The evolutionary consequences of niche construction: a theoretical investigation using two-locus theory

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Abstract

This paper addresses the joint evolution of environment-altering (niche constructing) traits, and traits whose fitness depends on alterable sources of natural selection in environments. We explore the evolutionary consequences of this niche construction using a two-locus population genetic model. The novel conclusions are that niche construction can (1) cause evolutionary inertia and momentum, (2) lead to the fixation of otherwise deleterious alleles, (3) support stable polymorphisms where none are expected, (4) eliminate what would otherwise be stable polymorphisms, and (5) influence disequilibrium. The results suggest that the changes that organisms bring about in their niche can themselves be an important source of natural selection pressures, and imply that evolution may proceed in cycles of selection and niche construction.

Introduction

Organisms, through their metabolism, their activities, and their choices, define and partly create their own niches. They may also part destroy them (Lewontin, 1978, 1983; Odling-Smee, 1988). We refer to these phenomena as either positive or

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negative 'niche construction'. Here, we argue that modern evolutionary theory does not fully take account of the evolutionary consequences of niche construction (Lewontin, 1978, 1983; Odling-Smee, 1988; Odling-Smee et al., 1996). Classically, adaptation is conceived of as a process by which natural selection effectively 'molds' organisms to fit a pre-established environmental 'template'. The environment is seen as posing problems, and those organisms best equipped to deal with the problem leave the most offspring. Although the environmental template may be dynamic, in the sense that forces independent of the organism may change the world to which the population adapts (Van Valen, 1973), the changes that organisms themselves bring about are rarely considered in evolutionary analyses. However, to varying degrees, organisms choose their own habitats, choose their mates, choose and consume resources, generate detritus, and construct important components of their own environments (such as nests, holes, burrows, paths, webs, dams and chemical environments). In addition, many organisms choose, protect, and provision 'nursery' environments of their offspring. On the basis of this kind of evidence, Lewontin (1978, 1982, 1983) has argued that the 'metaphor of adaptation' should be replaced by a 'metaphor of construction'. The key point is that many of the selection pressures to which an organism is exposed exist partly as a consequence of the niche constructing activities of past and present generations of organisms.

There are numerous examples of organisms changing their selection pressures in non-trivial ways, by changing their surroundings or constructing artifacts (Frisch, 1975; Hassell 1984). For instance, Darwin himself pointed out that earthworms are important niche constructors (Darwin, 1881). Through their burrowing activities, their dragging organic material into the soil and mixing it with inorganic material, and their casting which serves as the basis of microbial activity, they dramatically change the structure and chemistry of the soil (Hayes, 1983; Lee, 1985). As a result of the accumulated effects of past generations of earthworm niche construction, present generations of earthworms now inhibit radically altered environments, and are exposed to changing sets of selection pressures.

In many cases there is evidence of an evolutionary response to these self-induced selection pressures. For instance, webs are probably important sources of selection for web spiders: species of Theridion, Tetragnatha and Araneus hide under a leaf of twig which they place in the web (Bristowe, 1958; Edmunds, 1974); species of Cyclosa, Gasteracantha and Uloborus construct "dummy spiders" to divert the attention of avian predators (Hingston, 1927; Edmunds, 1974); and species of the Segestria and Mallos genera differentially respond to particular frequencies of web vibration (Bristowe, 1958; Hassell, 1984). Social bees, wasps, ants and termites also construct elaborate nests which are themselves the source of selection for many nest regulatory, maintenance and defense behaviors. For example, thermo-regulatory behavior has evolved in Vespoid wasps and honeybees. Workers maintain the comb at an appropriate temperature, heating it through muscular activity, or moistening the cells so they are cooled by evaporation (Spradbery, 1973; Mathews and Mathews, 1978). Many ant and termite species regulate temperature by plugging entrances at night or in the cold, adjusting the height or shape of the
mound to optimize the intake of the sun’s rays, or carrying the brood around the
nest to the place with the optimal temperature and humidity for their development
(Frisch, 1975; Hassell, 1984). Hygienic behavior has also evolved in many social bee
and wasp species (Frisch, 1975). For instance, worker honey bees uncap sealed
cells, and remove dead pupae from the nest. Rothenbuhler (1964) established that
the uncutting and removal behaviors are influenced by genetic variation. In all of
these cases, without the prior existence of the web, hive or nest, selection for these
behaviors would be impossible.

Niche construction is equally common amongst vertebrates. For example, many
species of fish, amphibians, reptiles, birds and mammals build nests. In addition,
many mammals (for example, moles, rats, badgers, marmots), construct complex
burrow systems with underground passages, interconnected chambers, and multiple
entrances. Here again there is some evidence that nest and burrow defense,
maintenance, and regulation behaviors, and some components of mating rituals,
have evolved as a response to selection initiated by the construction of the burrow
or nest (see Frisch, 1975; Hassell, 1984).

Niche construction does not always involve the building of an artifact. For
example, the Galapagos “woodpecker” finch has probably modified how it is
affected by its environment simply by employing twigs or cactus spine tools to grub
for insects in the bark of trees. In consequence, these birds have not evolved either
the specialized beaks or elongated tongues of more conventional woodpeckers
(Wilson, 1992). This example illustrates that niche construction can be a conserva-
tive, as well as an innovative, force in evolution. Niche construction is not restricted
to animals. Plants can change the chemical nature, pattern of nutrient cycling,
temperature, humidity, and fertility of the soils in which they live (Ricklefs, 1990).
They may even affect the local climate, the amount of precipitation, and the water
cycle (Shukla et al., 1990). Many plants also change both their own and other
species’ local environments via allelopathy (Rice, 1984). For instance, Rasmussen
and Rice (1971) found that a small grass species (Sporobolus pyramidalis) is able to
spread rapidly into heavy sods of bermudagrass and buffalograss by exuding toxins
from its living roots, or diffusing toxins from its decaying shoots, thereby inhibiting
seed germination and growth in other species. To survive the same allelopathic
effects on itself, however, Sporobolus must move constantly into new areas, which
also means that it has to be less inhibitory to itself than to the surrounding
vegetation. Again we see evidence for feedback from plant niche construction.

The above examples all illustrate that niche construction can have evolutionary
as well as ecological consequences. However, the changes that organisms cause in
their niches, and the resulting dynamics, are seldom investigated in empirical
evolutionary studies, or incorporated into population genetic models. One theoreti-
cal construct which captures some, but not all, of the consequences of niche
construction is Dawkin’s (1983, 1989) ‘extended phenotype’. Dawkins argues that
genes can express themselves outside the bodies of the organisms that carry them.
For instance, the beaver’s dam is an extended phenotypic effect of beaver genes.
However, a dam sets up a host of selective pressures which feed back to act on the
beaver’s teeth, tail, feeding behavior, its susceptibility to predation, its social
system, and many other aspects of its phenotype. It may also affect the many future generations of beavers that may 'inherit' the dam, its lodge, and the altered river, as well as many other species of organisms that now have to live in a world with a dam in it. In pointing out the evolutionary consequences of niche construction, we therefore wish to emphasize that these consequences sometimes persist, or accumulate, or are constantly repeated in the environment, to the point where they significantly influence selection on the organism. This self-induced selection acts on many other aspects of the phenotype, and on many other organisms, even those distant in space and time, and can influence the spread of genetic variation at many loci, including some unconnected with the original phenotypic effect.

