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Non-equilibrium genetic structure is insensitive to the shape of the dispersal distribution

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ABSTRACT

Questions: How does rare, long-distance dispersal affect spatial genetic structure on ecological temporal scales? What is the magnitude of its effect relative to the effects of the mean extent of dispersal and of fecundity?

Model features: One-locus, two-allele individual-based simulation of a sessile, annual organism, without mutation.

Key variables: The shape of the dispersal distribution is leptokurtic or platykurtic, with kurtosis varying by a factor of 3; the spatial variance of dispersal and fecundity vary by a factor of 5.

Conclusions: Effects of the shape of the dispersal distribution and of fecundity on within-population spatial genetic autocorrelation are small compared with the strong effect of dispersal variance. Additional processes such as spatial population expansion can increase the effect of long-distance dispersal for some time, and may contribute to studies showing a large impact of dispersal distribution shape. Analysis of different trajectories for populations not in mutation-drift equilibrium provides key information about biological mechanisms and explains why long-distance dispersal is important to some ecological processes and not others.

Keywords: dispersal kernel, genetic structure, kurtosis, long-distance dispersal, pattern formation, spatial autocorrelation.

INTRODUCTION

Understanding dispersal is central to spatial ecology. The ways in which organisms redistribute themselves in space can affect the dynamics of competition (e.g. Bolker and Pacala, 1999; Neuhauser and Pacala, 1999; Chesson, 2000), disease (e.g. Thrall and Burdon, 1999), spatial population spread (e.g. Kot *et al.*, 1996; Clark, 1998), occupation of patchy habitats (Hanski and Gilpin, 1997), such as marine reserves (e.g. Botsford *et al.*, 2001; Lockwood *et al.*, 2002; Gerber *et al.*, 2003), and other spatial ecological processes (e.g. Tilman and Kareiva, 1997). Measuring dispersal directly, however, is notoriously difficult (Bullock and Clarke, 2000; Cain *et al.*, 2000; Nathan and Muller-Landau, 2000), and

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researchers frequently assess dispersal indirectly by inferring it from genetic patterns (Ouborg *et al.*, 1999; Cain *et al.*, 2000; Nathan *et al.*, 2003). Issues surrounding these inferences are prominent in marine systems as well as in terrestrial ones (Kinlan and Gaines, 2003).

A rich body of modelling work predicts a strong relationship between dispersal and the development of patches of genetically similar individuals (Wright, 1943; Kimura and Weiss, 1964; Malécot, 1969; Rohlf and Schnell, 1971; Sawyer, 1977b; Sokal and Wartenberg, 1983; Slatkin, 1985b; Epperson, 1995a; Rousset, 2000). This work, which has proved useful for estimating the average extent of dispersal from genetic structure in scores of empirical studies (reviewed in Slatkin, 1985a; Neigel, 1997; Bohonak, 1999; Ouborg *et al.*, 1999), shows that long-distance average dispersal reduces patch formation. The role that rare long-distance dispersal may play in this well-developed theoretical framework, however, is less well understood. If extreme, infrequent dispersal events affect genetic structure appreciably, then estimates of dispersal from genetic data may be inaccurate in cases where such dispersal occurs. On the other hand, a quantitative relationship between the frequency of long-distance dispersal and the genetic patterns that result would be a useful tool for assessing long-distance dispersal in the field. The strengths and shortcomings of other empirical methods for quantifying long-distance dispersal, such as parentage analysis, are reviewed elsewhere (see, for example, Cain *et al.*, 2000; Nathan *et al.*, 2003), and we do not consider them here.

Empirical evidence indicates that many species experience some long-distance dispersal. Measured dispersal distributions are frequently leptokurtic (see Portnoy and Willson, 1993; and references in Kot *et al.*, 1996). Leptokurtic distributions have more items in their centres and tails and fewer in their shoulders than a Gaussian distribution with the same variance, while platykurtic distributions have more items in their shoulders and fewer in their centres and tails (Fig. 1). The ubiquity of leptokurtic dispersal distributions thus indicates that many species effectively disperse over disparate spatial scales, with most dispersers staying relatively close to their origin and some travelling very long distances. Mechanistic models demonstrate how various dispersal distributions may arise from different assumptions about aerodynamics, the behaviour of pollinators or other animal dispersal vectors, or other means of dispersal (see, for example, Morris *et al.*, 1995; Neubert *et al.*, 1995; and references in Higgins *et al.*, 2003).

Gathering data on the tails of a dispersal distribution is inherently difficult (Bullock and Clarke, 2000). Therefore, modelling has been instrumental in demonstrating that long-distance dispersal may have profound effects on ecological processes. Recent modelling studies show that dispersal kurtosis may have important repercussions for the dynamics of spatial host–parasitoid interactions (Wilson *et al.*, 1999), disease (Brown, 2001) and hybrid zones (A. Hastings and C.T. Lee, unpublished work). Studies in spatial population spread show that the tails of dispersal distributions are critical determinants of rates of spread of introduced organisms (Clark, 1998; Higgins and Richardson, 1999; Lewis and Pacala, 2000). Given that long-distance dispersal is so common, important and difficult to measure directly, being able to quantify the phenomenon using genetics would be very valuable.

