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Asymmetric Evolvability Leads to Specialization without Trade-Offs

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ABSTRACT: Many organisms are specialized, and these narrow niches are often explained with trade-offs—the inability for one organism to express maximal performance in two or more environments. However, evidence is lacking that trade-offs are sufficient to explain specialists. Several lines of theoretical inquiry suggest that populations can specialize without explicit trade-offs, as a result of relaxed selection in generalists for their performance in rare environments. Here, I synthesize and extend these approaches, showing that emergent asymmetries in evolvability can push a population toward specialization in the absence of trade-offs and in the presence of substantial ecological costs of specialism. Simulations are used to demonstrate how adaptation to a more common environment interferes with adaptation to a less common but otherwise equal alternative environment and that this interference is greatly exacerbated at low recombination rates. This adaptive process of specialization can effectively trap populations in a suboptimal niche. These modeling results predict that transient differences in evolvability across traits during a single episode of adaptation could have long-term consequences for a population's niche.

Keywords: evolvability, generalists, specialists, habitat choice, niche evolution.

Introduction

Species have limits that emerge from complex evolutionary processes. The breadth of a niche is circumscribed by competition with other species but also by constraints internal to the population: for example, limits to local adaptation with gene flow (Kirkpatrick and Barton 1997). Variation in niche breadth is a key dimension of biodiversity and is typically quite high (Poisot et al. 2015), but it is declining as specialists are disproportionally at risk as a result of environmental change (Clavel et al. 2011). Rapid evolution of the niche is most obvious in microorganisms, in forms like host shifts in pathogens (Hall et al. 2011; Longdon et al. 2014), evolution of cell-type tropism inside an infected host (Regoes and Bonhoeffer 2005; Vignuzzi et al. 2006), and broadening of the host ranges of plasmids that spread antibiotic resistance among bacteria (Loftie-Eaton et al. 2016). In multicellular eukaryotes, the propensity for niche evolution has been linked to rates of diversification (Kozak and Wiens 2010) and, inversely, to declines from anthropogenic stresses and risks of extinction (Lavergne et al. 2013). Theory is just beginning to explore how evolutionary rescue in threatened populations might lead to niche reduction (e.g., Schiffers et al. 2013), but understanding niche evolution, particularly as it parallels adaptation to a deteriorating environment, may be essential to predicting responses to climate change.

One highly influential explanation for restricted niche breadths is trade-offs, such that a specialist can exceed the maximal performance of a generalist in the same environment. Despite the intuitive appeal of trade-offs, empirical evidence that they actually explain niche breadths has been hard to find (Futuyma and Moreno 1988; Fry 1996; Remold et al. 2012). Trade-offs should result in strong, consistent costs of adaptation, whereby populations that adapt to specialize in one environment lose fitness in other environments. Experimental evolution methods have a long history of application to this question (Kassen 2002; Bono et al. 2020) and continue to help uncover complex variation in these costs. For example, costs have been observed to be greater in larger populations (Chavhan et al. 2020), to be highly variable across replicate experiments (Jerison et al. 2020), and to appear late in adaptation (Satterwhite and Cooper 2015). In the field, reciprocal transplant experiments with plant and animal species reveal highly variable costs of adaptation with only a weak signal of trade-offs (Hereford 2009). The vast number of mostly specialized phytophagous insect species has inspired a vigorous but largely unsuccessful search for trade-offs limiting niche breadth (Hardy et al. 2020). These shortfalls of the

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trade-off hypothesis have motivated the search for alternative theories that incorporate behavior, population genetics, and macroevolution into a synthetic theory of niche limits (Poisot et al. 2011; Sexton et al. 2017).

A major insight from population-genetic models of niche breadth is that the evolution of preference for a particular habitat—and performance in that habitat—can be linked in a positive feedback loop (Holt 1985; Crespi 2004). Such models elaborate on optimal foraging perspectives by allowing the value of resources to change with an organism's degree of local adaptation, feeding back to determine optimal preferences (Futuyma and Moreno 1988). The cause of this feedback is that the intensity of selection in a specific habitat is proportional to the number and reproductive success of organisms within it (Holt and Gaines 1992). This concept is identical to the more familiar example of weakening selection with increasing age as a result of declining reproductive value (Holt 1996*b*); essentially, rarely encountered or unproductive environments are similar to rarely attained age classes. If organisms evolve to prefer a habitat where they are fitter, selection will shift to favor further improvement in that habitat. This prediction that stronger selection improves the degree of adaptation is quite general, emerging from models with trade-offs but also from those without them (e.g., Kawecki et al. 1997). Greater adaptation in one environment can then drive still greater levels of preference, completing the feedback loop. In such models, specialization can therefore arise from even minor asymmetries in performance across a broad niche (Fry 1996).

Any model of niche breadth considering both performance traits and habitat selection incorporates this positive feedback loop. Such models are plentiful and highly diverse (see reviews in Futuyma and Moreno 1988; Wilson and Yoshimura 1994; Ravigné et al. 2009), but most inherit the idea from Levins (1962) of representing trade-offs with, in two dimensions, a curve of the maximum fitness in one environment given each possible fitness in the other environment (essentially, a Pareto front; see Shovel et al. 2012). Evolution of the degree of specialization is modeled as movement in a single dimension, constrained to this optimal curve. Modeling two dimensions of performance in a single variable is an appealing simplification but cannot adequately represent a scenario in which a population is suboptimally adapted to both environments. This scenario might arise when an invasive species adapts to the multiple novelties of a new environment (Prentis et al. 2008) or as a result of antagonistic coevolution with multiple hosts or prey species (e.g., Hall et al. 2011). In these circumstances, the rate of adaptive evolution, or evolvability, in each environment now dynamically shapes the fitness in each environment and could therefore drive evolution of preferences for those environments.

Several studies have proposed the idea that generalists may suffer deficits in evolvability, even when trade-offs are avoided through separate genetic bases for performance in each environment (Kawecki 1994; Fry 1996; Holt 1996*a*; Whitlock 1996). As discussed above, the strength of selection for performance in any one of a generalist's repertoire of environments is weaker in comparison to a specialist population that reproduces only in that single environment. Because of this relaxed selection alone, specialists may replace generalists because they can adapt faster (Whitlock 1996; Kawecki 1998), and generalist niches may shrink when they cannot maintain fitness for rare environments in the face of deleterious mutation (Kawecki et al. 1997). While these studies present intriguing alternatives to trade-offs, they predict specialization only under quite restricted conditions. Deleterious mutations drive specialization only when the alternative habitat is very rare or unproductive (Kawecki et al. 1997), and specialization via differential adaptation has been demonstrated only in the presence of strong, continuous antagonistic coevolution (Kawecki 1998). Neither framework explicitly considers linkage between the genetic bases of fitness in each environment or potential ecological costs of specialism, such as the search costs that are typically associated with a narrow niche in classical models of optimal foraging (Charnov 1976; Rosenzweig 1981). Therefore, we still lack a clear demonstration that evolvability deficits in generalists can cause specialization in the face of search costs, as well as an understanding of the roles of linkage and the form of selection in this process.

