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LETTER

The spatial scale of local adaptation in a stochastic environment

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Abstract

The distribution of phenotypes in space will be a compromise between adaptive plasticity and local adaptation increasing the fit of phenotypes to local conditions and gene flow reducing that fit. Theoretical models on the evolution of quantitative characters on spatially explicit landscapes have only considered scenarios where optimum trait values change as deterministic functions of space. Here, these models are extended to include stochastic spatially autocorrelated aspects to the environment, and consequently the optimal phenotype. Under these conditions, the regression of phenotype on the environmental variable becomes steeper as the spatial scale on which populations are sampled becomes larger. Under certain deterministic models – such as linear clines – the regression is constant. The way in which the regression changes with spatial scale is informative about the degree of phenotypic plasticity, the relative scale of effective gene flow and the environmental dependency of selection. Connections to temporal models are discussed.

Keywords

Gene flow, local adaptation, phenotypic plasticity, quantitative genetics, stochastic environments.

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INTRODUCTION

The optimal phenotype is likely to vary in space because of changes in both the biotic and abiotic environment. To some degree populations can track these optima by individuals responding plastically to cues that predict what the optimal phenotype should be (Via & Lande 1985) and/or through selection increasing the frequency of alleles that confer a local advantage (Kawecki & Ebert 2004). These processes give rise to a better fit between phenotype and environment but are limited by intrinsic costs (van Tienderen 1991) and imperfections (de Jong 1999) to plasticity and/or gene flow between populations with different optima (Lenormand 2002). The discrepancy between the optimal and observed distribution of phenotypes caused by gene flow has two main components: the first is from gene flow shifting the population mean from the optimum resulting in local directional selection, and the second is caused by gene flow increasing the genetic variance resulting in higher genetic variance load from stabilising selection (Barton 2001).

When dispersal between populations is equally likely regardless of their position in space (the island model), the degree to which population means deviate from local optima is determined by the ratio of migration to the strength of stabilising selection around the local optimum (Bulmer 1971), similar to results from single-locus population genetic models (Wright 1931; Moran 1962). For populations living on continuous landscapes, but where the environment changes discretely, models predict that population means should deviate from the optimum in the transition zone between the two environments before reaching their optimal values (Slatkin 1978). The spatial scale over which population means are intermediate between the two selective optima can be characterised by the cline-width (Endler 1977) which is determined by the relative strengths of migration and stabilising selection, as in the island model and also single-locus models (Haldane 1948). In contrast, models that explore the effect of a linearly changing environment find that population means track the optima perfectly (Felsenstein 1977; Slatkin 1978) and the cline in phenotype provides no information about the relative strength of selection and migration, unlike in single-locus models (Slatkin 1973).

The absence of local directional selection in a linearly changing environment is a consequence of assuming symmetric dispersal: the number of immigrants with trait values below the local optimum of a focal population exactly balances the number of immigrants with trait values greater than the local optimum. When this assumption is relaxed, areas that receive more net immigration tend to show greater deviations from the local optima. Non-symmetric dispersal can occur actively because of directional dispersal (Slatkin 1978) or as a passive consequence of non-uniform population densities; net movement of gametes from high to low-density areas occurs even when individuals disperse randomly (Pease *et al.* 1989). When population density declines from the centre of a species range, this results in a cline in phenotype that is shallower than the change in the optimum, and peripheral populations experience directional selection (Haldane 1956; Garcia-Ramos & Kirkpatrick 1997).

Here, this body of theory is extended so that changes in the environment are not fully deterministic (such as with latitude) but also have a stochastic spatially autocorrelated component (see also Engen & Sæther 2016). Such a component is typical of many important biotic and abiotic factors (Legendre 1993). It is found that the joint spatial distribution of the mean phenotype and driving environmental variable can be partitioned into two parts: a deterministic part determined by the optimal trait-environment relationship as in Slatkin (1978) and a

stochastic part determined by the relative spatial scales of environmental fluctuations and gene flow. In contrast to linear deterministic environments with symmetrical migration, stochasticity introduces local directional selection. It is also shown that the joint distribution can be summarised by how the regression of phenotype on the environmental variable changes as the spatial scale of sampling changes. Aspects of this relationship (intercept, asymptote and initial rate of change) are informative about the magnitude of plasticity, the optimal trait value and the relative strength of stabilising selection and migration. This work connects directly with similar work on temporal fluctuations in selection (Hansen *et al.* 2008; Michel *et al.* 2014; Tufto 2015) and suggests that scaling temporal fluctuations by a species generation time and scaling spatial fluctuations by a species dispersal distance puts them on a common scale by which they can be compared.

METHODS

Below, a spatial evolutionary model is developed for which the joint distribution of a trait and a driving environmental variable in space can be solved for. Only the key equations are presented in the main text and the full derivations can be found in the Supporting Information. Mean phenotype at location x at time t is of the form

$$\bar{z}(x,t) = \bar{a}(x,t) + b\epsilon(x,t). \quad (1)$$

where $\bar{a}$ is the mean breeding value and b is the plastic response to the driving environmental variable, ϵ. In what follows ϵ is assumed not to fluctuate in time, but the likely consequences of relaxing this assumption are discussed in the discussion. The variable x is a position on the real line in which the organisms live. Generations are assumed to be discrete, and all quantities, including the plastic response, are assumed to be measured prior to selection at the start of each generation, effectively at the zygote stage.