Much previous theoretical work that considers the effect of different dispersal distributions on genetic patterns assumes genetic and demographic equilibrium. These studies exploit the non-trivial equilibrium spatial patterns that result when local genetic drift, caused by restricted dispersal, is opposed by a diversifying evolutionary force such as mutation or speciation (Weiss and Kimura, 1965; Malécot, 1969, 1975; Sawyer, 1977a; Rousset, 2000; Chave and Leigh, 2002), global migration, which allows dispersal from any point in space to reach any other point with some non-zero frequency (Kimura and Weiss, 1964; Crawford, 1984), or balancing

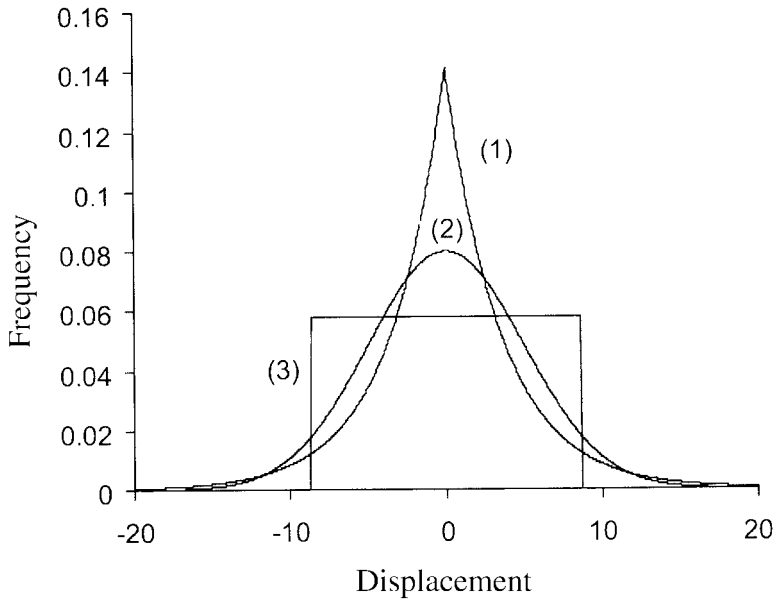

Fig. 1. Leptokurtic, neutrally kurtotic and platykurtic dispersal distributions in one spatial dimension. (1) Laplacian or 'double exponential' (leptokurtic) function. (2) Gaussian (neutrally kurtotic). (3) Uniform or 'top hat' (platykurtic) function. The three curves have a variance of 5 spatial units squared. Note that because the distributions are symmetric around zero, mean dispersal distance and skew are zero in all three cases.

selection (Lande, 1991). Most of these studies find little effect of non-Gaussian restricted dispersal on equilibrium genetic patterns or on indirect measures of dispersal based on them. These results are important, but their applicability to populations not in genetic equilibrium is unclear. In particular, in the absence of any diversifying force, no equilibrium genetic pattern exists in infinite space (see, for example, Sawyer, 1977b), while equilibria in finite populations involve fixation of one allele. To investigate any influence of long-distance dispersal on spatial genetic patterns in populations where fixation of neutral alleles due to drift is a real possibility, we require a framework in which mutation and other diversifying evolutionary forces are absent.

Simulation studies that investigate how the shape of the dispersal distribution affects spatial genetic structure in the absence of mutation show large effects of kurtosis. Levin and Kerster (1975), Le Corre *et al.* (1997) and Austerlitz and Garnier-Géré (2003) demonstrated dramatic differences in genetic structure resulting from different dispersal distributions, but did not control the variances of these distributions to ensure that they differed only in shape. Ibrahim *et al.* (1996) did compare Gaussian and leptokurtic distributions with equal variances, and found that the latter generates much more patchy genetic patterns. Like Le Corre *et al.* (1997) and Austerlitz and Garnier-Géré (2003), however, Ibrahim *et al.* (1996) examined genetic structuring in the context of active population expansion into previously unoccupied habitat. These studies confound the effects of colonization history and ongoing, though rare, long-distance dispersal. An approach that independently varies dispersal variance and kurtosis in a population that is not expanding would clarify the

relative contributions of these two factors. Finally, few studies on the shapes of dispersal distributions have considered the effects of platykurtic dispersal. Although it is not often observed in nature, published simulation models of isolation by distance often assume platykurtic dispersal for computational convenience (Kimura and Weiss, 1964; Sokal and Wartenberg, 1983; Epperson, 1995b; Doligez *et al.*, 1998). Understanding the effects of platykurtic dispersal is important in order to apply these models to real biological systems.