Here, I show that specialization can evolve during a single period of adaptation to two environments and that this adaptive specialization can occur without trade-offs and in opposition to substantial search costs. Populations can evolve niche reduction adaptively; although the fitness landscape allows for no-cost generalists that are competitively superior to any specialist, specialization, once evolved, is persistent. Therefore, a continuously fluctuating environment is not required for this mechanism of specialization.

A key discovery of this work is that a population's chance of specializing is strongly driven by recombination between the genetic bases of fitness in each environment. Recombination helps maintain generalism because it alleviates clonal interference—competition between concurrently segregating beneficial mutations (Gerrish and Lenski 1998)—that further impedes adaptation in traits with already low evolvability. Recent theory focusing on asexual evolution has predicted that adaptation among sites with small selection coefficients can be effectively stalled by interference from rapidly evolving sites with larger effects (Schiffels et al. 2011). Extending this model to multiple traits, Gomez et al. (2019) showed that clonal interference can produce a false signature of trade-offs

where none exist, and Gomez et al. (2020) showed that a trait with more frequent or larger beneficial mutations can effectively stall adaptation in a trait with lower evolvability, an effect that Venkataram et al. (2019) recently demonstrated experimentally. Here, specialization is seen to evolve when a rare environment becomes unprofitable in comparison to the more common environment, solely because of slow relative improvement in how organisms can exploit the rare environment. Asymmetric evolvability can drive specialization regardless of recombination rate, but it has a particularly potent effect when the genetic bases of fitness are strongly linked.

Model

Overview of Ecology and the Life Cycle

The basic Wright-Fisher model was modified to allow for a potentially costly preference for the common environment while retaining several traditional features: nonoverlapping generations and construction of a fixed number of adults via random sampling from an unlimited pool of gametes. Each organism has three traits—a preference trait (f) and two performance traits, w_A and w_B—that are entirely determined genetically by three sets of distinct loci (for model parameters, see table 1). The number of adults is limited to a global carrying capacity K; each of these individuals then attempts to settle in a habitat of either environment A or B, as described below. The expected fecundity (W) of an individual i in environment x is

$$\mathrm{E}(W_{i,x}) = \frac{w_{i,x}}{\sum_{i\in\alpha} w_{i,\mathrm{A}} + \sum_{i\in\beta} w_{i,\mathrm{B}}} K. \tag{1}$$

Here, α is the set of individuals that have settled in habitats of environment A, and β is the set in environment B. Because of search costs, as detailed below, $|\alpha| + |\beta| \le K$; that is, the total number of individuals settled in each environment is no greater than K but can be less than that. Following the taxonomy summarized in Ravigné et al. (2009), this is a model with global regulation of population size and variable habitat outputs, and it is therefore a model of "hard selection."

In fully asexual simulations, the fecundities given by equation (1) are used as weights in a multinomial distribution from which K parents are selected, with replacement, to reproduce and form the next generation. In simulations with recombination, $2K$ parents are drawn with replacement, and offspring are determined via the linkage relationships described below. All organisms modeled here are haploid.

Table 1: Important model parameters

Symbol	Description
w_A, w_B	Fitnesses in environments A and B
G_A, G_B	Sets of loci determining fitness in environments A and B
s_A, s_B	Fitness effects of the favored alleles in environments A and B
μ_{pref}	Rate of mutations in the preference trait per individual per generation
μ_A, μ_A	Rates of mutations in the sets of loci determining w_A and w_B, per individual per generation
g_A, g_B	Numbers of beneficial alleles in G_A and G_B for a given genotype
f	Probability of rejecting environment B for a given genotype
K	Carrying capacity
N	Number of adults in habitats of environment A or B
c	Search cost, equal to the chance of dying while seeking another habitat
p	Frequency of encountering environment B
b	Number of possible alleles at a locus
r	Rate of recombination between neighboring blocks of loci (i.e., between the preference locus and G_A and between G_A and G_B)

Environment Preference and Specialism

The model considers two environments with unequal frequencies; without loss of generality, we label the common environment A and the rare one B. The preference trait causes aversion to the rare environment (and therefore a preference for the common environment A). The preference trait f is therefore treated as a probability to reject environment B when encountered. While this model could be extended to allow preferences for either environment to evolve, this complication is not necessary to model the process of specialization on the common environment. Therefore, the model does not allow an environment B specialist to evolve.

Each adult searches for a habitat, encountering environment A with probability $1 - p$ and environment B with probability p, independent of genotype. An individual will reject environment B with probability f and experience a search cost, c, representing the probability of death while searching for a new habitat. If the individual avoids death, it once again encounters environment A or B with the same probabilities and may again reject B with probability f, continuing until it has perished or been assigned to an environment. The probabilities of assignment to A, B, or death are given by the sums of geometric series as follows:

$$P(\mathrm{A}) = \frac{1-p}{1-pf(1-c)}, \tag{2a}$$

$$P(\mathrm{B}) = \frac{p(1-f)}{1-pf(1-c)}, \tag{2b}$$

$$P(\mathrm{death}) = \frac{pfc}{1-pf(1-c)}. \tag{2c}$$

This model of search costs is similar to that in Forbes et al. (2017) but is generalized to allow for a probabilistic preference.

Mean fitness of a given genotype can then be written as a sum of equations (2a) and (2b), weighted by that genotype's fitness in those environments:

$$\mathrm{E}(w) = \frac{1-p}{1-pf(1-c)}w_{\mathrm{A}} + \frac{p(1-f)}{1-pf(1-c)}w_{\mathrm{B}}. \tag{3}$$

The derivative of equation (3) with respect to f is positive when the following inequality is met:

$$(1-c)(1-p)w_{\mathrm{A}} > (1-p(1-c))w_{\mathrm{B}}. \tag{4}$$

Using equation (3), we can also calculate when $\mathrm{E}(w|f = 1)$ is greater than $\mathrm{E}(w|f = 0)$ and obtain the same condition. Therefore, whenever equation (4) is true, selection favors specialization, up to complete specialization ($f = 1$).

Two simplifications of equation (4) help establish some intuition. When c is zero, then specialism on A can start to evolve whenever $w_{\mathrm{A}} > w_{\mathrm{B}}$. When instead p is small, we can approximate the condition in equation (4) as $(1-c)w_{\mathrm{A}} > w_{\mathrm{B}}$; that is, specialism starts to be favorable when it is more profitable to risk death than to settle for environment B. This latter result highlights that search costs make specialization unprofitable without a compensatory difference in the realized value of the environments.