Following Slatkin (1978) the model has three components. *Selection:* the mean breeding value in population x within-generation t shifts because of selection causing zygotes to produce a differential number of gametes. *Migration:* gametes then migrate into population x which may cause a further within-generation shift in mean breeding value. *Reproduction:* gametes in population x then unite at random to form the zygotes of generation $t + 1$. The order of events is therefore trait determination, including plasticity, followed by selection, gamete dispersal and then fertilisation.

Population density is assumed to be constant in space (i.e. selection is soft) and the probability that a gamete disperses from location x' to x is assumed to depend only on the distance between the two populations ($|x - x'|$). $M(x - x')$ is used to denote this probability distribution. A Gaussian fitness function is assumed where the optimum (θ) varies in space but the width (ω) remains constant (Haldane 1954). The within population additive genetic variance (G_a) and phenotypic variance (P) are also assumed to remain constant in space such that strength of stabilising selection is constant: $\gamma = (\omega^2 + P^2)^{-1}$ (Lande 1976).

Assuming equilibrium has been reached we can drop the notation with time (i.e. $\bar{a}(x, t + 1) = \bar{a}(x, t)$ for all locations) and (Slatkin, 1978, eqn 13) demonstrated that

$$\bar{a}(x) = (1 - G_a\gamma)(M * \bar{a})(x) + G_a\gamma(M * \theta)(x) \quad (2)$$

where * stands for convolution. When plasticity exists, eqn 2 becomes

$$\bar{a}(x) = (1 - G_a\gamma)(M * \bar{a})(x) + G_a\gamma(M * \psi)(x) \quad (3)$$

where $\psi(x) = \theta(x) - b\epsilon(x)$ is the short fall between the optimum and the plastic response and gives the optimal breeding value rather than the optimal phenotype (Michel *et al.* 2014).

If dispersal events are assumed to follow a Laplace distribution (i.e. dispersal distances have an exponential distribution), then the Fourier transform of eqn 3 allows for a simple solution

$$\mathcal{F}\{\bar{a}(x)\} = \frac{\lambda_s^2}{\lambda_s^2 + \xi^2}\mathcal{F}\{\psi(x)\} \quad (4)$$

where $\lambda_s = \lambda\sqrt{G_a\gamma}$ and λ is the rate parameter of the Laplace distribution. λ^{-1} is equal to the mean dispersal distance and so λ_s increases as effective gene flow diminishes; dispersal distances decrease and/or selection around the optimum increases. The dispersal displacements have standard deviation $\sqrt{2}\lambda^{-1}$ and $\sqrt{2}\lambda_s^{-1}$ is referred to as the characteristic length by Slatkin (1978). The simplicity of eqn 4 arises because the Fourier transform is a representation of the spatial function in the frequency domain (ξ is the unitary ordinary frequency) and convolution in the spatial domain corresponds to ordinary multiplication in the frequency domain.

A fixed linear relationship between the optimal phenotype and the environmental variable (B) is assumed and different spatial distributions of the environmental variable considered. Under this scenario $\psi(x) = (B - b)\epsilon(x)$ and so both selection and plasticity are assumed to depend on the same environmental variable, ϵ. This assumption is relaxed in the Supporting Information and discussed in the discussion. To place the results from stochastic environments in context, two models with deterministic environments presented in Slatkin (1978) are analysed, one with a linear change in the environment and one with a discrete change. The results are not presented (see SI), since in the absence of plasticity the Fourier transform method gives identical results to those in Slatkin (1978). However, they do appear as dashed lines in Fig. 1. In a linear environment the optimum is tracked perfectly (see also Felsenstein 1977) and does not depend on the amount of plasticity. In a discrete environment, mean phenotype is a sigmoid function of x reaching the two optima some distance from the transition zone. The rate at which the optima are reached, the inverse of cline-width, depends on λ_s. With plasticity, a discontinuity occurs at the transition zone because of the different plastic responses in the two environments.

To analyse models with stochastic environments, spatial changes in the environmental variable are split into a deterministic part and a stochastic part,

$$\epsilon(x) = \epsilon_\mu(x) + \epsilon_e(x) \quad (5)$$

where $\epsilon_\mu(x)$ is a deterministic function, such as a linear or discrete change, and $\epsilon_e(x)$ is some zero-mean stationary random field. Under these conditions it can be shown that the expected mean breeding values of populations (where the

