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The consequences of phenotypic plasticity for ecological speciation

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Abstract

We use an individual-based numerical simulation to study the effects of phenotypic plasticity on ecological speciation. We find that adaptive plasticity evolves readily in the presence of dispersal between populations from different ecological environments. This plasticity promotes the colonization of new environments but reduces genetic divergence between them. We also find that the evolution of plasticity can either enhance or degrade the potential for divergent selection to form reproductive barriers. Of particular importance here is the timing of plasticity in relation to the timing of dispersal. If plasticity is expressed after dispersal, reproductive barriers are generally weaker because plasticity allows migrants to be better suited for their new environment. If plasticity is expressed before dispersal, reproductive barriers are either unaffected or enhanced. Among the potential reproductive barriers we considered, natural selection against migrants was the most important, primarily because it was the earliest-acting barrier. Accordingly, plasticity had a much greater effect on natural selection against migrants than on sexual selection against migrants or on natural and sexual selection against hybrids. In general, phenotypic plasticity can strongly alter the process of ecological speciation and should be considered when studying the evolution of reproductive barriers.

Introduction

Ecological speciation occurs when divergent natural selection causes adaptive divergence, which then leads to reproductive isolation (Schluter, 2000; Rundle & Nosil, 2005). The importance of ecological speciation is now supported by a broad array of theoretical models (Dieckmann & Doebeli, 1999; Doebeli & Dieckmann, 2003; Fry, 2003; Gavrilets & Vose, 2005, 2007; Gavrilets *et al.*, 2007; Thibert-Plante & Hendry, 2009), meta-analyses (Funk *et al.*, 2006) and empirical studies of specific natural systems (Rundle *et al.*, 2000; Barluenga *et al.*, 2006; Nosil, 2007; Grant & Grant, 2008). At the same time, it is increasingly apparent that divergent selection does not always cause substantial progress towards ecological speciation, because this progress can depend on genetic architecture, the dimensionality of selection, the nature of mating systems and the extent of dispersal (Berner *et al.*, 2009; Hendry, 2009; Nosil *et al.*, 2009; Labonne & Hendry, 2010). Our goal here is to explicitly examine one additional factor, phenotypic plasticity, that might influence progress towards ecological speciation.

Phenotypic plasticity is the tendency of a particular genotype to produce different phenotypes under different environmental conditions. Phenotypic plasticity is very common in nature (DeWitt & Scheiner, 2004) and often appears to be adaptive (Sultan, 2000; Pigliucci, 2001). That is, phenotypic plasticity allows organisms to deal with unpredictable environments by altering their phenotype to be better suited to local conditions, as opposed to genetic canalization that would eliminate this option. In particular, theoretical models have shown that phenotypic plasticity can be favoured over adaptive genetic divergence for organisms living in environments that vary in space (Via & Lande, 1985; Zhivotovsky *et al.*, 1996; Alpert & Simms, 2002; Schlichting, 2004; Richter-Boix

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et al., 2006; Lind & Johansson, 2007; Hollander, 2008) or time (Ernande & Dieckmann, 2004; Gabriel, 2005; Stomp *et al.*, 2008; Svanbäck *et al.*, 2009; Lande, 2009). And yet, phenotypic plasticity is not a panacea because it has limits (information reliability, lag time, developmental range and epiphenotype) and costs (maintenance, production, information acquisition, developmental instability, pleiotropy and epistasis) (reviewed in DeWitt *et al.*, 1998; van Buskirk & Steiner, 2009). Moreover, phenotypic plasticity can be maladaptive under some circumstances (Langerhans & DeWitt, 2002; Grether, 2005).

How might phenotypic plasticity influence progress towards ecological speciation? In such cases, reproductive barriers are ecologically based, including natural and sexual selection against migrants and hybrids (Schluter, 2000; Rundle & Nosil, 2005). Also note that we are not considering reproductive barriers such as intrinsic genetic incompatibilities. We here organize some of the possibilities around previous conclusions about ecological speciation.

Intermediate levels of divergent selection are most conducive to ecological speciation

This is a frequent outcome of theoretical models (Gavrilets *et al.*, 2007; Thibert-Plante & Hendry, 2009) and results from the intersection of two contrasting effects. On the one hand, an increase in divergent selection increases adaptive divergence, which should result in stronger ecologically driven reproductive isolation. On the other hand, increasing divergent selection reduces the chances that a new environment will be colonized, thus reducing the opportunity for ecological speciation (Garant *et al.*, 2007; Thibert-Plante & Hendry, 2009). This expectation is certainly borne out in natural populations as all lineages occupy only a limited range of available environments, although this constraint would be less for species with very wide ecological niches. For instance, threespine stickleback (*Gasterosteus aculeatus*) can adapt to stream environments as long as the habitat is not too extreme (Moore & Hendry, 2009). We propose that plasticity might here have two potentially opposing effects (Baldwin, 1896; Price *et al.*, 2003; Ghalambor *et al.*, 2007). First, it might reduce progress towards ecological speciation, because adaptive plasticity should reduce divergent selection on the genetic component of the traits. Second, it might increase progress towards ecological speciation, because plasticity should enhance colonization of new environments.

Adaptive divergence leads to natural selection against migrants

This is one of the most obvious, general and widely supported predictions of ecological speciation (Via *et al.*, 2000; Hendry, 2004; Nosil *et al.*, 2005; Thibert-Plante & Hendry, 2009). The reason is that organisms adapted to local environments should, by definition, perform better in those environments than individuals adapted to other environments (Schluter, 2000; Hereford, 2009). Indeed, many studies of natural populations have documented strong natural selection against migrants (Nosil *et al.*, 2005). For instance, pea aphids (*Acyrthosiphon pisum* Harris) that move between different host plants have dramatically lower survival than those that move between similar host plants (Via *et al.*, 2000). We suspect that any modifying influence of plasticity on selection against migrants will depend on whether the plastic response occurs before or after dispersal. If plasticity occurs before dispersal (e.g. migration of adults), it might increase selection against migrants because plasticity would tailor phenotypes to their original environment, making them less well suited for the environment to which they migrate. If plasticity occurs after dispersal (e.g. migration of juveniles), then it might decrease selection against migrants because developing individuals could then change their phenotypes to better suit the new environment. To be concise, we will use dispersal before or after plasticity to describe the two developmental scenarios.

Adaptive divergence can lead to the evolution of female preferences for local males (i.e. sexual selection against migrants)

This process for the origin of positive assortative mating is expected to occur most easily when a trait under divergent selection also pleiotropically influences mate choice, such as in 'magic trait' models (where the exact same trait is under both natural and sexual selection) (Dieckmann & Doebeli, 1999; Gavrilets, 2004). Such processes are also possible if females have a preference gene that targets the male ecological trait or sometimes even if the female preference gene targets marker traits in males that are separate from the ecological trait (Kondrashov & Kondrashov, 1999; Fry, 2003; Doebeli, 2005; van Doorn *et al.*, 2009). In these latter cases, female preferences can evolve as a result of direct or indirect selection to avoid mating with locally maladapted males (Kirkpatrick, 2001; Servedio & Noor, 2003). Empirical work on natural populations suggests that adaptive divergence sometimes leads to positive assortative mating, such as in benthic-limnetic stickleback (Rundle *et al.*, 2000) and Timema walkingsticks (Nosil *et al.*, 2002), but not always, as in Clouded Sulphur butterflies (Ellers & Boggs, 2003). We suspect that the influence of plasticity on the evolution of assortative mating will depend on when plasticity occurs relative to dispersal, for the same reasons as described in the previous section. In particular, sexual selection against migrants might be strengthened by plasticity that occurs before dispersal but weakened by plasticity that occurs after dispersal.

