Version: 27 May 2010

The 'struggle for existence:' why the mismatch of basic theory in ecology and evolution?

James Mallet

Radcliffe Institute, Harvard University,
Byerly Hall, 8 Garden St., CAMBRIDGE, MA 02138, USA

and:

Galton Laboratory, Department of Genetics, Evolution and Environment, University College London,
4 Stephenson Way, LONDON NW1 2HE, UK

Corresponding author: James Mallet (j.mallet@ucl.ac.uk)
tel: +44 - (0)20 7679 7412
or: +1 (617) 496 2780

Keywords: Density-dependent selection, frequency-dependent selection, hard selection, r selection, K selection, Lotka-Volterra competition, logistic population growth.

Darwin's 'struggle for existence' was based on Malthus's demography: competition among species should be similar to natural selection among genotypes within species. Yet the role of population ecology in evolution remains debated. In particular genetic variation for intrinsic rate of increase *r* is seemingly neutral in conventional 'rigid carrying capacity' (*r*-*K*) demographic models, whereas *r* is equivalent to fitness in simple population genetic models. Due to focus on conventional models, known results linking ecological competition and natural selection have been obfuscated.

Here I show that simple theory based on birth and death rates instead supports an 'elastic carrying capacity' reinterpretation of population regulation. This model solves theoretical paradoxes introduced by the conventional model, and explains hitherto puzzling experimental results. Furthermore, selection of many different kinds, including simple Fisherian *r* selection, density-dependent and frequency-dependent selection, as well as hard and soft selection all emerge cleanly from the resulting haploid demographic formulation. Diploidy and sexuality add complexity, but the same principles apply.

These findings clarify earlier debates about *r* and *K* selection, and can lead to better understanding of spatial evolution, life history evolution, and genetic loads. They demonstrate the fundamental unity between population ecology and evolution.

Introduction: Evolution and ecology

It is a curious feature of the theoretical core of evolutionary biology – population genetics – that it often ignores the original impetus of its creation, population ecology. The idea of natural selection was triggered only after Darwin read a treatise on overpopulation and human suffering (Malthus 1826). The earliest ecologists to investigate competition were motivated by an interest in Darwin's 'struggle for existence,' or natural selection (Gause 1934; Scudo and Ziegler 1978), but later ecologists focused mainly on population densities of competing species. In contrast, the originators of population genetic theory, Ronald A. Fisher, J.B.S. Haldane and Sewall Wright, based their theories on population ecology, but generalized selection almost exclusively in terms of gene frequencies within species. As a result, textbooks today treat population ecology and evolution by natural selection as almost entirely separate topics.

This separation is reasonable if gene frequencies and population density do not interact. Unfortunately, interaction is likely: selection is caused by differences in fertility and survival, the same parameters that affect population density. A deliberate fusion between evolution and ecology, 'population biology,' was attempted in the 1960s and 1970s (Wilson and Bossert 1971; Roughgarden 1979). Today, a common opinion is that the effort has failed (Lewontin 2004).

Competition among species closely resembles natural selection among genotypes, so it ought to be possible to build theories of population genetics equivalent to ecological competition. However, efforts to unify ecology and evolution have been frustrated because theories of population growth and competition conflict with common sense about evolution. In particular, ecological theory seems to demand that genotypes with the highest carrying capacities K will be fitter, while intrinsic growth rate r does not affect the

outcome (MacArthur 1962; Roughgarden 1971); meanwhile classical evolutionary theory employs *r* as the basis of fitness (Fisher 1918; Fisher 1922; Fisher 1930)?

Here I use simple models to demonstrate the fundamental unity between population ecology and evolution. The literature on combining demography with evolution is voluminous and has a long history. Many problems have been solved repeatedly (Kostitzin 1937; MacArthur 1962; Smouse 1976; Slatkin 1979; Asmussen 1983a; Asmussen 1983b; Christiansen 2004). Crow and Kimura (1970) and Kimura (1978) made major inroads. However, the results were never linked and the overall problem remains elusive. Smouse's important paper (1976), for example, is rarely cited, perhaps because it did not appear to address a key debate of its time, MacArthur and Wilson's hypothesis of *r* and *K* selection (MacArthur and Wilson 1967).

The overall topic is often known as 'density-dependent selection.' The issue has sometimes been viewed as little more than an interesting backwater, an embellishment on simpler population genetics to 'allow for' selection in density regulated populations. However, because natural selection depends on differences in fertility and viability that also affect population growth and density regulation, the topic is fundamental: its solution leads to a clearer understanding of natural selection in general.

I first briefly review paradoxes that have appeared in demographic models, before applying their resolutions to natural selection.

Population growth and competition: problems with conventional formulations

The per capita instantaneous growth rate, $R = B - D$ is sometimes called the 'Malthusian parameter' (Fisher 1930) after Malthus' original insight (Malthus

1826). It is the excess of births over deaths in the population, both of which may be functions of population density:

$$\frac{1}{N}\frac{dN}{dt} = R(N) = B(N) - D(N) \qquad (1)$$

At its simplest, populations are unregulated, and both $B(N) = b$ and $D(N) = d$ are constants: the intrinsic growth rate itself becomes constant, $r = b - d$. Then:

$$\frac{1}{N}\frac{dN}{dt} = r \qquad (2)$$

On integration, this gives exponential growth, $N_t = N_0 e^{rt}$, where N_0 is the initial number at time $t = 0$.

When populations are regulated, growth rate is expected to decline monotonically as a function of density, N. (I comment on low density 'Allee effects' below, but ignore them for now). A well-known function is the logistic formulation introduced by Gause (1934), in which growth declines linearly to 0 when $N = K$ and becomes negative when $N > K$. The new parameter K is the equilibrium population density, known as 'carrying capacity:'

$$\frac{1}{N}\frac{dN}{dt} = r\left(1 - \frac{N}{K}\right) \qquad (3)$$

Logistic growth is the basis for many models in population biology, and the integral for this equation is also well known (e.g. Crow and Kimura 1970; Roughgarden 1979; Pastor 2008).

In the present context, it is worth noting that equation 3 has often been claimed to be unrealistic, and indeed it can behave paradoxically. The best known case occurs when $r < 0$, and $N_0 > K$ as a starting condition: equation 3

predicts a singularity when population growth accelerates until $dN/dt \rightarrow +\infty$ after a finite time $t = \frac{\log\{N_0/(N_0 - K)\}}{|r|}$ (Kuno 1991). This is known as Levins' paradox (Hutchinson 1978; Gabriel et al. 2005). A number of other paradoxes are known (Kuno 1991; Ginzburg 1992); for instance, when $r = 0$, the population neither grows nor declines no matter how large N is, even if larger than K (Kuno 1991).