Other topics of interest in population biology are concerned with the evolutionary consequences of the changes that organisms bring about in their own, and in other populations', selective environments. For example, habitat selection, frequency-, and density-dependent selection are all areas of analysis in which a phenotypic effect feeds back to affect fitness (Felsenstein, 1976; Rosenweig, 1987; Wright, 1984; Slatkin, 1979a, b). So far, however, these analyses have only focused on those loci which influence the production of the niche-constructing phenotype itself. What is missing is an exploration of the feedback effects on other loci. Studies of maternal inheritance also explore the consequences of female (and male) parents selecting and provisioning the environments in which their zygotes develop, in the process of which they modify the selection pressures to which their offspring are exposed (Grun, 1976; Schluter and Gustafsson, 1993). There is some theoretical evidence that non-Mendelian forms of inheritance can have more general effects on evolutionary dynamics (Feldman and Cavalli-Sforza, 1976; Cavalli-Sforza and Feldman, 1981; Kirkpatrick and Lande, 1989; Cowley and Atchley, 1992), but so far, these models have limited the number of generations involved in this inheritance to two. So, even though each of these topics clearly overlaps with examples of niche construction, at present there exists no body of theory which explores the evolutionary consequences of niche construction in a systematic manner.

Models

Potentially, niche construction is itself an evolutionary player, which can co-direct and regulate natural selection, and other evolutionary forces (Lewontin, 1983; Odling-Smee, 1988, Odling-Smee et al., 1996). Incorporating niche construction into a theoretical analysis is no trivial matter, however, since it involves feedback, or frequency-dependent effects. This feedback imposes constraints on the type of theoretical analysis that might be appropriate. The approach we adopt involves a simple idealized model and uses two-locus population genetic theory. Our theoretical models assume that a population's capacity for niche construction is influenced by the frequency of alleles at a first locus. We label this the E locus, to emphasize that alleles at this locus influence the changes that the organisms bring about in their environment. We define the amount of some resource R in the environment to be dependent on the niche constructing activities of past and present generations of
organisms. More specifically, \( R \) is a function of the frequency of alleles at the \( E \) locus over a number of recent generations. \( R \) may represent the availability of a food or water source, the presence of a predator, the amount of detritus, or any significant component of the population's niche. The amount of the resource in the environment is assumed to vary between 0 (where it is absent) to 1 (where it is accessible to all members of the population). In turn, the amount of this resource in the environment determines the contributions to fitnesses made by a second locus \( A \). Hence, the genotypic fitnesses in part depend upon the frequency over a number of generations of alleles at the \( E \) locus. We argue that this type of model minimally captures the essential dynamics of the evolutionary consequences of niche construction.

The main problem with developing a niche construction theory that is based on a two-locus frequency-dependent selection model is that an infinite number of frequency-dependent relationships are conceivable, and it is not certain that any useful general properties of two-locus frequency-dependent systems will emerge. Consequently, there is a danger that the results of the analysis will be difficult to interpret. In order to circumvent this problem, we have used as a baseline for our model an established fixed-fitness two-locus model well studied in the theoretical population genetics literature, the multiplicative model (Bodmer and Felsenstein, 1967; Karlin and Feldman, 1970; Feldman et al., 1975; Karlin and Liberman, 1979; Hastings, 1981; Christiansen, 1990). We consider the effects of perturbing the standard multiplicative model by introducing frequency dependent fitnesses. This allows us to analyze and describe the effects of niche construction relative to a comparatively well analyzed body of theory. As two-locus theory has been concerned with the effects of recombination, and how its interaction with selection determines the dynamics of the evolving system, we will also focus on these topics in our analysis. In addition, we consider the effects of both niche construction and destruction, and the consequences of manipulating the functional form of the relationship between the fitness of the genotypes at the \( A \) locus and the amount of the resource \( R \).

We consider an isolated population of randomly mating, diploid individuals, defined at two di-allelic loci, \( E \) (with alleles \( E \) and \( e \)) and \( A \) (with alleles \( A \) and \( a \)). In generation \( t \), the frequencies of the four gametes (\( EA, Ee, eA, \) and \( ea \)) are given by \( x_1(t), x_2(t), x_3(t), \) and \( x_4(t) \), respectively, so that the frequencies of alleles \( E \) and \( A \) are

\[
\begin{align*}
p(t) &= x_1(t) + x_3(t), \\
q(t) &= x_2(t) + x_4(t).
\end{align*}
\]

Genotypes at the second locus \( A \) make contributions to the two-locus fitnesses which are assumed to be functions of the frequency with which a resource \( R \) is encountered by organisms in their environment (where \( 0 < R < 1 \)). This frequency is, in turn, a function of the amount of niche construction over \( n \) generations (including the present one), relative to an implicit rate of spontaneous resource recovery or resource dissipation, due to other independent factors in the environment. This treatment of the interaction between the population and the resource is extremely simplified. A more realistic treatment would involve general distributions of the resource, as well as simultaneous ecological dynamics of resource and population (e.g. MacArthur and Levins, 1967; Schoener, 1974). In each generation, the amount of the resource created or
destroyed as a consequence of niche construction is assumed to be a function of the frequency of allele \( E(p_i) \) at the time \( t \). The simplest such function is linear, in which case we may write

\[
R = \sum_{i=-n+1}^{1} \pi_i p_i,
\]

where \( \pi_i \) represents the weight attributed to the impact of the \( i \)th generation's niche construction. We consider three idealized functions, in which the amount of the resource is (i) equally dependent on each generation’s niche construction, (ii) has greater dependency on the activities of more recent generations (for example, if the effects of earlier generations’ activities have partially dissipated), and (iii) has greater dependency on the activities of earlier generations (for example, as may be the case when a founder population moves to a new environment).

(i) \textit{Equal weighting}: All \( n \) generations of niche construction equally influence the frequency of the resource in the environment at time \( t \), so that \( \pi_i = \pi \) for all \( i \). Thus

\[
\pi_i = \pi_{i-1} = \pi_{i-2} = \cdots = \pi_{n+1} = \frac{1}{n}.
\]

so that the frequency of the resource at time \( t \) is given by

\[
R = \frac{1}{n} \sum_{i=-n+1}^{1} p_i.
\]  

(ii) \textit{Recency effect}: Recent generations of niche construction have a greater effect on the frequency of the resource at time \( t \) than have earlier generations of niche construction. Thus

\[
\pi_i > \pi_{i-1} > \pi_{i-2} > \cdots > \pi_{n+1}.
\]