Adaptive divergence leads to natural and sexual selection against hybrids

The reason for this prediction is that hybrids are often phenotypically intermediate to the parental forms and should therefore be maladapted in both parental environments. Fitting with this expectation, selection against hybrids appears to be a common contributor to ecological speciation (Schluter, 1995, 2000; Rundle & Nosil, 2005). For example, hybrids are disfavoured in both parental environments for benthic-limnetic threespine stickleback (Schluter, 1995) and pea aphids (Via *et al.*, 2000). We suspect that plasticity will weaken this effect because hybrids produced in a given environment might be able to shift their phenotypes closer to those that are locally adapted for that environment.

Ecological speciation might be detectable by reduced gene flow at unlinked neutral genetic markers

This idea has received mixed support from empirical studies, with some finding that divergent selection (or adaptive divergence) reduces neutral gene flow (Grahame *et al.*, 2006; Nosil *et al.*, 2008; Berner *et al.*, 2009), but others not finding such a pattern (Emelianov *et al.*, 2004; Aguilar *et al.*, 2005; Crispo *et al.*, 2006). Theoretical models have helped to understand this variation by revealing (1) that divergent selection can indeed cause a generalized barrier to gene flow when strong selection acts against the whole genome of migrants and first-generation hybrids (i.e. before recombination between parental genomes) but (2) that this effect is relatively weak, inconsistent and difficult to detect empirically, because selected genes and unlinked neutral markers become decoupled as a result of recombination in the gametes of hybrid offspring (Via, 2001; Wu, 2001; Gavrilets & Vose, 2005; Nosil *et al.*, 2007; Thibert-Plante & Hendry, 2009; Feder & Nosil, 2010; Thibert-Plante & Hendry, 2010). We suspect that plasticity will further weaken any ecological patterns in neutral marker data because phenotypic divergence becomes less closely tied to genetic divergence.

We here study these topics by incorporating phenotypic plasticity into individual-based numerical models of ecological speciation. We focus on two spatially discrete environments with different ecological conditions (i.e. different optimal trait values). We start with a population adapted to one of the environments, with some individuals then moving to the other environment. If a population becomes established in the new environment, then adaptation proceeds under continuing dispersal between the environments (i.e. parapatry). Given that the two environments impose divergent selection that causes adaptive divergence, ecological speciation is expected. Onto this basic framework we add plasticity in the ecological trait. This plasticity can occur before or after dispersal, can have limits and can be costly. Importantly, we do not impose a particular level of plasticity but rather allow it to evolve.

Modelling framework

Our work adopts several important elements from previous numerical models of adaptive radiation and ecological speciation (Gavrilets & Vose, 2005; Yukilevich & True, 2006; Gavrilets *et al.*, 2007; Gavrilets & Vose, 2009; Thibert-Plante & Hendry, 2009). In particular, we use hard selection (Christiansen, 1975) rather than soft selection (Kisdi & Geritz, 1999; Spichtig & Kawecki, 2004) to provide a more realistic dynamic for changing population size (during colonization). We also here employ reasonable mutation rates (Dallas, 1992; Brinkmann *et al.*, 1998; Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007; Gavrilets & Vose, 2007, 2009). At the same time, we implement two major differences from those previous models. First, we do not here, nor in Thibert-Plante & Hendry (2009), model the evolution of habitat choice. The main reason is that this situation has been frequently modelled (Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007; Gavrilets & Vose, 2009), and we are here more interested in what happens when individuals from different environments encounter each other. Second, the optimal phenotypes in our model are not at the extremes of the theoretically possible range (as they are in Dieckmann & Doebeli, 1999; Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007; Gavrilets & Vose, 2009). The reason for this choice is that we want to allow phenotypes to potentially overshoot their optima, which might reduce a bias towards the optimal phenotypes. The code is written in Fortran 90 and available upon request.

Environment and dispersal

The environment is represented by an optimal trait value θ and has a maximum carrying capacity of K_0. The actual carrying capacity (K) is a function of the level of adaptation of the population (Kot, 2001; Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007). Two environments (two θ) are present and they are not spatially overlapping, which is a frequent occurrence in nature. Examples include guppies (*Poecilia reticulate*) in high-predation versus low-predation environments (Crispo *et al.*, 2006; Labonne & Hendry, 2010), threespine stickleback (*Gasterosteus aculeatus*) in lake versus stream habitats (Berner *et al.*, 2009; Moore & Hendry, 2009) and insect host races on (sometimes) abutting host plant patches (Nosil *et al.*, 2002).

Dispersal of individuals from a given environment (all of which make it to the other environment) occurs as a fixed proportion (d) of the population size (N) in the originating environment. That is, each population contributes (on average) Nd randomly chosen individuals to the other population. This dispersal is random with respect to phenotype, although phenotype-biased dispersal (Edelaar *et al.*, 2008) would be useful to

consider in future work. In addition, individuals do not have a particular habitat preference, although this could be considered implicit in the sense that dispersal occurs with a limited probability. We nevertheless do not allow the evolution of habitat preference and therefore the evolution of dispersal rate. However, the proportion of foreign individuals entering a given environment will change with population size in that environment. For example, the same number of migrants from a given source population will become a smaller proportion of the recipient population as that recipient population grows following colonization (Hendry, 2004).

Individuals

The individuals are diploid hermaphrodites (monoecious). They have different traits that are each controlled by L different unlinked additive loci with three possible alleles at each locus ($\{-1,0,1\}$). At reproduction, each offspring receives, at each locus, one allele from each of the two parents. Individuals have an ecological trait x', a male signalling trait (m), a female target preference (f), a strength and direction of female preference (c), and a level of plasticity (r). f and c are only expressed when the individual acts as a female, and m is only expressed when the individual acts as male. All traits are genetically unlinked and are scaled to be between extreme possible values of zero and one. Each trait value z is function of the alleles (z_i) coding for that trait as given by Eqn 1.

$$z = \frac{L + \sum_{i=1}^{2L} z_i}{2L} \tag{1}$$

Note that although the number of allelic states per locus is small, this is typical of other theoretical models (Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007; Duenez-Guzman *et al.*, 2009; Bridle *et al.*, 2010; Thibert-Plante & Hendry, 2010). Moreover, because the traits are polygenic, this leads to a near-continuous distribution of phenotypic states, although other models, such as an infinite alleles model, could have been considered. This sort of genetic architecture (multiple loci each with modest effect) appears to be a common feature of adaptation (Hill, 2010; Michel *et al.*, 2010). In addition to these phenotypic traits, each individual has two alleles at each of 16 unlinked neutral loci that behave like microsatellite markers.

Life cycle

The life cycle of individuals is as follows: birth, development, density-dependent viability selection, mating and death. Random dispersal between environments occurs either just before (as in De Jong & Behera, 2010) or after development. As plasticity might have different effects if development occurs before or after dispersal, we look at the two scenarios independently.