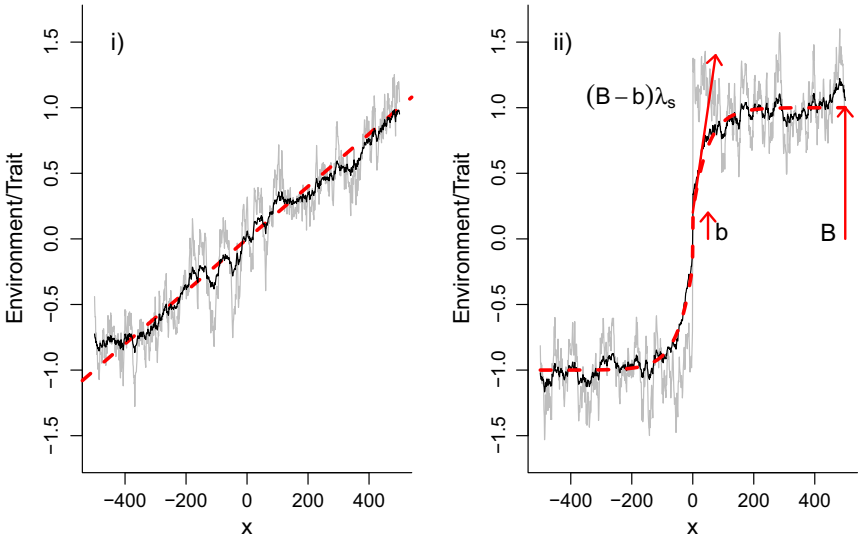

Figure 1 Simulated environmental variable (grey) and equilibrium mean phenotype (black) when the environment has a deterministic component ((i) linear change, (ii) discrete change) and a stochastic component. $B = 1$ and so the grey trace also depicts the optimal phenotype. The dashed red line gives the equilibrium mean phenotype in the absence of stochasticity, which is also the expected mean phenotype with stochasticity where the expectation is taken over realisations of the spatial process. This figure constitutes one such realisation. The arrows in (ii) represent aspects of the deterministic/expected phenotypic cline in a discrete environment in terms of the biological parameters: intercept ($b = 0.2$), asymptote (B) and maximum rate of change [$(B - b)\lambda_s$], the reciprocal of which is often referred to as the cline-width.

expectation is taken over the spatial process) is equivalent to the mean breeding values of populations subject to purely deterministic environments:

$$\mathcal{F}\{E[\bar{a}(x)]\} = \frac{\lambda_s^2}{\lambda_s^2 + \xi^2}\mathcal{F}\{\psi_\mu(x)\}. \quad (6)$$

To analyse the consequences of spatially stochastic changes in the environmental variable we need to consider its autocovariance function $C_{\epsilon_e}(x, x')$. This is the expected covariance between the environmental variable at site x and site x' where the covariance is taken over realisations of the spatial process. Assuming that the covariance is defined solely in terms of distance d, the cross-covariance function between the environmental variable and the de-trended trait ($\bar{z}_e(x) = \bar{z}(x) - \bar{z}_\mu(x)$) is given by

$$\mathcal{F}\{C_{\bar{z}_e,\epsilon_e}(d)\} = \left(\frac{\lambda_s^2}{\lambda_s^2 + \xi^2}\right)\mathcal{F}\{C_{\epsilon_e}(d)\}(B - b) + b\mathcal{F}\{C_{\epsilon_e}(d)\} \quad (7)$$

where the first term represents the effect of local adaptation and the second term represents the effect of plasticity.

One of the simplest stochastic processes is the Ornstein–Uhlenbeck process, which is the continuous analogue of a first-order autoregressive process. This generates an exponential autocovariance function, $C_{\epsilon_e}(d) = \sigma_\epsilon^2 e^{-d/\phi_\epsilon}$, where σ_ϵ^2 is the stationary variance, and e^{-d/ϕ_ϵ} gives the correlation in the environmental variables of populations separated by distance d (see also Tufto 2015). As the scale parameter ϕ_ϵ increases the environmental variable becomes correlated at greater distances. In Fig. 1 environmental variables are simulated at a thousand equal spaced points according to an Ornstein–Uhlenbeck process around a linear trend and around a step change. By discretising the landscape into a thousand populations, the expected phenotype in each population was obtained by solving the discrete-space equation analogue of eqn 2. The environmental variable is in grey, the mean phenotype in black and the dashed red line corresponds to the theoretical prediction for the mean phenotype in a fully deterministic environment.

The de-trended fluctuations (i.e. the fluctuations around the dashed line in Fig. 1) of the environmental variable and the phenotype are correlated, but the fluctuations in the phenotype are smaller in magnitude and tend to be smoother. We can characterise this by obtaining the modal 'phenotype-environment association' (PEA) had a pair of populations been sampled at distance d. Conceptually, this is like taking a random pair of populations d units apart and asking what the modal regression coefficient would be if their mean phenotypes were regressed on their environmental variables. Practically, the distance-based PEA could be measured by estimating the parameters of the cross-covariance function between the phenotype and the environmental variable (eqn 7) and using the methods in the SI to derive to the distance-based PEA. For an environmental variable with an exponential autocovariance function this evaluates to

$$\kappa_{\mathrm{PEA}}(d) = (B - b)\frac{\lambda_s\phi_\epsilon}{1 - \lambda_s^2\phi_\epsilon^2}\left[\frac{1 - e^{-d\lambda_s}}{1 - e^{-d\phi_\epsilon^{-1}}} - \lambda_s\phi_\epsilon\right] + b \quad (8)$$

The function $\kappa_{\mathrm{PEA}}(d)$ is plotted for a range of parameters in Fig. 2.

