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RESEARCH ARTICLE

Habitat amount, not habitat configuration, best predicts population genetic structure in fragmented landscapes

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Abstract
Context Landscape structure shapes the genetic structure of populations by delimiting spatial patterns of dispersal and reproduction across generations. Thus, descriptions of human-altered landscapes can be used to predict demographic and evolutionary outcomes of populations. Effectively measuring landscape structure to predict genetic structure requires that we understand the relative importance of distinct components of landscape structure (e.g., habitat amount and configuration) in creating spatial patterns of genetic variation.
Objectives We thus developed an individual-based simulation model to test predictions about the relative importance of habitat amount and configuration in producing genetic structure. We also investigated the independent relationships between components of landscape structure and the population dynamics that underlie genetic effects.
Methods We ran experiments in which we allowed gene flow and population size to vary as emergent outcomes of the interactions between hypothetical populations and heterogeneous landscapes.
Results We found that the amount of habitat in a landscape is a much better predictor of genetic structure than is habitat configuration. This pattern holds across a range of landscapes and dispersal distances and behaviors. When habitat is non-contiguous (i.e., fragmented), habitat amount mediates production of genetic differentiation by regulating both the size and isolation of habitat patches, which in turn regulate population size and gene flow.
Conclusions These results suggest that habitat amount, a simple measure that is easy to calculate, may often be the best metric for predicting population genetic structure and that when possible, measures of habitat amount and population size should be incorporated into landscape genetic studies.

Keywords Connectivity · Conservation genetics · Habitat fragmentation · Habitat loss · Individual-based model · Landscape metric

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Introduction

Spatial structure of habitat within landscapes shapes spatial structure of genetic variation within and among

populations (Templeton et al. 1990; Young et al. 1996; Funk et al. 2005). Yet, despite ample research documenting this association in a wide range of species (reviewed in Keyghobadi 2007), little is known about how distinct components of landscape structure (e.g., the amount and configuration of habitat) contribute to genetic divergence, or about the mechanisms that underlie these independent relationships. Understanding these relationships has important implications for conservation management and research. For example, all else being equal, if our goal is to reduce genetic isolation among populations, should we focus on managing the spatial arrangement of habitat (habitat configuration) or simply on conserving as much habitat as possible?

One of the underlying mechanisms governing spatial patterns of genetic variation (i.e., creating genetic structure) is restricted dispersal (and hence, gene flow) across the landscape. Dispersal can be restricted through either (1) the removal of habitat (habitat loss), which results in the division of habitat into increasingly isolated patches (Fig. 1a), or (2) the fragmentation of habitat (a metric of habitat configuration), which renders a given patch of habitat into multiple fragments (Fig. 1b). Individuals inhabiting more isolated habitat genetically diverge over time due to genetic drift, resulting in genetic structure (Wright 1931; Slatkin 1987). Although habitat fragmentation per se—the subdivision of habitat, independent of habitat amount (Fahrig 2003)—increases the number of distinct patches, it also decreases the average distance among patches (Fig. 1b). Thus, the overall effect of habitat fragmentation on genetic structure via habitat isolation is unclear.

A second factor contributing to the creation of genetic structure is population size, which is constrained by the amount of habitat in the landscape (Fahrig 2003). Fragmentation may also influence population size, such as in the case of negative edge effects. The rate at which genetic drift leads to differential frequencies and fixations/losses of alleles in isolated populations directly depends on population size (Wright 1931; Kimura and Ohta 1969; Slatkin 1987). If some limitation in gene flow between two habitat patches exists, then as mean patch size (and thus population size) decreases, genetic divergence becomes more probable due to accelerated genetic drift. For reduced population size to contribute to genetic structure, some habitat isolation must exist initially; but once it does, reductions in population size may significantly affect the extent of genetic structure observed across landscapes.

In spite of the theoretical contribution of population size to genetic structure, demographic effects are often minimized or ignored in simulation studies (e.g., Bruggeman et al. 2010; Cushman et al. 2012, 2013;

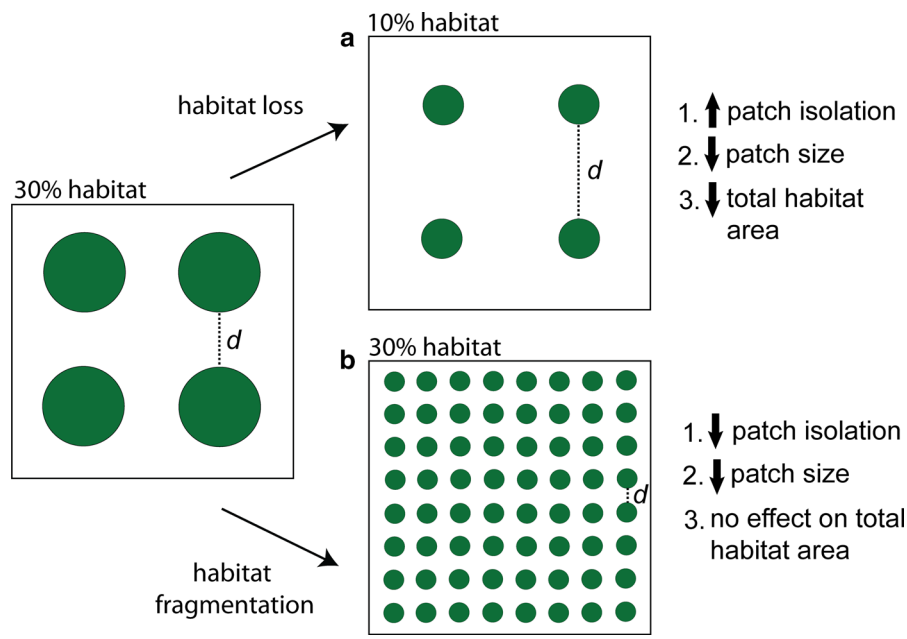

Fig. 1 Effects of habitat loss (composition) and fragmentation (configuration) on habitat isolation (*green/dark* = habitat; *white* = non-habitat). **a** Habitat loss increases the average distance among patches (*d*, shown by the *dotted line*) while decreasing both average patch size and the total amount of habitat available to a species. **b** Habitat fragmentation decreases both the average distance among patches and patch size while keeping the total amount of habitat constant. (Color figure online)

Graves et al. 2012) and are generally underappreciated in landscape genetics, where much focus has been on how genetic structure is shaped by impacts of habitat isolation on dispersal and gene flow (Holderegger and Wagner 2008; Balkenhol et al. 2009). In one prevailing approach, effective geographic distances among sampled individuals are calculated based on the hypothesized permeability of various landscape features and cover types to movement and gene flow (e.g., using resistance distances, McRae 2006, or cost distances, Bunn et al. 2000), and subsequently fitted to genetic distances among samples. This is a powerful approach for exploring effects of landscape structure on population connectivity and gene flow (e.g., Cushman et al. 2006; McRae and Beier 2007). However, as these effective distances do not explicitly incorporate information about population size, these studies cannot address the role of population size in shaping genetic structure. In another approach, impacts of habitat structure on genetic structure are investigated by fitting genetic distances to different measures of landscape composition or configuration, calculated either at the level of a patch (Honnay et al. 2006), a landscape (Schmidt et al. 2009; Kanuch et al. 2012; Lange et al. 2012; Telles et al. 2014; van Strien et al. 2014), or both (Mapelli et al. 2012; Balkenhol et al. 2013; Millette and Keyghobadi 2015). Such studies use a wide range of "landscape metrics" (Gustafson 1998; McGarigal et al. 2002) to optimize measurement of the aspects of landscape structure that most influence population connectivity (e.g., patch cohesion, Schumaker 1996; proximity index, Gustafson and Parker 1994; or patch density). However, these metrics typically incorporate aspects of both habitat amount and habitat configuration (see Neel et al. 2004), so the relative influence of these two factors (or of population size and gene flow) on genetic differentiation is not clear. If the configuration of habitat drives genetic outcomes, we should continue to focus on the effects of landscape on gene flow and on the development and testing of configuration metrics. If the amount of habitat is the better predictor, we should concentrate on improving our definitions of "habitat" for the various species in question such that its proportion in landscapes can be accurately calculated (Kozakiewicz 1995; Betts et al. 2014) and incorporated into landscape genetic studies.

Because a reduction in both population connectivity and population size may theoretically contribute to patterns of genetic structure, it is important to track both processes when investigating the effects of landscape structure on genetic structure. Recent simulation studies that explore the relative importance of habitat amount and configuration on genetic divergence explicitly limit demographic fluctuations (Bruggeman et al. 2010), or disallow them altogether (Cushman et al. 2012). These studies thus generate predictions about the importance of landscape structure components on genetic structure via their effects on connectivity. However, if demographic fluctuations play an important role in mediating genetic structure in real populations, the relative importance inferred might be substantially different.

In this study, we use an individual-based simulation model to investigate the relative importance of habitat amount and configuration for creating population genetic structure across landscapes. In doing so, we allow patterns of both gene flow and population size to organically emerge from interactions of individuals with landscapes that vary independently in the amount and configuration of habitat. We also clarify the mechanisms by which components of landscape structure shape genetic structure by inquiring into how these two factors predict emerging patterns of gene flow and population size, and how these emerging patterns in turn predict genetic structure. To increase the generality of our findings, we carry out simulations for hypothetical species that vary in dispersal distance and behavior. Our ultimate goal is to establish predictions about the shape, relative strength, and underlying causes of the distinct relationships between two major components of landscape structure (habitat amount and habitat configuration) and genetic structure. These predictions will help to refine our measures of landscape structure for use in landscape genetic and conservation research.