In this study, we investigate whether a platykurtic dispersal distribution and a comparable leptokurtic distribution give rise to different patterns of spatial correlation in a single diallelic locus, assuming that genes are neutral and that mutation is absent. We evaluate the importance of changes in the shape of the distribution relative to changes in dispersal variance and fecundity. We develop and describe our model for a population of annual plants, but our approach and results apply equally to sessile marine organisms, which are of much current interest (Gerber *et al.*, 2003; Grantham *et al.*, 2003). We vary the distribution shape, dispersal variance and fecundity independently through ranges that are reasonably wide for each factor given the model's generality. We select initial conditions that provide little opportunity for colonization processes, and discuss how the choice of initial conditions can reveal important information in non-equilibrium analyses.

METHODS

The simulation model

Our stochastic, individual-based simulation tracks a single, neutral, diallelic locus in a population of diploid, self-compatible annual plants without a seed bank. Each cell of a two-dimensional grid may or may not be occupied by a single plant at a given time. Each simulation run begins with 10% of the cells occupied. For runs described here, these initial individuals' positions are random, and their genotypes are distributed in Hardy-Weinberg proportions; the initial frequency of each allele is 0.5. At the beginning of each generation, plants produce a number of pollen grains chosen from a Poisson distribution with a mean of 100. A random choice between the parental plant's two alleles determines each pollen grain's genotype, and a random choice from a dispersal distribution dictates its destination grid cell. Pollen grains that fall in unoccupied cells or beyond the borders of the grid are lost.

Plants make seeds by pairing the pollen grains that they receive with randomly chosen alleles from their own genotypes. Depending upon the amount of pollen available, each plant produces seeds up to a maximum number chosen from a Poisson distribution (see 'Factorial experiment', below). A plant that receives more pollen grains than this maximum number of seeds chooses the pollen to be used at random and discards the rest. Seeds receive a destination according to a choice from the same dispersal distribution used for pollen. From the seeds that arrive at a given grid cell, one randomly selected individual survives. The removal of adult plants from the previous generation and their replacement by successful seeds completes each simulation generation.

As described, the model is appropriate for plants and for similarly sessile, hermaphroditic animals such as some marine invertebrates. The concepts involved in pollen and seed dispersal are general enough, however, to apply to animal species where one sex is sedentary before mating, including insect species where males mate with females as soon as the adult females emerge. In that case, positions on the grid represent not the lifetime position of a

plant but the position of an animal censused before dispersal, pollen dispersal corresponds to the movement of the mobile parent to find its mates, and seed dispersal represents the movements of the offspring – inside a parent or out – after mating and until census. Finally, for gonochoristic animals the simulation should be modified so that only females produce offspring. To extend the model to species where both sexes move before mating, one could census males before their mating dispersal and females afterwards. Pollen dispersal would represent the mating movement of the males, while seed dispersal would correspond to the mixture of male offspring dispersal (from fertilization to birth, for instance) with female offspring dispersal followed by adult female mating dispersal (fertilization to mating). Structural changes to the model could also capture these differences in dispersal strategies. We do not address these complexities here, and simply refer to pollen and seed dispersal. Similarly, we do not deal with the situation where adults live longer than one reproductive season.

To execute dispersal, we used movement distances chosen from continuous distributions and binned the resulting positions onto the simulation grid. In keeping with other theoretical work on dispersal (e.g. Neubert *et al.*, 1995; Kot *et al.*, 1996), we work with distributions of displacements rather than distances. Displacement includes direction and thus can be positive or negative; consequently, displacement distributions are symmetric around zero in one dimension (Fig. 1). Average dispersal displacement and higher odd moments are zero, while the variance and kurtosis of displacement provide measures of the average extent of dispersal and the shape of the distribution, respectively. Dispersal distance distributions, in contrast, are distributions of non-negative numbers and are not symmetric around zero; the average dispersal distance (rather than the variance) captures the extent of dispersal. We convert one-dimensional distributions of dispersal displacement into two-dimensional ones for use on our grid by rotating the one-dimensional distributions around zero.

The model allows for separate dispersal distributions for pollen and seeds, but we used the same distributions for both. We recognize that such a situation is unlikely in nature, but we are not concerned in this study about the relative importance of the two kinds of dispersal. Other studies have examined the effects of allowing these two dispersal stages to differ independently (e.g. Levin and Kerster, 1975; Doligez *et al.*, 1998; Epperson, 2003). Because the model considers only one species, seed and pollen output actually refer to seeds and pollen grains that survive predation, interspecific competition, and all such perils other than intraspecific competition, dispersal beyond the population boundaries and, in the case of pollen, failure to land on a receptive stigma. Finally, we used absorbing boundary conditions, where we assume that habitat beyond the grid's borders is unsuitable, because we believe that absorbing boundaries may more accurately reflect the boundary conditions that real plant populations experience. We ran the base simulations using a 100×100 grid, which allows a maximum of 10,000 individuals. An independent set of runs with a 200×200 grid yielded indistinguishable results, and a parallel analysis of an infinite population revealed similar behaviour (Lee, 2002). Thus, we are confident that the essential dynamics of our small model population are representative for a range of population sizes. We acknowledge, however, that real populations could contain more than 10,000 individuals.