RESULTS

A number of interesting results emerge from this theory.

(1) Equation 6 shows that the expected population mean at a location follows the deterministic prediction (See Fig. 1). In the case of a linear change in the environmental variable this

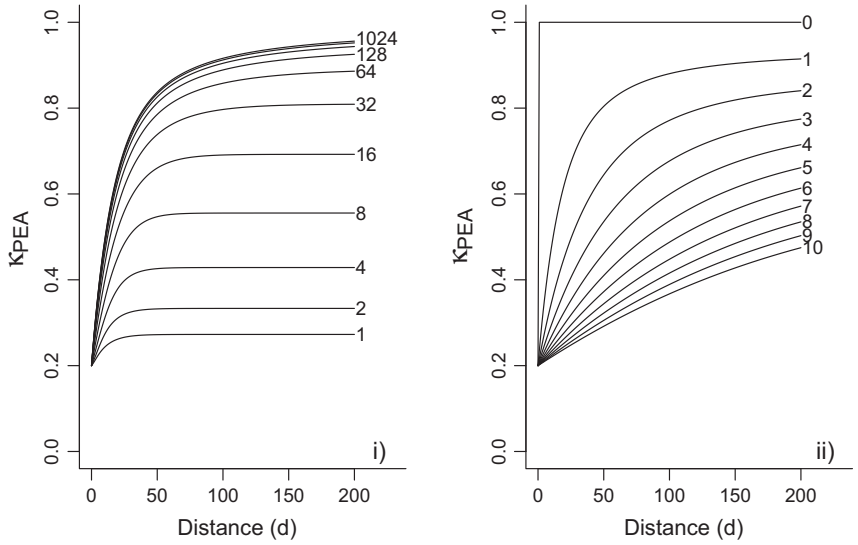

Figure 2 Regression of the de-trended trait on the environmental variable (κ_{PEA}) as a function of sampling distance d with (i) different spatial autocorrelation in the environmental variable (ϕ_ϵ) holding the mean dispersal distance at $\lambda^{-1} = 1$ or ii) different mean dispersal distances holding the spatial autocorrelation in the environmental variable at $\phi_\epsilon = 100$. In both scenarios and $B = 1$ and $b = 0.2$ $G_a = 0.2$ & $\gamma = 0.05$ (Johnson & Barton 2005).

implies that the optimal relationship between the trait and the environmental variable can be obtained from the ratio $B = \beta_{z,x}/\beta_{\epsilon,x}$ where the regression coefficients are obtained by individually regressing the trait and the environmental variable on location respectively. Equation 8 demonstrates that simply regressing the phenotype on the environmental variable will under estimate B if there is stochasticity, because the fluctuations in each are not perfectly correlated because of gene flow (See Fig. 1).

Equation 8 gives the modal PEA when assayed populations are separated by some distance. In Fig. 3, $\kappa_{PEA}(d)$ is plotted for a specific set of parameters (see figure legend) and properties of the function are characterised in terms of the biological parameters. The relationship between the shape of this function and key biological processes constitutes the main results:

(2) At distance zero eqn 8 simplifies to

$$\kappa_{PEA}(0) = b \tag{9}$$

and is equal to the plastic response. This logic was used by Phillimore *et al*. (2010) to estimate b by regressing the phenotype (spawning dates of the Common frog, *Rana temporaria*) on the environmental variable (temperature) at the same site over multiple years, under the assumption that micro-evolution at that site had not built up a large association. When temporal replication does not exist at a site, eqn 8 suggests that if the environmental variable has a stochastic component, then in a spatially explicit model b can be estimated by estimating the function $\kappa_{PEA}(d)$ and finding the intercept.

(3) At large distances, eqn 8 simplifies to

$$\kappa_{PEA}(\infty) = (B - b)\frac{\lambda_s\phi_\epsilon}{1 + \lambda_s\phi_\epsilon} + b \tag{10}$$

and tends to the optimal slope as $\lambda_s\phi_\epsilon$becomes large (i.e. dispersal is short with respect to the scale of environmental fluctuations).

(4) The initial (i.e. at $d = 0$) rate of change in $\kappa_{PEA}(d)$ is given by

$$\lim_{d\to 0} \kappa'_{PEA}(d) = \tfrac{1}{2}\lambda_s(\kappa_{PEA}(\infty) - \kappa_{PEA}(0)) \tag{11}$$

which demonstrates that the relative strength of selection vs. migration (λ_s) can be independently estimated, and is related to the cline-width in deterministic models.

(5) If the environment does not have a deterministic component, and so B cannot be estimated according to (1), results (2–4) demonstrate that the function $\kappa_{PEA}(d)$ provides independent information about b, λ_s and B conditional on ϕ_ϵ.

