caused by each change occurring in isolation (see equations

2-3, 2-4, 2-6, and 2-7). This means that aside from changes in individual-level cue errors, which influence the equilibrium phenotypic variance and therefore contribute to all four load terms, the only parameter combinations with non-linear interactions are environmental variance and autocorrelation (both found in the environmental fluctuation term $\mathcal{F}$), and the environmental mean and two cue biases (which are found in the environmental shift term $\mathcal{S}$).

There are some combinations of changes that result in large reductions in population growth regardless of the population's evolutionary history. For example, environmental variance affects the environmental fluctuation load $\mathcal{F}$ and the juvenile cue bias affects the environmental shift load $\mathcal{S}$, so if both environmental variance and juvenile cue bias increase, the effect on population growth can be visualized simply by superimposing figures 2-5*B* and 2-5*C* (as well as figures 2-6*B* and 2-6*C*). The fact that these figures are almost reflections of each other across a diagonal means that if the organism evolved in past conditions where one environmental change has low costs, the other has high costs. Therefore, these two changes produce complementary effects on population growth that tend to produce substantial reductions in growth rate regardless of pre-change conditions (but see Supplement 2-6 for when the genetic cue is present). The only exception is for populations that evolved in environments in which all cues were already unreliable (the upper right corner of figures 2-5*B* and 2-5*C*). These are, however, precisely the environments in which populations have low growth rates to begin with and therefore might be particularly vulnerable to even minor reductions in population growth rate. Thus, a simultaneous increase in environmental variation and juvenile cue bias represents a "no-win" scenario.

Discussion

Almost all species are facing one or more aspects of rapid environmental change including climate change, habitat loss or fragmentation, exotic enemies, increased human exploitation, and novel chemical stressors (Candolin and Wong 2012). Strategies that allow populations to persist in variable environments differ in the speed of their response to rapid environmental change, with

within-generational plasticity (WGP) being the fastest, transgenerational plasticity (TGP) slower, and rapid evolution slower still. Thus whether a population is able to adjust rapidly and persist after environmental change depends, in part, on which of these mechanisms was evolutionarily favored in the past. Building on recent work (Kuijper and Hoyle 2015; Leimar and McNamara 2015; McNamara et al. 2016; Rivoire and Leibler 2014; Uller et al. 2015) that used an informational framework to predict how environmental conditions should influence the relative importance of WGP and TGP in governing an individual's phenotype (Harney et al. 2017; McIntyre and Strauss 2014; Sultan et al. 2009; Walsh et al. 2016, 2015), we showed that the ability to cope with environmental change depends critically on the degree of mismatch between new environmental conditions and the strategies which evolved in pre-change environmental conditions.

Patterns of plasticity before environmental change

We found that selection for WGP and TGP tends to be negatively correlated across a wide range of environmental histories. This is because TGP was stronger when environments are more autocorrelated and when juvenile cues were less reliable, while WGP predominates when juvenile cues are accurate and previous environmental conditions are poor predictors of the current environment. These results are in line with previous theory (Colicchio and Herman 2020; English et al. 2015; McNamara et al. 2016).

We also found selection for bet-hedging when environmental cues are consistently unreliable (i.e., high environmental variance, low autocorrelation, and high population-level cue error). This is consistent with previous studies that predict bet-hedging when the environmental fluctuation load is high relative to the width of the fitness function (Bull 1987; Scheiner 2014; Tufto 2015). These results also highlight how the effects of cue error differ when shared by the entire population versus experienced independently by each individual. While McNamara et al. (2016) found that high individual-level error mimics randomization by increasing phenotypic variation (Donaldson-Matasci et al. 2013), we found that population-level error can increase the favorability of randomization as the only mechanism to achieve bet-hedging (figure S2-1). Population-level

errors also have a stronger effect on optimal levels of WGP and TGP, as well as on population growth rate, than do individual-level errors (figures S2-1 and S2-2).

Strikingly, we find that the genetic cue was not utilized for any of our main results (i.e., rapid evolution in the reaction norm elevation was not favored and the phenotype was always genetically canalized). This is consistent with McNamara's et al. (2016) observation that utilizing the genetic cue is only optimal when the environment is stable and the other cues are inaccurate (see also figure S2-5C). To explain this result we show analytically, in Supplement 2-3, that populations relying on both TGP and rapid evolution tend to be worse at tracking the environment than populations that rely on either one or the other and that plasticity reduces genotype-environment covariance.

Our prediction that complete genetic canalization is often evolutionarily favored is belied by the presence of heritable fitness-related traits in many natural populations. One explanation is that complete genetic canalization is indeed favored, but prevented by genetic or developmental constraints on the cue integration system. In Supplement 2-6, we replicate our results under the assumption that the cue integration system does not control the genetic cue weight. While we see mostly similar results, changes in mean environment and cue bias do not have long-term fitness effects because the population rapidly evolves in response to these changes. Additionally, the maternal phenotype cue is significantly lower and may even be negative. This is consistent with previous work showing that, in the presence of heritable variation, negative maternal cue weights cause maternal effects to become negatively correlated with offspring genotype, thereby reducing phenotype variance and the variance load on fitness (Hoyle and Ezard 2012; Kuijper and Hoyle 2015). Overall, these results suggest that the degree of developmental control exercised by the cue integration system may have significant impacts on the evolution of plasticity.

Determining the historical environment in terms of variance, autocorrelation, and cue reliability is challenging in an empirical setting, but can be accomplished, for example, by using a blend of long-term studies and paleoecological records (Barnosky et al. 2017; Colicchio and Herman 2020; Merilä and Hoffmann 2016), by comparing plasticity patterns across populations with differing

and documented long-term environmental regimes (Walsh et al. 2016) or by measuring the genetic variation in the cue integration system in response to environmental variance (Harney et al. 2017). Determining the patterns of adaptive plasticity, their interactions, and their evolution are ripe areas for future empirical work.

Evolutionary history and the response to environmental change

Our model results clarify, and in some cases differ from, previous theoretical predictions of how past evolutionary history pre-adapts organisms to cope with environmental change. Two common ideas are: 1) Organisms that have evolved greater plasticity (e.g., generalists) should be able to adjust well to environmental change (Sih et al. 2011; Snell-Rood 2013), and 2) organisms that have evolved with high environmental variation should, all else being equal, cope better with environmental change. Reviews of organismal responses to specific changes suggest that these trends often hold, but with exceptions (Beever et al. 2017; Kuparinen and Festa-Bianchet 2017; Langkilde et al. 2017; Legrand et al. 2017; Saaristo et al. 2018).

When does greater plasticity help organisms respond well to environmental change?

The idea that generalists or flexible (plastic) organisms might do well with environmental change depends on the assumption that they exhibit adaptive plasticity. If environmental change does not reduce the reliability of WGP or TGP, our results concur with this prediction (figure 2-3*A*). Indeed, numerous empirical examples show that many organisms exhibit adaptive plasticity in response to environmental change (Bonamour et al. 2019; Charmantier et al. 2008; Nicotra et al. 2010). If, however, change reduces cue reliability (i.e., causes a previously adaptive cue-response system to become maladaptive), organisms can suffer substantial fitness losses associated with falling into evolutionary traps (figure 2-3*B*; Ashander et al. 2016; Reed et al. 2010). Empirically, these include settlement in low quality habitat (Crowley et al. 2019; Delibes et al. 2001; Robertson et al. 2013) and lack of avoidance of novel predators (Ehlman et al. 2019) or novel toxic resources (Savoca et al. 2017; Shine 2010). Our model corroborates recent models (Crowley et al. 2019; Ehlman et al. 2019)

and conceptual reviews in suggesting that organisms that had highly reliable cues in the past and that thus exhibit strong WGP might be particularly likely to exhibit costly, maladaptive responses to habitat change, exotic predators or novel toxic 'foods' (Pollack et al. 2021).

In contrast, organisms that evolved with low cue reliability should exhibit little WGP, and thus weak responses to environmental change. For example, while some organisms have exhibited an adaptive phenological response to climate warming (e.g., by germinating, reproducing, migrating or changing color earlier in the season), others have not (reviewed in Chmura et al. 2019). Our model predicts that organisms that evolved with unreliable environmental cues (e.g., late winter temperature has not reliably indicated the best time to breed or germinate in the spring) should not exhibit WGP in response to warmer late winter temperatures, even if those temperatures signal a consistently earlier spring (McNamara et al. 2011). For another example, if the conditions in the wintering grounds of migrating organisms are poor indicators of conditions in their spring breeding grounds (e.g., birds migrating from the tropics to temperate regions), we predict that migrant organisms exhibit smaller shifts in breeding time with global warming than resident populations (Moller et al. 2003).

