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Quantifying the lag time to detect barriers in landscape genetics

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Abstract

Understanding how spatial genetic patterns respond to landscape change is crucial for advancing the emerging field of landscape genetics. We quantified the number of generations for new landscape barrier signatures to become detectable and for old signatures to disappear after barrier removal. We used spatially explicit, individual-based simulations to examine the ability of an individual-based statistic [Mantel's *r* using the proportion of shared alleles' statistic (Dps)] and population-based statistic (F_{ST}) to detect barriers. We simulated a range of movement strategies including nearest neighbour dispersal, long-distance dispersal and panmixia. The lag time for the signal of a new barrier to become established is short using Mantel's *r* (1–15 generations). F_{ST} required approximately 200 generations to reach 50% of its equilibrium maximum, although G'_{ST} performed much like Mantel's *r*. In strong contrast, F_{ST} and Mantel's *r* perform similarly following the removal of a barrier formerly dividing a population. Also, given neighbour mating and very short-distance dispersal strategies, historical discontinuities from more than 100 generations ago might still be detectable with either method. This suggests that historical events and landscapes could have long-term effects that confound inferences about the impacts of current landscape features on gene flow for species with very little long-distance dispersal. Nonetheless, populations of organisms with relatively large dispersal distances will lose the signal of a former barrier within less than 15 generations, suggesting that individual-based landscape genetic approaches can improve our ability to measure effects of existing landscape features on genetic structure and connectivity.

Keywords: computer simulation, connectivity, conservation genetics, gene flow, habitat fragmentation, landscape modelling, power analysis, resistance surfaces, spatial analysis

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Introduction

A primary goal for landscape genetics research is to understand how landscape features and environmental factors influence population structure, gene flow and genetic drift (Manel *et al.* 2003; Holderegger & Wagner 2006; Storfer *et al.* 2007; Balkenhol *et al.* 2009a). Classical population genetics analysis generally assumes sampling groups of individuals from predefined, isolated populations and then estimates parameters, such as F_{ST}, to assess genetic differences among these subpopulations. However, in continuously or widely distributed populations, where there often exists substantial internal structure (e.g. isolation by distance (Wright 1943) or landscape resistance (Cushman *et al.* 2006; McRae 2006)), population-based approaches can produce misleading results (Pritchard *et al.* 2000; Schwartz &

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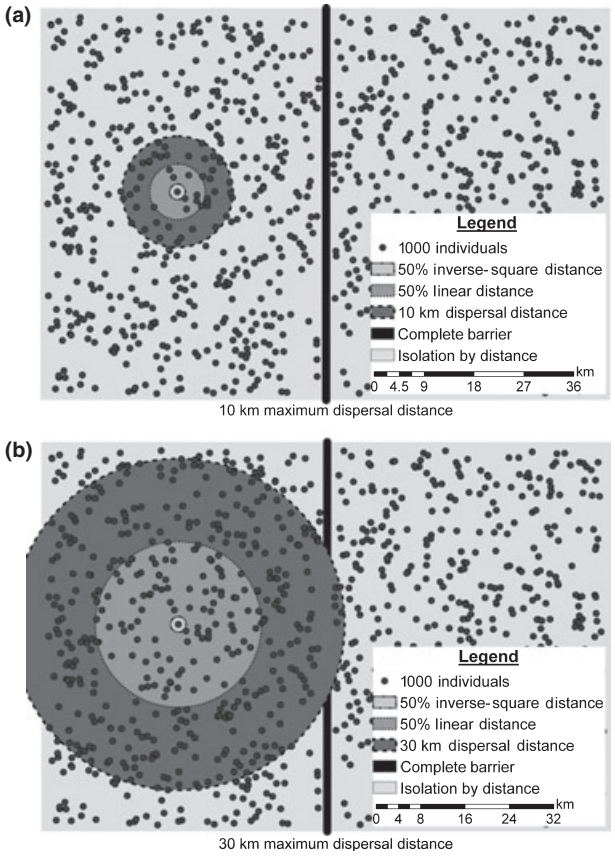

Fig. 1 An example of 1 simulation of 1000 randomly located individuals (dots) on a resistance surface of isolation by Euclidean distance with a complete barrier (vertical black line) for two maximum dispersal distances (a) 10 km and (b) 30 km. The largest dark circle (outer dashed line) represents the maximum dispersal distance. The two circles within the maximum dispersal distance correspond to the 50% dispersal distances for the centre individual under a linear (middle dotted line) and inverse-square movement strategy (tiny inner dashed-dotted line), respectively (i.e. the centre individual will mate and have offspring that disperse within this circle 50% of the time).

McKelvey 2009; Murphy *et al.* 2008; Cushman & Landguth 2010). One of the biggest contributions from landscape genetics research is the generalization of spatial genetic analysis to include either groups of samples or individual samples distributed across the landscape (e.g. Cushman & Landguth in press).

Individual-based landscape genetics approaches involve sampling of many individuals across broad landscapes, assessment of genetic differentiation between individuals and evaluation of relationships between inter-individual genetic distance (relatedness) and landscape gradients (Manel *et al.* 2003; Cushman *et al.* 2006; Storfer *et al.* 2007; Balkenhol *et al.* 2009a). The genetic signals of individuals sampled across landscapes allow localization of genetic discontinuities, identification of subpopulations and quantification of the influence of landscape features on gene flow.

Patterns of genetic distance among individuals or groups (e.g. Spear *et al.* 2005; Epps *et al.* 2007) can be tested with correlations between landscape features by assigning different resistance-to-movement values to different landscape features (e.g. a high resistance-to-movement might be assigned to a known barrier). The approach is based on raster maps that represent the cell-level resistance to gene flow related to different habitat or vegetation types, elevation, slope or other landscape features (see Cushman *et al.* 2006; Spear *et al.* 2010). Cells or pixels are given weights or 'resistance values' reflecting the presumed influence of each variable on movement of the species in question. A matrix of movement costs can then be computed based on shortest cost paths algorithms between all pairs of individuals. Common programmes to calculate cost-distance matrices in landscape genetics include Dijkstra's algorithm (Dijkstra 1959) [e.g. implemented through programmes like UNICOR (http://cel.dbs.umt.edu/software.php)], CIRCUITSCAPE (McRae & Beier 2007), PATHMATRIX (Ray 2005), and COSTDISTANCE in ArcGIS (ESRI Corp., Redlands, CA, USA).