Factorial experiment

We allowed pollen and seeds to disperse according to either: (1) a platykurtic, uniform or 'top hat' function, which is constant within a certain radius and zero outside it, and which

has kurtosis 1.8 in one dimension (as opposed to the value of 3 for a Gaussian distribution); or (2) a leptokurtic, Laplacian or 'double-exponential' function with kurtosis 6 (Fig. 1). To compare the shapes of these different dispersal distributions while holding the overall extent of dispersal equal, we chose parameters for the two distributions such that their spatial variances were equal in one dimension (Fig. 1). We independently allowed dispersal variance to be either high or low. Independent simulations show that binning the continuous dispersal distributions onto an integer grid along their major axes does not appreciably change their kurtosis, given the range of dispersal variance considered here. Finally, we examined the effects of changing the mean number of seeds that plants can produce when saturated with pollen. The full factorial design thus includes leptokurtic and platykurtic dispersal distributions, dispersal variance of 5 and 25 grid cells squared, and an average of 2 and 10 seeds produced by plants with enough pollen. We ran the model 500 times for each experimental treatment, yielding a total of 4000 landscapes in this $2 \times 2 \times 2$ factorial design.

The overall distribution for the dispersal of genes is the mixture of the seed dispersal distribution (for female genes) with the convolution of the pollen and seed distributions (for male genes). Gene dispersal is frequently more difficult to measure independently than pollen or seed dispersal, however. Because our interest is in establishing whether genetic patterns respond to concrete quantities that ecologists could measure in the field, we chose to vary the dispersal distributions of pollen and seeds rather than to manipulate the distribution of gene dispersal that results.

Our choices for the high and low levels for each of the three independent variables reflect our desire to obtain reasonably general results from the model. To choose natural levels of variation is difficult without specifying a particular species or even a life-history category for the modelled organism. Dispersal variance can vary by several orders of magnitude in nature (Kinlan and Gaines, 2003); fecundity, particularly as we describe it here, is hard to measure and surely similarly variable. We feel that varying dispersal variance and average fecundity by a factor of 5 is reasonable. The variation in kurtosis is only a factor of about 3, and dispersal distributions with kurtosis more extreme than 6 do occur in nature. We reiterate that few empirical examples of platykurtic dispersal distributions exist: a uniform distribution, with kurtosis 1.8, is certainly extreme compared with what occurs in nature. We explore the issue of kurtosis higher than 6 further in the Discussion section.

Assessing spatial genetic structure

To measure spatial genetic structure, we used Moran's *I*-statistics for individual gene frequencies, following Heywood (1991):

$$I = \frac{n}{\sum_{i,j} w_{ij}} \frac{\sum_{i,j} w_{ij}(x_i - \bar{x})(x_j - \bar{x})}{\sum_i (x_i - \bar{x})^2},$$

where n is the population size, w_{ij} is a weight, x_i and x_j are individual allele frequencies (1 for genotype AA, 0.5 for Aa, and 0 for aa) for individuals i and j, and $\bar{x}$ is the mean population allele frequency. If we assign w_{ij} a value of 1 for all pairs of individuals i and j separated by a given distance, and a value of 0 for all other pairs, this formula takes the form of a

landscape-average measure of the genetic correlation between individuals separated by the chosen spatial lag, and therefore generally varies between −1 and +1. Positive values at short lags indicate clumping of like genotypes and thus the development of genetic structure. Values of Moran's I of 0 indicate a random spatial arrangement of genotypes, and negative values imply over-dispersion. Many theoretical and empirical studies of genetic structure include calculation of Moran's I as described here (Le Corre *et al.*, 1997; Epperson, 2003), or of spatial correlation functions that are conceptually similar (e.g. Kimura and Weiss, 1964; Malécot, 1969; Sawyer, 1977a). Additionally, correlation functions are becoming more common as measures of ecological pattern formation (e.g. Bolker and Pacala, 1999; Lewis and Pacala, 2000; Engen *et al.*, 2002).

