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Competition enhances spatial genetic differentiation

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ABSTRACT

Questions: How does the number of species in a community affect the level of genetic diversity of its constituent species? What is the relation between competitive interactions and the spatial distribution of genetic variation? When spatially structured, how is the genetic differentiation among subpopulations affected by competition and size of the community?

Model features and key assumptions: A model combining one-locus two-allele genetics with density-dependent regulation in population renewal (Ricker function) and demographic stochasticity is extended to incorporate *S*-species Lotka-Volterra competition. The system is embedded into a spatial context where 1000 populations are connected with stepping-stone dispersal. Beginning from random genotype composition, the system is simulated and the resulting community composition and genetic diversity across space are recorded.

Conclusions: Genetic differentiation in the ensemble increases with the number of competing species in the community (0.30 ± 0.13, normalized coefficient with 95% confidence limit) and with the intensity of pair-wise competition (0.20 ± 0.05) but most strongly with their interaction (0.62 ± 0.22). Although the system-wide differentiation increases, one finds that the process leads to local paucity of genotypes and hence a negative correlation between species diversity and local genetic diversity.

Keywords: community genetics, competition, F_{ST}, Lotka-Volterra, spatial structure, species diversity, stepping-stone.

INTRODUCTION

A major aim of conservation biology is to maintain, and even to restore, biological diversity. This is genuinely a challenging task, as there are at least two differing meanings of biological diversity. Ecologists study the number of species (e.g. Preston, 1948; Diamond and Case, 1986): the focus is on species assemblages (communities), habitats, and even on ecosystems. In turn, geneticists focus on genetic differences among individuals in a population: the number of genotypes, level of heterozygosity or frequency of rare alleles (Frankham *et al.*, 2002).

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Ecologists study the causes and consequences of population fluctuations that result from local (births and deaths) and larger-scale processes (immigration, emigration) influencing population renewal (Ranta *et al.*, 2006). Often individuals are assumed equal, allowing at best age-group related differences in vital rates. These, together with external (e.g. climate) forcing, result in the observed fluctuations in population time series (augmented by sampling error). When extending to more than one-species systems, the significance of biotic interactions (e.g. competition, predation, diseases) in moulding diversity enters the picture.

Geneticists are ultimately concerned with maintenance of the adaptive potential of populations (Lande and Shannon, 1996; Willi *et al.*, 2006) and the avoidance of inbreeding depression (Crnokrak and Roff, 1999; Hedrick, 2000; Keller and Waller, 2002). Seen from this perspective, the essential processes include the dynamic distribution of genotypes and the rate of loss of genetic diversity. The level of individual variation is often investigated empirically using neutral genetic markers or quantitative traits. Both demographic and environmental processes determining the extent of fluctuations in population sizes have an obvious influence on the effective genetic population size (Ewens, 1982; Caballero, 1994; Vucetich *et al.*, 1997; Kaitala *et al.*, 2006; Turner *et al.*, 2006).

Both ecological and genetic approaches often take the spatial dimension for granted. Nonetheless, we know that by increasing the area of samples, one also increases the number of different kinds of biological entities encountered. Hence, with increasing area, biological diversity should increase. But there may also be contrasting elements in the system. For example, processes maintaining low diversity at a local level (e.g. drift) might result in the presence of different species or different genetic structure at different locations. When several spatial units are pooled, the composite diversity is likely to be richer than that of the components. However, only recently has the genetic perspective been applied to communities with a collection of species (see Vellend and Geber, 2005 and references therein).

Our scope here is to address the relationship between number of species and genetic differentiation in a community of S competing species. The system is put into a space of n population units in a string joined with stepping-stone dispersal (corresponding to dispersal between nearest neighbours). For a population genetic model, we begin with the classical one-locus two-allele system for each of the S species. The three genotypes in each species are assumed to be neutral.