In modern theories of the evolution of geographic range limits of species, for example, it would be sensible to allow $r < 0$ in unfavourable regions (Kirkpatrick and Barton 1997; Polechová et al. 2009), and stochastic fluctuations should sometimes lead to a population exceeding equilibrium density. Models of population dynamics should deal with such situations. If $r < 0$, with $N_0 > K$, we intuitively expect the population to decline to extinction rather rapidly since exceeding the carrying capacity should reduce population growth still further below zero. Instead, in Levins' paradox, a product of two negatives in equation 3 becomes positive and the population increases to infinity. Therefore, some ecologists now interpret these paradoxes as serious failures rather than just annoyances (Kuno 1991; Ginzburg 1992; Gabriel et al. 2005; Pastor 2008).

Another paradox is produced in theories of competition that are extenstions of equation 3. The best known model is Lotka-Volterra competition:

$$\frac{1}{N_i}\frac{dN_i}{dt} = r_i\left[1 - \left(N_i - \sum_{j \neq i}\alpha_{ij}N_j\right)\Big/K_i\right] \qquad (4)$$

where the subscripts refer to species i and j and α_{ij} represents the density-dependent interaction effect of species j on species i. Conditions for coexistence or exclusion of one by the other are well known and readily demonstrated graphically by the method of isoclines (Gause and Witt 1935).

The inequalities that determine outcomes (Table 1) for two species have appeared in many ecology texts since then. Yet it is paradoxical that outcomes of competition depend only on α and K, and not on intrinsic growth rates, r. The rapidity with which equilibrium is attained does depend on r (Roughgarden 1971), as does the stability of equilibria in models with ≥ 3 species (Strobeck 1973). However the paradox of the lack of involvement of r in outcomes remains puzzling (Maynard Smith 1989): p. 19; (Begon et al. 1996): 278). This is also a major problem for natural selection (discussed below). Noting these issues, John Maynard Smith argued:

> There is an important difference in kind between equations [2, this paper] and [3, this paper]. This can be expressed by saying that equation [2] can be microscopically justified, whereas equation [3] is descriptive and phenomenological. ... The justification for equation [3] is that it accurately describes some cases of change in population number, and that it is mathematically simple, but not that it is derived from what individuals are doing (Maynard Smith 1989): 19, see also (Maynard Smith 1974): 18-19).

Maynard Smith and others have therefore argued that the logistic is inadequate to incorporate our intuitions about r.

Population growth and competition: paradox resolution via the 'elastic K' logistic

However, equations 3 and 4 can in fact be justified microscopically from equation 1 in the same way as equation 2. This justification was first published in the 1930s (Kostitzin 1937; Kostitzin 1939); since then, it has been independently re-derived several times from the underlying birth-death model (Prout 1980; Bell 1997; Christiansen 2004; Gabriel et al. 2005; Pastor 2008; Gotelli 2008):

$$\frac{1}{N}\frac{dN}{dt} = r - cN \qquad (5)$$

This follows from first principles if $B(N)$ and $D(N)$ in equation 1 are both linear, where $r = (b - d)$ as before, and a 'crowding effect' $c = (\beta + \delta)$ is the sum of density-dependent effects on birth and death (Appendix 1). K is no longer a 'rigid' carrying capacity; instead, setting equation 5 to zero results in a dynamic or 'elastic' equilibrium, $K = (b - d)/(\beta + \delta) = r/c$, a balance between density-dependent and density-independent birth and death parameters. For competition, equation 4 is modified similarly (Appendix 1):

$$\frac{1}{N_i}\frac{dN_i}{dt} = r_i - c_i(N_i + \sum_{j \neq i} \alpha_{ij} N_j) \qquad (6)$$

where the crowding effect on an individual of species i by another species j is α_{ij} relative to that of its own species, c_i.

Density-independent elements, r, and density-dependent parameters, c, are completely separated in equations 5-6. In contrast, the classic 'rigid K' parameterization (equations 3, 4) enforces a paradoxical effect of r on density-dependent regulation (because $c = r/K$). The 'elastic K' logistic (equation 5) was the form first proposed for population dynamics (Verhulst 1838), and was used to derive all the major results in ecological competition (Lotka 1925; Volterra 1927; Lotka 1932), such as the outcome inequalities in Table 1. As it is based on a non-linear birth-death process, it is also formally equivalent to stochastic, dynamic equilibrium models of colonization and extinction such as the Levins metapopulation model and the MacArthur-Wilson theory of island biogeography (Hanski and Gilpin 1991).

Levins' paradox no longer applies because c must be positive to give population regulation with a positive equilibrium density (Kuno 1991).

Crowding will always reduce population growth in equation 5; therefore, when $r < 0$ the 'carrying capacity' K is also negative. A negative K implies an absence of a positive equilibrium rather than an actually negative N: the population always declines to extinction when $r < 0$ or $r = 0$, as seems most logical. In competition, both density-dependent c and density-independent r parameters in equation 6 are directly involved in the outcome of competition, even for two species. The Lotka-Volterra paradox is thus resolved: relative values of r are now important for outcomes. For example, stable coexistence, condition 4 (Table 1), now requires $1/\alpha_{21} > r_1c_2/r_2c_1 > \alpha_{12}$. The elastic K formulation thus resolves all known paradoxes of the rigid K formulation (Kuno 1991; Christiansen 2004; Lewontin 2004; Gabriel et al. 2005; Pastor 2008). Therefore, it is perhaps best not to think of K as a rigid feature of the environment, as seems implied by the term 'carrying capacity.'

These discoveries have not yet penetrated into mainstream ecology, although some textbooks do now mention the Kostitzin derivation (Begon et al. 1996; Pastor 2008; Gotelli 2008). Of course, the logistic is only the simplest of a family of models for population regulation, and higher-order models may sometimes be required (Ayala et al. 1973; Ginzburg 1992). Nonetheless, paradoxes similar to those above will arise in more complex rigid K models.