Genetic Bases of Traits and Mutation

The fitness in environment A, w_{A}, is determined by a set of loci G_{A}. This set consists of L perfectly linked loci with b possible alleles each. Fitness in environment B is determined in exactly the same way, by an independent set of L loci G_{B}; G_{A} has no effect on fitness in environment B and vice versa. Within a set each locus is interchangeable with the others, so we can fully represent the genotypic basis of fitness for, say, environment A by the number of beneficial alleles, g_{A}, it contains. Fitness effects are multiplicative across loci and are normalized such that the maximum fitness is 1; therefore, the fitness of an individual in environment x is

$$w_x = \frac{(1+s_x)^{g_x}}{(1+s_x)^{L}}. \tag{5}$$

Throughout this article, $s_{\mathrm{A}} = s_{\mathrm{B}}$. Of b alleles, only one of the alleles at a given locus is beneficial, while $s = 0$ for the remainder. Mutation is modeled as changing the allele to one of the other $b-1$ possibilities. The probability that a mutation in G_{A} is beneficial (increasing g_{A}) is

$$p_{\mathrm{bene}}(g_{\mathrm{A}}) = \frac{L-g_{\mathrm{A}}}{L(b-1)},$$

the probability that it is deleterious (decreasing g_{A}) is

$$p_{\mathrm{del}}(g_{\mathrm{A}}) = \frac{g_{\mathrm{A}}}{L},$$

and remaining mutations are neutral because they swap one unfavored allele for another. Because there are a finite number of sites that can be improved, the rate at which new beneficial mutations arise decreases linearly with the degree of adaptation.

A preference locus, represented by a single number in $[0, 1]$, is also encoded in each genotype. In simulations with linkage, these sets are on the same chromosome in the order of preference locus, G_{A}, and G_{B}. This is clearly not realistic for polygenic traits; the model is best seen as representing complete linkage with specific, purposeful deviations from that pattern. Linkage between G_{A} and G_{B} is a focus of the model, while linkage between the preference locus and $G_{\mathrm{A}}/G_{\mathrm{B}}$ is a nuisance factor—therefore, in some simulations the preference locus is modeled as if it were on an independent chromosome to assess the impact of this choice. Mutation of the preference locus is implemented by simply redrawing the value from a uniform distribution. This broad distribution was chosen to increase the variation in preference within a population and highlight the action of selection rather than possible mutational constraints.

The number of mutations in each trait in a generation is drawn from Poisson distributions with means of $\mu_{\mathrm{pref}}N$ for the preference trait, $\mu_{\mathrm{A}}N$ for G_{A}, and $\mu_{\mathrm{B}}N$ for G_{B}. Individuals are selected to receive mutations with replacement.

Density Dependence

While the basic model does not include density dependence within environments, in one section of "Results" an extension of the model is used to explore the effects of crowding. In the absence of any habitat preferences, we expect $(1-p)K$ adults to reproduce in environment A and pK in environment B. We refer to these values as the neutral expectations for densities in each environment. I implemented density-dependent growth as a discrete option: either density in each habitat was ignored, as described above, or a density higher than the neutral expectation for that environment caused reduced reproduction for everyone in that environment. The reduction in growth was calculated to be proportional to the percentage

by which density exceeded the neutral expectation. If N_A is the number of breeding adults in environment A and adults encounter environment A with probability $1 - p$, then reproduction in environment A was divided by a factor $N_A/((1 - p)K)$ whenever $N_A > (1 - p)K$. If N_A did not exceed $(1 - p)K$, then fitness was calculated normally—there was no positive effect of low density. The corresponding calculation was also performed for environment B; by definition, only one environment could suffer negative density dependence in a given generation.

Simulation Approach and Statistics

Simulations were written in R with integrated C++ algorithms for the sake of speed. In general, between 250 and 1,000 replicates were performed for each treatment; confidence intervals are included in all figures in which they are not negligible. Confidence intervals for proportions were generated by bootstrapping: the 2.5% and 97.5% quantiles of the mean proportions of 100,000 resamplings of each data set were used to construct a 95% confidence interval. All code is available in the Dryad Digital Repository (https://doi.org/10.5061/dryad.pc866t1nc; Draghi 2021).

Results

Linkage Promotes Adaptive Aversion to the Rare Environment

To understand how performance in each environment was shaped by adaptation to the other, I first performed simulations with generalists that could not evolve into specialists (i.e., $\mu_{pref} = 0$) in large populations ($K = 10{,}000$) in which environment B was encountered by 25% of individuals ($p = 0.25$). Performance in each environment was determined by a distinct set of 100 loci ($L = 100$) for each environment. At each of these 200 loci, one of 21 ($b = 21$) alleles conferred a small multiplicative benefit ($s_A = s_B = 0.01$) in the corresponding environment. The initial genotypes had 31 adaptive alleles in each environment, yielding starting fitnesses of approximately 0.503 in A and B. For the set of loci corresponding to environment A, all alleles are neutral in environment B and vice versa; there were no trade-offs. Populations were then allowed to improve performance in one or both environments, by setting the mutation rates to ($\mu_A = 5 \times 10^{-4}$, $\mu_B = 0$), ($\mu_A = 0$, $\mu_B = 5 \times 10^{-4}$), or ($\mu_A = 5 \times 10^{-4}, \mu_B = 5 \times 10^{-4}$).

With these parameter values, the expected number of beneficial mutations arising in the population per generation for fitness in environment A is the product $\mu_A K(L - 31)/((b - 1)L)$, which is 0.1725. The effective selection coefficient in environment A is approximately 0.0075 (ps_A; Whitlock 1996). The fraction expected to fix, ignoring clonal interference, is about twice this effective selection coefficient; we can therefore calculate that a beneficial mutation escapes loss by drift at a rate of $2 \times 0.0075 \times 0.1725$, or about every 400 generations. Once again ignoring clonal interference, we can estimate the fixation time for such mutations at approximately 2,500 generations (Otto and Whitlock 2013, eq. [6]). Therefore, we expect beneficial substitutions to overlap in time and interfere with each other.

Figure 1 plots mean fitness in each environment, comparing treatments when each trait evolves alone (no mutation in the other trait) versus when it evolves simultaneously with the other fitness trait. With complete linkage ($r = 0$) and therefore clonal interference between traits (see Schiffels et al. 2011; Gomez et al. 2020), adaptation to the less common environment is greatly slowed when the population is also adapting to environment A; this slowdown is largely but not entirely eliminated with free recombination between the genetic bases of each trait, G_A and G_B. Recombination does not eliminate all interference between traits because the strength of selection in an environment is still dependent on that environment's contribution to the total pool of offspring; w_A increases faster than w_B because $p < 0.5$. The asymmetry makes the initial rate of adaptation proportional to p even without clonal interference (Whitlock 1996); as the difference in fitnesses grows, the force of selection is expected to be further biased toward environment A (Holt and Gaines 1992).

