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SHORT COMMUNICATION

Crossing the threshold: gene flow, dominance and the critical level of standing genetic variation required for adaptation to novel environments

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Abstract

Genetic architecture plays an important role in the process of adaptation to novel environments. One example is the role of allelic dominance, where advantageous recessive mutations have a lower probability of fixation than advantageous dominant mutations. This classic observation, termed 'Haldane's sieve', has been well explored theoretically for single isolated populations adapting to new selective regimes. However, the role of dominance is less well understood for peripheral populations adapting to novel environments in the face of recurrent and maladaptive gene flow. Here, we use a combination of analytical approximations and individual-based simulations to explore how dominance influences the likelihood of adaptation to novel peripheral environments. We demonstrate that in the face of recurrent maladaptive gene flow, recessive alleles can fuel adaptation only when their frequency exceeds a critical threshold within the ancestral range.

Introduction

The genetic architecture of traits under selection has important consequences for the process of adaptation (e.g. Lande, 1983; Orr & Unckless, 2008; Gomulkiewicz *et al.*, 2010). A classic example is the expectation that adaptation will more often be driven by dominant rather than recessive alleles (Haldane, 1924; Turner, 1981; Charlesworth, 1992). This expectation derives from the fact that rare advantageous alleles are more 'visible' to selection when dominant and thus will increase in frequency more efficiently than recessive alleles (reviewed in Orr, 2010). The decreased probability of establishment for advantageous recessive alleles has been referred to as 'Haldane's sieve' (Turner, 1981; Charlesworth, 1992).

Theoretical work suggests that Haldane's sieve becomes 'leaky' – allowing the establishment of recessive alleles – under certain conditions. Specifically, when a single panmictic population is confronted with a novel environment, the probability of fixation can be independent of dominance if adaptive alleles are at mutation selection-balance prior to the environmental shift (Orr & Betancourt, 2001). Although the role dominance plays in adaptation within a single panmictic population is well understood, its influence on adaptation to novel peripheral environments is less clear. Theoretical results demonstrate that the bias against establishment of recessive alleles is even stronger when populations adapt to divergent environments in the presence of gene flow (Haldane, 1930; Nagylaki, 1975, 1977). Recessive, advantageous alleles simply cannot increase in frequency when confronted with a continuous influx of maladaptive alleles from the ancestral range. Thus, existing theory suggests that adaptation to novel peripheral environments should almost never be driven by the spread of recessive, adaptive alleles.

In contrast to these theoretical results, empirical studies demonstrate that recessive alleles can be responsible for adaptation to novel environments. For instance, a recessive allele has been implicated in the evolution of adaptive blanched coloration in the lizards of White Sands, New Mexico. White Sands is a geologically recent formation (~6000 years old, Kocurek *et al.*,

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2007) and is a unique 'island' of white gypsum surrounded by the dark soil substrate of the Chihuahuan desert. Three lizard species have blanched, substrate-matching coloration at White Sands and dark coloration in the surrounding habitat and the rest of their ranges (Rosenblum, 2006). Blanched coloration in one of the white sands lizard species, the Little Striped Whiptail (*Aspidoscelis inornata*), is caused by a recessive mutation in the melanocortin-1 receptor gene (*Mc1r*) (Rosenblum *et al.*, 2010). There is also strong evidence for ongoing migration between white sands and dark soils populations of *A. inornata* (Rosenblum, 2006; Rosenblum & Harmon, 2011). Although the extent of maladaptive gene flow is not always quantified, additional examples of adaptation to novel environments conferred by recessive alleles exist (e.g. body armour in *Gasterosteus* stickleback fish (Chan *et al.*, 2010), colour in mimetic *Heliconius* butterflies (Chamberlain *et al.*, 2009) and colour in *Mimulus* monkeyflowers (Bradshaw & Schemske, 2003)). Thus, in contrast to predictions of existing theory, recessive alleles seem quite capable of driving adaptation to novel environments even in the face of maladaptive gene flow.

Here, we develop an extension of the mainland–island model originally developed by Haldane (1930) in an effort to reconcile existing theory with these recent empirical observations. Using a combination of analytical approximations and individual-based simulations, we demonstrate that the potential for recessive alleles to contribute to adaptation in the face of maladaptive gene flow rests on the amount of standing genetic variation initially present in the ancestral range.