As an example of a more complex model, consider 'Allee effects.' These are beneficial effects of population size at low density, and can lead to an unstable threshold density below which the population crashes to extinction. Allee effects are not incorporated into the logistic, but it is not a problem to add parameters to do so. Such models give logistic-like behaviour when N is greater than the Allee threshold (Kostitzin 1940; Asmussen 1979). Thus although the lack of Allee effects in equations 3-6 can be unrealistic, this is not a good argument against logistic approximations near equilibrium.

Rigid *K* formulations appear very ingrained, and to some appear intuitively correct. For example, Ginzburg's preference was to "side with intuition" when confronted with a problem caused by the rigid *K* logistic: his intuition dictated that equilibrium density should remain constant under changing constant mortality (Ginzburg 1992). It is here suggested that, as with relativity in physics, siding with logic rather than intuition can resolve the paradoxical 'thought experiments' given above.

An important justification for the elastic *K* logistic is that it can be derived from explicit consideration of births and deaths (Kostitzin 1937; Prout 1980; Pastor 2008), while the rigid *K* model cannot (Appendix 1). Logistic-like models can also be derived from physical chemistry models of consumers exploiting either finite or constantly renewed non-living resources (Williams 1972; Pastor 2008). Similar, but more general justifications for logistic-type regulation have been based on ideas of energy conservation, efficiency, and energy flow (Lotka 1925; Slobodkin 1960).

Finally, perhaps the most powerful evidence in favour of elastic *K* formulations in population dynamics is empirical.

Empirical evidence for elastic K population regulation

At first sight, it seems unlikely that mere parameterization of the logistic could be at all important in nature. However, if *r* and *c* are mechanistically independent, as in Kostitzin's version of the logistic, this might be demonstrable in real organisms. Under elastic *K*, we might expect *r* to be positively correlated with $K = r/c$. In fact, tests of life-history trade-offs between *r* and *K* in experimental data have often produced results that were highly puzzling under a rigid *K* interpretation. For example, experiments on *Paramecium* showed strong positive correlations of fitted *r* and fitted *K* among

strains of *P. primaurelia*, and among different species of *Paramecium* and other ciliates (Luckinbill 1979). Positive correlations were also found for inbred strains of *Drosophila melanogaster* when grown alone (Mueller and Ayala 1981). Furthermore, when populations of *P. primaurelia* were selected for higher *r*, a positive response was seen in *K* as well (Luckinbill 1979). Although quite unexpected under the *r* vs. *K* trade-off ideas of their time (see below), these results are perfectly in accord with elastic *K* theory.

Similar positive correlations between *r* and *K* occurred among laboratory populations of *Nephotettix* leafhoppers feeding on a fixed input of resource (rice seedlings) at different temperatures (Fig. 1). Crowding effects $c = r/K$ were uncorrelated with *K* both within and among species normally feeding on rice (Valle et al. 1989; Kuno 1991). These results are expected if the crowding effects *c* are relatively independent of temperature, while maximal per capita rate of increase *r* (as well as its effect on an elastic *K*) is more strongly affected. Possibly, *r* depends strongly on metabolic processes in these species, explaining the temperature dependency within species of *r* and the consequent response of equilibrium density *K*.

Toxic chemicals provide another means to alter population growth. Data on many organisms showed that *r* was directly proportional to *K* as *r* declined after treatment with toxins (Hendriks et al. 2005). These results are again as expected under elastic *K*, in which density-independent death rates *d*, and therefore *r* (Appendix 1) should be directly proportional to equilibrium density *K*. If *K* were not elastic, pesticides would be considerably less useful.

Theory of natural selection based on clonal genotype competition

The simplest case of natural selection supposes that populations of haploid alleles, haplotypes, or clonal genotypes compete only by growing at different

rates. The goal here is to understand how changes in frequency p_1 of a focal allele (allele 1) emerges from population growth. By analogy with equation 1, we compare the growth in density, n_1, of the focal allele 1 with that of all other allelic types, n_2, in a population of total density $N = n_1 + n_2$:

$$\frac{1}{n_1}\frac{dn_1}{dt} = R_1 = B_1 - D_1 \qquad (7a)$$

$$\frac{1}{n_2}\frac{dn_2}{dt} = R_2 = B_2 - D_2 \qquad (7b)$$

Meanwhile, the entire population grows according to:

$$\frac{dN}{dt} = R_1 n_1 + R_2 n_2 \qquad (7c)$$

B_1, D_1, B_2, and D_2 and therefore R_1 and R_2 are functions that may depend on allelic densities n_1 and n_2. Then it is true in general that the rate of change of allele frequency is as follows (from Appendix 2, equation A2.4):

$$\frac{dp_1}{dt} = \{R_1 - R_2\} p_1(1 - p_1) \qquad (8)$$

$S = R_1 - R_2$ measures the strength of natural selection, while the values of R measure allelic fitnesses; these derived functions also depend, in general, on allelic density because of their basis in viability and fertility.

Exponential growth

A special case of equation 8 supposes that the population is not subject to regulation, and genotypes differ only in constant intrinsic growth rates, so that $B_1 - D_1 = r_1$ and $B_2 - D_2 = r_2$ are constants. The parameters r_1 and r_2 were

Fisher's (Fisher 1922; Fisher 1930) measure of fitness. Substituting into equation 8, the change of allele frequency is given by:

$$\frac{dp_1}{dt} = \{r_1 - r_2\} p_1(1-p_1) = sp_1(1-p_1) \qquad (9)$$

The selection function, S becomes a constant $s = r_1 - r_2$; s measures the strength of selection. Equation 9 is itself a kind of logistic (here p_1 is analogous to N/K in equation 3), even though both types increase without limit (equation 8, Fig. 2A). Integration gives (Fisher 1918; Fisher 1922; Haldane 1924; Lotka 1925; Crow and Kimura 1970):

$$\log_e\left[\frac{p_1}{(1-p_1)}\right] = C + st \qquad (10)$$

Here, $C = \log_e[p_{1,0}/(1-p_{1,0})]$, which depends on the starting allele frequency, $p_{1,0}$. Thus, when $\log\left[p_1/(1-p_1)\right]$ (the 'logit' transformation of p_1) is plotted against time, the sigmoid growth of p_1 is straightened, giving a constant slope s (Fig. 2B).

This linear relationship of logit allele frequency to time, equation 10 (Fig. 2B), suggests a kind of 'gold-standard' for Fisherian constant fitness (Crow and Kimura 1970; Smouse 1976). Conformity in more complex models to logit-linear natural selection would imply that 'Fisherian' constant, density-independent fitnesses, as well as weak 'Wrightian' selection in discrete generations (Appendix 3) provide a good representation of natural selection.

