

REVIEW AND SYNTHESIS

The evolution of species interactions across natural landscapes

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Abstract

Given the potential for rapid and microgeographical adaptation, ecologists increasingly are exploring evolutionary explanations for community patterns. Biotic selection can generate local adaptations that alter species interactions. Although some gene flow might be necessary to fuel local adaptation, higher gene flow can homogenise traits across regions and generate local maladaptation. Herein, I estimate the contributions of local biotic selection, gene flow and spatially autocorrelated biotic selection to among-population divergence in traits involved in species interactions across 75 studies. Local biotic selection explained 6.9% of inter-population trait divergence, an indirect estimate of restricted gene flow explained 0.1%, and spatially autocorrelated selection explained 9.3%. Together, biotic selection explained 16% of the variance in population trait means. Most biotic selection regimes were spatially autocorrelated. Hence, most populations receive gene flow from populations facing similar selection, which could allow for local adaptation despite moderate gene flow. Gene flow constrained adaptation in studies conducted at finer spatial scales as expected, but this effect was often confounded with spatially autocorrelated selection. Results indicate that traits involved in species interactions might often evolve across landscapes, especially when biotic selection is spatially autocorrelated. The frequent evolution of species interactions suggests that evolutionary processes might often influence community ecology.

Keywords

Community ecology, evolutionary ecology, evolving metacommunity, gene flow, landscape genetics, local adaptation, meta-analysis, metapopulation.

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INTRODUCTION

Evolution can occur in parallel with ecological dynamics (Birch 1960; Hairston *et al.* 2005) and result in novel eco-evolutionary dynamics (Pelletier *et al.* 2009; Post & Palkovacs 2009). The integrative fields of evolutionary ecology, ecological genetics and community genetics seek to discover evolutionary explanations for ecological patterns and dynamics (Orians 1962; Ford 1964; Antonovics 1992; Holt 2005; Thompson 2005; Whitham *et al.* 2006; Fussmann *et al.* 2007; Johnson & Stinchcombe 2007). In particular, recent efforts have focused on determining how local adaptation and gene flow interact to produce novel community dynamics across heterogeneous landscapes (Thompson 2005; Urban & Skelly 2006; Urban *et al.* 2008). However, our understanding of the overall degree to which species interactions evolve across natural landscapes remains limited. Herein, I quantify how antagonistic biotic selection and regional gene flow shape the adaptive differentiation of traits involved in species interactions through a meta-analysis.

Local adaptation depends, in part, on the strength of gene flow relative to antagonistic selection. Populations can adapt to local conditions when antagonistic local natural selection (s) overcomes gene flow (m) from nearby populations ($s > m$) (Wright 1969; Slatkin 1985) as long as sufficient additive genetic variation exists to fuel local adaptation. Moderate gene flow also can supply the necessary genetic variation to facilitate adaptation, especially in small, inbred populations or for coevolving partners (Holt & Gomulkiewicz 1997; Wade 2000; Greischar & Koskella 2007). However, higher gene flow ($s < m$) dilutes local gene pools and generates local maladaptation (Crespi 2000). The relative strengths of gene flow and selection not only

determine if adaptation occurs but also the degree to which gene flow displaces trait means from local optima (Hendry *et al.* 2001). Adaptive divergence also can feed back to decrease gene flow if local adaptation promotes the evolution of reproductive isolation (Rasanen & Hendry 2008). Many populations exhibit local adaptation (Hereford 2009), but still receive gene flow from neighboring populations (Morjan & Rieseberg 2004). Therefore, most populations probably occur somewhere between absolute maladaptation and optimal local adaptation (Hendry *et al.* 2001; Nosil & Crespi 2004; Bolnick & Nosil 2007; Garant *et al.* 2007; Hendry & Gonzalez 2008).

The source and spatial distribution of natural selection also determines how gene flow affects local evolutionary dynamics (Ehrlich & Raven 1969; Gillespie 1974; Garant *et al.* 2007). Genes originating from populations facing different natural selection than the recipient population can swamp adaptation; genes originating from populations facing similar natural selection can facilitate adaptation (Bulmer 1972; Gillespie 1974). Individual empirical studies as well as a growing body of theory demonstrate that both local selection heterogeneity and gene flow can shape evolutionary dynamics among interacting species to varying degrees (Thompson 1999, 2005; Nosil & Crespi 2004; Urban *et al.* 2008). However, further progress requires a more synthetic understanding of the importance of these processes in wild populations.

I estimated the relative contributions of biotic selection and gene flow to traits that mediate species interactions. Toward this end, I performed a meta-analysis on published studies that measured traits that directly affect the outcomes of species interactions across multiple natural populations (see Table S1 in Supporting Information). Previous evolutionary syntheses on population differentiation

ignored gene flow and the spatial distribution of selection as explanatory variables (Nosil *et al.* 2005; Hereford 2009) or focused on coevolutionary dynamics between hosts and parasites (Greischar & Koskella 2007; Hoeksema & Forde 2008). I conducted my analysis in four steps. First, I modelled the spatial autocorrelation of population trait means in each study as a proxy for the homogenizing influence of gene flow on genetic divergence from local trait optima (Sokal & Oden 1978; Epperson & Li 1996; Hardy & Vekemans 1999; Diniz-Filho *et al.* 2009). Second, I derived statistical models to explain the among-population divergence in trait means based on trait spatial autocorrelation and local biotic selection. Third, I partitioned the variance in trait means into contributions from selection, spatial autocorrelation and spatially autocorrelated selection, and then estimated the mean contributions across all studies. Fourth, I tested if the connectivity of study populations, in terms of species-specific estimates of gene flow relative to the mean distances among sampled populations, affected these estimated contributions to population trait divergence. Throughout, I use the term gene flow to indicate the process by which genes from another population contribute to the reproductive pool of a recipient population. I use the term population connectivity to define distances among study populations in terms of species-specific estimates of gene flow.