The most widely used method to associate genetic distances with landscape cost distances is the Mantel test (Mantel 1967; see also Sokal & Rohlf 1995; Storfer *et al.* 2010). Using partial Mantel tests to test for correlation between genetic distance (relatedness) and alternative ecological distances enables tests of hypotheses regarding the effects of landscape structure on gene flow (e.g. Coulon *et al.* 2004; Broquet *et al.* 2006; Cushman *et al.* 2006; Schwartz *et al.* 2009; Cushman & Landguth 2010, Shirk *et al.* 2010). By comparing genetic distances between individuals with ecological cost distances between them, researchers can test specific hypotheses about the influences of landscape features and environmental conditions on gene flow (Cushman *et al.* 2006; Epps *et al.* 2007; Cushman & Landguth 2010, Shirk *et al.* in press).

Recent empirical studies have detected the effects of landscape and seascape structure on gene-flow patterns and suggest that spatially explicit, individual-based approaches in heterogeneous landscapes might be the best means to help establish mechanistic explanations of landscape connectivity (Cushman *et al.* 2006; Antolin *et al.* 2006; Neville *et al.* 2006; Selkoe *et al.* 2008; Pavlacky *et al.* 2009; Cushman & Landguth 2010; Shirk *et al.* in press). Furthermore, landscape genetic approaches can provide insights to key biological processes, such as individual movement, mating or dispersal, and could complement traditional population genetic approaches, for example, to localize genetic

Table 1 Maximum and 50% movement distance radius and number of individuals that occupy the corresponding areas based on a population density for the given simulation landscape of 1000 individuals in the total extent area of 7235.60 km^2. Note that the 50 and 60 km movement distances include individuals outside the focal study area of 70 × 100 km and not included in the simulation study

Movement distance	Maximum movement distance area (km^2)	Number of individuals in area	50% Linear movement distance area (km^2)	Number of individuals in linear 50% area	50% Inverse-square movement distance area (km^2)	Number of individuals in inverse-square 50% area
Panmictic	7235.60	1000.00	NA	NA	NA	NA
60 km	11 309.73	1563.07	2827.43	390.77	6.28	0.85
50 km	7853.98	1085.47	1963.50	271.37	6.28	0.85
40 km	5026.55	694.70	1256.64	173.67	6.28	0.85
30 km	2827.43	390.77	706.86	97.69	6.28	0.85
20 km	1256.64	173.67	314.16	43.42	6.28	0.85
10 km	314.16	43.42	78.54	10.85	6.28	0.85

discontinuities (barriers) and thus to understand how landscape features influence movement and gene flow (reviewed in Balkenhol *et al.* 2009a).

Applications of individual-based landscape genetic analysis techniques are increasing rapidly, yet there have been few rigorous studies to establish the sensitivity and reliability of these techniques (but see Murphy *et al.* 2008; Balkenhol *et al.* 2009b; Cushman & Landguth 2010). One of the most important research threads needing exploration is the temporal dynamics of landscape genetic processes. Little is known about how rates and patterns of landscape change affect the emergence, change and loss of genetic structure (but see Ezard & Travis 2006; Murphy *et al.* 2008; Cushman & Landguth 2010). Given that species conservation and management is primarily concerned with recent or future changes, if landscape genetics is to be used in this context then there is an urgent need to rigorously assess both the effects that legacies of past landscape change have on observed genetic patterns and the speed at which these genetic patterns change in response to alterations to existing landscapes. Simulation studies provide the most practical means to investigate the interactions between landscape change and spatial genetic processes, as they enable control over the pattern–process relationship and replicated simulation over long temporal periods (see Cushman & Landguth 2010, Epperson *et al.* 2010, Landguth *et al.* 2010; Bruggeman *et al.* 2010).

We conducted spatially explicit, individual-based simulations using the CDPOP model (Landguth & Cushman 2010) to investigate the power of two of the most commonly used metrics of genetic structure from population genetics and landscape genetics research (F_{ST} and Mantel's r, respectively) to detect landscape change. Specifically, we evaluate the time after establishment of a barrier between two populations until a barrier genetic signal is detected and stabilizes, and the time after the removal of a barrier until the barrier signal is lost. We simulate a range of individual movement strategies (how individuals mate and disperse with respect to the landscape's resistance) and maximum dispersal distances (how far an individual will move through mating and dispersal in terms of the landscape's resistance). This allows us to explore the interaction between species-specific dispersal characteristics and the emergence and loss of genetic structure following establishment and removal of dispersal barriers.

This type of study is crucial for understanding if contemporary genetic patterns are caused by extant landscapes or are a function of historical events. Understanding how rapidly a new barrier can be detected will allow us to infer if landscape genetic tools can be used to detect recent landscape fragmentation events, which is the focus of many ecological and conservation studies. In addition, it is critical to understand the length of time over which the signal of historical population barriers remains detectable. By exploring the spatio-temporal dynamics of landscape genetic processes under controlled simulated conditions, this research helps provide a foundation for landscape genetics in spatially complex and temporally dynamic landscapes.

Models and methods

Simulation programme

We used a spatially explicit, individual-based landscape genetics programme (Landguth & Cushman 2010) to simulate individual genetic exchange across 500 nonoverlapping generations among 1000 randomly spatially located individuals as functions of individual-based movement through mating and dispersal. All simulated populations were initiated with 30 loci and a 30 alleles

per locus maximum (resulting in 900 total possible alleles and mean H_o = 0.967), a *k*-allele model mutation rate of 0.0005 in a two-sex, random assignment of offspring sex, female individuals mating without replacement and male individuals mating with replacement. Initial genotypes were assigned by randomly drawing an allele for each locus from the 30 available alleles.