Logistic population regulation

The implicit assumption that populations grow without limit does not at first sight seem to provide a particularly fruitful foundation on which to build a

population genetics of natural selection (Leigh 1971; Wagner 2010). When population numbers are regulated, for instance via competition among alleles, haplotypes, or clonal genotypes, the mathematics of natural selection should become considerably less simple. Assuming Lotka-Volterra competition, equations 6 and 7 specify overall population growth as follows:

$$\frac{dN}{dt} = \left[r_1 - c_1(n_1 - \alpha_{12}n_2)\right]n_1 + \left[r_2 - c_2(n_2 - \alpha_{21}n_1)\right]n_2 \qquad (11)$$

Substituting these functions into equation 8, gene frequency evolves as follows (Appendix A2.6, see also Smouse 1976):

$$\frac{dp_1}{dt} = \left\{(r_1 - r_2) - N\left[(c_1 - \alpha_{21}c_2)p_1 - (c_2 - \alpha_{12}c_1)(1 - p_1)\right]\right\}p_1(1 - p_1) \qquad (12)$$

The terms in brackets before $p_1(1 - p_1)$ form the new natural selection function $S(N, p_1)$ of overall population density and gene frequency. S is now dependent on values of c and α, as well as r for each allele. Equation 12 is the general model for natural selection corresponding to Lotka-Volterra competition among haploid or asexual populations. This selection is both frequency- and density-dependent since equation 12 depends both on allele frequencies p_1 and on overall population density N.

Some special cases are especially interesting:

Logistic regulation special case 1: 'r selection'

If heritable differences among alleles affect only r_1 and r_2 , all other parameters being equal (i.e. $\alpha_{12} = \alpha_{21} = 1$, $c_2 = c_1 = c$), equation 12 reduces to:

$$\frac{dp_1}{dt} = \{r_1 - r_2\}p_1(1 - p_1) \qquad (13)$$

... in other words, identical to equation 9, giving a Fisherian gold-standard type of evolution, even though the population is now regulated (Crow and Kimura 1970; Smouse 1976). The same density-independent result is also true somewhat more generally for variable c and α, if $c_1/c_2 = \alpha_{21} = 1/\alpha_{12}$ (equation 12). Evolution is identical however far either allele is from its equilibrium density. This is because only density-independent parameters r differ. Perhaps Fisher (1930) knew this: he did not need to make an assumption of exponential growth, as is sometimes believed.

This result (Volterra 1927; Leslie 1957; Smouse 1976) has gone largely unrecognized (MacArthur 1962; Roughgarden 1971; Desharnais and Constantino 1983; Asmussen 1983a), because r is convoluted with K in rigid K population regulation. As we have seen, with $K_1 = K_2 = K$ and $\alpha_{12} = \alpha_{21} = 1$, values of r have no effect on outcomes: coexistence of alleles is neutrally stable (Table 1). Curiously, only density dependent selection is possible under conventional *r-K* regulation, thereby making impossible the most basic, Fisherian population genetic model. One apparent exception depends on the ruse of forcing density regulation of each allele to depend in part on the value of r in the other haplotype as well as overall density N (case 2 in Crow and Kimura 1970: 27); this hardly seems justified. Even when recognized, the elastic K logistic was characterized as "weaker population control," a situation that "is probably quite unusual in nature" (case 3 in Crow & Kimura 1970: 28). A suitable name for the current case is 'pure r selection,' which has a different meaning to MacArthur & Wilson's term when $n_i >> 0$ (see below). Because c is constant, equilibrium density K_i can vary, and is directly proportion to r_i.

As in exponential growth, this pure r selection is independent of density and gene frequency. However, selection does strongly affect density, and is therefore also a form of 'hard selection' (*sensu* Christiansen 1975; the alternative, 'soft selection,' would not affect population density). Population

geneticists often assume soft selection implicitly, and ignore population density. This is clearly not a valid assumption even in this, the simplest form of Fisherian natural selection.

Logistic regulation special case 2: 'c selection'

If selection is primarily controlled by heritable differences in crowding effects *c* (or equivalently in equation A2.6b, variable *K*), but all other effects are again identical, (i.e. $\alpha_{12} = \alpha_{21} = 1$ and and $r_2 = r_1 = r$). Equation 12 reduces to:

$$\frac{dp_1}{dt} = \{N(c_2 - c_1)\}\, p_1(1 - p_1) \qquad (14a)$$

This is similar, again, to the Fisherian gold-standard of equation 9, but is now directly dependent on population density *N*. Any selection to minimize crowding is most effective in high density populations. The equivalent result from the traditional rigid *K* logistic (equation A2.6b) appears somewhat less elegant (case 4 in Crow and Kimura 1970: 29):

$$\frac{dp_1}{dt} = \left\{ rN\left[\frac{K_1 - K_2}{K_1 K_2} \right] \right\} p_1(1 - p_1) \qquad (14b)$$

This type of selection might be termed 'pure *c* selection.' Unlike *r* selection, *c* selection is density-dependent, but like *r* selection it is independent of gene frequency and hard – it again tends to increase equilibrium population size.

Logistic regulation special case 3: 'K selection' sensu MacArthur & Wilson

An interpretation of the intent of MacArthur's pioneering paper (MacArthur 1962) and of the '*K* selection' of MacArthur & Wilson (MacArthur and Wilson 1967) would allow both r_i and K_i are allowed to vary among alleles, while

crowding is still effected by all alleles identically ($\alpha_{ij} = 1 \; \forall \; i,j$). This combines special cases 1 and 2 and gives the expected combination of equations 13 & 14:

$$\frac{dp_1}{dt} = \left\{(r_1 - r_2) + N\left(c_2 - c_1\right)\right\} p_1(1 - p_1) \qquad (15a)$$

Or expressed in rigid K formulation:

$$\frac{dp_1}{dt} = \left\{(r_1 - r_2) - N(r_1 / K_1 - r_2 / K_2)\right\} p_1(1 - p_1) \qquad (15b)$$

As with equation 13, selection is density-dependent, frequency-independent, and hard. Equilibrium densities K always increase (as in MacArthur 1962), because selection favours both increased r and reduced c. Interior equilibria are impossible under either r or c selection or their combinations in these haploid models; Lotka-Volterra conditions 1 & 4 cannot be met (Table 1). Virtually identical results for r selection, c selection and their combination (Macarthurian K selection) were obtained in another elastic K model differing only in logarithmic rather than linear density dependence (Kimura 1978).