Our results also suggest that when environmental cues during development are unreliable, TGP may play an important role in facilitating responses to environmental change (Donelan et al. 2020). Organisms that evolved to ignore juvenile environmental cues may still respond adaptively to exotic predators or warming conditions (albeit with a one generation delay) if they have evolved to utilize cues passed down from their parents (Burgess and Marshall 2011; Shama et al. 2014; Sobral et al. 2021; Walsh et al. 2015). TGP is expected to be strong when WGP is ineffective (juvenile cue reliability is low) and the environment is relatively stable (low to moderate environmental variation and moderate to high autocorrelation) so that parental environments are good predictors of offspring environments. Organisms that have evolved strong TGP and weaker WGP can cope with reduced juvenile cue reliability (figure 2-5*B*); i.e., parents can keep offspring from falling into evolutionary traps. However, organisms that exhibit strong TGP and weaker WGP should be susceptible to reduced environmental stability (i.e., increased environmental

variation, or reduced autocorrelation; figures 2-5*C*,2-5*D*; Donelan et al. 2020).

On the other hand, if organisms evolved in conditions that favor neither WGP nor TGP, then they will likely exhibit non-plastic phenotypic strategies, such as consistently breeding at the same time each year or bet hedging (e.g., have offspring that vary in when they germinate; Cuello et al. 2019; Simons 2014). In either case, such populations would not show adaptive shifts to climate change and could experience reduced growth rate.

When does past environmental variation prepare organisms for future change?

For organisms that evolved in very stable environments, our model confirms there is selection for low WGP and low TGP, and a single, generally successful phenotype. Since these organisms are relatively inflexible, they are vulnerable to large fitness losses with either an increase in environmental variation or a change in the environmental mean (figures 2-6*A*,2-6*C*), but aren't as affected by changes in cue reliability. When organisms evolved with a wide range of possible conditions (high environmental variance), then our results show that they respond well to a shift in environmental mean so long as cues have been reliable and autocorrelation was sufficiently high. In that case, organisms should exhibit strong WGP and high overall plasticity, resulting in adaptive responses to the shift if cue reliability remains high (figures 2-5*A*,2-5*C*). Because these organisms exhibit strong WGP, they do not necessarily cope with decreases in juvenile cue reliability (reviewed in Bonamour et al. 2019). For example, plants that evolved in conditions where the timing of snowmelt was a good indicator of each year's best time to germinate would suffer fitness loss if the environment changed so historically early snowmelts were often followed by a later lethal freeze (Gezon et al. 2016).

If environmental variation is high, but juvenile cue reliability was historically low, then as long as environmental autocorrelation is high, organisms should exhibit strong TGP. For example, for shifts in seasonal phenology in the absence of good reliable cues, we and others (e.g., Burgess and Marshall 2011; Salinas et al. 2013) predict that TGP should be important in areas with strong interannual correlation (and low interannual variation) in temperature and precipitation. Colicchio

and Herman's (2020) geographic analysis of predicted TGP levels in the United States indicates substantial regional differences in the favorability of TGP. Taken with our results, this suggests that regional variation in response to climate change could in part be explained by whether regional conditions favor WGP, TGP, or bet-hedging.

If, on the other hand, conditions are unpredictable due low autocorrelation, then contrary to previous predictions, organisms that evolved in these environments do not necessarily cope well with rapid change. Populations that evolved with low autocorrelation should exhibit far lower levels of total plasticity than populations that evolved with high autocorrelation (especially if WGP is somewhat unreliable), so they should be particularly vulnerable to changes in the mean environment. At the extreme end, if organisms evolved in variable environments with relatively little useful environmental information of any sort (e.g., with low juvenile cue reliability and low autocorrelation), they should exhibit diversified bet hedging. In this situation, bet-hedging reduces the fitness consequences of environmental change, but pre-change fitness is low enough that even small reductions in growth rate could drive populations to extinction.

Comparison to rapid evolution of plasticity

By assuming that the cue integration system does not have time to adjust to rapid environmental changes, our results complement previous studies that focus on rapid evolutionary response to environmental change (e.g., Botero et al. 2015; Kuijper and Hoyle 2015; Lande 2009). When the evolution of plasticity is possible, populations respond to abrupt changes in the mean environment by rapidly evolving high WGP (or utilizing existing plasticity) and then slowly reducing plasticity as the reaction norm elevation gradually evolves to match the new environment (Chevin and Lande 2010; Lande 2009). When maternal effects are present, they show a similar transient increase with smaller peak levels of WGP (Hoyle and Ezard 2012; Kuijper and Hoyle 2015). This aligns with our observation that in the absence of rapid evolution (no genetic cue), populations that already possess high levels of total plasticity (WGP + TGP) cope the best with changes in mean environment.

Botero et al. (2015) model how historical environments affect population responses to environmental changes by selecting for different evolutionary strategies (reversible and irreversible plasticity, conservative and diversified bet-hedging, and adaptive tracking). They find abrupt shifts in strategies between different environmental conditions. Extinction risk is greatest for environment change across one of these evolutionary tipping points. Broadly, this agrees with our finding that populations suffer most when previously adaptive strategies become maladaptive, but contrasts with our observation that cue weight and fitness loss change continuously with the environment. This discrepancy is likely due to differences in model assumptions; their model's genetic architecture and fixed costs of plasticity lead Botero et al. (2015) to find that evolution is faster within than between strategy types (except for transitions between reversible and irreversible plasticity). We do not consider the genetics or costs of the cue integration system, leading to a gradient of strategies rather than discrete regimes.

Future Directions

Our simplifying assumptions provide areas of possible future study. Like McNamara et al. (2016), our model of the cue integration system was phenomenological. Future modeling could feature a more mechanistic description of phenotypic determination and organism fitness, including a non-linear cue integration system and more realistic interactions between the environment, cues, and fitness (e.g., asymmetric fitness surfaces; Lof et al. 2012, or asymmetric cue error). In particular, our model did not address the underlying genetics of the cue integration system. Genetic constraints (e.g., genetic correlations or lack of genetic variation) might keep organisms from reaching the optimal level of plasticity. Recent evidence has shown genetic correlations involving TGP (Townley and Ezard 2013) and between TGP and WGP (Auge et al. 2017). Additional, we did not account for the costs of plasticity and how they might differ for WGP versus TGP. We anticipate, for example, that accounting for costs of plasticity will reduce plasticity and likely increase the range of conditions favoring bet hedging or rapid evolution.

In addition, we assumed that organisms do not have repeated opportunities to adjust their

traits. In reality, parents might often provide several pieces of information to offspring over time (e.g., via epigenetic carryovers or yolk hormones before birth, and parental care after birth) and offspring can exhibit both developmental plasticity based on early sensitive windows as well as adjustments via reversible, contextual plasticity in response to later experiences throughout their lifetime (English et al. 2016; Snell-Rood 2013; Stamps and Groothuis 2010). Future work could begin to address these multi-step options by adding a second opportunity for information transfer and adjustment associated with TGP and WGP.

Finally, we assumed that patterns of environmental variation remained stationary over evolutionary time so that the evolved levels of plasticity represent evolutionary optimal; in reality, environmental conditions may be subject to long-term trends and periodic disturbance. This means that not only the nature and strength of rapid environmental change important, but also its timing.

By accounting for these details, future work may examine evolution following environmental change and, in particular, the role of previously adaptive cue integration systems in shaping subsequent evolutionary rescue (Lande 2009). Though the effect of fluctuating environments on the likelihood of rescue following a change in the mean environment has long been studied (Burger and Lynch 1995; Chevin 2013; Chevin et al. 2017; Lande and Shannon 1996), to date there has been little study of evolutionary rescue under environmental changes other than a change in mean environment. This is a promising area for future research using our conceptual framework.