The programme represents landscape structure through resistance surfaces whose values represent the cost of crossing each location (i.e. grid cell or pixel on the landscape). Mating and dispersal are modelled as probabilistic functions of cumulative cost across these resistance surfaces (e.g. random, linear or inverse-square relationships between movement and cumulative cost; Landguth & Cushman 2010). These movement (mating and dispersal) strategies were scaled to maximum movement distances (i.e. to restrict an individual from moving across the entire landscape). For the presented simulations, probabilities for mating and offspring dispersal were identical. All cells were parameterized with identical resistance values except for barrier cells, whose resistance created a complete barrier and individuals could not move across the barrier. In the CDPOP output, genotypes at each generation for all spatially referenced individuals was produced from probabilistic cost-distance mating with Mendelian exchange and probabilistic cost-distance dispersal of offspring (Landguth & Cushman 2010). These spatially located genotypes can then be related to population substructure by either assigning spatial regions to different populations and calculating classical population genetic parameters (e.g. F_{ST}) or by analysing pairwise genetic distances among individuals with Mantel and partial Mantel tests (Mantel 1967).

Simulation scenarios

Our goal in designing our simulation scenarios was to represent a wide range of biologically plausible combinations of movement function and dispersal distance to make our results as generalizable as possible. Our general approach involves simulating emergence and dynamics of genetic substructure associated with the creation of a new barrier dividing the population, followed by subsequent removal of the barrier.

We stipulated a population of 1000 individuals and simulated all combinations of three movement strategies (linear, inverse-square, panmictic) scaled to six maximum movement distances (10, 20, 30, 40, 50 and 60 km). The maximum extent for the simulation area was 70 × 100 km (see Fig. 1 and Table 1).

The simulation involved two components. First, for each combination of movement strategy and distance, we placed a barrier, dividing the population in half, and ran the simulation for 500 generations. Genetic equilibrium was reached within 300 generations in all cases. Then, in the second step, we then removed the barrier and ran the model for an additional 500 generations. For each combination of movement strategy and maximum movement distance, we simulated 10 Monte Carlo replicate runs and calculated F_{ST}, Mantel's *r* with permutation tests for statistical significance, and two measures of effect size (generations until 50% and 90% of equilibrium maximum value of F_{ST} and Mantel's *r*) for detection of the barrier separating the subpopulations for each run of each scenario at each of 500 time steps.

Statistical analysis of simulation results

For all simulation scenarios, we computed F_{ST} of the populations on either side of the barrier and Mantel's *r* for each time step of each replicate run. F_{ST} was calculated as

$$F_{ST} = 1 - \frac{1}{2}\left(\frac{H_1 + H_2}{H_T}\right) \quad (1)$$

or the proportion of the total genetic variance contained in the subpopulation (Wright 1969). H_1, H_2 and H_T are calculated at each simulation time as the estimated fraction of all individuals who would be heterozygous based on allele frequency in an ideal Hardy–Weinberg population for the left subpopulation, right subpopulation and total population, respectively. We computed partial Mantel tests to correlate genetic distance to barrier-cost distance over time factoring out Euclidean distance for the partial tests. The test statistic for partial Mantel tests is calculated by constructing two matrices of residuals for (i) the regression of the genetic distance to barrier-cost distance (x) and (ii) the regression of the barrier-cost distance to Euclidean distance (y). Then the two residual matrices are compared by the standard Mantel test,

$$r = \frac{1}{n-1}\sum_{i=1}^{n}\sum_{j=1}^{n}\frac{x_{ij} - \bar{x}}{s_x}\frac{y_{ij} - \bar{y}}{s_y} \quad (2)$$

where n is the total population, and $\bar{x}$, s_x, $\bar{y}$ and s_y are the means and standard deviations of the respective residual matrices. The inter-individual genetic distance was calculated following the proportion of shared alleles (Dps; Bowcock *et al.* 1994) and barrier-cost distance was represented as a matrix representing Euclidean distance (and the one control simulation of panmixia) on either side of a complete barrier separating the two subpopulations.

We use two measures of barrier detection effects. First using the partial Mantel's *r*, we evaluate the

generations until a statistically significant signal is detected for barrier establishment and until a significant signal is lost following barrier disappearance. Because distances are not independent, we used permutation tests with 1999 permutations to test the significance of Mantel's correlation. The generation lag times for barrier establishment and disappearance were recorded for each scenario for $\alpha < 0.05$ with the Mantel's r statistic. All Mantel tests were conducted using the library ECODIST v1.1.3 (Goslee & Urban 2007) in the statistical software package R (R Development Core Team 2007).

The significance of statistical tests such as the Mantel's r is highly dependent on sample size. When sample sizes are large, differences that are ecologically trivial may still be statistically significant. In addition, statistical significance based on permutation (i.e. Mantel r values are not independent) and significance of difference from zero based on parametric test statistics (i.e. F_{ST} values are independent) are not comparable measures. Therefore, we also evaluated the effect size of the barrier. In all simulations, genetic equilibrium was achieved and F_{ST} and Mantel's r reached an approximate asymptote within 300 generations. We use the equilibrium maximum value as a reference point and calculate the number of generations required achieving 50% and 90% of this equilibrium maximum following creation of the barrier, and the number of generations following the removal of the barrier until 50% and 90% of the signal is lost.

Results

Time until barrier signal arises

Under nearly all barrier appearance scenarios, the individual-based analyses show a positive, significant Mantel's correlation appearing in less than 15 generations. Large correlation values ($r > 0.5$) occur within the first 100 generations, and all scenarios trend towards an equilibrium of $r = 1.0$ within 300 generations (Fig. 2, Table 2). A noticeable difference occurs in the scenarios in which the maximum movement distance is low (10 km); the highly constrained (or short distance) mating and dispersal movements in these scenarios result in substantially slower rate of approach to equilibrium.

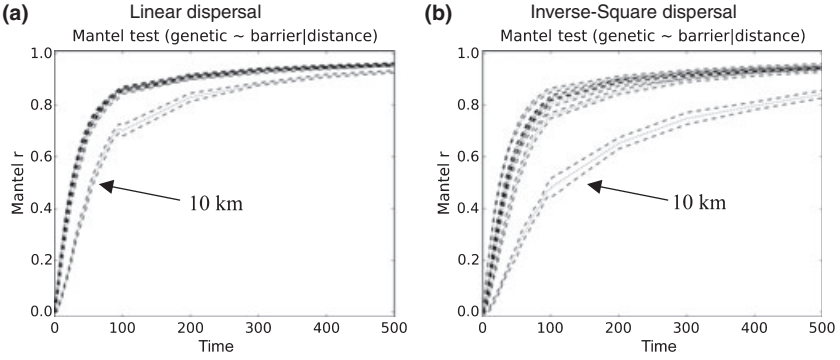

Fig. 2 Time (in generations) for barrier signal establishment based on significance of Mantel's r where we examined the correlation of genetic distance to barrier-cost distance over time factoring out Euclidean distance for the partial tests. Mantel's r values for the barrier appearance scenarios for (a) linear dispersal and (b) inverse-square dispersal over the seven maximal dispersal distances (10, 20, 30, 40, 50 and 60 km, panmictic). Dashed lines are confidence intervals for the averaged 10 replicates (dotted lines). The notably different scenario (movement threshold 10 km) is indicated in both graphs.