Logistic regulation special case 4: 'α selection'

Suppose selection does not alter equilibrium density, i.e. $K_1 = K_2 = K = r_1/c_1 = r_2/c_2$. The case is mathematically identical to the general equation 12:

$$\frac{dp_1}{dt} = \left\{(r_1 - r_2) - N\left[\left(c_1 - \alpha_{21}c_2\right)p_1 - \left(c_2 - \alpha_{12}c_1\right)(1 - p_1)\right]\right\} p_1(1 - p_1) \qquad (16)$$

It may seem puzzling that equilibrium density K may go up, down, or not change at all during evolution, while gene frequency evolution obeys the same equation 12 = 16. This is explained because in equation A2.6b K_i parameters appear always in the combinations r_i/K_i or K_i/K_i and so can be

substituted by others. Equilibrium density K is important for population dynamics, and in evolution when $\alpha_{ij} = 1 \; \forall \; i,j$, but has variable effects in more general cases. This type of selection might be termed 'α selection' (Gill 1974; Joshi et al. 2001). An interesting simplification occurs when all solitary allelic growth parameters are identical, $r_1 = r_2 = r$ and $c_1 = c_2 = c = r/K$, so that alleles compete only via differing α interactions, and equilibrium population density K does not evolve:

$$\frac{dp_1}{dt} = \left\{ cN\left[(1-\alpha_{12})(1-p_1) - (1-\alpha_{21})p_1\right]\right\} p_1(1-p_1) \qquad (17)$$

This kind of selection might be termed 'pure α selection,' since only values of α differ among alleles.

Even when density remains the same during evolution, we see a two-phase evolutionary process on a logit scale (equation 12, as in Figs. 3-6, discussed below). Thus selection is frequency-dependent. Overall density N remains approximately constant during replacement evolution, although 'blips' in population density can occur when selection is strong (Figs. 5A, 6A). Pure 'α selection' can be both effectively density-independent and frequency-dependent. Furthermore, because equilibrium density does not change, selection can be approximately 'soft' (*sensu* Christiansen 1975). Frequency-dependent selection emerges from these simple demographic models only via differences in α (interaction) among alleles.

Interior equilibria are possible, both stable and unstable, and are given by conditions 1 and 4 for Lotka-Volterra competition (Table 1). A special case of this in equation 17 occrs when $\hat{p}_1 = (1-\alpha_{12}) / \left[(1-\alpha_{21}) + (1-\alpha_{12})\right]$. Evolution towards or away from interior equilibria will, however, give hard selection, since overall density at equilibrium will be higher than K for stable equilibria, or lower for unstable, even when K does not differ among alleles.

Logistic regulation: general case of haploid selection

For the most general kinds of selection, equations 12 and A2.6b behave similarly to that under α selection. Evolution will consist of two approximately logit-linear phases; when allele 1 is rare, $n_1 \approx 0$, and if the population is at approximate density equilibrium, $n_2 \approx K_2$ (as in Fig. 3), then the rate of evolution will be:

$$\frac{dp_1}{dt} \approx \frac{r_1(K_1 - \alpha_{12}K_2)}{K_1} p_1(1-p_1) = \left(r_1 - \frac{r_2 \alpha_{12} c_1}{c_2} \right) p_1(1-p_1) \qquad (18a)$$

Similarly, when allele 1 is common, the rate of evolution will be:

$$\frac{dp_1}{dt} \approx -\frac{r_2(K_2 - \alpha_{21}K_1)}{K_2} p_1(1-p_1) = -\left(r_2 - \frac{r_1 \alpha_{21} c_2}{c_1} \right) p_1(1-p_1) \qquad (18b)$$

Provided that density equilibration is rapid compared with the rate of evolution, we expect initial evolution of a rare advantageous allele at one constant rate, followed by a shift during evolution to a different constant rate mode when the allele becomes more common, when plotted on a logit scale as in Figs. 3-6. Even with slow replacement evolution (and therefore, weak selection), significant differences in *r*, *K* and α among alleles will mean that replacement evolution is frequency-dependent and deviates from the Fisherian gold-standard: it is no longer linearized by a logit transformation (Figs. 3B, 4B, 5B, 6B). In equations 12, 16 & 17, stable or unstable polymorphisms are possible, as in generalized Lotka-Volterra competition. Condition 4 for Lotka-Volterra coexistence must be met for a polymorphic equilibrium to be stable, and condition 1 for it to be unstable (Table 1).

Equation 12 also shows that natural selection will generally be density-dependent and frequency-dependent; it will also typically affect population density (either up or down), so selection is hard. Even weak selection can result in major changes in population size and variable rates of evolution during replacement. Results of the various cases are summarized in Table 2.

Natural selection and competition in diploids: the MacArthur and Wilson hypothesis of r and K selection

The results so far apply only to the simplest haploid asexuals. More generally, selection might entail competition among sexual diploids: at its simplest among genotypes at a biallelic locus, A_1A_1, A_1A_2, and A_2A_2. This introduces interactions among individuals due to mating, genotypic interactions due to dominance and heterosis, and variation among progenies of each genotype. All these combine in complex ways with the purely competitive interactions discussed above for haploids. The difficulty of analysing diploid models has been exacerbated still further by opaqueness introduced by the rigid K logistic formulation, and in many cases by additional complexity due to modelling in discrete generations. Here, I discuss results for diploids by referring back to the haploid, continuous time cases to which they are most similar.

Diploidy has been explored both in continuous-time models (Kostitzin 1937; MacArthur 1962; Smouse 1976; Desharnais and Constantino 1983), and in discrete-time Wrightian models (Anderson 1971; Charlesworth 1971; Roughgarden 1971; Asmussen and Feldman 1977; Anderson and Arnold 1983; Asmussen 1983b). Since weak selection in discrete generations can be approximated by a continuous-time equivalent, I here ignore the extra paradoxes and chaotic behaviour introduced by discrete time models (Charlesworth 1971; Asmussen and Feldman 1977; Asmussen 1983b). These problems do not occur under weak selection and can be eradicated by using

arguably more appropriate discrete time formulations (e.g. equation 20 in Asmussen and Feldman 1977).