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Decomposing the effects of demographic, sex-ratio, and phenotypic stochasticity on extinction during evolutionary rescue

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Abstract

Predicting when and how populations go extinct is a critical task for ecologists. Because of uncertainty in individual-level processes such as death or reproduction, populations with the same initial state experiencing identical environments may have different fates. Individual-level variability manifests as a variety of population-level processes – such as demographic stochasticity, sex-ratio stochasticity, and phenotypic stochasticity – that are often studied separately. Understanding how stochasticity drives divergence in population outcomes requires studying these process under a unified framework. I present a general framework for quantifying and partitioning the effects of stochasticity on future population states and apply it to the question of evolutionary rescue: When can rapid evolution save a population from going extinct in a changing environment? I decomposed the effects of demographic stochasticity, sex-ratio stochasticity, and genetic drift on per-capita growth rate and then quantify how well the effects of each form of stochasticity can predict time to extinction or time to rescue. I found that phenotypic stochasticity contributed the most to the predictability of extinction early during rescue while the contributions of demographic stochasticity and sex-ratio stochasticity increase in importance as time goes on. I further found that phenotypic stochasticity was significantly more important for predicting time to recovery than for predicting time to extinction, while the opposite is true for demographic and sex-ratio stochasticity. The greater importance of phenotypic stochasticity to recovering populations reflects the fact that rapid evolution is required for successful recovery and hence faster-than-average evolution should lead to sooner than average recovery. Overall, these results present a nuanced picture of how stochasticity results in divergent population outcomes and illustrate how my framework for partitioning the effects of stochasticity may be used to derive novel insights into the dynamics of small populations and extinction.

Introduction

Anthropogenic environmental change has produced an unprecedented decline in natural populations and many species are at significant risk of extinction (Ceballos et al. 2017; Román-Palacios and Wiens 2020). Many populations are exhibiting phenotypic change in response to these changes (Hendry et al. 2017), raising the hope that rapid adaptation may blunt the effects of a changing environment (but see Radchuk et al. 2019) and leading to greater focus on evolutionary potential as part of species conservation (Ashley et al. 2003; Carroll et al. 2014; Kinnison and Hairston 2007; Lankau et al. 2011; Stockwell et al. 2003). At the same time, humans are attempting to suppress a variety of species that are deemed harmful- including pathogens, agricultural pests, and invasive species- and wish to prevent these species from evolving resistance to the measures used to control them (reviewed in Bell 2017). These areas of interest are united by the potential for rapid adaptation to enable populations to persist in the face of otherwise fatal environmental changes, a phenomenon referred to as evolutionary rescue.

Evolutionary rescue has been studied extensively in both theoretical and laboratory settings (reviewed in Bell 2017; Carlson et al. 2014; Vander Wal et al. 2013). One of the central conclusions of this research is that the success of evolutionary rescue depends critically on the rate of evolution and the rate of demographic decline. We know, for example, that both the likelihood of persistence and the time to recovery are closely tied to the initial genetic variance of the population and the rate of beneficial mutations (Agashe et al. 2011; Barrett and Schluter 2008; Burger and Lynch 1995; Orr and Unckless 2008; reviewed in Bell 2017). Similarly, populations with low initial size or those experiencing stronger or more rapid environmental changes are more likely to go extinct (Bell 2017; Bell and Gonzalez 2009; Gomulkiewicz and Holt 1995; Lynch and Lande 1993).

Because the likelihood of evolutionary rescue depends on the relative rates of population decline and phenotypic evolution, the interaction between ecological and evolutionary dynamics is particularly important. Populations undergoing rescue may become quite small and vulnerable to stochastic extinction due to the intrinsic variability in the births and deaths of individuals in

the population (Bell and Gonzalez 2009; Engen et al. 1998, 2001; Lande 1988, 1998). This intrinsic variability may induce variation in population size (demographic stochasticity), variation in allele frequency (genetic drift), and in dioecious populations, variation in sex ratio (sex stochasticity, sensu Melbourne and Hastings 2008), which all contribute to extinction in their own ways.

The impacts of different forms of stochasticity on extinction have individually been extensively studied: There is a substantial body of work on the effect of demographic stochasticity (Engen et al. 2001; Gabriel and Bürger 1992; Jeppsson and Forslund 2012; Lande 1993, 1998) and the effect of sex-ratio stochasticity (Gabriel and Bürger 1992; Melbourne and Hastings 2008) on a population's time to extinction, and it is well known that genetic drift may contribute to extinction through mutational meltdown (in which deleterious alleles rise in frequency in small populations, reducing population size and resulting in stronger drift; Coron et al. 2013; Lande 1994; Lynch et al. 1995*a*,*b*; Zeyl et al. 2001), by reducing the efficacy of natural selection, and by eroding genetic variation (Lande 1988; Luque et al. 2016; Whitlock 2000; Willi et al. 2006). Because the interplay between ecology and evolution drives the success or failure of evolutionary rescue, the interaction between these forms of stochasticity is critical to understanding the process of extinction in evolving populations and remains an important and understudied (but see Melbourne and Hastings 2008) avenue of inquiry.

This paper presents a general theoretical framework for quantifying the effects of stochasticity on the outcomes (such as extinction) of individual populations, and partitioning these effects by the form of stochasticity (e.g. demographic stochasticity and genetic drift), and measuring how the effects of stochasticity change over time. I then apply this method to examine the relative importance of demographic stochasticity, sex-ratio stochasticity, and phenotypic stochasticity on the persistence or extinction, time to extinction, and time to recovery of populations undergoing evolutionary rescue. I focus on populations that rely on standing quantitative genetic variation to adapt an abrupt environmental change. This leads to a sharp initial decline in the population, followed by rapid evolution and either recovery or extinction, leading to a characteristic U-shaped curve in population abundance (Gomulkiewicz and Holt 1995). I chose this mode of environmental

change (as opposed to slower, but sustained environmental changes; e.g. Chevin et al. 2010; Lande and Shannon 1996; Lynch and Lande 1993; Osmond and Klausmeier 2017) because populations either recover or go extinct within a relatively short window of time, allowing for a more fine grained analysis of the effect of stochasticity in each generation.

This framework represents a novel approach to thinking about uncertainty during evolutionary rescue. Previous models of evolutionary rescue from standing quantitative genetic variation have focused on how the initial conditions of the population affect the likelihood of rescue (e.g. Barfield and Holt 2016; Orive et al. 2019). The present paper focuses instead on how stochastic events over the course of evolutionary rescue itself change the likelihood of rescue. This approach allows me to quantify which forms of stochasticity, at which points in time, are most critical in determining the persistence or extinction of a population in which the outcome of evolutionary rescue is initially uncertain.

Methods

Model Methods

Consider a sexually reproducing, dioecious population of semelparous individuals with synchronized reproduction. Each generation consists of two demographic periods: survival to reproduction and reproduction. Natural selection acts during reproduction and the population is censused prior to reproduction. At census in generation t, there are N_t individuals characterized by their sex ($f_i = 1$ for females and 0 for males), a continuous trait ($z_i \in \mathbb{R}$), and the breeding value, underlying that trait ($g_i \in \mathbb{R}$), with $i \in \{1, 2, ..., N_t\}$ where N_t is the population size at time t.

Mating is female limited. During reproduction, females experience fecundity selection while males experience mate choice or gamete selection. The offspring produced by each female are sired independently by males in proportion to each male's fitness. This mating system describes broadcast spawners, animal species with widespread promiscuity, and dioecious plants. Individual

i produces a Poisson distributed number of offspring with mean $f_i\lambda(z_i)$ (i.e. $\lambda(z_i)$ offspring if individual i is female and 0 otherwise), where $\lambda(z_i)$ is a Gaussian function of z_i with center μ_w, width σ_w^2, and maximum mean fecundity λ_{max}:

$$\lambda(z_i) = \lambda_{max} \exp\left[-\frac{(z_i - \mu_w)^2}{2\sigma_w^2}\right]. \tag{3-1}$$

The probability that a given individual i sires a given offspring, $p_m(z_i)$, is weighted by a Gaussian function of phenotype (with the same center μ_w and width σ_w^2) that is transformed into a probability by dividing by total male fitness in the population:

$$p_m(z_i) = \frac{(1 - f_i) \exp\left[-\frac{(z_i - \mu_w)^2}{2\sigma_w^2}\right]}{\Sigma_{j=1}^{N_t}(1 - f_j) \exp\left[-\frac{(z_j - \mu_w)^2}{2\sigma_w^2}\right]}. \tag{3-2}$$

If we assume that the genetic basis for z is purely additive with no dominance or epistasis, z_i may be given as the sum of an individual's breeding value g_i (henceforth "genotype") and a normally distributed random deviation due to environmental effects (e_i with mean zero and variance σ_e^2). For the breeding value, I assume the Gaussian descendants approximation (sensu Turelli 2017) of the infinitesimal model of quantitative genetics (Fisher 1918; Walsh and Lynch 2018). Then, an individual's breeding value is the average of its parents' breeding values (denoted g_i^f for its mother and g_i^m for its father) plus a normally distributed random deviation from the midparent breeding value due to recombination (s_i with mean zero and segregation variance σ_s^2):

$$g_i = \frac{1}{2}(g_i^f + g_i^m) + s_i \tag{3-3}$$

$$z_i = g_i + e_i. \tag{3-4}$$

Finally, offspring survive to reproduce with probability p_s, which is independent of phenotype.