The model

We study the process of adaptation to a novel 'island' environment in the face of gene flow from a large 'mainland' population. Adaptation is assumed to be mediated by a single diallelic locus, and individuals are assumed to mate at random. Each generation, a proportion, m, of individuals move from the mainland to the island, with negligible movement in the opposite direction (from the island to the mainland). We assume the '*A*' allele is maladaptive within the mainland and maintained at a fixed (small) frequency, p_m, determined by the balance of local selection and mutation. In contrast, the '*A*' allele is favoured within the novel island environment where it has frequency p_i. Specifically, we assume that the fitnesses of genotypes on the island are:

$$W_{AA} = 1 \tag{1a}$$

$$W_{Aa} = 1 - (1 - h)s \tag{1b}$$

$$W_{aa} = 1 - s \tag{1c}$$

where h is the dominance coefficient and determines the extent to which allele '*A*' is dominant ($h = 1$) vs. recessive ($h = 0$), and s measures the strength of selection against '*a*' alleles in the novel environment.

We take two approaches to analysing this general model. In the first, we make several sets of simplifying assumptions that allow development of analytical approximations that yield simple but valuable insights. In the second, we use individual-based simulations to relax the assumptions of our approximations and evaluate the robustness of our analytical results.

Analytical approximations

The case in which the advantageous allele, *A*, is very rare in the ancestral environment

We begin by re-deriving several classical population genetic results originally developed by Haldane (1930) and Nagylaki (1975, 1977). Our goal is to show how these results have important but underappreciated implications for the genetic basis of adaptation to novel environments. We then explore these implications more thoroughly in the next section using an approximation that relaxes a key assumption of this classical work. For simplicity, we assume selection on the island is weak (s of small order ε), the rate of gene flow is small (m of small order ε), population sizes are sufficiently large for the effects of genetic drift to be ignored and that mutation is negligible. In addition, we assume the frequency of the '*A*' allele on the mainland is low (p_m of small order ε), such that levels of standing genetic variation for adaptation to the novel environment within the ancestral mainland population are negligible. Under such conditions, the change in the frequency of the adaptive allele in the novel island environment is given by:

$$\Delta p_i \approx s p_i q_i^2 - m p_i + O(\varepsilon^2) \tag{2a}$$

for the case where the adaptive allele is dominant, and by:

$$\Delta p_i \approx s p_i^2 q_i - m p_i + O(\varepsilon^2) \tag{2b}$$

for the case where the adaptive allele is recessive.

Analysis of eqn (2) reveals a very bleak fate for rare, advantageous, recessive alleles. Specifically, if $m > s/4$, an advantageous allele is inevitably lost, and even if $m < s/4$, the allele must exceed a critical frequency threshold in the island population in order to increase in frequency and contribute to adaptation within the island population:

$$P_{i,\mathrm{crit}} = \frac{1}{2} - \frac{\sqrt{-4m + s}}{2\sqrt{s}} \tag{3}$$

(Appendix S1, Haldane, 1930; Nagylaki, 1975). If this critical threshold is exceeded, an advantageous recessive allele can reach an appreciable equilibrium frequency in the island population representing a balance

between selection and gene flow:

$$\hat{p}_i = \frac{1}{2}\left(1 + \sqrt{\frac{-4m+s}{\sqrt{s}}}\right) \quad (4)$$

Together, these results demonstrate that adaptation to a novel island environment cannot occur through the deterministic spread of new mutations if such mutations are recessive, because the starting frequency will not exceed the critical threshold, even in small populations. These results are summarized graphically in (Fig. 1a).

In contrast to the stringent conditions for the spread of advantageous recessive alleles, advantageous dominant alleles face no special hurdles when confronted with gene flow. Specifically, as long as the strength of local selection exceeds the rate of gene flow ($s > m$), adaptation occurs and the allele reaches an equilibrium allele frequency equal to:

$$P_i \to 1 - \frac{\sqrt{m}}{\sqrt{s}} \quad (5)$$

(Appendix S1). Consequently, adaptation to novel environments can readily occur through the spread of new advantageous dominant mutations even in the face of substantial maladaptive gene flow (Fig. 1b).