A simple example is expressible in terms of a parameter \( \mu \) which represents the decrement in relative weighting of each generation with time:

\[
\pi_i = \frac{1}{\varphi}, \quad \pi_{i-1} = \frac{\mu}{\varphi}, \quad \pi_{i-2} = \frac{\mu^2}{\varphi}, \ldots, \pi_{n+1} = \frac{\mu^{n+1}}{\varphi},
\]

where \( 0 < \mu < 1 \), and \( \varphi = 1 + \mu + \mu^2 + \cdots + \mu^n = 1 - \mu^{n+1} \). Hence the frequency of the resource at time \( t \) is given by

\[
R = \frac{1 - \mu^{n+1}}{1 - \mu^{n+1}} \sum_{i=-n+1}^{1} \mu^{i-1} p_i.
\]

(iii) \textit{Primacy effect}: Earlier generations of niche construction have a greater effect on the frequency of the resource time \( t \) than more recent generations. Here

\[
\pi_i < \pi_{i-1} < \pi_{i-2} < \cdots < \pi_{n+1}.
\]

As in (ii), a simple example employs the parameter \( \mu \) to represent an increment in the relative weighting of each generation with time:

\[
\pi_i = \frac{\mu^{n-i}}{\varphi}, \quad \pi_{i-1} = \frac{\mu^{n-i-1}}{\varphi}, \quad \pi_{i-2} = \frac{\mu^{n-i-2}}{\varphi}, \ldots, \pi_{n+1} = \frac{1}{\varphi}.
\]
where $0 < \mu < 1$, and $\phi = 1 + \mu + \mu^2 + \cdots + \mu^{n-1} = 1 - \mu^n / (1 - \mu)$. Here the frequency of the resource at time $t$ is given by

$$R = \frac{1 - \mu}{1 - \mu^n} \sum_{i=1}^{n} \mu^{i-1} p_{i-t, i} \ldots$$  \hspace{1cm} (2iii)

Note that in all three cases, if $n = 1$, then the frequency of the resource at time $t$ is equal to the frequency of allele $E$ in that generation. The key point is that in this model the effect of niche construction on the resource, and hence on selection, is reduced to a function of the frequency of allele $E$.

Genotypic fitnesses are given in Table 1. It can be seen that the fitnesses are assumed to be functions of a fixed viability component and a frequency-dependent viability component. The fixed component is given by the $\gamma_i$ and $\beta_i$ terms, which are the fitnesses of genotypes in the standard two-locus multiplicative viability model. The frequency-dependent component is a function of the frequency of the resource $R$, given by equations (1) and (2), and hence the terms involving $c$ express the deviations from constant viabilities due to niche construction, where the size of $c$ scales the amount of niche construction, with $-1 < c < 1$. The frequency-dependent components of the contribution to fitness of genotypes $AA$, $Aa$ and $aa$ are functions of $R$, $\sqrt{R(1-R)}$ and $1-R$, respectively, chosen so that allele $A$ will be favored by this component of selection when the resource is common, and allele $a$ when it is rare. The particular functions chosen here are specified by a parameter $f$ which determines the powers of the relationship between the contribution of locus $A$ to genotypic fitnesses and the frequency of the resource, and a coefficient of proportionality $(c)$ which determines the strength of the frequency-dependent component of selection relative to the fixed fitness component. We analyze this model for the cases when $f = 2$, $1$, and $0.5$, which represent simple examples of relationships between the contribution of locus $A$ to genotypic fitnesses the frequency of the resource $R$. Positive values of $c$ represent niche construction or resource accumulation, while negative values represent niche destruction or resource depletion. It is assumed that the double heterozygotes have equal fitness. The entries in Table 1 give rise to the four standard gametic recursions

Table 1. Multiplicative viabilities with niche construction

<table>
<thead>
<tr>
<th>$\gamma_i$</th>
<th>$\beta_i$</th>
<th>$c(\gamma_i)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$AA$ ($\gamma_i$)</td>
<td>$w_{11} = x_1 x_2 + c R^f$</td>
<td>$w_{11} = x_1 x_2 + c R^f$</td>
</tr>
<tr>
<td>$Aa$ ($\beta_i$)</td>
<td>$w_{21} = x_1 + 4R(1-R)</td>
<td>^{0.5}$</td>
</tr>
<tr>
<td>$aa$ ($\beta_i$)</td>
<td>$w_{31} = x_1 \beta_2 + (1 - R)^f$</td>
<td>$w_{31} = x_1 \beta_2 + (1 - R)^f$</td>
</tr>
</tbody>
</table>

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where $r$ is the recombination rate, $D = x_1 x_4 - x_2 x_3$ and $W$ is given by the sum of the right hand sides of $3a$–$3d$.

**Results**

In order to investigate the effects of niche construction we explore its effects when an external source of selection (independent of the niche construction), represented by the fixed fitness component of selection, favors one of the alleles at one of the loci. Then we focus on the effects of niche construction when the external selection generates overdominance at both loci. To provide a baseline for the subsequent analysis, we first describe the behavior of the model when there is no external source of selection, in which case the fixed multiplicative viabilities ($\alpha$'s and $\beta$'s) may all be taken to be 1.

**No external selection**

Since there is no external source of selection ($x_1 = \beta_1 = x_2 = \beta_2 = 1$), genotypes at the E locus make the same contribution to fitness. Here, the amount of the resource is constant, and given by the frequency of allele $E$. Since $R$ is now unaffected by $n$, equations (1) and (2) simplify to the $n = 1$ case. However, the niche construction generates selection which changes the frequency of alleles at locus A. Where niche construction increases the amount of the resource $R$ (when $\epsilon$ is positive), then in the selective environment characterized by high frequencies of $E$ (i.e. where $R$ is common), allele $A$ will be favored, while in the selective environment provided by high frequencies of $e$ (i.e. where $R$ is rare), allele $a$ will be favored. The reverse is true where niche construction decreases the amount of the resource $R$ (when $\epsilon$ is negative). Figure 1 illustrates that under these conditions the system exhibits a

![Fig. 1. Niche construction can generate selection even when no external source of selecting is acting. The arrows represent the trajectories of a population with a frequency of allele $E$ and $A$ of $p$ and $q$ respectively. The heavy line represents neutrally stable equilibria, and the dashed line unstable equilibria.](image-url)
The evolutionary consequences of niche construction curve of equilibria, similar to that found in sexual selection models (Kirkpatrick, 1982; Gomulkiewicz and Hastings, 1990). The theoretical details are included in Appendix 1. With no external selection, when \( e \) is positive, mean fitness is highest with either \( A \) and \( E \) fixed, or with \( a \) and \( e \) fixed.

**External selection at the E locus**

We first consider the case in which external selection acts only at the \( E \) locus \((x \neq 1, \beta_1 \neq 1, x_2 = \beta_2 = 1)\), representing selection on a niche constructing activity, such as nest building or resource depletion. When allele \( E \) is favored \((x_4 > 1 > \beta_4)\), positive values of \( e \) result in convergence to a single equilibrium point with \( E \) and \( A \) fixed \((x_3 = 1)\), while negative values result in fixation of \( E \) and \( a \) \((x_4 = 1)\). If allele \( e \) is favored by external selection \((x_3 < 1 < \beta_3)\), then positive values of \( e \) result in fixation of \( e \) and \( a \) \((x_4 = 1)\), and negative values cause fixation of \( e \) and \( A \) \((x_2 = 1)\).