To understand how these asymmetries in evolvability interact with the evolution of preference, I performed simulations in which the preference trait was also allowed to evolve. Figure 2 shows two depictions of the same four representative examples of simultaneous evolution of all three traits. Figure 2*A* plots the ratio of mean fitnesses, $\bar{w}_A/\bar{w}_B$, as both adapt. Evolution of the preference, depicted with the changing colors in figure 2, is predicted by the horizontal dashed line drawn from equation (4). Figure 2*B* shows the same data with each performance trait shown on its own axis, with the line defined by equation (4) now appearing as a diagonal line.

Stochastic effects are evident in figure 2 ($r = 0.5$), in which two initially similar trajectories reach divergent outcomes. For $r = 0.5$, 204 out of 400 replicate populations evolved into specialists (for $r = 0$, all 400 evolved into specialists). In these 204 populations, the frequency of strong specialists (those with a 90% or greater chance to reject environment B) crossed 50% when the ratio $\bar{w}_A/\bar{w}_B$ had a mean value of about 1.493—close to the value of 1.444 calculated from equation (4). About 82% of the populations that remained generalists never exceeded the threshold ratio of $\bar{w}_A/\bar{w}_B = 1.444$. Selection

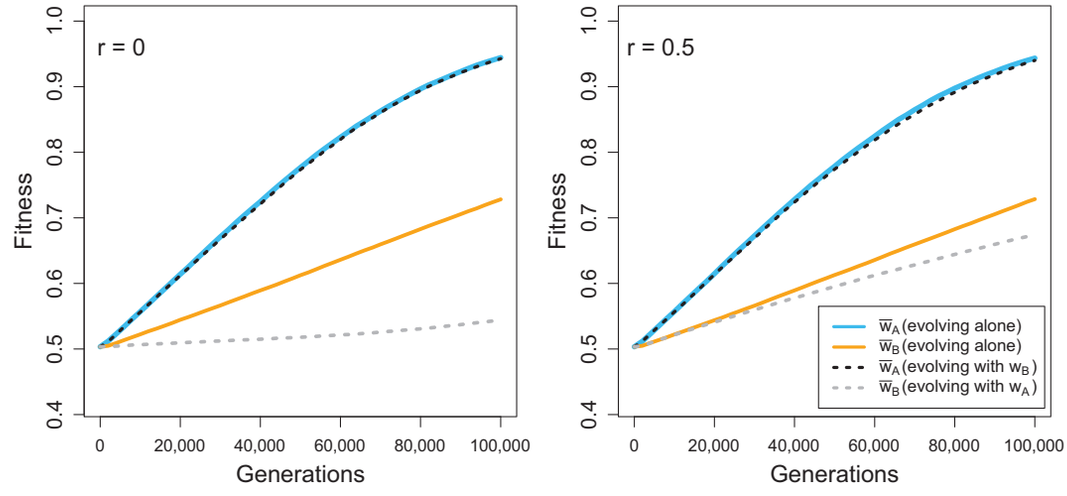

Figure 1: Mean fitness in each environment when each performance trait evolves alone (solid lines) or together (dashed lines). *Left*, no recombination between the sets of loci for performance in each environment ($r = 0$); *right*, full recombination between sets of loci. Four hundred replicates are averaged; 95% confidence intervals (not plotted) span less than 0.01 units on the *Y*-axis. $K = 10{,}000$, $L = 100$, $s_A = s_B = 0.01$, $b = 21$, and mutation per locus is either 5×10^{-4} (when allowed to evolve) or zero (when not allowed to evolve).

for specialists is expected to be weak near the line predicted from equation (4), allowing the random effects of drift and mutation to sometimes override that prediction.

The examples in figure 2 illustrate how asymmetries in evolvability can lead to specialization—operationally defined here based on preference, not performance—as well as the positive feedback that can lock in a narrow niche and prevent the emergence of a superior generalist genotype. These positive feedbacks are evident in the further skewing of the ratio of fitness after environmental preference evolves; as seen in figure 2*B*, fitness in environment B can decay below its initial level even as fitness in environment A is maximized. This decay is a consequence of very weak selection in environment B in a highly specialized population that rarely experiences that environment (Kawecki et al. 1997), as well as a bias toward deleterious mutations (when $b = 21$ and, e.g., half the loci are adapted, then the deleterious mutation

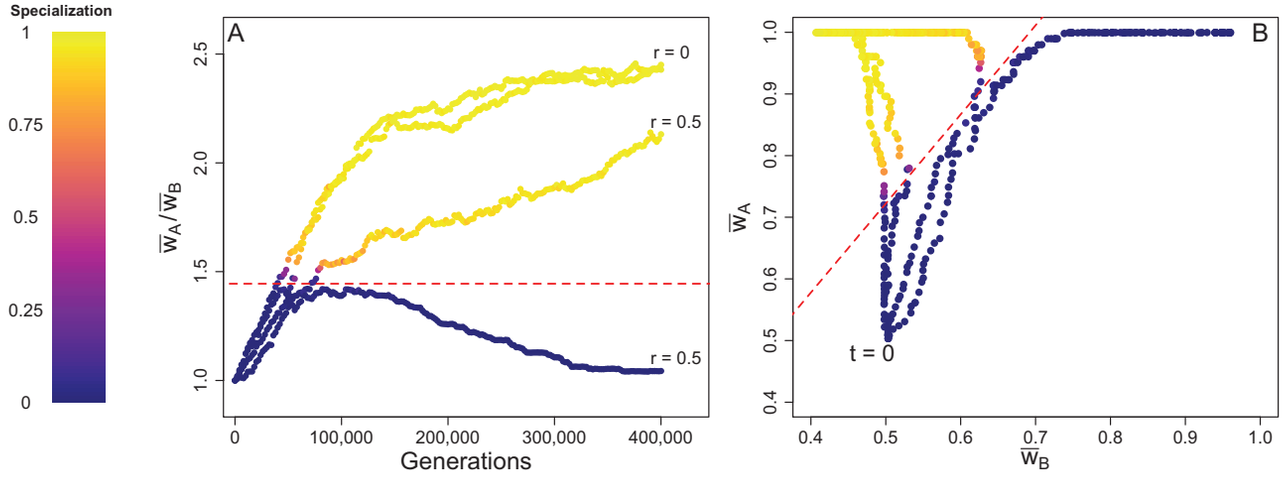

Figure 2: Representative samples of fitness evolution with complete linkage ($r = 0$) and free recombination ($r = 0.5$) between the genetic bases of fitness in environment A and B, as well as the preference locus. Each point is a mean over all individuals in a single replicate population. Specialization is measured as the fraction of the population with a strong preference for environment A (90% chance or greater to reject environment B when encountered). $K = 10{,}000$, $L = 100$, $s_A = s_B = 0.01$, $m_A = 5 \times 10^{-4}$, $\mu_B = 5 \times 10^{-4}$, $\mu_{pref} = 0.001$, $p = 0.25$, $b = 21$, and $c = 0.25$.

rate is twentyfold greater than the beneficial mutation rate).