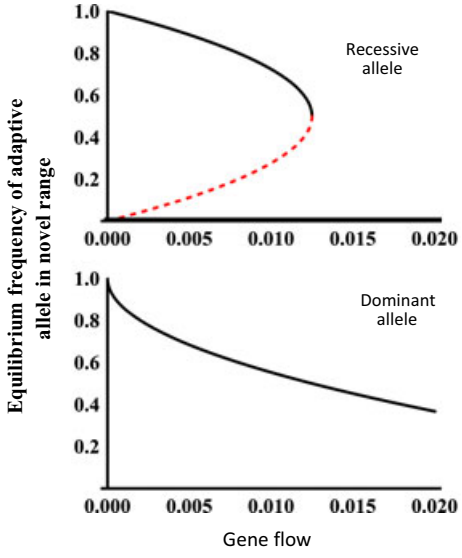

Fig. 1 Equilibrium frequencies of the adaptive '*A*' allele on the island as a function of the rate of gene flow from the mainland, *m*. The top panel shows the three equilibria for recessive alleles. The black line indicates a locally stable equilibrium, and the red dashed line indicates a locally unstable equilibrium. If the initial allele frequency is above the red dashed line, the allele increases in frequency, whereas if initial allele frequency is below the red dashed line the allele frequency decreases. The bottom panel shows the single (locally stable) equilibrium for dominant alleles. The strength of selection favouring the '*A*' allele within the novel island population was $s = 0.05$ in both panels.

Together, the results re-derived in this section suggest that an important dichotomy exists between adaptation to novel environments fuelled by recessive and dominant alleles. Specifically, advantageous dominant alleles can increase in frequency irrespective of their initial frequency. Therefore, dominant alleles can easily spread and contribute to adaptation, whether they are already segregating in the ancestral range or whether they arise from new mutations. In contrast, advantageous recessive alleles can spread only if their initial frequency exceeds a critical threshold. Thus, adaptation to novel environments mediated by recessive alleles may require a threshold level of standing genetic variation within the ancestral range. In the next section, we develop an approximation that allows us to confirm the existence of such a threshold.

The case in which the advantageous allele is not rare in the ancestral environment

A key assumption of the results presented in the previous section is that the '*A*' allele – which is advantageous within the novel island environment – is rare within the ancestral, mainland habitat. Thus, the results ignore the possibility that adaptation to the novel environment has its origins in standing genetic variation within the mainland population. In this section, we relax this assumption and explore how the frequency of the '*A*' allele within the mainland population influences adaptation to the novel island environment.

When the frequency of the '*A*' allele is not negligible within the mainland population, the change in its frequency within the novel environment is given by:

$$\Delta p_i \approx s p_i q_i^2 + m(p_m - p_i) + O(\varepsilon^2) \quad (6a)$$

for the case where the adaptive allele is dominant and by:

$$\Delta p_i \approx s p_i^2 q_i + m(p_m - p_i) + O(\varepsilon^2) \quad (6b)$$

for the case where the adaptive allele is recessive (Appendix S1).

Analysis of eqns (6) reveals that a critical threshold level of standing genetic variation exists for adaptation via recessive alleles anytime $m < s/3$ (Appendix S2). Under such conditions, adaptation is inevitable if the frequency of the '*A*' allele within the ancestral mainland population satisfies:

$$p_m > \frac{(9ms - 2s^2 + 2\sqrt{-27m^3s + 27m^2s^2 - 9ms^3 + s^4}}{27ms} \quad (7)$$

If eqn (7) is not satisfied, but the frequency of the '*A*' allele within the ancestral mainland population does satisfy:

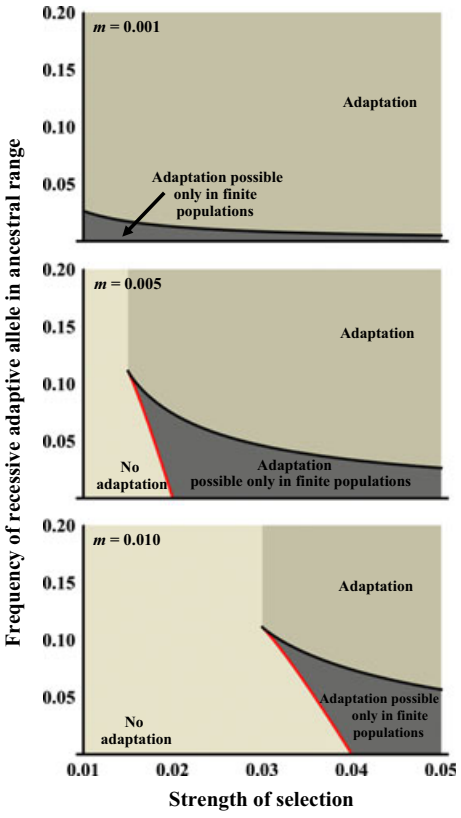

Fig. 2 Plots of critical values of mainland allele frequencies given by (eqn 7) and (eqn 8) as a function of the strength of selection within the island population for three different rates of gene flow. Critical value (eqn 7) is indicated by black line, and critical value (eqn 8) by the red line. Within the parameter space defined by the region labelled 'no adaptation', recessive adaptive alleles will inevitably be lost from the island population irrespective of island population size. In contrast, within the region labelled 'adaptation possible only in finite populations', a recessive adaptive allele can increase in frequency and become common if the island population is sufficiently small for random genetic drift to increase the frequency of the allele above a critical frequency threshold. In the region labelled 'adaptation', a recessive adaptive allele will increase in frequency and become common irrespective of island population size.

$$p_m > \frac{(9ms - 2s^2 - 2\sqrt{-27m^3s + 27m^2s^2 - 9ms^3 + s^4}}{27ms} \quad (8)$$

adaptation can occur if the frequency of the adaptive allele on the island exceeds a threshold value. In such cases, deterministic adaptation is impossible, but stochastic effects within small, island populations could, in principle, result in adaptation. Finally, if neither (eqn 7) nor (eqn 8) is satisfied, adaptation to the novel environment is impossible even in small, finite populations. In contrast to recessive alleles, analysis of (eqn 6) shows that no such critical threshold exists for dominant alleles. For dominant alleles, the frequency of the adaptive '*A*' allele inevitably increases to an equilibrium value where selection and gene flow are balanced (Appendix S2). These results are summarized in Fig. 2.

Individual-based simulations

Our analytical approximations suggest that – in the face of recurrent gene flow from a mainland population – a critical threshold level of standing genetic variation is required for advantageous recessive alleles to establish in a novel environment. In contrast, no such threshold level of standing genetic variation appears to exist for dominant alleles. However, our analytical approximations make several potentially important assumptions, including large population size, weak selection, low rates of gene flow and complete dominance ($h = 1$ or $h = 0$). To relax these assumptions, we developed and analysed individual-based simulations. These simulations assume an explicit two population structure consisting of an ancestral mainland population with a fixed frequency of the '*A*' allele, p_m, and a novel population where the '*A*' allele is favoured. Within each generation, island individuals first experience viability selection, which they survive with probabilities given by (eqn 1). Individuals then move from the mainland to the island with probability, m. After movement, mating occurs by drawing two individuals at random from within the island population and producing an offspring following standard rules of Mendelian inheritance. Mating continues until K_i offspring individuals have been produced in the island population, after which point the parental generation dies. Finally, mutation occurs within the island population at a per-allele rate μ.

Simulations were initialized by assigning genotypes to individuals within the mainland and island populations. Mainland individuals were assigned genotypes by drawing alleles at random with the probability of drawing the '*A*' allele equal to the fixed frequency of the allele within the mainland population, p_m. In contrast, the '*A*' allele was assumed to be initially absent from the island population such that all individuals were initially of genotype '*aa*'. Simulations were then run for 6000 generations, and final allele frequencies and genotype frequencies recorded for the island population.

We used these simulations to study a broad range of parameter combinations including two strengths of selection {0.05, 0.25}, two island population sizes {250, 5000}, six rates of gene flow ranging between $m = 0.013$ and $m = 0.065$, fourteen frequencies of the '*A*' allele within the mainland population ranging from $p_m = 0.00$ to $p_m = 0.13$ and three levels of dominance {$h = 0$, $h = 1$, and $h = 0.1$}. The mutation rate within the island population was set to $\mu = 1 \times 10^{-6}$ for all simulations.

Analysis of simulation results provides strong support for our analytical predictions. Specifically, as long as the population size within the novel island environment is not too small, simulation and analytical results are highly concordant (Fig. 3). Even when island population size becomes small, agreement remains quite good, although random genetic drift in small island populations generates significant noise (Fig. 3). One apparent discrepancy between our analytical prediction and simulation results is the slight underestimation of equilibrium allele frequency in some cases (e.g. Recessive advantageous allele with large population size). This slight discrepancy is most likely a simple consequence of our assumption of weak selection which ignores terms of order s^2. In addition to providing support for our analytical predictions, our simulations allow us to investigate whether a critical threshold level of standing genetic variation remains when the adaptive allele is only partially recessive. This investigation revealed that a critical value of standing genetic variation persisted even for partially recessive alleles, although its value was reduced (Figure S1).

