POPULATION GENETIC CONSEQUENCES OF DIFFERENT DISPERSAL-DISTANCE DISTRIBUTIONS IN A CONTINUOUS LANDSCAPE.

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Abstract

Dispersal is a pervasive life history trait, yet there is relatively little understanding of the evolutionary forces affecting variation in dispersal distances or the consequences of that variation. In natural populations, the effects of dispersal are often confounded by other factors including local selection or population history. To isolate the population genetic effects of different dispersal distributions in continuous space, modeling approaches must be used. We developed a cellular automata model and used it to quantify spatial genetic variation for 18 different dispersal distance distributions. We developed and used a measure of spatial autocovariance to quantify the population genetic consequences of dispersal. We found that population genetic structure is persistent (for thousands of generations) and that landscape size and initial distribution of individuals may affect the genetic structure by increasing the importance of demographic stochasticity early in the population history. Our results indicate that the relationship between dispersal and resultant genetic structure is scale-dependent. As expected, a few long-distance dispersal events diminished patch size (clusters of individuals of the same genotype) at the landscape scale. We expected that philopatry would result in increased patch formation at local and neighborhood scales and for some dispersal distributions this expectation was met. However, some combinations of philopatry and dispersal yielded surprisingly low levels of local patch formation.

Population genetic consequences of different dispersal-distance distributions in a continuous landscape

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Running head: Spatial genetic structure

Abstract

Dispersal is a pervasive life history trait, yet there is relatively little understanding of the evolutionary forces affecting variation in dispersal distances or the consequences of that variation. In natural populations, the effects of dispersal are often confounded by other factors including local selection or population history. To isolate the population genetic effects of different dispersal distributions in continuous space, modeling approaches must be used. We developed a cellular automata model and used it to quantify spatial genetic variation for 18 different dispersal distance distributions. We developed and used a measure of spatial autocovariance to quantify the population genetic consequences of dispersal. We found that population genetic structure is persistent (for thousands of generations) and that landscape size and initial distribution of individuals may affect the genetic structure by increasing the importance of demographic stochasticity early in the population history. Our results indicate that the relationship between dispersal and resultant genetic structure is scale-dependent. As expected, a few long-distance dispersal events diminished patch size (clusters of individuals of the same genotype) at the landscape scale. We expected that philopatry would result in increased patch formation at local and neighborhood scales and for some dispersal distributions this expectation was met. However, some combinations of philopatry and dispersal yielded surprisingly low levels of local patch formation.

Introduction

In recent years, there has been a congruence of interest in dispersal from the separate fields of population ecology and population genetics. Population ecologists have become increasingly aware of the importance of natal dispersal in metapopulation (Hanski and Gilpin1997, and references therein) and range dynamics (Lewis 1997), and population geneticists continue to be fascinated by the fundamental role of dispersal and gene flow in affecting the geographic distribution of genetic variability. Of all the fundamental life history processes that affect population biology, dispersal is probably the most pervasive and least understood. A great deal of theory has been developed to predict the proportion of offspring that disperse (e.g., Johnson and Gaines 1990; Hamilton and May 1977). However, given that some offspring disperse, there is relatively little understanding of the evolutionary forces affecting variation in dispersal distances or the consequences of that variation (e.g., Ezoe 1998; McCarthy 1996; Pärt 1990; Payne 1991).

Given the difficulty of measuring dispersal distance distributions directly (Barrowclough 1978; Koenig et al. 1996) indirect methods have often been the only means available. These have been limited largely to the quantification and statistical evaluation of the spatial patterns of genetic variation. The distribution of this genetic variability varies geographically in response to the effects of drift, vicariance, selection, and dispersal (Nürnberger and Harrison 1995; Templeton 1998). Population geneticists have applied a steadily increasing suite of tools to the analysis of genetic population structure, and a number of methods for inferring patterns of gene flow (and by extension

dispersal) from molecular data have been developed (see reviews in Neigel 1997; Avise 1994; Hillis and Moritz 1990). The use of selectively neutral genetic markers and the development of statistical models for evaluating genetic data (e.g., Beerli and Felsenstein 1999; Beerli 1998; Rousset and Raymond 1997) offer the prospect of disentangling the evolutionary forces that generate population genetic structure.

Because the spatial distribution of neutral molecular markers has the potential to provide estimates of the frequency and distances of dispersal (Neigel et al. 1991; Bohonok 1999), ecologists have begun to show considerable interest in spatial genetic population structure. Despite marked improvements in the methodology, the effects of history (e.g., Nürnberger and Harrison 1995; Bowen and Avise 1990; Templeton et al. 1995) and environmental variation (e.g., Koehn et al. 1976) often confound interpretations of genetic structure in nature as indirect measures of dispersal. To critically evaluate the link between dispersal behavior and population structure free of these confounding factors, it is necessary to adopt a modeling approach.

Such an approach would be most efficient and general if the dispersal process and its effects on structure could be modeled analytically or with accurate approximations. However, models incorporating sufficient structure to address complex questions are intractable analytically and there is increasing evidence (Hiebeler, in press) that approaches such as the "mean field" approximation of spatial processes can yield very misleading conclusions about spatial pattern. We therefore use simulation to investigate the dependence of genetic spatial structure on dispersal behavior.

A large body of theoretical work has indicated that spatial distributions of genetic

variation should differ from random or uniform distributions (Wright 1943; Felsenstein 1975; 1976) and many empirical studies of spatial structure support theoretical predictions (e.g., Slatkin 1985; 1987; Bradshaw 1984). A general result of these investigations is that neighborhood size is inversely correlated with the degree of spatial structure.

Many of the theoretical investigations have relied solely on normal distributions of dispersal distance in which neighborhood size is allowed to vary (e.g., Sokal and Wartenberg 1989; Sokal et al. 1989; Epperson 1993; Kawata 1995; 1997). Ibrahim et al. (1996) examined the impact of three models of dispersal (stepping-stone, normal and leptokurtic) on spatial genetic structure in expanding populations. In their model, the landscape was divided into a number of demes and the ultimate measure of population genetic structure was correlation between the allele frequencies in pairs of populations as a function of the number of demes separating them. Not surprisingly, they found that patchiness was more pronounced when the dispersal distribution was leptokurtic because rare long-distance migration led to the establishment of "pocket populations" in advance of the main invasion front. However, because they were interested in population expansion, it is possible that their results reflect founder effects as well as dispersal distribution effects on subsequent population genetic structure.

Here, we develop a model to explicitly examine the effects of different dispersal distance distributions on the spatial population genetic structure. In our model reproduction is asexual and dispersal is entirely passive; individuals have no prior assessment of the patch to which they ultimately disperse. Density-dependent survival

probabilities impose the limit to the number of individuals at any one site. We assess population genetic structure at equilibrium conditions (i.e. after the population has reached its carrying capacity and spatial population genetic structure as we measure it is no longer noticeably changing).

Different measures for spatial structure have been developed and include join-count statistics for continuous populations and Moran's "I" statistics for discrete subpopulations (reviewed in Epperson 1993). To analyze our results we developed a spatially continuous measure of autocovariance, thus avoiding the need to artificially divide our landscape into demes. Because we have haploid data, our measure of genetic structure is inversely related to the join-count statistics (Epperson 1993).

Our specific goal was to evaluate a number of different dispersal distance distributions and to assess the consequences of changes in the shape and variance of those functions for population genetic structure. In this paper we limit ourselves to the neutral case in which the genetic variation modeled has no selective effect on life history variation.

Methods

Model structure

Our model was developed in Matlab 5.2 and subsequently converted to C in order to maximize the efficiency of running simulations and evaluating results. We conducted extensive preliminary investigations to settle upon a "standard model" to evaluate basic methodological concerns.

A three dimensional matrix was used to record the number of individuals that were present at each location (i,j) within a two-dimensional landscape for each state (k) where the state is a combination of genotype and age class. Individuals were assigned to one of two age classes (juvenile or adult) and to one of three haplotypes; this resulted in six different state categories. The landscape for our standard model (to be described later in this section) is a square homogeneous environment of size 100 x 100. The initial choice of size was somewhat arbitrary, but chosen small enough to avoid excessively long run times. We later tested the effects of landscape size (see Methodological concerns). Each location or site can support more than one individual, though density-dependent survival eventually becomes limiting (see below).

The initial population consisted of 1,000 adults with equal frequencies for each of the three haplotypes. For our standard model, the initial population was seeded in the middle quarter of the landscape, i.e. a 50 x 50 area at the center of the landscape. The starting location for each individual was randomly selected within this region.