I predicted that local selection and regional gene flow would explain significant variation in traits involved in species interactions based on accumulated evidence for local adaptation in populations (Hereford 2009) and the maladaptive effect of migrants (Hendry *et al.* 2001; Hendry & Taylor 2004; Nosil & Crespi 2004; Moore *et al.* 2007). I predicted that spatially autocorrelated selection, characterised by clines or patches, would explain substantial trait divergence because populations can evolve traits closer to their optima if genes flow from similar, rather than dissimilar, selection regimes (Jain & Bradshaw 1966; Nosil & Crespi 2004; Urban 2007). I tested if estimated gene flow influences adaptation by evaluating the contributions of each factor along a gradient of inter-population connectivity. I expected that selection would explain more trait divergence when populations were connected by less gene flow and that traits would be more spatially autocorrelated when populations were connected by high gene flow (Wright 1946). Lastly, I predicted that spatially autocorrelated selection would explain the most trait variance at intermediate levels of population connectivity, where both gene flow and selection interact. Results suggest that local adaptation commonly occurs for traits involved in species interactions, especially in systems, where biotic selection is spatially autocorrelated. This outcome indicates the need to consider evolutionary as well as ecological mechanisms underlying community diversity patterns.

MATERIALS AND METHODS

Literature search

I searched all science journals in the Web of Science using the keywords 'adapt*', 'evol*' and 'population' for all years in the index up to 2008, when I completed the search. I refined this list of studies to those that measured interaction trait variation among eight or more natural populations and for which I could obtain the geographical location and local biotic selection regime of each population (Table S1). I defined an interaction trait as any characteristic that directly modifies a species interaction including traits such as competitive ability, predator defense, prey preference and host

resistance (hereafter 'interaction traits'). I only included studies for which trait differences were determined to be genetic by measuring trait divergence in common environments or by establishing discrete genetic polymorphisms through reproductive crosses. Most studies did not evaluate contributions from maternal effects, and so I cannot exclude their effect on common garden estimates. I geo-referenced published maps in Google Earth (Mountainview, CA) to obtain geographical coordinates. I directly contacted authors for missing data. Datasets on different species or from populations from different sub-regions and treated as separate in the original manuscript were likewise treated as separate here; including sub-region as a random effect had little effect on results (Table S2). I only included studies for which authors indicated evidence for biotic selection.

Biotic selection

I estimated the degree to which biotic selection explained trait differences among populations in a generalised linear model with population trait means as response variables and putative local selection agents as explanatory factors. I analysed studies that documented inter-population variation in traits such as pigmentation, growth, defensive structures or in fitness components such as survival or fecundity in relation to selection resulting from a species interaction (Table S1). Authors generally reported biotic selection regimes as habitat differences that produce antagonistic selection rather than as explicit estimates of selection intensity. For instance, authors reported different habitat colours when quantifying selection for crypsis or the presence or abundance of interacting species in other studies. In 89% of cases, these categorizations of selection were underlain by explicit measures of selection, including the estimation of selection differentials or gradients or direct measurements of fitness differences among selection regimes (Table S1). In six cases, biotic selection characterization was based on selection estimates performed on similar species, and in two cases the researchers relied on field observations of fitness variation among phenotypically divergent populations. Of the studies that relied on selection estimates, 85% of these estimates originated from mortality selection, 13% evaluated growth or development rate, 9% assessed fecundity selection and 3% analysed overall fitness (some studies measured multiple components such that percentages do not sum to 100%). I assigned binary variables or rank values to selection recorded in categories. I excluded coevolutionary studies when traits could not be tested independently because I could not differentiate between the agent and target of selection. I averaged percent variances explained from each trait whenever authors measured more than one trait. However, not every trait identified by authors might confer fitness benefits or evolve. Therefore, I also estimated contributions when I only included the trait in each multi-trait study that was correlated most strongly with selection differences. In the absence of other information, I assumed that the original authors had identified a legitimate source of biotic selection. Non-experimental field explorations of selection rely on correlations, rather than causation, and therefore caution is always warranted in this regard (Reznick *et al.* 2001).

Spatial autocorrelation and gene flow modelling

Few studies meeting the above criteria also measured gene flow with neutral markers. Instead, I measured the spatial autocorrelation of population trait means, after accounting for biotic selection, to estimate gene flow's effect on population trait means. Longstanding

evidence indicates that gene flow generates clines in gene frequencies, assuming finite populations and sufficient time (Wright 1943; Slatkin 1985; Lande 1991; Hardy & Vekemans 1999). Weak to moderate gene flow and neutral differentiation in finite populations generates positive spatial autocorrelation in gene frequencies and quantitative trait means (Epperson & Clegg 1986; Epperson 1995; Epperson & Li 1996; Hardy & Vekemans 1999; Manel *et al.* 2003). Statistical measures of spatial autocorrelation in gene frequencies and quantitative trait means correspond accurately with trait distributions simulated under low to moderate gene flow (Sokal & Oden 1978; Epperson & Li 1996; Hardy & Vekemans 1999; Diniz-Filho *et al.* 2009). For traits under selection, the spatial autocorrelation of trait means, after accounting for local selection, indicates how much gene flow from nearby populations explains remaining trait variance (Jain & Bradshaw 1966; Argyres & Schmitt 1991; Urban 2007). For instance, a step-function in selection along a linear spatial gradient will generate a step-function (vertical transition) in trait values without gene flow. With gene flow, the same step-function selection will generate a wider, sloping transition in trait means (Jain & Bradshaw 1966). As a result, such clines can be used to estimate migration when selection intensity and its spatial distribution are known (Slatkin 1973).