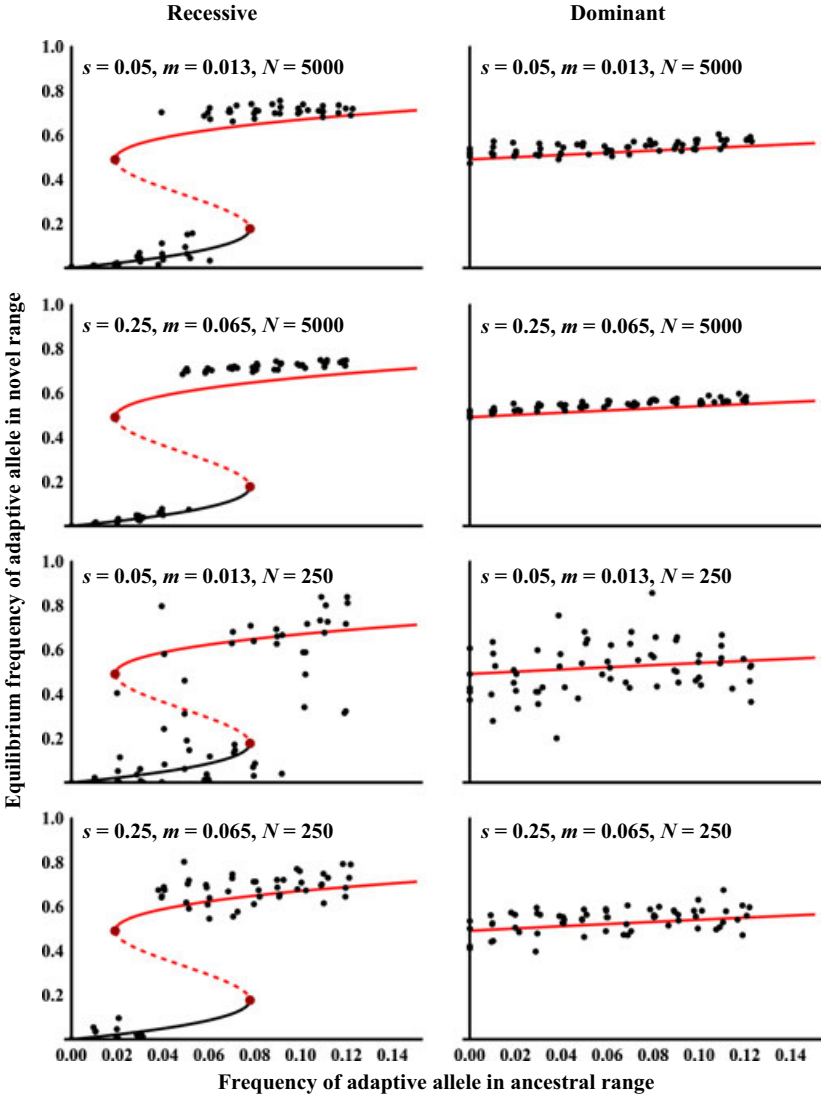

Fig. 3 Analytical predictions for the equilibrium frequency of the adaptive allele in the island population as a function of its frequency within the mainland population. The solid black line indicates a locally stable equilibrium, the dashed red line a locally unstable equilibrium and the solid red line an additional locally stable equilibrium. Large, red dots indicate the critical values predicted by (eqn 7) and (eqn 8). Small black dots indicate allele frequencies in generation 6000 of individual-based simulations for the parameter combinations shown in each individual panel.

Discussion

Our analyses clarify how standing genetic variation, dominance and gene flow interact to shape the process of adaptation to novel peripheral environments by extending classical population genetic results originally derived by Haldane (1930) and Nagylaki (1975). Specifically, we develop a new approximation that allows for standing genetic variation within the ancestral population. We then use this approximation to explore the bias against recessive alleles ('Haldane's Sieve') in the specific case where gene flow connects two differentially adapted populations (i.e. the ancestral 'mainland' and the novel 'island'). Understanding the role of allelic dominance in this context is particularly important given the recent interest in understanding the dynamics of divergence and speciation when they occur in the face of gene flow (e.g. Pinho & Hey, 2010; Smadja & Butlin, 2011)