Development and plasticity

In the absence of plasticity ($r = 0$), individuals develop their ecological trait x as coded in their genotype x'. Equation 2 describes how the environment modifies the ecological trait (x),

$$x = \begin{cases} x', & \text{if no plasticity : } r\alpha_r = 0 \\ x' + sign\,(\theta' - x') * r * \alpha_r, & \text{if } |\theta' - x'| \text{ exceeds the maximum plasticity: } |\theta' - x'| > r\alpha_r \\ x' + (\theta' - x') = \theta', & \text{if } \theta' \text{ is within the range of possible plasticity values: } |\theta' - x'| \leq r\alpha_r \end{cases} \tag{2}$$

where $sign()$ is the function that returns the sign of a number, x' is the genotypic value for the ecological trait, x is the phenotype of the ecological trait, α_r controls the limit of plasticity and θ' is the 'perceived optimum' by an individual drawn from a normal distribution with mean θ and standard deviation of θ_{err}. We kept this error small because we are interested in the effect of plasticity on ecological speciation and not on the role of developmental noise. Thus, we include imperfection in adaptive plasticity by assuming that individuals vary in their ability to perceive correctly the true optimum phenotype. That is, even if all individuals have the developmental ability to reach the new optimum, some will fall short or overshoot as a result of their limited perceptual ability (θ'). Note that with this scheme, it is possible for a single individual with an intermediate genotype ($x' = 0.5$) and large plasticity (r) to be adapted to both environments. In our model, we decided not to allow the evolution of maladaptive plasticity ($r < 0$) as it is unlikely in the context of adaptive divergence.

Viability selection

The viability of an individual is a function of its ecological trait, the environment and the population density. The fitness of an individual (ω') is a function of the distance to the optimal phenotype in that environment (θ) and the strength of selection around that optimum (σ_s):

$$\omega' = \exp\left[-\frac{(x-\theta)^2}{2\sigma_s^2}\right], \tag{3}$$

where ω' represents fitness before a cost of plasticity is added. As σ_s decreases, the strength of stabilizing selection around the optimum increases and the fitness valley between the peaks get deeper. With a cost to plasticity (C_r), fitness becomes:

$$\omega = \omega'(1 - C_r|r|). \tag{4}$$

Thus, the fitness cost of an individual expressing plasticity will increase with the magnitude of plasticity that is actually expressed. Finally, density dependence is

included by specifying the probability that an individual survives to the reproduction stage (v) according to the Beverton–Holt model (Kot, 2001):

$$v = \frac{K}{K + N(b-1)}, \tag{5}$$

where b is the average number of offspring produced by a female, K is the carrying capacity adjusted by individual fitness ($K = K_0 w$) and K_0 is the maximum carrying capacity (Kot, 2001; Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007).

Mating preference

Individuals who survive past the viability selection stage can mate. Every individual is chosen once as a female and will produce on average b offspring drawn from a Poisson distribution. Every surviving individual is also a potential father, but there is no self-fertilization. Potential fathers have a probability Ψ of being chosen by a given female with a given target preference (f) and a given preference direction and strength (c) for the male signalling trait (m) [modified from Bolnick (2004, 2006); Doebeli (2005) by Gavrilets *et al.* (2007)]:

$$\Psi(m,f,c) = \begin{cases} \exp\left[-(2c-1)^2 \frac{(f-m)^2}{2\sigma_a^2}\right], & \text{if } c \geq 0.5 \\ \exp\left[-(2c-1)^2 \frac{(f-(1-m))^2}{2\sigma_a^2}\right], & \text{if } c < 0.5. \end{cases} \tag{6}$$

At $c = 0.5$, every surviving male has the same probability of being chosen, and mating is therefore random. At $c > 0.5$, positive assortative mating is present with respect to the male signalling trait (m) and the female target preference trait (f). At $c < 0.5$, negative assortative mating is present. We also consider a magic trait model (Dieckmann & Doebeli, 1999; Gavrilets, 2004) in which we replace m and f with the ecological trait x. The parameter σ_a controls the width of the mating probability distribution as a function of the male trait, and as females become choosier, σ_a decreases. We only consider cases where there is no direct cost to signalling and choosiness.

Neutral loci

The neutral loci act like microsatellites, with high mutation rates (10^{-3}) (Dallas, 1992; Weber & Wong, 1993; Brinkmann *et al.*, 1998; Drake *et al.*, 1998) that change the number of repeats in a sequence (Valdes *et al.*, 1993; Di Rienzo *et al.*, 1994). Mutations are stepwise and consist of an increase or decrease in the number of repeat units (Kimura & Ohta, 1975). A mutation that occurs at the boundary of the allelic range changes the allele value to the closest other possible value (−1 where the allele is at the maximum of 15 repeats and +1 when the allele is at the minimum of one repeat). The neutral loci are unlinked to each other and unlinked to selected loci, equivalent to a recombination rate of 0.5 between all loci. We chose free recombination because that is the desired property of markers in most population genetic studies and because it is also the situation where neutral markers might have the most difficulty inferring progress towards ecological speciation (Charlesworth *et al.*, 1997; Thibert-Plante & Hendry, 2010).

Initial conditions

Initially, all of the loci controlling the ecological trait have a value of −1, thus $x = 0$ for all individuals (Eqn. 1). The same initial conditions apply to the loci controlling the plasticity trait r. The male signalling trait (m), the female target preference (f), and the preference and direction trait (c) loci are all set at 0, and so $m = f = c = 0.5$ for all individuals. All of these loci have the same mutation probability (10^{-5}) (Drake *et al.*, 1998; Gavrilets & Vose, 2005, 2007; Gavrilets *et al.*, 2007). A mutation changes the allele by a value of plus one or minus one with the same probability, relative to the current allele. Mutations at the boundary of the allele range are designed to keep the value within the possible range, by changing the sign of the mutation if necessary. Another genetic architecture was tested on a subset of the simulations (only two alleles with back and forth mutation starting with no genetic variance), and it did not qualitatively alter the conclusions. In general, the details of allelic coding matter little in a polygenic model such as ours.

The two environments are spatially discrete and both have the same maximum carrying capacity (K_0), but they have different optima: $\theta_1 = 0.2$ and $\theta_2 = 0.8$. Initially, only one environment is seeded by 10 individuals (θ_1) and the other remains empty until a dispersal event takes place (as in Gavrilets *et al.*, 2007).

Parameters

Table 1 lists the parameter space explored. All combinations were simulated except those that were unnecessary, such as the costs of plasticity when plasticity is absent. All simulations were run for 20000 generations with 10 replicates for each parameter combination. The values of the parameters were chosen to be comparable to those simulated in previous models (Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007). The specific time frame was chosen because many studies of ecological speciation do not examine longer time frames, such as those of post-glacial divergence (Schluter, 2000; Rundle & Nosil, 2005). Also, this time frame excludes transient dynamics, and little change occurs over the last generations. We do not expect that our results would qualitatively change if we ran the simulations longer. Note that to estimate directly the effect of selection on an adapted migrant in a way that is comparable to Gavrilets *et al.* (2007), we need

Table 1 Parameter space explored.

Parameter	Symbol (if any)	Values
Natural selection	σ_s	{0.24, 0.30}
Sexual selection	σ_a	{0.05, 0.1}
Maximum carrying capacity	K_0	{512, 2048, 4096}
Number of loci	L	{4, 8}
Cost of plasticity	C_r	{0, 0.1}
Error plasticity	θ_{err}	{0.05}
Maximum Plasticity	α_r	{0, 0.3, 0.6}
Average number of offspring	b	{4}
Time of dispersal		{before, after} plasticity
Magic trait		{yes, no}
Dispersal rate (%)	d	{0.01, 0.1, 0.2}

to divide our σ_s by 0.6 – because of the above-mentioned differences between studies in the position of the adaptive peaks. The strength of selection can be placed in the context of empirical studies by reference of reciprocal transplant experiments that estimate the total strength of selection (difference in fitness) between resident and foreign individuals. Based on a recent review of this literature, we chose as our 'weak selection' value $\sigma_s = 0.3$, which corresponds $\left(2\left(1 - \exp\left(\frac{-0.3^2}{2\sigma_a}\right)\right)\Big/\left(1 + \exp\left(\frac{-0.3^2}{2\sigma_a}\right)\right)\right)$ to a measure of local adaptation of 0.49 as calculated by Hereford (2009), when we compare a specialist ($x = \theta$) to a generalist ($x = 0.5$). This value is about the average of local adaptation found by Hereford (2009). Our 'strong selection' value ($\sigma_s = 0.24$) was chosen to correspond to a level of local adaptation of 0.74. In the simulations, the number of loci per trait was the same (4 or 8) for all traits in that simulation.