I used Moran's eigenvector maps to estimate trait spatial autocorrelation. Moran's I provides a direct analogue of Malecot's estimator of spatial correlations of gene frequencies (Malecot 1955; Epperson 2005) and describes accurately how gene flow influences neutral gene frequencies across space (Sokal & Oden 1978; Epperson & Li 1996; Hardy & Vekemans 1999; Epperson 2005). However, Moran's I only indicates the spatial genetic correlation across one distance class at a time, and these correlations cannot be entered directly into a linear model. Moran's Eigenvector Map (MEM) solves this problem by deriving explanatory orthogonal vectors that maximise Moran's I for variables across multiple spatial scales (Borcard & Legendre 2002; Dray *et al.* 2006). In doing so, MEMs capture realistically complex nonlinear spatial structures (Borcard & Legendre 2002; Dray *et al.* 2006) and accurately characterise simulated neutral allele patterns subject to gene flow better than other tested methods (Diniz-Filho *et al.* 2009). In Appendix S1, I demonstrate with a simulation study that MEMs represent changes in the spatial autocorrelation of trait means that occur when gene flow integrates local dynamics across different spatial scales.

This indirect method of gene flow estimation requires that gene flow is not so strong that it homogenises trait means across populations (less than one gene flow neighborhood radii, Appendix S1). However, none of the studies in this analysis were performed at distances much less than one gene flow neighborhood radius. Also, removing the few studies conducted at less than one gene flow neighborhood radius had little effect on variance estimates (see Results: Partitioning of variance).

I included a fixed number of three forward-selected spatial vectors because variance explained would otherwise increase with the number of populations evaluated in each study. I also examined spatial trait variance explained by latitude, longitude and their interaction using the alternative method of trend-surface analysis (Legendre & Legendre 1998) and obtained similar results as those obtained using MEMs (Table S2).

Variance partitioning

I partitioned the variance in trait means owing to biotic selection, trait spatial autocorrelation and spatially autocorrelated biotic selection.

Community ecologists apply this same approach to evaluate the contributions of environmental gradients and the homogenizing effect of migration to among-community variation in species (Borcard & Legendre 2002; Cottenie 2005; Holyoak *et al.* 2005; Legendre *et al.* 2005). In this case, I apply this method to understand contributions to differences in genetically determined trait means among populations. Although gene flow can sometimes promote local adaptation, gene flow more commonly diverts local trait means away from selective optima by lowering fitness in recipient populations (Crespi 2000; Hendry *et al.* 2001; Hendry & Taylor 2004; Nosil & Crespi 2004). Hence, this partitioning approach provides a means to evaluate contributions from selection gradients and the homogenizing effect of migration to adaptive divergence when direct gene flow estimates are not available (Urban 2007; Diniz-Filho *et al.* 2009).

For each study, I partitioned the divergence of trait means among populations (T) into that owing to local biotic selection alone (B), trait spatial autocorrelation alone (S), spatially autocorrelated biotic selection ($B:S$) and residual error (ϵ)

$$T = B + S + B : S + \epsilon. \quad (1)$$

Variances were estimated from the coefficients of determination (R^2) obtained in generalised linear models using standard methods (Legendre & Legendre 1998). I first calculated the total variance explained by the full model containing factors related to both selection and trait spatial autocorrelation. Next, I calculated the variances explained by the set of reduced models containing only variables related to selection and spatial autocorrelation and their error terms. The variance explained by the full model minus the summed variances explained by the two reduced models estimates the $B:S$ term. This shared variance term can be positive or negative depending on whether the shared fraction acts synergistically or antagonistically. By this simple partitioning model, I do not mean to imply that selection and gene flow solely determine evolution by natural selection. In particular, I lacked information on the availability of additive genetic variation needed to fuel adaptive responses in each population. The error term includes these additional factors that I could not quantify.

I interpret variance components in a manner analogous to the standard interpretations for the variance partitioning of among-community beta diversity (see Cottenie 2005). I infer that the inter-population trait variance explained by differences in selective environments among patches occurs owing to local adaptation. I attribute the variance explained by spatial autocorrelation, independent of selective environment, to gene flow from nearby populations as supported by the simulation results provided in Appendix S1. The variance explained by the $B:S$ term can reflect either the contribution of spatially autocorrelated selection to trait patterns (Urban 2007) or a spurious relationship with an unmeasured and spatially autocorrelated selection regime (Peres-Neto & Legendre 2010). I directly assessed evidence for the first explanation by regressing shared explained variances against the spatial autocorrelation of biotic selection itself. I found that spatial autocorrelation in biotic selection, measured as the Mantel correlation between Euclidean landscape distance and the squared differences between selection regimes, strongly predicted the trait variance explained by the $B:S$ term. Hereafter, I refer to this shared term as the contribution of spatially autocorrelated selection to trait divergence throughout the manuscript, but readers should recognise the potential for additional contributions to this term.

The explained variance estimated from any statistical model upwardly biases effect size (Helland 1987). Therefore, I randomly

permuted data 1000 times, calculated variances for each random data set and then subtracted the mean random explained variance from the observed explained variance to obtain a bias-free estimate. Sometimes this process resulted in negative variances, which is expected if random variation exists around near-zero numbers. I also evaluated how results changed if negative variances were set to zero in Table S2.

Meta-analysis

Meta-analytic approaches weight the contribution of each study by its expected variability to reduce the influence of less replicated studies that can be less reliable (Gurevitch & Hedges 1999; Rothstein *et al.* 2005). I calculated the signed correlation coefficient for each value and transformed it into Fisher's Z_r to calculate an effect size estimate with well-known variance properties (Lipsey & Wilson 2000). I used the inverse standard error of Z_r -transformed correlation coefficients to weight response variables. All proportional response data were arcsin-transformed to correct for non-normality. The weighted means of each variance estimate were calculated using random-effects models in S-plus v. 8.0.4 (Tibco, Somerville, MA). Mean variance models included study number nested within species because I expected that species might respond differently to selection and gene flow. Adding species as a random effect reduced the degrees of freedom by the number of species.