Decomposition of Stochasticity

In this section I present a theoretical framework for decomposing the effects of different forms on stochasticity on a particular population outcome, such as extinction or persistence. We may represent an individual mathematically as the collection of characteristics (in this case sex,

phenotype, and genotype) that most influence its fate, which is to say the likelihood of the individual giving birth, dying, or changing state. These characteristics are "*i*-state variables" and their values constitute the "*i*-state" of an individual (Diekmann et al. 2010; Metz and Diekmann 1986). The "*p*-state" of the population is the density of i-states across i-state-space (Diekmann et al. 2010; Metz and Diekmann 1986). The *p*-state variables are then the frequency distributions of each *i*-state variable, plus the population size N, which scales these frequency distributions into densities. For the model above, the *p*-state variables are population size N and the vectors of individual sexes $\mathbf{F}$, phenotypes $\mathbf{P}$, and genotypes $\mathbf{G}$ (because the phenotypes are continuous, the frequency distributions of genotypes and phenotypes are collections of point masses and can therefore be represented as vectors).

Stochasticity at the population level (i.e. in the *p*-state) is driven by demographic changes at the individual level. If an individual dies, gives birth, or changes state, this event changes the distribution of *i*-states in the population. The effect of this individual event on future population states is mediated by each *p*-state variable. The death of a male individual with a low fitness phenotype necessarily reduces population size, but will also affect the population's sex ratio and phenotypic distribution. Each of these changes may have different knock-on effects on the population state at a future time. We may therefore think of each *p*-state variable as a distinct pathway by which individual events affect future states of the population. To partition the effects of different forms of stochasticity on future population dynamics, we must partition variability in the total *p*-state into variability in particular *p*-state variables.

One can use the law of total probability to partition the probability distribution of the *p*-state into the marginal and conditional distributions of the *p*-state variables. I begin with the marginal distribution of N, $f_N(n)$. I condition the distribution of $\mathbf{F}$ on population size, $f_{\mathbf{F}|N}(\mathbf{f}|n)$. Then, I condition the joint distribution of phenotypes and genotypes on population size and the sex of individuals, $f_{\mathbf{P},\mathbf{G}|N,\mathbf{F}}(\mathbf{p},\mathbf{g}|n,\mathbf{f})$. This gives

$$f_{N,\mathbf{F},\mathbf{Z},\mathbf{G}}(n,\mathbf{f},\mathbf{g},\mathbf{p}) = f_N(n) f_{\mathbf{F}|N}(\mathbf{f}|n) f_{\mathbf{P},\mathbf{G}|N,\mathbf{F}}(\mathbf{g},\mathbf{p}|n,\mathbf{f})$$

This assumes that *i*-state variables are discrete, but an analogous expression exists for continuous states; Kallenberg 2017. Decomposing the joint state distribution in this way requires explicitly ordering the *p*-state variables based on their conditional dependence. This ordering is an important choice that defines the pathways through which individual stochasticity affects future population states, as well as the dependency of these pathways on one another. In this formulation, individual stochasticity is partitioned into three population-level pathways: demographic stochasticity, sex ratio stochasticity, and phenotypic stochasticity.

In general there is no single correct answer, but some orderings are more natural than others. This choice will depend on the state variables of interest and the underlying demography of the population. For the present model, writing the distribution of N as marginal is a natural choice because the remaining variables depend on the number of individuals in the population. For example, in a single-locus bi-allelic model the distribution of allele frequencies in the next generation, conditional on population size, matches the usual definition of genetic drift while the distribution of N conditional on an allele frequency of, say, 0.5 has the bizarre property that only even values of N have positive probability. The decision to condition the phenotypic and genotypic distributions on sex is more situational. If phenotypes are sexually dimorphic or the genotype of interest is sex-linked, then it makes sense to condition the phenotypic distribution on the sex ratio. If the genotype of interest is located at a locus associated with sex-determination, then the reverse ordering is more logical. Similarly, writing genotype and phenotype as a the joint distribution implies that stochasticity in these *p*-state variables constitute a single pathway of interest. In the presence of phenotypic plasticity, for example, phenotypic stochasticity within a given genotype might be sufficiently important to be considered as a separate pathway.

I now consider the effect of stochasticity over the time period from t to $t+1$ on the probability of persistence to time horizon T_{max}. Let I_{pst} equal 1 if $N_{T_{max}} > 0$ and zero otherwise. At time t, the probability of persisting to T_{max} is $\mathbb{P}[N_{T_{max}} > 0|\mathcal{S}(t)] = \mathbb{E}[I_{pst}|\mathcal{S}(t)]$, where $\mathcal{S}(t) = (N, \mathbf{F}, \mathbf{G}, \mathbf{P})$ is the *p*-state of the population at time t. As we gain information about the events of from time t to $t+1$, probability of persistence changes to reflect this new information: $\mathbb{E}[I_{pst}|\mathcal{S}(t+1)]$. The

realized effect of stochasticity from $(t, t+1]$ on persistence probability may then be defined as

$$\Delta_t I_{pst} := \mathbb{E}[I_{pst}|\mathcal{S}_{t+1}] - \mathbb{E}[I_{pst}|\mathcal{S}_t]. \tag{3-5}$$

If we partition time into unit intervals, we can decompose the outcome of persistence or extinction (I_{pst}) at time T_{max} into the effect of stochasticity in each generation on persistence probability:

$$\mathbb{E}[I_{pst}|\mathcal{S}_{T_{max}}] = \mathbb{E}[I_{pst}|\mathcal{S}_0] + \Sigma_{t=0}^{T_{max}-1}\Delta_t I_{pst}. \tag{3-6}$$

The realized stochasticity from t to $t+1$ may be partitioned into components corresponding to phenotypic stochasticity $\Delta_t^P I_{pst}$, sex ratio stochasticity $\Delta_t^F I_{pst}$, and demographic stochasticity $\Delta_t^N I_{pst}$ between $(t, t+1]$:

$$\begin{aligned}\Delta_t I_{pst} = \Delta_t^P I_{pst} + \Delta_t^F I_{pst} + \Delta_t^N I_{pst} :=\\ (\mathbb{E}[I_{pst}|\mathcal{S}_{t+1}] - \mathbb{E}[I_{pst}|\mathcal{S}_t, N_{t+1}, \mathbf{F}_{t+1}])+\\ (\mathbb{E}[I_{pst}|\mathcal{S}_t, N_{t+1}, \mathbf{F}_{t+1}] - \mathbb{E}[I_{pst}|\mathcal{S}_t, N_{t+1}])+\\ (\mathbb{E}[I_{pst}|\mathcal{S}_t, N_{t+1}] - \mathbb{E}[I_{pst}|\mathcal{S}_t])\end{aligned} \tag{3-7}$$

We then may define the cumulative effects of phenotypic stochasticity, sex ratio stochasticity, and demographic stochasticity on persistence probability by summing the effects across generations: $\mathcal{C}_{T_{max}}^P(I_{pst}) := \Sigma_{t=1}^{T_{max}}\Delta_t^P I_{pst}$, $\mathcal{C}_{T_{max}}^F(I_{pst}) := \Sigma_{t=1}^{T_{max}}\Delta_t^{\mathbf{F}} I_{pst}$, and $\mathcal{C}_{T_{max}}^N(I_{pst}) := \Sigma_{t=1}^{T_{max}}\Delta_t^N I_{pst}$, respectively. This method of partitioning stochasticity may be applied the additional population outcomes of time to recovery ($T_{rec} = \min\{t \geq 0 : N_T \geq N_0\}$) and time to extinction ($T_{ext} = \min\{t \geq 0 : N_T = 0\}$) to yield $\mathcal{C}_{T_{max}}^P(T_{rec})$, $\mathcal{C}_{T_{max}}^F(T_{rec})$, $\mathcal{C}_{T_{max}}^N(T_{rec})$, $\mathcal{C}_{T_{max}}^P(T_{ext})$, $\mathcal{C}_{T_{max}}^F(T_{ext})$, and $\mathcal{C}_{T_{max}}^N(T_{ext})$.