Provided all alleles are initially present, a numerical analysis has established that under these conditions populations will always converge to these equilibria. When allele \( E \) is favored, the niche constructing behavior spreads through the population, increasing the amount of the resource, and changing the selection pressure at the second locus so that allele \( A \) is favored. If the amount of the resource present depends only on the present generation's niche construction \((n = 1)\), for example, where the resource is a spider's web, then the response at the \( A \) locus is fairly immediate. However, less predictable patterns emerge when the amount of the resource \( R \) depends on multiple past generations' niche construction \((n > 1)\), for instance, as occurs when earthworms generate topsoil. Under such circumstances there is a timelag between the change in frequency of alleles at the first locus \((E)\), and the response to the frequency dependent selection at the second locus \((A)\).

The effect of increasing the number of generations of niche construction on the response to selection at the second locus is illustrated in Fig. 2. The timelag between the response to the selection at the two loci creates an evolutionary inertia. The inertia occurs because when selection favoring allele \( E \) begins, the resource cannot accumulate as rapidly as allele \( E \) can spread. This means that, at the \( A \) locus, populations will take a number of generations to move away from an equilibrium. In addition, while the resource accumulates to the level necessary to reverse selection at the second locus, the allele favored by the frequency-dependent selection will have dropped in frequency because selection will still be operating in the original direction, and this "lost ground" will have to be made up. The inertia is greatest if there is a primacy effect, since here the amount of the resource depends heavily on earlier generations, when allele \( E \) was rare, and niche construction had had little effect. In contrast, the inertia is at its weakest if there is a recency effect, since here the amount of the resource depends on more recent generations, when allele \( E \) has increased in frequency, and hence niche construction is beginning to have some impact. Although not illustrated in Figure 2, the timelag can also generate an evolutionary momentum. This occurs because when selection at the \( E \) locus stops, or reverses, the amount of the resource continues to accumulate for a
As a consequence of the spread of allele \( E \), niche construction increases the amount of a resource in the environment, which generates selection favoring allele \( A \). The response to selection at the second locus is dependent upon the number of generations of niche construction (\( n \)) which influence a resource \( R \). The Figure plots the frequency of \( E(p) \) and \( A(q) \) against time for \( n = 1, 25, 50 \) and 100. Considerable time-lags may occur between the initial niche construction and the response to the selection it generates. \((x_1 = 1.1, \beta_1 = 0.9, x_2 - \beta_2 = 1, \epsilon = 0.3, r = 0.5, f = 1)\).

number of generations so that, at the \( A \) locus, populations will continue to evolve in the original direction, despite selection favoring the opposite allele. The momentum is also greatest for a primacy effect, and weakest for a recency effect, for similar reasons.

**External selection at the \( A \) locus**

Niche construction creates interesting results when the selection it generates opposes external selection acting at the \( A \) locus (when \( x_1 = 1, \beta_1 = 1, x_2 \neq 1, \beta_2 \neq 1 \)). This would occur if some external agent depletes the resource accrued through niche construction, or accumulates the resource depleted through niche destruction. The results of a numerical analysis are shown in Fig. 3, which illustrates the behavior of populations under external selection favoring \( A \) (\( x_2 > 1 > \beta_2 \)). When there is no niche construction (\( \epsilon = 0 \)), and when the selection generated by the niche construction is very weak (\( 1 > x_2 > \epsilon < 1 - \beta_2 \)), all populations become fixed for \( A \) (assuming that the \( A \) allele is present) (Fig. 3a).

Using the local stability conditions in Appendix 2, it is seen that near \( x_4 = 1 \), if \( \epsilon > 1 - \beta_2 \), fixation of the chromosome \( ea \) is neutrally stable. That is, the niche construction can reverse the instability of this fixation state (Fig. 3b). In the same way, near \( x_1 = 1 \), if \( \epsilon < 1 - x_2 \), alleles \( a \) and \( e \) can increase when rare. Numerical iteration reveals that in this case a stable fully polymorphic equilibrium is possible (Fig. 3c). Again, the niche construction has altered the evolutionary outcome. In this case, it appears that selection at the \( A \) locus is unaffected by the number of generations of niche construction (\( n \)) influencing \( R \). The qualitative behavior of this
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The system is also little affected by the amount of recombination ($r$) or the power of the frequency dependence ($f$).

External selection with overdominance

Finally, we consider the case where external selection generates overdominance ($\alpha_1, \beta_1, \alpha_2, \beta_2 < 1$) (e.g. Bodmer and Felstenstein, 1967). Two locus, two allele
viability models appear to have a maximum of fifteen possible equilibria; four chromosome fixation states, four allelic fixation states, and seven interior equilibria. In the multiplicative model with heterozygote advantage, and internal equilibrium with no linkage disequilibrium \((D = 0)\) exists for all levels of recombination \((r)\), while for tightly linked loci (small \(r\)), two equilibria with disequilibrium \((D \neq 0)\) exist (Moran, 1964; Bodmer and Felsenstein, 1967; Karlin and Feldman, 1970, Karlin, 1975). For small values of \(r\), with overdominance at both loci, the \(D \neq 0\) equilibria are stable (Karlin, 1975), and the internal equilibrium with \(D = 0\) is unstable, although, for an intermediate range of recombination rates, \(D = 0\) and \(D \neq 0\) may both be stable (Franklin and Feldman, 1977; Karlin and Feldman, 1978; Hastings, 1981). One of these \(D \neq 0\) equilibria is characterized by positive linkage disequilibrium, and the other by negative linkage disequilibrium. As a convenient shorthand, we refer to these equilibria as the \(+D\) and \(-D\) equilibria, respectively. As the amount of recombination increases from zero, the amount of linkage disequilibrium at equilibrium decreases, and these two stable equilibria approach the internal equilibrium with \(D = 0\), although they do reach it at the same \(r\) value (Karlin and Feldman, 1978).

First we explore the behavior of the model when there is no linkage disequilibrium (which occurs when \(r > r^*\), defined in Appendix 3). At the \(D = 0\) polymorphic equilibrium, the frequency-dependent selection does not change the equilibrium frequency of \(E\), but \(q\) becomes a function of \(c\), and, for \(f = 1\), the equilibrium is given by

\[
\dot{p} = \frac{(1 - \beta_1)}{(2 - z_1 - \beta_1)},
\]

\[
\dot{q} = \frac{(1 - \beta_2)(1 - z_1 \beta_1) - c(1 - z_1 + \sqrt{(z_1 - 1)(\beta_1 - 1)})}{(2 - z_2 - \beta_2)(1 - z_1 \beta_1) - c(2 - z_1 - \beta_1 + 2 \sqrt{(z_1 - 1)(\beta_1 - 1)})}.
\]