MODEL OF POPULATION DYNAMICS, GENETICS, AND COMPETITION

How might intraspecific competition on limited resources among S species influence genetic differentiation in spatially structured populations? Our model incorporates density dependence in the renewal process. A simple one-locus two-allele (alleles 1 and 2) sub-model represents the genetic system. We denote the numbers of parent individuals of three different genotypes of species s in location i at time t with $X^{11}_{t,s,i}$, $X^{12}_{t,s,i}$, and $X^{22}_{t,s,i}$, where $X^{TOT}_{i,s,t}$ is their total. The model here is a multi-species extension of that of Kaitala *et al.* (2006) and Ranta *et al.* (2008). Within each species, the three genotypes are phenotypically identical and not subject to mutation or selection. The Ricker model (May, 1973) serves as the kernel for the density-dependent renewal, with r (here we use $r = 0.5$ but our results hold for other values of $r < 2.7$) as the intrinsic growth rate and K_s (here we set $K = 1$ for all species in each unit) as the carrying capacity of a local sub-unit (both are assumed equal for each unit):

$$X^{TOT}_{t+1,s,i} = X^{TOT}_{t,s,i} \exp\left[r_s \left(1 - \frac{X^{TOT}_{t,s,i} + \sum_{j=1, j\neq s}^{S} \alpha X^{TOT}_{t,s,i}}{K_s} \right) \right]. \quad (1)$$

Here the S species are otherwise identical but the term α_{ij} indicates how intense resource competition is (0 no competition, 1 the species are identical); we use the following values for α_{ij}: 0, 0.25, 0.5, and 0.75.

For each species, the individual genotype frequencies in the mating generation are $p^{11}_{t,s,i}$, $p^{12}_{t,s,i}$, and $p^{22}_{t,s,i}$ at time t in sub-population i. The species-specific total amount of resources to produce offspring is $\Phi_{s,i,t}$. Following Kaitala *et al.* (2006), but extending for S species, the expected number of offspring from each possible pairing within the species s is given as:

$$\begin{array}{cccc} & 11_s & 12_s & 22_s \\ 11_s & (p^{11}_{t,s,i})^2 \Phi_{s,i,t} & p^{11}_{t,s,i} p^{12}_{t,s,i} \Phi_{s,i,t} & p^{11}_{t,s,i} p^{22}_{t,s,i} \Phi_{s,i,t} \\ 12_s & p^{11}_{t,s,i} p^{12}_{t,s,i} \Phi_{s,i,t} & (p^{12}_{t,s,i})^2 \Phi_{s,i,t} & p^{12}_{t,s,i} p^{22}_{t,s,i} \Phi_{s,i,t} \\ 22_s & p^{11}_{t,s,i} p^{22}_{t,s,i} \Phi_{s,i,t} & p^{12}_{t,s,i} p^{22}_{t,s,i} \Phi_{s,i,t} & (p^{22}_{t,s,i})^2 \Phi_{s,i,t} \end{array} \quad (2)$$

Keeping species separate, we next pool, for each location i, the total number of genotypes arising from the different pairings of parent genotypes. Here local carrying capacity is small ($K = 50$), but the number of local units – tied to redistribution of individuals – is large ($n = 1000$). It follows that demographic stochasticity (e.g. May, 1973) plays an important role locally. To achieve the effect we take

$$\tilde{X}^{\bullet}_{t+1,s,i} = Poisson(X^{\bullet}_{t+1,s,i}), \quad (3)$$

where $\bullet$ denotes genotypes 11_s, 12_s, and 22_s, and ~ is for population size prior to redistribution. This procedure makes use of the deterministic genotype numbers as expectations of the Poisson random process, and the Poisson operator ensures that we are dealing with integer individuals.

Local habitable units make a string that has bouncing boundaries at the two ends (not a critical assumption). Redistribution of individuals in the basic setting occurs after reproduction and $(1 - m)$ $X^{\bullet}_{t+1,s,i}$ remains in the natal population i, while the dispersers go to the two neighbouring units (half to $i - 1$ and the other half to $i + 1$). Thus, the redistribution obeys the stepping-stone model, used in population genetic models ever since Kimura (1953) introduced it.

To illustrate the emergence of local allele fixation, the model (equations 1, 2, and 3) was initialized with each sub-unit receiving random numbers ($< K$) of individuals of either of the homozygous genotypes. The numbers of individuals in the two remaining genotypes were determined using the Hardy-Weinberg equilibrium. The system was left running for 1000 generations. At the termination of the runs, we scored, for a randomly selected focal species, the F_{ST} statistic as a measure of genetic structuring among the n units (e.g. Hartl and Clark, 1997). This measure (ranging from 0 to 1) is higher the more pronounced the genetic differentiation is among the units studied. Of course, the F_{ST} values keep changing even after $t = 1000$ but the rate of change slows dramatically quite early in development (Kaitala *et al.*, 2006). We also rely on the fact that we sample the data consistently using a two-factor design and vary one factor at one level per run, with runs replicated.