Time was indexed by generation, and the biological processes of reproduction, dispersal and survival were sequentially simulated each generation. The following were assumed about reproduction: 1) only adults reproduce, 2) reproduction is asexual, 3) fecundity is independent of density and genotype, and 4) offspring are of the same haplotype as the parent. The fecundity (number of offspring per adult) was the closest non-negative integer to a random draw from a normal distribution with a mean of 15 and standard deviation of 7.5.

During dispersal, each juvenile chose one of three behaviors: juveniles could "stay

at home", they could disperse a short distance or they could disperse a longer distance. If a juvenile dispersed, a new location was randomly selected in the landscape. The dispersal location was selected from a bivariate normal distribution centered on the current location with mean of zero and a predetermined variance (σ_S^2 for short distance dispersal or σ_L^2 for long distance dispersal). We used absorbing boundaries, i.e. if the new location was outside of the 100 x 100 landscape, those individuals were lost from the population. For the standard model we used a dispersal distribution with 50% stayers, 40% dispersing a short distance with standard deviation of $\sigma_S = 0.95$, and 10% dispersing a long distance with standard deviation of $\sigma_L = 2.53$. These proportions and variances (including the stayers) resulted in an overall variance of 1 for the dispersal distances (See Appendix I for calculation of overall variance).

The last biological process in the model was survival to the next generation. We incorporated density dependence in this part of the life-cycle with the following equation:

$$p_r = p_0\left(\frac{2}{1+e^{0.05n}}\right)$$

where p_r = realized survival probability, p_0 = survival probability in the absence of density dependence, and n = the number of individuals at the location. The density independent probability of survival, p_0, did not vary with genotype or age class and was fixed at 0.75 in the standard model. The realized survival probability was therefore a monotonically decreasing function of n. The density dependence was calibrated so that p_r dropped to 0.5 at a density of n=15. Iteration of the standard model resulted in

asymptotic population growth; the carrying capacity for the landscape was reached within the first 100 generations. All simulations reported here are for at least 1000 generations, well beyond the period required to reach the carrying capacity.

Shapes of dispersal functions

It is widely hypothesized that a few long distance dispersers may have a strong effect on spatial patterns (e.g., Ibrahim et al. 1995, Lewis 1997). The distributions for the short- and long-distance dispersers were each chosen to be bivariate normal, though, of course, the three-component mixture is not a normal distribution. Changing the proportions of stayers, and of short and long dispersal distances simultaneously affects the shape and variance of the overall dispersal-distance distribution. To isolate whether any observed effects on genetic structure were merely due to changes in dispersal variance we also simulated dispersal using distributions with equal overall variances but different shapes (Table 1, Figure 1).

Mechanistically, the shape is determined by a two-component dispersal strategy. The first component is the movement strategy; individuals move with probability 0.5 or 1.0. The first movement strategy includes stayers (st) while the latter does not include stayers (ns). The second component is a combination of the proportions and distances moved by the juveniles that disperse. For the distance strategy there are three alternatives: all dispersing juveniles move a short distance (S), many move a short distance and some move a long distance (SL), or most move a short distance and a few move a very long distance (SVL). Different combinations of the two components result in six different shapes for the dispersal distance distributions (Figure 1). Although the

shapes are also influenced by the overall variance, for a given variance, the shapes can be summarized by the parameters of the two-component dispersal strategy alone. Throughout the remainder of the text the "shapes" or dispersal strategies will be referred to by the code for their dispersal parameters in Table 1.

We calculated mean dispersal distance (Table 1) for each distribution using the formulae in Appendix I. The relationship between mean dispersal distance and overall variance of the dispersal distance distribution is sensitive to the shape of the dispersal strategy (Figure 2). At the extremes, when all individuals move a short distance (nsS) there is a large effect of variance on mean dispersal distance and conversely, there is very little effect of variance when the strategy includes stayers and very long distance dispersers (stSVL).

Analysis methodology

One of the assumptions of our model is that space is continuous. Because we have no basis upon which to divide our landscape into subpopulations, the usual F_{ST} measure of correlation (Wright 1921) does not apply. Therefore, to analyze our results we developed a spatially continuous measure of autocovariance, which is derived from the usual calculation of covariance for a binary data set:

$$Cov(x,y) = P\{x=1, y=1\} - P\{x=1\}P\{y=1\}.$$

Biologically, we wanted to describe the probability that two individuals separated by a distance d would have the same genotype after subtracting the probability it would happen randomly. This is given by

$$P\{x=y \mid d\} - \sum_{g} P\{x=g\}^2$$

where g is the index for genotypes. We then plotted this covariance versus d to display the "structure" of the autocovariance.

These "structure" plots have a positive y-intercept (y-intercept is the probability that individuals within a site have the same genotype) followed by an approximately exponential decay as distance between sites increases (Figure 3). To compare structure plots in a quantitative manner, we summarized the plots by log transforming the y values and then fitting a straight line through the new coordinates. To estimate the slope of the structure plot, we used only the first six values of d, excluding 0, and thus including distance measures from 1 to 7. In analyzing structure plots, we relied on the slope, as well as M_0 (the corrected probability of a matching genotype within a site) and M_1 (the corrected probability of a matching genotype in the adjacent eight cells, i.e. the cells at distance 1).

Methodological issues

While developing the model, we addressed the following methodological concerns: 1) What are the consequences of landscape size? 2) Are there edge effects? i.e. is there a difference in the distribution of individuals along the edges of the landscape when compared with the central portions of the landscape? 3) What effects do fecundity parameters (mean and variance) have on population genetic structure? 4) How stable is the genetic structure? That is, does it change over large numbers of generations? 5) What consequences does the distribution of the initial population have for eventual population structure? The first two questions are largely related to potential artifacts of

model structure; the latter three are linked to biological processes.

To address the potential effect of landscape size we ran the standard model 5 times for each of three different landscape sizes: 100 x 100, 200 x 200, and 300 x 300. Each simulation was run for a period of 1000 generations (the number of generations was chosen on the basis of long runs for two dispersal distributions – see results below). Analysis of variance was used to test for an effect of landscape size on the three structure plot parameters: M_0, M_1 and slope. We used a Tukey test for multiple comparisons among the three different landscape sizes.

Additionally, we used these results to address edge effects by comparing the adult abundance among sites with different numbers of neighboring sites: corners, edges, and interior sites with 4, 6 and 9 neighboring sites respectively, including the site itself. Mean abundance was calculated for each site type for each of the 15 simulations; single factor ANOVA tested for a significant effect of site type on mean number of adults per site.

We predicted that an increase in mean fecundity would reduce the patchiness of the resulting population genetic structure. This could be expressed as a smaller value for M_0 and M_1 or as a more rapid decay in the structure plot and therefore a more negative slope. Similarly, we predicted that an increase in variance of fecundity would lead to increased patchiness with higher values for M_0 and M_1 and a smaller absolute value for the slope. To test these ideas we compared five replicates of three model simulations: the standard model and two other models in which fecundity parameters were manipulated. The mean and standard deviations for fecundity were as follows: the standard model (μ = 15, σ^2 = 7.5), increased mean (μ = 25, σ^2 = 7.5) and increased variance (μ =15, σ^2 = 15).

Summary data were compared using single factor ANOVA. All parameters other than mean and variance of fecundity were as described in the standard model.

To address the question of long-term stability we first simulated two of the dispersal distributions for 10,000 generations on a 100 x 100 lattice. The dispersal parameters were $P_{stay} = 0.5$, $P_{short} = 0.5$, $P_{long} = 0$, $\sigma_S = 1.41$, $\sigma_L = 0$ for the first run (stS with overall variance of 1.0, as in Table 1) and $P_{stay} = 0$, $P_{short} = 0.98$, $P_{long} = 0.02$, $\sigma_S = 0.95$, $\sigma_L = 7.48$ for the second run (i.e. nsSVL with an overall variance of 2.0). We generated a time series of structure plots and graphed the corresponding parameters (M_0, M_1, and slope) as a function of time. The time at which they no longer changed (by visual inspection of the plots) was designated as the cutoff for comparing the model simulation results for the 18 different dispersal distributions.

In order to evaluate the concern about the geography of founding populations, we ran the standard model 5 times with the following initial distributions of adults: 1) randomly seeded in the middle (50 x 50) of the landscape, 2) randomly seeded in one corner of the landscape where the corner is one fourth of the entire landscape and 3) randomly seeded over the entire landscape. The first two starting configurations mimic a biological situation in which range expansion occurs. In the former case, range expansion can occur in all directions while in the latter case expansion can proceed in only two directions because of absorbing boundaries. The final case addresses a biological scenario in which many individuals have successfully colonized the available habitat and the population continues to grow from that initial colonization. We predicted that an initial population in one corner of the landscape is more likely to have founder effects that

lead to persistent genetic structure when compared with the other two starting conditions. Mean values of the resulting structure plot parameters were compared with a single factor ANOVA and we used Levene's test (homogeneity of variance) to determine whether initial distribution caused increased variability in any of the parameters.

