



Population structure inhibits evolutionary diversification under competition for resources

Troy Day

Department of Zoology, University of Toronto, 25 Harbord St., Toronto, ON, Canada M5S 3G5 (Phone: (416) 946-5563; Fax: (416) 978-8532; E-mail: dayt@zoo.utoronto.ca)

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Abstract

A model is presented that explores how population structure affects the evolutionary outcome of ecological competition for resources. The model assumes that competition for resources occurs within groups of a finite number of individuals (interaction groups), and that limited dispersal of individuals between groups (according to Wright's island model of population structure) results in genetic structuring of the population. It is found that both finite-sized interaction groups and limited dispersal can have substantial effects on the evolution of resource exploitation strategies as compared to models with a single, infinitely large, well-mixed interaction group. Both effects, in general, tend to select for less aggressive competitive strategies. Moreover, both effects also tend to reduce the likelihood of the evolutionary diversification of resource exploitation strategies that often occurs in models of resource competition with infinite populations. The results are discussed in the context of theories of the evolutionary diversification of resource exploitation strategies and speciation.

Introduction

The role that microevolutionary processes such as natural selection play in the process of speciation has long been debated. One of the primary ways in which natural selection has been implicated in speciation is through the evolutionary reinforcement of partial reproductive isolation between incipient species (Dobzhansky, 1940; Butlin, 1989; Howard, 1993). In particular, it has been suggested that if two groups of organisms produce hybrid offspring through interbreeding, but that these offspring suffer some degree of reduced fitness, then natural selection will favour the evolution of additional isolating mechanisms that reinforce this partial isolation. This hybrid disadvantage is often considered to occur as a result of intrinsic genetic incompatibilities that build up during a period of geographic isolation followed by secondary contact (Coyne & Orr, 1998), but it is also possible that hybrid disadvantage is mediated through ecological interactions, and occurs in the absence of any geographic isolation (Coyne & Orr, 1998; Schluter, 1998).

In fact, one view of how sympatric speciation might be initiated is that ecologically mediated disruptive selection favours two extreme types, and that the intermediate forms that result from hybridization suffer from being maladapted to the environment (Maynard Smith, 1966; Rice & Hostert, 1993; Bush, 1994; Doebeli, 1996; Johnson & Gullberg, 1998 and references therein). In other words, the ecological environment contains two distinct niches (two adaptive peaks on the fitness landscape), and the resulting disruptive selection promotes the evolution of two species.

In his 1959 paper, Hutchinson focused attention on such ecological issues by considering how they might set an upper limit for the number of animal species that coexist. Later Felsenstein (1981) suggested that, although such ecological processes likely do set an upper limit, evolution might not produce enough species to attain this limit. One of Felsenstein's main points was that the number of species that actually occur in nature is far smaller than we would expect based on a purely ecological explanation, and therefore he sought to find an explanation for why this

might be so. His answer was that genetic constraints arising from a tension between linkage disequilibrium and recombination between loci associated with adaptation and those associated with assortative mating can often prevent the occurrence of speciation, even in the face of disruptive selection. In essence, speciation requires a statistical association to build up between alleles involved with adaptation and those involved in assortative mating, and recombination between loci continually erodes this association. This study was highly influential, and it highlighted two important phases of evolution that occur during the process of speciation in the absence of geographical isolation. First, disruptive selection sets the stage for speciation by driving the evolution of two extreme types. Second, reproductive isolation must then evolve between these two types to complete the process of speciation (Kondrashov & Mina, 1986; Johnson & Gullberg, 1998). It is this second phase that is hindered by the genetic constraints highlighted by Felsenstein.

Although Felsenstein's paper cast some doubt on the plausibility of speciation in the absence of geographic isolation, several more recent theoretical studies seem to reaffirm the idea that such sympatric speciation can easily occur (Doebeli, 1996; Law et al., 1997; Kondrashov et al., 1998; Dieckmann & Doebeli, 1999; Kisdi, 1999; Kondrashov & Kondrashov, 1999; Doebeli & Dieckmann, 2000; Gertiz & Kisdi, 2000). Not all of these studies deal with speciation *per se* according to the biological species concept (Mayr, 1963) because some deal with asexual organisms, but much of this recent work has again focussed on the role that ecological interactions play in generating disruptive selection, and on how this disruptive selection might then, in turn, ultimately lead to the evolution of reproductive isolation in sexual species. The central idea of these recent theories builds upon Rosenzweig's (1978) notion of competitive speciation. Competition for resources will first cause a single population to evolve a strategy that best exploits the most abundant resource available. At this point, however, if competition is strong enough, then selection will favour individuals that specialize on extreme resources because they experience reduced competition. Thus disruptive selection is generated endogenously by the ecological interactions among individuals (Doebeli & Dieckmann, 2000). This disruptive selection can then result in speciation if reproductive isolation evolves (e.g., Dieckmann & Doebeli, 1999).

There is growing evidence that disruptive selection is relatively common in natural populations (reviewed

in Endler, 1986; Kingsolver et al., *in press*), and that it can play an important role in the evolution of reproductive isolation in some organisms (Rice & Hostert 1993; Feder, 1998; Schluter, 1998). Although it is not yet clear how many of these empirical examples are instances of the kind of ecologically mediated disruptive selection characteristic of competitive speciation, one of the most interesting conclusions of this recent theory is that we might often expect natural selection to drive the evolution of single populations to phenotypic values at which disruptive selection occurs. Therefore this process of speciation in the absence of geographic isolation might be quite common in nature. Indeed, in many of these models speciation seems to occur quite easily, suggesting that a reconsideration of Felsenstein's original question might be worthwhile. If such ecological factors do play a pre-eminent role in evolutionary diversification and speciation, then why are not more species found in nature? What limits this process of seemingly inevitable diversification and speciation that is seen in some of these models (Bridle & Jiggins, 2000)?

As already mentioned, Felsenstein's original answer to this question was a genetic one. My intention here is to suggest that, for these recent models similar to Rosenzweig's (1978) notion of competition speciation, another partial answer might be found at the interface between ecology and genetics. Much of the recent theory demonstrating that competitive interactions can result in disruptive selection and thereby drive speciation is based on models that assume competitive interactions take place among all individuals of an effectively infinite population, and that the population is 'well-mixed' (e.g., Doebeli & Dieckmann, 2000). Here I present a very simple model of resource competition that allows competitive interactions to take place within finite-sized 'interaction groups', and that also relaxes the assumption that the population is well-mixed by allowing individuals to exhibit limited dispersal. I use this model to ask two questions. (1) Is the resource exploitation strategy that evolves under competition affected by these realistic biological features? (2) Is competition for resources more or less likely to result in disruptive selection once these factors are incorporated? The combination of finite-sized interaction groups and the population viscosity that results from limited dispersal, causes populations to become genetically structured. If such structuring makes the disruptive selection that arises from competition for resources less likely, then this interaction between genetics and ecology provides one possible

factor that, when incorporated into models of competitive speciation would reduce the frequency with which they predict speciation. The difference between this type of mechanism and that of Felsenstein is that the present mechanism would reduce the extent to which disruptive selection occurs thereby preventing the first phase of sympatric speciation (selection favouring extreme types), whereas Felsenstein's mechanism prevents the evolution of reproductive isolation once disruptive selection is present (Kondrashov & Mina, 1986; Johnson & Gullberg, 1998).

Theoretical development

Modeling approach and method of analysis

I take a very simple game-theoretic approach. In particular, I consider competition for resources among individuals of a single species and I determine the conditions that must be satisfied for there to be a single, evolutionarily stable resource exploitation strategy (i.e., an ESS). This analysis is used for two purposes. First, it is used to determine how such ESS's are affected by the genetic structuring of the population which is inherent in the model (to be described shortly). The answer to this question provides some insight into how such population structure can affect the direction of evolution of a single species in which there is competition for resources. Second, the analysis is used to determine how the conditions necessary for there to be an ESS with a single type of consumer, are affected by this population structure. The answer to this second question is important because when an ESS with a single type of consumer does *not* exist, we then expect that the competition for resources within the species results in disruptive selection, favouring an evolutionary diversification of strategies. It is under such conditions that phenomena like character divergence and speciation might occur (Johnson & Gullberg, 1998).