Table 2 Time (generations) to detection of a barrier using Mantel P-value <0.05 in which a significant generation of a barrier was detected across the movement strategies (linear, inverse-square) and scaled to the maximum movement distances (10, 20, 30, 40, 50 and 60 km, Pan). For the individual-based barrier establishment scenarios, the starting generation for significant detection is reported ($P < 0.05$). The starting generation for insignificant detection ($P \geq 0.05$) is reported for when a barrier signal is no longer detectable for the barrier removal scenarios. Generations >500 indicates that all r values were significant

	10 km	20 km	30 km	40 km	50 km	60 km	Pan
Barrier establishment							
Linear movement (generations)	6	2	1	1	1	1	1
Inverse-square movement (generations)	14	5	3	3	2	2	1
Barrier removal							
Linear movement (generations)	>500	>500	25	17	8	5	1
Inverse-square movement (generations)	>500	>500	>500	32	27	24	1

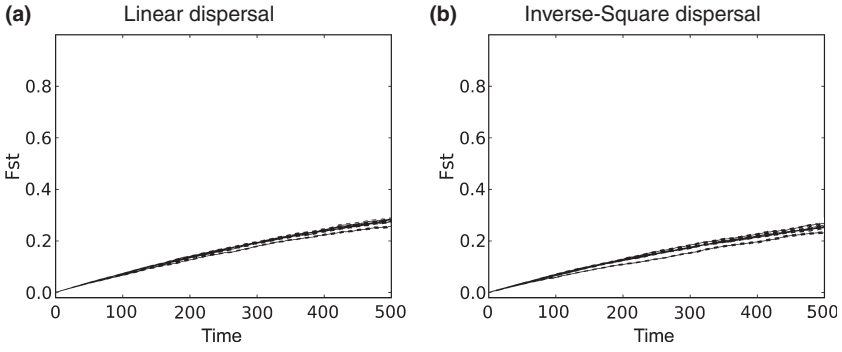

Fig. 3 Time for barrier signal establishment based on significance of F_{ST}. F_{ST} values for the barrier appearance scenarios for (a) linear dispersal and (b) inverse-square dispersal over the seven maximal dispersal distances (10, 20, 30, 40, 50 and 60 km, panmictic). Dashed lines are confidence intervals for the averaged 10 replicates (dotted lines).

The rate of substructure appearance using F_{ST} is slow and nearly linear across the range of maximum dispersal distances (Fig. 3). The partial Mantel's *r* is much more sensitive than F_{ST} in detecting the effects of recent landscape changes on spatial genetic substructure, as indicated by much more rapid rise towards an asymptote at the equilibrial maximum (Fig. 2).

The boxplots presenting the effect-size results for the three-way factorial of statistical measure (Mantel's *r* vs. F_{ST}), dispersal threshold (10, 20, 30, 40, 50 and 60 km) and movement function (linear and inverse-square) indicate major differences between Mantel's *r* and F_{ST} in the rate at which a new genetic signal emerges following the advent of a new barrier dividing a population with isolation by distance (Fig. 4). There are three major insights provided by Fig. 4. First, Mantel's *r* responds much faster to the advent of a new barrier dividing a population regardless of movement function and dispersal threshold (a, c vs. b, d in Fig. 4). Second, the generations required for F_{ST} to achieve 50% or 90% of its maximum equilibrium value (210 and 420 generations, respectively) is independent of movement threshold or movement strategy (Fig. 4). Third, Mantel's *r* is sensitive to both movement function and movement threshold (a, b vs. c, d in Fig. 4). For example, it takes approximately 50 generations for Mantel's *r* to reach 50% of its equilibrium maximum when the dispersal function is linear and the dispersal threshold is 10 km, while it takes approximately 100 generations to reach the same level when the dispersal function is inverse-square and the threshold is 10 km. In addition, the difference in time to reach a given proportion of equilibrium maximum between the two movement functions disappears as the dispersal threshold increases above 20 km (e.g. 15 generations to reach 50% and 100–150 generations to reach 90% of the equilibrium Mantel's *r* at all movement threshold distances above 20 km).

Time until barrier signal disappearance

A significant barrier signal is retained over the entire 500-generation simulation time for scenarios involving relatively short-distance movement (e.g. linear movement strategy with 10 km movement distance and inverse-square movement strategy with 10–30 km movement distances). Time until loss of the barrier signal is highly dependent on maximum movement distances. The signal of a former barrier is lost rapidly when maximum dispersal distances are relatively large (Figs 5 and 6 and Table 2).

In strong contrast to the pattern observed following the advent of a new barrier, there are no clear differences between Mantel's *r* and F_{ST} in terms of the generations required for a genetic signature of a former barrier to be lost in the population (Fig. 7). While there are no clear differences between the two statistical measures of population differentiation, there is a strong effect of both movement function and dispersal threshold on the generations required before a genetic signal of a former barrier is lost from a population (a, b vs. c, d in Fig. 7). Similarly, the genetic signal of a former barrier is also lost faster when dispersal distances are relatively large. For example, it takes an average of approximately 100 generations to lose 50% of the barrier signal when the dispersal threshold is inverse-square and the movement threshold is 10 km, while it only takes 40 generations when the movement threshold is 20 km and drops to <10 generations when the movement threshold is 30 km or more (Fig. 7). These results show that there is considerable effect of organism vagility on the time required for the signal of a former landscape condition to be lost from the genetic makeup of a population, while there is no difference in the performance of F_{ST} and Mantel's *r* in how rapidly they respond to removal of a past barrier.