(6) In a temporally autocorrelated environment with adaptive plasticity, (Michel *et al*. 2014; eqn 6) derive the expected temporal PEA using a continuous time approximation (see eqn 4c in Tufto 2015, for discrete-time). In the notation used here, their result is

$$\kappa_{PEA} = (B - b)\frac{G_a\gamma\phi_\epsilon}{1 + G_a\gamma\phi_\epsilon} + b \tag{12}$$

where ϕ_ϵ now specifies temporal autocorrelation in the environment in units of generations. This is identical to our result at large spatial distances when the mean dispersal distance is one, except that $\sqrt{G_a\gamma}$ appears in the spatial model (eqn 10) rather than $G_a\gamma$. The square-root probably appears because 'dispersal' in time is only in one direction (forward) whereas dispersal in a linear habitat can be in two directions and so all 'sites' are connected. This result suggests that a natural way of comparing temporal and spatial fluctuations in selection is to scale them by generation time and average dispersal distance respectively.

DISCUSSION

Compared to theoretical studies on the effects of selection, migration and drift on gene frequencies (e.g. Haldane 1930; Wright 1931; Levene 1953), work on quantitative traits has a shorter history and remains less complete (Barton & Turelli

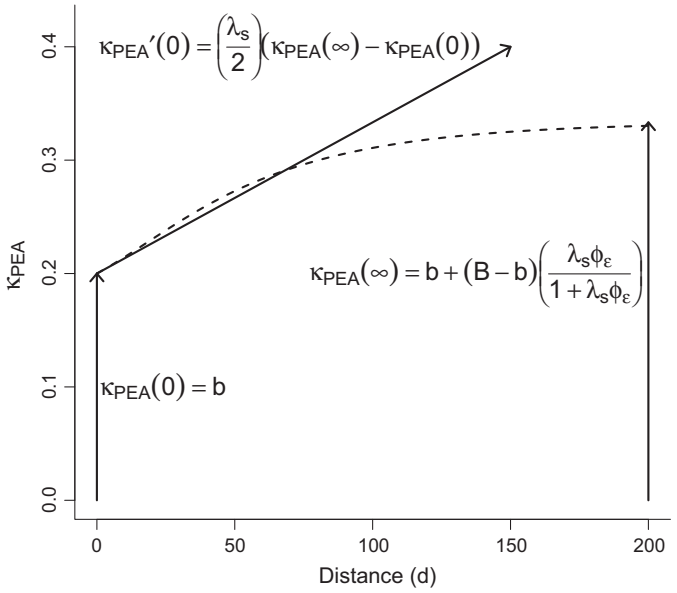

Figure 3 Regression of the de-trended trait on the de-trended environmental variable (κ_{PEA}) as a function of sampling distance d. The parameters are the same as those used to generate the simulations presented in Fig. 1. The arrows represent aspects of the function, $\kappa_{PEA}(d)$ in terms of the biological parameters: intercept [$\kappa_{PEA}(0)$], asymptote [$\kappa_{PEA}(\infty)$] and initial rate of change [$\kappa'_{PEA}(0)$].

1989; Barton 1999). Nevertheless, a substantial theoretical literature does exist exploring genetic differentiation, not only in the presence of divergent selective forces (Bulmer 1971) but with the added complexity of phenotypic plasticity (Via & Lande 1985), demographic variation (Pease *et al*. 1989), drift (Lande 1991) and realistic genetic architectures (Yeaman & Guillaume 2009). Currently, only environments that change deterministically in space have been considered, and this study adds to this body of work by considering environments that also have a stochastic component (see also Engen & Sæther 2016).

The effect of stochasticity is most clearly illustrated by contrasting it with genetic differentiation under a deterministic linear change in the environment (Felsenstein 1977; Slatkin 1978). In the deterministic case, population means track the optimum perfectly and so the optimal PEA (B) (also called the environmental sensitivity of selection; Chevin *et al*. 2010) can be directly observed as the rate at which the phenotype changes as a function of the environmental variable. Equivalently, B can also be indirectly obtained as the rate at which the phenotype changes as a function of location multiplied by the rate at which location changes as a function of the environmental variable. With stochasticity, the expected (as opposed to the observed) population means behave the same as in the deterministic case, and are therefore also informative about B. However, B cannot be measured directly from the observed PEA. This is because fluctuations in phenotypes and environmental variables around their expectations are not perfectly correlated and so weaken the relationship, as in models of temporally fluctuating selection (Michel *et al*. 2014; Chevin *et al*. 2015). However, in contrast to temporally fluctuating selection, B can be readily obtained by independently regressing phenotype on space (e.g. latitude) and the environmental variable on space and taking the ratio of the coefficients.