Data tracking

Genetic measures and statistical tests were performed after reproduction but before dispersal. During the simulations, we tracked the means and standard deviations of all traits (x', x, m, f, c, r). We also tracked these parameters in the migrants and first-generation hybrids. A migrant is defined as a first-generation immigrant into one environment from the other. A hybrid is the offspring of a cross between a migrant and a nonmigrant. A resident is an individual that is neither a hybrid nor a migrant. These definitions are approximations, because, for instance, a cross between two migrants will be considered a resident. However, the probability of such events is so low that we can safely ignore them, especially in the presence of divergent natural selection. Viability and the 'contribution' to the next generation (number of offspring produced by an average individual that survives to reproduce) is tracked for the residents, migrants and hybrids. Average F_{ST} between the two populations across the 16 neutral markers is calculated each generation (after reproduction) following Weir (1996).

Results

Statistical analyses of each response variable involved a general linear model with fixed main factors and two-way interactions. Higher-level interactions were not considered here because they explained almost none of the variation and because we were mainly interested in main effects (see Introduction). In the following sections, we explicitly mention the effects that were most relevant to our hypotheses (see Introduction), as well as those that explained the most variation in a particular analysis. Statistical analyses of all main effects and two-way interactions are shown in Tables S1–S10 in Supporting Information. The relative importance of these different factors can be roughly judged by reference to estimates of variance explained. Because of the artificially controlled number of replicates in individual-based modelling, the level of statistical significance needs to be considered with caution (Grimm & Railsback, 2005).

Plasticity evolution

We considered plasticity to have evolved in a given simulation if the mean plasticity (r) was substantially greater than zero, here defined as $\bar{r} - SD(r) > 0$, where SD is the standard deviation across individuals. In general, plasticity evolved less often when (1) the number of loci (L) controlling plasticity (r) was large, (2) dispersal (d) was high, (3) dispersal occurred after rather than before plasticity, (4) selection (σ_s) was strong, (5) maximum plasticity (α_r) was low and (6) plasticity was costly ($C_r > 0$). This last effect was the strongest (Table S1 in Supporting Information); of the simulations where plasticity did not evolve (723 of 11520), all but 91 occurred when plasticity was costly. A number of two-way interactions among these main effects were also evident (Table S1 in Supporting Information). Although all of the above effects were statistically significant, the variance explained (r^2) was low because plasticity evolved nearly all of the time.

When plasticity did evolve, its magnitude (r) was most dependent on the maximum plasticity possible (α_r) (Table S2 in Supporting Information). For instance, the mean values of plasticity were $\bar{r} = 0.14$ and $\bar{r} = 0.25$ for $\alpha_r = 0.3$ and $\alpha_r = 0.6$, respectively. In the second strongest effect, plasticity was lower when the carrying capacity (K_0) was lower. Also, a higher cost of plasticity (C_r) reduced the magnitude of plasticity (r) that evolved ($\bar{r} = 0.22$ without cost and $\bar{r} = 0.18$ with cost). Finally, plasticity was greater when dispersal occurred before plasticity than when it occurred after plasticity ($\bar{r} = 0.22$ and $\bar{r} = 0.18$). The variance explained by the above effects was quite high, ranging from 4% to 28% (Table S2 in Supporting Information). Although a number of two-way interactions were evident among these main effects, none explained more than 2% of the variance.

Colonization

We considered colonization of the second environment ($\theta_2 = 0.8$) to have successfully occurred when the number of offspring produced was > 60% of the carrying capacity (K_0) of that environment and this level was maintained until the end of the simulation. This threshold was high enough to eliminate cases where only immigrants were present in a given environment, while also being low enough not to exclude established populations held below carrying capacity by migration load. Time to colonization was then the number of generations from the beginning of the simulation until the first occurrence of the above criterion.

Successful colonization occurred 40% of the time without plasticity ($\alpha_r = 0$) and 87% of the time with plasticity. A particularly important main effect was that colonization was more likely when the maximum plasticity was higher (Table S3 in Supporting Information). Dispersal rate, selection strength and carrying capacity were also important, including some interesting interactions among these variables (Table S3 in Supporting Information). At a low dispersal rate ($d = 0.01$), colonization was not strongly influenced by selection (it took

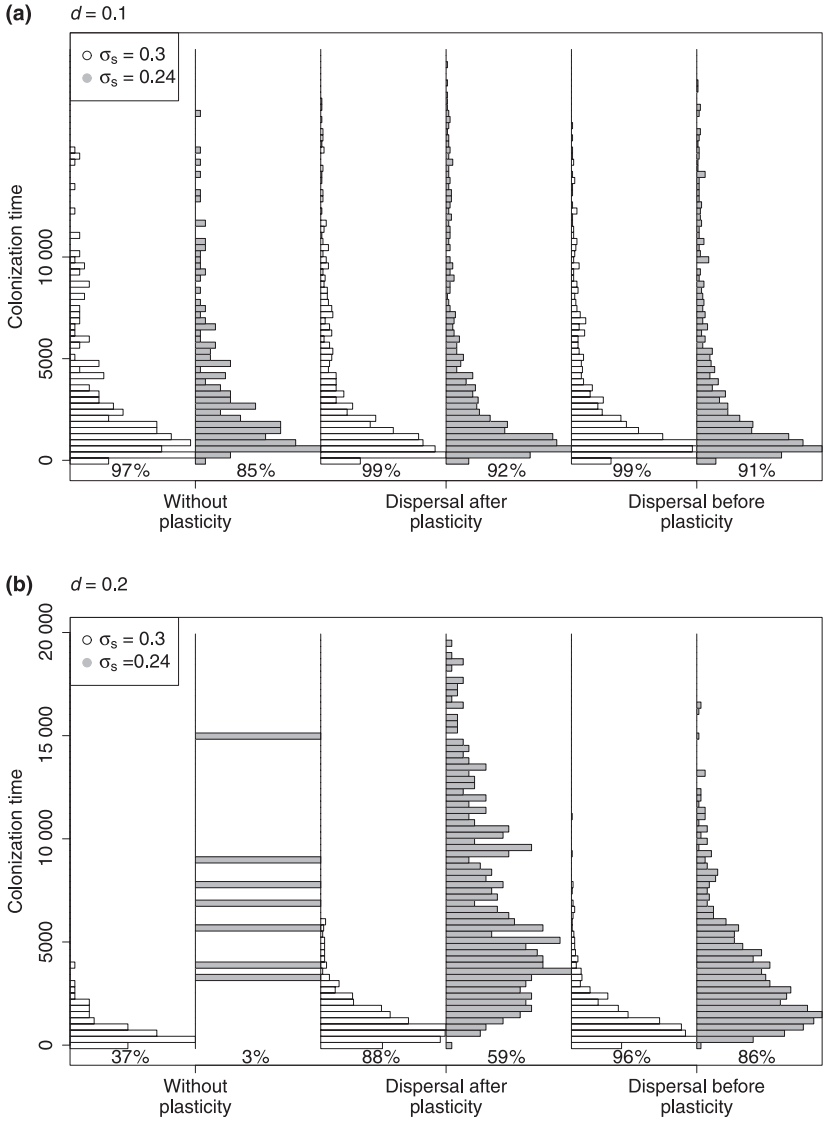

Fig. 1 Time required to colonize the second environment ($\theta_2 = 0.8$) as a function of the plasticity scenario and the strength of selection at different dispersal rates ($d = \{0.1, 0.2\}$). The percentage shown at the bottom of each panel represents the frequency of colonization over the number of simulations.

only slightly longer under stronger selection ($\sigma_s = 0.24$)) or plasticity (Fig. 1a). At higher dispersal rates ($d \geq 0.1$), colonization was less likely and often took longer, particularly when selection was stronger ($\sigma_s = 0.24$ for $d = 0.2$, see Fig. 1b; results for $d = 0.01$ not shown), and this was because migration load was higher. That is, the few residents of the second environment were likely to mate with migrants, and migrants to mate among themselves at high dispersal rates, thus slowing down adaptation. When selection was weak ($\sigma_s = 0.3$), plasticity had little influence on colonization rate. When selection was strong ($\sigma_s = 0.24$), plasticity increased the likelihood of colonization, as well as its speed (Fig. 1b). This effect was strongest when dispersal occurred before plasticity (Tables S3 and S4 in Supporting Information).

