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Why So Variable: Can Genetic Variance in Flowering Thresholds Be Maintained by Fluctuating Selection?

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ABSTRACT: We use integral projection models (IPMs) and individual-based simulations to study the evolution of genetic variance in two monocarpic plant systems. Previous approaches combining IPMs with an adaptive dynamics–style invasion analysis predicted that genetic variability in the size threshold for flowering will not be maintained, which conflicts with empirical evidence. We ask whether this discrepancy can be resolved by making more realistic assumptions about the underlying genetic architecture, assuming a multilocus quantitative trait in an outcrossing diploid species. To do this, we embed the infinitesimal model of quantitative genetics into an IPM for a size-structured cosexual plant species. The resulting IPM describes the joint dynamics of individual size and breeding value of the evolving trait. We apply this general framework to the monocarpic perennials *Oenothera glazioviana* and *Carlina vulgaris*. The evolution of heritable variation in threshold size is explored in both individual-based models (IBMs) and IPMs, using a mutation rate modifier approach. In the *Oenothera* model, where the environment is constant, there is selection against producing genetically variable offspring. In the *Carlina* model, where the environment varies between years, genetically variable offspring provide a selective advantage, allowing the maintenance of genetic variability. The contrasting predictions of adaptive dynamics and quantitative genetics models for the same system suggest that fluctuating selection may be more effective at maintaining genetic variation than previously thought.

Keywords: genetic variance, adaptive dynamics, quantitative genetics, flowering threshold, integral projection model.

Introduction

Many recent studies have emphasized the interplay between ecological and evolutionary processes leading to eco-evolutionary dynamics (e.g., Hendry and Kinnison 1999; Hairston et al. 2005; Smallegange and Coulson 2013; Thompson 1998, 2013; Hendry 2017). To understand this interplay, a wide range of methods have been developed to predict and understand the determinants of trait change. Integral projection models (IPMs) are one widely used framework for projecting trait dynamics (Easterling et al. 2000; Ellner and Rees 2006; Rees and Ellner 2009). These models project a continuous trait distribution from one census to the next by allowing the processes that determine an individual's fate (e.g., survival, growth) to depend on an individual's state (e.g., size, age, sex). However, they typically have not dealt with the transmission of genetic quantities (e.g., alleles or breeding values; but see Vindenes and Langangen 2015; Childs et al. 2016; Rees and Ellner 2016; Coulson et al. 2017, 2011).

Evolution in structured populations has commonly been studied by combining ideas from adaptive dynamics (Geritz et al. 1997) with structured population models (Caswell 2001; de Roos and Persson 2013; Ellner et al. 2016) to predict evolutionarily stable strategies (ESSs). In particular, IPMs have often been used to study evolution of size-dependent flowering in monocarpic perennial plants (e.g., Rees and Rose 2002; Childs et al. 2004; Rees et al. 2006; Metcalf et al. 2008; Rees and Ellner 2016). The strength of this approach is that selection arises from the ecological interactions built into the model rather than from arbitrarily fixed fitnesses. A weakness of this approach is that it assumes mutation-limited evolution, such that adaptive evolution can be viewed as a series of nonoverlapping trait substitutions. Consequently, the condition for maintaining genetic variation in the adaptive dynamics framework is that no genetically monomorphic population can repel invasion by all rare mutants with a different trait value. The assumption of mutation-limited evolution is at odds with the substantial genetic variation found in many natural populations (e.g., Geber and Griffen 2003; Charmantier et al. 2014). In both constant and stochastic environment models, previous studies of the relationship between the probability of flowering and plant size using adaptive dynamics methods predicted evolution to a single ESS with a step function relationship, such that plants smaller than some critical size T never flowered, whereas plants

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larger than T always flowered (Rees and Rose 2002; Childs et al. 2003, 2004; Rees and Ellner 2009, 2016). This prediction of a single critical size for flowering (the flowering threshold) implies that no genetic variation for flowering threshold can be maintained by selection, even when the environment fluctuates from year to year.

In a fluctuating environment, there are five processes by which it is currently thought that selection might favor the maintenance of adaptive genetic variability. These processes are listed below.

Heterozygotes advantage. Temporally fluctuating selection can give heterozygotes the highest geometric mean fitness, even though one or both homozygotes have higher arithmetic mean fitness (Gillespie 1991). Wittmann et al. (2017) recently demonstrated that this mechanism can maintain multilocus polymorphisms in a seasonal environment, if seasonal reversal in dominance at each locus allows heterozygotes to track the seasonally varying optimal phenotype accurately enough, while each homozygote has high fitness in one season and low fitness in the other.

The storage effect. If the optimal genotype varies either spatially or temporally, then genetic variability can be maintained by the storage effect when the variance in the trait optimum is sufficiently high (Ellner and Hairston 1994; Svardal et al. 2015). The maintenance of genetic variance with purely temporal variation requires generational overlap (see Ellner and Hairston 1994; Sasaki and Ellner 1995; Svardal et al. 2015) so that genotype fitnesses are buffered in the way required for coexistence through the temporal storage effect (Chesson 1994).

Tracking. When the environment changes in a predictable manner either deterministically or with substantial temporal autocorrelation, then genetic variance can be maintained because this allows the population to track changes in the environment (Mather 1943; Slatkin and Lande 1976; Charlesworth 1993). This effect cannot occur in the adaptive dynamics analysis of conditions maintaining genetic variation because both the resident and the invader populations are monomorphic, and so all offspring play the same strategy.

Disruptive selection. In the quantitative genetics framework, disruptive selection occurs when the covariance between relative fitness and the squared deviations from the trait mean is positive (Lande and Arnold 1983, eq. [13b]). This implies that individuals in the tails of the trait distribution have higher relative fitness, which selects for increased variance. When the trait optimum varies, a large mismatch between the current optimum and the current genotype distribution can produce disruptive selection (Layzer 1980; Kawecki 2000). For example, the standard nor-optimal fitness function, $W = \exp(-(x - \theta_t)^2/2\omega^2)$, has a positive second derivative whenever phenotype x is more than $\pm\omega$ units from the optimum θ_t, and so large shifts in θ_t can generate disruptive selection. If x is Gaussian with mean $\bar{x}$ and variance V_x, then the population will experience disruptive selection (quadratic selection gradient, $\gamma > 0$; Lande and Arnold 1983, eq. [14a]) if $(\bar{x} - \theta_t)^2 > V_x + \omega^2$. In the adaptive dynamics framework, this mode of disruptive selection cannot contribute to the predicted conditions for maintaining genetic variance because the adaptive dynamics analysis is local; branching from monomorphism to polymorphism occurs because of a concave-up fitness function at the singular trait value. A quantitative genetics model can also have disruptive selection because of what the fitness function looks like at the tails of a broad trait distribution.

Bet hedging. This refers to variation among the offspring of an individual that reduces the between-year temporal variance in fitness, thus increasing geometric mean fitness, at the expense of not maximizing average fitness (Seger and Brockmann 1987). As with tracking, this effect cannot occur in an adaptive dynamics analysis of the conditions for stable genetic polymorphism, as both the resident and invader populations are monomorphic and so all offspring play the same strategy.

In addition to these mechanisms where variation is adaptive, nonadaptive variation can be maintained by mutation-selection balance; for an overview, see Bulmer (1989). Roff (1998) considers mutation-selection balance for threshold traits, such as the flowering threshold undergoing directional selection.

The analyses of genetic variation maintained by the storage effect of Ellner and Hairston (1994) uses adaptive dynamics methods to determine when alternative alleles can invade. The conditions for maintenance of genetic variance are sufficiently large generation overlap and high enough temporal variation in the optimal trait value that there is disruptive selection operating on the trait at the evolutionarily singular strategy (where there is no directional selection); in adaptive dynamics this is called a branching point (Geritz et al. 1997). Svardal et al. (2015) recently extended this analysis, finding conditions for adaptive branching when selection varies spatially as well as temporally. Within the adaptive dynamics approach, the condition for polymorphism to arise is derived by considering invasion of a monomorphic resident population by a monomorphic invader and determining when no monomorphic resident can repel all monomorphic invaders. Within that scenario, the first, third, fourth, and fifth of the processes listed above cannot occur. The conditions for maintaining genetic variation in the adaptive dynamics framework are thus rather restrictive. When there is an ESS (i.e., no branching) and the population has evolved to the ESS, the storage effect also cannot operate because there is stabilizing selection and rare alleles cannot invade. The key conceptual difference between the adaptive dynamics and quantitative genetics analysis of the conditions for maintaining genetic variation is that the former looks at the conditions for a rare invading allele to spread, while the

latter asks how moments of the breeding value distribution evolve.