RESULTS AND DISCUSSION

We built a model that combines one-locus two-allele genetics with density-dependent regulation in the population renewal (Ricker function) with demographic stochasticity. The system is embedded into a spatial context, where $n = 1000$ populations are connected with stepping-stone dispersal. The model incorporates S-species Lotka-Volterra competition. At the termination of the runs ($t = 1000$), genetic differentiation was scored using the F_{ST} statistic (e.g. Hartl and Clark, 1997).

The output of one simulation of the model [equations 1–3; with $n = 1000$ units, and two species ($\alpha_{ij} = \alpha_{ji} = 0.5$), terminated at $t = 1000$] is given as an example in Fig. 1. A notable tendency is that there are regions where the homozygous genotypes (11 and 22) tend to dominate in numbers (Fig. 1). The results reflect those observed in a single-species version of the present model (Kaitala *et al.*, 2006), where regional allele fixation without global loss of alleles is the characteristic feature of spatial genetic differentiation. From Kaitala *et al.* (2006), we also know that evolution of clearly pronounced allele fixation regions, with heterozygous areas in between, take quite a time to evolve (although fingerprints of such patterns are clearly observable with stepping-stone dispersal as early as $t = 100$–500).

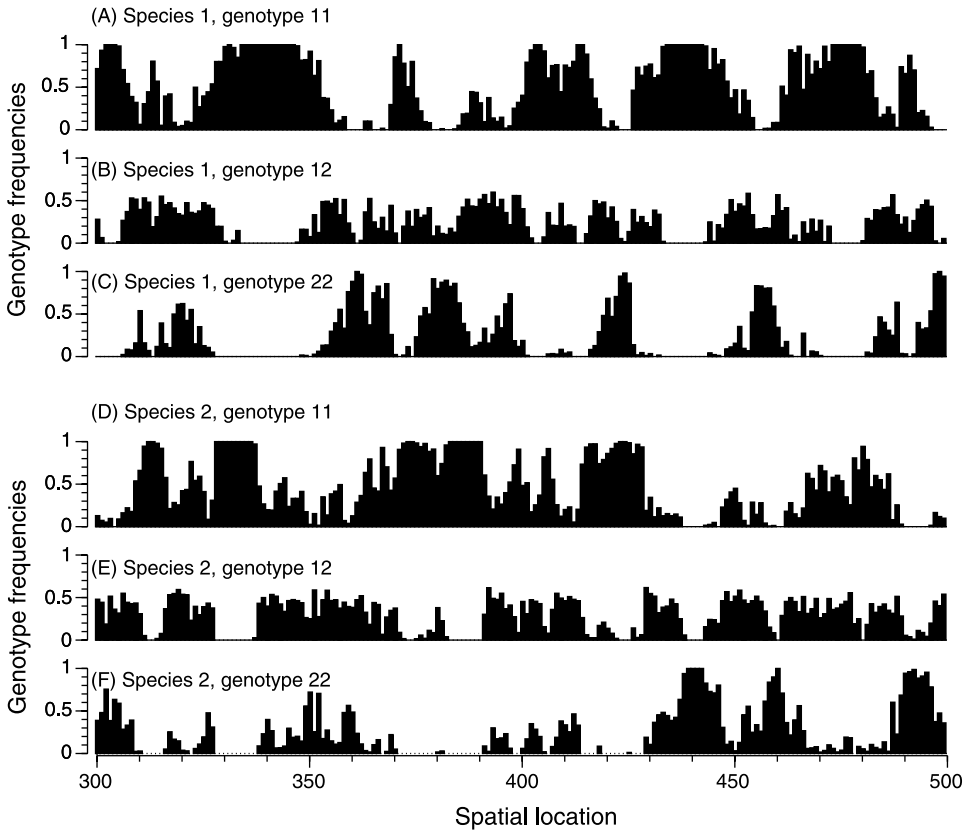

Fig. 1. Genotype frequencies in species 1 and 2 (at $t = 1000$) shown for population units in spatial locations from 301 to 500.