For each model landscape, we calculated Moran's I after 50 generations (I_{50}) for the ten distance classes consisting of nearest neighbours only, next-nearest neighbours, and so on up to a lag of ten. We assessed differences in correlograms between treatment groups using multivariate analysis of variance (MANOVA). Raw values of I_{50} were strongly heteroscedastic, as were values transformed according to Fisher's z-transformation for Pearson's correlation coefficients, but the arcsine-square-root transform substantially relieved both heterogeneity of variance and deviations from univariate normality. The arcsine-square-root transformation yields complex numbers for raw data that are negative. Of the 40,000 values of I_{50} we included in this analysis – we calculated I_{50} at each of ten lags for 4000 landscapes – only two were negative (see Results section). We performed the transformation and MANOVA with these two values (-1.2×10^{-3} and -7×10^{-4}) set to zero, and again with the two points omitted from the analysis. The results from these two analyses were virtually identical. We report here numbers from the test with the two values set to zero.

RESULTS

Effects of experimental treatment on I_{50}

More restricted dispersal generates more genetic structure across all measured distance classes (MANOVA test for main effect of variance, $P < 0.0001$; Table 1). Values of I_{50} are approximately five times greater for low dispersal variance treatments than for high dispersal variance treatments at each spatial lag (means and standard errors for first lag: low variance 0.1907 ± 0.0013, high variance 0.0372 ± 0.0004; for tenth lag: low variance 0.1119 ± 0.0012, high variance 0.0228 ± 0.0003).

Table 1. Multivariate analysis of variance for dispersal variance, dispersal distribution shape, fecundity, and their interactions

Treatment	Pillai's Tr	F	d.f.	P
Dispersal variance	0.941	6314.25	10, 3983	<0.0001
Distribution shape	0.440	313.41	10, 3983	<0.0001
Fecundity	0.180	86.94	10, 3983	<0.0001
Variance × shape	0.051	21.25	10, 3983	<0.0001
Variance × fecundity	0.960	16.73	10, 3983	<0.0001
Shape × fecundity	0.004	1.63	10, 3983	0.0906
Variance × shape × fecundity	0.005	1.92	10, 3983	0.0376

Note: Values of Wilks' lambda, Hotelling-Lawley trace, Pillai's trace and Roy's greatest root yield identical values of F and P. Pillai's trace is shown here.

Differences in genetic structure due to dispersal distribution shape and fecundity are small compared with differences caused by dispersal variance (Figs. 2 and 3). Leptokurtosis results in higher average values of I_{50} than platykurtosis (MANOVA test for main effect of shape, $P < 0.0001$; for the shape–variance interaction, $P < 0.0001$; Table 1), but the difference is slight even for nearest neighbours, where the effect is largest (Fig. 2). Similarly, low-fecundity treatments generate more structure than high-fecundity treatments (MANOVA for fecundity, $P < 0.0001$; for the fecundity–variance interaction, $P < 0.0001$; Table 1), but the effect is relatively small (Fig. 3).

A marginally significant interaction between fecundity and shape of the dispersal distribution (MANOVA, $P = 0.0906$; Table 1) is stronger when the two variance treatments are considered separately (MANOVA for three-way interaction, $P = 0.0376$; Table 1). For all distance classes, the extent to which the Laplacian dispersal distribution increases the value of I_{50} over the 'top hat' dispersal distribution is greater at high fecundities than at low fecundities when dispersal variance is low (Fig. 4). When dispersal variance is high, the reverse occurs: the effect of shape is more pronounced at low than at high fecundities (Fig. 4).

Behaviour of Moran's *I* with time

From initial conditions of zero genetic spatial correlation, Moran's *I* for measured lags increases to positive values within the first few generations in all cases. Moran's *I* is highest for short lags and decreases with increasing distance, indicating some clumping of like genotypes in space. In independent model runs, we followed populations to fixation of

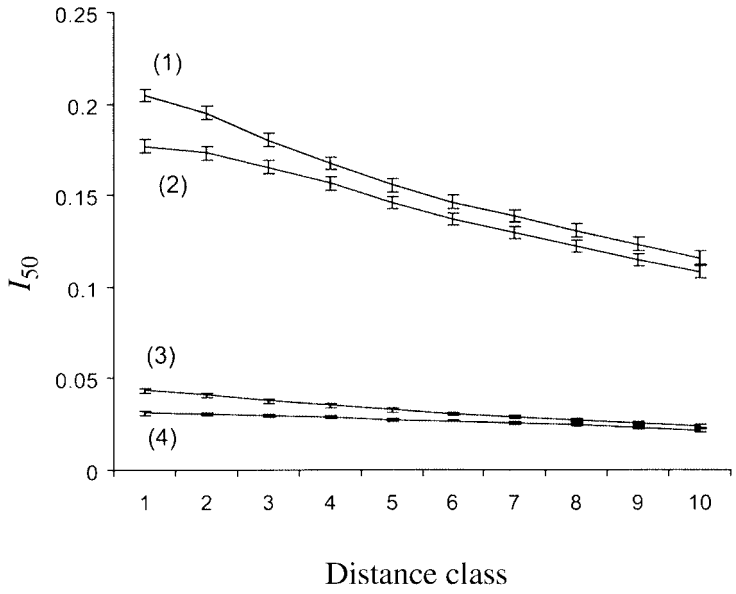

Fig. 2. Restricted variance and leptokurtosis result in higher mean Moran's *I*, measured after 50 simulation generations, for the first ten distance classes. (1) and (2) low dispersal variance; (3) and (4) high dispersal variance; (1) and (3) Laplacian dispersal distribution; (2) and (4) uniform distribution. Error bars show ± one standard error.