Numerical Methods

I consider a single, abrupt environmental change to which the population must rapidly adapt in order to avoid extinction. Prior to the environmental change, the optimal trait value is $\mu_w = 0$ and

at $t = 0$, the optimal trait value shifts to $\mu_w = \mu'_w$ where it remains indefinitely. All populations are assumed to start with 1000 individuals at $t = 0$. These initial individuals are drawn from the population's equilibrium phenotypic distribution in the limit of infinite population size, with $\mu_z = 0$ and genetic variance satisfying the equation $\sigma_g^2 = \frac{1}{2}(\frac{\sigma_w^2+\sigma_g^2}{\sigma_w^2+\sigma_g^2})\sigma_g^2 + \sigma_s^2$ (derived in supplement 3-1; Bulmer 1971; Walsh and Lynch 2018). Simulations are run until the population goes extinct, recovers to its initial size, or until time $T_{max} = 100$ (though in practice all populations either recover or go extinct by generation 70).

At each time step t, the probability of persistence $\mathbb{E}[I_{pst}|\mathcal{S}_t]$, expected time to extinction $\mathbb{E}[T_{ext}|\mathcal{S}_t]$, and expected time to recovery $\mathbb{E}[T_{rec}|\mathcal{S}_t]$ are computed by running 1000 sub-simulations, initialized at the current population state, and recording whether these population recovered or went extinct and how long it took to achieve this outcome. The expected outcomes conditional on $N(t+1)$ are computed similarly except that each sub-simulation is initialized by drawing from the distribution of possible population states at time t conditional on $N(t+1)$ in the primary simulation. The computation of the expected outcomes conditional on both $N(t+1)$ and $F(t+1)$ follows the same method, but with the distribution of possible initial conditions in the secondary simulations being conditioned on both N and F in the primary simulation. The conditional expectations are then used to compute the effects of demographic stochasticity, sex ratio stochasticity, and phenotypic stochasticity on persistence probability, extinction time, and recovery time in generation t following equation 3-7 and its equivalents for T_{ext} and T_{rec}.

Because the number of simulations are finite (1000), the computed expected recovery time is not accurate when the persistence probability is close to zero and the expected extinction time is not accurate when the probability of recovery is close to one. For this reason, extinction time or recovery time are only analyzed for parameters where at least 25% of populations go extinction or recover, respectively.

Eight parameter values were selected in a full factorial combination of low and high environmental shift ($\mu_w = 5.2$ and 5.5), initial heritability ($h^2 = 0.25$ and 0.75), and initial phenotypic variance ($\sigma_z^2 = 1$ and 3). The width of the fitness function was held constant at $\sigma_w^2 = 10$, represent-

ing weak to moderate stabilizing selection, and the maximum per-capita growth rate was fixed at $\lambda_{max} = 1.5$.

For each parameter set, h^2 and σ_z^2 are converted into the genetic and environmental variances (σ_g^2 and σ_e^2). Genetic variance was assumed to initially be at its equilibrium in the limit of infinite population, and the segregation variance σ_s^2 was chosen to produce the appropriate equilibrium genetic variance (by satisfying the equation $\sigma_s^2 = \sigma_g^2 - \frac{1}{2}(\frac{\sigma_w^2+\sigma_g^2}{\sigma_w^2+\sigma_g^2})\sigma_g^2$, see supplement 3-1). In general, segregation variance and initial genetic variance correspond closely enough that treating one or the other as the manipulated parameter does not significantly alter the chosen parameters values.

To measure how well population outcomes can be predicted prior to their occurrence, I use the covariance across simulations between the predicted outcome at time t and the actual outcome, scaled by the variance in the actual outcome. This is equivalent to the linear regression coefficient of the predicted outcome on the actual outcome. If we let $\mathcal{S}_t^k$ denote the state at time t of simulated population k and let I_{pst}^k denote I_{pst} for simulation k, then this standardized covariance (SCV) is given by

$$SCV_{I_{pst}}(t) := \frac{Cov[\mathbb{E}[I_{pst}^k|\mathcal{S}_t^k], I_{pst}^k]}{Var[I_{pst}^k]}. \tag{3-8}$$

where I_{pst}^k is used as shorthand for the final outcome of persistence or extinction for simulation k ($\mathbb{E}[I_{pst}^k|\mathcal{S}_{T_{max}}^k]$). As time approaches T_{max}, this standardized covariance increases to 1.

Using equations 3-6 and 3-7, $SCV(t)$ may be decomposed into the contributions of demographic stochasticity, sex ratio stochasticity, phenotypic stochasticity:

$$SCV_{I_{pst}}(t) \quad = \quad SCV_{I_{pst}}(0) \; + \; \frac{Cov[\mathcal{C}_{k,t}^N(I_{pst}), I_k]}{Var[I_{pst}^k]} \; + \; \frac{Cov[\mathcal{C}_{k,t}^F(I_{pst}), I_k]}{Var[I_{pst}^k]} \; + \; \frac{Cov[\mathcal{C}_{k,t}^P(I_{pst}), I_k]}{Var[I_{pst}^k]}. \tag{3-9}$$

Equivalents of equations 3-8 and 3-9 may be used to decompose the contributions of stochasticity to extinction time T_{ext} and recovery time T_{rec}.

To ensure that these metrics are robust, I also use an alternative method of measuring how well population outcomes can be predicted prior to their occurrence. The variance in expected outcomes at time t is divided by the variance in actual outcomes at time T_max. This variance ratio

(VR) for persistence is given by

$$VR_{I_{pst}}(t) := \frac{Var[\mathbb{E}[I^k_{pst}|\mathcal{S}_{k,t}]]}{Var[I^k_{pst}]}. \tag{3-10}$$

Using equations 3-6 and 3-7, the variance in the numerator may then be decomposed into components of variance due to each form of stochasticitiy and covariances between forms of stochasticity:

$$\begin{aligned} VR_{I_{pst}}(t) = VR_{I_{pst}}(0) + \frac{Var[\mathcal{C}^N_{k,t}(I_{pst})]}{Var[I^k_{pst}]} + \frac{Var[\mathcal{C}^F_{k,t}(I_{pst})]}{Var[I^k_{pst}]} + \frac{Var[\mathcal{C}^P_{k,t}(I_{pst})]}{Var[I^k_{pst}]} + \\ \frac{Cov[\mathcal{C}^N_{k,t}(I_{pst}), \mathcal{C}^F_{k,t}(I_{pst})]}{Var[I^k_{pst}]} + \frac{Cov[\mathcal{C}^N_{k,t}(I_{pst}), \mathcal{C}^P_{k,t}(I_{pst})]}{Var[I^k_{pst}]} + \frac{Cov[\mathcal{C}^F_{k,t}(I_{pst}), \mathcal{C}^P_{k,t}(I_{pst})]}{Var[I^k_{pst}]}. \end{aligned} \tag{3-11}$$

The standardized covariance decomposition is used in the main results while supplement 3-2 recapitulates the main results using the variance ratio decomposition (figures S3-1 to S3-6).

Results

Figure 3-1 illustrates the core dynamics of evolutionary rescue. After the environmental change, populations decline towards extinction while simultaneously evolving toward the new phenotypic optimum. Some populations (black lines) evolve sufficiently fast that they are able to recover while the remainder (red lines) decline to extinction while exhibiting increasingly erratic changes in mean phenotype.