Discussion

Rapid detection of recent landscape change

A primary goal in landscape genetics is to detect pattern–process relationships in contemporary landscapes, such as the effects of recent landscape change on population connectivity. The most important finding of this

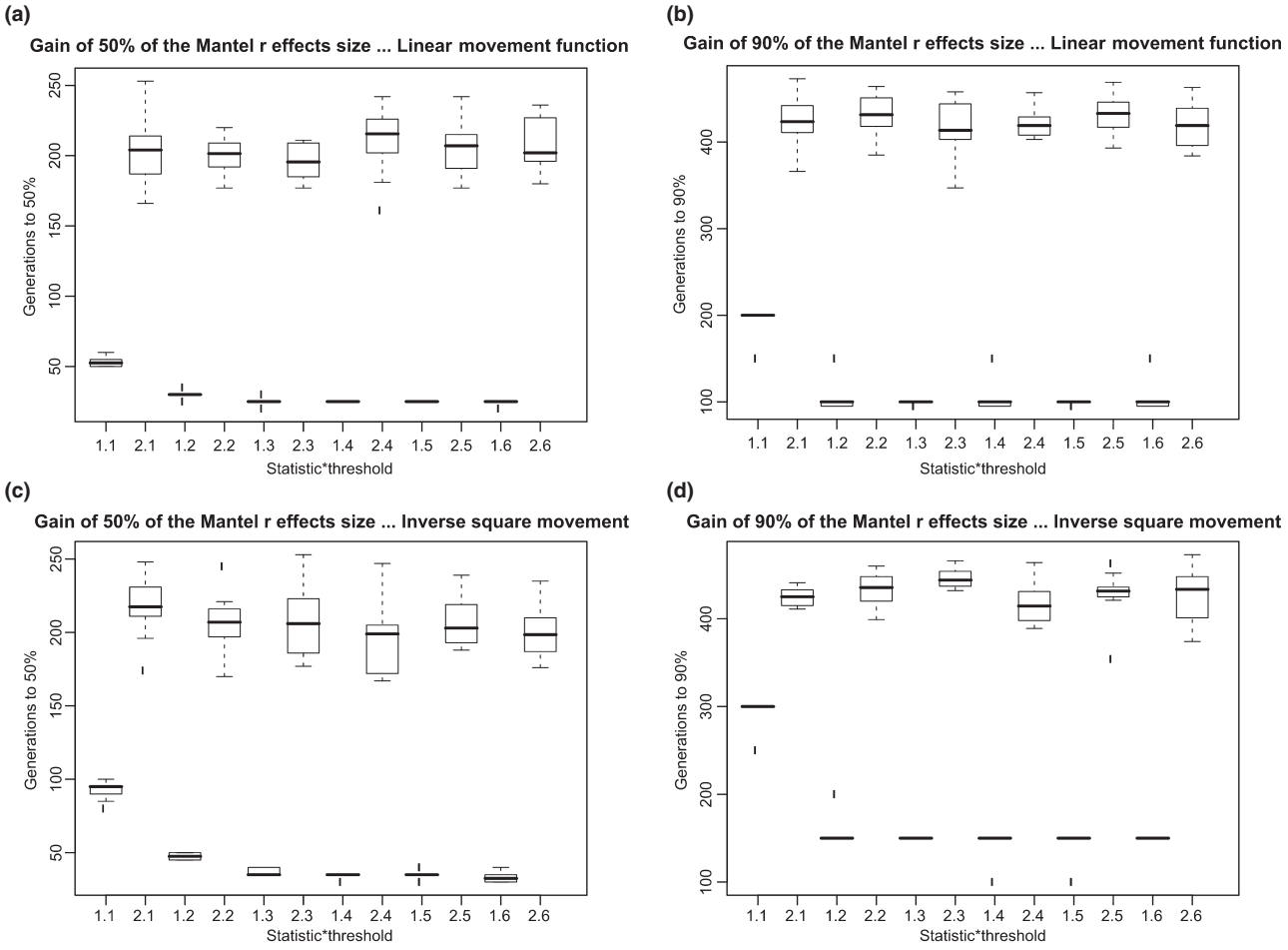

Fig. 4 Time for barrier signal establishment based on effect size for Mantel's r and F_{ST}. The boxplots collectively present a three-way factorial of differences among two statistical measures of genetic differentiation (Mantel's r and F_{ST}), across six dispersal thresholds (10, 20, 30, 40, 50 and 60 km), and dispersal and mating movement function (linear and inverse-square). Boxplots (a) and (b) compare generations to 50% and 90% of equilibrium value of Mantel's r and F_{ST}, respectively, across six dispersal and mating threshold distances and a linear dispersal function simulated in CDPOP. Boxplots (c) and (d) compare generations to 50% and 90% of equilibrium value of Mantel's r and F_{ST}, respectively, across six dispersal threshold distances and an inverse-square dispersal function simulated in CDPOP. The first digit of the x-axis label indicates which statistical measure is used: 1—Mantel's r, 2—F_{ST}. The second digit of the x-axis label indicates which threshold distance is used: 1—10 km, 2—20 km, 3—30 km, 4—40 km, 5—50 km, 6—60 km.

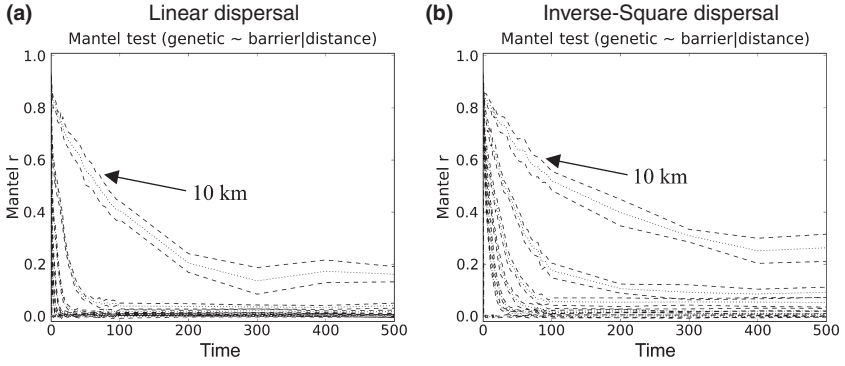

Fig. 5 Time until barrier signal disappearance based on significance of Mantel's r. Mantel's r values for the barrier removal scenarios for (a) linear dispersal and (b) inverse-square dispersal over the seven maximal dispersal distances (10, 20, 30, 40, 50 and 60, panmictic). Dashed lines are confidence intervals for the averaged 10 replicates (dotted lines). The notably different scenario (movement threshold 10 km) is indicated in both graphs.