To further explore these results, I next focused on the quantitative effect of recombination. Figure 3 sweeps across a range of p as well as intermediate levels of recombination. A population is classified as specialist if at least 90% of the individuals reject environment B with at least a 90% chance. Given that the model implements hard selection, frequency-dependent polymorphisms are not expected, and classifying the entire population as either specialist or generalist can therefore be appropriate. However, these thresholds are obviously somewhat arbitrary, and increasing the required number of specialist individuals does reduce the number of populations classified as specialists, particularly at small values of p when selection on environment preference is weak (fig. S1; figs. S1–S3 are available online). Regardless, there is a clear overall pattern: recombination lowers the chance of niche reduction, allowing initial generalists to maintain that generalism as they adapt across a larger range of prevalences of the rare environment. At each level of recombination, specialization can evolve despite substantial search costs if environment B is sufficiently rare. Analysis of the mean preference, rather than the fraction above a threshold, yields the same pattern as figure 3 (fig. S1*C*). Furthermore, in the absence of asymmetry in opportunity for fitness gain, specialists are disfavored (fig. S1*D*).

Figure 2 hints that specialization, once evolved, may persist even after populations have stopped adapting; I next validated this prediction for the set of simulations in figure 3. Among those populations (28,000 populations across all treatments), more than 99% could be classified as either specialist (containing at least 90% of individuals with a preference of at least 0.9 for environment A) or generalist (containing at most 10% of such specialist individuals) by generation 200,000. By the end of the simulations, at generation 400,000 every population could be classified as one or the other extreme. Moreover, none of the populations classified as generalist or specialist at the midpoint had changed their classification by the end. This stability indicates that while the process of adaptation was transient, its effects on preferences and therefore the utilized niche were long-lasting. These results also confirm that polymorphisms do not persist indefinitely under the conditions simulated here.

In figure 3, recombination between the preference locus and the neighboring block of loci for performance in environment A also varies on the basis of the stated r values. Additional simulations with free recombination between the preference locus and performance loci yield indistinguishable results (fig. 2), confirming that the effect of recombination in figure 3 is caused by relieved interference between the genetic bases of performance in the two environments.

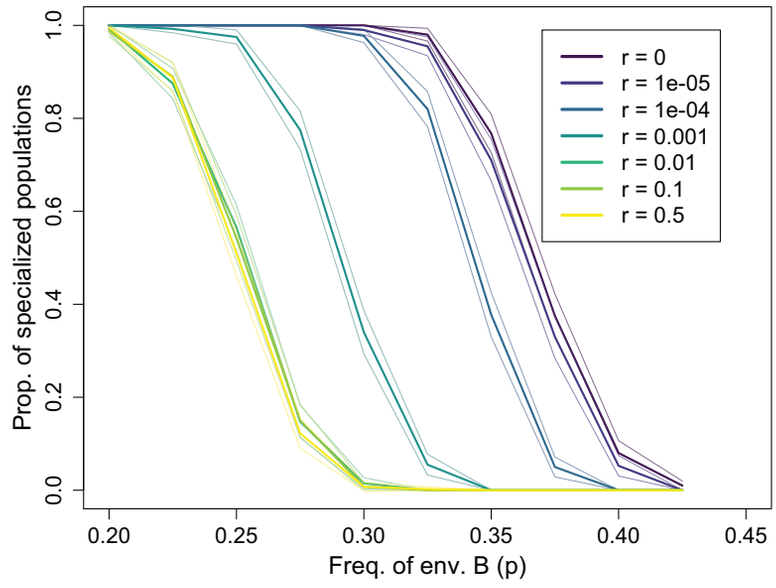

Figure 3: Evolved niche breadths across a range of values of p (X-axis) and r (colors). Thin lines indicate 95% confidence intervals generated by bootstrapping. Four hundred replicates were performed for each combination of parameters. $K = 10{,}000$, $L = 100$, $\mu_A = 5 \times 10^{-4}$, $\mu_B = 5 \times 10^{-4}$, $b = 21$, $\mu_{pref} = 0.001$, and $c = 0.25$.

The Propensity for Adaptive Specialization Is Sensitive to Determinants of Evolvability

In assessing the causes and generality of asymmetric evolvability and the resulting evolution of specialism, I applied the results of Schiffels et al. (2011) and Gomez et al. (2020) to make two predictions. First, I expected the likelihood of specialization to be sensitive to population-genetic parameters determining evolvability, principally N, μ, s, and L. Second, in asexual populations, increasing N or μ will worsen clonal interference and exacerbate the asymmetry between the rates of evolution in the common and rare environments. Also, when simulating populations with different values of these parameters, I evaluated whether asymmetry in the rates of improvement in w_A and w_B sufficed to predict the likelihood of specialization.

Figure 4*A* shows that the product $N\mu$ does correlate positively, in the absence of recombination, with asymmetry of adaptation rates. In this figure, the ratio of fitnesses w_B/w_A is the grand mean of the means of populations, each assayed when the mean of w_A crosses 0.8 (as determined by linear interpolation). This procedure allowed asymmetry to be assessed at a consistent degree of adaptation, to make a fair comparison across treatments with different rates of evolution. Also, μ_{pref} is set to zero in these simulations to measure asymmetry arising from ecology and clonal interference, apart from effects of habitat choice. Finally, to maximize the sensitivity of our outcome measure, note that the values of p (the frequency of the rare environment B) for the $r = 0$ and $r = 0.5$ treatments shown here are those for which half the populations were expected to specialize as estimated from figure 3: $p = 0.251$ for $r = 0$ and $p = 0.367$ for $r = 0.5$. Figure 4*B* shows that this ratio of mean fitnesses does explain the likelihood of specialization that occurred in separate simulations with $\mu_{pref} = 0.001$.

I next explored how these results would change if each trait were determined by fewer loci of larger effect. Figure 5 shows that the degree of asymmetry is generally robust to increasing s and decreasing L (such that sL remains equal to 1 and the initial number of beneficial alleles is adjusted to approximately 50% of maximal fitness in each environment) and is well explained by the ratio of fitness gains in populations with complete linkage, although not in populations with free recombination. Populations with high values of s adapt much faster—when $r = 0.5$, populations achieve a mean w_A of 0.8 in about 56,000 generations when $s = 0.01$ but in only about 2,800 generations when $s = 0.05$, with a similar pattern of about 67,000 generations at $s = 0.01$ and 3,300 generations at $s = 0.05$ for $r = 0$. The window of time for evolution of specialization is therefore likely to be much lower with large s, although why this shorter window might affect populations with recombination specifically (fig. 5*B*) is not clear.