The chosen parameters result in four parameter values where the persistence probability is non-trivial (two with persistence probability above 50% and two with persistence probability below 50%), two where the persistence probability is close to zero, and two where the persistence probability is 100% (table 3-1). This allows for six parameter sets in which time to extinction can be examined and six parameter sets in which time to recovery can be examined. As expected, greater heritability, greater phenotypic variance, and less severe environmental change improve population outcomes across the board: increasing persistence probability, increasing the time to

extinction, and decreasing the time to recovery (table 3-1). Increasing phenotypic variance while lowering heritability had the effect of increasing the variance in the environmental component of phenotype (σ_e^2), which lowered the persistence probability.

Figure 3-2 shows the standardized covariances between persistence or extinction at time T_{max} and the predicted probability of extinction at time t (or "total stochasticity", dashed grey lines), the effect of demographic stochasticity on this prediction (black lines), the effect of sex-ratio stochasticity (blue lines), and the effect phenotypic stochasticity (red lines). Figure 3-3 shows the covariances between predicted time to extinction and actual extinction times and Figure 3-4 shows the covariances between predicted times to recovery and actual recovery times. Figures 3-2 and 3-3 are accompanied by histograms of extinction times, while figure 3-4 is accompanied by a histogram of recovery times.

There are several general observations that hold across the parameter choices. The covariance between the predicted probability of persistence and actual persistence is low at the time of first extinction (between 0.2 and 0.3) and only approaches 1 when most of the populations that will go extinct have already gone extinct. This implies that the ability to predict which populations will go extinction and which will persist, even given perfect information, is limited unless the populations are very close to extinction. This is confirmed by figure S3-8, which shows (for $\mu_w = 5.2$, $\sigma_z^2 = 3$, and $h^2 = 0.25$) that while most population's predicted probabilities of persistence are near zero in the one or two generations before extinction, this predictability rapidly evaporates as one looks further back in time, to such a degree that 14% of populations that eventually go extinct actually have a higher predicted probability of persistence a mere four generations before extinction than they did at time 0. The predictability of extinction time exhibits a similar pattern to persistence, with low predictability at the time of first extinction and high predictability only when many populations have already gone extinct. On the other hand, there is a high covariance between predicted recovery times and actual recovery times prior to the time of first recovery. This indicates that the time to recovery for most recovering populations becomes largely deterministic before they reach their initial population size.

The effect of phenotypic stochasticity on persistence probability early during rescue has a larger covariance with persistence or extinction at T_{max} than does the effect of demographic and sex-ratio stochasticity on persistence probability (figure 3-2). Later during rescue, the covariance between the effect of demographic and sex-ratio stochasticity on persistence probability and I_{pst} increases at a faster rate than for the effect of phenotypic stochasticity. This is particularly apparent in figures 3-2a and 3-2b, where the cumulative effects of phenotypic stochasticity, demographic stochasticity, and sex ratio stochasticity on population persistence ultimately have similar covariance with I_{pst} to the cumulative effect of phenotypic stochasticity despite the cumulative effect of phenotypic stochasticity having a higher covariance prior to generation 15. The covariance between cumulative effect of phenotypic stochasticity and I_{pst} is also greater when phenotypic variance is larger. This is consistent with the fact that phenotypic stochasticity includes both stochasticity due to random mating and recombination (which scales with genetic variance σ_g^2) and stochasticity due to developmental noise (which scales with σ_e^2).

The most apparent difference between figures 3-2 and 3-3 is that the covariance between final extinction times and the effect of phenotypic stochasticity on mean extinction times is substantially less than the covariance between I_{pst} and the effect of phenotypic stochasticity on extinction probability. The relative covariances of demographic stochasticity and extinction time versus sex-ratio stochasticity and extinction time vary substantially across parameter values, with the effect of sex-ratio stochasticity exhibiting the largest covariance with extinction time when persistence probability is highest and the effect of demographic stochasticity exhibiting the largest covariance with extinction time when persistence probability is lowest (figure 3-3 a and e). It is not immediately clear why this is the case, but the pattern appears to be related to the proportion of populations that go extinct due to mating failure (when their are either no males or no females) versus reproductive failure. In the case of figure 3-3a, a full 86.8% of populations go extinct due to mating failure while for figures 3-3e and 3-3f, only 73.0% and 71.6% of populations go extinct due to mating failure. Panels b, c, and d all have intermediate values (83.2%, 78.2%, and 78.2% respectively).

The effect of phenotypic stochasticity on recovery time has a higher covariance with final recovery time than the effects of demographic or sex-ratio stochasticity when either phenotypic variance or heritability are low, but not when both are high (in which case the effects are comparable; compare figures 3-4a,b,c, and d to figures 3-4e and f). Figure S3-7 demonstrates that this is not because, all else being equal, the magnitude of phenotypic stochasticity is small when phenotypic variance and heritability are high, but rather because the effect of phenotypic stochasticity on recovery time is greatest when populations have yet to fully adapt to the changed environment. When heritability or phenotypic variance are low, adaptation occurs more slowly, increasing the period of time in which phenotypic stochasticity may significantly affect the speed of recovery.

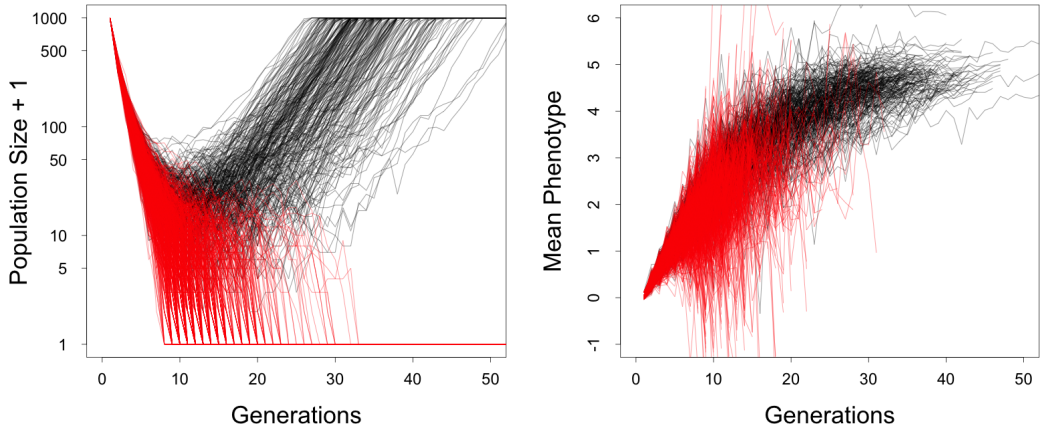

Figure 3-1: The change in log population size (a) and mean phenotype (b) over the course of evolutionary rescue for 1000 simulated populations. Black lines denote populations that recover to their initial size ($N_0 = 1000$) while red lines denote populations that go extinct. Simulations are truncated at extinction and recovery. The parameter values are $\mu_w = 5.5$, $h^2 = 0.25$, $\sigma_z^2 = 3$, and $\lambda_{max} = 1.5$

Parameters			Outcomes		
σ_z^2	h^2	μ_w	$\bar{I}_{pst}$	$\bar{T}_{ext}$	$\bar{T}_{rec}$
1	0.25	5.2	0.3%	9.7	NA
1	0.25	5.5	0.0%	8.1	NA
1	0.75	5.2	76.6%	14.9	27.3
1	0.75	5.5	41.7%	12.2	29.0
3	0.25	5.2	52.7%	15.4	34.0
3	0.25	5.5	27.2%	13.0	34.8
3	0.75	5.2	100%	NA	13.1
3	0.75	5.5	100%	NA	14.9

Table 3-1: The persistence probabilities ($\bar{I}_{pst}$), mean extinction times ($\bar{T}_{ext}$) and mean recovery times ($\bar{T}_{rec}$) over 1000 simulations for factorial combinations of high and low phenotypic variance (σ_z^2), heritability (h^2), and change in optimal phenotype (μ_w).