With a deterministic linear change in the environment, the change in phenotype with location does not depend on the relative strengths of migration and stabilising selection (Felsenstein 1977; Slatkin 1978), or the magnitude of plasticity (Via & Lande 1985). However, with stochasticity, the relative spatial scales at which phenotypes and the environment fluctuate around their expected values tells us something about these processes. This result can also be understood in terms of the PEA, which gets shallower as we sample populations at smaller distances. We can understand this result in terms of a more familiar phenomenon. A regression coefficient is defined as $\mathrm{cov}(\bar{a}, \epsilon)/\mathrm{var}(\epsilon)$. If ϵ is an imperfect measure with random error around the true values, then these errors do not contribute to the covariance between $\bar{a}$ and ϵ, but they do contribute to the variance in ϵ. The resulting slope is shallower than it would have been, had ϵ been measured without error. The Fourier transform represents spatial variation in ϵ as a weighted sum of sinusoids with different wavelengths, and this idea can be used to gain biological insight into why the PEA changes with spatial scale (Fig. 4). Fluctuations at short wavelengths (over short distances) are like noise, occurring at such small spatial scales that the population cannot locally adapt. These fluctuations contribute to $\mathrm{var}(\epsilon)$ but do not cause correlated fluctuations in breeding value and so do not contribute to $\mathrm{cov}(\bar{a}, \epsilon)$. However, populations can locally adapt to environmental fluctuations that operate at long wavelengths (over large distances), resulting in an association between breeding value $\bar{a}$ and ϵ. As we increase the scale of sampling we pick up more and more of the long-wavelength variation, whereas the short-wavelength variation remains constant. Consequently, $\mathrm{cov}(\bar{a}, \epsilon)$ decreases more quickly than $\mathrm{var}(\epsilon)$ at short distances and so the regression slope gets shallower, eventually tending to zero. In contrast, plasticity allows a direct response to fluctuations in ϵ, irrespective of their spatial scale, and therefore adds a constant to the regression of phenotype on ϵ (as opposed to the regression of breeding value on ϵ).

These features of stochastic models have analogues in both quantitative (Slatkin 1978) and population genetic (Haldane 1948) models with a deterministic step change in the environment (Figs 1b and 3). In these models, the mean breeding value (or allele frequency) is intermediate between the two optima at the transition zone, and tends to the optimal value with increasing distance. The distance over which the transition happens increases with dispersal distance (which tends to move alleles favoured on one side of the transition zone deeper into the other side) but decreases with selection (because alleles on the wrong side of the transition zone are then eliminated more quickly). The relative strength of these two processes is measured by the maximum rate of change in mean breeding value in space: the reciprocal of cline-width. In stochastic models the degree to which the association between $\bar{a}$ and ϵ is maintained is also determined by the relative strengths of dispersal and stabilising selection. Similar to deterministic step change models, it is shown that the relative strength of these two processes can be measured by the initial

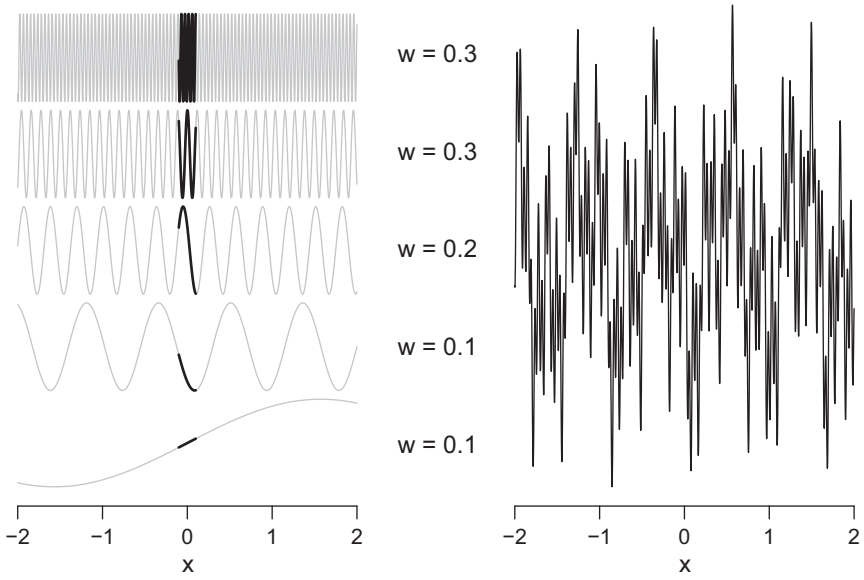

Figure 4 The five sinusoids in grey are weighted by their respective weights (w) and then added together to form the function describing how the environmental variable changes in space [$\epsilon(x)$, on the right]. Any function can be decomposed into a weighted sum of sinusoids using the Fourier transform. We can imagine populations trying to locally adapt to each sinusoid. For the low-frequency sinusoid at the bottom this is relatively easy because the environment is changing slowly compared to dispersal. For the high-frequency sinusoid at the top this is relatively hard because the environment is changing quickly compared to dispersal, and an allele experiences very different environments each generation. If we sample populations close together (those parts of the sinusoids in black) we can see that we are picking up much of the high-frequency variation but little of the low-frequency variation. Because it is not possible to adapt to the high-frequency variation, the covariance between the environment and breeding value is not built up and so the PEA is shallow and mainly due to plasticity. If we sampled populations further away we would be picking up more of the low-frequency variation, for which a covariance can build up, and so the PEA gets steeper. The rate at which the PEA changes with distance depends on the degree of plasticity, the scale of dispersal and also the scale of autocorrelation in the environment. On the frequency scale, the autocorrelation is determined by the magnitude of the different weights, with the scale of autocorrelation increasing as more weight is placed on the low-frequency sinusoids.

rate at which the between-population regression changes with spatial sampling.