The effects of the shape of the dispersal distribution

In order to evaluate the effects of dispersal strategy (shape) and overall variance of the dispersal distance distribution, we ran 10 replicate simulations for each of 18 different dispersal distance distributions. A 2-way ANOVA was used to test for main effects of dispersal strategy (shape) and overall variance on the structure plot variables; a Tukey test was used to make multiple comparisons of the shapes of dispersal strategies within each level of overall variance. In addition, we used a 3-way ANOVA to test the effects of overall variance and the two components of the dispersal strategy that generate the shape of the distribution. The three factors in this analysis are variance (3 levels: 1, 1.5, 2), movement strategy (2 levels, no stayers or stayers), and distance strategy (3 levels: short only, short and long, or short and very long). We included mean dispersal distance (μ_d) as a covariate in this 3-way analysis. All statistical analyses of quantitative measures (with the exception of Levene's test for homogeneity variance—for which we used Minitab) were done using SAS Version 7.0 (SAS Institute Incorporated, Cary, North Carolina, USA).

Results

Structure plot parameters

Interpretation of the results requires a biological interpretation of the relative values for the structure plot parameters. Large values for M_0 and M_1 indicate the formation of small patches of individuals of similar genotype. We propose using the terms "local aggregation" and "neighborhood aggregation" as interpretations for increased values of M_0 and M_1, respectively. The slope is always negative and smaller (more negative) values therefore represent a rapid decay of matching genotype with distance while larger (less negative) values indicate that larger patches of similar genotypes are present. Thus, values of the slope represent patch formation or aggregation over a larger spatial scale than the local or neighborhood scales above.

Methodological concerns

Results of running the standard dispersal distribution on different landscape sizes (100 x 100, 200 x 200, 300 x 300) are illustrated in the structure plots of Figure 3. As landscape size increases, M_0, M_1 and the slope also increase. ANOVA revealed a significant effect of landscape size on M_0, M_1, and slope (Table 2). There were no significant differences between the 200 x 200 and the 300 x 300 landscape sizes for any of our quantitative measures of genetic structure.

Comparison of the mean number of individuals per site for corner, edge and central sites from the same 15 runs provided no evidence of significant edge effects ($F= 1.80$, $p=0.18$). This result implies that we can safely use all of the data from the landscape to evaluate genetic structure of the population.

An increase in either the mean or the variance of fecundity had little effect on the structure plots and their corresponding parameters (Figure 4). The distance at which the probability of a matching genotype decays to random was more variable (visual inspection of the plots in Figure 4) when either the mean or variance of fecundity was increased. However, we were unable to detect significant overall effects of changes in the fecundity on the structure plot parameters (Table 3).

When two of the dispersal distributions were run for 10,000 generations, preliminary evaluation of the results (plots of M_0, M_1 and slope versus time; not shown) suggested that a quasi-stable population structure was reached by 1000 generations. Because we were unsure of the effects of all 18 dispersal distributions on long-term stability, subsequent runs of all distributions were iterated for 2000 generations, however, inspection of these results indicates that 1000 generations was sufficient with all parameter values tested to reach definitive structure plots. Thus, we report results at 1000 generations.

Structure plots for each of the starting populations (corner quarter, center quarter or random distribution over the entire landscape) appeared more variable if the initial distribution of adults was restricted to one corner of the landscape (Figure 5). However, Levene's test for homogeneity of variance was significant only for the M_0 parameter (test statistic = 5.013, p-value = 0.026). One-way ANOVA revealed a significant effect of starting conditions on the slope parameter (Table 4). For simulations in which the initial population was located in the corner, the slopes were significantly higher (closer to zero), corresponding with a spatially longer exponential decay of the structure plots and

indicating larger patch size. Interestingly, large clusters of individuals of the same genotype formed and persisted for at least 1000 generations, despite the fact that the entire landscape reached carrying capacity within 100 generations. There is no effect of confining the initial population to the central portion of the landscape; range expansion in all directions is similar to an initial random colonization over the entire habitat (Figure 5B and C).

Results of these investigative runs provided the guidance to choose landscape size, number of generations, and starting populations for our analysis of the effects of different dispersal distributions. We chose to run ten replicates of 18 different dispersal distance distributions on a 200 x 200 landscape for 2000 generations. The starting population was randomly seeded in the center quarter of the landscape.

Effects of Dispersal Distribution

The dispersal distance distribution affected the structure plots and their corresponding parameters in interesting ways. The results of the 2-way ANOVA revealed significant effects of overall dispersal variance, dispersal strategy and the interaction term on each of the response variables M_0, M_1, and the slope (Table 5A). It is interesting to note that the relative contribution (proportion of variance) of each main effect or interaction term is different for each of the structure plot parameters. M_0 was strongly influenced by both overall variance and shape, M_1 was affected most by the overall variance, and the slope was most affected by the shape of the dispersal distance distribution.

As expected, the probability of a matching genotype within a site (M_0) decreased with an increase in the overall variance of the dispersal distribution (Figure 6A). We expected that a dispersal strategy that included stayers would increase the value of M_0. Surprisingly, this expectation did not hold for the strategy that included stayers and short-distance dispersers only (stS). Because of our practice of keeping the overall variance constant within variance-groups, the short-distance dispersers in the stS group actually dispersed farther than individuals in the stSL and stSVL strategies (Table 1—see also Discussion). For the other strategies that included stayers (stSL and stSVL), the values for M_0 (within each variance) were consistently higher than those observed for the other four strategies (Figure 6A).

In analyzing the effects of two components of the dispersal strategy, we use the shorthand "movement" to refer to the presence of organisms that disperse or not (i.e. "ns" vs. "st") and "distance" to refer to the mix of dispersal distances in the dispersal strategy (i.e. "S", "SL", or "SVL"). The results of the 3-way ANOVA (Table 5B) revealed that the pattern in Figure 6A and the multiple comparison test is largely due to the significant interaction term of movement*distance. This interaction term was significant and accounted for 17% of the variability in M_0 while the main effects of overall variance and distance accounted for 8% and 14% of the variability, respectively. One other interaction term (variance*movement) was significant but accounted for only 4% of the variability in M_0.

The effect of overall variance on the probability of a matching genotype at the adjacent site (M_1) is similar to the effect observed for M_0. As overall variance increased

M_1 decreased (Figure 6B). We expected that the dispersal strategies with stayers would result in relatively higher values of M_1. Again, this expectation held for the stSL and stSVL dispersal strategies, but the stS had significantly smaller values of M_1 than all other dispersal strategies. The dispersal strategies that resulted in the highest values for M_1 included nsS, stSL, and stSVL. Additionally, the Tukey test failed to differentiate between nsSL, nsSVL, stSL and stSVL. Taken together, these results indicate that the effect of movement on M_1 is complex and confounded by interaction with distance. The 3-way ANOVA results revealed a significant effect of overall variance on M_1 and overall variance contributed 12 % to the total sum of squares (Table 5B). The interaction effect of movement*distance was also significant and accounted for an additional 9% of the total sum of squares. While many of the other main and interaction effects were significant, they contributed little to the total sum of squares.

An increase in the overall variance of the dispersal distribution did not produce a general pattern in the slope parameter (Figure 6C). Rather, three of the dispersal strategies (nsSVL, stSL and stSVL) exhibited dramatic changes in slope as overall variance increased while the slopes for the remaining dispersal strategies were relatively similar across all three levels of variance. The 3-way ANOVA results (Table 5B) for this parameter indicate that the main effect of distance contributes most (24%) to the total sum of squares. The variance*distance interaction contributed an additional 11% to the total sum of squares and the significance of this interaction is reflected in the increased disparity among shapes as overall variance was increased (Figure 6C). The movement*distance interaction accounted for an additional 9% of the total sum of

squares. When some individuals disperse very long distances, the inclusion of stayers ameliorates the reduction in slope. Thus nsSVL has a steeper slope than stSVL within each level of variance.

Discussion

Although our modeling efforts were specifically designed to evaluate the population genetic consequences of a number of dispersal distance distributions, there are many general results worthy of discussion. Among these is the number of statistically significant effects of the dispersal distance distribution characteristics (variance, movement, and distance) on the measures of genetic structure. However, because our data are derived from simulations and are thus well replicated, controlled experiments, we recognize that statistical significance will occur more frequently than it might with real data. We thus limit our discussion to those results that account for more than 5% of the total sum of squares in each statistical model.