It is well established that gene flow can retard local adaptation due to the swamping effects of maladaptive alleles (e.g. Haldane, 1930; Slatkin, 1973, 1987; Lenormand, 2002). However, our results show that the consequences of gene flow for adaptation in peripheral populations are qualitatively different for dominant vs. recessive alleles. Dominant alleles can become frequent within the novel environment even when confronted with maladaptive gene flow from the ancestral range. Thus, as long as the strength of local selection exceeds the rate of gene flow, advantageous dominant alleles can contribute to adaptation, regardless of whether they arise from new mutation or standing genetic variation. In contrast, recessive alleles can overcome the swamping influence of maladaptive gene flow only when already present above a critical threshold frequency within the ancestral range. The reason recessive alleles are so sensitive to maladaptive gene flow is that selection acting on rare recessive alleles is inefficient due to masking. If the adaptive recessive allele is absent or rare within the ancestral range, gene flow prevents the frequency of the recessive allele from ever rising sufficiently for selection to gain traction on it within the novel range. Thus, standing genetic variation (SGV) within the ancestral range is required for recessive alleles to escape the swamping influence of gene flow. Obviously, the presence of standing genetic variation within the ancestral range also promotes adaptation to novel environments by eliminating the waiting time required for novel mutation. However, this positive influence of standing genetic variation applies to both dominant and recessive alleles.

Our work extends results derived for isolated populations adapting to environmental change (Orr & Betancourt, 2001). Specifically, Orr and Betancourt (2001) demonstrated that the probability of fixation for an advantageous allele is generally independent of dominance when the allele is at mutation–selection equilibrium prior to environmental change. Thus, when adaptation occurs in response to environmental change within a single population, recessive alleles may be just as likely to contribute to adaptation as dominant alleles. Our results concur with Orr and Betancourt (2001) in that we show SGV greatly enhances the likelihood that recessive alleles overcome the swamping influence of maladaptive gene flow. However, our results differ from Orr and Betancourt (2001) in one critical way. When divergence occurs in the face of gene flow, SGV does not level the playing field between dominant and recessive advantageous alleles. When gene flow is considered, recessive alleles are still at a disadvantage. All else being equal, dominant advantageous alleles are therefore more likely to fuel adaptation to novel peripheral environments in the face of maladaptive gene flow from the ancestral range. Thus, it is critical to consider the context in which adaptation occurs to fully understand the role of dominance in adaptation to novel environments.

Theoretical models on the role of dominance in adaptive evolution generate straightforward predictions to evaluate in natural systems. Many previous authors have discussed the bias against recessive alleles in adaptive evolution (e.g. Turner, 1981; Charlesworth, 1992). However, our results generate an additional prediction, which applies anytime adaptation to a novel environment is driven by recessive alleles and occurs in the face of maladaptive gene flow. Specifically adaptive, recessive alleles are likely derived from standing genetic variation in the ancestral range rather than new mutation. Consequently, in the cases of adaptation from recessive alleles in the face of gene flow, we expect the recessive allele to be segregating at an appreciable frequency within the ancestral range.

The prediction that adaptive recessive alleles likely derive from standing genetic variation receives at least some empirical support. For instance, in stickleback, reduced body armour in freshwater fish is controlled by a recessive allele at the *Eda* locus, and this allele is found at low frequency in the ancestral marine population (Colosimo *et al.*, 2005). Similarly, blanched coloration in *A. inornata* at White Sands is controlled by a recessive allele despite ongoing migration between differentially adapted populations (Rosenblum, 2006). The derived recessive allele in this system is found in dark soil populations (Rosenblum *et al.*, 2010), suggesting the allele may have arisen from standing genetic variation. There are other examples of recessive mutations contributing to adaptation (e.g. colour in mimetic *Heliconius* butterflies (Chamberlain *et al.*, 2009) and colour in *Mimulus* monkeyflowers (Bradshaw & Schemske, 2003)), which could also be used to evaluate our prediction.

In summary, our results demonstrate that standing genetic variation within the ancestral range is essential if recessive alleles are to overcome the swamping

influence of maladaptive gene flow and contribute to adaptive evolution in a novel environment. Although existing empirical studies provide some qualitative support for this result, additional empirical work in natural systems and using experimental evolution could offer valuable tests of the theory.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Conditions for adaptation to novel environment when mainland standing genetic variation is absent.

Appendix S2 Conditions for adaptation to novel environment when mainland standing genetic variation is present.

Figure S1 The frequency of the adaptive allele within the island population as a function of its frequency within the mainland population.

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