Methods

Model overview

We developed an individual-based model (Grimm and Railsback 2005), that simulates generations of population dynamics for hypothetical species inhabiting heterogeneous landscapes (based in part on a previous model; Jackson and Fahrig 2014). The model first produces stochastic landscapes that vary independently

in the amount and configuration of habitat; a total of 25 landscape types were simulated. Then, within these landscapes, the model simulates birth, dispersal, mating, reproduction, and death of individuals over time. At the end of each simulation run, we sampled genetic structure of populations to determine how these patterns are governed by variation in landscape structure (see Table 1 for a list of model parameters).

Because dispersal behavior of species is expected to mediate relationships between landscape structure and population responses (Nichols and Hewitt 1994; Jackson and Fahrig 2012), we varied the average distance traveled by individuals during dispersal (six levels) and their dispersal behavior in response to non-habitat (two levels): random and informed. Random dispersal (RD), commonly observed in plants (e.g., wind dispersed seeds) and in passively dispersing invertebrates, results in movement paths that are independent of underlying landscape structure. Thus, under RD, landscape structure influences population dynamics through differential rates of reproduction in habitat and non-habitat (where the rate is zero). Informed dispersal (ID) allows movement decisions to be influenced by environmental cues, which occurs to some extent in most terrestrial animals (e.g., individuals distinguish habitat from non-habitat via food availability or the presence of conspecifics; Reed and Dobson 1993; Bowler and Benton 2005). Under ID, landscape structure affects population dynamics by directing dispersal such that individuals tend to move and settle in habitat (Conradt et al. 2000).

Landscape simulation

We simulated stochastic landscapes that vary independently in the amount and "clumpiness" (the

Table 1 Input and output parameters of the simulation model used in this study

Parameters	Values/description
Input parameters (varied)	
Proportion of habitat (P)	0.1, 0.3, 0.5, 0.7, 0.9
Clumpiness of habitat (H)	0.1, 0.3, 0.5, 0.7, 0.9 (measuring degree of spatial autocorrelation)
Average dispersal distance (D_{av})	Step length = exponential (D_{av}) where D_{av} = 1, 2, 3, 4, 5, 6 cells
Dispersal behavior (RD and ID)	Random dispersal (RD): disperse in random direction, into matrix or habitat
	Informed dispersal (ID): disperse in random direction, into habitat only
Input parameters (constant)	
Landscape size	101 × 101 cell grid
Initial population size	5000 individuals
Run length	1000 generations
Replicates per treatment	100 runs
Death rate	100 % per generation
Sex ratio	1:1
Reproductive rate (λ)[a]	Two individuals
Carrying capacity (K)[a]	Two individuals per cell (or 1.8 individuals per cell in edge habitat under ID + NEE)
Number of offspring (R)	$2\left[\frac{\lambda}{1+\left(\frac{\lambda-1}{K}\right)N_F}\right]$
Output parameters[b]	
Population size (N)	Total number of individuals
Genetic distances (Dps)	Proportion of shared alleles, calculated pairwise among sample of 200 individuals
Genetic structure (Mantel r)	Correlation coefficient from Mantel test of genetic distances against geographic distances
Realized dispersal distance (D_{real})	Mean, median, or maximum distance travelled by reproducing individuals

N_F number of females in the cell

[a] Note that these are values used in the logistic growth equation; reproductive rate and carrying capacity within a given cell may exceed these values as number of offspring is determined probabilistically

[b] All outputted parameters are averaged across the last ten generations of each run

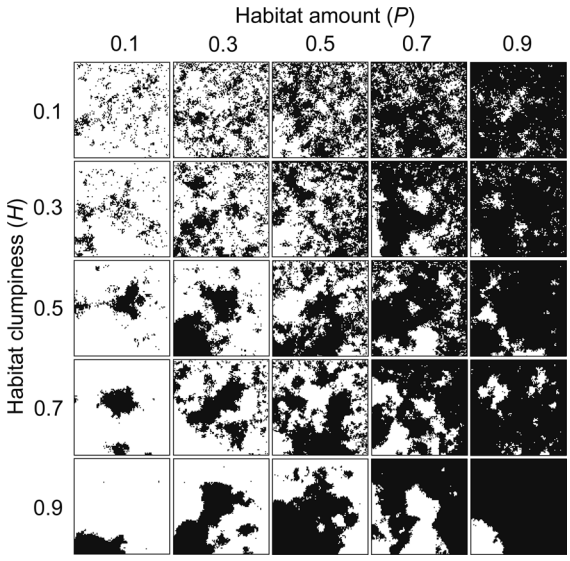

Fig. 2 An illustration of landscapes simulated under varying amounts (x-axis) and clumpiness (y-axis) of habitat. *Black* cells represent habitat and white cells represent matrix

inverse of fragmentation) of habitat using the midpoint displacement algorithm (Saupe 1988), a method commonly used to simulate variation in habitat configuration (With and King 1999; Flather and Bevers 2002; Neel et al. 2004; Cushman et al. 2012). This algorithm produces realistic-looking landscapes based on a specified proportion of habitat (P) and degree of habitat spatial autocorrelation, which is controlled by the index H ($H = -D/3$, where D is the fractal dimension). H ranges from zero (extremely scattered, i.e., fragmented, habitat) to 1 (extremely clumped habitat). We simulated five amounts of habitat ($P = 0.1, 0.3, 0.5, 0.7, 0.9$) and five levels of clumpiness ($H = 0.1, 0.3, 0.5, 0.7, 0.9$) in a full factorial design (Fig. 2). Landscapes were comprised of a 101×101 cell grid, where each cell was classified as habitat or non-habitat ("matrix"). Although we simulated landscape structure using discrete cells to facilitate model tractability, we emphasize that it is the properties of emergent habitat patches, not of habitat cells, that are expected to shape population genetic patterns.

Population setup

In this model, landscapes are initially populated with a large number of individuals (5000) to ensure that only inhospitable landscapes result in extinction. Coordinates of individuals are randomly assigned, and can fall in habitat or matrix, ensuring that average initial population densities within habitat are the same, regardless of the underlying landscape structure (average initial population density in habitat is $\sim 1/4$ the carrying capacity). Because individuals in matrix die without reproducing (discussed below), after the first generation, population sizes vary greatly depending on the amount of habitat in the landscape and are thereafter completely free to respond to underlying landscape structure and specified species parameters. Each individual is randomly assigned to one of two sexes, with equal probability. All individuals possess ten diploid loci that are each randomly assigned two of 20 alleles initially available per locus. These values have been shown to be adequate for accurately detecting genetic structure at fine spatial and temporal scales (Landguth et al. 2012).

Movement

All individuals disperse a single step. Step length (the number of cells across a continuous surface) is drawn from a negative exponential distribution with a mean equal to the specified average dispersal distance (D_{av}). This produces distributions of dispersal distances that are "fat-tailed," with more long-distance dispersers than expected under a Gaussian distribution (e.g., Kot et al. 1996; Chapman et al. 2007). Fat-tailed dispersal kernels are observed in many species (Okubo 1980; Turchin 1998) and their long-distance dispersers likely contribute disproportionately to the evolutionary dynamics of populations (Nathan 2006; Lowe 2010).

Under informed dispersal (ID), prior to movement, combinations of a random step direction and a negative exponentially distributed step length are drawn in an exploratory loop until a step into habitat results, in which case individuals settle at that location. If no habitat is located after 100 loops, the individual does not move. Thus, under ID, a step ending in matrix never takes place, although matrix can be traversed as long as settlement is in habitat and the drawn D_{av} is sufficiently large. Although dispersal to isolated patches of habitat is thus possible, the negative exponential dispersal kernel ensures that most movements occur within the individual's patch of origin (for example, the median movement distance observed across all our simulation treatments was 1.9 cells). In contrast to ID, under random dispersal (RD), step direction is drawn from a

uniform distribution, resulting in settlement locations that are unaffected by cell conditions (i.e., whether habitat or matrix). Thus, our approach was to simulate the extreme ends of a continuum of behaviors running from complete knowledge to complete ignorance of underlying landscape structure.

Grid boundaries are reflective for both types of dispersal and, to prevent aggregation of individuals along the edge of the grid, individuals located in an outer-most cell of the grid disperse by iteratively drawing step directions until a random draw results in a heading that is away from (or parallel to) the grid boundary. D_{av} was experimentally varied from 1 to 6 cells. This range produced nearly all the variation in genetic divergence observed in preliminary simulations using D_{av} values between 0.5 and 10 cells.

Reproduction

All females that have settled in habitat are given the opportunity to reproduce. If one or more adult males are located nearby (within a radius of two cells of the female), she mates with the closest one. Individuals that settle in matrix (allowable under RD) do not mate, which is equivalent to instant mortality given that individuals only live a single time step. Number of offspring is regulated by logistic growth and drawn from a Poisson distribution with a mean based on the growth rate (λ) and carrying capacity per cell (K), which are each set at two (Table 1). These values result in population sizes that never exceed our computational resources (<25,000 individuals) while allowing for adequate persistence of populations inhabiting unfavorable landscapes (i.e., when P and H are low). Offspring occupy the coordinates of their mothers and randomly inherit one allele per locus from each parent. Prior to inheritance, an allele can mutate with a probability of 0.0001 into any one of the other 19 available alleles, selected at random with equal probability (a k-allele mutation model). The effect of mutation on this study is likely very small given the short time scales simulated.