Initially, we were concerned that genetic structure would fade given a long enough time period. Our results suggest that the structure observed by generation 100 persisted for as long as 10,000 generations. In the biological world it is unlikely that life history traits of reproduction, survival, or dispersal would remain constant for that length of time. However, it is important to realize that the population genetic consequences of dispersal are persistent, even for the neutral case (i.e. no selective forces acting on any portion of the life history).

A second general result is the observed effect of population history on the

resulting genetic structure. The initial seeding of the population in our model is the simulation analogue of a colonization event in the natural world. If individuals were initially seeded in one corner of the landscape, then the structure plots were variable and the slopes were often less steep, suggesting that groups of individuals of similar genotype progressed across the landscape. Basically, if the colonization event is small and decentralized relative to the available habitat, the resulting genetic structure will be less predictable because of the relative importance of stochastic processes in the formation of clusters of similar genotypes (Lewis 1997). In contrast, if individuals were initially distributed over the entire landscape or in the central portion, the genetic structure was less variable among runs and the slopes of the structure plots were steeper, indicating smaller clusters of individuals of similar genotype.

The effect of landscape size is in some ways similar to the effects of the initial population distribution. If there is relatively more space available for the development of larger clusters of similar genotypes, those clusters will form and the slopes of the structure plots are less steep. Thus, whenever there is a potential for larger clusters or patches to form, whether due to increased landscape size or a colonization event that is decentralized, stochastic effects play an important role in determining consequent genetic structure.

Effects of landscape size may interact with dispersal distances in a way that allows a homogeneous environment to become "intrinsically" heterogeneous due to the development of patches of similar genotypes. Here, we can easily imagine that the environmental grain (sensu Levins 1968) defined by the size of clusters of individuals of

similar genotype will be a product of an interaction between dispersal distance and available habitat space. Because of the density-dependent nature of survival in our model, the establishment of these clusters of similar genotypes induces an "unseen" environmental heterogeneity for dispersing juveniles. Juveniles may disperse to a site that has a low density of their own genotypes but if that site is already occupied by a cluster of individuals of another genotype, there is a small probability of successful invasion by a new genotype.

Lewis (1997) investigated spatial simulation models in which individuals spread or invaded across a two-dimensional landscape. He found that an increase in mean fecundity resulted in lower variability in spread rates and reduced patchiness while an increase in the variance of fecundity increased spatial correlation and thus led to higher variability in the spread rates and increased patchiness. Although we do not quantify asymptotic rates of spread in our model, our investigation of genetic structure evaluates the patchiness of the three different genotypes. We were unable to detect any effect of changes in the fecundity distribution parameters (i.e. mean and variance) for our measure of population genetic structure.

Clearly, our results indicate that the population genetic consequences of dispersal distance distribution depend on the spatial scale at which genetic structure is measured. On the large or landscape scale, our results confirm the homogenizing effect of long distance dispersal. This effect was manifested in a significantly lower slope of the structure plot when long distance dispersers were included (i.e. for dispersal distributions that had some proportion of juveniles that dispersed across distances with a standard

deviation that was greater than 7.5). Such leptokurtic dispersal distributions (see Figure 1) are common in natural populations (e.g., Wilson 1993; Fitt et al. 1987; Taylor 1978). Additionally, we observed that an increase in the overall variance enhanced the homogenizing effect while philopatry reduced the homogenizing effect.

In contrast, the local and neighborhood measures of genetic structure are more complicated. For example, we predicted that M_0 and M_1 would increase when half of the juveniles remained at their natal sites. Careful examination of Table 1 may provide a potential explanation for the observations on M_1. When half of the juveniles remain at their natal sites, the standard deviation of the short distance dispersal must be increased (relative to the strategies with no stayers) in order to accommodate the overall variance requirements of the distribution. Therefore, the short distance essentially becomes longer when there are stayers and short distance dispersers only ($\sigma_S = 1.41$, $\sigma_S = 1.73$ and $\sigma_S = 2.0$ for overall variance of 1.0, 1.5 and 2.0, respectively). Additionally, the standard deviation of short-distance dispersal increases with the overall variance. With the addition of long or very long dispersal distances, the standard deviation of the short-distance dispersal was held constant at the same level ($\sigma_S = 0.95$) as in the strategies that had no stayers.

We propose a similar explanation for the observations on the M_0 parameter. In this case, the juveniles of each subsequent generation that are dispersing are less likely to move back to a site (of their grandparent) if they are moving a greater distance. The effect is not as strong as what was observed for M_1. We note, however, that variation in both M_0 and M_1 may be further complicated in models that include a mating system

because mating is often limited to local or neighborhood scales.

What, if any, utility do theoretical models such as ours have for empirical studies? We have found that even the simplest model can result in persistent population genetic structure and that the relationship between genetic structure and dispersal distance distribution depends critically on the spatial scale at which the genetic structure is measured. Perhaps the most important result of our analysis is that knowledge of mean dispersal distance and overall variance in dispersal distance is not sufficient to predict the consequent population genetic structure. Mean dispersal distance was a significant covariate for only one parameter, M_0 and spatial structure of the population varied (for all three of our metrics) among the different dispersal strategies within the same level of variance. An understanding of the effects of dispersal on genetic population structure requires knowledge of the entire dispersal distance distribution, not just its first two moments.

Our results suggest that the simplest model (i.e. no environmental heterogeneity, no selection, asexual reproduction) yields stable and persistent genetic structure over a broad range of dispersal distributions. A challenge for future work is how the signature of this structure changes when selection and directional movement are added to the mix of factors affecting the population.

Acknowledgments

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Appendix I

To following formulae were used to calculate mean dispersal distance for each of the dispersal distributions:

$$E\{|z|\} = \int_{-\infty}^{+\infty} \frac{|z| e^{-z^2/2}}{\sqrt{2\pi}} dz$$

$$= 2 \int_{0}^{+\infty} \frac{z e^{-z^2/2}}{\sqrt{2\pi}} dz$$

$$= \sqrt{\frac{2}{\pi}} \int_{0}^{\infty} z\, e^{-z^2/2}\, dz$$

$$u = z^2/2$$

$$du = z dz$$

$$E\{|z|\} = \sqrt{\frac{2}{\pi}} \int_{0}^{\infty} e^{-u}\, du = \sqrt{\frac{2}{\pi}} = 0.798$$

Given a normal distribution with standard deviation $=\sigma$

$$E\{|x|\} = E\{|\sigma z|\}$$

$$= \sigma E\{|z|\} = 0.798\sigma$$

and for a mixture distribution

$$E\{|x|\} = \left[0 \cdot P\{stay\} + \sigma_{short} \cdot P\{short\} + \sigma_{long} \cdot P\{long\}\right] \cdot 0.798 \quad \text{or}$$

$$E\{|x|\} = \left[0 \cdot P\{stay\} + \sigma_{short} \cdot P\{short\} + \sigma_{verylong} P\{verylong\}\right] \cdot 0.798$$

Overall variance for each dispersal distance distribution was calculated as follows:

$$Var(X) = P\{stay\} \cdot 0 + P\{short\} \cdot \sigma^2_{short} + P\{long\} \cdot \sigma^2_{long}$$

$$Var(X) = P\{stay\} \cdot 0 + P\{short\} \cdot \sigma^2_{short} + P\{verylong\} \cdot \sigma^2_{verylong}$$

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Table 1. Parameters used to generate dispersal distributions with different variances and shapes. Symbols are as follows: σ_S and σ_L are the standard deviation for the short and long dispersal distances respectively and μ_d represents the mean dispersal distance for each distribution. The code provided in the second column is used as shorthand to refer to each group of parameters elsewhere in the paper, where "ns" = no stayers, "st" = stayers, "S = short distance dispersal, "L" = long distance dispersal, and "VL" = very long distance dispersal.

		Variance								
		1			**1.5**			**2.0**		
Distribution	**Code**	σ_S	σ_L	μ_d	σ_S	σ_L	μ_d	σ_S	σ_L	μ_d
100 % short	ns S	1	-	0.80	1.22	-	0.97	1.41	-	1.13
80% short, 20% long	ns SL	0.95	1.18	0.79	0.95	1.97	0.92	0.95	2.53	1.01
98% short, 2% very long	ns SVL	0.95	2.43	0.78	0.95	5.56	0.83	0.95	7.48	0.86
50% stay, 50% short	st S	1.41	-	0.56	1.73	-	0.69	2	-	0.80
50% stay, 40% short, 10%long	st SL	0.95	2.53	0.51	0.95	3.38	0.57	0.95	4.05	0.63
50% stay, 49% short, 1% very long	st SVL	0.95	7.48	0.43	0.95	10.29	0.45	0.95	12.49	0.47

Table 2. One-way ANOVA results testing the effect of landscape size on quantitative variables associated with the structure plots. Mean parameter values and standard errors (in parentheses) are given for each of 3 landscape sizes, df =2.