To evaluate overall adaptation of the population (i.e. mean population fitness), we compared population size after reproduction (i.e. number of offspring produced) to carrying capacity. Another measure of adaptation could have been the distance of the mean phenotype to the optimal phenotype. We chose the first measure because it is more closely related to the mean fitness of populations (Hendry & Gonzalez, 2008). Deviations of trait values from optima might not have large fitness effects if the trait is not that closely related to fitness or when the cost of plasticity is high. Regardless, qualitative results would have been the same if we had used trait deviations from the optimum as our measure of adaptation. Dispersal rate had a large influence on overall adaptation: population sizes were often lower when dispersal rates were higher, again because of migration load owing to maladaptive gene flow (Fig. 2). Dispersal rate also showed an important interaction with plasticity (Table S5 in Supporting Information); plasticity influenced population size only when dispersal rates were high ($d > 0.1$) In this case, population size was lowest without plasticity, intermediate when dispersal occurred after plasticity and highest when dispersal occurred before plasticity. In short, plasticity often promoted local adaptation and colonisation of new environments.

Genetic and phenotypic divergence

Phentoypic divergence (x) of the ecological trait was similar with or without plasticity (Fig. 3). Genetic adaptation, however, was quite different. When plasticity was not allowed to evolve, genetic divergence in the ecological trait was high between the two environments. When plasticity was present, however, genetic divergence was much lower and this reduction was more important when dispersal occurred before plasticity. With plasticity, both scenarios lead to an intermediate genotype ($x' = 0.5$), but with dispersal after plasticity, the genotypic distributions in each environment were skewed in opposite directions (Fig. 3). Also, the distribution of phenotypes and genotypes in the two environments were not mirror images of each other, even if only the position of the optima (θ) was different, because of the initially monomorphic genotype being located closer to the optimum in the first environment (θ_1). In some cases, there was no colonization of the second environment (θ_2), only newcomers adapted to the first environment (θ_1) were present in the second environment. The reciprocal case never occurred.

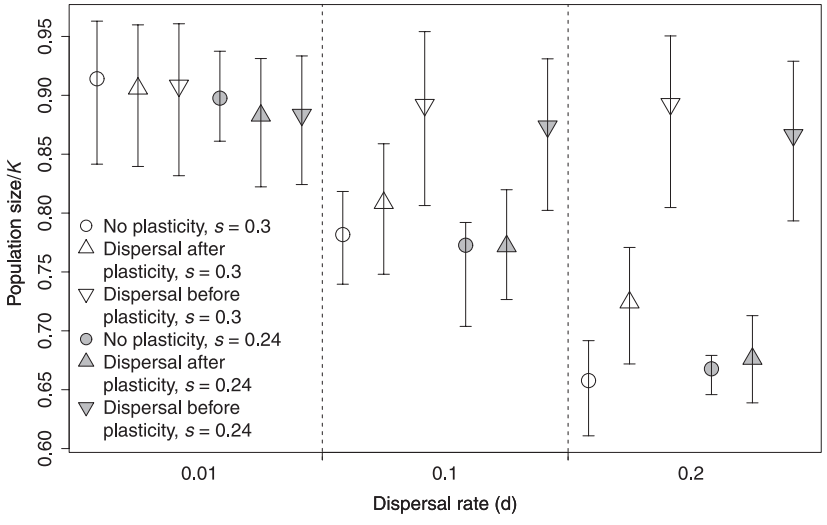

Fig. 2 Population size relative to the carrying capacity (a surrogate for mean population fitness) in the second environment ($\theta_2 = 0.8$), as a function of dispersal rate, the plasticity scenario and the strength of selection after 20000 generations. The symbols represent the median of the distribution, and the bars delimit the range where 68.2% of the values were located (equivalent to one standard deviation if the distribution was normal).

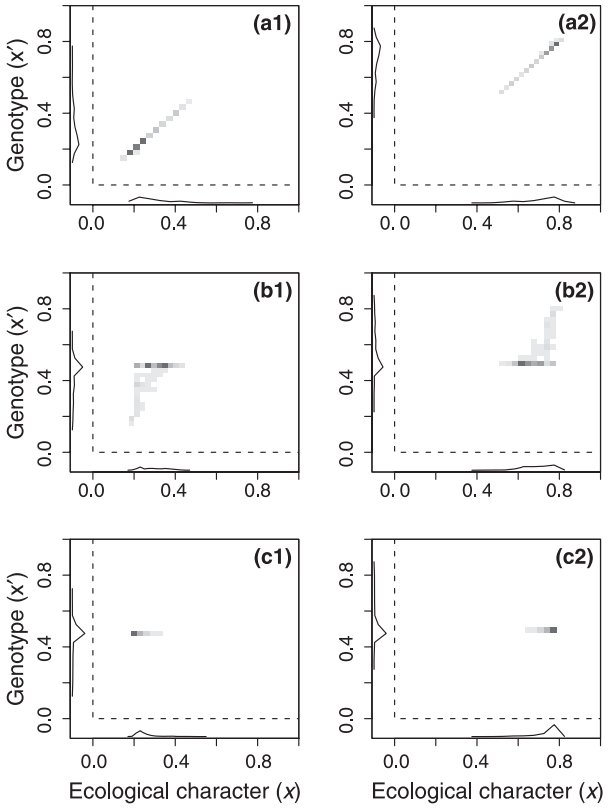

Fig. 3 Density of mean ecological trait (phenotype x and genotype x') after 20000 generations: (a) without plasticity, (b) with dispersal after plasticity and (c) with dispersal before plasticity, in the first environment ($\theta_1 = 0.2$): (a1, b1, c1) and the second environment ($\theta_2 = 0.8$): (a2, b2, c2). Darker areas represent higher density. Under the dash line is the distribution of phenotypes along the x-axis and genotypes along the y-axis.

Natural selection against migrants

Plasticity and its timing had by far the largest effect on natural selection against migrants (Table S6 in Supporting Information). Without plasticity, the average survival of migrants was always lower than the average survival of residents (Fig. 4). When dispersal occurred before plasticity, survival did not differ appreciably between migrants and residents. When dispersal occurred after plasticity, migrants had the lowest survival relative to residents. In short, plasticity somewhat increased selection against migrants between ecological environments when dispersal occurred after plasticity and greatly decreased it when dispersal occurred before plasticity.