Death

Once reproduction has finished, all adults die and all juveniles become adults. A single non-overlapping generation consists of a completed cycle of movement to death.

Model output

At the end of each simulation run, we calculated emergent population size (N) and estimates of population connectivity and genetic structure across the landscape (Table 1). In this study, we took an individual-based, landscape approach (e.g., Cushman et al. 2006; Murphy et al. 2008) as opposed to a population-based approach (e.g., Coulon et al. 2012). Instead of defining and sampling distinct populations occupying discrete patches, we sampled individuals across a continuous (but heterogeneous) surface. This approach has the advantages that it does not presuppose the population spatial structure, and it can account for fine-scale, continuous spatial genetic structure that is not well captured in a population-based framework (Schwartz and McKelvey 2009). However, note that although we do not explicitly simulate or sample patches of habitat, discrete patches nonetheless emerge from the model and it is the properties of these patches (i.e., their size and isolation) that largely govern expected genetic effects (e.g., Fig. 1). We estimated genetic structure (the spatially non-random distribution of genetic variation) by calculating the correlation between genetic and geographical distances (i.e., the strength of isolation by distance) among individuals. We expected that isolation by distance among individuals would linearly increase with decreasing population connectivity at equilibrium. This is because genetic distance among spatially proximate individuals in the same "population" will remain low while genetic distance among spatially distant members of different "populations" will continuously increase with time. Thus, we expected that habitat loss and fragmentation would increase genetic distance per unit of geographic distance among individuals (e.g., Stow et al. 2001; Coulon et al. 2010). This is in contrast to the expected pattern of isolation by distance among populations, which can decrease over time if gene flow is highly restricted (Hutchison and Templeton 1999).

To estimate genetic structure, we randomly sampled 200 individuals after 1000 generations from each landscape (sampling all individuals was too computationally intensive) and estimated pairwise genetic distances among individuals by calculating the proportion of shared alleles averaged across loci (Dps; Bowcock et al. 1994). We estimated the correlation between genetic and geographic distance matrices

using Mantel tests performed using the vegan package in R (R Development Core Team 2012) and used Pearson's correlation coefficient, *r*, as our estimate of population structure. Mantel *r* is a sensitive predictor of genetic structure (Landguth et al. 2010), and is commonly used in individual-based studies (e.g., Peakall et al. 2003; Cushman et al. 2006). Note that we used Mantel *r* here to assess the relative strength of genetic structure across simulated treatments, not to test whether the null hypothesis of no genetic structure can be rejected; thus the observation that Mantel tests may sometimes possess low power to detect a true correlation with statistical significance (Legendre and Fortin 2010) should not affect our results.

Population connectivity is often defined as the rate of movement among populations as scaled by effective population size (i.e., *Nm*; Wright 1969). However, we were interested in comparing the independent effects of dispersal and population size on genetic structure among individuals. Thus we defined connectivity (or gene flow) here as simply the rate of gene movement (Mallet 2001). Because we wanted to measure this rate across a continuous landscape rather than among discrete patches (which are not defined in our landscapes), we calculated the average, median, and maximum distances moved by reproducing individuals (i.e., the realized dispersal distance of alleles or D_{real}) to approximate the rate of gene movement. Because we are only tracking reproducing individuals, D_{real} directly measures the movement of alleles. The farther that alleles tended to move under a given treatment, the more gene flow became possible among geographically isolated areas of the landscape. D_{real} is positively correlated with simulated (input) average dispersal distance (D_{av}; R_2 across all treatments is 0.926); thus, we performed all analyses for each D_{av} level separately such that all variation observed in D_{real} could be assumed to emerge from population interactions with the landscape. As an alternative approach, we also defined the rate of gene movement as the proportion of females in the sample that produced offspring; this assumes that gene flow is greater when a higher proportion of alleles are reproduced, and thus "dispersed," across generations. This metric produced relationships to landscape structure that were broadly similar to those using D_{real} (Appendix A in Supplementary material), suggesting that our results are somewhat robust to how we measure gene movement. To minimize effects of generation-specific stochasticity on parameters, we used average values of Mantel *r*, *N*, and D_{real} for the final 10 generations of each simulation.

Simulation experiments

Using a full factorial design we varied habitat amount (*P*; five levels), habitat clumpiness (*H*; five levels), average dispersal distance (D_{av}; six levels), and dispersal behavior (random or informed). In the case of informed dispersal, we also ran simulations with reduced habitat quality in edge habitat to evaluate the potential impacts of negative edge effects on the relative influence of habitat loss and fragmentation on genetic structure. In this treatment (ID + negative edge effects, or NEE), habitat cells adjacent to non-habitat cells (i.e., edge habitat) were assigned a lower carrying capacity ($K = 1.8$) than other habitat ($K = 2$). In all, we considered 450 treatment combinations. We performed 100 replicates of each treatment combination, each time using a different, randomly generated landscape, and each simulation ran for 1000 generations to allow for population sizes and genetic outcomes to stabilize. Because population structure cannot be measured in extinct populations, replicates that resulted in global extinction prior to the 1000th generation were discarded and additional runs were performed until 100 viable populations were obtained for each treatment combination.

Statistical analysis

Relative effects of habitat amount and configuration on genetic structure (I)

We determined the relative effects of two categorical predictors, habitat amount (*P*) and clumpiness (*H*), and their interaction ($P \times H$), on a single response, genetic structure (Mantel *r*), using analyses of variance (ANOVA; question I in Fig. 3). We repeated this analysis across six dispersal distances (D_{av}), two dispersal behaviors (random and informed), and with and without negative edge effects to explore how the relative effects of *P* and *H* on Mantel *r* depend on these species traits. We quantified relative effects using the percent sum of squares (%SS), which gives the proportion of variation in the data explained by a variable (Fletcher 2006). We focused on %SS rather than tests of significance because we were interested in

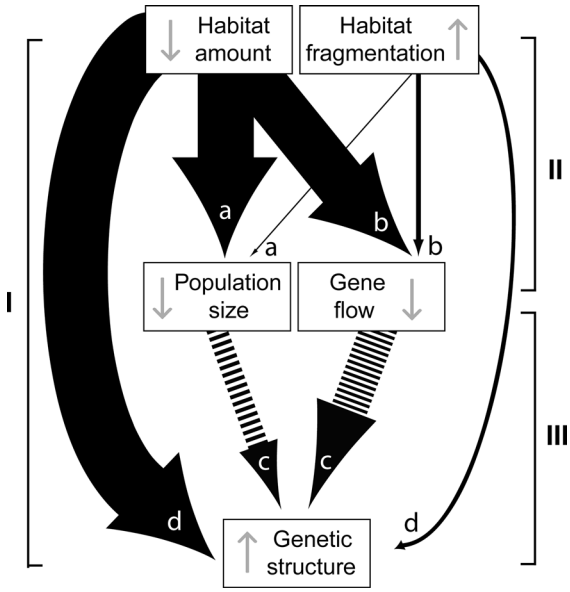

Fig. 3 Summary of relationships inferred in this study between landscape structure (habitat amount and fragmentation) and genetic structure via the effects of landscape structure on population dynamics (population size and gene flow). Numerals I–III partition three major sets of relationships (matching heading labels in the methods and results). *Small grey arrows* show the general directions of effects brought on by habitat loss and fragmentation. *Black arrows* indicate the polarity and relative size of distinct relationships. Relative thickness of *arrows* within a pair (pairs are labeled with *lower case letters*) represents quantitative differences in the importance of one relationship over another, where *arrow* widths are scaled to percent sum of squares values from ANOVAs, averaged across all analyses depicted in Figs. 3, 4, 5 and 6 for a given response variable. *Dashed arrows* indicate when the relative importance of relationships strongly depends on the conditions simulated

estimating relative effects and because the large number of replicate simulations rendered even small differences statistically significant. The car package in R was used for %SS.

Effects of landscape structure on population size and connectivity (II)

We next investigated the influence of the two components of landscape structure (P and H) on the population dynamics hypothesized to drive genetic structure (population size and gene flow; question II in Fig. 3). To do this, we performed two sets of ANOVAs across all treatments: (1) where total population size was regressed on P, H, and $P \times H$ and (2) where mean realized dispersal distance (D_{real}) was regressed on P, H, and $P \times H$. We assessed relative effects of the predictors on population dynamics using %SS.

Relative effects of population size and connectivity on genetic structure (III)

To investigate the relative effects of population size and connectivity on genetic structure (question III in Fig. 3), we fit (1) Mantel r to population size and (2) Mantel r to D_{real} for each treatment combination separately (450 analyses) using multiple linear regressions. A negative relationship between Mantel r and population size would suggest that reduced population size increased genetic structure; a negative relationship between Mantel r and D_{real} would suggest that reduced movement of alleles increased genetic structure. Relative effects were calculated by taking the difference between the absolute value of standardized regression coefficients for the two predictors ($RE = |\beta_{gene\ flow}| - |\beta_{population\ size}|$). Predictor variables were standardized by subtracting the mean from each value and dividing by the standard deviation. Positive REs indicate that gene flow is a better predictor of genetic structure; negative REs indicate that population size is a better predictor.