Examining the species-specific heterozygote scores in this two-species system at $t = 1000$ reveals a clear bimodality (peaks at zero and close to 0.5), suggesting that even this system, given long enough, would evolve towards more clearly pronounced spatial segregation of the two alleles in both species (Fig. 2A,B). An examination of the spatial-unit-specific heterozygous scores in the two species reveals that the spatial genetic differentiation in one species is entirely independent of the presence of another species (Fig. 2C).

To explore the significance of intensity of competition and the number of species in the Lotka-Volterra competition, the two parameters α_{ij} and S were allowed to vary ($\alpha_{ij} = 0$, 0.25, 0.5, and 0.75; $S = 1$, 2, 3, 4, and 5). As $n = 1000$ and $t = 1000$ at the end of a run, the computations are time-consuming (especially with $S > 2$). Therefore, for each value of S and α_{ij} used, we replicated the procedure ten times and subjected the F_{ST} data to an analysis of variance (ANOVA) with α_{ij} and S as factors. The results are rather straightforward [note that when $S = 1$ (no competition), F_{ST} is about 0.48) (Table 1). Genetic differentiation (F_{ST}) among the population units increases not only with increasing number of species in the Lotka-Volterra competitive community, but also with increasing intensity of the interactions in pairs, α_{ij} (Fig. 3). Calculating normalized coefficients gives 0.30 ± 0.13 ($\pm$ 95% confidence limits) for species, 0.20 ± 0.05 for the intensity of pair-wise competition, and 0.62 ± 0.22 for the interaction of the two terms.

The observed acceleration of genetic differentiation as a result of larger numbers of species or intensified competition means that local populations can become more prone to genetic drift and thus local loss of alleles. Effective population size (N_E) decreases locally with increasing competition, but in addition there is an effect of smaller gene flow as the neighbouring sites have an increased probability of being uninhabited. Because the rate of loss of heterozygosity depends on the local effective population size affected by the gene flow, we can expect greater genetic differentiation to accompany stronger competition. For example, in a two-species case with $N_1 = N_2 = 50$, and initial $H_0 = 0.5$, we find that after 100 generations heterozygosity decreases to 0.18, 0.11, 0.068, and 0.041 for $\alpha = 0$, 0.25, 0.5, and

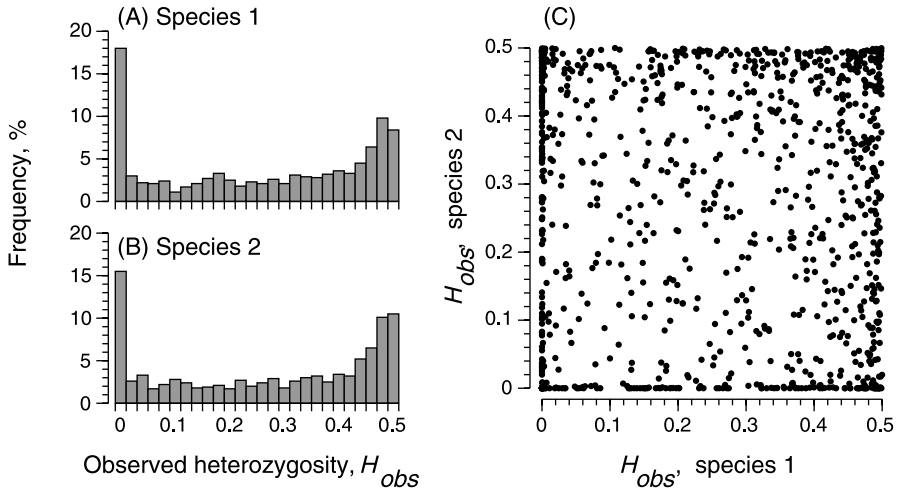

Fig. 2. Frequency distributions of observed heterozygotes in species 1 (A) and 2 (B) in a stepping-stone distribution system of two competing species in a universe of $n = 1000$ units. (C) The same data in a scatter plot.