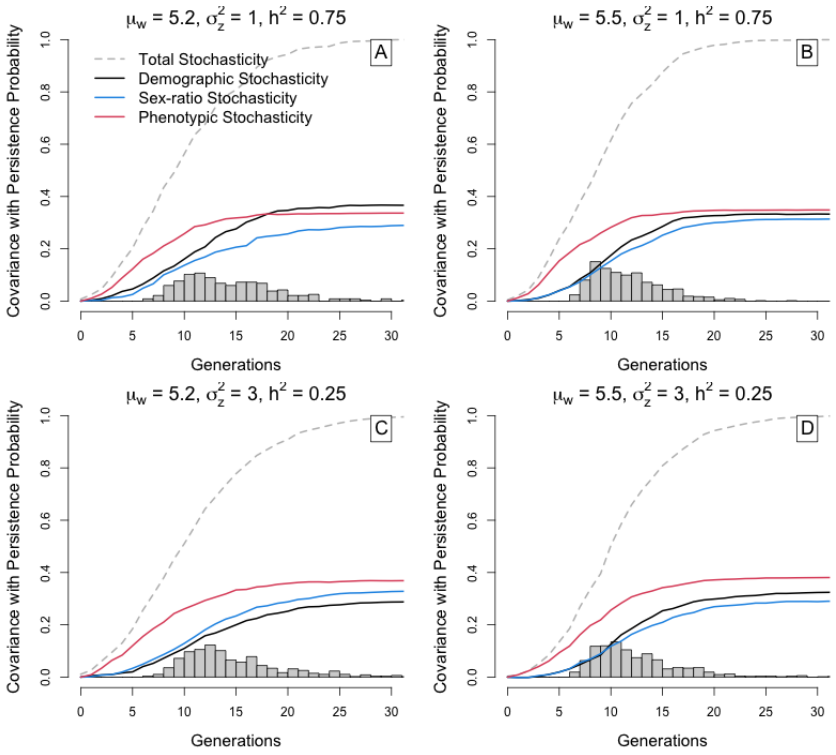

Figure 3-2: The covariances between the cumulative effects of demographic stochasticity (black line), sex-ratio stochasticity (blue line), and phenotypic stochasticity (red line) on persistence probability and the binary indicators I^k_{pst} of extinction or persistence at time T_{max}, scaled by the variance across I^k_{pst}'s. Covariances in each panel are computed over 1000 simulations. The histogram in each panel represents the simulated extinction times for that parameter value.

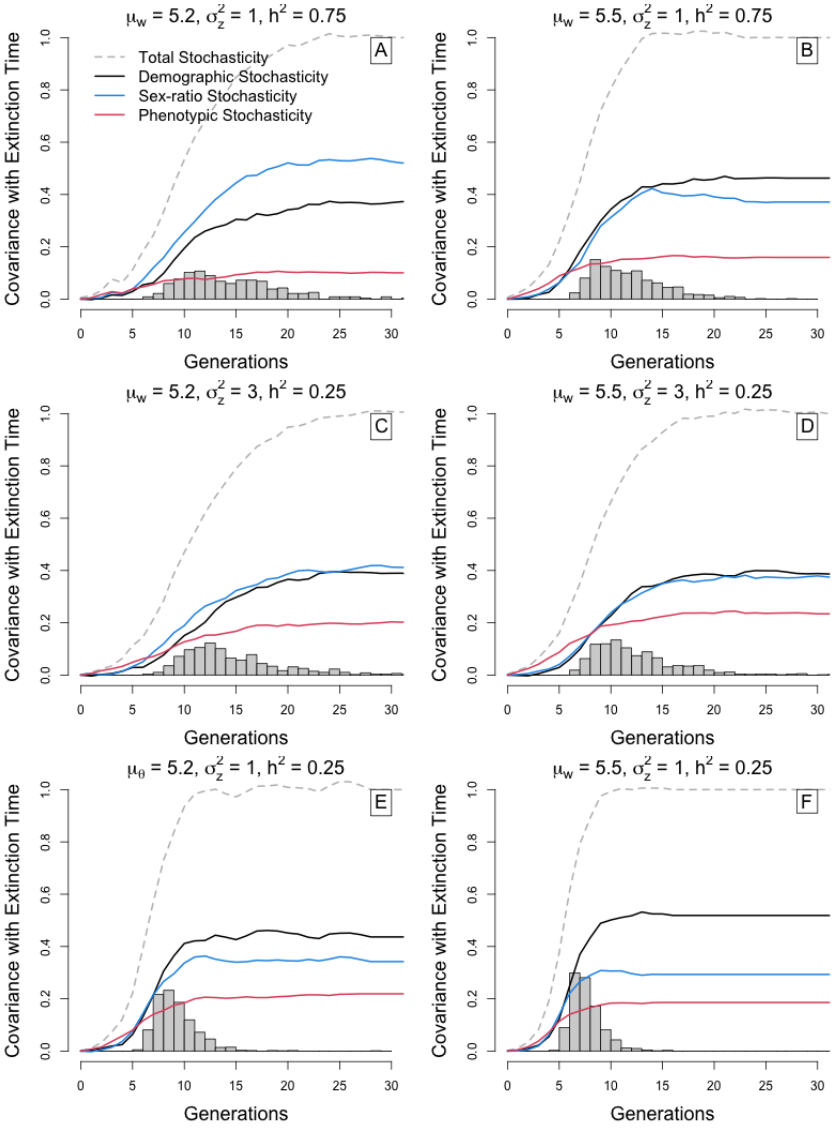

Figure 3-3: The covariances in the cumulative effects of demographic stochasticity (black line), sex-ratio stochasticity (blue line), and phenotypic stochasticity (red line) on mean extinction time and realized extinction times T_{ext}^{k}, scaled by the variance across T_{ext}^{k}'s. Covariances are computed over 234 (a), 583 (b), 473 (c), 728 (d), 997 (e), and 1000 (f) extinction-bound populations. The histogram in each panel represents the simulated extinction times for that parameter value.

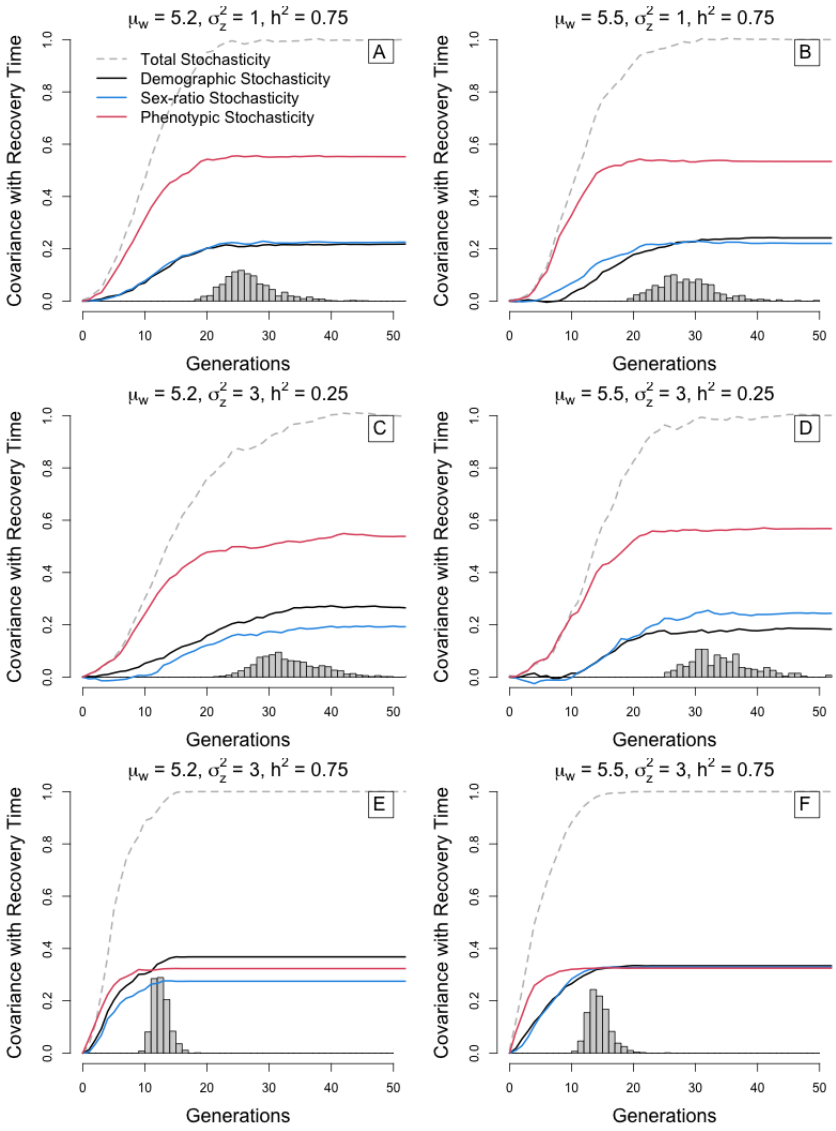

Figure 3-4: The covariances in the cumulative effects of demographic stochasticity (black line), sex-ratio stochasticity (blue line), and phenotypic stochasticity (red line) on mean recovery time and realized recovery times T_{rec}^{k}, scaled by the variance across T_{rec}^{k}'s. Covariances are computed over 766 (a), 417 (b), 527 (c), 272 (d), 1000 (e), and 1000(f) recovery-bound populations. The histogram in each panel represents the simulated recovery times for that parameter value.