To derive conditions that characterize an ESS we first need to formulate an expression for the fitness of an adult with a rare mutant phenotype, x , in a population with resident phenotype, \hat{y} (Taylor & Frank, 1996) (which I denote by $W(x, \hat{y})$). Given this fitness function, an ESS is then a strategy which is uninvadable by all alternative strategies (i.e., all alternative strategies have a lower fitness). If y^* is an ESS, then mathematically this requires that $W(x, y^*) \leq W(y^*, y^*)$ for all $x \neq y^*$. This is a global condition

stating that W must be maximized in x at $x = y^*$ when the predominate population strategy is y^* . It is usually much easier, however, to restrict attention to local ESS's by working with the corresponding local conditions,

$$\left. \frac{\partial W}{\partial x} \right|_{x=\hat{y}=y^*} = 0, \quad (1)$$

$$\left. \frac{\partial^2 W}{\partial x^2} \right|_{x=\hat{y}=y^*} < 0. \quad (2)$$

Condition (1) will be referred to as the equilibrium condition since when it holds, directional selection ceases ($\partial W / \partial x|_{x=\hat{y}=y^*}$ is a measure of the strength of directional selection). Strategies that satisfy (1) have also been termed 'evolutionarily singular strategies' by some authors (Geritz et al., 1998) since they are not necessarily ESS's. To be an ESS, y^* must also satisfy condition (2) (which guarantees that W is maximized rather than minimized), and therefore I will refer to this as the ESS condition. When the ESS condition is *not* satisfied, then selection becomes disruptive at these singular points, and we therefore expect some form of evolutionary diversification. In asexual populations this is always evolutionary branching, but in sexual populations the kinds of evolutionary diversification that can occur are more varied (Christiansen, 1991; Abrams et al., 1993; Taylor & Day, 1997; Geritz et al., 1998 and references therein).

Notice, however, that the above analysis is meaningful only if directional selection *near* the evolutionary equilibrium actually drives the population towards it since it is only such equilibria that the population will experience. This means that we require directional selection to be positive (favouring larger values of y) when $\hat{y} < y^*$ and for it to be negative (favouring smaller values of y) when $y^* < \hat{y}$. This implies that the derivative of $\partial W(x, \hat{y}) / \partial x|_{x=\hat{y}}$ with respect to \hat{y} is negative at $\hat{y} = y^*$ (Eshel, 1983; Taylor, 1989; Christiansen, 1991); that is,

$$\left. \frac{d}{d\hat{y}} \left\{ \left. \frac{\partial W}{\partial x} \right|_{x=\hat{y}} \right\} \right|_{\hat{y}=y^*} < 0. \quad (3)$$

I will refer to condition (3) as the convergence stability condition. Some authors (Geritz et al., 1998) have termed values of y that satisfy conditions (1) and (3) but that do not satisfy the ESS condition (2) 'evolutionary branching points' since natural selection drives the population towards these strategies, at which point selection becomes disruptive, sometimes resulting in phenomena like sympatric speciation.

A model of resource competition

I consider a model of a haploid asexual organism in a very large (effectively infinite) population, structured into patches containing exactly N individuals each (i.e., Wright's island model of population structure). It might seem paradoxical to construct a model that purports to have implications for theories of speciation by using an assumption of asexuality, but my goal here is to explore how population structure affects the form of selection that results from competition for resources rather than to explore speciation itself. An assumption of asexuality is well suited to this goal since (providing there is ample genetic variation as I assume below) the long-term course of evolution is then largely determined by selection. It is quite conceivable that the quantitative details of the results presented below will be altered for a diploid, sexual organism, since restricted movement of individuals (i.e., partial dispersal in an island model) causes genetic structuring of the population, and the exact nature of this structuring may depend on the genetic system under consideration. Nevertheless, the qualitative nature of this structuring should be similar across different genetic systems, and the main objective here is to qualitatively explore how this genetic structuring interacts with the ecological phenomenon of competition for resources to determine the form of selection, and thus the potential outcome of evolution.

The life cycle of the organism occurs as follows. Each generation the N adults compete for resources locally within each patch, and each adult produces a large number of offspring in accordance with its competitive ability (the precise form of competition will be described shortly). All patches are assumed to contain an identical resource distribution in the absence of any consumers. Following reproduction, a proportion, d , of the offspring disperse globally, and each survives dispersal with probability $1 - c$ (c is the cost of dispersal). The surviving dispersers then settle at random on some patch in the population. The remaining proportion, $1 - d$, of offspring stay on their natal patch. All adults survive to the next year with probability s , in which case they retain their spot on the patch with certainty. There is then a process of genotype-independent culling in which the population size on each patch is reduced back to N . In this process, the probability of any individual obtaining one of the newly vacated spots is inversely proportional to the number of individuals competing for that spot. The life cycle then begins anew.

Within each patch there is a distribution of resources that can be indexed along a one-dimensional continuous axis, with all patches having the same distribution. For example, resources might be characterized by their size. The abundance of resources of type z in each patch is denoted by $K(z)$, and following much previous work on evolution under resource competition (e.g., Slatkin, 1980; Rummel & Roughgarden, 1985; Brown & Vincent, 1987; Taper & Case, 1992; Vincent et al., 1993; Doebeli, 1996; Day, 2000 and references therein), a Gaussian form for $K(z)$ is used

$$K(z) = \kappa \exp \left\{ -\frac{z^2}{2\sigma_k^2} \right\}. \quad (4)$$

This formulation assumes that the resource axis has been scaled so that the type of resource with the greatest abundance is labelled 0. The parameter κ specifies the abundance of this type 0 resource, and σ_k specifies how quickly the abundance of resources declines as we consider types further away from type 0. Note that the resources do not move between patches.

Competition for resources is exploitative, and each individual's ability at consuming resources of type z is determined by a quantitative character. In particular, I suppose that this character maps directly on to the resource axis such that an individual with phenotype x specializes at (i.e., is best at) consuming resources of type x (I will use 'phenotype' and 'genotype' interchangeably). Two individuals with different phenotypes, x and y , nevertheless have some overlap in their resource consumption, and thus they compete with one another for resources. This competition is stronger the more similar are their phenotypes, and it decays to zero as the phenotypes become very different. This form of competition, along with the above specification of the resource distribution, results in two qualitative selective pressures on the evolution of resource exploitation strategies. First, selection is stabilizing, favouring strategies that are close to 0 because they benefit from a high resource abundance. Second, there is also a disruptive aspect of selection favouring individuals that are 'different' because they experience less competition. These two forms of selection that arise from competition for resources are present in all models related to Rosenzweig's notion of competitive speciation (Rosenzweig, 1978).

To model these two forms of selection, I use the following expression for, ϕ , the number of offspring produced by an individual with phenotype x , when in a patch where the remaining individuals have phenotypes y_1, \dots, y_{N-1}

$$\begin{aligned} \phi(x; y_1, \dots, y_{N-1}) \\ = K(x) \frac{\alpha(x, x)}{\alpha(x, x) + \alpha(x, y_1) + \dots + \alpha(x, y_{N-1})} \end{aligned} \quad (5)$$

$$= K(x) \frac{1}{1 + \sum_{i=1}^{N-1} \alpha(x, y_i)}. \quad (6)$$

This form is similar to a model of exploitative competition presented by Schoener (1976). Here the function, $\alpha(x, y)$, gives the competitive effect of a y -individual on an x -individual (as felt by an x -individual), and it is analogous to the competition coefficients of the Lotka–Volterra competition equations. I assume that $\alpha(x, x) = 1$, and that α decays to zero as the two phenotypes become sufficiently different. Notice that if all individuals have the same phenotype, x , then each has offspring production $K(x)/N$, which can be interpreted roughly as though each individual gets a competitive share, $1/N$, of the type x resources available. If some of the competitors have a different phenotype, however, then an individual with phenotype x gets a greater than $1/N$ competitive share of the type x resources. Moreover, if all the remaining $N - 1$ competitors have phenotypes sufficiently different from x (so that $\alpha(x, y_i) \approx 0$ for $i = 1, \dots, N - 1$) then the x -individual gets essentially all of the resources of type x . Importantly, this form for ϕ also results in the same predictions as infinite population size models based on the Lotka–Volterra equations as $N \rightarrow \infty$ as will be detailed shortly. Consequently, the results obtained here are directly comparable with earlier results. An important difference between these earlier models and the form of (6), however, is that here a single individual has a non-negligible effect on resource availability within its patch.