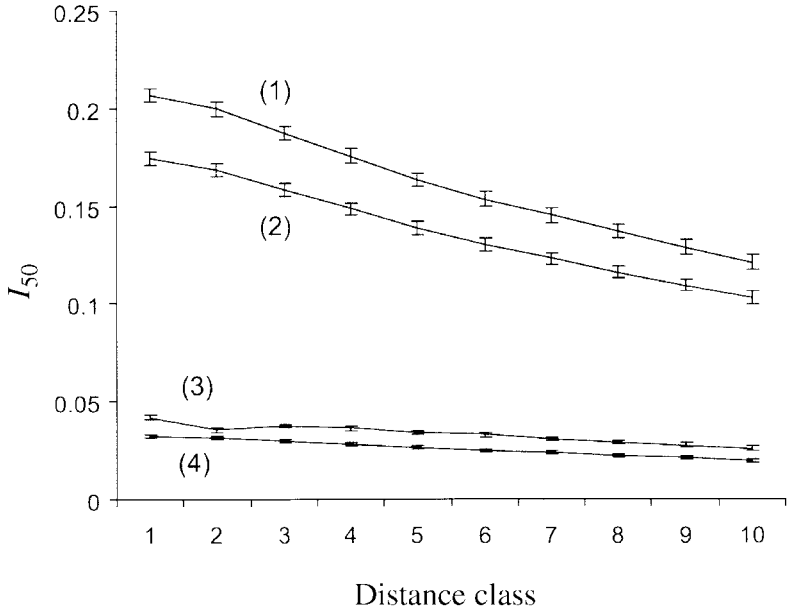

Fig. 3. Restricted variance and lower fecundity generate more genetic structure. (1) and (2) low dispersal variance; (3) and (4) high dispersal variance; (1) and (3) low fecundity; (2) and (4) high fecundity.

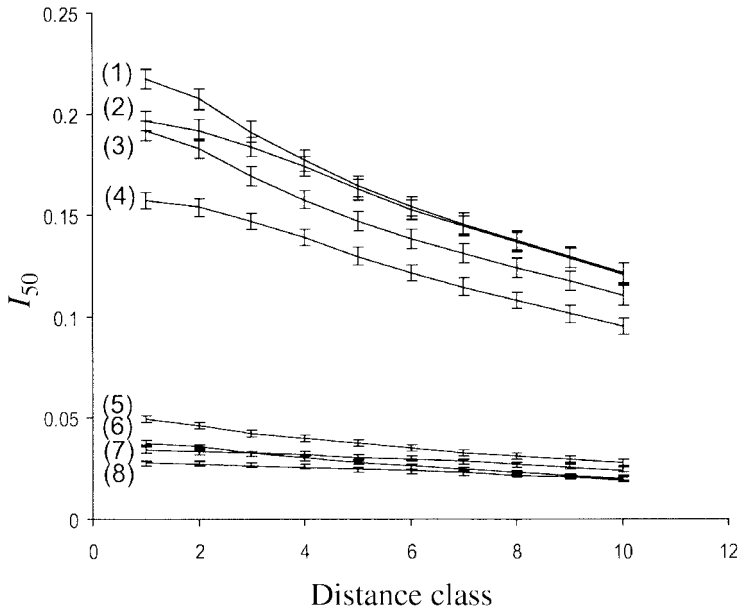

Fig. 4. Variance × shape × fecundity interaction. (1–4) low dispersal variance; (5–8) high dispersal variance. (1) and (2), (5) and (7) low fecundity; (3) and (4), (6) and (8) high fecundity. (1) and (3), (5) and (6) Laplacian dispersal distribution; (2) and (4), (7) and (8) uniform distribution.

either allele to determine how Moran's I for given lags behaves over periods of time longer than 50 generations. In high dispersal variance treatments, Moran's I rises from zero to a low level in the first few generations, where it remains steady until becoming undefined at fixation in 2000 ± 199 generations on average ($n = 40$). In these high-variance runs, I_{50} is representative of I measured at any time not immediately after the start of the simulation or immediately before fixation, and values of Moran's I that are zero or negative occur occasionally as part of random variation around the low quasi-steady value. In individual low-variance treatments, no steady state is achieved: Moran's I increases and then generally decreases somewhat before finally becoming undefined at fixation (284 ± 9.2 generations; $n = 40$). Values of I_{50} correlate strongly, however, with the maximum I achieved in all independent runs (means ± standard errors for nearest neighbours: I_{50} 0.11 ± 0.02, I_{max} 0.24 ± 0.01; Pearson's correlation coefficient: $r = 0.84$; $n = 80$), so we accept I_{50} as a reasonable indicator of the behaviour of Moran's I over longer periods of time. Higher fecundity and platykurtic dispersal delay fixation slightly (means ± standard errors of fixation times: high fecundity 1200 ± 205 generations, low fecundity 1000 ± 184 generations; platykurtic dispersal 1200 ± 199 generations, leptokurtic dispersal 1000 ± 192 generations; $n = 40$ in each case).