The prediction that genetic variation in the flowering threshold could not be maintained adaptively is, however, at odds with the shallower relationships between plant size and the probability of flowering observed in natural populations (Metcalf et al. 2003) and with the extensive genetic variation in the threshold size for flowering found in breeding experiments (Wesselingh and de Jong 1995; Wesselingh and Klinkhamer 1996; Simons and Johnston 2000). Given the mismatch between theory and data, it is natural to ask whether this is a consequence of the genetic assumptions used in adaptive dynamics theory and whether these predictions are robust to the inclusion of more realistic genetics and breeding systems. In particular, quantitative genetics models assume that all matings produce a range of genotypes, and so it is possible that genetic variation could be maintained by tracking, disruptive selection, and bet hedging.

To test this hypothesis, we develop IPMs for cosexual plant species and embed within them a quantitative genetic model for evolution of the flowering threshold. Quantitative genetic analysis of data from field populations has developed rapidly in recent years (Hadfield 2010; Charmantier et al. 2014), and our models illustrate how this information can be merged with realistic ecological models. We use our models to ask whether the production of genetically variable offspring (as assumed by quantitative genetic models) can be selectively advantageous by modeling the joint evolution of flowering threshold and a mutation rate modifier (Koren et al. 2014; Raynes and Sniegowski 2014) affecting the loci controlling the flowering threshold.

Previous studies (reviewed in Wittmann et al. 2017) have concluded that when a trait is determined by many loci with additive effects within and among loci, fluctuating selection can maintain genetic variability at only a very small number of loci. Our approach leads to the opposite conclusion. There is no contradiction here; the previous studies do not include several features of our models, including overlapping generations, strong selection with large between-year variation, and selection that results from data-driven models for the ecological interactions between competitors differing in phenotype. Which (or how many) of these differences are responsible for the difference in outcomes is an important question but beyond the already broad scope of this article.

A crucial aspect of our approach is that size per se (or size at a given age, time, or event) is not the evolving trait in our models, nor should it ever be when heritable trait evolution is combined with a size-structured IPM. Please read the preceding sentence again because it is essential for understanding this article, but it may be exactly opposite to what you were expecting. In this article, the evolving heritable trait is not size at any given time or age, and it is not the actual size at flowering. Because flowering decisions are made only once a year, and because growth has a random component in our models, individuals with a given flowering threshold will flower at a range of different sizes at or above that threshold. The evolving trait in our framework can be something that impacts the size dynamics of individuals, such as the flowering threshold or the intercept parameter in a regression equation for growth rate versus size. But making size per se the evolving trait—using observed parent-offspring size correlations to model inheritance—has both conceptual and practical problems that are explained by Wilson et al. (2010), Chevin (2015), and Janeiro et al. (2017). Instead, in our approach, size at any age or size at an event such as flowering is an emergent consequence of prior growth rates, which are affected by individuals' traits and by chance variation in growth.

This article is structured as follows. We first briefly review the context for this study: demographic models for monocarpic perennial plants with size-dependent flowering probability and adaptive dynamics predictions of flowering strategy evolution. We then introduce models in which flowering strategy is modeled as a heritable quantitative trait using the infinitesimal model from quantitative genetics (Bulmer 1971; Barton et al. 2017; Turelli 2017). The standing level of heritable variation in those models, as measured by the genic variance, is determined by mutation-drift balance (genic variance is the value that the additive genetic variance would take if all loci on all chromosomes were independent, given the population-level allele frequencies at each locus [Walsh and Lynch 2018, ch. 16]). Therefore, to study selection on the level of heritable variation, we introduce new models where the mutation rate at flowering strategy loci is determined by a mutation rate modifier, and we study the mechanisms whereby selection can favor increased mutation rate and high levels of heritable variation.

Background

Demographic Models

Our IPMs are based on field studies of two monocarpic plants, *Oenothera glazioviana* and *Carlina vulgaris*. *Oenothera* demography is described by Rose et al. (2002), and *Oenothera* demography was studied by Kachi and Hirose (Kachi 1983; Kachi and Hirose 1983, 1985). The models have been described in detail elsewhere (Rees and Rose 2002; Ellner et al. 2016), so we will be brief here.

The general IPM equation for a size-structured population is

$$n(z', t+1) = \int (F(z', z) + P(z', z)) n(z, t)\, dz. \qquad (1)$$

Here $n(z, t)$ is a density function describing the distribution of size z individuals at time t, $F(z', z)\,dz'$ describes the production by size z individuals of offspring whose size is in the range from z' to $z' + dz'$, and $P(z', z)dz'$ describes survival and growth of size z individuals into range from z' to $z' + dz'$.

The size measures in the field studies were log-tranformed rosette diameter for *Oenothera* and log-transformed length of the longest leaf for *Carlina*. The fecundity kernel F includes both seed production and survival of seeds to become new recruits of various sizes the following year. Seed production depends on parent size and on parent genotype in our evolutionary models. In all of our models, seed survival is the same for all seeds, regardless of any parent or seed attributes.

The demographic functions in both models are simple linear or generalized linear models for the effect of current size on demographic rates, with random year effects in the *Carlina* model. For example, the probability of survival (if not flowering) is modeled by a fitted logistic regressions in which the logit of survival probability is a linear function of size z. Because flowering decisions are made once a year, on the basis of size at that time, individuals with the same flowering threshold will flower at a range of ages and sizes because of variability in how many years it takes them to reach a size at which flowering is likely. The *Oenothera* model is deterministic (constant environment); the *Carlina* model is stochastic, with environmental variation modeled through partially correlated random effects in the regression models and random variation in the number of new recruits each year.

Both models incorporate negative density dependence, acting at only the recruitment stage, such that total recruit number is independent of the number and size of the parents in the previous year. The underlying biological assumption is that recruitment is limited by the number of available sites suitable for new recruits to establish successfully, and sufficient seeds are always produced to saturate those sites. The *Oenothera* IPM can therefore be written as

$$n(z', t + 1) = \mathcal{R}c_0(z') + \int P(z', z)n(z, t)\,dz, \qquad (2)$$

where $\mathcal{R}$ is the number of new recruits. The seedling contribution to the subsequent year's population is $\mathcal{R}c_0(z')$, the number of recruits multiplied by the frequency distribution of seedling size, $c_0(z')$. The survival kernel is $P(z', z) = (1 - p_b(z))s(z)G(z', z)$, where $p_b(z)$ and $s(z)$ are the probabilities of flowering and survival (conditional on not flowering), and the growth kernel $G(z', z)$ is the probability density for survivors' size z' conditional on their initial size z. The demographic rate equations and parameters (e.g., regression coefficients) are given in table 1.

The *Carlina* model is very similar in structure, the key difference being that the parameters for the demographic functions and the total number of recruits $\mathcal{R}$ vary from year to year. Rees and Ellner (2009) give a detailed description of model construction and parameter estimation, and the parameter estimates are given in table 2.

In both cases, the predictions of the published models are in good agreement with empirical observations (Rose et al. 2002; Rees and Ellner 2009). We therefore use the published models here rather than considering more general nonlinear alternatives (Rees et al. 2014). Except when stated otherwise, all simulations and analyses in this article use the model equations and parameter values in tables 1 and 2. In both models the time unit is 1 year. For a description of the IBMs, see "Testing the Approximations" in appendix F (apps. A–K are available online).