I assume that the competition coefficients used in (6) all have a general, exponential form that allows for competitive asymmetry;

$$\alpha(x, y) = \exp \left\{ \frac{\beta^2}{2} \right\} \exp \left\{ -\frac{(x - y + \sigma_\alpha \beta)^2}{2\sigma_\alpha^2} \right\} \quad (7)$$

(Kisdi, 1999). Notice that $\alpha(x, x) = 1$ for all x . To get a better feel for this expression, first consider the case whereby $\beta = 0$. The parameter σ_α in (7) governs that rate at which α decreases as the phenotypes become more different. A large value of σ_α means that α decreases slowly with increasing phenotypic distance, and $\alpha(x, y)$ gets smaller than unity as y becomes more different from x . Now the parameter β (which

is non-negative) allows for the possibility of competitive asymmetry. If $\beta > 0$, then individuals with large phenotypic values have a competitive advantage over those with small values, and the larger the value of β , the greater is this advantage. This means that $\alpha(x, y)$ will then be greater than unity if y is slightly greater than x , and less than unity if the reverse holds. And as the phenotypic distance becomes very large in either direction, α eventually decreases back to zero.

The fitness function

With the above definitions I now formulate an expression for the fitness of a rare mutant adult with phenotype x , in a population where the predominate phenotype is \hat{y} . Fitness here is measured as the expected total number of offspring produced that, themselves, go on to become reproductively active adults. First suppose that a mutant finds itself in a patch where the remaining individuals have phenotypes y_1, \dots, y_{N-1} . It is necessary to keep track of all of the phenotypes of the individuals in the patch because, even though I will suppose that the mutant type is rare, more than one individual in the patch might be a mutant due to restricted dispersal. The mutant's fitness, $G(x; y_1, \dots, y_{N-1}; \hat{y})$, is given by

$$\begin{aligned} G(x; y_1, \dots, y_{N-1}; \hat{y}) \\ = \phi(x; y_1, \dots, y_{N-1}) \\ \left\{ \frac{d(1-c)N(1-s)}{(1-cd)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N} + \frac{(1-d)N(1-s)}{(1-d)\bar{\phi} + d(1-c)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N} \right\} + s \end{aligned} \quad (8)$$

$$\begin{aligned} = (1-s)\phi(x; y_1, \dots, y_{N-1}) \\ \left\{ \frac{d(1-c)}{(1-cd)\phi(\hat{y}; \hat{y}, \dots, \hat{y})} + \frac{(1-d)}{(1-d)\bar{\phi} + d(1-c)\phi(\hat{y}; \hat{y}, \dots, \hat{y})} \right\} + s, \end{aligned} \quad (9)$$

where

$$\begin{aligned} \bar{\phi} = \frac{1}{N}\phi(x; y_1, \dots, y_{N-1}) + \\ + \frac{1}{N}\phi(y_1; x, \dots, y_{N-1}) + \dots + \\ + \frac{1}{N}\phi(y_{N-1}; y_1, \dots, x) \end{aligned} \quad (10)$$

is the average number of offspring produced by an individual in the patch containing the mutant. The two

terms in the braces of (8) represent the two different fates of the offspring produced. A proportion $d(1-c)$ disperse (and survive dispersal), and because the population size is very large and the mutant is rare, these will land on patches where all other individuals, both native and immigrant, have phenotype \hat{y} . Summing these two sources of wild type individuals gives a total of $(1-d)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N + d(1-c)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N$ or simply $(1-cd)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N$. Therefore, each (surviving) dispersing mutant offspring has a $1/(1-cd)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N$ chance of surviving culling to become an adult. Because all adults have a large number of offspring, this is true for all the $N(1-s)$ spots that become available in that generation, and this gives the first term of (8). Now a proportion $1-d$ of the mutant's offspring remain on their natal site, and the total expected number of juveniles on this patch after dispersal, from native and immigrant individuals, is $(1-d)\bar{\phi}N + d(1-c)\phi(\hat{y}; \hat{y}, \dots, \hat{y})N$. Thus, each non-dispersing mutant offspring has a $1/[(1-d)\bar{\phi} + d(1-c)\phi(\hat{y}; \hat{y}, \dots, \hat{y})]N$ chance of surviving culling to become an adult, and again this is true for all the $N(1-s)$ spots that become available that generation, giving the second term of (8). The third term, s , is simply the probability that the mutant adult will survive to the following year.

Expression (9) is the expected fitness of a mutant given it finds itself on a patch where the remaining individuals have phenotypes y_1, \dots, y_{N-1} . To obtain the final fitness function, $W(x, \hat{y})$, we then need to take the expectation of this expression over all possible types of patches in which the mutants are found. To obtain a simple expression for W , I make the simplifying assumption that selection is weak, so that the distribution of mutants on patches throughout the population reaches a statistical equilibrium while the mutant is still rare. In particular, I assume that the mutant allele is neutral (and rare) to calculate this equilibrium (which represents a balance between drift and dispersal within each patch). This means that, although virtually no patches contain mutants (because they are rare), wherever a mutant is found, there will often be many due to limited dispersal. It is this distribution that is used to calculate the expression for $W(x, \hat{y})$. This type of assumption is implicit in many inclusive fitness models. Appendix A shows that the assumption of neutrality is valid for weak selection (i.e., to first order in the mutant deviation), and simulation results for the present model suggest that this simplification is quite reasonable.

Results

Before detailing the results of the present model, I first present analogous results for a model with an infinite, well-mixed population based on the Lotka–Volterra equations for comparison (I will refer to this as the LV model). The results are based on a model described by Doebeli and Dieckmann (2000) (see also Dieckmann & Doebeli, 1999) but use (7) as the competition coefficient instead of

$$\alpha(x, y) = \exp\left\{\frac{\sigma_\alpha^2 \beta^2}{2}\right\} \exp\left\{-\frac{(x-y+\sigma_\alpha^2 \beta)^2}{2\sigma_\alpha^2}\right\},$$

as used by Doebeli and Dieckmann (2000). Results are qualitatively similar in either case (see Rummel & Roughgarden, 1985; Taper & Case, 1992; Vincent et al., 1993, & Law et al., 1997 for other forms for α).

Condition (1) for this LV model yields

$$y^* = \frac{\sigma_K^2}{\sigma_\alpha} \beta \quad (11)$$

as the candidate ESS, and this value of y^* is easily shown to always satisfy the convergence stability condition (3). The ESS condition (2) for this model yields

$$\sigma_K^2 < \sigma_\alpha^2 \quad (12)$$

as the inequality that must be satisfied for y^* given in (11) to be an ESS (Roughgarden, 1976, 1983; Brown & Vincent, 1987; Vincent et al., 1993).

These results have interesting, and intuitive explanations. First it is clear that when a single evolutionarily stable resource exploitation strategy, y^* , exists (i.e., when (12) holds) it is located at the peak of the resource distribution, K , if competition is symmetric (i.e., $\beta = 0$). In other words, all individuals specialize on the most abundant resource. Under competitive asymmetry ($\beta > 0$) however, at the ESS, individuals do not specialize on resources that are most abundant. Rather, they have a phenotype that is larger than this because large trait values outcompete small trait values. In other words, despite the fact that a mutant individual with a slightly smaller phenotype would benefit from an increased resource abundance at the ESS, it would suffer greatly from competition with the remaining, larger individuals. The ESS value of y^* given in (11) is the phenotype at which these costs and benefits exactly balance.