Sexual selection against migrants

Overall, none of the factors or interactions explained more than 1% of the variation in sexual selection against migrants (Table S7 in Supporting Information). That is, when sexual preference evolved (i.e. $\bar{c} \pm SD(c)$ did not overlap with 0.5), surviving migrants and residents then produced approximately the same average number of offspring (Fig. 5). Some variation around this average, however, was seen in magic trait models (i.e. sexual selection against immigrants was not evident in any other situation). In this case, a number of simulations showed sexual selection against immigrants when plasticity was absent or when dispersal occurred after (but not before) plasticity. Interestingly, sexual selection also sometimes favoured migrants because of the drifting direction (whether females prefer males of the same or different trait values) of preference (c) owing to the lack of selection pressure on that trait early in the simulation (Fig. 6). We provide additional explanation in the Discussion.

Natural and sexual selection against hybrids

The survival of hybrids was lower than that of residents when plasticity was absent or when dispersal occurred after plasticity (Fig. 4). The reason for this was that the residents in these cases were better adapted (through genetic differences in the first case and through plasticity combined with higher survival as a result of the smaller population size, Eqn. 5, in the second case) than were hybrids. No survival differences were observed when dispersal occurred before plasticity, because hybrids could plastically adjust to the local conditions. Not surprisingly then, natural selection against hybrids was also weaker when the maximum plasticity was higher and the dispersal rate was higher (Table S8 in Supporting Information).

As in the case of migrants, much less of the variation for hybrids could be explained by sexual selection than by natural selection (Tables S8 and S9 in Supporting Information). Specifically, the average offspring production of hybrids (i.e. sexual selection against hybrids) was not different from that of residents with or without plasticity (Table S9 in Supporting Information and Fig. 5). Some variation, however, was present around this average. In particular, hybrids had a mating disadvantage in magic trait models because of the implicit prebuilt linkage between the ecological trait (x) and the female target preference (f). That is, residents automatically disfavoured hybrids because they were phenotypically different.

Unlinked neutral markers

As expected, divergence in unlinked neutral markers was mostly influenced by dispersal rate (Table S10 in Supporting Information); increasing dispersal reduced divergence (Fig. 7). Also as expected, divergence was influenced by carrying capacity: decreasing K_0 increased divergence because of increased drift. Plasticity did not have an appreciable influence on genetic divergence when dispersal occurred after plasticity. Neutral genetic

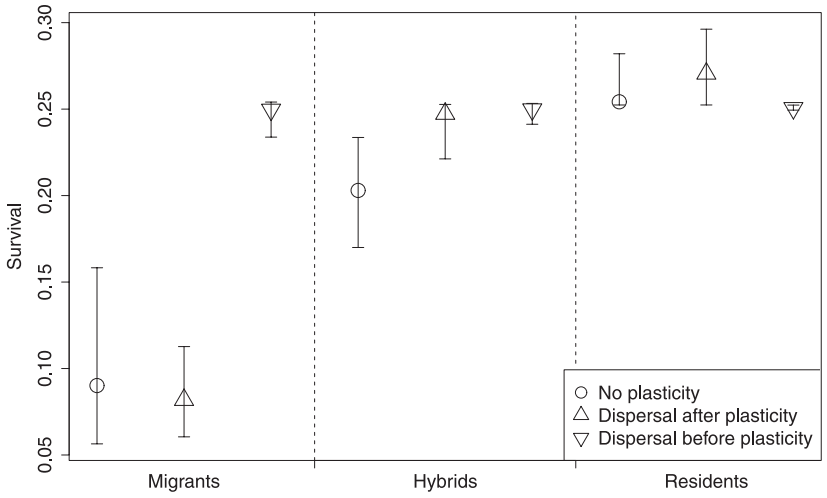

Fig. 4 Survival probability in the second environment ($\theta_2 = 0.8$) of immigrants, hybrids and residents under different plasticity scenarios after 20 000 generations. The symbols represent the median of the distribution, and the bars delimit the range where 68.2% of the values were located (equivalent to one standard deviation if the distribution was normal).

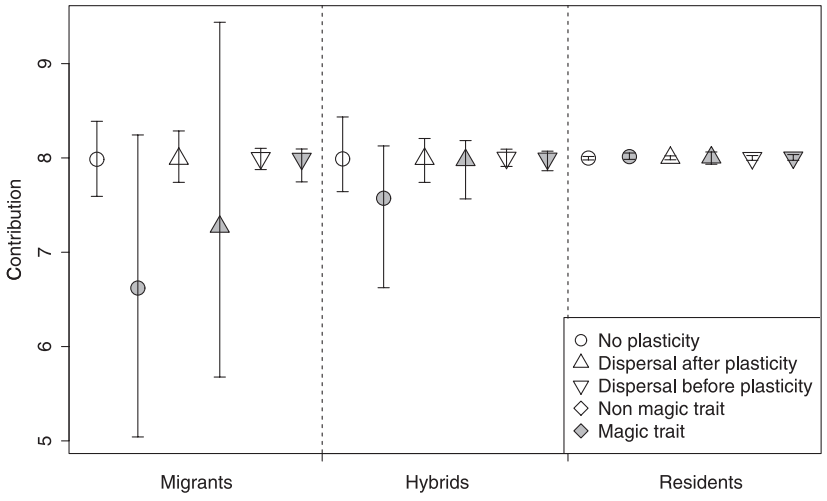

Fig. 5 Contribution (average number of offspring per parent) in the second environment ($\theta_2 = 0.8$) of migrants, hybrids and residents for different plasticity scenarios after 20 000 generations. Only simulations where female preference evolved are shown. The symbols represent the median of the distribution, and the bars delimit the range where 68.2% of the values are located (equivalent to one standard deviation if the distribution was normal); the grey symbols are used to differentiate magic from nonmagic traits.

divergence, however, was lower when plasticity occurred before dispersal, because the potential barriers to gene flow were inefficient when the ecological traits of the migrants and hybrids were indistinguishable from those of the residents.

Discussion

Plasticity is expected to evolve when environments fluctuate in time (Gabriel, 2005; Stomp *et al.*, 2008; Svanbäck *et al.*, 2009) or space (Via & Lande, 1985; Zhivotovsky *et al.*, 1996; Alpert & Simms, 2002; Lind & Johansson, 2007). We modelled the latter situation based on dispersal between two ecologically different environments. Plasticity evolved in nearly all of our simulations, confirming once again the evolutionary advantage of plasticity under these conditions. The rare occurrences where plasticity did not evolve were characterized by strong selection, high costs of plasticity, low dispersal rate and few loci controlling the nonplastic component of the trait. These are the same conditions expected to favour genetic divergence [strong selection, fewer loci (Gourbiere, 2004; Gavrilets *et al.*, 2007)] and disfavour plasticity [high costs (van Tienderen, 1997) and low dispersal (Sultan & Spencer,

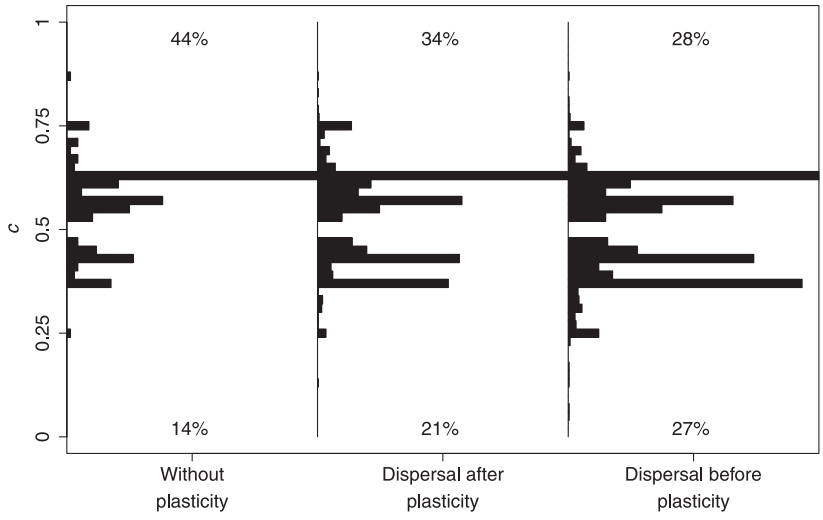

Fig. 6 The distributions of female preference trait (c) after 20000 generations as a function of different plasticity scenarios for magic traits only. We filtered out simulations where preference did not evolve ($\bar{c} \pm SD(c)$ overlap with 0.5). The numbers at the bottom of the panels are the percentage of simulations where negative assortative (i.e. disassortative) mating ($c < 0.5$) evolved. The numbers at the top of the panels are the percentage of simulations where positive assortative mating ($c > 0.5$) evolved.