Results

Simulation experiments

Extinction did not occur under informed dispersal. Under random dispersal, 54 % of treatments resulted in some extinction and, overall, extinction occurred in 30 % of runs (see Appendix B in Supplementary material for viability proportions across treatments). Extinction became more likely with decreasing habitat amount, increasing fragmentation, and increasing average dispersal distance, showing that population viability is related to patch size in our model. Similarly, population densities were reduced in landscapes with sparse or fragmented habitat (Appendix C in Supplementary material). For example, in landscapes composed of 30 % habitat, highly fragmented landscapes ($H = 0.1$) supported average densities of 0.9 individuals per habitat cell under informed dispersal (or 0.2 under random dispersal), whereas highly clumped landscapes ($H = 0.9$, also at 30 % habitat) supported average densities of 1.8 (or 1.3

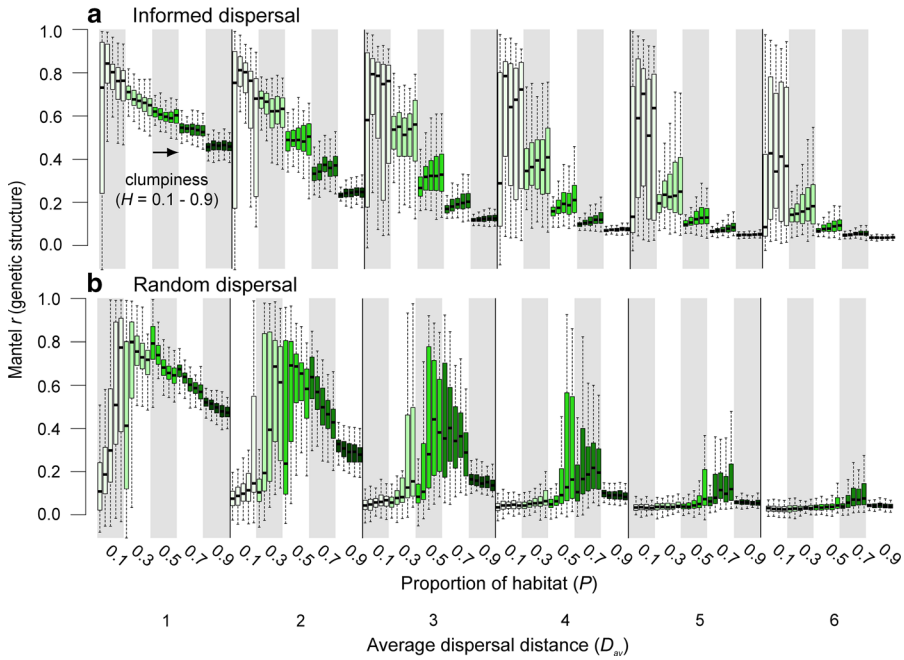

Fig. 4 Boxplots showing variation in Mantel r across a full factorial design of simulated treatments: increasing habitat amount, P; increasing habitat clumpiness, H; increasing average dispersal distance, D_{av}; and alternating dispersal behavior, informed versus random dispersal. Each box-and-whisker plot displays distributions in Mantel r for a particular treatment. **a** Informed dispersal (ID). **b** Random dispersal (RD). Increasing P is depicted by darkening color on the x-axis (to enhance readability, changing factors of P are also indicated by alternating *gray* and *white backgrounds*). Within each level of P, five bars represent increasing H, from left to right. *Boxes* extend from the 0.25–0.75 quartiles and whiskers extend to 1.5 times the interquartile range. *Horizontal lines* within boxes show median values. Outliers are excluded to enhance readability

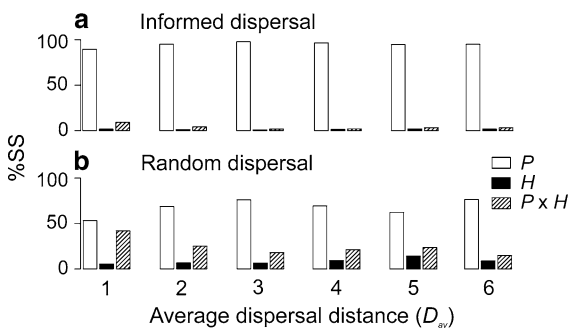

Fig. 5 The proportion of variation in Mantel r explained by habitat amount (P), habitat clumpiness (H), and their interaction ($P \times H$) are depicted using percent sum of squares (%SS) values. Values are shown for ANOVAs carried out for each dispersal behaviour—**a** informed and, **b** random—and for each average dispersal distance (D_{av}). Residual %SS (% sum of squares error) were 52, 41, 40, 43, 49, and 54 % for **a** (from $D_{av} = 1$–6) and 55, 58, 68, 77, 79, and 82 % for **b**

under RD). This shows that for both ID and RD there was a strong demographic penalty for inhabiting small patches. This is likely due to increased stochastic local extirpation in small habitat fragments, which may be exacerbated by higher per capita patch emigration due to a higher proportion of edge-dwelling individuals in small patches. The demographic cost to small fragments is even greater under RD where there is no restriction to emigration and where settlement in the matrix (allowed under RD) reduces the pool of potential immigrants by lowering population growth. The demographic penalty within small fragments is actually greater under RD than in the ID + NEE case where we explicitly added reduced habitat quality in edges. Under ID + NEE, extinction was only observed when both P and $H = 0.1$ or when $P = 0.3$, $H = 0.1$ and $D_{av} = 1$ (average extinction frequency in these treatments was 0.87).

Sub-selecting only viable populations did not appear to influence our conclusions about the effects of landscape treatments on genetic structure (Appendix D in Supplementary material). However, in two scenarios (when dispersal = random, P and $H = 0.1$, and $D_{av} = 5$ or 6) global extinction nearly

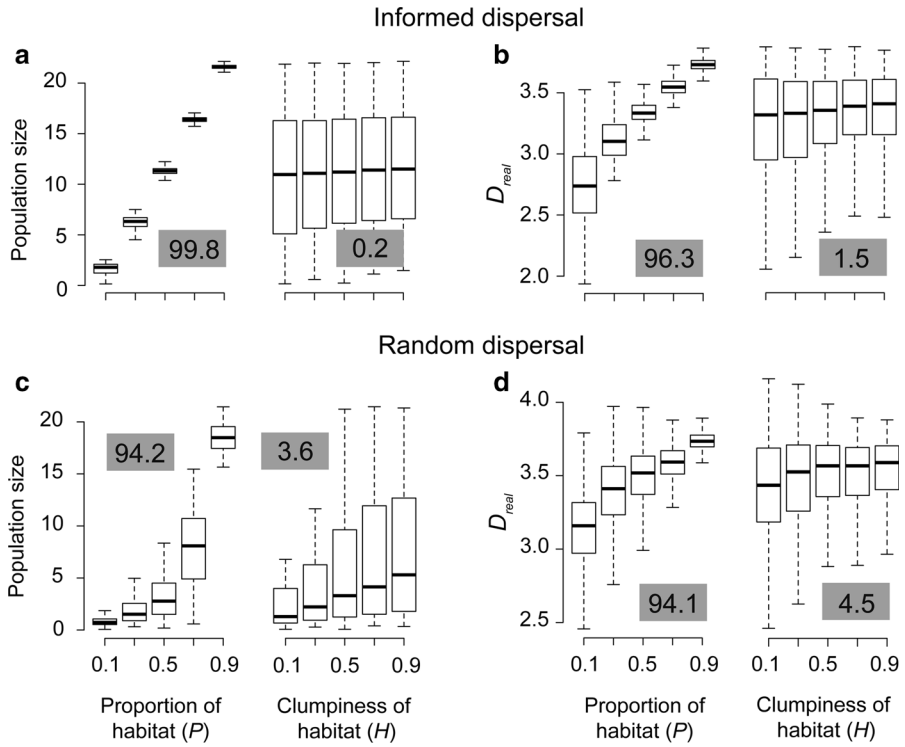

Fig. 6 Boxplots showing how emerging patterns of population size (N) and connectivity (D_{real}) vary with the amount (P) and clumpiness (H) of habitat. Panels **a** and **b** plot N and D_{real}, respectively, under informed dispersal; panels **c** and **d** plot N and D_{real}, respectively, under random dispersal. Percent sum of squares (%SS) values for the two predictors (P and H) of model outcomes (N and D_{real}) are given in gray boxes (note that $P \times H$ was also a predictor in each model; %SS values for this interaction term are not shown). For all plots, data are only shown for when average dispersal distance (D_{av}) = 4. Patterns are broadly similar across other dispersal distances, which are shown in Appendix I in Supplementary material. *Boxes* extend from the 0.25–0.75 quartiles and whiskers extend to 1.5 times the interquartile range. *Horizontal lines* within boxes show median values. Outliers are excluded to enhance readability

always occurred such that we could not obtain 100 viable replicates. These two treatment combinations were thus excluded from analysis.