Also of primary interest is when condition (12) does *not* hold. In this case, natural selection will still drive the population towards phenotype y^* , but once

the population is there, selection will become disruptive. This occurs because, when $\sigma_k^2 > \sigma_\alpha^2$, the resource distribution is very broad and/or the strength of competition is very high. Thus, selection to ‘be different’ is very strong so as to avoid the intense competition. Because the resource distribution is quite broad, little is lost in terms of resource abundance by deviating from y^* slightly. Interestingly, notice that the condition under which we expect such evolutionary diversification to occur (i.e., $\sigma_k^2 > \sigma_\alpha^2$) is independent of the degree of symmetry of competition, β .

General findings

All examples of potential ESS’s that were found in the present model appear to satisfy the convergence stability condition (3) based on simulation results. These candidate ESS values of y must satisfy condition (1), and Appendix A demonstrates that this is equivalent to satisfying the condition,

$$\left[\frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} (N\bar{r} - 1) \right]_{x=y_i=\hat{y}=y^*} = 0. \quad (13)$$

Here \bar{r} is the genetic relatedness of two randomly chosen patchmates with replacement (Michod & Hamilton, 1980), and $\partial G/\partial y_i$ is defined as $\partial G/dy_i$ for any i (notice that the derivative of G with respect to y_i is the same for all i because all y_i individuals play the same role). The constant, \bar{r} can also be interpreted as the expected fraction of individuals in a patch that are mutant as seen by a randomly chosen mutant individual. Therefore, using (9) in (13) gives

$$\begin{aligned} & \frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} (N\bar{r} - 1) \\ & \propto \frac{\partial \phi}{\partial x} + \frac{\partial \bar{\phi}}{\partial y} \left\{ N\bar{r} \frac{1 - k^2}{1 - \bar{r}k^2} - 1 \right\}, \end{aligned} \quad (14)$$

where $k = (1-d)/(1-cd)$ is the probability that a randomly chosen individual is native to that patch from the previous generation. Now using (6) in (14) and setting the resulting expression equal to zero, gives the (candidate) ESS resource exploitation phenotype to be

$$y^* = \frac{\sigma_k^2}{\sigma_\alpha} \beta \{1 - \bar{r}\xi\}, \quad (15)$$

which is expressed in terms of the relatedness parameter, \bar{r} , and the constant,

$$\xi = \frac{(1 - k^2) \left(N - k^2(N - 1) \frac{2s - ks + k}{k(1+s)} \right)}{N - k^2(N - 1) \frac{2s - ks + k}{k(1+s)} - k^2}$$

(compare this with the LV model result, [11]). Appendix B (see also Pen, 2000; Taylor & Irwin, 2000) shows that

$$\bar{r} = \frac{1}{N - k^2(N - 1) \frac{2s - ks + k}{k(1+s)}}. \quad (16)$$

Once the population has evolved to this candidate ESS, selection can then become either stabilizing or disruptive depending upon condition (2). If selection is disruptive we then expect some type of evolutionary diversification of resource exploitation strategies to occur.

A consideration of the form of (15), reveals some interesting general properties. First, if $\beta = 0$ (no competitive asymmetry) then $y^* = 0$, which reveals that neither interaction group size nor genetic structuring have any effect on the location of the candidate ESS. If there is competitive asymmetry (i.e., $\beta \neq 0$), however, then we can see from (15) that both factors can have an effect on the location of y^* . Unfortunately a general expression for condition (2) was not obtained, but as will be seen below, both interaction groups size and population viscosity affect whether selection is stabilizing or disruptive at $y^* = 0$ regardless of whether competition is asymmetric or not. Some special cases help to illustrate these phenomena.

Some special cases

Well-mixed populations

Suppose that all individuals disperse every generation ($d = 1$), that there is no cost of dispersal ($c = 0$), and that there is no adult survival ($s = 0$). Interactions still take place within finite-sized groups, but there is no genetic structuring of the population. In this case conditions (15) and (2) yield

$$y^* = \frac{\sigma_k^2}{\sigma_\alpha} \beta \left(1 - \frac{1}{N} \right) \quad (17)$$

and

$$\left(1 - \frac{1}{N} \right) \left(1 - \frac{\beta^2}{N} \right) \sigma_k^2 < \sigma_\alpha^2. \quad (18)$$

These results reveal several interesting effects of having finite-sized interaction groups. First, (17) shows that the predicted value of y^* converges to that of the LV model as $N \rightarrow \infty$ (as it should). It also reveals that (under asymmetric competition) the value of y^* decreases as the size of the interaction group gets smaller, and it is zero when $N = 1$ (Figure 1). This is intuitively reasonable since when

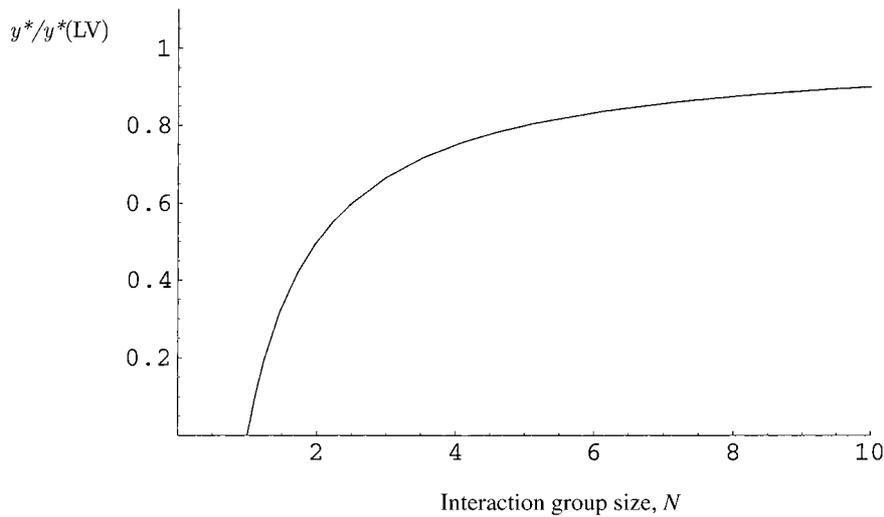


Figure 1. A plot of the ratio of y^* (i.e., the ESS) from the present model to that from the LV (i.e., the well-mixed, infinite population size Lotka–Volterra model) model. In other words, this is Equation (17) divided by Equation (11). The value of y^* from the LV model is denoted $y^*(LV)$. y^* from the present model is always smaller than that from the LV model, but they become more similar as the size of the interaction group, N , gets large.

$N = 1$ there is no competition and therefore natural selection should push the population towards a strategy that specializes on the most abundant resource. Figure 2 presents some simulation results in this setting (Appendix C provides details of the simulation).

The condition determining the form of selection that occurs at y^* also has interesting properties. If inequality (18) is satisfied, then selection will be stabilizing whereas if the reverse inequality holds, then we expect evolutionary diversification. Notice that, again as $N \rightarrow \infty$, this converges on the LV model result (12), but it differs (sometimes substantially) from this condition for any finite-sized interaction group. In the most extreme case, with groups of size $N = 1$, inequality (18) is always satisfied and selection will never be disruptive. Again this is intuitively reasonable since there is never anything for an individual to gain by deviating from using the most abundant resource. If $N > 1$ then there is some benefit of reduced competition that results from being different, but this benefit can be quite small for small N , since competition occurs among very few individuals. This thereby makes disruptive selection much less likely (Figure 3). Inequality (18) also shows that competitive asymmetry ($\beta > 0$) makes disruptive selection less likely as well (whereas it has no effect in the LV model; condition [12]). Simulation results also confirm this idea that finite-sized interaction groups

make disruptive selection less common (Appendix C; Figure 4).