	Landscape size			
Dependent variable	100 x 100	200 x 200	300 x 300	p-value
M_0	0.341	0.378	0.401	<0.01
	(0.010)	(0.013)	(0.02)	
M_1	0.195	0.26	0.294	<0.01
	(0.012)	(0.014)	(0.009)	
Slope	-0.113	-0.06	-0.04	<0.01
	(0.009)	(0.003)	(0.003)	

Table 3. One-way ANOVA results testing the effect of fecundity parameters on each of the quantitative variables derived from the structure plots. There were three different combinations for the mean and standard deviation in fecundity: (15, 7.5), (25, 7.5) and (15, 15). Means and standard errors (in parentheses) are given for each of the resulting structure plot parameters, df = 2.

	Fecundity mean and variance			
Dependent Variable	(15, 7.5)	(25, 7.5)	(15, 15)	p-value
M_0	0.339	0.356	0.356	0.33
	(0.007)	(0.010)	(0.010)	
M_1	0.194	0.225	0.221	0.19
	(0.009)	(0.014)	(0.013)	
Slope	-0.12	-0.10	-0.104	0.5
	(0.010)	(0.012)	(0.015)	

Table 4. One-way ANOVA results testing the main effect of the initial distribution of the population on each of the quantitative measures derived from the structure plots. There were three different starting conditions: randomly seeded in one corner of the landscape, randomly seeded in the center quarter of the landscape and randomly seeded over the entire landscape. Means and standard errors (in parentheses) are given for each starting distribution.

	Starting distribution			
Dependent Variable	corner	center quarter	entire	p-value
M_0	0.320	0.339	0.327	0.82
	(0.036)	(0.007)	(0.005)	
M_1	0.230	0.194	0.175	0.22
	(0.035)	(0.009)	(0.007)	
Slope	-0.058	-0.120	-0.14	<0.01
	(0.01)	(0.01)	(0.01)	

Table 5. ANOVA results for the effects of changes in the dispersal distribution in the standard 200 x 200 landscape at 1000 generations. A. Two-way ANOVA evaluated the main and interaction effects of variance and dispersal strategy (e.g. nsS, stSVL, etc.) on the dependent variables of the structure plots: M_0, M_1, and slope. The proportion of variance was calculated by dividing the Type III sum of squares (for each factor or interaction term) by the total sum of squares. As such it will not usually add to 100%. B. Three-way ANOVA results include main effects of overall variance, movement and distance; mean distance dispersed was included as a covariate in the model. The proportion of variance was calculated by dividing the Type III sum of squares (for each factor or interaction term) by the total sum of squares.

A.

Dependent variable: $\mathbf{M_0}$	df	Type III Sum of Squares	Proportion of Variance	F	p-value
Source: Variance	2	0.247439	35%	431.3	<0.001
Strategy	5	0.391226	55%	272.8	<0.001
Vari*Strategy	10	0.030258	4%	10.6	<0.001
Error	162	0.046469			
Dependent variable $\mathbf{M_1}$					
Source: Variance	2	0.272338	71%	355.5	<0.001
Strategy	5	0.033843	9%	17.7	<0.001
Vari*Strategy	10	0.012947	3%	3.4	<0.001
Error	162	0.062044			

Dependent variable:					
slope					
Source: Variance	2	0.014515	6%	49.5	<0.001
Strategy	5	0.165013	70%	225.2	<0.001
Vari*Strategy	10	0.032837	14%	22.4	<0.001
Error	162	0.023736			

B.

Dependent variable: $\mathbf{M_0}$	df	Type III Sum of Squares	Proportion of Variance	F	p-value
Source: Mean distance	1	0.000236	<1%	0.82	0.37
Variance	2	0.006822	8%	11.9	<0.001
Movement	1	0.000902	1%	3.1	0.078
Distance	2	0.012429	14%	21.7	<0.001
Variance*Movement	2	0.003430	4%	6.0	0.003
Movement*Distance	2	0.015030	17%	26.2	<0.001
Variance*Distance	4	0.001187	1%	1.0	0.39
Error	165	0.047354			
Dependent variable: $\mathbf{M_1}$					
Source: Mean distance	1	0.002070	2%	5.42	0.02
Variance	2	0.012228	12%	16.0	<0.001

Movement	1	0.001907	2%	5.0	0.03
Distance	2	0.003688	4%	4.8	0.01
Variance*Movement	2	0.005391	5%	7.1	<0.001
Movement*Distance	2	0.009458	9%	12.4	<0.001
Variance*Distance	4	0.004232	4%	2.8	0.03
Error	165	0.063021			
Dependent variable:					
slope					
Source: Mean distance	1	0.000002	<1%	0.02	0.9
Variance	2	0.000120	<1%	0.4	0.67
Movement	1	0.000005	<1%	0.04	0.85
Distance	2	0.010923	24%	36.6	<0.001
Variance*Movement	2	0.000622	1%	2.1	0.13
Movement*Distance	2	0.004086	9%	13.7	<0.001
Variance*Distance	4	0.005044	11%	8.4	<0.001
Error	165	0.024160			

FIGURE CAPTIONS

Figure 1. Dispersal distributions generated from parameters described in Table 1. Only four of the six distributions are shown in the interest of clarity. In each plot, the upper two curves are those with all juveniles dispersing (10nsS and 10nsSVL); and in the lower two graphs juveniles have a 50 % probability of remaining at their birthplace. For the lower two graphs there is a point mass at zero. The tails of the distributions are influenced by the presence of individuals who disperse very long distances. The SL distributions (not plotted) fall between the respective S and SVL distributions as expected. A. Overall variance = 1.0, B. Overall variance = 1.5, C. Overall variance = 2.0

Figure 2. Plot of the relationship between mean dispersal distance (μ_d) and the overall variance of the dispersal distance distribution. Abbreviations (codes) for each two-component dispersal strategy are found in Table 1.

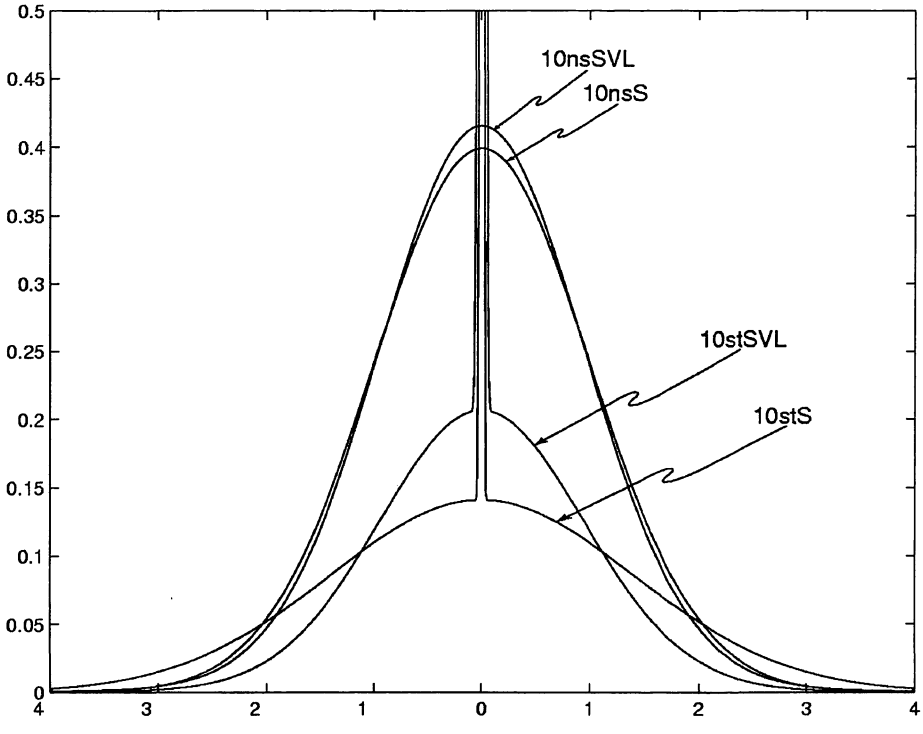
Figure 3. Five replicate structure plots for each of three different landscape sizes in which the probability of genotype sharing is plotted against distance. Structure plots were constructed for generation 1000. Because we are primarily interested in M_0, M_1 and the exponential decay at shorter distances (see text), plots are truncated. A. 100 x 100 landscape, B. 200 x 200 landscape, C. 300 x 300 landscape.