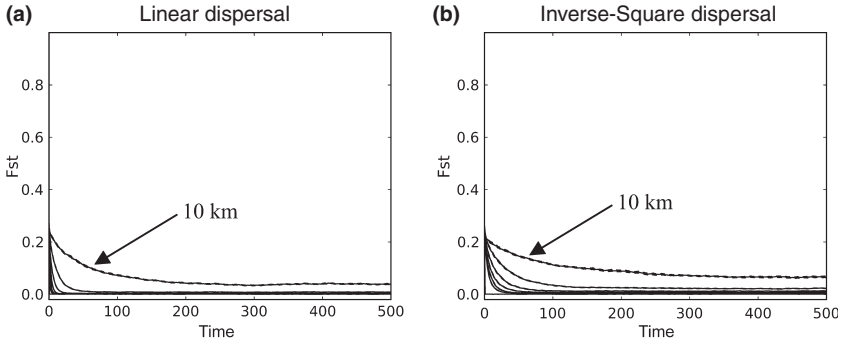

Fig. 6 Time until barrier signal disappearance based on significance of F_{ST}. F_{ST} values for the barrier removal scenarios for (a) linear dispersal and (b) inverse-square dispersal over the seven maximal dispersal distances (10, 20, 30, 40, 50 and 60 km, panmictic). Dashed lines are confidence intervals for the averaged 10 replicates (dotted lines).

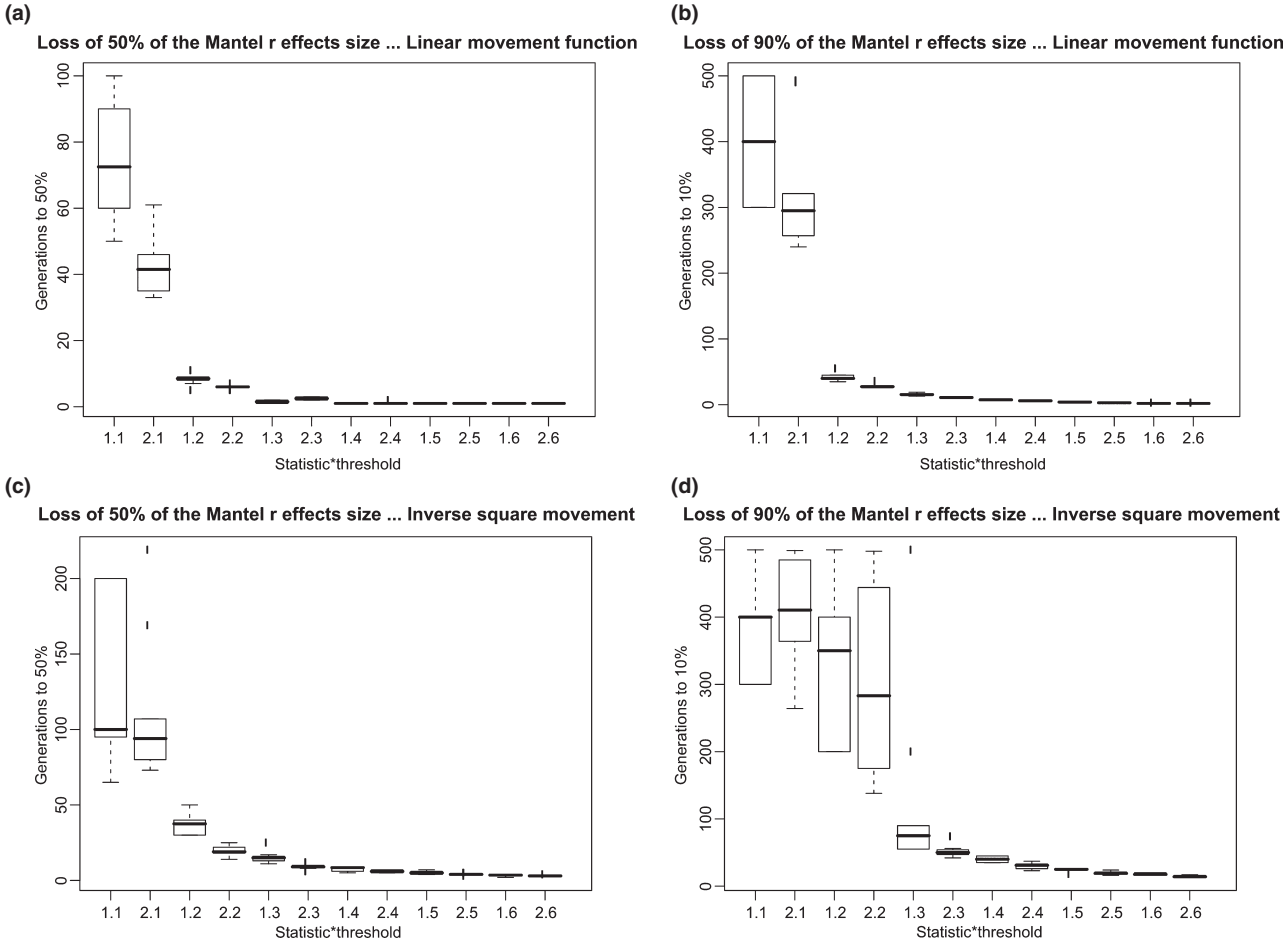

Fig. 7 Time for barrier signal disappearance based on effect size for Mantel's *r* and F_{ST}. The boxplots collectively present a three-way factorial of differences between two statistical measures of genetic differentiation (Mantel's *r* and F_{ST}), across six dispersal thresholds (10, 20, 30, 40, 50 and 60 km) and dispersal and mating movement function (linear and inverse-square). Boxplots (a) and (b) compare generations to loss of 50% and loss of 90% of equilibrium value of Mantel's *r* and F_{ST}, respectively, across six movement threshold distances and a linear dispersal and mating function simulated in CDPOP. Boxplots (c) and (d) compare generations to loss of 50% and 90% of equilibrium value of Mantel's *r* and F_{ST}, respectively, across six movement threshold distances and an inverse-square movement function simulated in CDPOP. The first digit of the *x*-axis label indicates which statistical measure is used: 1—Mantel's *r*, 2—F_{ST}. The second digit of the *x*-axis label indicates which threshold distance is used: 1—10 km, 2—20 km, 3—30 km, 4—40 km, 5—50 km, 6—60 km.

study, therefore, is that the lag time for a new barrier to become detectable is relatively short using the Dps statistic and Mantel tests. This is in contrast to empirical studies that found long lag time for detection of the genetic effects of landscape change using F_{ST} (Holzhauer *et al.* 2006; Keyghobadi *et al.* 2005). We found Mantel's *r* achieves an equivalent effect size roughly 10 times faster than $F_{ST.}$ This rapid detection of a barrier with individual-based approaches compared to F_{ST} confirms results from previous simulations under a one-dimensional stepping stone model (Murphy *et al.* 2008). It is also consistent with some recent empirical studies that have found allele frequency-based metrics respond rapidly to recent landscape change (Spear & Storfer 2008; Murphy *et al.* 2010a,b).