Potential bias

I plotted funnel plots in metafor (R, v. 2.9.1, R Development Core Team, Vienna, Austria) to explore the potential bias introduced by studies that remain unpublished because results were insignificant (the 'file drawer problem') (Rothstein *et al.* 2005). Published studies probably represent an unbiased subset of research if effect sizes follow a symmetrically narrowing cone with decreasing standard error and if data points occur just as frequently in regions with high and low statistical power. (Rothstein *et al.* 2005). Funnel plots for biotic selection and interaction variances revealed no strong bias (Figure S1).

Potential modifiers

Two distinct research areas (industrial melanism in insects and thrush predation on land snails) dominated the dataset. Therefore, I tested the sensitivity of results to removing these research areas. I also examined if generation length altered patterns. Shorter-generation species might evolve more readily because they can respond more quickly to changing selection regimes than species with longer generations (Kawecki & Ebert 2004). I included species as a random effect for the first analysis, but not the second, because these models failed to converge when species identity was included.

Population connectivity

I next examined if population connectivity affected patterns. Simulations indicate that the variance explained by trait spatial autocorrelation should decrease as populations become less connected by gene flow (Appendix S1). I measured population connectivity relative to each species' dispersal ability by collecting independent data on gene flow or dispersal for each species for which data were available (Table S1). Data were generally not available for the individual populations used in the study, meaning that I had to

assume that dispersal does not vary substantially between study populations and populations for which dispersal data were available. I used available dispersal data to parameterise a Gaussian dispersal kernel. From this kernel, I calculated the gene flow neighborhood, which estimates the radius at which panmixia can be assumed (Wright 1946). I then calculated patch connectivity as the number of Wright's (1946) gene flow neighborhood radii that span the mean minimum distance among sampled populations. Dispersal usually follows a more leptokurtic distribution than the Gaussian distribution assumed by Wright (1946), which would upwardly bias these gene flow estimates. However, limited data precluded fitting a species-specific dispersal kernel to each data set. This gene flow measure assumes that selection in unstudied intervening populations does not substantially alter the spatial pattern of selection encountered by the subset of studied populations. Generally, I expect that deviations in selection in intervening populations would be either random or would parallel existing spatial structure and would therefore add unexplained variance but not bias.

I found data on gene flow or dispersal for 92% of the species from collected studies. I located published values of gene flow neighbourhood radii for 64% of studies. In a few cases, I converted the regression slope of pairwise estimates of neutral genetic differentiation versus physical distance into a gene flow neighbourhood radius after assuming Gaussian dispersal using standard methods (Rousset 1997). For the remaining studies, I calculated gene flow neighbourhood radii from dispersal data using the appropriate one- or two-dimensional equation (Wright 1946). Dispersal need not correspond to gene flow because strong antagonistic selection and outbreeding avoidance can prevent immigrants from reproducing and contributing to the local gene pool (Nosil 2004) or because inbreeding provides a selective advantage to migrants (Ebert *et al.* 2002). These measures only provide a coarse estimate of gene flow.

I evaluated relationships with population connectivity using a mixed-effect regression with weights based on expected variance. I lacked data from most studies on additional influences on adaptation such as antagonistic selection strength, genetic variance and the constancy of the selection regime (Kawecki & Ebert 2004). These factors probably caused departures from the expected evolutionary response predicted from considerations of gene flow alone. Therefore, I also used weighted quantile regression to fit a relationship to the upper 90% of data to examine evidence for upper boundary conditions (Cade & Noon 2003). I included a 2nd-order polynomial in the model if it improved the fit substantially as indicated by a likelihood ratio test (Pinheiro & Bates 2000). I did not include species as a random effect because these more complex models failed to converge.

RESULTS

Studies

I found 75 datasets that documented interaction traits in 32 species (see Table S1). The number of populations sampled ranged from 8 to 528 with a median of 30. The most common trait type analysed was cryptic colouration in prey (72%). Most research on this trait type involved either *Cepaea* land snail colour variation across different terrestrial habitats (48%) or insect pigmentation differences across soot clines (20%). Remaining studies evaluated traits involved in predator escape (11%), competition (7%), parasite or pathogen avoidance (5%), herbivory (4%) and prey capture (3%).

Partitioning of variance

Biotic selection explained a significant weighted average of 6.9% of trait variance (Fig. 1; Mixed-effects ANOVA: $F_{1,43} = 17.1$; $P < 0.001$). Traits evolved significantly in response to biotic selection in 36% of studies based on the model results for each study. In contrast to predictions, trait spatial autocorrelation, acting as an estimate of gene flow, explained little population trait divergence overall (0.1%; $F_{1,43} = 0.3$; $P = 0.572$), but did explain significant trait divergence in 27% of the individual studies. Spatially autocorrelated selection explained the most trait divergence (9.3%; $F_{1,43} = 24.2$; $P < 0.001$) and accounted for significant trait divergence in 53% of the studies. Most interaction variances were positive (83%), even though negative terms are possible (Legendre & Legendre 1998). The variances explained by selection and spatially autocorrelated selection increased

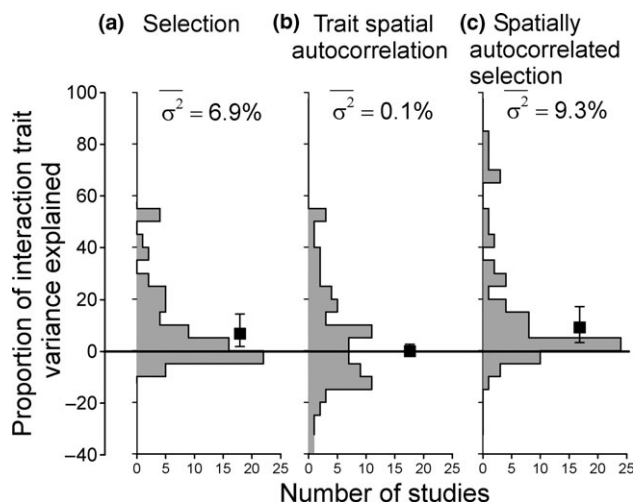


Figure 1 Histograms of the variance in traits explained by (a) local selection, (b) spatial autocorrelation and (c) spatially autocorrelated selection. Squares denote the mean variance explained, and error bars indicate the asymmetrical 95% confidence intervals. Negative variances are possible because values were corrected for positive bias, which leads some small estimates to become negative.