Adaptive Dynamics Modeling of Flowering Threshold Evolution

Previous studies of flowering threshold evolution in *Oenothera* and *Carlina* have all reached the same conclusion: the only evolutionarily stable state is monomorphic at a unique flowering threshold that is an ESS (Rees and Rose 2002; Childs et al. 2003, 2004; Rees and Ellner 2009, 2016). However, all of those studies used an adaptive dynamics

Table 1: Estimated demographic functions in the *Oenothera* integral projection model (Rees and Rose 2002)

Demographic process	Model	Parameter estimates
Recruit size c_0 (z')	$z' = a_R + \omega$	$a_R = -.08$, $\omega \sim N(0, .76)$
Rosette growth $G(z', z)$	$z' = a_0 + b_z z + \epsilon$	$a_0 = .96$, $b_z = .59$, $\epsilon \sim N(0, .67)$
Survival probability $s(z)$	$\text{logit}(s(z)) = m_0 + m_z z$	$m_0 = -.65$, $m_z = .75$
Flowering probability $p_b(z)$	$p_b(z) = 1$ if $z > T$, otherwise 0	$T = 2.64$, $V_0 = .04$
Seed production $b(z)$	$b(z) = \exp(A + B_z)$	$A = 1$, $B = 2.2$

Note: Here z is size (log-transformed rosette diameter) in year t, z' is the size the following year, and $N(\mu, \sigma)$ is a normal distribution with mean μ and standard deviation σ. The flowering threshold T is an evolving trait in our eco-evolutionary two-dimensional integral projection models with no assumed value. For flowering probability, the models used in this article use the sharp threshold equation given in the table, with the threshold as an evolving trait rather than a fixed parameter. The parameter column gives the parameters of the fitted logistic model described in app. E (available online); T is the value of z at which $p_b(z) = 0.5$ in the fitted logistic regression model. The seed production intercept $A = 1$ is arbitrary and immaterial because the total number of recruits each year is constant and allocated to parents of different types in proportion to their total seed production.

Table 2: Estimated demographic functions in the *Carlina* stochastic integral projection model (Rees and Ellner 2009)

Demographic process	Model	Parameter estimates
Rosette growth $G(z', z)$ and recruit size c_0 (z')	$z' = a_0 + b_z z + \epsilon$, $z' = a_R + \omega$	$b_z \sim N(.74, .13)$; $\epsilon \sim N(0, .29)$; $\omega \sim N(0, .50)$; a_0, $a_R \sim MVN(\mu, \Sigma)$; $\mu = (1.14, 3.16)$; $\Sigma = \begin{pmatrix} .037 & .041 \\ .041 & .075 \end{pmatrix}$
Survival probability $s(z)$	$\text{logit}(s(z)) = m_0 + m_z z$	$m_0 \sim N(-2.28, 1.16)$, $m_z \sim N(.90, .41)$
Flowering probability $p_b(z)$	$p_b(z) = 1$ if $z > T$, otherwise 0	$T \sim N(4.18, .54)$, $V_0 = .12$
Seed production $b(z)$	$b(z) = \exp(A + Bz)$	$A = 1$, $B = 2$

Note: Here z is size (log-transformed length of the longest leaf) in year t, z' is the size the following year, $N(\mu, \sigma)$ is a normal distribution with mean μ and standard deviation σ, and $MVN(\mu, \Sigma)$ is a multivariate normal distribution with mean vector μ and variance-covariance matrix Σ. For flowering probability, the models used in this article use the sharp threshold equation given in the table, with the threshold as an evolving trait rather than a fixed parameters. The parameter column gives the parameters of the fitted probit model described in app. E (available online); T is the value of z at which $p_b(z) = 0.5$ in the fitted probit regression model. The seed production intercept A is arbitrary; the number of recruits each year is drawn at random from the set of observed numbers in the original field study and allocated to parents of different types in proportion to their total seed production. The seed production slope B represents the assumption that seed production is proportional to (maximum leaf length)^2, a measure of rosette area.

approach (Geritz et al. 1998) rather than quantitative genetic modeling of trait evolution.

The adaptive dynamics approach used in these previous studies is illustrated in figure 1*A*, a pairwise invasion plot (PIP) for this article's *Carlina* model. The PIP was constructed by simulating pairwise competition between a resident population monomorphic for the threshold size for flowering and an invader with a different threshold and computing the long-run stochastic growth rate λ_S for the invader (for a full description of the methods, see Ellner et al. 2016, sec. 9.5). Adaptive dynamics assumes mutation-limited evolution, proceeding by sequential selective sweeps as a successful invader (one with $\lambda_S > 1$) replaces the resident. The *Carlina* PIP in figure 1*A* predicts that a series of sweeps (such as those indicated by the arrows) will converge onto the ESS (open circle), which could not be invaded. The mutual invasion plot constructed from the PIP (fig. 1*B*) shows pairs of strategies such that each can invade the other, leading to coexistence in a protected polymorphism. However, the coexistence region's boundary curves intersect at less than 90°, which implies that these polymorphisms are not evolutionarily stable (Geritz et al. 1999).

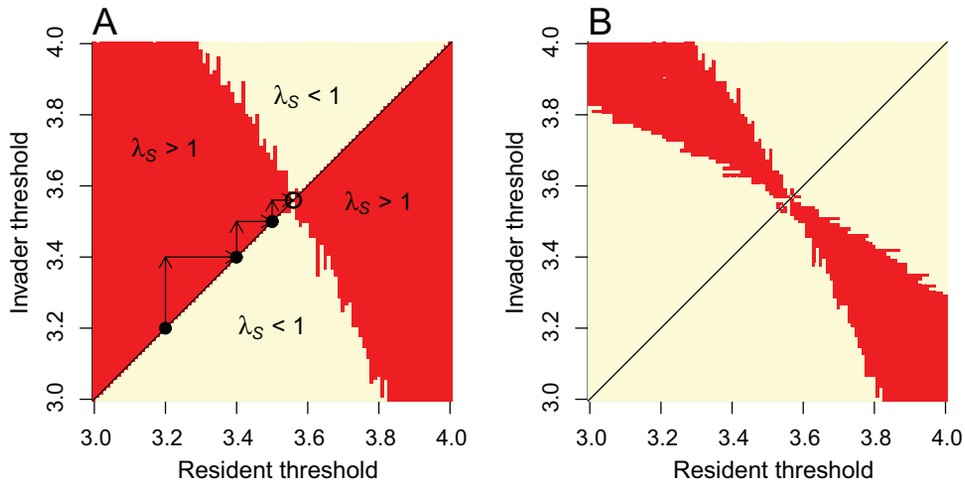

Figure 1: *A*, Pairwise invasion plot (PIP) for the threshold size for flowering T in *Carlina*. Each square represents one stochastic simulation of competition between a resident population with one value of T and a rare invader with a possibly different value of T (both modeled as haploids with offspring inheriting exactly their parent's T value). Resident and invader T values ranged from 3 to 4 in steps of size 0.01. Stochastic growth rate λ_S for the invader was estimated by allowing 500 years for the resident to reach steady state and then averaging invader low-density population growth rate over 10,000 years. The estimates of λ_S were smoothed slightly (using a bivariate tensor spline) before plotting. Open circle indicates the one noninvadable (evolutionarily stable strategy [ESS]) trait value; the shape of the red region where $\lambda_S > 1$ (i.e., invasion is successful) implies that successive invasions (such as the sequence illustrated by the arrows) will converge to the ESS, which is therefore convergence stable. *B*, Mutual invasion plot (red) squares are trait pairs such that each can invade the other on the basis of the PIP. Source file: Carlina PIP Thresh.R.

Resident and invader were both modeled as haploids for figure 1, with offspring inheriting exactly their parent's trait value, but the conditions for stability of a monomorphic ESS versus adaptive branching to a polymorphic population are the same under diploid or multilocus additive models for the underlying genetics (Geritz and Kisdi 2000; Spichtig and Kawecki 2004). The results in figure 1 therefore confirm that the previous conclusions still apply in this article's model, which assumes a sharp flowering threshold rather than a gradual increase in flowering probability with size.

Our models thus predict that heritable variation for flowering threshold cannot be maintained through fluctuating selection producing adaptive branching (in the adaptive dynamics sense) from a monomorphic ESS to an ecologically and evolutionarily stable genetic polymorphism of alleles coexisting through the storage effect (Ellner and Hairston 1994; Svardal et al. 2015). However, it is nonetheless possible that tracking, disruptive selection, or bet hedging could allow the maintenance of genetic variation, as explained in the introduction. To see whether this is true, we will now extend our models to ask whether heritable variation might be maintained when flowering threshold is modeled as a quantitative trait with multilocus inheritance.