The highly nonlinear relationship between time to barrier detection and dispersal strategy using Mantel tests with Dps has a number of important implications for research and management. For dispersal abilities larger than 10km (i.e. approximately 1/5th the distance across the study area), time to barrier detection was short (1–3 generations). However, there is a strong nonlinear threshold relationship, such that as dispersal ability becomes very low, time until barrier detection rises rapidly. For species that have naturally low dispersal ability, it will take much longer for a barrier to be detected than those with long-distance dispersal capability. This implies that genetic studies of species with low dispersal might not detect effects of landscape fragmentation for many generations, even if the landscape change has resulted in complete isolation of subgroups of a previously connected population (see Epperson 2007; Spear & Storfer 2008; Murphy *et al.* 2010b). This could easily lead to incorrect supposition that the landscape change has not resulted in population isolation, which could lead to failure to identify potentially serious conservation issues (e.g. Gerlach & Musolf 2000; Keyghobadi *et al.* 2005, Gerlach & Musolf 2000).

For most mobile animals, and plants with relatively broad pollen and seed dispersal distances, the lag time to barrier detection is short. This suggests that early detection of population fragmentation can be achieved using individual-based genetic methods on organisms with relatively long range mating and/or dispersal. In addition, landscape change can begin to influence the genetic structure and architecture of a population almost immediately. For example, genetic structure resulting from recent road fragmentation has been observed using individual-based measures (Keller *et al.* 2004; Clark *et al.* 2010).

The strong contrast between the sensitivity of Mantel's *r* and F_{ST} for detecting barrier signals has important implications that should guide the selection of methods used to measure the effects of recent landscape change on population connectivity. The explanation for the observation that time to emergence of a barrier signal was independent of movement strategy and was much slower using F_{ST} in that F_{ST} is based on heterozygosity. This makes it much less sensitive to recent landscape change than Mantel's *r*, because a shift in genotype frequencies is likely to happen more quickly than changes in heterozygosity (Luikart *et al.* 1998; Murphy *et al.* 2008, 2010b). This suggests that F_{ST} is a poor choice of response metric in studies aimed at measuring effects of recent landscape change on population connectivity (but see limitations of our work below) and that individual-based genetic distances coupled with the mantel test are more sensitive alternatives.

Genetic legacies of past landscape structure

Our results indicate that the effects of past landscape conditions from more than 100 generations ago might still be detectable with Mantel's *r* or F_{ST}, in species with limited dispersal abilities. This finding is important because it suggests that historical events and past landscape changes may have long-lasting legacy effects on the spatial genetic structure of populations from certain species (Dyer *et al.* 2010).

The strong relationship between dispersal ability and time for a barrier signal to be lost provides useful guidance to researchers and conservation managers. For organisms with large dispersal abilities relative to population density, a barrier signal is lost rapidly. Over 50% of the effect size (Mantel's *r*) of the signal of a past barrier is lost within 15 generations for populations of organisms with relatively large dispersal abilities. This implies that the legacy of past landscape structure may not be a major problem in studies of highly mobile organisms (but see Dyer *et al.* 2010). However, our simulation suggests that organisms with relatively restricted dispersal abilities will retain a genetic signal from a past barrier for tens to hundreds of generations. For example, it took over 300 generations to lose 90% of the barrier signal for organisms with inverse-square movement function and dispersal thresholds up to 20 km.

There is a strong nonlinear relationship between time for a former barrier to become undetectable and dispersal ability. This strong nonlinearity in time to loss of a past barrier signal as function of dispersal distance has important implications for studies that hope to separate the effects of current from past landscape conditions (Dyer *et al.* 2010). This 'ghosts of landscape past' effect suggests that analyses of relationships between spatial genetic structure and landscape features should carefully consider past landscape features, especially when the dispersal abilities of the study organism are limited.

Scope and limitations

The simulations framework used in this study has several advantages for landscape genetic analyses. It enabled us to simulate gene flow in modelling experiments in which the process was known and the key parameters of dispersal ability were systematically varied to explore the effects of population vagility on the rate of emergence and loss of a genetic signal caused by a population barrier. In addition, because the simulation we implemented is individual-based and spatially explicit, we were able to directly compare the sensitivity of Mantel's r to Dps and F_{ST} in detecting barrier effects from current and past landscape conditions. However, as in any simulation, our analysis is a simplification and has a limited scope.

There are three main simplifications in our analysis that should be considered when interpreting our results. First, our model is based on nonoverlapping generations. The effects of overlapping generations in long-lived species on the emergence of genetic structure are not considered in our analysis. Overlapping generations can have some impact on population genetic structure (e.g. Epperson 2003), but little research has been conducted in heterogeneous landscapes. Some longer-lived highly mobile species might not show effects of fragmentation on a timescale useful for making conservation and management decisions (Coulon *et al.* 2004) or only show weak effects (Coulon *et al.* 2006; Spear & Storfer 2008). In addition, many long-lived plant species show more continuous genetic structure where fragmentation effects are less easy to detect (e.g. Wagner *et al.* 2005). Future work should explore the effects of overlapping generations on landscape genetic inferences.

Second, our model is based on a constant-sized population. We stipulated a constant population across simulated generations because we wanted to explore the relationships between dispersal ability and genetic differentiation across a barrier without the confounding influence of the effects of a dynamic population on genetic change. However, real populations may have lower effective population size and/or effective population size may fluctuate over time. This can have little (Murphy *et al.* 2008) or substantial influences on the nature and rate of genetic change observed (e.g. Engen *et al.* 2005). It will be important for future work to explore the effects of dynamically varying population size in heterogeneous landscapes using genetic methods.