Figure 4. Structure plots from simulations (n=5 for each set) to evaluate the effects of changes in the fecundity parameters on geographic structure. A. Standard model with

fecundity parameters ($X=15$, $\sigma^2=7.5$) B. Increased mean fecundity ($X=25$, $\sigma^2=7.5$) and C. Increased variance of fecundity ($X=15$, $\sigma^2=15$)

Figure 5. Five replicate structure plots for each of three different starting conditions in which the initial population is randomly seeded as follows: A. in one corner of the landscape (50 x 50 box), B. in the central quarter of the landscape, C. over the entire landscape.

Figure 6. Boxplots for each of the structure plot variables plotted as a function of dispersal distance distribution. A: M_0, the probability of a matching genotype within a site, B: M_1, the probability of a matching genotype at an adjacent site, C: the slope at which the probability of a matching genotype decreases with distance. The key for the dispersal distribution codes on the x-axis is as follows: the overall variance (x 10), no stayers (ns) or stayers (st) and short (S), long (L) or very long (VL) dispersal distances. For further details see Table 1. The horizontal line represents the median value (from 10 runs), the box covers the range between the 25^{th} and 75^{th} percentiles and the vertical lines represent the entire range of values with the exception of the asterisks that represent statistical outliers (i.e. points that exceed $\pm$ 1.5*interquartile range). For all structure plot variables, the overall variance caused significant differences; letters above each plot for the middle variance reflect the effect of the dispersal strategy. Those plots with the same lower case letter within the same overall variance group are not significantly different from one another.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0.3
0.25
0.2
0.15
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0.05
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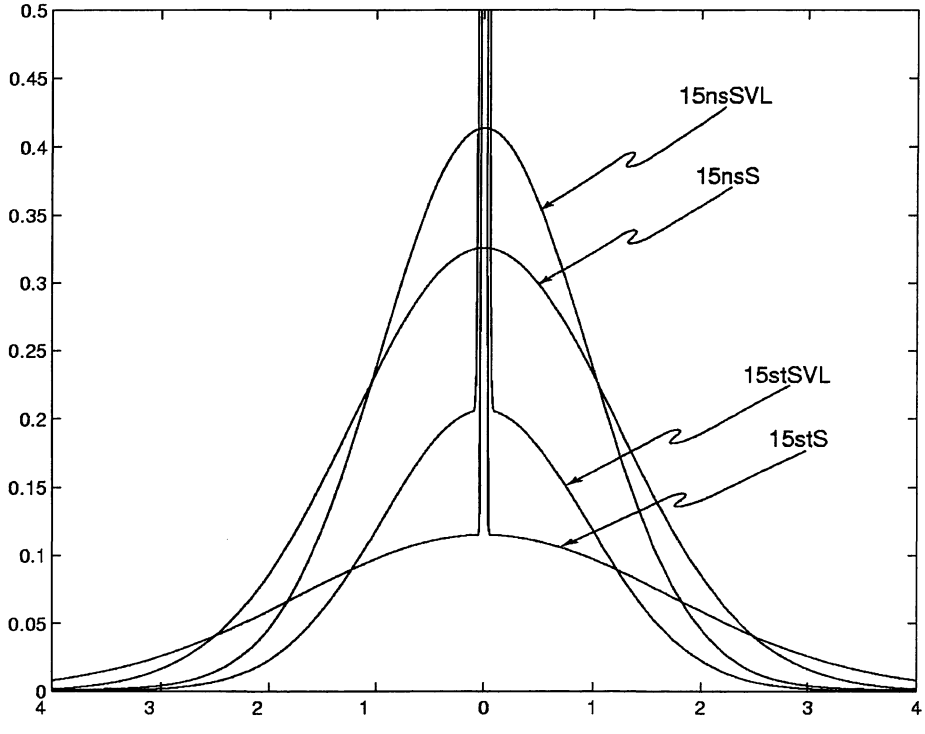
3
2
1
0
1
2
3
4
10nsSVL
10nsS
10stSVL
10stS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15nsSVL
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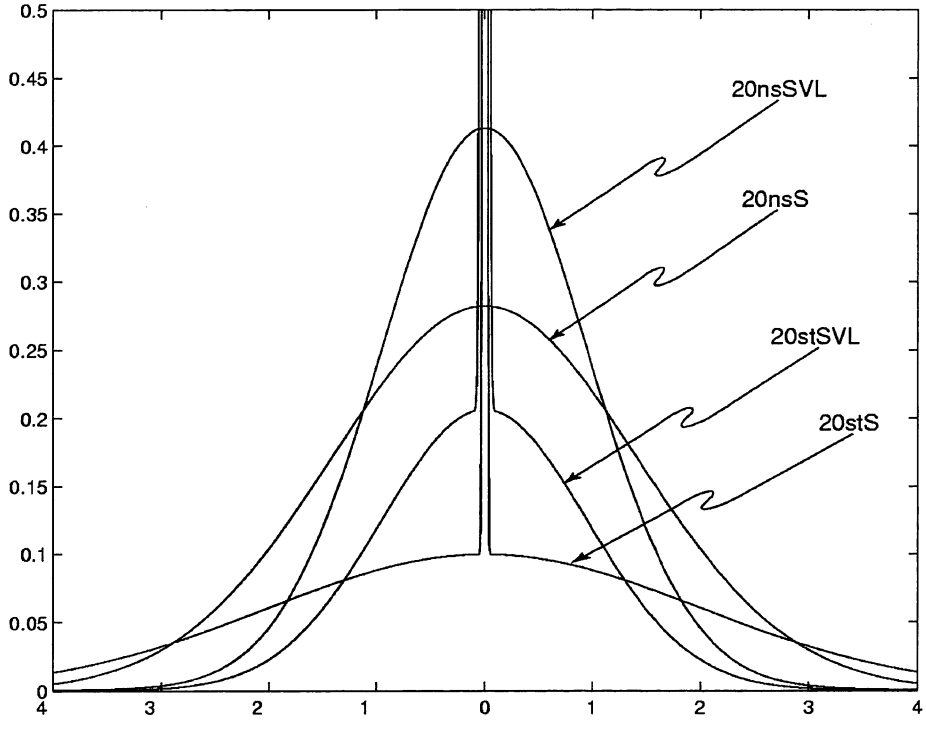
15stSVL
15stS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0
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2
3
4
20nsSVL
20nsS
20stSVL
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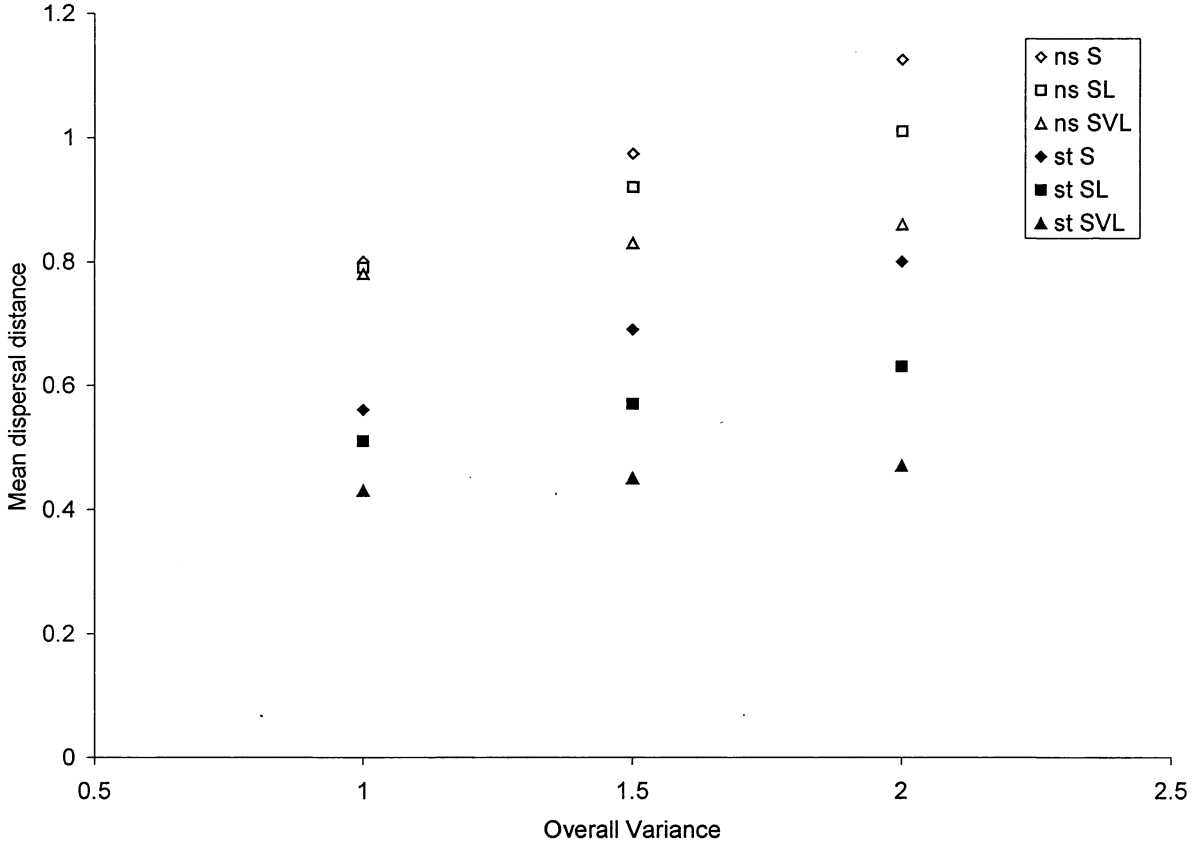
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1
0.8
0.6
0.4
0.2
0
0.5
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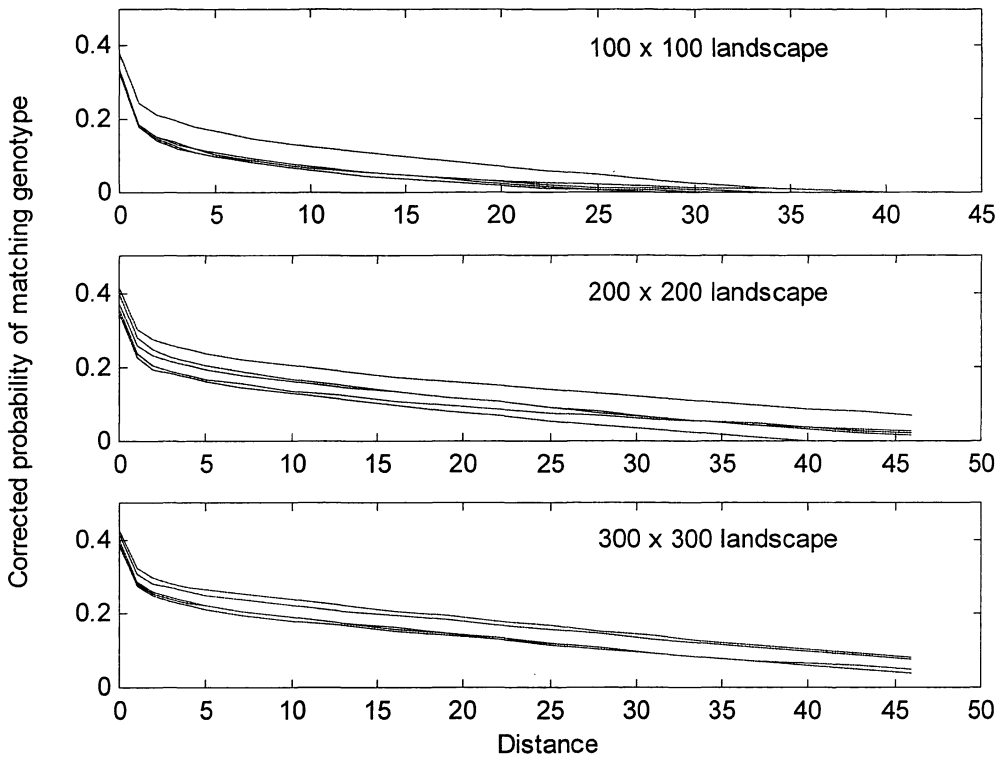
1.5
2
2.5
Mean dispersal distance
Overall Variance
ns S
ns SL
ns SVL
st S
st SL
st SVL