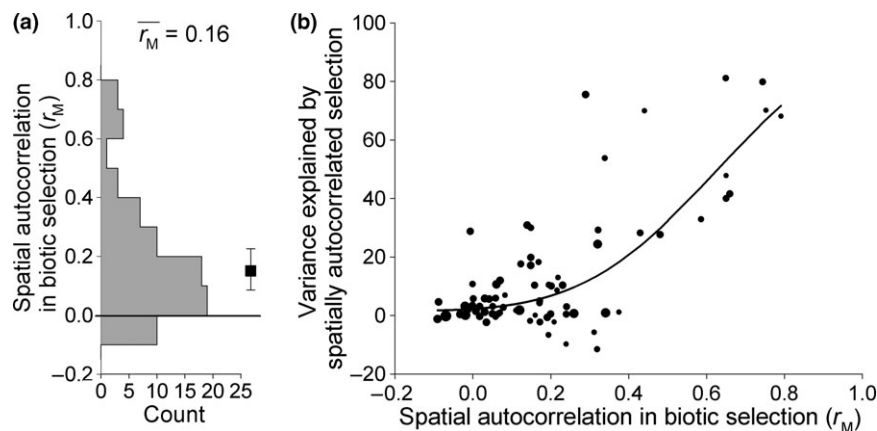


Figure 2 (a) Counts for the Mantel coefficients (r_M) for spatial autocorrelation in biotic selection regimes. Values greater than zero indicate positive spatial autocorrelation. The weighted average of these values is depicted by the square symbol and its 95% confidence intervals. (b) The variance explained by spatially autocorrelated selection increases with higher spatial autocorrelation in biotic selection regimes. The line indicates the best fit obtained with a mixed-effects polynomial regression weighted by sample size. The estimated relationship was back-transformed, which gives it additional curvature than that owing to the polynomial term alone. Symbol size indicates \log_{10} sample size.

to 9.1 and 12.7%, respectively, after restricting analyses performed on multiple traits to the single trait most correlated with selection regimes (Table S2). Eliminating negative variances owing to bias correction increased contributions from selection, trait spatial autocorrelation and autocorrelated selection to 10.5, 5.0 and 12.3, respectively (Table S2). Weighting study contributions based on sample size resulted in more conservative estimates for all measures; un-weighted estimates for selection, trait spatial autocorrelation and autocorrelated selection increased to 8.6, 4.7 and 14.1%, respectively (Table S2).

The mean Mantel autocorrelation coefficient between inter-population connectivity and selection regime dissimilarity was positive and significant at +0.16, indicating strong positive spatial autocorrelation in biotic selection (Fig. 2a; Mixed-effects regression: $F_{1,43} = 62.1$; $P < 0.001$). The spatially autocorrelated selection term explained more trait variance when biotic selection regimes were more spatially autocorrelated (Fig. 2b; Mixed-effects polynomial regression: Likelihood ratio = 54.6; $P < 0.001$). Hence, most of the trait variance explained by the $B:S$ term probably represents a response to spatially autocorrelated selection rather than responses to an unmeasured spatially autocorrelated selection regime.

Additional causes of variation

I explored several potential causes of bias and variation among studies. Funnel plots did not indicate any biases in mean variances (Figure S1), such as might occur if non-significant results remain unpublished (Rothstein *et al.* 2005). Removing the three highest weighted studies (population numbers > 200) affected estimates little ($< 0.2\%$ difference) (Table S2). Limiting analyses to populations connected by greater than one gene flow neighbourhood, one definition for a population boundary, did not alter results substantially ($< 1.5\%$ difference; Table S2). I next evaluated if two of the most common research areas (i.e., cryptic colouration in land snails and industrial melanism) influenced results. The variance explained by selection was not significantly affected by research area (Figure S2; Mixed-effects ANOVA; $F_{2,43} = 2.3$; $P = 0.110$). However, research area significantly affected the variance explained by trait spatial autocorrelation ($F_{2,43} = 3.7$; $P = 0.034$) and spatially autocorrelated selection ($F_{2,43} = 5.0$; $P = 0.011$). Traits were spatially autocorrelated in land