Inspection of figure 2 suggests that deleterious mutation plays a role in maladaptation in environment B in

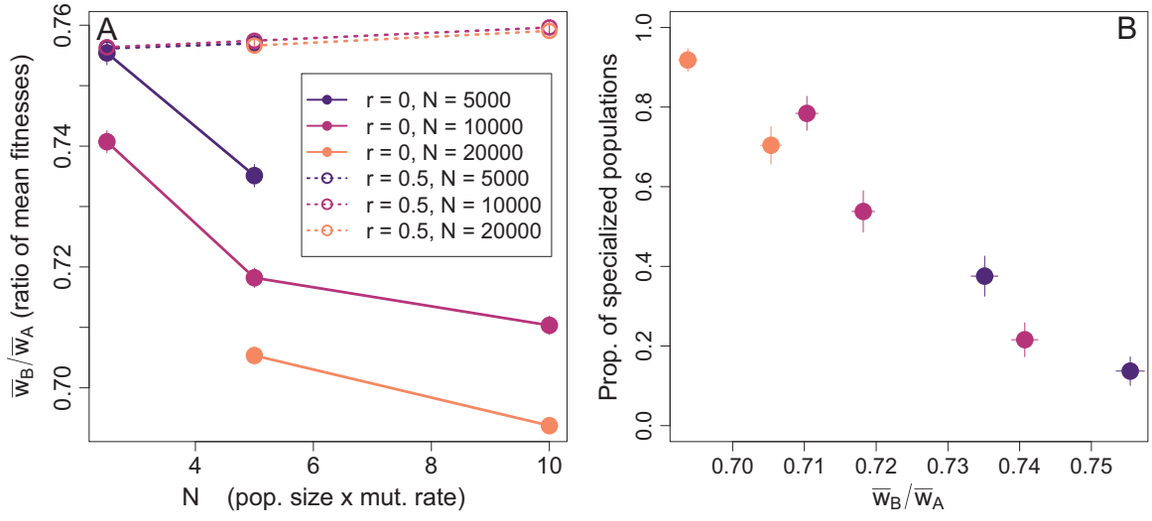

Figure 4: Asymmetry of adaptation and fraction of specialists for combinations of N and μ. *A*, With complete linkage, the ratio of fitness in environment B over environment A generally declines with increases in the product $N\mu$ (where $\mu = \mu_A = \mu_B$), indicating greater asymmetry of evolvability. Without linkage, there is no negative trend, and sensitivity of the ratio to $N\mu$ is very low. *B*, Fraction of replicate populations that end their simulations as specialists plotted against the ratio of fitnesses. As a result of their low sensitivity to $N\mu$ in *A*, populations with $r = 0$ are omitted. $L = 100$, $b = 21$, and $c = 0.25$.

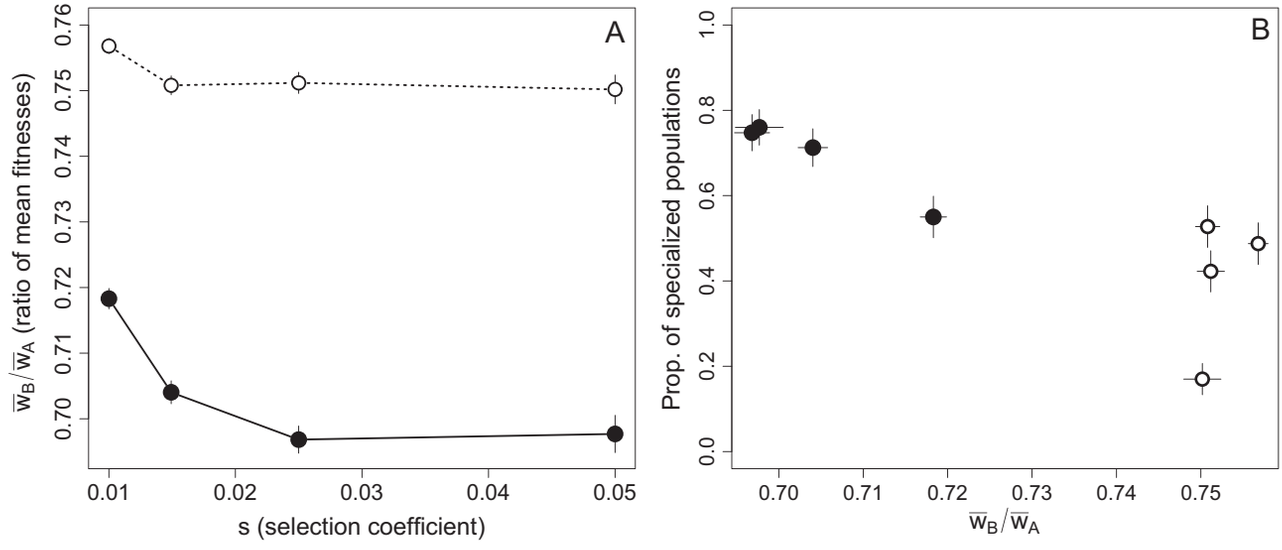

Figure 5: Asymmetry of adaptation and fraction of specialists for combinations of s and L. Filled circles are $r = 0$, and open circles are $r = 0.5$. *A*, The ratio of fitness in environment B over environment A is maintained or slightly declines with increases in s (where $s = s_A = s_B$). *B*, Fraction of replicate populations that specialized, by the criteria applied in figure 3 and elsewhere, plotted against the ratio of fitnesses. $K = 10,000$, $\mu_A = 5 \times 10^{-4}$, $\mu_B = 5 \times 10^{-4}$, and $c = 0.25$.

specialists, prompting the question of how much the bias toward deleterious mutation affects these results. I therefore explored simulations in which the b parameter was set to 2 rather than 21; essentially, this reduced the number of possible alleles at each locus to two, one beneficial and one deleterious. To maintain the overall beneficial mutation rate, μ_A and μ_B were each reduced by tenfold, to 5×10^{-5}. Figure S3 shows that this change reduced the likelihood of specialization, but only for populations with the highest rates of recombination.