Quantitative Trait Evolution in a Structured Population

In this section, we add to our demographic models evolution of a general quantitative trait, such as (but not exclusively) the flowering threshold. Previous models for quantitative trait evolution in these systems using IPMs (Rees and Ellner 2016) assumed haploid genetics where offspring inherit their parent's genotype plus a Gaussian random deviation modeling mutation. Here we use a more realistic evolutionary model. We consider an outcrossing, cosexual species, as this is very common in plants. Childs et al. (2016) presented a very general framework with separate sexes (and potentially age- as well as state-dependent demography); here we keep things much simpler by adding one heritable trait to the size-structured IPMs described above. The bookkeeping of the size × trait bivariate distribution is essentially the same as in the stage-structured case (Barfield et al. 2011) but notationally simpler because of the IPM formalism. Besides deriving the models for our case study systems, the main goal of this section is to clarify exactly what we are and are not assuming about the genetic basis of the trait.

Because our goal is to understand the maintenance of heritable variation, our models assume that the trait variation is the result of genetic variation rather than nonheritable phenotypic variation (e.g., plasticity or a phenotypic mixed strategy). We assume that the infinitesimal model describes the genetic basis of the trait (Barton et al. 2017). As Turelli (2017) notes, the infinitesimal model is really three nested approximations. The most basic is that the trait is determined by many loci with small additive effects within and across loci. The Gaussian descendants approximation further states that the distribution of offspring breeding values from a mating has a Gaussian distribution whose variance-covariance structure depends only on the relatedness of the parents and is unaffected by whether selection, mutation, or migration are occurring. Recent theory has rigorously validated this approximation, proving that its error decreases to 0 as the number of loci increases (Barton et al. 2017). The Gaussian population approximation further states that individuals on a pedigree have breeding values and phenotypes with a multivariate Gaussian distribution whose variance-covariance matrix depends only on the pedigree. The Gaussian population approximation is the basis for the animal model, one of the most widely used methods for estimating genetic parameters (Henderson 1950, 1975; Kruuk 2004). The Gaussian population approximation is the most questionable because trait distributions are affected by selection and so departures from Gaussian are expected (although often small even when selection is strong [Turelli and Barton 1994]).

Here we use the Gaussian descendants approximation but not the Gaussian population assumption. We assume that the parents of any new recruit are unrelated, so offspring from a mating have a Gaussian distribution of breeding values, with mean equal to the midparent value and a variance that is independent of parent breeding values and unaffected by selection. We make no assumptions about the population distribution of breeding values; it is a model output, calculated in the process of iterating the model. Because allele effects are additive, all genetic variance is additive genetic variance, and an individual's breeding value equals their expected phenotype.

To iterate our model, we need to compute the distribution of breeding values among survivors and new recruits. For survivors this is straightforward, as their breeding value does not change over time. The distribution of breeding values in recruits results from the distribution of breeding values in pollen and ovules produced by adults, the breeding values produced by random mating, and the additional variance in offspring breeding values from allele segregation.

The total abundance of pollen from parents with breeding value x is

$$S_{\mathrm{p}}(x) = \int p_{\mathrm{b}}(z,x)\, b_{\mathrm{p}}(z,x)\, n(z,x,t)\, dz, \tag{3}$$

where $b_{\mathrm{p}}(z,x)$ is the pollen production of plants of size z and breeding value x, $p_{\mathrm{b}}(z,x)$ is the probability of flowering, and $n(z,x,t)$ is a density function describing the distribu-

tion of individuals of size z and breeding value x. Note that we are assuming that flowering occurs before mortality; if mortality occurs first, then there would be a survival term in equation (3). To obtain the probability density function (i.e., relative abundance) of pollen breeding values, we normalize the abundance distribution by the total pollen abundance:

$$f_{\mathrm{p}}(x) = \frac{S_{\mathrm{p}}(x)}{\int S_{\mathrm{p}}(u)\,du}. \tag{4}$$

Similarly, defining $b_{\mathrm{o}}(z, x)$ as the ovule production of plants of size z and breeding value x, we have

$$f_{\mathrm{o}}(x) = \frac{S_{\mathrm{o}}(x)}{\int S_{\mathrm{o}}(u)\,du}. \tag{5}$$

Applying the Gaussian descendants property of the infinitesimal model, the expected breeding value of an offspring whose parents' breeding values are x_{p} (pollen parent) and x_{o} (ovule parent) is

$$x_{\mathrm{b}} = \frac{x_{\mathrm{p}} + x_{\mathrm{o}}}{2}. \tag{6}$$

The probability density function of expected offspring breeding values, assuming random mating, is therefore

$$f_{\mathrm{b}}(x) = 2\int f_{\mathrm{p}}(x_{\mathrm{p}}) f_{\mathrm{o}}(2x - x_{\mathrm{p}})\,dx_{\mathrm{p}} \tag{7}$$

(for the derivation, see app. A). Actual offspring breeding values under the infinitesimal model are the sum of expected breeding value and the segregation variance, which describes the variation among the offspring of any two parents. In the infinitesimal model, the segregation variance V_0 is half the genic variance σ_{a}^2 (recall that σ_{a}^2 is the value that the additive genetic variance σ_{A}^2 would take if allele frequencies at all loci on all chromosomes were independent, given the current allele frequencies at each locus [Walsh and Lynch 2018, chap. 16]). If x_b is the parental midpoint, then offspring breeding values (accounting for segregation) are given by

$$x^* = x_{\mathrm{b}} + \varepsilon, \tag{8}$$

where ε is a Gaussian random variable. So the probability density function of offspring breeding values is

$$f^*(x^*) = \int f_{\mathrm{b}}(x) f_{\mathrm{S}}(x^* - x)\,dx, \tag{9}$$

where f_S is the probability density function of a Gaussian random variable with mean 0 and variance V_0 (the segregation variance), describing the variance produced by random allele segregation (Slatkin and Lande 1976; Turelli and Barton 1994). Note that equation (9) incorporates the property of the Gaussian descendants approximation that the segregation variance is the same for all matings, regardless of the parents' breeding values. An important property of the infinitesimal model is that the genic variance and therefore the segregation variance V_0 are not changed by selection because each individual locus experiences very weak selection (Bulmer 1971); for simulations illustrating this property, see app. B. Extreme as this sounds, it has often been observed over many generations of directional selection (Barton and Keightley 2002).

The two-dimensional IPM describing the joint dynamics of size and breeding values then has the form

$$n(z', x, t+1) = \mathcal{R} c_0(z') f^*(x) + \int P(z', z; x) n(z, x, t)\,dz. \tag{10}$$

Note that in this equation $f^*(x)$ must be calculated each time step as the distribution of breeding values changes. The recruitment term in equation (10) assumes that the evolving trait x has no impact on offspring size, so that offspring initial size is independent of offspring breeding value. Throughout the text we refer to this model as the two-dimensional (2D) IPM. The term $\mathcal{R}$ and the parameters of the P kernel are constant in the *Oenothera* model, while for *Carlina* both of those vary randomly over time.

An important benefit of the infinitesimal model is that the 2D IPMs require only one parameter beyond those in the demographic IPMs (eq. [2]), the segregation variance V_0. In appendix C, we explain how we derived an estimate of V_0 for our case studies on the basis of observations of size at flowering and the predicted relationship between V_0 and trait variance. The estimate is an upper bound based on the assumption that all individuals have a perfectly sharp flowering threshold, but that is sufficient because we use it only as an initial value in evolutionary simulations to explore general properties of our models. One noteworthy conclusion from that analysis is that the equilibrium breeding value variance is dominated by the generation of new variance by segregation and is well approximated by the variance dynamics for a neutral trait (see figs. C1, C2; figs. B1, B2, C1, C2, E1, F1, F2, G1, I1, J1 are available online). This contrasts with the situation for models with haploid inheritance, where analogous approximations for the mean and variance of an evolving trait were quite inaccurate for both IPMs and individual-based models (Rees and Ellner 2016).

The usual assumption in quantitative genetics is that each individual's phenotype is the sum of a genetic contribution (equal to their breeding value, in our models) and an independent environmental effect. We assume for simplicity that the environment effect is small enough to ignore. However, if the environment component is expressed independently each year, a 2D model can be used in which fitness conditional on breeding value is calculated by integrating over the distribution of environment effects (Lande 1982; Barfield et al. 2011). A permanent environment effect

can also be incorporated through a three-dimensional IPM where individuals are cross-classified by size, breeding value, and environment effect (see app. G).