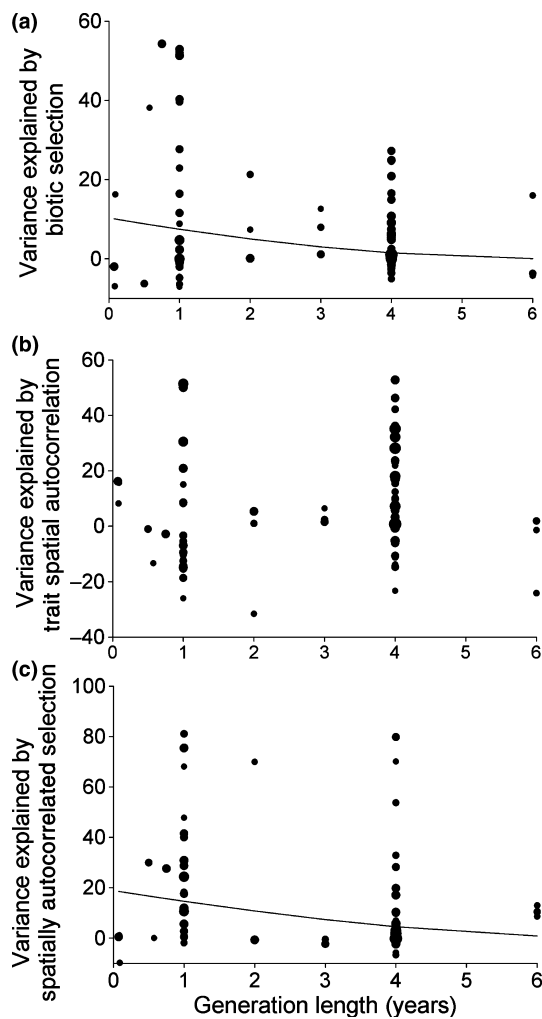


Figure 3 Regressions of the variance in traits explained by (a) biotic selection, (b) spatial autocorrelation and (c) spatially autocorrelated selection versus generation length. Lines indicate a significant ($P < 0.05$) weighted regression slope; its absence indicates a non-significant relationship. Symbol size indicates \log_{10} sample size. Observed curvilinearity results from back-transforming estimated slopes.

snails (5.8%), but not other species (0%). However, removing land snails from the analysis did not change the mean variance explained by trait spatial autocorrelation (0.1%; Table S2). Spatially autocorrelated selection explained high trait variance in industrial melanism studies (24.0%), but only 2–5% of trait variance in other studies. Removing studies on industrial melanism reduced variance estimates for both selection and spatially autocorrelated selection (Table S2). This last finding suggests caution in extrapolating results if industrial melanism studies are somehow exceptional.

Generation length

I evaluated if interspecific differences in generation lengths explained additional variance. Selection (Fig. 3; Mixed-effects regression: $F_{1,73} = 5.3$; $P = 0.025$) and spatially autocorrelated selection ($F_{1,73} = 6.1$; $P = 0.016$) explained more inter-population trait divergence in shorter-lived versus longer-lived species, suggesting that species with rapid generation times can track environmental changes better than longer-lived species.

Effects of population connectivity

The first set of analyses summarises mean contributions from local selection and spatial autocorrelation to trait patterns regardless of how distant populations were relative to their dispersal ability. If gene flow is important, then the degree to which populations are connected via gene flow should strongly influence trait evolution (Appendix S1). Biotic selection explained more trait variance when populations were farther apart in both standard and 90th-quantile regressions (Fig. 4; Mixed-effects regression: $F_{1,67} = 17.7$; $P < 0.001$; 90th quantile regression (Q_{90}): $t_{67} = 3.79$, $P < 0.001$), which is consistent with a homogenizing effect of localised gene flow. Population connectivity did not significantly explain the trait variance explained by spatial autocorrelation ($F_{1,67} = 0.5$; $P = 0.503$). However, the 90th quantile regression supported a diminishing upper limit to spatial autocorrelation's contribution when populations were farther apart (Q_{90} : $t_{67} = -2.86$, $P = 0.006$). Both the mean and 90th quantile of variance explained by spatially autocorrelated selection significantly also decreased when populations were farther apart ($F_{1,67} = 5.0$; $P = 0.046$; Q_{90} : $t_{66} = -2.52$, $P = 0.014$, 2nd-order: $t_{66} = 2.37$, $P = 0.021$), indicating that spatially autocorrelated selection occurred mostly at finer microgeographical spatial scales.

DISCUSSION

Ecologists increasingly recognise the possibility that evolution can alter ecological patterns and processes (Hairston *et al.* 2005; Holt 2005; Thompson 2005; Urban & Skelly 2006; Vellend 2006; Whitham *et al.* 2006; Fussmann *et al.* 2007; Johnson & Stinchcombe 2007; Pelletier *et al.* 2009). Evolution could play a particularly strong role in predicting species interactions and community patterns because: (1) traits are often under selection from biotic agents, (2) spatially heterogeneous species distributions often generate antagonistic biotic selection, (3) coevolution can induce greater antagonistic selection through space and time, and (4) multiple case studies indicate rapid evolution in response to species interactions (Thompson 1999, 2005; Hairston *et al.* 2005; Urban & Skelly 2006; Johnson & Stinchcombe 2007). Although case studies suggest that traits involved in species interactions can evolve (reviewed in Thompson 1999), those studies could represent rare cases and therefore overestimate the importance of evolution in community ecology. I used a meta-analysis to address this central question at the interface of community ecology and evolutionary biology in a more quantitative manner: to what degree does local adaptation versus gene flow influence traits involved in species interactions?

On the basis of this meta-analysis, I found that traits involved in species interactions commonly diverge in a manner consistent with local adaptation. An estimated 6.9% of the divergence in population trait means was attributed to local adaptive evolution alone, 0.1% was attributed to estimated gene flow alone and the largest proportion, 9.3%, was attributed to spatially autocorrelated selection. Combining these two estimates, local biotic selection explained 16% of the variance in population trait means – five times the variance explained, on average, than in biological research overall (Moller & Jennions 2002). Local adaptation explained even more variance (22.8%) relative to other aspects of biology when you compare the uncorrected R^2 from this study with other studies that generally do not apply this correction (Moller & Jennions 2002). These findings apply to traits involved in mediating interactions among species. Future work should examine if results also apply to traits under abiotic selection.