In MacArthur's original analysis (MacArthur 1962), genotypes were assumed to differ in values of K, but selection depended only on total density of all three genotypes $N = N_{11} + N_{12} + N_{22}$ equivalent in the logistic to the assumption that $\alpha_{ij} = 1 \; \forall \; i,j$ (where i and j represent combinations of diploid genotypes 11, 12, 22). This is MacArthur & Wilson's K selection. Unlike the equivalent haploid/asexual model (case 3, above), polymorphic equilibria are possible under diploid genetics. If heterozygotes are intermediate, $K_{11} \geq K_{12} > K_{22}$, then A_1 replaces A_2 , and vice-versa for opposite signs. If there is heterosis for K, i.e. $K_{12} > K_{11}$, K_{22}, stable polymorphisms result; a reverse inequality gives unstable polymorphic equilibria (Kostitzin 1936; Kostitzin 1937; MacArthur 1962). Purely competitive equilibria are still not possible when $\alpha_{ij} = 1 \; \forall \; i,j$, as in MacArthur's analysis (see special case 3 for haploids).

MacArthur went on to argue that K_i "substituted for fitness" in a density regulated population (MacArthur 1962). He suggested that equilibrium density K would be maximized in evolution, unless populations were frequently perturbed so that $N << K$, in which case selection on r should prevail (MacArthur and Wilson 1967). Thus arose an argument for trade-offs between r vs K selection, even though strictly only K seemed important in MacArthur's formulation. "In an environment with no crowding (r selection) genotypes which harvest the most food (even if wastefully) will rear the largest families and be most fit. Evolution here favours *productivity*. At the other extreme, in a crowded area, (K selection), genotypes which can at least replace themselves with a small family at the lowest food level will win, the food density being lowered so that large families cannot be fed. Evolution here favours *efficiency* of conversion of food into offspring ..." (ref. (MacArthur and Wilson 1967): 149).

However fitness is more complicated than implied by MacArthur. Even in haploid *c* selection (special case 2), relative fitness involves *r* and overall population density as well as *K*; in rigid *K* terms it is given by $rN(K_1 - K_2)/K_1K_2$ (equation 14b; Crow and Kimura 1970: 29); allowing *r* to vary as well makes relative fitness more complicated: $(r_1 - r_2) - N(r_1/K_1 - r_2/K_2)$ (equation 15). Given Kostitzin's elastic interpretation of $K = r/c$, maximization of *K* argues for maximization of intrinsic rates of increase, *r*, as well as minimization of crowding effects, *c*, since fitness differences are affected by both (Kostitzin 1936; Kostitzin 1939; Christiansen 2004). Any mortality or fertility differences among genotypes will affect birth or death functions $B_i(n_i, n_j)$ and $D_i(n_i, n_j)$ (equation 7), and therefore values of *r* or *c*. In short, *r* selection in the sense employed here (as in haploid special case 1) is bound to be involved in MacArthurian *K* selection (special case 3). This *r* selection will be equally important however near to or far from equilibrium the population density finds itself. As seems reasonable, under the elastic *K* formulation it is always beneficial to have higher birth and lower death rates, and therefore higher intrinsic growth rate *r*, even when crowded.

Pure *c* selection (with constant *r*), on the other hand, will be more efficient in direct proportion to *N*; it is strongly density-dependent. These types of selection are frequency-independent and hard, because p_1 does not appear anywhere within expressions for relative fitness (see haploid equations 13-15), and because selection alters the value of *K*.

Frequency-dependent selection, α selection, and the general importance of competitive models of selection

As we have seen, where crowding is effected by all genotypes identically ($\alpha_{ij} = 1 \; \forall \; i,j$), equilibrium density *K* increases monotonically under selection (León and Charlesworth 1978), as in haploids under *r* and *c* selection or classic *K*

selection. Interior equilibria occur only if heterozygotes are fitter or less fit than both homozygotes.

After relaxing this assumption, selection on genotype *i* can also be expected to increase crowding pressure on other genotypes (α_{ji}), and reduce crowding by others (α_{ij}), as well as increasing *i*'s own equilibrium density by maximizing $K_i = r_i/c_i$. Thus, when interaction coefficients $\alpha_{ij} \neq 1$, equilibrium densities K_i can remain constant, or be driven down by α selection (as in Fig. 4). This mode of selection is important in the history of life: large, less fecund, complex organisms have clearly sometimes evolved from small, highly fecund, simple organisms. Optimization principles found by MacArthur (MacArthur 1962) no longer apply, and a balance between maximizing $K = r/c$ and optimizing competition is expected (Anderson and Arnold 1983; Asmussen 1983b). As we have seen, this is true for haploids as well as for diploids.

For diploids, additional stable and unstable equilibria become possible (Smouse 1976; Asmussen 1983b). Equilibria representing positive or negative heterosis are added to the Lotka-Volterra competitive equilibria of conditions 1 and 4 (Table 1). Even during replacement evolution, the response on a logit gene frequency scale will be curvilinear and three-phased rather than two-phased as in haploid evolution: first the heterozygotes, and then the new homozygotes will successively replace the original homozygotes (Fig. 7). Similar complex behaviours are found in another elastic *K* type model (Poulsen 1979). Special cases of the general model for natural selection in diploids occur where $K = r/c$ is invariant among genotypes (as in haploid equations 16 & 17; Figs. 5, 6) and near population dynamic equilibrium. These can lead to purely frequency-dependent selection which does not alter population density, i.e. approximately soft frequency-dependent selection. Thus, frequency-dependent selection can emerge from simple demographic considerations involving interactions among alleles or genotypes.

Having said this, most small-scale evolution will involve only small fitness differences, mean fitnesses may often be similar, and selection will often produce evolution that reduces competition (i.e. towards $\alpha_{ij} < 1$). Therefore, it can be argued that population size and/or biomass will not usually change rapidly, and should generally increase under selection (León and Charlesworth 1978). Even though selection is probably never completely soft, or frequency- and density-independent, one will sometimes be able to assume it is so as an approximation.

However, one should not assume selection is soft when investigating problems such as the cost of natural selection, defined as the effect of selection on population numbers (Haldane 1957; Crow and Kimura 1970). Natural selection works through parameters that affect population growth and density and so will generally be hard. This leads to evolution by natural selection having a potential 'speed limit' (Haldane 1957; Kimura 1968; Barton and Partridge 2000; Barton et al. 2007). The costs of selection must be borne by the population as an allele heads to fixation (the substitution load) or segregates in polymorphic populations (segregational and mutational loads). Genetic load may cause severe problems in populations or species of small size where stochastic processes are important, or in those with low intrinsic rates of increase. Such populations may not be able to expunge deleterious mutations as rapidly as they arise by mutation and drift, causing a 'mutational meltdown' (Lynch et al. 1995). This could be a major problem for endangered species of complex organisms such as larger vertebrates.