Overall, we observed a broad range of demographic outcomes, with population sizes ranging from 12 to 22,364 individuals and population densities ranging from 0.01 to 2.4 individuals per cell of habitat (Appendix E in Supplementary material). Population size and Mantel r values were generally stable for a given landscape in the 500 generations prior to sampling (Appendix F in Supplementary material), indicating that sufficient time had passed to sample demographic and genetic outcomes. Casual analysis of data sampled at generation 500 yielded very similar results (not shown).

Relative effects of habitat amount and clumpiness on genetic structure (I)

Regardless of the average dispersal distance (D_{av}), dispersal behavior, or edge quality simulated, the amount of habitat in the landscape had a much larger influence on patterns of genetic structure than did habitat clumpiness (%SS values averaged across all ID and RD treatment combinations were 81.1 % for habitat amount and 4.9 % for habitat clumpiness; Fig. 3).

Under informed dispersal (ID), habitat amount had a steep negative relationship with genetic structure (Mantel r), as expected, whereas the relationship between Mantel r and clumpiness was slightly nega-

tive under low D_{av}, becoming relatively flat as D_{av} was increased (Fig. 4a; predicted values from ANOVAs are provided in Appendix G in Supplementary material). Also, variation in Mantel *r* tended to be greater at lower amounts of habitat, likely reflecting the larger range of demographic outcomes produced when landscapes contained little habitat; low habitat landscapes could support a single small subpopulation, resulting in low Mantel *r*, or several isolated subpopulations, resulting in high Mantel *r*. Note that we use the term "subpopulation" here loosely to refer to an emergent group of individuals that is locally distributed within (mostly) contiguous habitat and is (largely) isolated from other such groups. Not surprisingly, habitat-rich landscapes consistently supported well-connected populations, leading to universally low genetic structure. Under ID, %SS values ranged from 89.4 to 95.0 % for habitat amount and from 0.7 to 2.0 % for clumpiness (Fig. 5a), indicating that habitat amount is the main driver of genetic structure.

Although habitat amount was also the best predictor of genetic structure under random dispersal (RD; %SS ranged from 53.0 to 76.4 % for *P* and 5.3 to 14.3 % for *H*; Fig. 5b) the relationships were more complicated. First, the relationship between Mantel *r* and *P* was somewhat humped, with highest values of Mantel *r* occurring at intermediate amounts of habitat (Fig. 4b). The consistently small Mantel *r* values observed when *P* was high resulted from universal connectivity among subpopulations, as seen under ID. The relatively small values of Mantel *r* observed when *P* was low likely resulted from the high demographic cost of dispersal into the matrix imposed under RD. This cost rendered emergence of multiple subpopulations unlikely when habitat was scarce, resulting in landscapes that typically only produced a single subpopulation (usually occupying the landscape region with the highest density of habitat). Thus, under RD there was a "multiple subpopulation threshold," i.e., a threshold amount of habitat at which landscapes began to produce multiple subpopulations such that genetic structure could form. Structure (Mantel *r*) was consistently low when habitat was limited (in which case a single cohesive subpopulation typically formed) and when habitat was abundant (in which case a single cohesive global population usually formed), but ranged more widely when habitat amount was intermediate, i.e., was sufficiently high to result in multiple subpopulations, but not so high that connectivity always precluded differentiation.

Another characteristic of random dispersal was that this multiple subpopulation threshold shifted gradually to higher values of *P* as D_{av} was increased (from approximately $P = 0.1$ when $D_{av} = 1$ to $P = 0.7$ when $D_{av} = 6$; Fig. 4b). This is probably because the likelihood of leaving habitat and settling in matrix increased with rising dispersal distance (Murrell et al. 2002). Thus, larger amounts of habitat were required to produce multiple populations when D_{av} was high.

A third complication under RD was that the direction of the relationship between Mantel *r* and clumpiness switched, depending on the amount of habitat: the relationship tended to be positive when *P* was below the multiple subpopulation threshold and tended to be negative when *P* was above the threshold (Fig. 4b). This is seemingly because when *P* was so low as to make persistence challenging, increasing the clumpiness of habitat actually raised the probability that multiple subpopulations would form by reducing dispersal into the matrix (due to larger average patch sizes), thus strengthening recruitment. This occurrence of multiple subpopulations increased the probability that genetic structure would form. However, once landscapes contained enough habitat to consistently produce multiple subpopulations, raising clumpiness generally decreased genetic structure by consolidating habitat, and thus broadening gene flow.

Reducing carrying capacity in edge habitat (negative edge effects) produced results that were intermediate between RD and ID treatment results (Fig. H1 in Appendix H in Supplementary material). Adding NEE to ID reduced genetic structure when *P* and *H* were low (0.1). This is because when edge habitat dominates the landscape, NEE renders small patches even more difficult to inhabit such that a single unstructured subpopulation is the most common outcome, as under RD. For landscapes with more habitat (0.3 or greater), results become similar to those observed under strict ID, as the importance of edge habitat is reduced when patches are large. The relative effects of habitat loss and fragmentation were also similar to those observed under ID and RD treatments (Fig. H2), likely reflecting the fact that both habitat loss and fragmentation increase the edge to area ratio.

Effects of landscape structure on population size and connectivity (II)

Relationships between connectivity (i.e., realized dispersal distances, D_{real}) and landscape structure (P and H) and genetic structure (Mantel r) were nearly identical whether the mean or median D_{real} was used in analyses. In contrast to mean or median D_{real}, maximum D_{real} consistently displayed weak relationships with Mantel r (data not shown), indicating that maximum actual dispersal distance is here a poor measure of population connectivity across landscapes. We thus used mean (rather than maximum) D_{real} for all analyses.

Under ID, population size was almost completely a product of the amount of habitat in a landscape (%SS ranged from 99.0 to 99.8 % for P and from 0.1 to 0.8 % for H across values of D_{av}; Fig. 6a and Fig. I1a in Appendix I in Supplementary material). Clumpiness played a slightly larger role under RD (%SS = 0.2–23.7 % for H; Fig. 6b and Fig. I2a in Appendix I in Supplementary material), but habitat amount still dominated (%SS = 70.8–98.3 % for P). Results for ID + NEE were very similar to ID results (Fig. H3 in Appendix H in Supplementary material). In the case of RD, note that correlations between population size and habitat amount became smaller at lower values of P (R^2 averaged 0.915, 0.634, 0.377, and, 0.272 when $P \leq 0.9$, $P \leq 0.7$, $P \leq 0.5$, and $P \leq 0.3$ were analyzed, respectively), indicating that available habitat was increasingly unfilled as dispersal into matrix became more common.

The amount of habitat also best explained population connectivity across landscapes (Fig. 3). D_{real} was much better predicted by habitat amount (%SS = 64.4–98.3 % for P across all levels of D_{av} and dispersal behavior; Figs. 5c, d and Appendix I in Supplementary material) than by clumpiness (%SS = 0.2–24.6 % for H). In all cases, when a relationship existed between P or H and population size or D_{real}, it was positive (Fig. 6 and Appendix I in Supplementary material).

Relative effects of population size and connectivity on genetic structure (III)

Both population size and gene flow shaped genetic structure (Mantel r), but which of these dominated depended on landscape structure and species traits. Under ID, the relative effect (RE) of population size was greater than that of gene flow when habitat amount was low (Fig. 7). This is likely in part because the influence of genetic drift was strongest when population sizes were small (as observed when habitat was rare).

In contrast, under RD, D_{real} was generally a better predictor of genetic structure than was population size, particularly at intermediate amounts of habitat (when variation in Mantel r among replicates was greatest; Fig. 4b), although this pattern became less pronounced as D_{av} was increased (Fig. 7). D_{real} was a weaker predictor of genetic structure under more extreme values of P due to low variation in

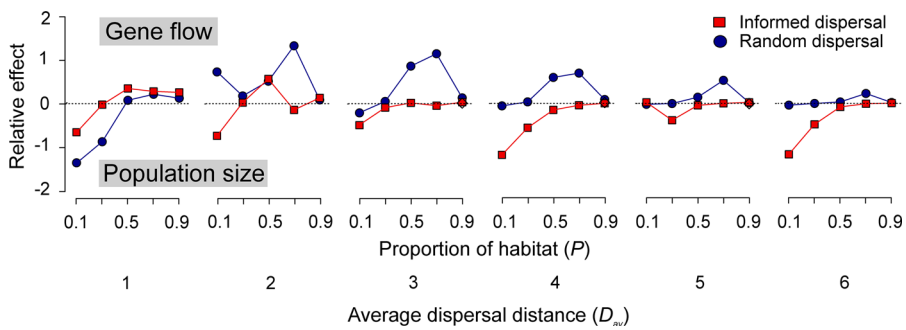

Fig. 7 Relative effect (RE) of population size and gene flow (D_{real}) on genetic structure (Mantel r) across increasing habitat amount (P) and increasing average dispersal distance (D_{av}). REs were calculated by taking the difference between the absolute values of standardized regression coefficients obtained from the equation, Mantel $r \sim P + H$, performed for each simulated treatment combination separately. At each level of P, REs were averaged across differing levels of clumpiness (H). Positive REs indicate treatment combinations under which gene flow was a stronger predictor of Mantel r; negative REs signal when population size was a stronger predictor. Coefficients for each treatment combination are shown in Appendix J in Supplementary material

connectivity among individuals when habitat was either rare (because poor recruitment under RD led to a preponderance of landscapes containing only a single small subpopulation) or abundant (due to the preponderance of landscapes containing a single global population). Population size was a weaker predictor of genetic structure under RD than under ID in part because when the potential influence of genetic drift was highest (at low population sizes), landscapes tended to only support a single subpopulation. Again, results for the ID + NEE treatments were intermediate between those observed under ID and RD (Fig. H4 in Appendix H in Supplementary material).