Population viscosity with no adult survival

Partial dispersal ($d < 1$, $c > 0$) will generate some degree of genetic structuring in the population, and it is of interest to examine the effect that this has on the location of y^* as well as on the likelihood of disruptive selection, both in the absence and in the presence of adult survival. In this case, again it is easy to obtain an analytical expression for y^* , but condition (2) can be extremely tedious to evaluate for deme sizes larger than 2. In the interest of clarity and ease of understanding, I present only the $N = 2$ case since the exact same phenomenon that occurs in this case will also occur for $N > 2$. Also, competitive asymmetry complicates the calculations, without altering the phenomenon to be described, and therefore I assume that $\beta = 0$ when calculating condition (2).

Interestingly, with no adult survival ($s = 0$), y^* is given by the exact same formula as above (i.e., [17]). This implies that the genetic structuring that results from partial dispersal has no effect on the location of y^* if there is no adult survival. This is an example of a well-known result from models for the evolution of altruism (Taylor, 1992; Wilson et al., 1992). Population viscosity (low dispersal) increases relatedness between interacting individuals, and one might expect this to increase the evolution of altruism (altruism

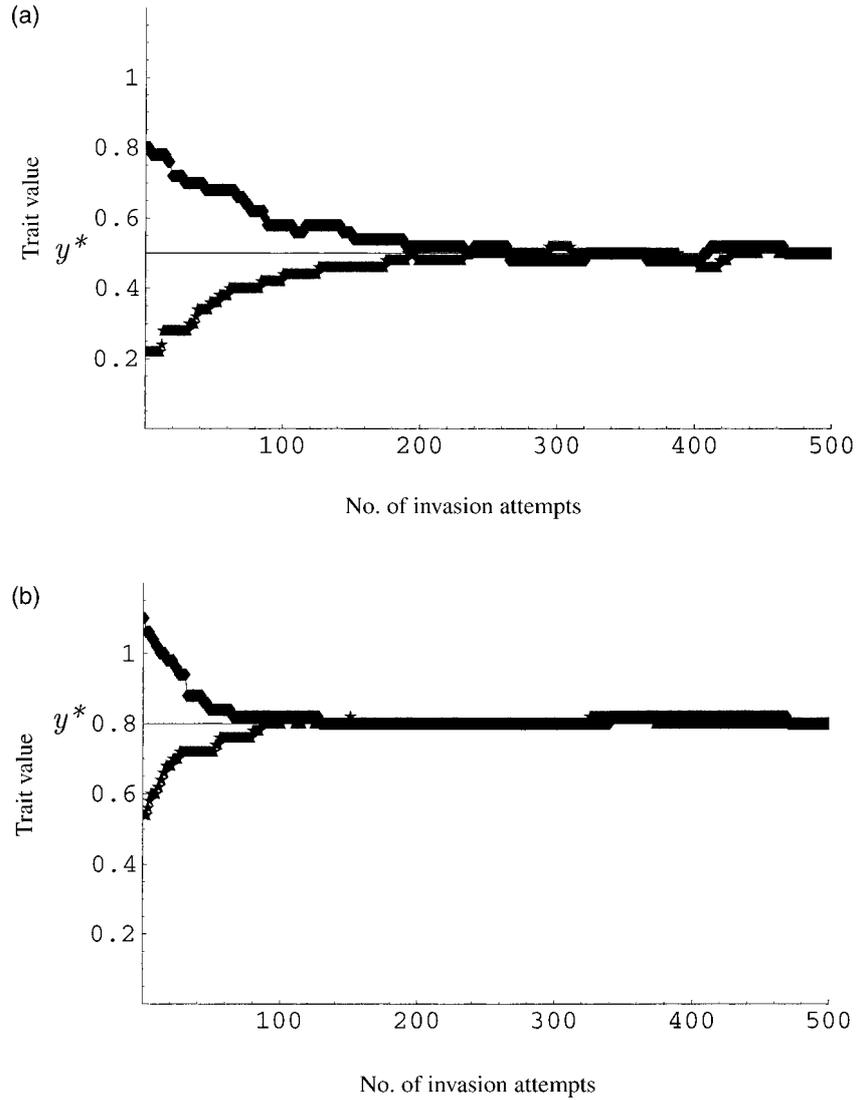


Figure 2. Simulation results demonstrating that the value of y^* is convergence stable. Each plot is the mean value of 5 separate runs of the simulation described in Appendix C. One of these plots is for 5 runs that begin above y^* and the other is for 5 runs that begin below y^* . The horizontal axis is the number of invasion attempts (i.e., the number of new mutants that have arisen and attempted to displace the resident strategy) from 0 to 500. The horizontal line in the figures is the analytical prediction for y^* . (a) Parameter values are $d = 1$, $c = 0$, $s = 0$, $\beta = 1$, $N = 2$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ where d is the dispersal rate, c is the cost of dispersal, and β is the competitive asymmetry parameter. The number of patches is 100. (b) Parameter values are $d = 1$, $c = 0$, $s = 0$, $\beta = 1$, $N = 5$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ and the number of patches is 100.

here, under competitive asymmetry, can be interpreted as a smaller phenotype). This is not necessarily the case, however, because this same viscosity guarantees that the extra offspring produced from altruistic behaviour will be competing with other related individuals due to their limited dispersal. These effects exactly cancel one another leaving y^* unaltered by population viscosity. This can also be understood from the general

expression (15) for y^* . Population viscosity increases relatedness, \bar{r} , but it decreases the factor ξ , and these two effects cancel provided that $s = 0$.

The ESS condition (2) for the case $N = 2$ and $\beta = 0$ yields the condition (Appendix D)

$$\frac{(1-c)d((1-d) + (1-cd))}{2(1-cd)^2} \sigma_K^2 < \sigma_\alpha^2. \quad (19)$$

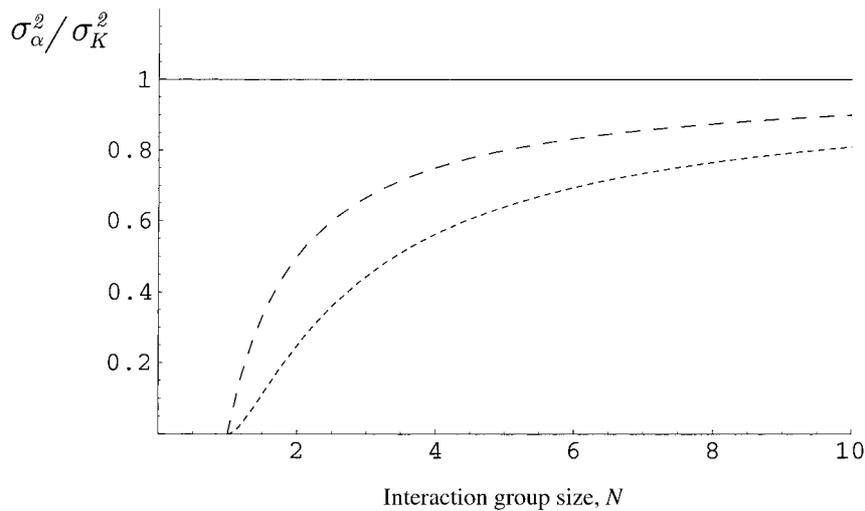


Figure 3. Plots of the threshold values of $\sigma_\alpha^2 / \sigma_K^2$ below which selection is disruptive (and consequently we expect some form of evolutionary diversification) against interaction group size, N . Solid, horizontal line at 1 is for the LV (i.e., Lotka–Volterra) model for comparison (which is independent of N). Dashed line is for competitive symmetry (i.e., $\beta = 0$) and dotted line is for $\beta = 1$.