Expressions for the two edge equilibria that are polymorphic at the \(A\) locus (with allele \(E\) or \(e\) fixed) are given in Appendix III. The effect of the frequency-dependent selection is to shift the internal equilibrium and these two edge equilibria in the direction of the arrows given in Fig. 1. It can be seen that positive values of \(\varepsilon\) will increase the equilibrium frequency, \(q\), of allele \(A\) when the resource is common (\(\dot{p} > 1/2\)) and decrease \(q\) when it is rare (\(\dot{p} < 1/2\)). Negative values of \(\varepsilon\) will have the reverse effect. The size of the change in frequency of \(A\) at equilibrium, as a consequence of the frequency-dependent selection, increases the further the frequency of \(E\) is from one half. This is illustrated in Fig. 4, which plots the magnitude of the difference between the equilibrium frequency of \(q\) with \((\dot{q})\) and without \((\dot{q}_0)\) the frequency-dependent selection, against the equilibrium frequency of \(E(\dot{p})\). The frequency-dependent selection thus has its most dramatic effect on the two edge equilibria at which \(E\) or \(e\) are fixed, often sweeping \(A\) or \(a\) to fixation. As might be expected, the larger the value of \(c\), the stronger the shift in equilibrium frequency. An expression for the equilibrium frequency of allele \(A\) as a function of \(c\) and \(f\) is given by (A4) in Appendix 3.
We now explore the behavior of the system when there is linkage disequilibrium. For illustration, consider the effects of frequency-dependent perturbations when $x_1 = \beta_1$ and $x_2 = \beta_2$, in which case the fixed-fitness component of the model is the symmetric multiplicative viability model (Lewontin and Kojima, 1960; Rodmer and Felsenstein, 1967; Karlin and Feldman, 1970). For values of $r$ below $r_0$, given by (A6), in addition to the $D = 0$ equilibrium, there are two symmetric equilibria, at which the frequencies of the gametes may be written as $x_1 = x_4 = \frac{1}{4} + \tilde{D}$ and $x_2 = x_3 = \frac{1}{4} - \tilde{D}$, where $\tilde{D}$ is given by

$$D = \pm \frac{1}{2} \sqrt{1 - \frac{4r(1 + \alpha(1/2)^f)}{(1 + x_1)(1 + x_2)}}.$$  

It is immediately apparent that positive values of $\alpha$ decrease the amount of linkage disequilibrium at equilibrium, while negative values of $\alpha$ increase it. The effect of $\alpha$ on the amount of disequilibrium, and the stability of the $D = 0$ equilibrium is illustrated in Fig. 5. Expressions (5) and (A6) show the effect of manipulating the power of the frequency dependence ($f$). In this case $f = 1/2$ increases the effect of $\alpha$ to a greater degree than $f = 1$, with $f = 2$ having the weakest effect. A similar analysis of the effects of niche construction on the asymmetric viability model is presented in Appendix 4b.

More generally, both positive and negative values of $\alpha$ can both increase and decrease the amount of disequilibrium at equilibrium, depending on the position of the equilibrium. Positive values of $\alpha$ increase the equilibrium frequency of gametes $AE$ and $Ae$ ($x_1$ and $x_3$) for equilibria at the frequency of $E$ is greater than one half ($p > 1/2$), and decrease the equilibrium frequency of gametes $aE$ and $ae$ ($x_2$ and $x_4$) for equilibria at which the frequency of $E$ is less than one half ($p < 1/2$). Negative values of $\alpha$ have the reverse effect. Since the frequency-dependent selection always

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**Fig. 4.** External selection favoring heterozygotes. The Figure shows the difference between the equilibrium frequency of $q$, $\bar{q}$, with ($\epsilon = 0.3$), and without, $\hat{q}_0$, ($\epsilon = 0$), the frequency dependent selection generated by niche construction, plotted against the equilibrium frequency of $p$. 
Fig. 5. External selection favoring heterozygotes, with low levels of recombination. The Figure gives the amount of linkage disequilibrium at equilibrium for the symmetric multiplicative model, plotted against $r$, for $\varepsilon = 0.3$, 0, and $-0.3$. ($x_1 = \beta_1 = x_2 = \beta_2 = 0.8$, $f = 1$). For each value of $\varepsilon$, the $D \neq 0$ points are stable for $r$ smaller than the value at which the curve cuts the $D = 0$ line, and $D = 0$ is stable for $r$ to the right of this value.

changes the frequency of two gametes in ways which result in an increase in linkage disequilibrium (for instance, increasing $x_1$ and decreasing $x_2$ for $\varepsilon > 0$), and alters the frequency of two gametes which result in a decrease in linkage disequilibrium (increasing $x_1$ and decreasing $x_2$), its effect at any point in time depends upon the gamete frequencies. A discussion of this point is given in Appendix 4c.

Discussion

Organisms choose and modify important components of their niches, thereby influencing some of the selection pressures which act on them, and on future generations. We have explored the evolutionary consequences of niche construction using two-locus theory. Our analysis assumes that a population's capacity for niche construction is influenced by the frequency of alleles at a first locus, and that this niche construction is expressed as a change in the frequency of a resource in the population's environment. The amount of this resource in the environment then determines the pattern and strength of selection acting at the second locus. The results suggest that the effects of niche construction can override external sources of selection to create new evolutionary trajectories and equilibria, make polymorphism where external selection could not, and generate unusual dynamics.

When a niche constructing behavior spreads through a population, as reflected in the increase in frequency of allele $E$, it changes the frequency of the resource $R$ in the environment, and therefore the selection pressures acting at the $A$ locus. If the fitness of allele $A$ is proportional to the amount of the resource (that is, when $\varepsilon$ is positive), the resource becomes more frequent as allele $E$ spreads, and the population eventually converges to the equilibrium at which $E$ and $A$ are both fixed. In contrast, if there is niche destruction (that is, $\varepsilon$ is negative), the resource becomes less frequent as allele $E$ spreads, so the population eventually converges to the equilibrium at which $E$ and $a$ are both fixed.
If the amount of the resource in the environment depends only on the current generation's niche construction \((n = 1)\), then the response to selection at the A locus is immediate. A good example of this kind of resource is a spider's web, which is not inherited from past generations, but built by each spider. Here, locus E would represent the genetic variation underlying web building behavior, while A might influence a web defense behavior. On the other hand, if the amount of the resource also depends on the niche constructing activities of past generations \((n > 1)\), a timelag is generated between the change in frequency of alleles at the first locus, and the response to the frequency-dependent selection at the second locus. This would be the case for the earthworm example, where R could represent the amount of topsoil, or soil nutrients, locus E could influence a soil processing, or burrow lining behavior, and locus A could specify some aspect of the phenotype affected by soil conditions, such as the structure of the epidermis, or the amount of mucus secreted.

This timelag, which can be considerably larger than \(n\), is due first to the time it takes for the resource to accumulate (or deteriorate) in the environment, and second to the continuing fall in the frequency of the allele that is eventually favored, before the resource has had time to accumulate (or deteriorate) sufficiently.

This timelag creates both an inertia and a momentum, with at least two consequences. First, assuming it takes an evolving population many generations to change its own selection pressures, it may not be able to do so fast enough to prevent the genetic variation on which its eventual response relies from being lost. Second, once a population reaches a stable equilibrium, it takes a greater period of time, or stronger selection, for the population to move away from it. These results are most pronounced when there is a primacy effect, and smallest when there is a recency effect. In Figure 2, the rate at which allele A replaces allele a, as indicated by \(q\), is the same, regardless of the duration of the preceding timelag, and therefore regardless of how many generations of niche construction \((n)\) are needed to change the selective landscape sufficiently to initiate this replacement. This suggests the presence of an evolutionary 'elasticity' which might facilitate the rapid passage of a population across a boundary between stable equilibria, as the population both crosses and alters its fitness trajectory under the influence of niche construction.