Standardized regression coefficients for the two predictors (N and D_{real}) of genetic structure (Mantel r) were usually negative, as expected if these emergent factors were driving patterns of genetic divergence (Appendix J in Supplementary material). An exception to this was when both habitat amount and clumpiness were low (P and $H \approx 0.1$), in which case the coefficients for population size (N) were often positive and large (Figs. J1A and J1B in Appendix J in Supplementary material). This is likely because only one or two subpopulations usually occurred in such inhospitable landscapes. If only one subpopulation occurred, Mantel r and population size were likely low. If two subpopulations occurred, this would have both greatly increased the probability of developing genetic divergence (i.e., a large Mantel r) and of expanding global population size (because two subpopulations will tend to contain more individuals than one), producing a positive relationship between N and Mantel r. In this scenario, N is not driving Mantel r, but is merely correlated with it. To minimize the influence of this phenomenon on our calculations of the relative effects of N and D_{real} on divergence, we removed treatment combinations resulting in positive coefficients for N when calculating relative effects (Fig. 7).

Discussion

How landscape structure creates genetic structure

Previous theoretical (Fahrig 1997, 1998; Jackson and Fahrig 2014) and empirical (Fahrig 2003) research suggests that the amount of habitat in a landscape is generally a stronger predictor of the abundance, persistence, and genetic diversity of populations than is the configuration of habitat in the landscape. However, because of the importance of population subdivision for creating genetic divergence (Wright 1931; Varvio et al. 1986; Slatkin 1987), there exists an intuition that configuration of habitat per se (i.e., independent of habitat amount) will be a stronger predictor in the case of genetic structure. Our results do not support this intuition: the amount of habitat in a landscape is also the biggest mediator of genetic divergence. Regardless of the dispersal capacity, dispersal behavior, or edge quality simulated here, landscape configuration effects on genetic differentiation are weak in comparison with habitat amount effects.

One reason that habitat amount has remained an underappreciated contributor to genetic structure is because the effects of demographic fluctuations on divergence, which are chiefly governed by the amount of habitat within landscapes, are often neglected in favor of a focus on dispersal effects (Balkenhol et al. 2009; Cushman et al. 2012). Here, we show that the influence of population size on genetic structure can be significant. This demographic effect is expected to be particularly strong when population sizes are small (as observed when habitat is rare) and when multiple subpopulations are present in the landscape (as observed under informed dispersal). The relative effects of population size on divergence (compared to the effects of gene flow) are reduced in large populations (e.g., when habitat amount is moderate or high) due to deceleration of genetic drift (Ezard and Travis 2006).

A second reason that the role of habitat amount in producing genetic structure has been overlooked stems from the conflation of habitat amount with habitat configuration in most studies (Fahrig 2003; Keyghobadi 2007). As habitat is lost from the landscape, discrete patches of habitat (and thus, subpopulations) become smaller and more distant from one another, even if the amount of fragmentation per se (such as the fractal dimension in our experiment) is held constant (Fig. 1). This increased isolation and decreased patch size in turn affects most measures of habitat configuration. Thus increased isolation of habitat (leading to decreased dispersal) reflects mainly habitat loss and not habitat fragmentation per se. This can be seen when inspecting relationships between genetic structure and landscape structure estimated using a variety

of landscape metrics (Appendix K in Supplementary material), which show that the predictive strength of a landscape metric is proportional to that metric's correlation with habitat amount. Because habitat amount has been shown to be strongly correlated with many metrics intended as measures of configuration (Neel et al. 2004) and is relatively easy to measure and interpret, there are good reasons to favor it over other generally less interpretable metrics. If, as in this study, the goal is to estimate the relative effects of habitat amount and configuration (here, fragmentation), this can only be done using a metric that is independent of habitat amount (e.g., CLUMPY).

Cushman et al. (2012) published a study with similar goals to ours, but arrived at the opposite conclusion: that genetic structure is largely governed by configuration. Although these authors also independently varied habitat amount and configuration and even included CLUMPY in their suite of configuration metrics analyzed, their conclusions are based on analysis of configuration metrics that are strongly correlated with habitat amount. For example, they observed that the metric GYRATE_AM (which describes the degree to which habitat is contiguous across a landscape) is a slightly better predictor of genetic differentiation than is habitat amount. However, given that habitat amount and GYRATE_AM are so strongly correlated (93 %; Neel et al. 2004), the main result could be interpreted to mean that nearly all the variation is explained by habitat amount, but that GYRATE_AM has a bit more explanatory power because it also accounts for the small effect of fragmentation per se. When the authors relied on an independent measure of fragmentation (CLUMPY), the effects of habitat amount were consistent with our results. Furthermore, the largest effect sizes were observed for metrics that are most strongly correlated with habitat amount, which is also consistent with our finding that habitat amount alone can explain much of the variation in genetic structure.

Although we simulated two ends of a continuum of dispersal behavior (i.e., complete ignorance or complete knowledge of the distribution of habitat), most animal species will likely fall somewhere between these two extremes. We expect that these species will generally exhibit patterns intermediate to those observed in the two simulated scenarios. Thus, a species with highly informed dispersal whose individuals may yet incur a small fitness cost by occasionally moving into or through the matrix (e.g., a small probability of dying in the matrix or a small decrease in fecundity after traversing habitat gaps) will likely exhibit slightly reduced persistence of subpopulations in low habitat landscapes, relative to those observed under ID. This would tend to reduce genetic structure in such landscapes by decreasing the probability that multiple isolated subpopulations will occur. The larger the demographic cost of crossing habitat boundaries, the narrower the range of habitat amounts that can result in genetic structure. In any case, regardless of where a species lies on the continuum of dispersal behaviors spanning from random to informed, our results suggest that realized patterns of genetic structure are much better predicted by the amount of habitat than by the configuration of habitat in a landscape.

As our simulations encompassed a wide array of demographic outcomes (see Appendix E in Supplementary material), our conclusions hold across a large demographic parameter space. Future work could investigate the impacts of altering reproductive rate per se—as an input parameter rather than an emergent variable—on these relationships, expanding our ability to predict the genetic impacts of landscape structure on a given species. We expect that increasing population densities beyond those observed here might decrease the spatial scale at which genetic structure can form and slow the rate of divergence due to larger effective population sizes; however, the relative impact of habitat amount and configuration on genetic structure would likely be unchanged.

Effects of habitat configuration on genetic structure

Although our results predict that habitat amount is the element of landscape structure largely driving patterns of genetic divergence, habitat configuration per se does have an influence. First, increasing habitat clumpiness can cause substantial increases in genetic structure when dispersal is random and when habitat amount is low. This is because larger average patch sizes result in the emergence of more subpopulations, which increases opportunities for genetic structure to form. However, this strong positive effect of clumpiness on genetic structure only occurs in the special case where habitat is scarce ($P \leq 0.3$), dispersal distance is low ($D_{av} \leq 2$), and dispersal has high

potential costs (random dispersal; e.g., see the far-left five bars in Fig. 4b). This suggests that, while the main conclusion of this study is that fragmentation per se overall adds little new information about population genetic structure, for poor moving species occupying landscapes that are strongly habitat-limited and hostile, incorporating fragmentation per se into landscape genetic models may significantly improve predictions of genetic structure. Thus, although the main goal of our study was to investigate relationships over a broad range of landscapes and dispersal abilities, future studies focused on exploring the genetic impacts of fragmentation in marginal landscapes would be particularly useful given their special conservation concern.

Once the threshold amount of habitat required for a landscape to support multiple subpopulations is reached, a general (but slight) negative relationship is observed between clumpiness and genetic structure, as long as D_{av} is low ($\leq$2 for RD and $\leq$1 for ID) and the amount of habitat is not too high (generally $\leq$0.3 for ID). This tendency for configuration to impose its greatest negative impact when habitat amount is low has been observed in other theoretical (Fahrig 1998; Flather and Bevers 2002) and empirical (Betts et al. 2007) studies and is here likely due to the existence of consistently high population connectivity once a certain amount of habitat is reached (an amount that increases with species dispersal distance), which dampens configuration effects on both gene flow and population size (Appendix L in Supplementary material). We note that configuration imposes a small independent effect on population size, similar to the size of its effect on gene flow (Appendix L in Supplementary material). This may in part result from increased emergence of subpopulations when habitat is increasingly clumped (Burkey 1997).

This study only focuses on fragmentation effects originating from the scatter of habitat and assumes that habitat gaps can be traversed if these gaps are small relative to the dispersal distance of an individual. However, some landscape features, such as rivers, urban structures, and gradients in elevation, may act as strong barriers to dispersal, regardless of their geographic extent, leading to short and long term effects on the evolution of populations (e.g., Cushman et al. 2006; Riley et al. 2006). If such features act as strong or complete barriers to a species, then fragmentation per se will restrict gene flow more than observed here. Also, our model assumes that all resources necessary for a species to live and reproduce are available within a habitat cell. If species (such as many amphibians) must disperse to different habitats to meet all life history requirements (e.g., breeding, nesting, or foraging; Pope et al. 2000), habitat fragmentation may impose additional reductions in population size, thus influencing genetic structure. However, we contend that for most species, it is not the number of pieces that habitat is divided into, but the distances among these pieces that contributes most to gene flow across landscapes. These distances are largely controlled by the amount of habitat in the landscape (Fig. 1).