More complex types of evolution often will also be affected in important ways by population density. When evolution occurs in subdivided populations and there is dispersal among them, population density matters. If a subpopulation decreases in size as a result of selection, it will emit fewer dispersers than surrounding populations, and will be more prone to genetic swamping by constant levels of immigration (Christiansen 1975; Wilson and Turelli 1986;

Kirkpatrick and Barton 1997; Bolnick and Nosil 2007; Polechová et al. 2009). Such spatial models typically require elastic *K* formulation, although they may sometimes appear not to do so (e.g. Appendix 4). Genetic swamping will occur even though, in the absence of any connection to other populations, one might often be able to understand evolution while ignoring population size. It is therefore perilous to assume that natural selection is perfectly 'soft' in such metapopulations. Even the simplest form, 'pure *r* selection' or classical density-independent selection (equation 13) will be hard – it affects population density – while conforming to the Fisher gold-standard of density-independence (Fig. 2).

Empirical importance of different modes of selection

Can these theoretical results be related to selection in nature? A thorough review is not possible here, but I make some general observations. Complications due to diploidy, such as heterosis, are ignored (equivalent to an assumption of additive fitnesses in heterozygotes).

r selection. – Although a large number of possible parameters (Table 1) might vary among genotypes, it is interesting that pure *r* selection provides a good fit to Gause's original studies of competition among *Paramecium* species (Leslie 1957). As another example, one can plausibly assume that bird attacks on melanic or non-melanic moths resting on bark of different colours might affect only density-independent death rates, *d*, and thus values of *r* only (Appendix 1). If so, then Haldane was quite justified in applying a constant selection model in his analysis of evolution of melanism in the peppered moth, *Biston betularia* (Haldane 1924). Similarly, it seems likely that death rates due to toxicants are mainly density-independent (Hendriks et al. 2005), as already mentioned. Selection for pesticide or antibiotic resistance will normally ameliorate density-independent death rates, so increasing *r*. Density-independent *r* selection will likely be a common form of selection in

nature as well: Ockham's razor suggests that *r* selection will at least provide a useful first-order approximation. Fisher's assumption of *r* selection seems vindicated, even in density regulated populations.

c selection. – This mode depends on interactions among individuals within species, loosely equivalent to MacArthur's *K* selection without effects of *r* selection. Selection to reduce crowding effects (*c* selection) is likely important in nature, but is not easily separated experimentally from *r* selection, which in elastic *K* formulations will cause increased *K* also. A possible example might be the much greater effects of crowding $c = r/K$ on rice as a host for *Nephotettix malayanus*, a leafhopper that normally feeds on grasses other than rice (Fig. 1B), in comparison to the three *Nephotettix* species that normally feed on rice (Valle et al. 1989). Adaptations to rice in these species evidently include a component that increases high density efficiency by reducing the effects of density-dependent crowding.

α *selection*. – A possible empirical example of α selection might occur within bird species limited by nesting sites. *K* is set by the environment, so that even if an allele has high *r*, its *c* parameters increase accordingly (alternatively reducing *c* reduces *r* accordingly), and no gain in equilibrium density results. However, if an allele *i* effects increased aggression (interference competition), this provides an example of an $\alpha_{12}\alpha_{21} > 1$ strategy (condition 1 in Table 1), which tends to destabilize polymorphism. The evolution of a novel warning colour pattern is another example of such destabilizing selection (Mallet and Joron 1999). Alternatively, if alleles specialize on different kinds of nest sites (exploitation competition), then $\alpha_{12}\alpha_{21} < 1$ among alleles (condition 4), an example of 'apostatic selection' which stabilizes polymorphism (Clarke 1962).

Trade-offs, and their resolution. – In nature, therefore, it is clear that all these kinds of selection can occur. Yet while elastic *K* logistic theory predicts mechanistic linkage between *r* and *K* as well as independence of *r*, *c* and α, it

says nothing about possible trade-offs among parameters. Obviously, resource-controlled trade-offs must exist or all species would evolve unlimited growth rates (Slatkin and Maynard Smith 1979; Mueller 1997). As a possible empirical example of trade-offs, when ciliates in Luckinbill's (Luckinbill 1979) experiments were placed in mixed-species culture, outcomes of competition were almost exactly the reverse of the prediction that high *K* would win: high *r* and high *K* species usually did *worse* in competition, while low *r*, low *K* species competed better (Luckinbill 1979). This suggests potential trade-offs between values of *r* and effects of interaction with other species, α. Trade-offs also seem likely between *r* and *c*, being roughly equivalent to those expected in *r* vs. *K* selection (MacArthur and Wilson 1967). In nature, resource allocation trade-offs are likely among all three groups of parameters and will control their joint evolution. However, the purely mechanistic linkage between *r* and *K* under the elastic *K* formulation probably explains experiments where *K* is often correlated to *r*, in spite of expected trade-offs between *r* and *c*.

Conclusions

Theories of the interaction of demography and evolution have rarely proved satisfying in the past. Evidence of the unclear situation is provided by extensive discussions of the involvement of population growth in evolution and of the validity or otherwise of the MacArthur & Wilson concepts of *r* and *K* selection over the last 40 years (Hairston et al. 1970; Charlesworth 1971; Roughgarden 1971; Pianka 1972; Gill 1974; Stearns 1977; Parry 1981; Boyce 1984; Mueller 1997; Joshi et al. 2001; Saccheri and Hanski 2006; Holt 2009). I argue here that resolution of the debate has been hampered by a historical accident (see also Christiansen 2004): recent modelers mostly used the paradoxical logistic formulation introduced by Gause (1934), or similar rigid *K* models. Gause's *r*-*K* formulation is very useful for fitting experimental data, but it has the curious side-effect of obfuscating the most basic kind of natural

selection, Fisherian *r* selection. Kostitzin's earlier, better justified, and mathematically more robust elastic *K* formulation, which does not cause this problem, was largely ignored. It seems astonishing that reparameterizing the logistic aids understanding in evolution, but given it is now clear that the same trick clears up paradoxes in demography (Kuno 1991; Gabriel et al. 2005; Pastor 2008), perhaps this should no longer seem so surprising.