Negative Density Dependence Restricts but Does Not Eliminate Adaptive Specialization

In addition to search costs, specialists might also suffer negative effects of crowding. If higher densities in environment A reduced individual reproductive success, then crowding could effectively reduce the ratio of w_A/w_B and therefore inhibit specialization. As described in the section "Model," the number of adults is regulated to a carrying capacity K before adults choose their reproductive environment. Without habitat preferences, we expect $(1 - p)K$ adults to reproduce in environment A and pK to reproduce in environment B. To implement a cost of high densities, I ran alternative simulations in which fecundity in an environment was reduced if the actual number of adults in that environment exceeded the expectation without any preferences (see "Density Dependence"). For example, if, because of habitat preference, environment A contained 20% more adults than the neutral expectation of $(1 - p)K$, then the fitness of all individuals in environment A was divided by a factor of 1.2.

This form of negative density dependence should make specialization less profitable and reduce the likelihood of transitions to specialism. It does—the frequency of the rarer environment at which 50% of replicate populations evolve into specialists (p_{50}) is smaller with crowding effects (fig. 6), indicating that generalism is maintained for a broader range of conditions.

Negative density-dependent effects on fitness, as implemented here, could potentially lead to a frequency-dependent polymorphism of more and less specialized strategies. Such polymorphisms would render the focal summary statistic—the fraction of populations where more than 90% of individuals have more than a 90% rejection rate of environment B—an incomplete picture of the evolving populations. However, after 500,000 generations of evolution only two treatments out of the 48 combinations of r and p examined showed evidence of intermediate polymorphisms in preference—genotypes with prevalences above 10% and preferences between 0.1 and 0.9. With $r = 0.5$, such polymorphisms were found in five out of 244 replicates performed with $p = 0.175$ and nine out of 244 replicates with $p = 0.2$. While polymorphisms in preference may play a role in adaptive evolution, by the end of the simulations most

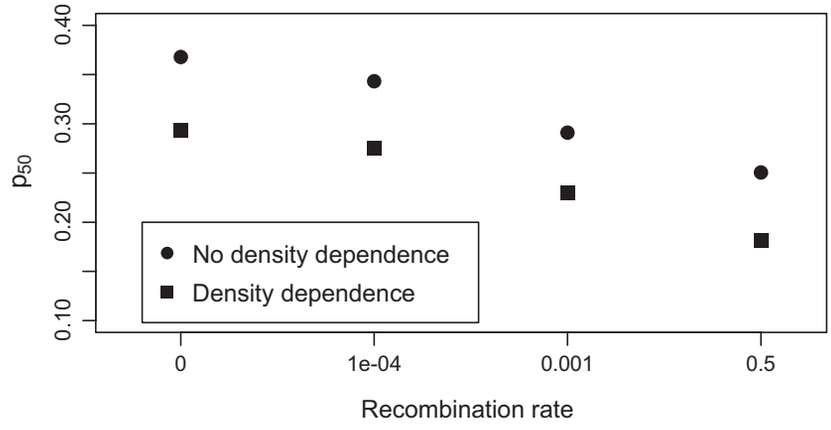

Figure 6: Value of p for which 50% of populations end the simulations as environment A specialists (p_{50}) with and without density dependence (squares vs. circles), across four values of the recombination rate. p_{50} was calculated by logistic regression; 95% confidence intervals were estimated by bootstrapping but were omitted as they were well approximated by the size of the plotting symbols. Data for the "no density dependence" treatment are the same as in figure 3. $K = 10{,}000$, $L = 100$, $\mu_A = 5 \times 10^{-4}$, $\mu_B = 5 \times 10^{-4}$, $b = 21$, $\mu_{pref} = 0.001$, and $c = 0.25$.

populations were essentially monomorphic for preference—as was the case without density dependence.

Discussion

The rejection of an unprofitable resource is a classical prediction (e.g., MacArthur and Pianka 1966), and evolved specialization in accordance with this prediction has been demonstrated empirically (Jasmin and Kassen 2007); the focus here is on the embedding of this process of adaptive specialization within a context in which an environment becomes relatively unsuitable because performance in that environment fails to increase as quickly as performance in an alternative environment. My approach is akin to models of habitat selection, in which populations may adapt either to better exploit unfavorable environments or to avoid them (Templeton and Rothman 1981; Castillo-Chavez et al. 1988;Rausher 1993; Feder and Forbes 2007; Ravigné et al. 2009). However, here the initial cause of a failure to adapt to an environment is that environment's rarity, alongside the potential of ongoing adaptation to a more common environment to interfere with adaptation to the less common environment; at an individual level, the initial and potential fitnesses in the two environments are the same.

While some aspects of these asymmetrical rates of adaptation have been previously predicted (e.g., Whitlock 1996), the analysis here yields several new insights. The first is that a single, prolonged bout of adaptation can cause specialization that persists despite the lack of trade-offs; fitness in the rejected environment cannot readily improve because specialists so rarely visit that environment (see also Kawecki et al. 1997). Specialization is not strictly a local optimum in this model because improvements in w_B are, at worst, neutral rather than deleterious. However, when deleterious mutations outnumber beneficial ones, the waiting time for a lineage to accumulate enough improvements in w_B to make a generalist mutation ($f = 0$) viable may be very long, essentially trapping the population in a suboptimal niche. Therefore, specialization can evolve and persist without trade-offs, with search costs favoring generalists, and without the necessity for continuous, antagonistic coevolution (Kawecki 1998) or an initially low contribution of the rejected environment (Kawecki et al. 1997). The results here also show that linkage, population size, mutation rate, and other contributors to evolvability can have substantial effects on the asymmetry of rates of adaptation and therefore the likelihood that specialization evolves. In this model, clonal interference changes the direction of evolution, not merely its speed.

Although rapid evolution is increasingly recognized as a contributor to ecological processes (e.g., Thompson 1998), much less is said about how the determinants of evolvability could explain patterns of biodiversity (but see Poisot et al. 2011). One argument is that regardless of whether adaptation is fast or slow, populations will still arrive at the same traits (although such predictions have limited usefulness if finding adaptive peaks is unreasonably slow; see Kaznatcheev 2019). This view is challenged by a primary role of rates of adaptations in a number of important scenarios: evolutionary rescue of populations fated for extinction (Bell 2017), rapid evolution in invasive species (Stapley et al. 2015), assembly of communities (Kremer and Klausmeier 2017), host-pathogen coevolution

(Abrams and Matsuda 1997; Cortez and Weitz 2014; Hiltunen et al. 2014), and coexistence among competitors (Lankau 2011). The results here add another such scenario, in which a type of "race" between the performances of two adapting traits can decide which of two distinct, stable outcomes is reached. This work is therefore allied with a diverse set of models that examine competition among distinct solutions to environmental heterogeneity (e.g., Bull 1987; Svardal et al. 2011; Tufto 2015). These models help to illustrate how subtle differences in rates of adaptation could matter, further motivating the idea that evolvability can act as an organizing framework for evolutionary biology (Wagner and Altenberg 1996; Pigliucci 2008).