Implications for research

Empirically testing our results would best be done using a multiple landscape-based approach. Sparse sampling of individuals from multiple independent landscapes rather than intense sampling of a single landscape (as is done in most patch-based studies) will minimize errors in inference due to pseudo-replication, which can result from treating genetic or landscape measures as independent when they are not (Legendre et al. 2002; Beale et al. 2010). A focal patch approach might also be useful in some cases, in which one could sample two isolated patches in replicate landscapes. The response variable would be genetic distance among comparable patches, correcting for the geographic distance between them. One problem with this approach however would be that if patches are too distant from one another, the expectation of isolation by distance disappears, and thus correcting for geographic distance becomes inappropriate. Employing multiple landscapes allows for more general extrapolation of inferred relationships beyond a particular landscape being investigated (Balkenhol et al. 2013). Dense sampling of a single landscape however may be more appropriate if the goal is to explore the genetic effects of particular features in a particular landscape.

Our results show that measures of habitat amount at the landscape scale can predict both patterns of gene flow and population size. Indeed, if the amount of genetic structure across a landscape can be adequately predicted by the amount of habitat, this suggests that when studying landscape effects on genetic structure at the landscape scale, rather than relying on a suite of

landscape metrics that may be difficult to interpret or to compare across studies (Haines-Young and Chopping 1996; Li and Wu 2004), and which are confounded with habitat amount to varying degrees (Neel et al. 2004), we can focus on a single, straightforward measure of landscape structure, the amount of habitat on a landscape, without losing much information. At the very least, habitat amount should be included in analyses by default, and other, more complex metrics should be added only if they are shown to have effects on genetic structure beyond those due to their correlations with habitat amount.

Measuring habitat amount at the patch scale (i.e., patch area) is not likely to have the explanatory value exhibited by habitat amount at the landscape scale (Fahrig 2013). Landscape-wide habitat amount is strongly correlated with both mean patch area (positive correlation) and mean patch isolation (negative correlation) measured at the patch scale (Fahrig 2003, 2013). To achieve the same explanatory power at the patch scale, patch area must be supplemented by additional metrics of local habitat isolation and regional patterns of habitat amount. Nevertheless, patch based studies can also benefit from the sampling of multiple independent landscapes (e.g., Balkenhol et al. 2013).

Studies that predict genetic structure using effective distances based on cost surfaces may also benefit by more explicitly incorporating measures of habitat amount or population size. For example, effective distances might be scaled to the proportion of habitat within a buffer around sampling points or by estimates of genetic diversity (Shirk and Cushman 2011) or the number of breeding individuals (Shirk and Cushman 2014) within pairs of sampled locations. Weckworth et al. (2013) included pairwise harmonic means of effective population sizes into partial Mantel tests along with genetic and resistance distance matrices, finding that correlations were strengthened when accounting for effective population size.

If habitat amount emerges as the best measure of landscape structure, improved predictions of genetic structure may be most effectively sought through development of more accurate and precise estimates of habitat amount for species under study. One way to improve habitat measures is through the application of habitat distribution modelling (e.g., Akcakaya and Atwood 1997). Such modelling can provide estimates of habitat amount that are tailored to the species and study area in question while potentially yielding habitat suitability estimates more accurate than those based on range maps derived from expert opinion and occurrence data (Bustamante and Seoane 2004). Habitat amount for a particular species can then be measured by summing occurrence probabilities across the landscape (Betts et al. 2007). In this way, measures of habitat amount can account for the continuum of habitat/matrix quality (e.g., negative edge effects) within a landscape.

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References

Akcakaya HR, Atwood JL (1997) A habitat based metapopulation model of the California gnatcatcher. Conserv Biol 11(2):422–434

Balkenhol N, Gugerli F, Cushman SA, Waits LP, Coulon A, Arntzen JW, Holderegger R, Wagner HH (2009) Identifying future research needs in landscape genetics: where to from here? Landscape Ecol 24(4):455–463

Balkenhol N, Pardini R, Cornelius C, Fernandes F, Sommer S (2013) Landscape-level comparison of genetic diversity and differentiation in a small mammal inhabiting different fragmented landscapes of the Brazilian Atlantic Forest. Conserv Genet 14(2):355–367

Beale CM, Lennon JJ, Yearsley JM, Brewer MJ, Elston DA (2010) Regression analysis of spatial data. Ecol Lett 13(2):246–264

Betts MG, Forbes GJ, Diamond AW (2007) Thresholds in songbird occurrence in relation to landscape structure. Conserv Biol 21(4):1046–1058

Betts MG, Fahrig L, Hadley AS, Halstead KE, Bowman J, Robinson WD, Wiens JA, Lindenmayer DB (2014) A species-centered approach for uncovering generalities in organism responses to habitat loss and fragmentation. Ecography 37(6):517–527

Bowcock AM, Ruiz-Linares A, Tomfohrde J, Minch E, Kidd JR, Cavalli-Sforza LL (1994) High resolution of human evolutionary trees with polymorphic microsatellites. Nature 368:455–457

Bowler DE, Benton TG (2005) Causes and consequences of animal dispersal strategies: relating individual behaviour to spatial dynamics. Biol Rev 80(2):205–225

Bruggeman DJ, Wiegand T, Fernandez N (2010) The relative effects of habitat loss and fragmentation on population genetic variation in the red-cockaded woodpecker (*Picoides borealis*). Mol Ecol 19(17):3679–3691

Bunn AG, Urban DL, Keitt TH (2000) Landscape connectivity: a conservation application of graph theory. J Environ Manag 59(4):265–278

Burkey TV (1997) Metapopulation extinction in fragmented landscapes: using bacteria and protozoa communities as model ecosystems. Am Nat 150(5):568–591

Bustamante J, Seoane J (2004) Predicting the distribution of four species of raptors (Aves: Accipitridae) in southern Spain: statistical models work better than existing maps. J Biogeogr 31:295–306

Chapman DS, Dytham C, Oxford GS (2007) Modelling population redistribution in a leaf beetle: an evaluation of alternative dispersal functions. J Anim Ecol 76(1):36–44

Conradt L, Bodsworth EJ, Roper TJ, Thomas CD (2000) Non-random dispersal in the butterfly *Maniola jurtina*: implications for metapopulation models. Proc R Soc B 267(1452):1505–1510

Coulon A, Fitzpatrick JW, Bowman R, Lovette IJ (2010) Effects of habitat fragmentation on effective dispersal of Florida scrub-jays. Conserv Biol 24(4):1080–1088

Coulon A, Fitzpatrick JW, Bowman R, Lovette IJ (2012) Mind the gap: genetic distance increases with habitat gap size in Florida scrub jays. Biol Lett 8(4):582–585

Cushman SA, McKelvey KS, Hayden J, Schwartz MK (2006) Gene flow in complex landscapes: testing multiple hypotheses with causal modeling. Am Nat 168(4):486–499

Cushman SA, Shirk A, Landguth EL (2012) Separating the effects of habitat area, fragmentation and matrix resistance on genetic differentiation in complex landscapes. Landscape Ecol 27(3):369–380

Cushman SA, Shirk AJ, Landguth EL (2013) Landscape genetics and limiting factors. Conserv Genet 14:263–274

Ezard THG, Travis JMJ (2006) The impact of habitat loss and fragmentation on genetic drift and fixation time. Oikos 114(2):367–375

Fahrig L (1997) Relative effects of habitat loss and fragmentation on population extinction. J Wildl Manag 61(3):603–610

Fahrig L (1998) When does fragmentation of breeding habitat affect population survival? Ecol Model 105(2–3):273–292

Fahrig L (2003) Effects of habitat fragmentation on biodiversity. Annu Rev Ecol Evol Syst 34:487–515

Fahrig L (2013) Rethinking patch size and isolation effects: the habitat amount hypothesis. J Biogeogr 40(9):1649–1663

Flather CH, Bevers M (2002) Patchy reaction-diffusion and population abundance: the relative importance of habitat amount and arrangement. Am Nat 159(1):40–56

Fletcher RJ Jr (2006) Emergent properties of conspecific attraction in fragmented landscapes. Am Nat 168(2):207–219

Funk WC, Blouin MS, Corn PS, Maxell BA, Pilliod DS, Amish S, Allendorf FW (2005) Population structure of Columbia spotted frogs (*Rana luteiventris*) is strongly affected by the landscape. Mol Ecol 14(2):483–496

Graves TA, Wasserman TN, Ribeiro MC, Landguth EL, Spear SF, Balkenhol N, Higgins CB, Fortin MJ, Cushman SA, Waits LP (2012) The influence of landscape characteristics and home-range size on the quantification of landscape-genetics relationships. Landscape Ecol 27(2):253–266

Grimm V, Railsback SF (2005) Individual-based modeling and ecology. Princeton University Press, Princeton