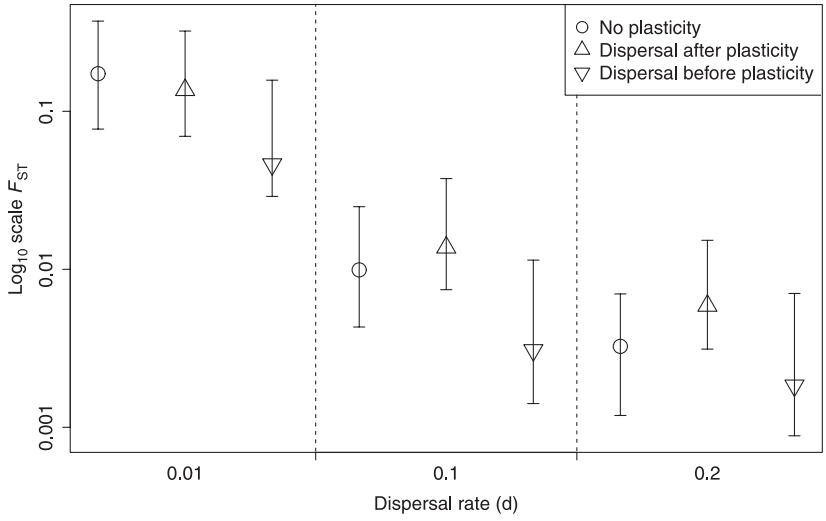

Fig. 7 F_{ST} as a function of dispersal rate and plasticity after 20 000 generations. Logarithmic scale (base ten) is used to better visualize values across different orders of magnitude. The symbols represent the median of the distribution, and the bars delimit the range where 68.2% of the values were located (equivalent to one standard deviation if the distribution was normal).

2002)]. The rationale behind the effect of the number of loci is that as the number of loci decreases, the allelic effects increase, leading to stronger selection on each loci, and thus promoting divergence at those loci (Gavrilets *et al.*, 2007). Overall, we find that plasticity in an ecologically important trait reduces genetic divergence in the non-plastic component of that trait. In short, the evolution of plasticity in our model was consistent with previous work, allowing us to turn our attention to how plasticity influences ecological speciation.

Colonization and population size

Ecological speciation is promoted by the colonization of highly divergent environments and the subsequent establishment of self-sustaining populations in each environment (Schluter, 2000; Rundle & Nosil, 2005). But herein lies the rub: highly divergent environments are also expected to be more difficult to colonize because the initial migrants are less well adapted for the new conditions. Accordingly, other modelling studies, as well

as ours, have found that colonization success is lower when the differences between ancestral and novel environments are greater (Holt & Gomulkiewicz, 1997; Gomulkiewicz *et al.*, 1999; Thibert-Plante & Hendry, 2009). One route to the partial alleviation of this problem might be when adaptive plasticity allows migrants to better match new environments, thus facilitating colonization and promoting positive population growth (Baldwin, 1896; Price *et al.*, 2003; Ghalambor *et al.*, 2007; Crispo, 2007, 2008). Our results confirm these expectations in that plasticity increased the speed of colonization of new environments, as well as the magnitude of environmental differences (here the depth of the fitness valley) that could be bridged (Fig. 1).

Once new environments are colonized, adaptation should often increase the local population size towards some theoretical maximum (Hendry, 2004; Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007; Thibert-Plante & Hendry, 2009). One exception occurs when ongoing dispersal between environments prevents strong adaptation and thereby imposes a migration load that keeps population sizes low (Kirkpatrick & Barton, 1997; Kawecki & Holt, 2002; Bridle *et al.*, 2010). Again, plasticity might partially alleviate this problem because individuals can adjust their phenotypes towards locally adaptive states and thereby not suffer the genetically expected fitness decrement. Our study also confirmed this expectation in that plasticity increased population sizes when dispersal rates were high enough to cause migration load. It is important, however, to remember that plasticity is not a panacea, because of its limits and costs (DeWitt, 1998). For instance, we confirmed that increasing costs of plasticity reduced its expected benefits for both colonization and final population size (results not shown).

Our results for spatial environmental heterogeneity can be contrasted with the expectation of temporal variation (Lande, 2009; Crispo *et al.*, 2010). In particular, the benefits of plasticity should be transitory, following an abrupt shift to a new environmental state. The reason is that plasticity can help individuals respond to the initial change but, if the environment then stabilizes, inherent costs to plasticity lead to its evolutionary reduction (i.e. canalization, Lande, 2009). In our simulations, however, plasticity remained high following colonization because ongoing dispersal maintained environmental heterogeneity experienced by any given metapopulation through time (see also Via & Lande, 1985). The same retention of plasticity is expected in the temporal context if the environment continues to fluctuate across generations (Gavrilets & Scheiner, 1993; De Jong, 1995).

Natural selection against migrants

A number of studies have argued that natural selection against migrants will be one of the most important and effective barriers to gene flow early in the course of ecological speciation (Via *et al.*, 2000; Hendry, 2004; Nosil *et al.*, 2005; Thibert-Plante & Hendry, 2009). One reason is that this barrier will act early in the life cycle, and so later-acting reproductive barriers can only make incremental reductions in gene flow. Another reason is that adaptive divergence implies by definition that individuals moving between different environments will be maladapted and therefore have lower fitness. In this sense, adaptive divergence can be considered the same thing as selection against migrants (Via *et al.*, 2000), and the many reciprocal transplant studies providing evidence for local adaptation (Schluter, 2000; Hereford, 2009) thus also provide evidence for selection against migrants. All of this could change,however, if plasticity alters the phenotype of migrants.

Our simulations confirmed that natural selection against migrants is likely to be one of the most important barriers during the early stages of ecological speciation (Fig. 4). Note, however, that we did not here consider the evolution of habitat preference, which could act even earlier in the life cycle and could therefore be even more important (Gavrilets & Vose, 2005; Gavrilets *et al.*, 2007). Our simulations revealed that plasticity can greatly reduce selection against migrants when dispersal occurs before plasticity (Fig. 4). On the other hand, plasticity can further reduce the fitness of migrants when dispersal occurs after plasticity. The reason is that plasticity here pushes phenotypes in the 'wrong' direction relative to the environment to which individuals then migrate. Based on this last observation, we suggest that plasticity could initiate reductions in gene flow before any genetic divergence takes place, a point to which we will later return.

Sexual selection against migrants

In most of our simulations, females did not evolve a strong preference for the locally adaptive male type, a result similar to that obtained by Gavrilets & Vose (2009). One reason might be that (as described earlier) natural selection is a very efficient filter against migrants when environments are quite different. In this case, so few migrants survive to reproduce that direct and indirect selection for females to mate with resident males is quite weak. As a result, sexual selection against migrants was only a minor contributor to ecological speciation in our model (Fig. 5). In contrast, when natural selection against migrants is not quite so strong, sexual selection against migrants can then make an important contribution to ecological speciation (Thibert-Plante & Hendry, 2009).