Modeling Evolution of the Segregation Variance for Flowering Threshold

As noted above, we found that the level of heritable flowering threshold variation maintained in the 2D IPMs for *Oenothera* and *Carlina* is primarily determined by the segregation variance V_0 and is well approximated by the steady-state variation that would be maintained in a neutral trait (see figs. C1*A*, C2*A*). Therefore, to see whether high additive genetic variance can be maintained in those systems by the mechanisms through which parents might benefit from producing heterogeneous offspring (i.e., tracking, disruptive selection, or bet hedging), we need to consider models where selection can alter the segregation variance V_0. In this section, we develop those models.

First, we extend the IBMs so that each individual is characterized by its size z, its threshold for flowering T, and additionally by a mutation rate modifier trait ξ (Gillespie 1981) that determines how much each parent contributes through mutation to the heritable variation in flowering threshold among their offspring. Apart from this effect, the modifier trait is assumed to be neutral. Koren et al. (2014) and Raynes and Sniegowski (2014) give examples of mutation rate modifiers and how they operate mechanistically. Reduced DNA repair rate is one common mechanism for achieving a higher overall mutation rate, with some mutator alleles (in bacteria) producing hundred-fold rate increases (Miller 1998; Raynes and Sniegowski 2014). Ness et al. (2015) observed sevenfold variation in the mutation rate among strains of *Chlamydomonas* and that mutator genotypes arose, increasing the mutation rate approximately eightfold in some replicates. They also found evidence for fine-scale heterogeneity in the mutation rate and clusters of multiple mutations occurring at closely linked sites. This provides a potential mechanism for evolution to adjust relative mutation rates at different loci.

Without mutation to counterbalance drift (and possibly selection), the genic variance σ_a^2 will eventually fall to 0, while large mutation rates will increase σ_a^2. Evolution of ξ thus provides a mechanism for natural selection to determine whether the segregation variance becomes large or small and thus for the genetic variance of the trait to increase or decrease over time. This is an indirect mechanism: ξ directly affects mutational variance, and this eventually changes the genic variance and segregation variance in the population. Similar approaches were used, for example, by Kondrashov (1995) to study selection on mutation rate when mutations are unconditionally deleterious and by Kawecki (2000) to study evolution of genetic canalization under constant and fluctuating selection resulting from its effects on trait distributions.

To develop the model, consider first the dynamics of the genic variance σ_a^2 for a fixed mutation rate. The mutational input to σ_a^2 is

$$\sigma_m^2 = 2\sum_i \mu_i \sigma_i^2(a), \tag{11}$$

where the sum runs over the loci controlling the trait, μ_i is the mutation rate at locus i, and $\sigma_i^2(a)$ is the variance of increments in allele effect due to mutation at locus i (Walsh and Lynch 2018, ch. 26). For the reasons explained above, we assume that the dominant forces affecting the genic variance σ_a^2 are drift and mutation. Then under the infinitesimal model with nonoverlapping generations, the expected variance in the next generation is

$$\sigma_a^2(t+1) = \left(1 - \frac{1}{2N_e}\right)\sigma_a^2(t) + \sigma_m^2, \tag{12}$$

where N_e is the effective population size (Walsh and Lynch 2018, ch. 24), the number of parents contributing to the next generation. The first term on the right-hand side is the reduction in variance due to drift, and the second is the injection of variance by mutation. The mechanism producing the factor $1 - 1/2N_e$ in equation (12) is inbreeding, causing identity by descent of a recruit's two alleles at a locus affecting the trait (Walsh and Lynch 2018, ch. 2). Equation (12) is the simplest possible model for the dynamics of σ_a^2, and factors absent from the infinitesimal model (e.g., inbreeding depression, epistasis) require more complicated models (Walsh and Lynch 2018, ch. 24).

Both terms in equation (12) must be modified when generations overlap, because new mutations and new instances of identity by descent occur only in new recruits. Let $p(t)$ be the fraction of recruits in the total population at time $t + 1$; we then have

$$\sigma_a^2(t+1) = \left(1 - \frac{p(t)}{2N_e}\right)\sigma_a^2(t) + p(t)\sigma_m^2. \tag{13}$$

Second, we add a heritable trait ξ that modifies the mutation rates μ_i in equation (11), such that an individual with trait value ξ has mutation rate $\xi\mu_i$ at locus i. We assume that ξ is also a multilocus quantitative trait whose dynamics follow the Gaussian descendents approximation of the infinitesimal model. Evolution of ξ provides a means for selection to alter the segregation variance on flowering threshold T and therefore to alter the level of heritable variation for T in the population.

The input to the genetic variance from mutations in new recruits will reflect the average value of ξ in their parents, a weighted average in which parents are weighted in proportion to the number of recruits they produce. Because re-

cruits inherit the midparent value of ξ under the infinitesimal model (plus segregation variance that does not affect their mean breeding value), the weighted average value in parents equals the average breeding value for ξ in recruits. Letting $\tilde{\xi}(t)$ denote this value in the new recruits that join the population at time $t + 1$, we then have

$$\sigma_a^2(t+1) = \left(1 - \frac{p(t)}{2N_e}\right)\sigma_a^2(t) + \tilde{\xi}(t)p(t)\sigma_m^2. \qquad (14)$$

Each of the parental ξ values (ξ_p and ξ_o) multiplies the mutation rate at half of each offspring genome, and the mutational input to an offspring's trait variance is proportional to the average mutation rate. The segregation variance for flowering threshold T in their offspring will therefore be

$$V_0 = \frac{\sigma_a^2(t)}{2} + \left(\frac{\xi_p + \xi_o}{2}\right)\sigma_m^2. \qquad (15)$$

We studied evolution of ξ using IBMs incorporating equations (14) and (15) and also through an invasion analysis using the 2D IPMs in which the parameter σ_m^2 is replaced by $\xi\sigma_m^2$. To generate a resident environment (with any value of ξ), we iterate the 2D IPM and record from a long simulation information on the following: the number of recruits $\mathcal{R}(t)$ each year, as this determines how the resident and an invading mutant interact; parameter values (table 2), which for *Carlina* vary from year to year; and the distribution of threshold breeding values T in pollen, as all mutant ovules are fertilized by resident pollen. We then calculate λ (stable population growth rate; Ellner et al. 2016) or λ_S (stochastic population growth rate; Ellner et al. 2016) for a rare mutant with mutation modifier value $\tilde{\xi}$. In order to do this we need to know how $\tilde{\xi}$ changes the genic variance as a consequence of breeding with the resident. This calculation (app. H) shows that on average, the genic variance of the mutant lineage, $\tilde{\sigma}_a^2(t)$, is

$$\tilde{\sigma}_a^2(t) \approx \sigma_a^2(t) + \frac{\bar{p}\sigma_m^2}{1 + \bar{p}/2N_e}(\tilde{\xi} - \xi), \qquad (16)$$

where $\sigma_a^2(t)$ is the genic variance in the resident lineage.