Third, this simulation is based on 1000 individuals initialized with 30 alleles and 30 loci per allele. The rate of detection of the genetic signal caused by landscape change is likely highly sensitive to variation in the proportion of population sampled, the number of loci analysed and the allelic richness and heterozygosity. This paper investigated a single combination of population size, number of alleles and number of loci. Ongoing work (E. L. Landguth, unpublished data) has shown that the number of loci and the number of alleles both strongly affect the equilibrium effect size and the power of causal modelling to detect the effects of landscape structure on genetic differentiation in complex landscapes. Further work is needed to explore how variation in sampled population size, number of loci and the rate at which the effect of landscape change on genetic differentiation becomes detectable (see Murphy *et al.* 2008).

In this manuscript, we chose to focus on what is far and away the most commonly used individual-based (Mantel's r) and population-based (F_{ST}) metric of gene flow. What is different about our simulation is that we were looking at transient states associated with the imposition or removal of a barrier. In these situations, F_{ST} is theoretically flawed, as it assumes constancy and equilibrium. In our simulations, initial heterozygosity was extremely high, much higher than would be likely in a natural population of the size simulated. Because heterozygosity was extremely high at $t = 0$, maximum F_{ST} was very low. With succeeding generations, heterogygosity dropped, and maximum F_{ST} increased leading to a signal, which started small and slowly increased—always limited by the maximum F_{ST}. This is clearly shown by applying Hedrick's transform from F_{ST} to G'_{ST} which scales F_{ST} to its maximum value (Hedrick 2005). Figure 8 shows clearly that a scaled F_{ST} is, in fact, as sensitive, or more so, than Mantel's r based on genetic distance. However, using to G'_{ST} is not a panacea; to G'_{ST} has many of the underlying assumptions

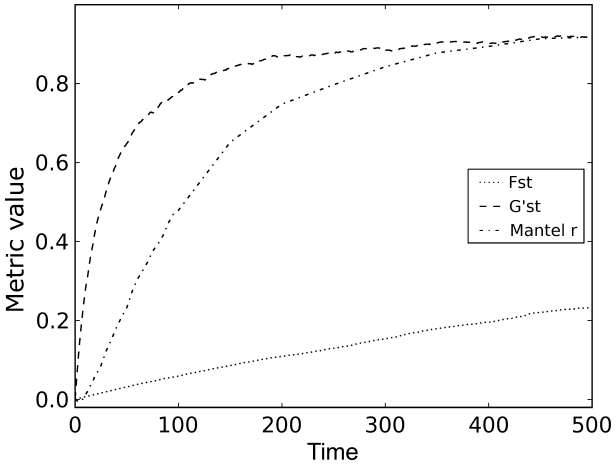

Fig. 8 G'_{ST} (dashed line), F_{ST} (dotted line) and Mantel's r (dash-dotted line) values for the barrier appearance scenario of inverse-square dispersal with the 10 km maximal dispersal. Dashed lines are confidence intervals for the averaged 10 replicates (dotted lines).

associated with the *F*-statistics and can be misleading under some circumstances such as high migration rates (Ryman & Leimar 2008). Mantel tests make few assumptions about the underlying population structure, equilibrium, neutrality of the markers or evolutionary model. Additionally, partial Mantel tests allow spatial patterns to be separated allowing barrier analysis in complex correlated environments.

The loss of heterozygosity across generations in our simulations may have been larger than is expected in natural populations, but we believe that rapid losses of heterozygosity associated with imposed barriers are not uncommon. In many cases, large contiguous populations have become highly fragmented in relatively short periods of time. Thus, the weaknesses we have demonstrated in the use of F_{ST} in these simulations will occur to one extent or another in nature.

There are, of course, many alternative methods that have been used to identify barriers. For example, Safner *et al.* (in press) reviewed a number of alternative individual-based approaches for the detection of barriers finding that Bayesian methods performed better than edge detection methods with GENELAND detecting the barriers in 49% of the simulation runs at generation 100. Additionally, there is a plethora of population-based metrics, including D_{ST} (Jost 2008). Also, the proportion of shared alleles was used as the distance metric for the Mantel tests. Many additional metrics with varying assumptions and power are available (for review, see Legendre & Legendre 1998), and future work should explore how choice of genetic distance measure affects the emergence and loss of genetic signals.

The simulation we conducted was designed to represent a broad range of dispersal abilities relative to population density and landscape heterogeneity. However, more work will be needed to evaluate the functional nature of the responses we identified. Specifically, our analysis suggests that there are strongly nonlinear relationships between time until a genetic signal appears or is lost following landscape change and dispersal ability. The simulations we ran do not enable generalization of the relationships between population density, movement strategy and dispersal distance. This is an important topic and more work is needed to quantify the threshold-like relationships suggested by this analysis between rate of emergence and loss of genetic structure and population density, dispersal distance, movement strategy and spatial complexity of the landscape.

Conclusion

These results overall are encouraging for those who wish to use individual-based landscape genetic approaches to infer the influences of current landscape features on gene flow and population connectivity. New landscape features, like roads, deforested areas, or other movement barriers, have rapid effects that are detectible almost immediately with individual-based landscape genetic approaches. F_{ST} is much less sensitive than Mantel's *r* on Dps (Keyghobadi *et al.* 2005; Murphy *et al.* 2010a,b), but initial simulations suggest that this is not because of individual-based approaches performing better than group-based statistics (which will occur in some circumstances), but rather because of the choice of an appropriate metric given the power of the metric for the question of interest.

Most combinations of movement strategy and dispersal threshold that we simulated indicated a rapid loss of the effects of previous landscape structure, and a decline to nonsignificance within a few hundred generations at most. This suggests that for organisms with relatively large dispersal abilities, legacy effects from past landscape conditions are likely to be lost within less than 15 generations, while they may remain for dozens to hundreds of generations for organisms with very limited dispersal abilities. This highlights the importance of considering organism vagility and life history when interpreting results of landscape genetic analysis. These results also indicate that longer-term changes, such as major post-Pleistocene climatic and biome shifts, are sufficiently distant to have no detectible effect on current genetics using microsatellites or other neutral genetic markers.

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