With a deterministic step change in the environment, plasticity causes a concomitant step change in phenotype despite mean breeding values changing smoothly. The magnitude of this step change is equal to the plastic response. In a stochastic model the regression of breeding value on ϵ tends to zero at short distances. However, plasticity allows a direct response to fluctuations in ϵ even over small distances and therefore the regression of phenotype on ϵ (as opposed to the regression of breeding value on ϵ) does not tend to zero but tends to the plastic response. Phillimore *et al.* (2010) used a similar logic to distinguish between plasticity and local adaptation in the spawning dates of the Common frog. However, the theory presented here shows that the analyses presented in Phillimore *et al.* (2010) could be extended to yield information on the relative strength of migration and selection and the optimal response to environmental change. Although superior information would be available from transplant or common garden studies that track individuals and their fitnesses (Hereford 2009), the results presented here would allow conclusions to be drawn from population means, which for many species is all that is available.

This work also connects with recent theoretical and methodological developments for studying the evolution of traits in temporally autocorrelated environments (Hansen *et al.* 2008; Michel *et al.* 2014; Chevin *et al.* 2015; Tufto 2015). Indeed one of the central results from work on temporal variation in selection is that the observed PEA is shallower than B, particularly when temporal autocorrelation is weak (Michel *et al.* 2014). This result is similar to our result when the PEA is measured across distant populations, although spatial autocorrelation must be scaled by mean dispersal distance and temporal autocorrelation by generation time. However, our results highlights the need to take care when interpreting temporal PEA: Michel *et al.*'s (2014) result only holds when environments are assayed at sufficiently long time intervals that temporal autocorrelation is negligible (Hansen *et al.* 2008). Moreover, it suggests that a complete analysis of the phenotype and ϵ time series would provide additional information. This result is analogous to that developed in a phylogenetic context (Hansen *et al.* 2008; see Burt (1989) also) where the observed PEA (called the 'evolutionary regression') is expected to be shallower than B (called the 'optimal regression'), particularly when the species are more closely related. The explanation for this phenomenon is analogous to the one put forward in a spatial context: closely related species living in two very divergent environments have had less time to adapt than distantly related species living in the same pair of divergent environments, and so the association between phenotype and environment is therefore weaker in the former (Burt 1989). The longer history of these ideas in the phylogenetic comparative literature has led to a wide understanding of why the difference between the observed PEA and B would change with taxonomic scale, and the development of statistical procedures for making inferences from data (Hansen *et al.*

2008; Bartoszek *et al.* 2012; Hansen & Bartoszek 2012). Similar methods could be developed in a spatial context by fitting spatial covariance functions for the phenotype and the environmental variable and then deriving the distance-based PEA in the same way that eqn 8 was derived. Although a wide variety of spatial models have been developed for analysing a single response variable (i.e. a trait *or* an environmental variable), bivariate analyses that allow a flexible model for the spatial cross-covariance function are relatively new. However, such models have been applied in a different context to short time series (Sy *et al.* 1997; Jaffrezic *et al.* 2004) and moderate sized spatial datasets (Gneiting *et al.* 2010), and new approaches and software are being developed for fitting multivariate models to very large spatial data sets (Lindgren *et al.* 2011; Schlather *et al.* 2015). These new frontiers in spatial data analysis should allow ecologists and evolutionary biologists to extract information from spatial patterns that has previously been ignored.

LIMITATIONS

The model makes several simplifying assumptions: population densities are (1) uniform and (2) large enough that genetic drift can be ignored; (3) plasticity and selection are driven by the same environmental variable and this variable has been correctly identified; (4) at a location the environmental variable is constant over time; (5) plasticity and (6) the additive genetic variance are fixed quantities that remain constant in space; (7) dispersal events are exponentially distributed in (8) a linear and infinite habitat. The likely consequences of these assumptions are addressed below.

(1) With random dispersal, gene flow tends to be from high- to low-density areas causing greater local maladaptation in low-density areas. When density declines from the centre of a species range, this causes peripheral populations to lie further off their local optimum resulting in an observed PEA that is shallower than B (Garcia-Ramos & Kirkpatrick 1997). It seems likely that in an environment with both a stochastic and deterministic part the expected population means would also follow this trend, and care would have to be taken in interpreting the ratio of the regressions of phenotype and environment on space as a measure of B. Likewise, obtaining a measure of B from the pattern of phenotype-environment fluctuations around the expectations would be compromised if fluctuations in the environmental variable also drive fluctuations in population density. Models that allow both density and trait to evolve (Pease *et al.* 1989) have shown that when the environment changes rapidly, and density regulation is weak, a feedback loop can occur (Haldane 1956; Holt & Gomulkiewicz 1997; Kirkpatrick & Barton 1997; Ronce & Kirkpatrick 2001). Such a process could generate a spatial cross-correlation between the environmental variable and population density (Polechová & Barton 2015), although it would seem that the magnitude of any feedback would be smaller than observed for deterministic changes.

(2) The effects of drift were ignored yet they are known to generate spatial autocorrelation in mean breeding value due to the fact that relatives exist closer in space with restricted dispersal (Lande 1991). However, spatial autocorrelation due to drift and random dispersal events in finite populations should be uncoupled to the environmental variable and so are unlikely to alter the conclusions of this study greatly (Engen & Sæther 2016).