The results here show one path to specialization without trade-offs, but do the data suggest that an alternative to trade-offs is required? Populations are often found to be adapted to their local conditions, but this fact alone does not allow the inference that trade-offs are responsible for specialism. Kassen (2002) reviewed selection experiments in single environments and concluded that most showed evidence of negative genetic correlations for fitness across environments after selection for specialization. However, only about a third of these showed clear-cut evidence for general costs of adaptation—a decrease in fitness in unselected environments in parallel with specialization to the selected environment. Costs of adaptation may be caused by trade-offs, accumulation of mutations deleterious in unselected environments (Kawecki 1994), or a mix of both (e.g., Reboud and Bell 1997). These distinct causes can be teased apart by experiments that challenge some populations to evolve to multiple environments while others specialize on particular resources or by examining parallel changes across replicate populations adapting to a single environment. Both approaches have yielded mixed results. For example, experiments with vesicular stomatitis virus have repeatedly found that the virus adapts differently to distinct cell types but can adapt to multiple cell lines simultaneously without apparent cost (Turner and Elena 2000; Remold et al. 2008; Smith-Tsurkan et al. 2010). Lenski's long-term evolution experiment with *E. coli* has been used to examine whether patterns in the decay of unselected functions indicates trade-offs or mere accumulation of unselected mutations—while an early analysis supported trade-offs (Cooper and Lenski 2000), a later reexamination supported mutational accumulation as the dominant factor (Leiby and Marx 2014). Replicate experiments that yield trade-offs sometimes but not always are expected if mutations vary in their degree of antagonistic pleiotropy (Bono et al. 2017). Detailed experimental evolution approaches can quantify causation in costs of adaptation; for example, one study with digital organisms distinguished the effects of beneficial and neutral mutations on nonselected traits (Ostrowiski et al. 2007). These approaches for investigating trade-offs are more broadly applicable than just laboratory experiments; for example, repeated evolutionary loss of eyes and pigment in cave fish has been studied with the same dichotomy of trade-offs and mutation accumulation (Jeffrey 2009).

The rather uncertain relevance of trade-offs for specialization in evolution experiments echoes the debate over the causes of specialization in the field (Fry 1996). There are a number of reasons why negative correlations in performance across environments may not be patent even if trade-offs are important (Joshi and Thompson 1995). In some cases, evolution may have also ameliorated significant trade-offs by reducing overlaps in the genetic bases of conflicting traits (Rausher 1988). Futuyma and Moreno (1988) point out that negative performance correlations, even when found, cannot simply be interpreted as the cause of niche breadth because those same performance traits are products of evolution. This dilemma motivates the theoretical approach pursued here in which the values of the two environments, in terms of both initial and potential fitness, are equal; differences in performance and therefore the payoff of exploiting each environment can then emerge only by the interplay of the performance and preference traits.

The question of whether specialization can evolve without being driven by existing trade-offs has been more recently discussed in relation to microbes, particularly viruses (Remold 2012), but earlier was inspired by patterns seen in phytophagous insects (Fry 1996). In light of the results presented here, one puzzling characteristic of phytophagous insects is the existence of highly successful asexual lineages with very broad host ranges (reviewed in Gibson 2019). The work presented here does not model competition between sexual and asexual lineages and therefore does not attempt to predict or explain this empirical pattern. However, there are several connections to be drawn between this model and relevant features of asexual generalist insects that could guide future work. First, as summarized in Gibson (2019), asexual generalist insects typically have limited ability to disperse and choose habitats; this feature could be approximated in this model as a high value of c, the cost of searching. Second, generalists may suffer a reduced ability to evolve in response to change in any one aspect of their niche, but this evolvability deficit might be compensated for by their increased population size (Whitlock 1996). The results presented here rely on a sizeable gap between an organism's current and optimal performance in each environment. Very large populations, whether sexual or asexual, may be able to keep pace with changing environments to such an extent that this performance gap does not arise. This

reasoning parallels that expressed by Gibson (2019), who suggested that large populations with diverse hosts may benefit less from the effects of sex on evolvability, allowing more efficient asexual modes of reproduction to thrive.

The evolution of reduced niche breadth is often studied in models in which disruptive selection leads to partitioning of a niche by coexisting specialists (e.g., Roughgarden 1972), a process that is often linked with sympatric speciation (Doebeli and Dieckmann 2000). My emphasis here was on the evolutionary transition from generalism to specialism in a single lineage, not niche partitioning; hence, important topics for niche partitioning like frequency dependence were relegated to the background. Similarly, there is a rich history of modeling competition among generalists and specialists, which shares overlapping concerns but foregrounds ecological factors like temporal heterogeneity, frequency-dependent selection, and adaptive behavior that allow coexistence (Wilson and Yoshimura 1994). Future work could build on the insights illustrated here to couple evolvability with a more fully realized ecological model, including different and possibly stronger forms of density dependence. Similarly, the absence of trade-offs is not a requirement for the results described here; trade-offs are excluded to focus on the role of evolvability. Future work could readily add trade-offs to the framework explored here to understand how genetic limitations on generalist fitness interact with evolvability differences between traits. The model here also does not consider philopatry or other scenarios in which a lineage experiences one environment for many sequential generations; this choice distinguishes the focus from models of local adaptation and specialization like Ronce and Kirkpatrick (2001).

This model relies on positive feedbacks between habitat preference and performance, mediated by the effect of each on the intensity of selection for the other. Other traits can show the same type of positive interdependence—for example, feeding efficiency in a forager changes the consumption of a given resource, and improvements in feeding efficiency and conversion efficiency of the same resource therefore interact synergistically (Vasconcelous and Rueffler 2020). More broadly, synergistic epistasis among aspects of fitness might lead to accelerated adaptation to one part of a niche, setting the stage for specialism to evolve. Based on this speculation, future work might profitably extend the model explored here to include more explicit links among traits, ecology, and fitness.

These results leverage the findings of Schiffels et al. (2011), as well as later work by Gomez et al. (2020) and Venkataram et al. (2019), to illustrate that asexual adaptation is not just slower than adaptation with recombination but differs in other significant aspects. Other models have predicted differences in the nature of adaptation in asexual versus sexual organisms, such as a difference in epistasis among fixed mutations (Livnat et al. 2008), and as discussed above asexuality is linked with extreme generalism in some insects (Gibson 2019). Still, there has not been a broad, synthetic effort to understand how sex and other important determinants of evolvability not only shape the rate of evolution but also bias adaptation toward distinct phenotypes, niches, or life histories. It is hoped that this work will help push us toward such an effort and start to fully realize the goal of integrating evolvability with our understanding of the forces shaping the niche.

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Data and Code Availability

The data are available in the Dryad Digital Repository (https://doi.org/10.5061/dryad.pc866t1nc; Draghi 2021).

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