100 x 100 landscape
200 x 200 landscape
300 x 300 landscape
Corrected probability of matching genotype
Distance
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0.2
0
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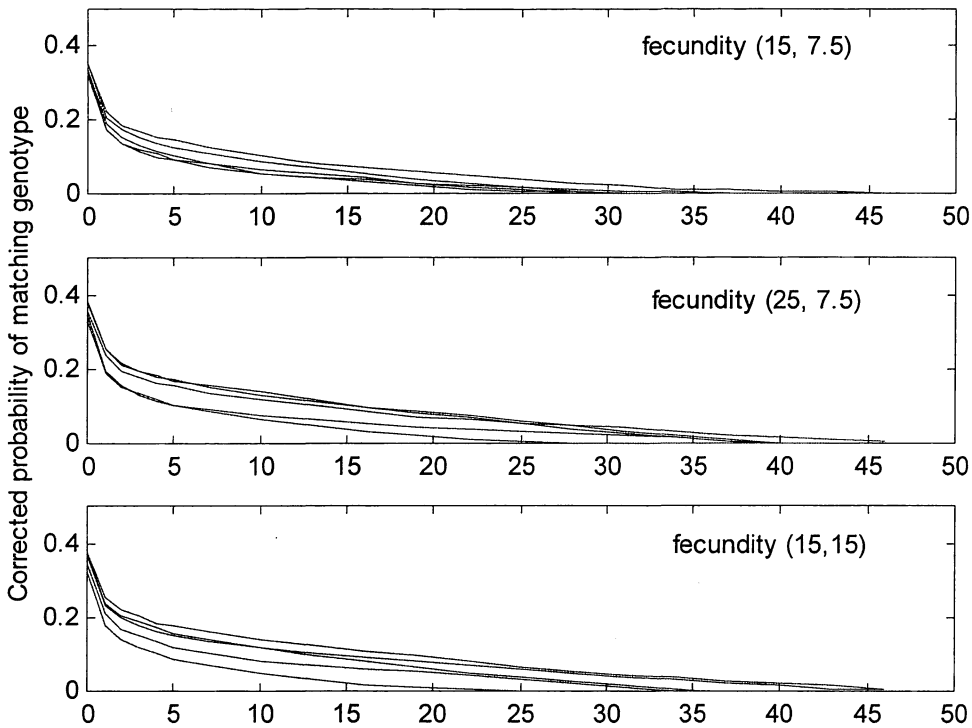
0 5 10 15 20 25 30 35 40 45 50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fecundity (15,15)
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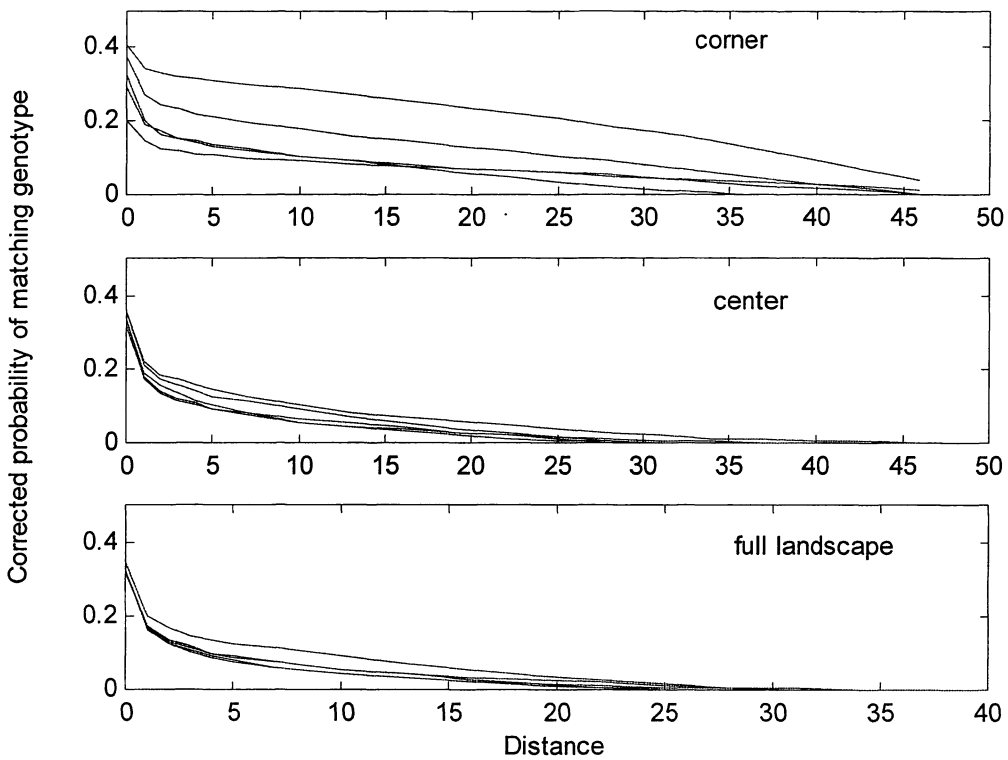
0 5 10 15 20 25 30 35 40 45 50