DISCUSSION

The idea that spatially limited dispersal should give rise to patchy patterns of genetic relatedness provides ecologists with methods for inferring properties of dispersal from spatial genetic data. As the results reported here confirm, the relationship between dispersal variance and spatial genetic structure is strong: a five-fold increase in dispersal variance results in an approximately five-fold reduction in genetic correlation as measured by Moran's I. These values are in line with the results of previous simulation studies with similar dispersal variances (Doligez *et al.*, 1998; Epperson, 2003). In contrast with situations in disease dynamics, spatial spread and hybridization, however, and with previous studies on non-equilibrium genetic structure, the shape of the dispersal distribution is surprisingly unimportant. This simulation study, with 500 landscapes per treatment, does detect differences in mean I as small as 0.01 due to these factors within high-variance treatments and as small as 0.03 within low-variance treatments. The standard deviations of the distributions of I, however, are large enough (0.02 in high variance and 0.65 in low variance) for these differences to be difficult to detect in a field study, in which only one or a few correlograms would be available for analysis.

Leaving aside for a moment the question of why dispersal distribution shape and fecundity are not very important, we must still understand the directions of their effects. One might intuit that leptokurtic dispersal distributions would result in less genetic structure than platykurtic ones, because the long-distance dispersers in the tails would serve to homogenize the population. Instead, we find that leptokurtosis results in more structure than platykurtosis for a given dispersal variance. This potentially puzzling result is a consequence of the 'clump-generating' properties of leptokurtic dispersal distributions (see, for example, Lewis, 1997). When comparatively more individuals remain in the vicinity of their parents than who travel long distances, clumps of related individuals form over time, increasing genetic structure. Thus, rare long-distance dispersal, which manifests itself in the shape of the dispersal distribution, and dispersal that is long-distance on average, which is reflected in the scale of the distribution, here have opposite effects on neutral genetic structure.

Higher fecundity decreases genetic structure because it decreases clump formation. Clumps of related individuals result from local genetic drift on spatial scales determined by dispersal variance. Just as large population sizes decrease the action of genetic drift within panmictic populations, higher fecundity retards the process of isolation by distance for a given dispersal variance. Finally, the interaction between dispersal shape and fecundity depends on dispersal variance. When variance is restricted, higher fecundity increases the ability of the leptokurtic dispersal distribution to generate clumps, because more individuals are retained close to their parents than under conditions of lower fecundity. When individuals can move long distances on average, however, nascent clumps are accessible to many other individuals that may be of a different genotype. Higher fecundity increases the numbers of these other individuals, thus decreasing the structure-generating power of dispersal kurtosis.

Why are the effects of dispersal kurtosis and fecundity so small? One possible explanation is that Moran's I is not sensitive enough to reveal them. Indeed, Moran's I is only one of many measures for genetic structure (see reviews in Heywood, 1991; Neigel, 1997). We used Moran's I here because it is appropriate for assessing patterns of identity by state within spatially continuous populations. Additionally, its interpretation as a measure of genetic correlation is intuitively accessible, does not depend on other assumptions about demographic or genetic equilibrium, and relates conceptually to previous work that measures genetic structure within or between populations using correlation functions (e.g. Kimura and Weiss, 1964; Malécot, 1969; Lande, 1991). Moran's I-statistics can be useful for analysis of multiple alleles or multiple loci, and several authors have derived relationships between I and other measures of genetic structure (Epperson, 2004). Finally, despite the quite variable values of I generated in this study from different realizations of the same ecological and genetic processes, the measure is sensitive enough to reveal changes in structure due to differences in dispersal variance. Other measures would have to be much more powerful to detect the smaller differences due to dispersal kurtosis and fecundity reliably. Alternatively, better measures might rely on aspects of genetic structure other than spatial correlation (for some alternatives, see Cain *et al.*, 2000; Nathan *et al.*, 2003).