Gustafson EJ (1998) Quantifying landscape spatial pattern: what is the state of the art? Ecosystems 1(2):143–156

Gustafson EJ, Parker GR (1994) Using an index of habitat patch proximity for landscape design. Landsc Urban Plan 29(2–3):117–130

Haines-Young R, Chopping M (1996) Quantifying landscape structure: a review of landscape indices and their application to forested landscapes. Prog Phys Geogr 20(4):418–445

Holderegger R, Wagner HH (2008) Landscape genetics. Bioscience 58(3):199–207

Honnay O, Coart E, Butaye J, Adriaens D, Van Glabeke S, Roldan-Ruiz I (2006) Low impact of present and historical landscape configuration on the genetics of fragmented *Anthyllis vulneraria* populations. Biol Conserv 127(4):411–419

Hutchison DW, Templeton AR (1999) Correlation of pairwise genetic and geographic distance measures: inferring the relative influences of gene flow and drift on the distribution of genetic variability. Evolution 53(6):1898–1914

Jackson HB, Fahrig L (2012) What size is a biologically relevant landscape? Landscape Ecol 27(7):929–941

Jackson ND, Fahrig L (2014) Landscape context affects genetic diversity at a much larger spatial extent than population abundance. Ecology 95(4):871–881

Kanuch P, Jarcuska B, Schlosserova D, Sliacka A, Paule L, Kristin A (2012) Landscape configuration determines gene flow and phenotype in a flightless forest-edge ground-dwelling bush-cricket *Pholidoptera griseoaptera*. Evol Ecol 26(6):1331–1343

Keyghobadi N (2007) The genetic implications of habitat fragmentation for animals. Can J Zool 85(10):1049–1064

Kimura M, Ohta T (1969) The average number of generations until fixation of a mutant gene in a finite population. Genetics 61(3):763–771

Kot M, Lewis MA, van den Driessche P (1996) Dispersal data and the spread of invading organisms. Ecology 77:2027–2042

Kozakiewicz M (1995) Resource tracking in space and time. In: Hansson L, Fahrig L, Merriam G (eds) Mosaic landscapes and ecological processes. Chapman & Hall, London, pp 136–148

Landguth EL, Cushman SA, Schwartz MK, McKelvey KS, Murphy M, Luikart G (2010) Quantifying the lag time to detect barriers in landscape genetics. Mol Ecol 19(19):4179–4191

Landguth EL, Fedy BC, Oyler-McCance SJ, Garey GL, Emel SL, Mumma M, Wagner HH, Fortin MJ, Cushman SA (2012) Effects of sample size, number of markers, and allelic richness on the detection of spatial genetic pattern. Mol Ecol Resour 12(2):276–284

Lange R, Diekoetter T, Schiffmann LA, Wolters V, Durka W (2012) Matrix quality and habitat configuration interactively determine functional connectivity in a widespread bush cricket at a small spatial scale. Landscape Ecol 27(3):381–392

Legendre P, Fortin MJ (2010) Comparison of the Mantel test and alternative approaches for detecting complex multivariate relationships in the spatial analysis of genetic data. Mol Ecol Resour 10(5):831–844

Legendre P, Dale MRT, Fortin MJ, Gurevitch J, Hohn M, Myers D (2002) The consequences of spatial structure for the design and analysis of ecological field surveys. Ecography 25(5):601–615

Li HB, Wu JG (2004) Use and misuse of landscape indices. Landscape Ecol 19(4):389–399

Lowe WH (2010) Explaining long-distance dispersal: effects of dispersal distance on survival and growth in a stream salamander. Ecology 91(10):3008–3015

Mallet J (2001) Gene flow. In: Woiwod IP, Reynolds DR, Thomas CD (eds) Insect movement: mechanisms and consequences. CAB International, Wallingford, pp 337–360

Mapelli FJ, Mora MS, Mirol PM, Kittlein MJ (2012) Population structure and landscape genetics in the endangered subterranean rodent *Ctenomys porteousi*. Conserv Genet 13(1):165–181

McGarigal K, Cushman SA, Neel MC, Ene E (2002) FRAGSTATS: spatial pattern analysis program for categorical maps. University of Massachusetts, Amherst. http://www.umass.edu/landeco/research/fragstats/fragstats.html

McRae BH (2006) Isolation by resistance. Evolution 60(8):1551–1561

McRae BH, Beier P (2007) Circuit theory predicts gene flow in plant and animal populations. Proc Natl Acad Sci USA 104(50):19885–19890

Millette KL, Keyghobadi N (2015) The relative influence of habitat amount and configuration on genetic structure across multiple spatial scales. Ecol Evol 5(1):73–86

Murphy MA, Evans JS, Cushman SA, Storfer A (2008) Representing genetic variation as continuous surfaces: an approach for identifying spatial dependency in landscape genetic studies. Ecography 31(6):685–697

Murrell DJ, Travis JMJ, Dytham C (2002) The evolution of dispersal distance in spatially-structured populations. Oikos 97(2):229–236

Nathan R (2006) Long-distance dispersal of plants. Science 313(5788):786–788

Neel MC, McGarigal K, Cushman SA (2004) Behavior of class-level landscape metrics across gradients of class aggregation and area. Landscape Ecol 19(4):435–455

Nichols RA, Hewitt GM (1994) The genetic consequences of long-distance dispersal during colonization. Heredity 72:312–317

Okubo A (1980) Diffusion and ecological problems: mathematical models. Springer, New York

Peakall R, Ruibal M, Lindenmayer DB (2003) Spatial autocorrelation analysis offers new insights into gene flow in the Australian bush rat *Rattus fuscipes*. Evolution 57(5):1182–1195

Pope SE, Fahrig L, Merriam NG (2000) Landscape complementation and metapopulation effects on leopard frog populations. Ecology 81(9):2498–2508

R Development Core Team (2012) R: a language and environment for statistical computing. R Foundation for Statistical Computing. Vienna, Austria. http://www.R-project.org

Reed JM, Dobson AP (1993) Behavioural constraints and conservation biology—conspecific attraction and recruitment. Trends Ecol Evol 8(7):253–256

Riley SPD, Pollinger JP, Sauvajot RM, York EC, Bromley C, Fuller TK, Wayne RK (2006) A southern California freeway is a physical and social barrier to gene flow in carnivores. Mol Ecol 15(7):1733–1741

Saupe D (1988) Algorithms for random fractals. In: Peitgen H-O, Saupe D (eds) The science of fractal images. Springer-Verlag, New York, pp 71–113

Schmidt T, Arens P, Smulders MJM, Billeter R, Liira J, Augenstein I, Durka W (2009) Effects of landscape structure on genetic diversity of *Geum urbanum* L. populations in agricultural landscapes. Flora 204(7):549–559

Schumaker NH (1996) Using landscape indices to predict habitat connectivity. Ecology 77(4):1210–1225

Schwartz M, McKelvey K (2009) Why sampling scheme matters: the effect of sampling scheme on landscape genetic results. Conserv Genet 10(2):441–452

Shirk AJ, Cushman SA (2011) sGD: software for estimating spatially explicit indices of genetic diversity. Mol Ecol Resour 11(5):922–934

Shirk AJ, Cushman SA (2014) Spatially-explicit estimation of Wright's neighborhood size in continuous populations. Front Ecol Evol 2(62):1–12

Slatkin M (1987) Gene flow and the geographic structure of natural populations. Science 236(4803):787–792

Stow AJ, Sunnucks P, Briscoe DA, Gardner MG (2001) The impact of habitat fragmentation on dispersal of Cunningham's skink (*Egernia cunninghami*): evidence from allelic and genotypic analyses of microsatellites. Mol Ecol 10(4):867–878

Telles MPD, Dobrovolski R, Souza KDE, Lima JD, Collevatti RG, Soares TN, Chaves LJ, Diniz JAF (2014) Disentangling landscape effects on population genetic structure of a Neotropical savanna tree. Nat Conserv 12(1):65–70

Templeton AR, Shaw K, Routman E, Davis SK (1990) The genetic consequences of habitat fragmentation. Ann Mo Bot Gard 77(1):13–27

Turchin P (1998) Quantitative analysis of movement: measuring and modeling population redistribution in animals and plants. Sinauer Associates Inc, Sunderland

van Strien MJ, Keller D, Holderegger R, Ghazoul J, Kienast F, Bolliger J (2014) Landscape genetics as a tool for conservation planning: predicting the effects of landscape change on gene flow. Ecol Appl 24(2):327–339

Varvio SL, Chakraborty R, Nei M (1986) Genetic variation in subdivided populations and conservation genetics. Heredity 57:189–198

Weckworth BV, Musiani M, DeCesare NJ, McDevitt AD, Hebblewhite M, Mariani S (2013) Preferred habitat and effective population size drive landscape genetic patterns in an endangered species. Proc R Soc B 280(1769):20131756

With KA, King AW (1999) Extinction thresholds for species in fractal landscapes. Conserv Biol 13(2):314–326

Wright S (1931) Evolution in Mendelian populations. Genetics 16:97–159

Wright S (1969) Evolution and the genetics of populations, vol. 2. The theory of gene frequencies. University of Chicago Press, Chicago

Young A, Boyle T, Brown T (1996) The population genetic consequences of habitat fragmentation for plants. Trends Ecol Evol 11:413–418