Evolution of Segregation Variance

For *Oenothera* we have a constant environment model, with stabilizing selection about an optimum. The expectation therefore is for selection against producing genetically variable offspring (Slatkin and Lande 1976), and indeed this is the case (fig. 2). In *Carlina* we have a stochastic environment model, and so it is possible that the production of genetically variable offspring could be advantageous as a result of tracking, disruptive selection, or bet hedging; we know from the PIP that the storage effect does not operate. Both the IBM and invasion analysis suggest that this is indeed the case (fig. 3). Note that in both cases the strength

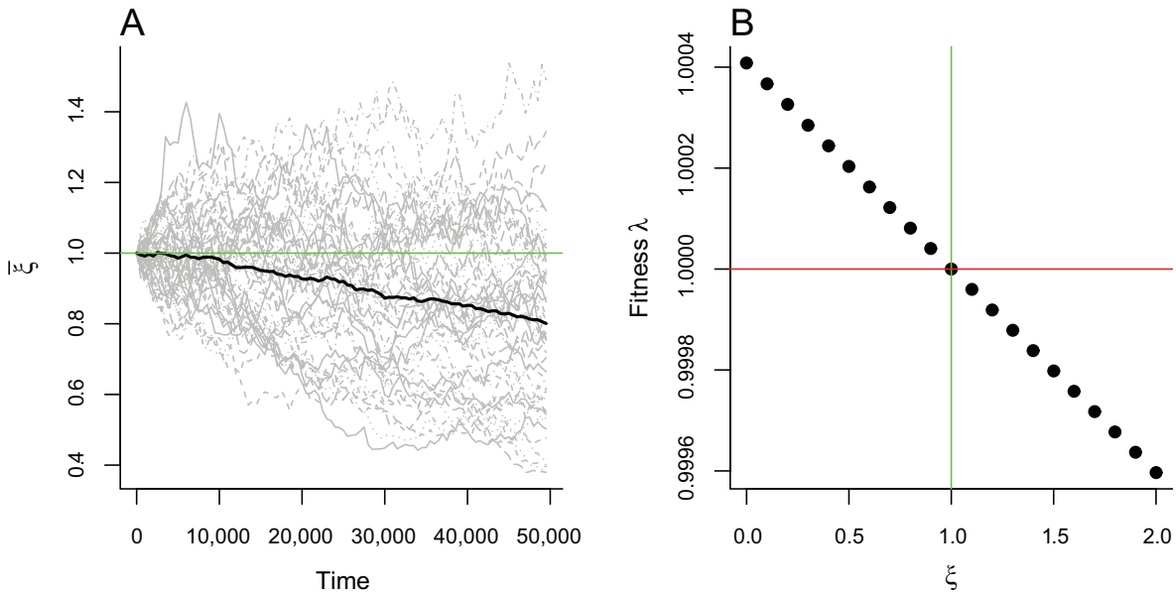

Figure 2: *A*, Evolution of the mutation modifier ξ for *Oenothera*. Green line is the initial value. Gray lines are from individual-based simulations, and black line is the overall mean. The segregation variance for ξ was 0.0025 in the individual-based simulations. *B*, Fitness landscape from the integral projection model for an invading strategy with different ξ; λ is the stable population growth rate. Green line is the resident ξ, and red line is $\lambda = 1$. Source file: Variance Dynamics Thresh Modifier.R.

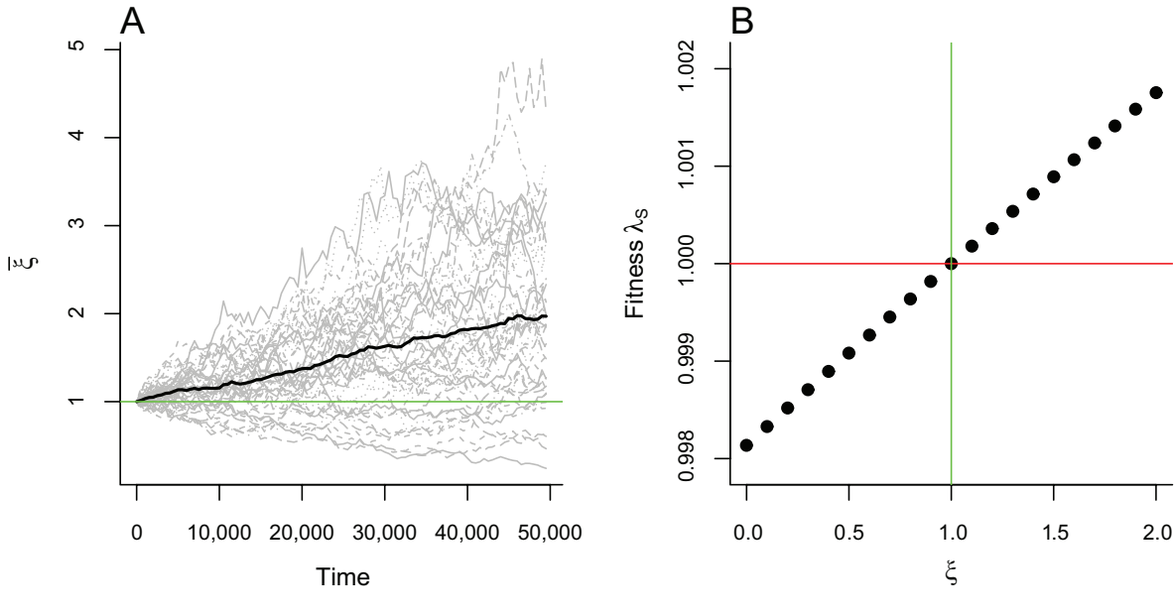

Figure 3: *A*, Evolution of the mutation modifier ξ for *Carlina*. Green line is the initial value. Gray lines are from individual-based simulations, and black line is the overall mean. The segregation variance for ξ was 0.0025 in the individual-based simulations. *B*, Fitness landscape from the integral projection model for an invading strategy with different ξ; λ_S is the stochastic population growth rate. Green line is the resident ξ, and red line is $\lambda_S = 1$. Source file: Carlina Evol Demog Dynamics Thresh Modifier 1.3.R.

of selection on the mutation modifier is very small and so the evolutionary trajectories are highly variable, even in a constant environment (fig. 2*A*).

To explore whether these results are simply artifacts of our specific speculative model in which evolution of a mutation rate modifier is the driving force in the dynamics of segregation variance, we also constructed and simulated models in which segregation variance V_0 or mutation variance σ_m^2 were themselves heritable quantitative traits on which selection could act directly (see app. I). We make no attempt to justify these models biologically; their only purpose is to ask whether our conclusions are robust to radical change in the mechanism whereby selection could affect heritable variation in flowering threshold. The models are both IBMs with *Carlina* demography.

The first is diploid, identical to the IPM with a mutation rate modifier except that the second heritable trait is the segregation variance V_0 itself, modeled as a multilocus trait evolving according to the infinitesimal model. Successful offspring inherit a segregation variance with mean equal to the average V_0 values of their two parents, with small variance lognormal variation. The second model is haploid, with individuals characterized by size, flowering threshold, and mutational variance σ_m^2. Offspring inherit their parent's flowering threshold with Gaussian noise having variance equal to the parent's mutational variance and their parent's mutational variance plus a small error. Simulation results for these model (fig. I1) agree exactly with our previous conclusions. In the first model, where selection can alter the magnitude of segregation variance, there is selection for increased segregation variance, resulting in high heritable trait variation in the population. In the second model, where all offspring closely resemble their parent (as in the adaptive dynamics analysis), there is a collapse of genetic diversity, as predicted from the PIP for the adaptive dynamics analysis of the *Carlina* model (fig. 1).

What Processes Maintain Heritable Trait Variance?

We have seen that fluctuating selection can maintain genetic variation for flowering threshold in the *Carlina* system when flowering threshold is modeled as a multilocus quantitative trait, opposite to what occurs in an adaptive dynamics analysis of flowering threshold evolution under the same selective regime. Here we ask which of the processes listed in the introduction is responsible for maintaining genetic variation. The three possibilities are tracking, disruptive selection, and bet hedging, as the adaptive dynamics analysis rules out storage effect as a possible mechanism, and our models do not include any heterozygote advantage.

Tracking seems unlikely, as successive environments are not correlated because parameters are drawn independently each year. As a consequence, successive changes in Δx are

negatively autocorrelated (partial autocorrelations ≈ -0.05 for lags 1–5), exactly the opposite of what we would expect if tracking were important.

Understanding the relative roles of disruptive selection and bet hedging is complicated because the two processes can overlap. In some situations, they are alternative descriptions of the same phenomenon because production of highly variable offspring is a mechanism for bet hedging (i.e., a means to increase geometric mean fitness at the expense of arithmetic mean fitness [averaged over offspring and year-to-year environmental variability]; for one example, see app. J). We therefore focus on disruptive selection on the variance, as it can be quantified using well-established methods.