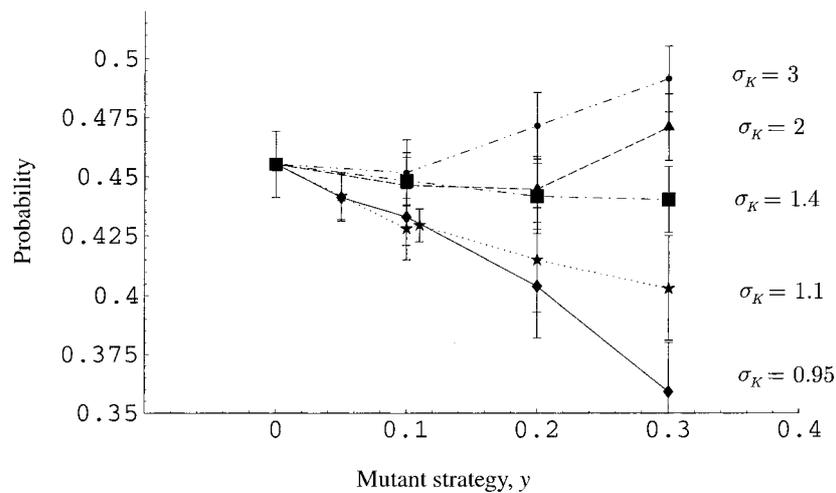


Figure 4. A plot of simulation results that examine whether selection is stabilizing or disruptive at $y^* = 0$. Mutant strategies that deviate different amounts from 0 in absolute value are plotted on the horizontal axis. Vertical axis is the probability of the mutant frequency hitting (or passing) a frequency twice as great as its initial frequency prior to hitting zero (as described in Appendix C), and is thus a measure of the selective advantage of the mutant. The value at $y = 0$ is the neutral probability, and anything above this is selectively favoured whereas anything below is selectively disadvantageous. The five plots are for different values of σ_K . Error bars are ± 2 S.E. Remaining parameter values are: $d = 1$, $c = 0$, $s = 0$, $\beta = 0$, $N = 2$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ where d is the dispersal rate, c is the cost of dispersal, and s is the probability of adult survival from year to year. The number of patches is 100. For these parameter values, condition (18) predicts that selection changes from stabilizing to disruptive as σ_K increases past $\sqrt{2} \approx 1.41$ which matches the simulation results quite well. Note that the threshold value of σ_K for the infinite, well-mixed population for these parameter values is $\sigma_K = 1$ (from condition [12]).

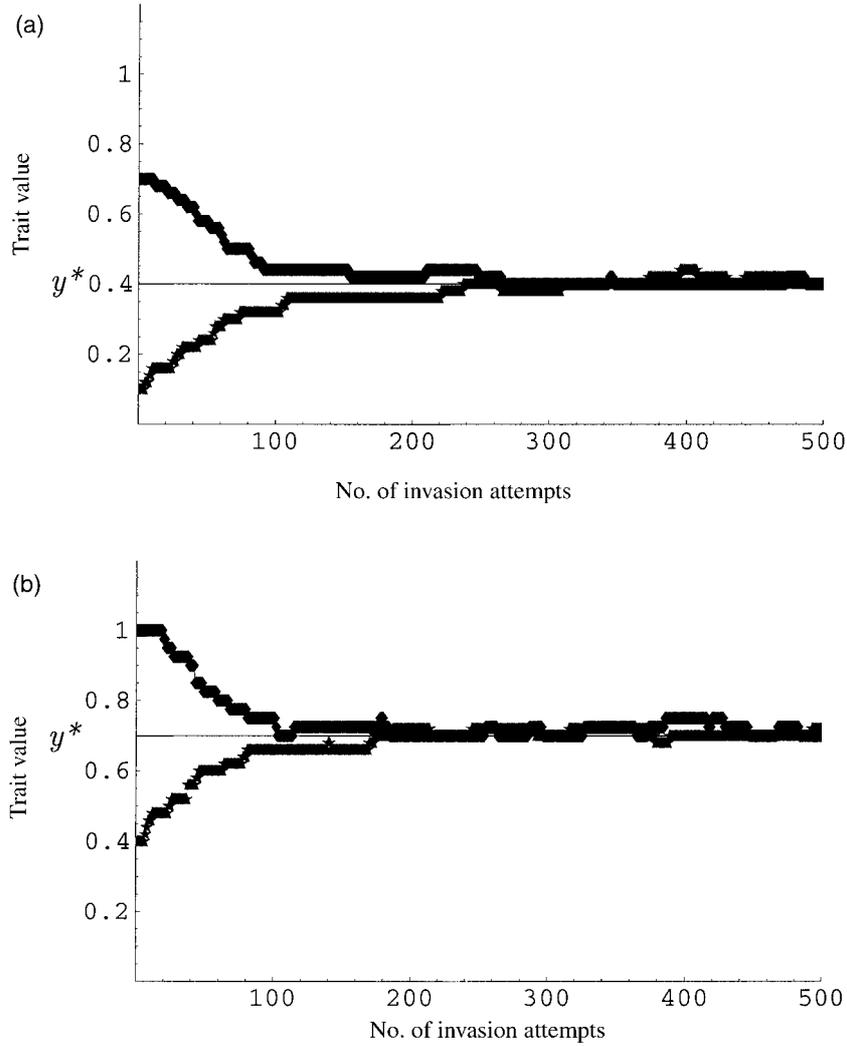


Figure 5. Simulation results demonstrating that the value of y^* is convergence stable. Each plot is the mean value of 5 separate runs of the simulation described in Appendix C. One of these plots is for 5 runs that begin above y^* and the other is for 5 runs that begin below y^* . The horizontal axis is the number of invasion attempts (i.e., the number of new mutants that have arisen and attempted to displace the resident strategy) from 0 to 500. The horizontal line in the figures is the analytical prediction for y^* . (a) Parameter values are $d = 0.4$, $c = 0.4$, $s = 0.75$, $\beta = 1$, $N = 2$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ where d is the dispersal rate, c is the cost of dispersal, s is the probability of adult survival, and β is the competitive asymmetry parameter. The number of patches is 100. (b) Parameter values are $d = 0.4$, $c = 0.4$, $s = 0.85$, $\beta = 1$, $N = 5$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ and the number of patches is 100.

As expected from (18), this reduces to $\frac{1}{2}\sigma_K^2 < \sigma_\alpha^2$ when the population is well-mixed (i.e., when $d = 1$, $c = 0$). Condition (19) illustrates that disruptive selection becomes even less likely as population viscosity increases because this condition is more likely to be satisfied. To better see this, consider the case where the cost of dispersal, c , is zero. Condition (19) then becomes

$$\frac{d(2-d)}{2}\sigma_K^2 < \sigma_\alpha^2. \quad (20)$$

The left-hand side of this inequality decreases to 0 as d decreases to zero, and therefore, when dispersal is very limited, selection will almost invariably be stabilizing, thereby preventing evolutionary diversification. Stated another way, for any set of parameter values for σ_K and σ_α , it is possible to choose a dispersal rate low enough to prevent evolutionary diversification.