In their theoretical analysis of the evolution of maternal characters, Kirkpatrick and Lande (1989) found that maternal inheritance can generate timelags in the response to selection, and that as a consequence populations may continue to evolve even after selection has ceased. Similar results have been generated by gene-culture coevolutionary models with uniparental transmission of a dichotomous trait (Feldman and Cavalli-Sforza, 1976). Our results are clearly in accordance with these earlier findings. In fact, if we were to set our parameters so that \(n = 2\), with a primacy effect, then niche construction could generate a form of maternal inheritance. That our models generate similar qualitative results to material inheritance models over a broader range of parameter space serves to support Kirkpatrick and Lande's intuition that these effects could be more general than the term 'maternal inheritance' implies.

The consequences of niche construction are interesting when the selection it generates opposes the action of an external source of selection acting at the A locus.
Niche construction which counteracts the action of an external source of selection is likely to be common in nature. Lewontin (1982, 1983) points out that many of the activities of organisms, such as migration, hoarding of food resources, habitat selection, or thermoregulatory behaviors, are adaptive precisely because they damp out statistical variation in the availability of environmental resources. The results of our analysis suggest that, despite this environmental variation, niche construction decreases the probability that such populations will maintain genetic variation. If the size of $c$ is greater than a threshold value, then the frequency-dependent selection overcomes the external selection. If the amount of the resource in the environment accumulates as $E$ increases (positive $c$), then, while the resource is rare, the frequency-dependent selection makes it more difficult for a novel advantageous mutation to invade. Here, despite an external source of selection favoring one allele, the niche construction can generate counter selection which takes an alternative allele to fixation, sending the population along a new evolutionary trajectory. If, on the other hand, the resource is common, then niche construction makes it easier for a novel advantageous mutation to invade (Fig. 3b). In contrast, where a population (say, a herbivore) exploits a naturally replenishing resource (a novel plant species), intermediate levels of the resource are maintained by the balancing of these opposing forces. This may, in turn, maintain genetic variation at loci for which genotype fitnesses depend upon the amount of this resource (for instance, loci affecting the herbivore’s digestive enzymes). Where the amount of the resource decreases as $E$ increases (negative $c$), then if the resource is common, niche construction makes it more difficult for a novel selectively favored mutation both to invade and fix. In this case the niche construction creates new polymorphic equilibria, and consequently increases the amount of genetic variation maintained (Fig. 3c).

With overdominance at both loci, unless the loci are very tightly linked, there is only one stable equilibrium which is completely polymorphic. The effect of the niche construction is to shift the frequency of this internal equilibrium in the direction of the arrows given in Figure 1. That is, positive values of $c$ increase the equilibrium frequency of allele $A$ for equilibria at which the frequency of the resource $R$ is greater than one half, and decrease the equilibrium frequency of allele $A$ for equilibria at which the frequency of $R$ is less than one half. Negative values of $c$ have the reverse effect. The size of the change in frequency of $A$, as a consequence of the frequency-dependent selection, increases the further the frequency of $R$ is from one half. By making the resource very common, or very rare, niche construction can generate selection which results in genetic variation being lost.

If, on the other hand, the two loci are tightly linked, then equilibria with linkage disequilibrium are possible. Both positive and negative values of $c$ can both increase and decrease the amount of disequilibrium at equilibrium, as well as the range of values of $r$ over which these equilibria are stable, depending on the position of the equilibrium. Since the frequency-dependent selection always changes the frequency of two gametes in ways which result in an increase in linkage disequilibrium, and two which result in a decrease, its effect at any point in time depends upon the gamete frequencies, as well as the sign of the disequilibrium.
Niche construction provides a way in which the differential phenotypic expression of genotypes at one locus can be influenced by the genotype at another locus, indirectly via the external environment (Odling-Smee et al., 1996). For instance, the pink coloration characteristic of flamingo species is extracted from the Carotenoid pigmentation of the crustacea they digest (Fox, 1979). Here the genes influencing flamingo prey choice interact with those underlying pigment extraction and utilization, via the food resources in their environment. The niche constructing outputs of individuals not only change selective environments, which feed back to alter the fitnesses of alleles at other loci, but they may also influence the phenotypic expression of those alleles in ontogenetic environments (West et al., 1988). The effect of these interactions, which influence both the nature of the variants subjected to selection, and the pattern of selection acting on those variants, is to introduce the kind of feedback loops between populations and their environments that Robertson (1991) suggests may be important in evolution.

Two other factors could add to the evolutionary significance of niche construction. Firstly, in our models, locus E and locus A occur in the same population, but in principle there is no reason why this could always be the case. Niche construction may also generate interactions between genes in different populations, thus providing a mechanism for driving populations to coevolve in ecosystems, not only by affecting each other directly in the ways which are already modeled by coevolutionary theory, as for instance, in host-parasite coevolution (Futuyma and Slatkin, 1983), but also by affecting each other indirectly via their impact on some intermediate abiotic component of a shared ecosystem, as in competition for a chemical or water resource. For example, if niche construction resulting from genes in a plant population causes the soil chemistry to change in such a way that the selection of genes in a second population, of plants or microorganisms, is changed, then the first population's niche construction will drive the evolution of the second population simply by changing the physical state of this abiotic ecosystem component. Since the dynamics of the intermediate abiotic component may be qualitatively quite different from either the frequency changes in the genes that underlie the niche construction, or the number of niche-constructing organisms in the first population, this indirect feedback between species may generate some interesting, and as yet unexplored behavior in coevolutionary systems.

Secondly, there is no requirement for niche construction to be directly determined by a gene at one locus, before it can alter the selection of a gene at a second locus. Niche construction can depend on learning (Bateson, 1988; Plotkin, 1988), as is the case for the British tits that have changed their selection pressures by opening foil milk bottle tops (Fisher and Hinde, 1949; Sherry and Galf, 1984). In terms of our model, this would evolve a gene at the E locus that would affect the capacity of these tits to learn in the first instance, and only then, and only indirectly, their niche construction. Yet the consequences for a gene at the A locus would still be the same, provided the effect of the niche construction on the environmental resource was unchanged. The opposite could also happen in other cases. Niche construction due to a gene at one locus, could change the distribution of a resource R in an environment in such a way that it forced organisms to learn more about their
environments. This, in turn, might change the selection of an allele at a second locus which influenced their learning capacities.

In conclusion, niche construction is probably pervasive in nature, and it very likely promotes genetic interactions in many situations, including all those mentioned in the introduction. This study is the first theoretical attempt to evaluate Lewontin’s (1983) original intuition that niche construction may be a major source of adaptation in evolution, and that its evolutionary effects deserve serious consideration.