corner
center
full landscape
Corrected probability of matching genotype
Distance
0.4
0.2
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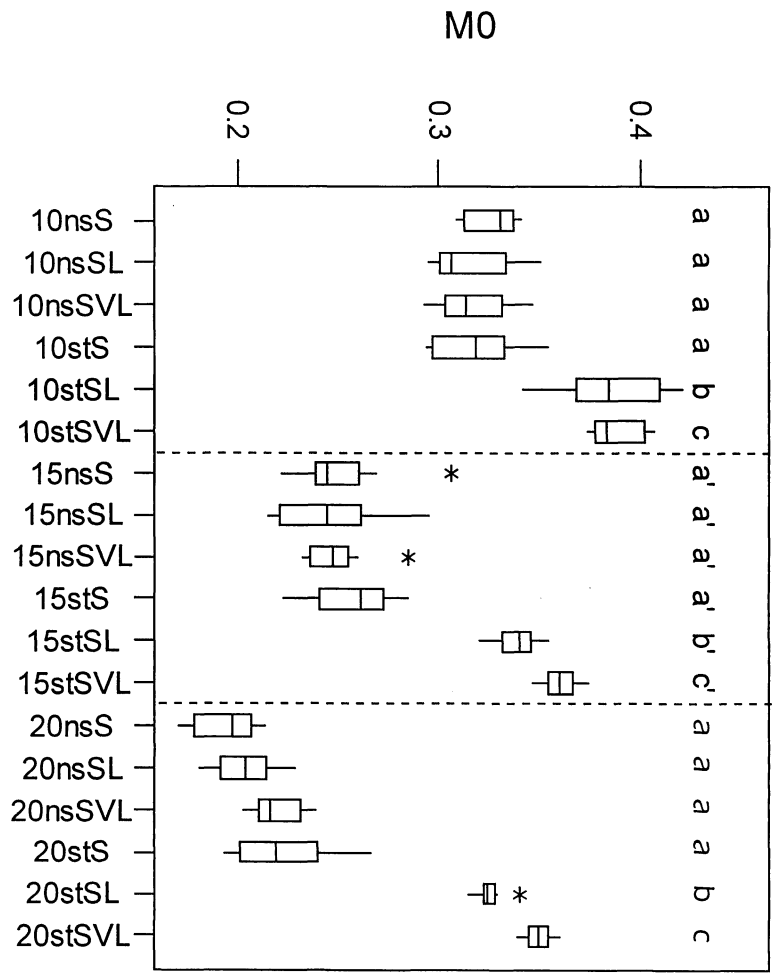
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10nsSVL
10stS
10stSL
10stSVL
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15nsSVL
15stS
15stSL
15stSVL
20nsS
20nsSL
20nsSVL
20stS
20stSL
20stSVL
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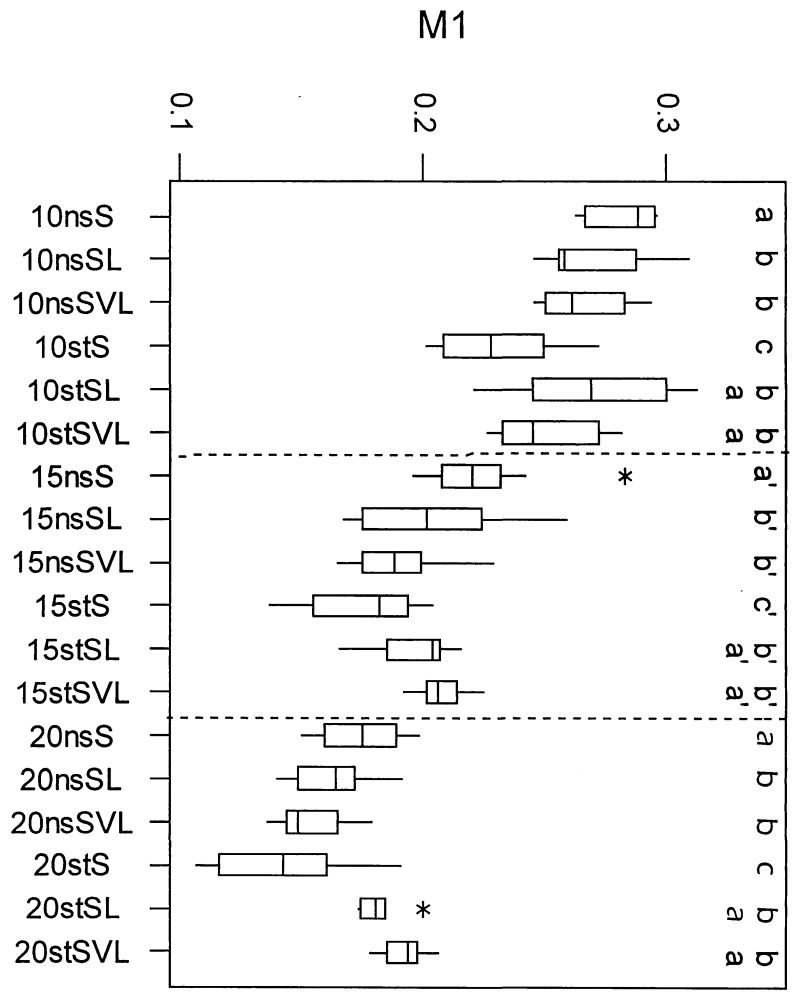
b
c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0.3
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10stSVL
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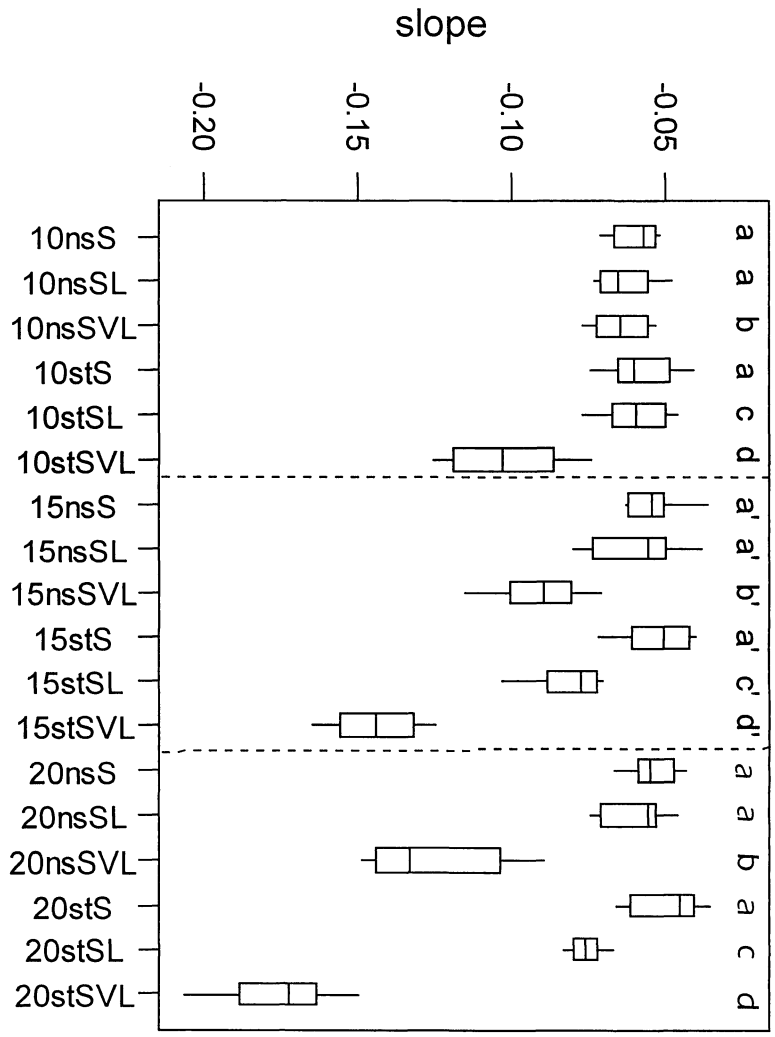
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a
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