The reason why genetic structuring can prevent evolutionary diversification is that, when dispersal is limited, most competitors will be genetically very

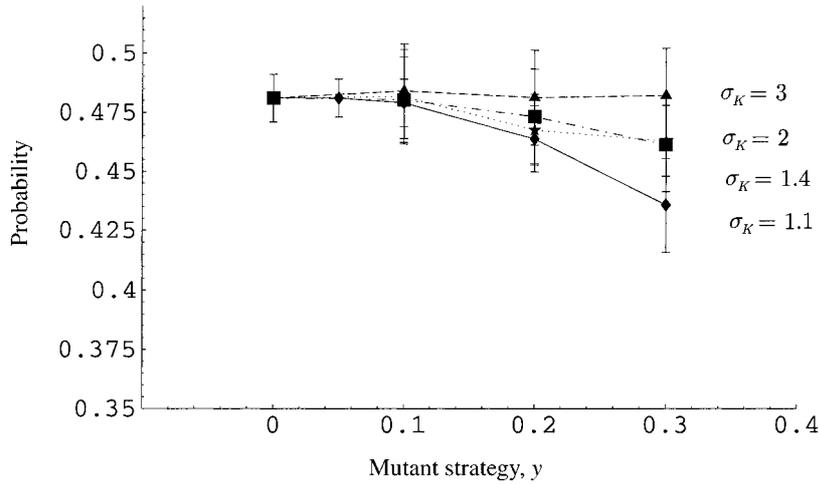


Figure 6. A plot of simulation results that examine whether selection is stabilizing or disruptive at $y^* = 0$. Mutant strategies that deviate different amounts from 0 in absolute value are plotted on the horizontal axis. Vertical axis is the probability of the mutant frequency hitting (or passing) a frequency twice as great as its initial frequency prior to hitting zero (as described in Appendix C), and is thus a measure of the selective advantage of the mutant. The value at $y = 0$ is the neutral probability, and anything above this is selectively favoured whereas anything below is selectively disadvantageous. The four plots are for different values of σ_K . Error bars are ± 2 S.E. Remaining parameter values are: $d = 0.4$, $c = 0.4$, $s = 0.75$, $\beta = 0$, $N = 2$, $\sigma_K = 1$, $\sigma_\alpha = 1$, $\kappa = 1000$ where d is the dispersal rate, c is the cost of dispersal, and s is the probability of adult survival from year to year. The number of patches is 100. For these parameter values, the complete version of condition (22) (not shown) predicts that selection changes from stabilizing to disruptive as σ_K increases past approximately 2.28, which matches the simulation results reasonably well.

similar. That means that the benefit that a mutant type gains from being different (which is a reduction in competition) will very seldom be realized because limited dispersal causes the mutant's competitors to be mutants as well. This means that the cost to being different (which is a reduced abundance of resources) must be extremely small in order for evolutionary diversification to occur. This will be true only when the resource distribution, K , is extremely broad, meaning that σ_K must be extremely large. This inhibition of diversification that arises from competition among relatives will occur if $N > 2$, as well as if $\beta \neq 0$.

Population viscosity with adult survival

If there is some adult survival, then both the expression for y^* , as well as the ESS condition (2) are altered by population viscosity. The candidate ESS is then given by

$$y^* = \frac{\sigma_k^2 \beta}{\sigma_\alpha} \frac{N-1}{N+\zeta}, \quad (21)$$

where $\zeta = k^2(1-\eta)/(1-k^2\eta)$, and $\eta = (2s - ks + k)/k(1+s)$. The constant ζ , although somewhat complicated, increases as dispersal rates go down, costs of dispersal go up, or adult survival rates go up. That implies that population viscosity selects for lower phenotypic values under more restricted dis-

persal if competition is asymmetric (Figure 5). Again this has a clear analogue in the altruism literature. It has been noted that overlapping generations in combination with population viscosity can select for greater altruism (Taylor & Irwin, 2000), which is analogous to smaller phenotypes under asymmetric competition. The reason is that the benefit gained by a mutant with a larger phenotype (which is reduced competition as well as the ability to outcompete smaller phenotypes) is reduced in this setting. Such larger mutants will tend to compete with genetically similar, and therefore large, mutants due to the population viscosity.

The ESS condition in this setting for symmetric competition and a patch size of 2 is quite complicated, but it can be simplified considerably in the case where there is no cost to dispersal;

$$\frac{2-d+ds}{2-d+s} \frac{d(2-d)}{2} \sigma_K^2 < \sigma_\alpha^2. \quad (22)$$

Again, this expression demonstrates that population viscosity has the same, inhibiting effects on evolutionary diversification here as it does when there is no adult survival (also see Figure 6). Moreover, as s increases, the left-hand side of (22) becomes even smaller for any given dispersal rate, making evolutionary diversification even less likely. The reason of

course, is that increasing adult survival increases the genetic similarity among competing individuals.

Discussion

Recent models have demonstrated that Rosenzweig's (1978) idea of competition speciation (and related phenomena) can, in theory, be a potent force driving evolutionary diversification (Doebeli, 1996; Law et al., 1997; Dieckmann & Doebeli, 1999; Kisdi, 1999; Doebeli & Dieckmann, 2000). So much so that one might be lead to feel that speciation occurs too easily in some of these models (Bridle & Jiggins, 2000). This echoes Felsenstein's (1981) feelings when he considered Hutchinson's ecological arguments for the determinants of current species numbers, and Felsenstein (1981) suggested that genetic constraints can hinder the process of speciation. Here I have demonstrated that an interaction between ecological aspects of competition for resources and the genetic structuring that results from finite interaction groups and population viscosity, can inhibit the occurrence of disruptive selection on which many recent models of sympatric speciation depend. This provides an additional factor limiting the occurrence of such evolutionary diversification in the absence of geographic isolation. Notice that the effect described here is one in which the occurrence of disruptive selection is inhibited, whereas Felsenstein's was one that inhibited the evolution of reproductive isolation once disruptive selection was in place.

The present study also found that finite interaction groups and population viscosity often select for less extreme competitive strategies when competition is asymmetric. This has a natural analogue in the literature on the evolution of altruism. Population structure increases the genetic relatedness between competitors, and this can select for increased altruism (less competitive strategies here). As with these inclusive fitness studies, this phenomenon seems to occur, only when there is some degree of adult survival. This causes overlapping generations and it increases the genetic relatedness among competitors (Taylor & Irwin, 2000).

Some of my finding are also closely related to a model of interspecific resource partitioning by Pacala (1988). Pacala modeled a community of sedentary individuals which compete for resources in spatially localized areas. He found that, as the resource use by each individual became more and more local, the extent

of resource partitioning that occurred among species decreased to zero. This is analogous to the present result in which, as patch size decreases, the likelihood of disruptive selection decreases. Moreover, disruptive selection never occurs once patch size equals 1. As with Pacala's result, this occurs in the present study because competition for resources diminishes as the number of competitors decreases, and there is no competition once resource use is completely local ($N = 1$ here).

The model presented here was chosen with an eye towards analytical tractability so that the phenomena of finite groups and genetic structuring could be understood relatively easily. As such it is not intended to be a completely faithful depiction of any particular biological organism. Nevertheless, it seems reasonable to expect that the phenomena described here will occur in more complex, realistic scenarios as well. Limited dispersal (whatever its form) should often result in competition between genetically similar individuals, and this should thereby reduce the evolutionary advantage of being different, which reduces the likelihood of disruptive selection. Being different is advantageous under competition because it allows an individual to escape competition. If one's competitors are always genetically similar to oneself, however, then being different is rarely possible. Mutant individuals with alternative resource exploitation strategies will compete predominately with other mutant individuals that have the exact same strategy because of limited dispersal. This enhances stabilizing selection for a strategy that specializes on the most abundant resource, and in general it should inhibit evolutionary diversification and thereby inhibit competitive speciation as well. It should be noted, however, that the importance of this effect for any particular biological system will depend upon at least two important factors, the size of the interaction groups, and the rate of dispersal. Larger interaction groups will require a smaller dispersal rate for the effects discussed here to play a significant role.

Finally, I close by mentioning an interesting caveat and possible extension. The results presented here assume that all patches are identical, and they make the prediction that the spatial structuring that accompanies reduced dispersal will reduce the likelihood of disruptive selection. If there was variation in patch type, then it seems likely that this would introduce an interesting effect that works in opposition to the effects detailed here. Previous results for two-patch models in which the patches contain different suites

of resources have shown that low dispersal between patches makes disruptive selection occur more readily (Brown & Pavlovic, 1992; Day, 2000). An important difference between the present results and this earlier work, however, is that competition occurs among a finite number of individuals here, whereas the patches in this previous work were effectively infinite. It would be very interesting to extend the present results by allowing for different patch types to examine how both of these factors play out in determining the form of selection.