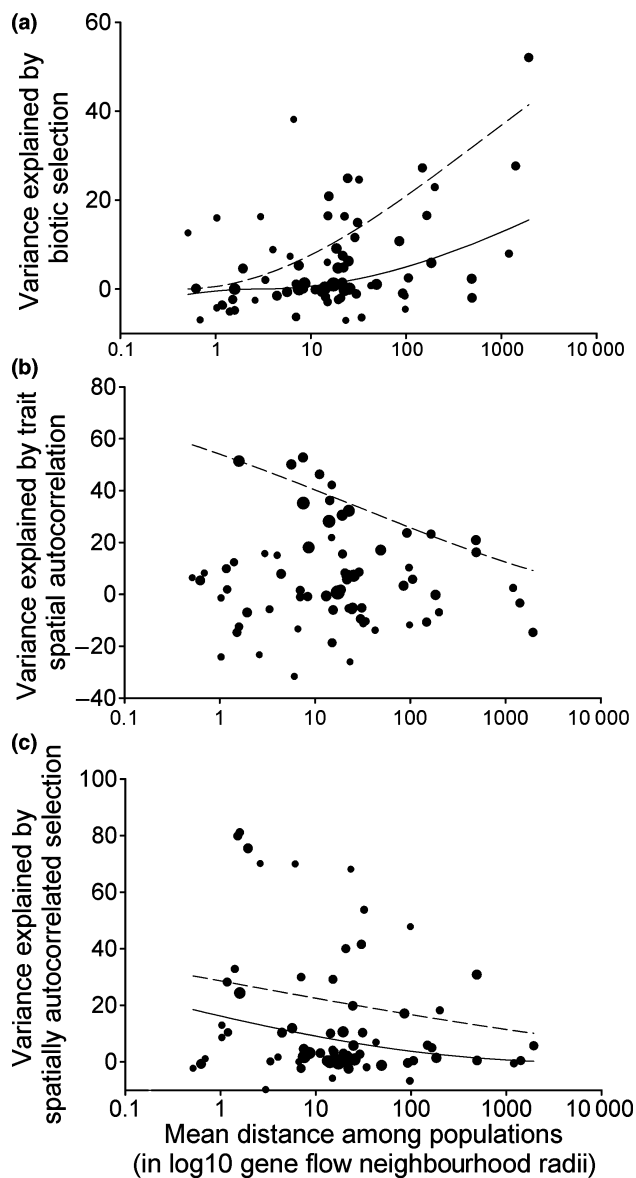


Figure 4 Regressions of the variance in interaction traits explained by (a) biotic selection, (b) spatial autocorrelation and (c) spatially autocorrelated selection versus log₁₀ inter-population connectivity measured as the mean minimum number of intervening gene flow neighborhood radii between sampled populations. The solid line indicates the estimated weighted regression slope. The dashed line indicates the estimated 90th-quantile weighted regression slope. The 90th-quantile relationship for spatially autocorrelated selection was analysed as a polynomial function. All other regressions were linear. Regression slopes were only plotted if the relationship was significant ($P < 0.05$). Symbol size indicates log₁₀ sample size.

No previous study has synthesised information on the variance in traits attributable to selection and gene flow. Three studies, however, synthesised data on relative fitness differences between local versus foreign populations. Based on transplant experiments, Hereford (2009) found that locally adapted populations performed 45% better than transplanted populations. Two studies focused on host-parasite interactions found local adaptation in parasites, but only when they migrated more than hosts, suggesting a positive influence of relative, but not absolute, gene flow (Greischar & Koskella 2007; Hoeksema & Forde). Hoeksema & Forde (2008) found no evidence that the maximum distance between allopatric populations (uncorrected for

species-specific dispersal ability) affected local adaptation, possibly because coevolution depends more on relative gene flow among interacting pairs than absolute gene flow (Hoeksema & Forde 2008). Although these studies do not provide variance estimates directly comparable to my results, they support that local adaptation can modify species interactions in nature.

Estimated gene flow explained little among-population trait divergence in the initial analysis, but had a strong effect once I controlled for population connectivity directly (Fig. 4). This initial result probably reflects both the operation of other factors that I could not account for in the model (e.g., strength of selection, levels of additive genetic variation) as well as inherent variability in the indirect approach used to estimate trait spatial autocorrelation. Biotic selection explained more among-population trait divergence when populations were less connected as expected based on the balance between selection and migration (Wright 1969; Slatkin 1985). Trait means became more spatially autocorrelated when populations were better connected, indicating a homogenizing effect of gene flow on population differentiation. Unidentified spatially autocorrelated selection provides an alternative explanation, but only if this selection becomes more spatially autocorrelated at finer spatial scales. However, unmeasured spatially autocorrelated selection probably occurs just as frequently at coarse spatial scales as would be the case for latitudinal or elevational clines in climate variables or species distributions.

Individual studies that explicitly relate gene flow to maladaptation in populations suggest an even stronger role for the homogenizing influence of gene flow on local adaptation (Hendry & Taylor 2004; Bolnick & Nosil 2007; Moore *et al.* 2007). For instance, Moore *et al.* (2007) suggested that gene flow from lake habitats caused lake outlet phenotypes to diverge by as much as 80% relative to what was expected based on natural selection alone. Thus, the indirect methods used in this article to uncover gene flow effects could underestimate gene flow's effect on evolution. More studies of this type are needed to generalise these empirical results across multiple taxa and ecosystems.

Conclusions should be tempered by the indirect methods used to estimate contributions from selection and gene flow. More direct assessments of effects of selection and gene flow will be needed to confirm the analyses presented here. In particular, I encourage future studies that integrate (1) direct estimates of selection measured in natural environments and attributed to specific causative agents, (2) common garden or genomic analyses of the relevant traits involved in species interactions collected for many (> 10) populations, and (3) information on gene flow based on neutral genetic variation, especially when this data can be used to assign migrants to other populations. Such integrated programmes of research, performed across multiple systems, would allow for a more direct assessment of the hypotheses explored here.

Adaptive divergence also can influence gene flow by imposing selection against migrants or hybrids. Therefore, a negative correlation between gene flow and adaptive divergence need not indicate unidirectional causality (Rasanen & Hendry 2008). The approach used here relies on physical distance, rather than gene flow, and thus limits this confounding of mechanisms. However, I cannot exclude the possibility that adaptive divergence constrains gene flow in some studies. This result could explain some instances of local adaptation at micro-geographical spatial scales by decreasing gene flow relative to what would be expected based on dispersal ability (e.g., Nosil *et al.* 2005).