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The evolutionary consequences of niche construction

Appendix I: No external selection

When all \( x, \beta \) are 1 the curve of equilibria illustrated in Figure 1 emerges from summing the \( x_i \) expressions given in (3), and expressing them in terms of \( p, q \) and \( D \) to give:

\[
Wq' = q\left[1 + \epsilon(qp' + (1 - q)(p(1 - p))^{p/2})\right] \quad (\text{Ala})
\]

\[
Wp' = p + \epsilon((pq + D)(qp' + (1 - q)(p(1 - p))^{p/2})
+ (p(1 - q) - D)(q(p(1 - p))^{p/2} - (1 - q)(1 - p)\epsilon)), \quad (\text{Alb})
\]

where

\[
W = 1 + \epsilon q^2 p^2 (1 - q)(1 - p)^{p/2} + 2q(1 - q)(p(1 - p))^{p/2}. \quad (\text{Alc})
\]

It can be seen that when \( p = 1/2 \), (Ala) simplifies to \( q' = q \); the vertical line of equilibria in Figure 1. It can also be seen that when \( q = 0 \) and \( q = 1 \), so that \( D = 0 \), (Alb) simplifies to \( p' = p \); the horizontal lines of equilibria at the top and bottom of Figure 1. The vertical curve of equilibria is structurally unstable since if \( x_1 \) and \( \beta_1 \) are not equal to 1 then it may disappear. From (Ala) and (Alc) we can derive an expression for the change in \( q \) each generation:

\[
\Delta q = q' - q = \frac{q(1 - q)[p^{p/2} - (1 - p)^{p/2}][qp^{p/2} + (1 - q)(1 - p)^{p/2}]}{W} \quad (\text{A2})
\]

from which it can be seen that the sign of \( \Delta q \) is dependent on the sign of \( \epsilon \); and that \( \Delta q > 0 \) when \( p > 1/2 \) and \( \Delta q < 0 \) when \( p < 1/2 \). Hence, if \( \epsilon > 0 \) and \( q = 1 \) fixation boundary is locally stable when \( p > 1/2 \) and the \( q = 0 \) boundary is locally stable when \( p < 1/2 \). Starting from any point on the line \( p = 1/2 \), although \( q \) remains constant it can be shown that \( D \) decreases to zero at the rate \( 1 - r \) per generation. Hence equilibrium points in the \( p = 1/2 \) line have \( D = 0 \), and thus if we write \( q = q_0 \), then \( x_1 = q_0/2 \), for instance.
Appendix 2: Corner eigenvalues

The eigenvalues for the local stability of the four chromosomal fixations are:

\[ x_1 = 1; \frac{1}{x_1}, \frac{1}{1/x_2}, \frac{1}{(x_1 - 1)} \]
\[ x_2 = 1; \frac{1}{x_1}, \frac{1}{1/x_2}, \frac{1}{(x_2 - 1)} \]
\[ x_3 = 1; \frac{1}{x_1}, \frac{1}{1/x_2}, \frac{1}{(x_3 - 1)} \]
\[ x_4 = 1; \frac{1}{x_1}, \frac{1}{1/x_2}, \frac{1}{(x_4 - 1)} \]

Appendix 3: Overdominance with no linkage disequilibrium

The two edge equilibria that are polymorphic at the A locus, with allele e or E fixed, are, respectively

\[ \hat{p} = 0, \quad \hat{q} = \frac{\beta_1(1 - \beta_2) - \varepsilon}{\beta_1(1 - x_2) - \beta_2 - \varepsilon} \] (A3a)
\[ \hat{p} = 1, \quad \hat{q} = \frac{x_1(1 - \beta_2)}{x_1(2 - x_1 - \beta_2) - \varepsilon} \] (A3b)

From Appendix 2, the equilibrium with gamete ae fixed \((x_4 = 1)\) is stable to invasion by A if \(\beta_1(1 - \beta_2) - \varepsilon < 0\), \(\beta_2 < \beta_1 \beta_2\), and \(1 - r < \beta_1 \beta_2 + \varepsilon\), and the equilibrium with gamete AE fixed \((x_1 = 1)\) is stable to invasion by a if \(x_1(1 - x_2) - \varepsilon < 0, x_2 < x_1 x_2\), and \(1 - r < x_1 x_2 + \varepsilon\). It can be seen that if e = 0, or is negative, then these corner equilibria are both unstable, and the polymorphic edge equilibria given by (A3a) and (A3b) will be stable along the edge, but not to invasion by E and e, respectively. Note also that (4) reduces to the edge equilibrium (A3a) if \(\beta_1 = 1, \) and to the equilibrium (A3b) if \(\beta_1 = 1\).

The general expression for the equilibrium frequency of allele A \((\hat{q})\), as a function of e and f, is given by

\[ \hat{q} = \frac{(x_1 + \beta_1 - 2)f(1 - \beta_2)(1 - x_1 \beta_2) + e(x_1 - 1)^{\beta_2}(x_1 - 1)^{\beta_2} - (\beta_1 - 1)^{\beta_2} + e(x_1 - 1)^{\beta_2} - (\beta_1 - 1)^{\beta_2})}{(x_1 + \beta_1 - 2)f(1 - x_1 \beta_2)(1 - x_1 \beta_1) + e(x_1 - 1)^{\beta_2}(x_1 - 1)^{\beta_2} - (\beta_1 - 1)^{\beta_2} + e(x_1 - 1)^{\beta_2} - (\beta_1 - 1)^{\beta_2})} \] (A4)

This reduces to (4) when \(f = 1\). When this is the case, if \(x_1 = \beta_1\), then \(\hat{p} = 1/2\), and \(\hat{q}\) reduces to \(\hat{q} = (1 - \beta_2)(2 - x_2 - \beta_2)\), which is the same equilibrium as in the standard multiplicative model (Moran, 1964), so that the frequency-dependent selection has no effect. Thus the internal equilibrium does not depend on the niche construction of \(p = 1/2\), and depends only weakly if \(p\) is close to 1/2.

Appendix 4: Overdominance with linkage disequilibrium

(i) The symmetric multiplicative viability model

Suppose first that \(x_1 = \beta_1\) and \(x_2 = \beta_2\) (where the fixed-fitness component of the model is the symmetric multiplicative viability model). Here, when \(\hat{p} = 1/2\) the equilibrium equations are
which are satisfied by \( x_1 = x_q \) and \( x_2 = x_3 \).

Substituting \( x_1 = x_q = a + \varepsilon D \), and \( x_2 = x_3 = a - \varepsilon D \), into the recursions, we obtain

\[
W = \frac{1 + x_1 (1 + x_2)}{4} + \varepsilon (1/2)^{1/2} + 4D \varepsilon (1 + a_1)(1 + a_2).
\]

When substituted into the equilibrium \( x_q \) recursion this produces the cubic equation:

\[
D [ (1 + x_q)(1 + x_2)(4D^2 - \frac{1}{4} + \varepsilon (1 + a_1)(1 + a_2)) ] = 0,
\]

whose solutions are \( \varepsilon = 0 \), and

\[
\varepsilon = -\frac{1}{4} \sqrt{1 - 4\varepsilon (1 + a_1(1/2)^{1/2})}.
\]

Thus for values of \( r < r_c \), in addition to the \( D = 0 \) equilibrium, there are two symmetric equilibria, at which the frequencies of the gametes are given by \( x_1 = x_4 = \frac{1}{4} + D \) and \( x_2 = x_3 = \frac{1}{4} - D \).