In order to characterize selection operating on the variance, we calculated the quadratic selection differential, using results from Lande and Arnold (1983). Specifically, Lande and Arnold (1983, eq. [13]) showed that the change in trait variance as a result of selection can be written as

$$V^* - V = \mathrm{cov}(w, (x - \bar{x})^2) - S^2, \tag{17}$$

where * indicates a quantity after selection, w is relative fitness, and $S = \bar{x}^* - \bar{x}$. This holds for arbitrary selection on an arbitrary trait distribution, so it applies to our models despite their complications (different selection on survivors and new recruits, and fitness resulting from demographic rates rather than a fitness function $w(x)$). The term $\mathrm{cov}(w, (x - \bar{x})^2)$ is the quadratic selection differential, denoted C. Variables V, V^*, and S are all quantities that our 2D IPMs can calculate at each time step (as we explain below), so we can calculate C as

$$C = V^* - V + S^2. \tag{18}$$

In a stochastic environment the expected change in the variance as a result of selection is (by rearranging eq. [18], taking expectations, and using the definition of S)

$$\mathbb{E}_e(V^* - V) = \mathbb{E}_e(C) - \mathbb{E}_e(\Delta\bar{x}^2), \tag{19}$$

where the expectation is with respect to the joint distribution of demographic parameters and the size/genotype distribution. The second term on the right-hand side of equation (19) is the change in variance resulting from selection on the mean. Therefore, the selection on the variance in a stochastic environment is characterized by the arithmetic mean quadratic selection differential $\mathbb{E}_e(C)$.

In these equations and the calculations that follow, it is important to remember that selection occurs within a year, and so the selection response does not include the reduction in variance from averaging of parental breeding values or the increase in variance due to allele segregation (eq. [C1]). To calculate $\mathbb{E}_e(C)$, we first calculate the means and variances of the trait (flowering threshold) distributions in survivors and gametes from the 2D IPM (eq. [10]; table 2), using the methods explained by Ellner et al. (2016, sec. 2.5.5). The mean trait after selection is then the weighted average of the means in gametes and surviving individuals, with weights equal to proportions of new recruits and survivors in the population. The variance after selection is calculated by combining the variances in survivors and gametes in simulations, using equation (D1). We then calculate C using equation (18). The corresponding quadratic selection gradient, γ, is $\gamma = C/V^2$. For a constant environment model, C or γ are equivalent for quantifying equilibrium selection on the variance because V converges to a constant value once the trait mean has equilibrated. However, in a stochastic environment, this is no longer true because the variance continues to fluctuate from year to year. We therefore present $\mathbb{E}_e(C)$ for *Carlina* and also give the distribution of γ_t to allow comparison with literature compilations of γ (Kingsolver et al. 2001; Stinch combe et al. 2008).

For *Oenothera*, $\gamma \approx -1$ at equilibrium, indicating stabilizing selection on the variance. For *Carlina*, the mean $\pm$ SE of the distribution of year-specific C and γ values are 0.02 ± 0.0005 and 0.35 ± 0.01, respectively, and ≈53% of the values are positive, which is consistent with disruptive within-year selection. The disruptive selection in *Carlina* occurs as a result of fluctuation in the environment, which results in a mismatch between the current breeding value distribution and the optimal flowering threshold in that year. If C_r is the quadratic selection coefficient operating on gametes and C_S is another operating on survivors, then the overall quadratic selection differential is

$$C = pC_r + (1 - p)C_s \tag{20}$$

(see app. K). The mean values are 0.003 ± 0.0006, 0.03 ± 0.0009, and 0.02 ± 0.0005 for survival, gamete production, and total C, respectively. The distributions of γ_r, γ_S, and the overall γ are shown in figure 4. The distributions of all three γ's are very similar, with negative modes and a long tail of positive values. This suggests that stabilizing selection frequently occurs (70% are negative for survival, 56% negative for gamete production, and 47% for total γ).

Some further insight in the selection operating on the variance can be obtained using the approximate equations for the variance dynamics (eqq. [F4], [F6]). Matching terms between the approximate variance equations and equation (20), we find

$$C \approx V^2 \left[p \frac{W_r''(x|\bar{x})}{2\bar{W}_r} + (1 - p) \frac{W_s''(x|\bar{x})}{\bar{W}_s} \right], \tag{21}$$

and so for small variance ($V \approx 0$), $C \approx 0$ as it is of order V^2. The change in mean due to selection is also $O(V)$, so S^2 is also $O(V^2)$. The actual year-to-year variance changes (in the 2D IPM or corresponding IBM) result from selection, mating

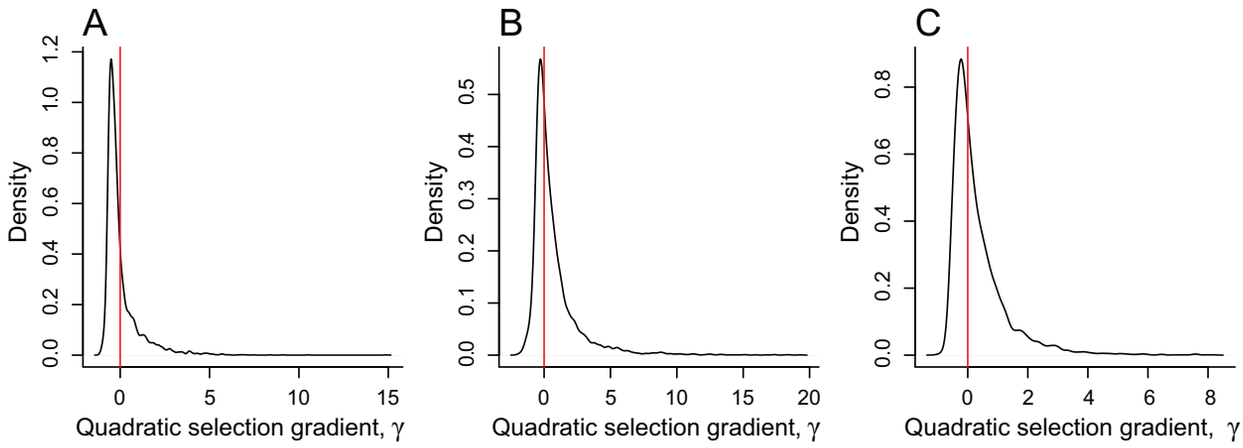

Figure 4: Distribution of within-year γ values for *Carlina* for survivors (*A*), gametes (*B*), and in total (*C*), combining the effects of selection through both routes. In each case, γ is calculated after survival and gamete production but before mating occurs and the segregation variance is added to the distribution of offspring breeding values. Source file: Carlina Evol Demog Dynamics Thresh.R.

(averaging of parent breeding values), and segregation variance. The latter two processes both have effects of magnitude $O(V) = O(V_0)$ and therefore dominate the effect of selection when V is small, which explains the accuracy of the neutral trait approximation for the steady state value of V (fig. C1).

The positive mean of C for flowering threshold provides a mechanism for the initial upward evolution of the mutation rate modifier ξ (fig. 3). A mutant with higher ξ has higher segregation variance for flowering threshold (app. H), so mutant individuals will be overrepresented at the tails of the flowering threshold distribution, more or less symetrically when segregation variance is the dominant term in the variance dynamics. If ξ mutants have the same mean flowering threshold as residents but higher variance, then whenever C is positive the mutants will have higher mean fitness, and the population mean ξ would evolve upward. Upward selection on ξ would cease when the variance in flowering threshold becomes high enough that individuals in the tails are less fit on average than those near the mean.

Discussion

Our key biological finding is that fluctuating selection can select for genetic variation in the threshold size for flowering when it is modeled as a multilocus quantitative trait. In contrast to Wittmann et al. (2017), our conclusion is not a consequence of mechanisms giving an intrinisic benefit to heterozygosity per se. Previous work using adaptive dynamics and IPMs to model the evolution of the relationship between plant size and flowering probability in *Carlina* showed that without constraints the ESS is a step function (Childs et al. 2003, 2004; Rees and Ellner 2009, 2016), a single threshold without any genetic variation. Interestingly, there is a similar discrepancy between theory and data with regard to seed dormancy. Simple adaptive dynamics models predict an ESS germination fraction (Ellner 1985), whereas many populations harbor a wide range of germination fractions that have a genetic basis (e.g., Wehner 1984; Koornneef et al. 2002; Saeidi 2008; Alonso-Blanco et al. 2009). As with flowering threshold, the trait mean is predicted well (Gremer and Venable 2014) but not the trait variance.