An important nuance to the above generalization emerges from a consideration of the variation among simulations when plasticity was absent or when dispersal occurred after plasticity (Fig. 5). Under these conditions, sexual selection sometimes influenced the mating success of migrants relative to residents. This variation was the result of differences among replicates in the magic trait

model only, because in this situation, assortative mating evolves most easily (Gavrilets, 2005). This occurs, of course, because the ecological trait was perfectly linked to male signalling and female target preference (i.e. they were all the same trait).

In the magic trait simulations where assortative mating evolved, it was usually positive (leading to sexual selection against migrants) but was also sometimes negative (resident females sometimes preferred migrant males) (Fig. 6). Similar results were obtained by Gavrilets & Vose (2009). One reason for this seemingly counter-intuitive result is that, in absence of costs to female preference, drift in the direction of preference (c) can lead to arbitrary mating patterns if natural selection is sufficient to largely prevent hybridization and therefore eliminate selection on mate choice. In these situations, however, so few migrant individuals survive to reproduce that the finding of disassortative mating is largely irrelevant to general inferences about ecological speciation.

The above results were modified to some extent by plasticity. When dispersal occurred after plasticity, the even stronger selection against migrants increased the effects of drift and therefore the frequency of negative, relative to positive, assortative mating. When dispersal occurred before plasticity, assortative mating with respect to phenotype (but not genotype) was higher because plasticity increased phenotypic similarity of individuals from the same environment. However, this also increased the frequency of disassortative mating with respect to site of origin (i.e. residents versus migrants). In short, sexual selection against migrants was only present in magic trait scenarios without plasticity or when dispersal occurred after plasticity, and even then only sometimes.

Selection against hybrids

It is generally expected that natural and sexual selection against hybrids often will be an important contributor to ecological speciation (Schluter, 2000; Rundle & Nosil, 2005). This selection is expected, at least early in ecological speciation, to have a primarily ecological basis resulting from maladaptation of the phenotypically intermediate hybrids to either parental environment (Rundle & Whitlock, 2001). Theoretical models have supported this intuition (Kruuk *et al.*, 1999; Servedio, 2004; Thibert-Plante & Hendry, 2009), and empirical studies have confirmed its presence in nature (Schluter, 1995; Vamosi & Schluter, 1999; Via *et al.*, 2000; Rundle, 2002; Gow *et al.*, 2007). When plasticity was absent, our model paralleled these results in finding that natural selection against hybrids was about half as strong as natural selection against migrants (Fig. 4). This was expected (Rundle & Whitlock, 2001) because additive gene action in our model dictated that hybrids were phenotypically intermediate between migrants and residents. Sexual selection against hybrids, however, was largely absent for the reasons discussed earlier for migrants. That is, natural selection against migrants was so efficient in removing selection for assortative mate choice. Also, for the reasons discussed earlier for migrants, sexual selection for or against hybrids sometimes arose in the magic trait models.

Plasticity, however, largely eliminated any selection against hybrids (Fig. 4), regardless of the timing of plasticity relative to dispersal. This was because most hybrids developed in the environment where natural selection occurred, and so adaptive plasticity eliminated the genetic disadvantage that hybrids would have otherwise suffered. These results highlight the critical importance of studying plasticity of adaptive traits for hybrids during the course of ecological speciation.

Neutral markers

As in many models, we found that the rate of dispersal was the primary determinant of divergence in unlinked neutral markers (Fig. 7). Nested within this predominant influence of dispersal rate was an additional influence of selection and plasticity. The influence of selection was to sometimes reduce neutral gene flow between populations (results not shown here, but see Thibert-Plante & Hendry, 2009, 2010; Labonne & Hendry, 2010). The influence of plasticity depended on when it occurred. Relative to in the absence of plasticity, neutral genetic divergence was lower (gene flow was higher) in the case of dispersal before plasticity but not in the case of dispersal after plasticity (Fig. 7). These results suggest that the role of adaptive divergence between populations in different environments in reducing gene flow at neutral genetic markers, already shown to be weak and inconsistent under most conditions (Thibert-Plante & Hendry, 2009, 2010; Labonne & Hendry, 2010), is even further weakened when migrants and hybrids can plastically adjust their phenotypes to suit local conditions. Plasticity thus further diminishes the utility of using such markers for inferring progress towards ecological speciation. Of course, the situation is different when one considers neutral loci physically linked to selected loci (Thibert-Plante & Hendry, 2010).

Integration

Several studies have called for an increasing consideration of plasticity in studies of ecological speciation (Crispo, 2008; Svanbäck *et al.*, 2009; Pfennig & McGee, 2010; Pfennig *et al.*, 2010). We agree, having found that several potentially important ecologically driven reproductive barriers can be influenced by phenotypic plasticity, as well as the particulars of its expression and evolution. A remaining question is what happens when integrating all of these influences to infer the effects of plasticity on overall reproductive isolation. The first

important point here is that adaptive plasticity is always conducive to colonization of highly divergent environments, and so aids ecological speciation in that sense. The situation is more complicated, however, when considering populations after they have successfully colonized new environments. To consider this situation, we calculated the overall fitness of migrants as the proportion of migrant genes in the recipient population two generations after a migration event (Fig. S1 in Supporting Information). In this more inclusive measure of reproductive isolation, nearly all effects were driven by natural selection against migrants. As was explained earlier, this correspondence arose because in our model, selection against migrants was the first to act in the life cycle and it was very powerful. This left little room for later-acting barriers. Overall, we can conclude that plasticity can strongly reduce progress towards ecological speciation when dispersal occurs before plasticity, and it can modestly increase progress towards ecological speciation when dispersal occurs after plasticity.

We wish to close with a discussion of the last point noted previously: plasticity can sometimes increase reproductive barriers. In particular, adaptive plasticity that occurs before dispersal can reduce the fitness of migrants even without any genetic divergence between populations. In this case, reproductive isolation owing to divergent environments can actually commence before any adaptive genetic divergence. This might be particularly likely in the case of two effects not considered here: imprinting on local habitats (thereby leading to habitat isolation) or imprinting on local individuals (thereby leading to assortative mate choice). In both cases, the very act of developing in a given environment confers reproductive barriers between groups in different environments. This inverts the causal pathway assumed in most studies of ecological speciation where adaptive divergence must be present to cause reproductive isolation (Räsänen & Hendry, 2008; Crispo, 2008). Moreover, this plasticity can then aid adaptive genetic divergence in cases where dispersal rates would otherwise be too high. That is, adaptive plasticity can cause initial reductions in gene flow, which can then allow adaptive divergence, which can then further reduce gene flow. In short, phenotypic plasticity might sometimes be an important catalyst in the progress towards ecological speciation.

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Supporting information

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

Figure S1 The proportion of migrant genes over two generations when dispersal is low.
Table S1 Analysis of variance Table for the occurence of plasticity.
Table S2 Analysis of variance Table the for magnitude of plasticity (r).
Table S3 Analysis of variance Table for the occurence of colonization.
Table S4 Analysis of variance Table for colonization time.
Table S5 Analysis of variance Table for overall adaptation (population size over carrying capacity).
Table S6 Analysis of variance Table for natural selection (viability) of migrants.
Table S7 Analysis of variance Table occurence for sexual selection (contribution) of migrants.
Table S8 Analysis of variance Table for natural selection (viability) of hybrids.
Table S9 Analysis of variance Table occurence for sexual selection (contribution) of hybrids.
Table S10 Analysis of variance Table occurence for genetic divergence (F_{ST}) at unlinked neutral markers.

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