(3) In their island model, Via & Lande (1985) showed that if there is no cost to plasticity, perfect plasticity evolves (i.e. $b = B$) and all populations track the local optima without genetic differentiation. Although simulation work with realistic genetic architecture challenged this view (Scheiner 1998) analytical studies of the simulation model demonstrated that deviations from perfect plasticity were in fact due to the cue for plasticity being imperfect. Then, spatial differentiation is a mixture of plastic and genetic responses (de Jong 1999; Tufto 2000). The model presented here assumes that plasticity and selection act simultaneously such that the environment of development and the environment of selection are perfectly correlated (Moran 1992). In the Supporting Information this assumption is relaxed and it is shown that a pair of PEAs, one for each environmental variable, could be constructed that would allow all relevant parameters to be assessed. Although empirically challenging this could also be extended to the case where there are multiple environmental variables driving selection and plasticity (Chevin & Lande 2015). A more difficult problem for empiricists will be to identify and measure the driving environmental variable(s) rather than an environmental variable that is merely correlated with them. In the Supporting Information, it is shown that if the correlations between and within all environmental variables decay in space at the same rate then eqn 8 would remain valid. However, the observed environmental sensitivities of selection and observed plasticities should then be thought of as effective: B and b multiplied, respectively, by the regressions of the environments of development and selection on the measured variable (Michel *et al.* 2014). When the rate of decay is different between or within environmental variables then an incorrectly identified driving variable could result in spurious inferences. However, two diagnostics are available to assess whether this might be the case. The first is that if the measured variable is the driving environmental variable, then the cross-correlation between the mean phenotype and the environmental variable should decay at a slower rate than the autocorrelation in the environmental variable. This should happen because gene flow increases the spatial scale of fluctuations in the phenotype with respect to the environmental variable. In cases where data have also been collected at multiple times at the same location then a second diagnostic would be to obtain a second direct estimate of b as in Phillimore *et al.* (2010) and see if the two estimates of plasticity differ.

(4) Using the within-site temporal PEA to measure plasticity as in Phillimore *et al.* (2010) assumes that micro-evolution has not generated an association between breeding value and the environmental variable across years. As shown in Michel *et al.* (2014), and consistent with the spatial results presented here, this will only hold when the temporal autocorrelation in the environmental variable is zero. Building temporal variation into the model presented here should be straightforward if the temporal and spatial processes are separable, and it is

envisaged that an additional temporal PEA similar to that presented in eqn 8 would emerge. This would allow the degree of plasticity and temporal adaptation to be assessed from time series even when individual-level data are not available (Chevin *et al*. 2015).

(5) The model described follows the evolution of a linear reaction norm where the intercept is allowed to evolve in space but the slope is fixed. Models that allow the slope to evolve have shown that populations experiencing more extreme environments may evolve greater plasticity (Tufto 2000) when the trait is canalised in the average environment (Lande 2009). With a deterministic linear trend in the environment this results in the evolution of greater plasticity at the margin of a species range (Chevin & Lande 2011). It is not clear what effect stochastic changes in the environment would have on the evolution of the slope, although it seems likely that they would induce spatial fluctuations in the slope as they do for the intercept. The magnitude of these fluctuations is hard to gauge, and although it seems that they would most likely be small compared to those for the intercept, further study would be required to confirm this.

(6) The genetic variance was assumed to be constant, although it is expected to be elevated in regions where the mean breeding value changes relatively quickly, such as at the boundary of two discrete environments (Nurnberger *et al*. 1995; Barton 1999). However, these effects will be mitigated in finite populations with hard selection because local population size will be reduced in such regions and the ensuing drift will act to reduce the genetic variance (Polechová & Barton 2015). Understanding the balance between these two processes in stochastic environments would require more work, although for highly polygenic traits in smoothly varying environments the expectation is that genetic variances may remain roughly constant.

(7) The Laplace distribution was used as a dispersal kernel due to its analytical properties, and other reasonable dispersal kernels could have been used. However, the deterministic results coincided with those of Slatkin (1978) who did not assume a specific form for the dispersal kernel, but used a Taylor approximation that assumed that mean breeding values change over large distances relative to dispersal. Consequently, although the details of the model are expected to change with different dispersal kernels, it is believed that the general results would still hold.

(8) An infinite habitat was assumed and the results will almost certainly breakdown at the boundary of a species range where edge effects become important. However, simulation work (see SI) suggests that these edge effects may be quite restricted and that the results would still hold throughout much of a species range. Like the majority of the theory exploring spatial evolution a one-dimensional habitat was also assumed, despite most organisms primarily living in two-dimensional habitats. Extending the Fourier analyses to two dimensions is possible, and has been used in an ecological context (e.g. Lande 2009), although little success was had in obtaining results using a multivariate Laplace dispersal kernel. A future aim is to extend these models to two dimensions and develop statistical tools that can be used to estimate the relevant parameters in a two-dimensional context.

In the most comprehensive empirical review of spatially varying selection to date (Siepielski *et al*. 2013) the authors state that the degree of spatial replication in selection studies is so low that we have little understanding of the scale of spatial variation in selection. The theory presented in this study will be useful to empiricists as it suggests that under certain conditions spatially replicated data can be used to estimate key evolutionary parameters without the need to collect individual-level fitness data from multiple populations.

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