The mismatch between the adaptive dynamics and quantitative genetics models' predictions is a consequence of how disruptive selection arises in our quantitative genetics models: individuals in the tails of a highly variable offspring distribution have higher fitness on average than those in the middle. Such effects cannot occur in the adaptive dynamics analysis because the conditions for maintaining an evolutionarily stable polymorphism are derived under a scenario where the resident and invader populations both consist of a single genotype and are similar in trait values. The breeding system also turns out to be important. In a haploid system, where offspring inherit their parent's genotype plus some small mutational error, the fact that genetic variance would grow without limit in the absence of selection means that any positive amount of mutation leads to a situation where trait variance is stabilized by the mutation-selection balance. Hence there is a genetic load due to the constant removal of low-fitness individuals from the tails of the trait distribution, which selects for reduced mutation rates (fig. I1). In contrast, in a diploid outcrossing species (as is assumed in our quantitative genetics models) there is a very rapid approach to an equilibrium variance largely determined by the loss of variation through averaging of parental breeding values and gen-

eration of variance through allele segregation in offspring. This means that an optimal level of trait variation (if there is one) could be achieved by evolution of the mutation rate, without producing overly extreme individuals that are culled by selection.

The quadratic selection gradient depends on the entire distribution of breeding values within a population. The quadratic selection differential is $C = \mathrm{cov}(w, (x - \bar{x})^2)$, and so if we Taylor expand relative fitness, w, we find (using the formula $\mathrm{cov}(X, Y) = \mathbb{E}(XY) - \mathbb{E}(X)\mathbb{E}(Y)$)

$$C \approx \mathrm{cov}\left(w_0 + w_1(x - \bar{x}) + \frac{w_2}{2}(x - \bar{x})^2 + \frac{w_3}{6}(x - \bar{x})^3 + \frac{w_4}{24}(x - \bar{x})^4 + \ldots, (x - \bar{x})^2\right) \tag{22}$$

$$= w_1 m_3 + \frac{w_2}{2}(m_4 - m_2^2) + \frac{w_3}{6}(m_5 - m_2 m_3) + \frac{w_4}{24}(m_6 - m_2 m_4) + \ldots, \tag{23}$$

where $w_k = \partial^k w / \partial x^k$ evaluated at $\bar{x}$ and m_k is the kth central moment of x. For a quadratic fitness function and Gaussian distribution of x, we then have $C = w_2 m_2^2$, and so we would expect quantitative genetic and adaptive dynamics models to be in agreement because the condition for evolutionary stability is $w_2 < 0$, which implies stabilizing selection on the variance. Turelli and Barton (1994) demonstrate that for the infinitesimal model even with fairly extreme truncation selection the deviations from a Gaussian distribution of breeding values will be small. However, all the models considered by Turelli and Barton (1994) assume nonoverlapping generations. With overlapping generations, the distribution of breeding values at the next time step is a mixture of the distribution in survivors and the distribution in new recruits. In general, this will not be Gaussian if selection is different in survivors and recruits, and it can be bimodal and skewed. For example, selection may be in opposite directions in survivors and recruits; in the present case, there is selection for small flowering thresholds in recruits and large ones in survivors (Rees and Ellner 2016). An IPM is useful in such systems because it projects the complete distribution of breeding values without any prior assumptions about the shape of the distribution. It is also worth emphasizing that with overlapping generations, where individuals experience multiple bouts of selection, the combined effects of multiple bouts of selection can be counterintuitive (McGlothlin 2010). For example, if there is only directional selection and it is always in the same direction, then there will nonetheless be disruptive selection affecting the variance.

The accuracy of the neutral approximations for the variance in both species—despite substantial selection on the variance—is surprising. This suggests that neutral variance models may often be a good approximation for quantitative traits in diploid outcrossing species. The neutral model works so well because large changes in the variance occur as a result of random breeding (halving the variance) and the generation of new variance from segregation. For *Carlina*, the within-year quadratic selection gradients, γ, are positive ≈50% of the time with a mode of ≈0. Interestingly, this pattern in γ is similar to that recorded in compilations of γ from the literature (Kingsolver et al. 2001; Stinchcombe et al. 2008), suggesting that the pattern of selection observed in *Carlina* is not unusual.

Whether the 2D IPM or various approximations provide a reasonable description of the eco-evolutionary dynamics depends on how robust and appropriate the infinitesimal model is as an approximation of the underlying genetic system. Barton et al. (2017) suggest that the infinitesimal model will hold under general conditions; for the additive case these include arbitrary selection and population structure, provided that the segregation variance is not too small or the traits too extreme (i.e., close to the maximum or minimum possible values of the trait). Their mathematical analysis suggests that the model's error is at most of order $1/(M)^{1/2}$, where M is the number of loci. The responses observed in long-term selection experiments are also often in good agreement with the infinitesimal model (Weber and Diggins 1990; Johnson and Barton 2005), suggesting that the model is a good starting point. However, the actual underlying mechanisms may be different, in particular for the genic variance remaining constant under directional selection. It occurs in the infinitesimal model because, with infinitely many loci determining the trait, changes in trait variance are entirely due to changes in linkage disequilibrium, while allele frequencies at each locus are unchanged (Bulmer 1971). Hill (1982) argued that new mutations are an important factor, and Barton and de Vladar (2009) suggested that genic variance remains roughly constant under directional selection because increased variance due to rare alleles becoming more common is balanced by the decrease due to common alleles approaching fixation.

The infinitesimal model is also challenged by the many empirical examples of heritable trait change within a few generations that involve substantial allele frequency change at a few loci (Hanski 2012; Thompson 2013). This has been observed in artificial selection in the laboratory and also in natural populations (Messer et al. 2016); for example, Bergland et al. (2014) observed consistent seasonal frequency oscillations in hundreds of SNPs in a natural orchard population of *Drosophila*. However, it is not clear how representative these examples are or to what extent the infinitesimal model's key predictions are compromised as a result.

When genetic variability is maintained by the storage effect, the theoretically predicted form of polymorphism is either a small number of diallelic or triallelic loci with alleles of large effect or an abrupt shift from zero to many polymorphic loci, with a small number of alleles at each locus when the environmental variance becomes sufficient to maintain genetic variability, depending on the distribution of environmental fluctuations (Ellner and Sasaki 1996). The latter situation would be consistent with the infinitesimal model, but the former would be inconsistent with any multivariate Gaussian or near-Gaussian model, including the infinitesimal model. Ellner and Sasaki (1996) emphasized that testing their models is "complicated by factors which are difficult to measure, such as the distribution of environmental fluctuations and the shape of the selection function." For IPMs there is now a well-developed set of statistical tools for estimating the distribution of environmental effects on demography (Metcalf et al. 2015), and once this has been done, the shape of the selection function is an emergent feature of the fitted model.

We have emphasized that size per se should not be treated as the evolving heritable trait in a size-structured IPM. Apart from this restriction, any heritable trait can be modeled using the approach we present here (e.g., seed size, seed dormancy, size-dependent sex allocation), so long as it is possible to model demographic rates as joint functions of an individual's size and their breeding value for the evolving trait, and we have described how extensions of our approach can be used when demographic rates depend on the actual trait value. We consider here a single heritable trait, interacting with size as a dynamic but nonheritable trait, but in principle the approach can be used with multiple traits using quantitative genetic theory for multivariate trait evolution (as in Barfield et al. 2011).

The methods we used to estimate segregation variance in both field populations are rather crude and are based on an upper bound on the additive genetic variance present. However, in an animal population with a known pedigree, more accurate estimates of the additive genetic variance are possible (e.g., Childs et al. 2016), and then IPMs can be used to refine the resulting neutral estimate of the segregation variance by accounting for the effects of selection on the relationship between the segregation variance and the additive genetic variance. Breeding experiments can also be used to estimate the additive genetic variance (Wesselingh and de Jong 1995; Wesselingh and Klinkhamer 1996; Zas and Sampedro 2015). In a selection experiment on *Cynoglossum officinale*, another monocarpic perennial, Wesselingh and de Jong (1995) were able to create populations with nonoverlapping flowering threshold distributions after one generation of selection, demonstrating that flowering thresholds have a genetic basis and that natural populations harbor extensive genetic variation. The results presented here suggest that temporal variation in the environment may well be important in maintaining the high levels of genetic variation observed in these systems.

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Typical *Carlina vulgaris* that, having waited to flower for several years, is about to flower and then die. Photo credit: Mark Rees.