Discussion

Evolutionary rescue, the ability of evolution to prevent species extinction, is a topic of great interest for conservation, medicine, and pest management (reviewed in Bell 2017). The success or failure of evolutionary rescue depends on the relative rates of evolution and population decline, and at small population sizes variation in individual births and deaths generates stochasticity that affects both of these rates. I developed a general method for quantifying how the timing and form of stochasticity affects future population outcomes. I then applied this method to examine the relative importance of demographic stochasticity, sex ratio stochasticity, and phenotypic stochasticity on probability of persistence, time to extinction, and time to recovery for populations undergoing evolutionary rescue in response to an abrupt environmental change. This work elucidates how the effect of stochasticity on population outcomes changes over the course of evolutionary rescue and depends on subtly on type of outcome under consideration.

I observed low predictability (defined as the regression coefficient of predicted outcomes at time t on the actual outcomes) of persistence and extinction time prior to the time of first extinction (figures 3-2 and 3-3). This suggests limited ability to forecast when and how populations will go extinct rather than just compute the probability of extinction. Indeed, many extinction-bound populations had a substantial persistence probability as few as three generations prior to their extinction (figure S3-8). In the case of time to extinction, part of this low predictability is due to the fact that a population's expected time to extinction increases the longer the population survives. Even for a very small population, unless extinction is guaranteed in the next generation, the distribution of extinction times will always be skewed towards persisting longer, inflating the expected extinction time. This, in turn, means that the final stochastic event that results in extinction has a disproportionately large influence on the regression coefficient of predicted extinction times on actual extinction times.

However, recovery time was highly predictable prior to the time of first recovery (figure 3-4). This is likely because the magnitude of stochasticity is inversely proportional to population size.

The initial population size ($N_0 = 1000$) was large enough that by the time the first population recovered to its initial size, the remaining recovery-bound populations had recovered to a point where subsequent stochasticity had a negligible effect on the time at which they recovered to their initial size.

I further found that early during evolutionary rescue, phenotypic stochasticity had a greater covariance with population persistence than did demographic stochasticity or sex ratio stochasticity, while later on the covariances of the three forms of stochasticity with persistence were comparable (figure 3-2). This indicates that phenotypic stochasticity was the most important form of stochasticity for persistence early during evolutionary rescue, but that demographic stochasticity and sex-ratio stochasticity increase in relative importance as time goes on. The importance of phenotypic stochasticity also scales with developmental error, which is unsurprising given that it represents an increase in the stochasticity of phenotypic determination. This suggests that during the declining phase (see figure 3-1), when the populations had not yet adapted to the new environment, the effect of stochasticity on the rate of adaptation is more important than the effect of stochasticity on population declines. When populations have declined to low size, demographic stochasticity and sex ratio stochasticity increase in importance as chance extinction events become more likely.

Phenotypic stochasticity is substantially less important in determining extinction time than it is in determining persistence (compare figures 3-2 and 3-3). This difference may be explained by the fact that extinction or persistence ultimately depends on the success of rapid evolution, while populations on extinction-bound trajectories are necessarily those that fail to evolved rapidly enough to be rescued. Extinction is ultimately a demographic phenomenon, so while phenotypic stochasticity may be an important component of variation in extinction times early on, demographic stochasticity and sex-ratio stochasticity are ultimately the deciding factors of when a population goes extinction. This observation is further supported by the fact that the relative importance of demographic stochasticity and sex-ratio stochasticity is associated with the odds of a population going extinct due to reproductive failure relative to mating failure.

Conversely, phenotypic stochasticity is more important for recovery time than for persistence (compare figures 3-2 and 3-4). Time to recovery is ultimately dependent on how quickly rescue occurs, which in turn is dependent on the rate of evolution. When genetic variance is high, the average rate of evolution is high and variation in that rate due to phenotypic stochasticity is small, in part due to the fact that the population spends less time at low population size (figures S7e and f). When genetic variance is low, the average rate of evolution is lower and variation in the rate due to phenotypic stochasticity is larger (figures S3-7a, b, c, and d). This leads to the conclusion that relative importance of phenotypic stochasticity in determining time to recovery depends heavily on the rate of evolution, all else being equal.

Many other results align with previous work: Greater heritability and phenotypic variance promote greater likelihood of persistence and faster recovery, while larger environmental shifts have the opposite effect (Agashe et al. 2011; Barrett and Schluter 2008; Bell 2017; Burger and Lynch 1995; Gomulkiewicz and Holt 1995; Lynch and Lande 1993; Orr and Unckless 2008). Most extinction occurs within a short window of time (Barfield and Holt 2016) and the distribution of both extinction times and recovery times is skewed towards longer times (e.g. Barfield and Holt 2016; Orr and Unckless 2014).

To the author's knowledge, the approach used here has no direct comparison within the existing evolutionary rescue literature. The closest analogs are previous studies, examining evolutionary rescue at a single locus, that have derived analytical expressions for the probability of rescue in order to measure how the likelihood of rescue through de novo mutation changes over time (Orr and Unckless 2008), to calculate the relative likelihood of rescue through standing variation versus de novo mutation (Orr and Unckless 2014), and to classify scenarios of rescue based on the number and effect size of de novo mutations (Osmond et al. 2020). These approaches assume asexual reproduction to allow for analytic tractability. For a sexually reproducing population whose fitness is determined by a quantitative trait, the focus of the present study, this analytic tractability is lost as distinct allelic combinations can no longer be represented by independent lineages. By directly calculating the probability of rescue using simulations, the approach

presented here allows for evolutionary rescue in populations with more complex demographies to be classified into qualitatively different scenarios based on how stochastic events during rescue impact population trajectories.

In an effort to isolate the effects of stochasticity during evolutionary rescue, the present study intentionally minimized the effect of variation in the initial population state due to stochasticity prior to the environmental change on the likelihood of persistence or extinction. As noted by Barfield and Holt (2016), variation in the genotypic distribution due to pre-change genetic drift may induce substantial variability in the probability of evolutionary rescue (though not in the time to extinction for a population experiencing gradual environmental change; Orive et al. 2019). The effect of this variability may easily be measured within the general framework presented here by comparing the probability of extinction given the distribution of possible initial states to the probability of extinction when the exact initial state is known. This comparison would also allow for the effect of measurement error on extinction probability to be quantified (at least within a modelling framework where the true parameters and true initial state are known).

I have assumed throughout that fitness is determined solely by an individual's phenotype and is not affected by population size. This was done to focus solely on the relationship between different forms of stochasticity and extinction. However, deterministic Allee effects, such as mate limitation or the accumulation of deleterious alleles, also play an important role in driving the extinction of small populations (Lande 1988). It is well know that stochasticity may contribute to extinction by driving populations below Allee thresholds (Dennis 1989, 2002) and there has been recent interest in characterizing the interactions between different forms of genetic Allee effects, such as inbreeding depression, mutation accumulation, and the loss of variation at loci under balancing selection, that may generate "eco-evolutionary extinction vortices" (Luque et al. 2016; Nabutanyi and Wittmann 2021*a*,*b*; Wittmann et al. 2018). An important future direction is to utilize the method presented here for partitioning the effect of stochasticity on extinction risk to examine how different forms of stochasticity influence time to extinction in these complex extinction scenarios.

This study has provided a detailed examination of how stochasticity affects the likelihood of persistence, the time to extinction, and the time to recovery of populations undergoing evolutionary rescue. I found that while the ability to predict in advance whether a population will persist or go extinct is low, the best early predictor is whether the population has evolved more quickly or slowly than expected (i.e. the effect of phenotypic stochasticity). This study has also presented a general framework for partitioning the effects of different forms of stochasticity on population outcomes that can be used as a template for understanding the process of extinction beyond evolutionary rescue. It therefore represents an important step in better understanding when, how, and why populations go extinction.

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