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Appendix A

The derivation here follows that of Day and Taylor (1998). Consider a patch-structured population in which there are N individuals per patch, and every individual plays the same role. Suppose that the fitness of an individual using the (mutant) strategy x in a patch where the remaining $N - 1$ individuals are using strategies y_1, y_2, \dots, y_{N-1} is given by the function $G(x; y_1, \dots, y_{N-1}; \hat{y})$. Then if ρ_k is the probability that a mutant individual finds itself in a patch with a total of k mutants, the expected fitness of this mutant allele is given by

$$W(x; y_1, \dots, y_{N-1}) = \sum_{k=1}^N \rho_k G(x; x, x, \dots, \hat{y}_{N-1}; \hat{y}). \quad (\text{A.1})$$

Here, x appears k times after the semicolon because that gives the fitness of a mutant given it is found in a population with a total of k mutants. Recall that all individuals play the same role and so all that matters in determining fitness is the total number of mutants, not which particular individuals are mutant and which are not. Because of this, all of the partial derivatives, $\partial G/\partial y_i$ are identical and I will simply denote them

by $\partial G/\partial y$. Now if y^* is an ESS, then (A.1) must be maximized in x at $x = y^*$ when y^* is substituted for all the y_i . The first order condition for this is that the first derivative with respect to x be zero at this point. This derivative is given as

$$\frac{\partial W}{\partial x} \Big|_{x=y_i=y^*} = \sum_{k=1}^N \frac{d\rho_k}{dx} G + \sum_{k=1}^N \rho_k \left(\frac{\partial G}{\partial x} + (k-1) \frac{\partial G}{\partial y} \right) \quad (\text{A.2})$$

$$= \frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} \sum_{k=1}^N \rho_k (k-1) \quad (\text{A.3})$$

$$= \frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} (N\bar{r} - 1), \quad (\text{A.4})$$

where $\bar{r} = \sum_k \rho_k (k/N)$ is the relatedness of two randomly chosen patch members with replacement (Michod & Hamilton, 1980; Day & Taylor, 1998). All the derivatives above are evaluated at $x = y_i = y^*$, and expression (A.2) must equal zero. The derivatives $d\rho_k/dx$ in the above expression reflect the fact that the distribution, ρ_k , will change with changes in the mutant strategy, x , but the first summation term in (A.2) is zero when evaluated at $x = y_i = y^*$ because it simplifies to $G \sum_{k=1}^N (d\rho_k/dx)$. Because the ρ_k sum to unity, the sum of their derivatives must equal zero. Notice that the only place that the spatial structuring of types enters into the final result (A.4) is in the relatedness, \bar{r} , and that this is calculated under the assumption that the population is monomorphic (or equivalently, that the mutant allele is selectively neutral). Thus, to first order in the mutant deviation, the distribution of mutants in the population can be calculated by assuming that the mutant allele is selectively neutral.

Appendix B

Here I calculate relatedness of two patch members chosen randomly, with replacement (i.e., \bar{r}) following Pen (2000) and Taylor and Irwin (2000). Suppose that \bar{r}_t is the relatedness in generation t . Then the relatedness in generation $t + 1$ is given by the recursion

$$\bar{r}_{t+1} = \frac{1}{N} + \left(1 - \frac{1}{N}\right) (s^2 r_t + 2s(1-s)k\bar{r}_t + (1-s)^2 k^2 \bar{r}_t), \quad (\text{B.1})$$

where r_t is the relatedness of two different members on the same patch in generation t , and is defined by the relation $\bar{r} = 1/N + r(1 - 1/N)$. With probability $1/N$ the same individual is chosen which gives the first term, and with probability $(1 - 1/N)$ a different individual is chosen, giving the second term, which itself is composed of three different pieces. First, with probability s^2 both individuals are surviving adults from the last generation, in which case their relatedness is r_t . With probability $2s(1 - s)k$ one of the individuals is a surviving adult and the other is a new adult that arose from an offspring native to the patch from the prior generation. With probability $1/N$ this offspring is from the surviving adult under consideration (in which case relatedness is 1), and with probability $(1 - 1/N)$ it is not (in which case relatedness is r_t). This gives a total of \bar{r}_t . Lastly, with probability $(1 - s)^2 k^2$ both individuals are new adults that arose from offspring native to the patch from the last generation. The same considerations as those for the last case give their relatedness to be \bar{r}_t , which completes the above recursion. At equilibrium, $\bar{r}_t = \bar{r}_{t+1} = \bar{r}$, and substituting this into the above recursion results in an equation that can be solved for \bar{r} .

Appendix C

Simulations were carried out using Mathematica 4 (Wolfram research). Simulations were initialized by setting up a population of demes of size N in which most individuals were of a given phenotype except for an initial, small number of mutants (usually with frequency 0.005). The mutants were placed such that there was never more than one mutant on any given patch initially. Then the life cycle described earlier was simulated by first generating offspring and then calculating the proportion of mutant offspring there were on each patch after dispersal. Each adult on each site then either survived or died independently according to the survival probability, s . Then there was fair competition for the newly opened sites on each patch (which amounts to Binomial sampling according to the proportion of mutants on each patch), and the cycle was then repeated. This procedure was continued until either the mutant frequency dropped to zero, or it increased to 1. At this point, a new mutant was generated by altering the resident strategy by a small amount (usually ± 0.1), and the entire invasion attempt simulation started again. This whole procedure continued for a total of 500 potential invasions/replacement.

To use the simulations for calculating whether or not there is disruptive selection at y^* , I needed to obtain a measure of the selective advantage (or disadvantage) of a mutant type when rare. This was done by starting the mutants at a frequency of 0.005 (as above) and then calculating the probability that the frequency hits (or passes) 0.01 before it hits 0 using the results of several runs of the simulation. This was carried out for different mutant values, y , when the wild type (resident) strategy was at y^* . These values can then be compared to the neutral probability obtained by carrying out the exact same simulation but with both strategies being set to y^* .

Appendix D

Here I evaluate condition (2) explicitly for the case where $N = 2$ and $\beta = 0$. In this case, (A.1) simplifies to

$$W(x; y) = \rho_1 G(x; \hat{y}) + \rho_2 G(x; x). \quad (\text{D.1})$$

The first derivative, (1), is given by

$$\begin{aligned} \frac{\partial W}{\partial x} &= \frac{d\rho_1}{dx} G(x; \hat{y}) + \rho_1 \frac{\partial G}{\partial x} + \frac{d\rho_2}{dx} G(x; x) + \\ &+ \rho_2 \left(\frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} \right) \end{aligned} \quad (\text{D.2})$$

prior to evaluating at $x = y_i = y^*$. Then ESS condition (2) is

$$\begin{aligned} \frac{\partial^2 W}{\partial x^2} \Big|_{x=y_i=y^*} &= \frac{d^2 \rho_1}{dx^2} G + 2 \frac{d\rho_1}{dx} \frac{\partial G}{\partial x} + \rho_1 \frac{\partial^2 G}{\partial x^2} + \frac{d^2 \rho_2}{dx^2} G + \\ &+ 2 \frac{d\rho_2}{dx} \left(\frac{\partial G}{\partial x} + \frac{\partial G}{\partial y} \right) + \end{aligned} \quad (\text{D.3})$$

$$+ \rho_2 \left(\frac{\partial^2 G}{\partial x^2} + 2 \frac{\partial^2 G}{\partial x \partial y} + \frac{\partial^2 G}{\partial y^2} \right) \quad (\text{D.4})$$

$$= \frac{\partial^2 G}{\partial x^2} + 2 \frac{d\rho_2}{dx} \frac{\partial G}{\partial y} + \rho_2 \left(2 \frac{\partial^2 G}{\partial x \partial y} + \frac{\partial^2 G}{\partial y^2} \right). \quad (\text{D.5})$$

The second equality above uses the fact that, because $\rho_1 + \rho_2 = 1$, we have $d\rho_1/dx + d\rho_2/dx = 0$ and $d^2 \rho_1/dx^2 + d^2 \rho_2/dx^2 = 0$. Using the definition of G given in the text, Equation (D.5) then evaluates to give conditions (19) and (22) of the text.

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