Another possibility is that the differences in dispersal distribution shape and in fecundity that we explored here are too small. Indeed, in the case of distribution kurtosis, empirical dispersal distributions may be so leptokurtic that exponential distributions provide a poor fit (Portnoy and Willson, 1993; Kot *et al.*, 1996; Clark *et al.*, 1999; Bullock and Clarke, 2000). Chave and Leigh (2002) reported a difference in equilibrium spatial structure when dispersal occurs according to an extremely fat-tailed Cauchy rather than a Gaussian distribution, although the Cauchy's infinite variance makes direct comparison to a Gaussian difficult. For expanding populations, Le Corre *et al.* (1997) and Austerlitz and Garnier-Géré (2003) report a dramatic effect of extremely kurtotic distributions in a situation where variance is similarly uncontrolled. Ibrahim *et al.* (1996) report that much less non-equilibrium structuring results from Gaussian dispersal than from a distribution with comparable variance and a kurtosis of about 17. These results suggest that more extreme dispersal kurtosis than we considered here might influence genetic structure. On the other hand, an analytic model that systematically explores the effects of more extreme dispersal distributions in an infinite (non-expanding) population reveals that kurtosis differences on the order of hundreds is necessary to affect genetic structure as much as dispersal variance does here, and that increasing kurtosis beyond a moderate value actually results in less structure (Lee, 2002).

The small effects reported here for treatments other than dispersal variance provide clues as to the conditions under which long-distance dispersal is and is not important. In cases where the shape of the dispersal distribution has proved important, long-distance dispersers experience environments that are very different from the ones they leave behind, even in the absence of explicit distance-dependent selective forces. In spatial population spread, disease dynamics and host–parasitoid pattern formation, the rare individual that travels a long way may escape density dependence or colonize new patches of susceptible hosts, consequently enjoying an advantage over less mobile individuals. In these situations, being the first to reach a new location is much better than arriving after the location has been occupied by conspecifics, and long-distance dispersal therefore has profound population consequences. These advantages to long-distance dispersal are not present in the situation modelled here, where genes that are not subject to any selective forces redistribute within a population that is initially more or less uniformly distributed in space. We expect that the introduction to this model of some types of self-incompatibility, or of selection for or against heterozygotes, would result in advantages or disadvantages to long-distance dispersal and yield greater differences between dispersal distributions.

Introducing greater spatial patterning in density might also increase the effects of dispersal kurtosis. We suggest that Ibrahim *et al.* (1996), Le Corre *et al.* (1997) and Austerlitz and Garnier-Géré (2003) detect greater patterning due to kurtotic dispersal because their populations are undergoing active spatial expansion, where long-distance dispersers enjoy release from density dependence as explained above. As these authors point out, leptokurtic dispersal in a colonization setting results in multiple local founder events, which drives initial patchiness in genetic relatedness (see also Kot *et al.*, 1996; Lewis and Pacala, 2000). Over time, however, as the habitat fills up, Ibrahim *et al.* (1996) show that repeated leptokurtic dispersal results in a faster decay of patchiness than does Gaussian dispersal. This decay after the end of the colonization phase is consistent with the result that more extreme kurtosis reduces patterning in a non-expanding population (Lee, 2002).

We tested the idea that spatial population spread can increase the effects of dispersal kurtosis on genetic patterning by running our model with a different set of initial conditions. Instead of distributing the 1000 initial individuals randomly across the landscape, we distributed them randomly within the 1000 grid points forming a 10-point-deep strip along one of the landscape's edges. As expected, because this situation provides more empty space for the population to spread, more genetic structure results in each treatment. Dispersal variance still has the largest effect (means ± standard errors of I_{50} for first lag: low variance 0.4008 ± 0.0101, high variance 0.0694 ± 0.0038; $n = 200$ for each group), but the differences between dispersal shape treatments are larger (0.06 in the low-variance treatment and 0.05 in the high-variance treatment, as opposed to 0.03 and 0.01, respectively, previously (Fig. 2)). These results confirm the primacy of dispersal variance in determining spatial patterning, but also confirm that a population's colonization history can affect non-equilibrium genetic structure via the influence of spatial population expansion. Over time, the mean I for samples initialized with different initial conditions should converge to a quasi-stationary value (Sokal and Wartenberg, 1983), but they follow different trajectories and may take different amounts of time to converge. Together the two contrasting trajectories demonstrate that, in non-equilibrium situations, strategic choices of initial conditions and examination of system dynamics can provide extremely useful information regarding the operation of ecological processes.

In conclusion, we have demonstrated that non-equilibrium spatial correlation in neutral genes is more sensitive to changes in dispersal variance than to moderate changes in the kurtosis of dispersal, though the action of a process such as active spatial expansion can accentuate the effects of the shape of the dispersal distribution. This finding means that genetic correlations are unlikely to be helpful for inferring the shape of dispersal distributions. On the other hand, this work confirms that inferences of dispersal variance from genetic structure (e.g. Epperson, 2003) are valid despite discrepancies between the dispersal assumed by models and the dispersal that occurs in nature. Measurements of dispersal variance from genetic data are particularly informative, therefore, in situations where the tails of dispersal distributions are not important, as in the design of marine reserves (Lockwood *et al.*, 2002).

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