Table 1. A two-factor ANOVA table for assessing the significance of the number of species ($S = 2$, 3, 4, and 5) and intensity competition ($\alpha_{ij} = 0.25$, 0.5, and 0.75) for genetic differentiation, measured in terms of F_{ST}

Source	SS	d.f.	MS	F	P
Species	0.419	3	0.140	187.1	<0.001
α_{ij}	1.974	2	0.987	987.0	<0.001
Species × α_{ij}	0.179	6	0.030	30.0	<0.001
Error	0.107	144	0.001		
Total	68.438	160			

Note: The following statistics are indicated: SS, sum of squares (type III); d.f., degrees of freedom; MS, mean square; F, ratio and its statistical significance, P.

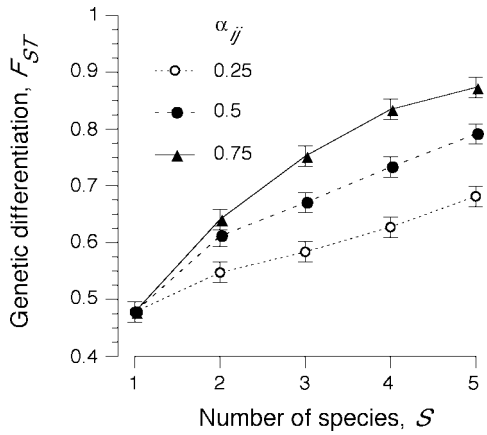

Fig. 3. Genetic differentiation, F_{ST} (e.g. Hartl and Clark, 1997), among the $n = 1000$ units at $t = 1000$. The data are averages (with 95% confidence limits) for ten replicated runs per number of competing species and the intensity of interspecific competition, α_{ij}. Details of the dispersal-structured system, density dependence in renewal, demographic stochasticity, population genetic model, and Lotka-Volterra competition in the community of S species are given in the text.

0.75 respectively. In other words, higher values of α accelerate the loss of heterozygosity, corresponding to depression of the local effective population size. In addition, however, local extinction prevents gene flow. Together, these factors will interact to increase the spatial genetic differentiation (higher F_{ST}). We expect that non-neutral genetic variation may also be eroded by drift before it has a chance to serve as the basis for divergent selection. Non-neutral genetic variation is compulsory for selection to drive species in differing directions in the competitive dimensions. One would therefore expect an upper limit to the capacity for species-packing and competitive intensity when it comes to reliance on divergent selection as the agent promoting co-existence. In contrast, our results suggest an increased potential for divergence among competitors, stemming from random genetic effects (although our mechanism should be a much less efficient force of divergence). This is clearly an issue deserving further research.

Theory provides examples of the formation of both clusters and sharp spatial borders between genotypes/populations. In general, these theories come in two versions, one in the

context of speciation and one in the context of environmental gradients. In the context of speciation, Gavrilets (2004 and references therein) used different spatial models and introduced mutation(s) that created reproductive incompatibility. Using this approach, he was able to show that clusters of reproductively isolated groups would evolve despite gene flow. He also showed that the spatial structure itself is of importance. In the second approach, several researchers – starting with Endler (1977) and summarized in Gavrilets (2004) – have studied the genetic structure of populations along an environmental gradient. Surprisingly, the genetic structure evolves into clusters with sharp boundaries despite the continuous gradient. In both these cases, selection was invoked: in the first case, selection against heterozygotes due to a built-in barrier to certain genotype pairings, and in the second case, selection along the gradient. Neither kind of selection is present in this study; the model has only competition between species. Thus, we can show that clustering can evolve as an emergent property by drift (and dispersal) alone as a result of competition.

Although the literature on community genetics is far from overwhelming, recent studies focusing on the relationship between species diversity and genetic diversity suggest a positive correlation between genetic variability and species diversity (Vellend and Geber, 2005; Vellend, 2006; Wehenkel *et al.*, 2006). Our results open the possibility for negative correlation between the number of species (and the strength of their competition) and genetic diversity. But we note that strongly differentiated local populations are associated with increased random drift and are therefore prone to loss of genetic variability due to a lowered effective population size. On the positive side, however, are indications from empirical studies showing that genotypic diversity may be able to replace the role of species diversity as a buffer against extreme climate events through enhanced population recovery (Hughes and Stachowicz, 2004; Reusch *et al.*, 2005).

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