Strong evidence for spatially autocorrelated selection suggests one reason why adaptive population differentiation can occur despite

evidence for high dispersal: if most gene flow arrives from nearby populations experiencing similar natural selection, then it will not swamp local adaptations with maladapted genes and could even facilitate adaptation by supplying additive genetic variation, creating heterosis and decreasing inbreeding depression (Ebert *et al.* 2002; Garant *et al.* 2007). Spatially autocorrelated selection explained the most variance in traits involved in species interactions. Eighty seven percent of biotic selection regimes were positively spatially autocorrelated. Spatially autocorrelated selection explained $\sim 20\%$ of trait variance at the fine scale of one gene flow neighbourhood radius, suggesting that local adaptation frequently occurs at microgeographical scales, because gene flow from nearby patches of similar natural selection does not swamp local adaptation. Gene flow from neighbouring patches with similar selection regimes can reinforce, rather than dissipate, local adaptive evolution because most gene flow originates from populations experiencing more similar selection than expected by chance (Ehrlich & Raven 1969; Gillespie 1974). For instance, a butterfly's preference for a host plant increases in a metapopulation, where this host plant dominates (Kuussaari *et al.* 2000). However, a population facing selection that differs greatly from most neighbours will become more maladapted than expected under randomly distributed selection (Kawecki & Stearns 1993; Thompson *et al.* 2002; Hendry & Gonzalez 2008).

Despite explaining more variation than the average for all meta-analyses in ecology and evolutionary biology (Moller & Jennions 2002), substantial variation remains unexplained by selection and gene flow ($\sim 84\%$). Some of this variation could be explained by random drift. Multiple additional deterministic factors besides drift, gene flow and the spatial distribution of natural selection can affect the match between population traits and local biotic selection and thereby explain remaining variance in population trait means. In particular, I did not have information on whether additive genetic variation was available in the past to allow for an evolutionary response to selection. A lack of additive genetic variation in some populations would decrease the variance explained by natural selection. I tested generation length and found that it significantly influenced the match between population traits and current selection. Greater adaptive responses occurred for shorter-generation organisms, providing support for the idea that shorter-generation organisms track temporally varying selection regimes more rapidly than longer-generation organisms (Kawecki & Ebert 2004). Most selection regimes were measured coarsely as the presence or absence of a given selective agent at a given snapshot in time. Longer-term, continuous measures of natural selection probably would have improved correlations between selection and trait means. Multiple unidentified selection agents could have affected trait means, leading to underestimated contributions from local adaptation or to spurious correlations between traits and biotic selection (Nuismer *et al.* 2010). Discerning the causative agent of evolutionary change remains a foremost challenge, especially if multiple putative selection regimes are correlated across habitats (Reznick *et al.* 2001). An important goal of ecological research should be to measure selection regimes accurately across space and time (Kingsolver *et al.* 2001; Grant & Grant 2002; Siepielski *et al.* 2009).

Other unknown factors could have affected results. Information on the strength of antagonistic selection for interaction traits would probably explain additional variation in trait divergence. Contributions from maternal effects could upwardly bias estimates of the contributions of selection to trait divergence, but these effects were seldom

estimated or controlled for. Several studies have shown that gene flow is often non-random with respect to the selection regime, and this habitat preference could reinforce patterns of local adaptation (Edelaar *et al.* 2008). However, if habitat preference generates correlations between selection and trait means, correlations should remain the same or weaken rather than strengthen when populations are sampled across coarser distances as results showed. I found a large number of studies that documented cryptic colouration as a defense against visual predators because most colour differences involved simple genetic polymorphisms that could be estimated from many populations in the field and thus eliminate the difficult work of common garden experimentation. I found only a minor effect of eliminating studies on cryptic colouration on snails. However, industrial melanism studies contributed strongly to the variance explained by spatially autocorrelated selection. Hence, the importance of spatially autocorrelated selection could have been biased by the large number of industrial melanism studies performed along regular clines in soot pollution. If adaptation to linear gradients of biotic selection occurs rarely, then this study might have overestimated contributions from spatially autocorrelated selection. However, biotic selection regimes often vary along natural gradients, such as those based on species distributed along latitudinal and altitudinal clines. Thus, biotic selection clines might often structure evolutionary dynamics in nature.

CONCLUSIONS

In summary, I estimated the proportion of trait differentiation among populations that could be explained by biotic selection, gene flow and spatially autocorrelated biotic selection. Empirical patterns were consistent with the proposition that traits that modify species interactions frequently evolve in natural communities. Most biotic selection was positively and spatially autocorrelated – that is, it occurred in clumps or along a cline. Spatially autocorrelated natural selection can provide an avenue for microgeographical adaptation even when gene flow is moderate, because most genes arrive from populations facing similar selection. Given that sets of population often face similar biotic selection, the largest trait-selection mismatches will occur in populations facing a locally rare selection regime or in populations situated at the intersection of regions characterised by divergent natural selection regimes.

Mismatches between population traits and local biotic selection might create strong feedbacks on community dynamics (Thompson 2005; Urban & Skelly 2006; Urban *et al.* 2008; Pelletier *et al.* 2009; Post & Palkovacs 2009). For instance, locally adapted and cryptically coloured stick insects and moths are 2–3 times more likely to survive predator attacks than those with maladaptive prominent colouration (Clarke & Sheppard 1966; Nosil 2004). These differences in mortality, in turn, could alter predator–prey dynamics across natural landscapes. Ultimately, we need to understand better how gene flow and the strength and spatial distribution of biotic selection determine local adaptation and maladaptation, and hence mediate ecological dynamics.

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SUPPORTING INFORMATION

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

Appendix S1 Simulation analysis of MEM estimation of trait spatial autocorrelation depending on different assumed levels of gene flow.

Figure S1 Funnel plots.

Figure S2 Effects of research area on estimates.

Table S1 List of studies, organisms, their characteristics and individual results.

Table S2 Robustness results for variances explained by biotic selection, spatial autocorrelation and spatially autocorrelated selection.

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