The eigenvalues of the linearized recursions at the point \( x_q = \frac{1}{4} \), i.e. \( D = 0 \), are

\[
\frac{2x_1(1 + x_1) + 4\varepsilon (1/2)^{1/2}}{(1 + x_1)(1 + x_2) + 4\varepsilon (1/2)^{1/2}},
\]

\[
\frac{2x_1(1 + x_1) + 4\varepsilon (1/2)^{1/2}}{(1 + x_1)(1 + x_2) + 4\varepsilon (1/2)^{1/2}},
\]

\[
\frac{2[1 + x_1, x_2 + 2\varepsilon (1/2)^{1/2} - 2\varepsilon (1 + a_1(1/2)^{1/2})]}{(1 + x_1)(1 + x_2) + 4\varepsilon (1/2)^{1/2}}.
\]

which produce the following three conditions for stability

(i) \( (1 + x_1)(1 - x_2) > 0 \)

(ii) \( (1 - x_1)(1 + x_2) > 0 \)

(iii) \( r > \frac{(1 - x_1)(1 - x_2)}{4(1 + \varepsilon (1/2)^{1/2})} = r_c. \) (A6)

The first two conditions require overdominance at both loci, while the last gives the critical value of \( r \) below which \( D_0 \) is unstable. It is clear that in this case \( r_c \) will be larger for negative values of \( \varepsilon \) and smaller for positive values of \( \varepsilon \). Expressions (A5) and (A6) give a clear indication of the effect of manipulating the power of the frequency dependence (f). In this case \( f = 1/2 \) increases the effect of \( \varepsilon \) to a greater degree than \( f = 1 \), with \( f = 2 \) having the weakest effect.
(ii) The asymmetric multiplicative viability model

In the case where \( x_1 = \beta_1 \) but \( x_2 \neq \beta_2 \), the fixed fitness component of the model is asymmetric (Karlin and Feldman, 1970). Here the condition for the stability of the \( D = 0 \) equilibrium is given by

\[
r > \frac{(1 - x_1)(1 - x_2)(1 - \beta_2)}{2(2 - x_2 - \beta_2)(1 + \alpha(1/2)^\gamma)} = r_0.
\]  

which simplifies to (A6) in the case where \( x_2 = \beta_2 \). The effects of \( \varepsilon \) and \( f \) are the same as in the symmetric viability model. For \( r < r_0 \), the \( x_1 = \beta_1 \), \( x_2 \neq \beta_2 \) condition generates equilibria with \( p \neq 1/2 \). This means that if the \( E \) and \( A \) loci are loosely linked, then \( x_1 = \beta_1 \), always gives a frequency of \( E \) of \( p = 1/2 \) at the stable polymorphic equilibrium, but if they are tightly linked then \( p \neq 1/2 \) (unless \( x_2 = \beta_2 \)). This is of interest here, since the frequency-dependent selection has no effect on the frequency of allele \( A \) if \( p = 1/2 \), but does if \( p \neq 1/2 \). Consider the example \( x_1 = 0.8 \), \( \beta_1 = 0.8 \), \( x_2 = 0.95 \), \( \beta_2 = 0.8 \), which, for \( r > r_0 \), has a stable equilibrium at \( p = 0.5 \), \( q = 0.8 \). At \( r = 0 \), with no frequency-dependent selection (\( \varepsilon = 0 \)), this equilibrium has been shifted to \( p = 0.5057 \), \( q = 0.7747 \) due to linkage disequilibrium. The fact that \( p \) is greater than \( 1/2 \) here means that positive values of \( \varepsilon \) now increase, and negative values decrease the equilibrium frequency of \( A \), for instance \( \varepsilon = 0.3 \) and \( \varepsilon = -0.3 \) give \( q = 0.7833 \), and \( q = 0.7654 \) respectively. Hence, even with \( x_1 = \beta_1 \), the frequency-dependent selection can change the equilibrium frequency of allele \( A \), for small \( r \).

(iii) More complex cases

With \( x_1 \neq \beta_1 \), analytical solutions to the multiplicative model become intractable, and we carried out a numerical analysis. Since the frequency-dependent selection always increases the frequency of two gametes that increase linkage disequilibrium, and decreases the frequency of two gametes that decrease linkage disequilibrium, its effect at any point in time depends upon the gamete frequencies. If \( p \) is small, then the dynamics are dominated by \( x_1 \) and \( x_2 \). Since positive \( \varepsilon \) increases \( x_4 \), and decreases \( x_3 \), it increases \( +D \), and decreases \( -D \). Thus, the reverse is true for negative \( \varepsilon \). For larger values of \( p \), but with \( p < 1/2 \), positive values of \( \varepsilon \) tend to increase the equilibrium amount of disequilibrium at both \(+D\) and \(-D\) equilibria, by increasing the equilibrium value of \( x_4 \) and \( x_2 \) respectively, although large positive values of \( \varepsilon \) reduce the \(-D\) equilibrium by increasing \( x_4 \). For these values of \( p \), negative values of \( \varepsilon \) always reduce the equilibrium amount of linkage disequilibrium, for both the \(+D\) and \(-D\) equilibria.

The reverse pattern is found in the region \( p > 1/2 \), where negative values of \( \varepsilon \) tend to increase the equilibrium amount of disequilibrium at both \(+D\) and \(-D\) equilibria, by increasing the equilibrium value of \( x_4 \) and \( x_2 \) respectively, although large negative values of \( \varepsilon \) reduce the \(-D\) equilibrium by increasing \( x_4 \). When \( p > 1/2 \), positive values of \( \varepsilon \) reduce the equilibrium amount of linkage disequilibrium.
librium, for both the $+D$ and $-D$ equilibria. However, for $p$ close to 1, the dynamics are dominated by $x_1$ and $x_2$. Since positive $c$ increases $x_1$, and decreases $x_2$, it tends to increase the $+D$, and decrease the $-D$ equilibria, with the reverse true for negative $c$. Thus, whether or not positive or negative values of $c$ increase or decrease the amount of linkage disequilibrium depends upon the frequency of allele $E$, and the sign of the disequilibrium.

While it is not generally true that the absolute change in the amount of linkage disequilibrium at equilibrium due to the frequency-dependent selection is greater as $p$ approaches 1 and 0, and moves away from 1/2, the proportional change in the amount of linkage disequilibrium (the absolute change divided by the amount of linkage disequilibrium at $c = 0$), does fit this pattern. This is because while the frequency-dependent selection acting on locus A becomes stronger as the frequency of $E$ moves away from 1/2, typically the amount of disequilibrium shows the reverse pattern.

Although the above results are for $n = 1$, the effect of increasing the number of generations of niche construction that affect $R$ is only to change the rates at which populations converge on the above equilibria, and their stability, and not to change the equilibria themselves.