Models analogous to Kostitzin's elastic *K* population regulation will also apply in more complex situations involving discrete generations (Asmussen 1983a; Asmussen 1983b), stochasticity, multiple loci and quantitative inheritance (Kirkpatrick and Barton 1997; Polechová et al. 2009), life-history variation (Charlesworth 1980), and spatial models of evolution (Slatkin 1973; May et al. 1975; Barton 1979; Barton 1983; Barton and Hewitt 1989; Barton and Gale 1993). Employing the simplest continuous time, haploid logistic or equivalent additive diploid models, as here, clarifies how natural selection can emerge from demography, and thereby demonstrates the fundamental unity of population ecology and evolution.

Elastic *K* models provide more natural theories of density-dependent and density-independent selection, whereby frequency-dependent selection, and hard or soft selection can emerge from microscopic justification of population modelling rather than being imposed *a priori*. Their use is essential if *r* and *c* parameters are mechanistically decoupled in nature, as suggested by my brief review of empirical evidence. Energetic trade-offs are likely, and will undoubtedly affect evolution, but it is important to understand first the mechanistic background against which these evolutionary constraints operate. Furthermore, demographically realistic models of natural selection play a key role in understanding genetic loads and Haldane's 'cost of natural selection', as well as in spatial modes of evolution. Combining ecological and evolutionary models can yield a better understanding of many persistent problems on the interface between ecology and evolution.

Acknowledgments

This project was in part triggered by my supervision of the honours project of Wei-Chung Liu (then an undergraduate at UCL) on Allee effects in 1998. As part of that project, Liu independently derived Kostitzin's justification for the logistic and Lotka-Volterra equations (Appendix 1), although neither of us had heard of Kostitzin then. After reading the current manuscript, Liu made the excellent suggestion to derive first the most general $B(n_i,n_j)$ and $D(n_i,n_j)$ form for natural selection; this is now incorporated in Appendix 2. Jitka Polechová first pointed out that the Barton-Kirkpatrick model is in fact equivalent to an elastic K model, in spite of appearances (see Appendix 4). I am also grateful for helpful discussions with Deepa Agashe, Nick Barton, Kanchon Dasmahapatra, Kevin Foster, Ben Green, Larry Lawlor, Egbert Leigh, Richard Lewontin, Andrea Nahmod, John Pastor, Gigliola Staffilani and John R.G. Turner. I thank the Radcliffe Institute of Advanced Studies, Harvard University, for fellowship support.

Appendix 1. Kostitzin's derivation of population growth models

With density regulation, crowding can affect instantaneous rates of birth and death, $B(N)$ and $D(N)$, so that $dN / Ndt = B(N) - D(N)$ (equation 1). The simplest means of achieving this is to imagine that the effect of crowding depends linearly on density:

$$B(N) = b - \beta N \qquad \text{(A1.1a)}$$

$$D(N) = d + \delta N \qquad \text{(A1.1b)}$$

Expressions A1.1 can be regarded as forming the first two terms of Taylor expansions of the complete birth and death functions. These models revert cleanly to exponential growth (equation 2) when $\beta = \delta = 0$ so that $B(N) = b$ and $D(N) = d$, giving a constant growth rate $r = b - d$. (In contrast, the classical logistic, equation 3, reverts to constant growth rate only when $K \to \infty$). When growth is density-dependent:

$$\frac{1}{N}\frac{dN}{dt} = (b-d) - (\beta + \delta)N \qquad \text{(A1.2a)}$$

or:

$$\frac{1}{N}\frac{dN}{dt} = (b-d)\left[1 - \frac{(\beta+\delta)}{(b-d)}N\right] \qquad \text{(A1.2b)}$$

Equation A1.2 can be expressed in the familiar *r-K* format (equation A1.2b) or the Verhulst *r-c* format (equation 1.2a), where $r = (b - d)$, density-dependent effects $c = (\beta + \delta)$, and equilibrium density is $K = \frac{(b-d)}{(\beta+\delta)} = \frac{r}{c}$, a ratio of the density-independent effects *r* and density-dependent effects *c*. Lotka-Volterra competition is a simple extension. Suppose birth and death rates are affected

by crowding not only by members of the same species, but also by members of other species. Overall rates of change in numbers are now affected by the densities of both species:

$$\frac{1}{N_1}\frac{dN_1}{dt} = B_1(N_1, N_2) - D_1(N_1, N_2) \quad \text{(A1.3a)}$$

$$\frac{1}{N_2}\frac{dN_2}{dt} = B_2(N_1, N_2) - D_2(N_1, N_2) \quad \text{(A1.3b)}$$

By analogy with A1.1:

$$B_1(N_1, N_2) = b_1 - \beta_{11}N_1 - \beta_{12}N_2 \quad \text{(A1.4a)}$$

$$D_1(N_1, N_2) = d_1 + \delta_{11}N_1 + \delta_{12}N_{12} \quad \text{(A1.4b)}$$

$$B_2(N_1, N_2) = b_2 - \beta_{22}N_2 - \beta_{21}N_1 \quad \text{(A1.4a)}$$

$$D_2(N_1, N_2) = d_2 + \delta_{22}N_2 + \delta_{21}N_1 \quad \text{(A1.4b)}$$

Therefore,

$$\frac{1}{N_1}\frac{dN_1}{dt} = (b_1 - d_1) - (\beta_{11} + \delta_{11})N_1 - (\beta_{12} + \delta_{12})N_2 \quad \text{(A1.5a)}$$

$$\frac{1}{N_2}\frac{dN_2}{dt} = (b_2 - d_2) - (\beta_{22} + \delta_{22})N_2 - (\beta_{21} + \delta_{21})N_1 \quad \text{(A1.5b)}$$

Equations A1.5 convert to equations 4 and 6, where $r_1 = (b_1 - d_1)$, $r_2 = (b_2 - d_2)$, $c_1 = (\beta_{11} + \delta_{11})$, $c_2 = (\beta_{22} + \delta_{22})$, $K_1 = r_1/c_1$, $K_2 = r_2/c_2$, $\alpha_{12} = (\beta_{12} + \delta_{12})/c_1$ and $\alpha_{21} = (\beta_{21} + \delta_{21})/c_2$. The α_{ij} are here parameterized relative to within-species crowding effects c_i on births and deaths to aid comparison with classic formulations used in most textbooks, and for easier comparison of interspecific versus intraspecific effects, expressed via $\alpha_{ij}\alpha_{ji} > 1$ or $\alpha_{ij}\alpha_{ji} < 1$ (Table 1).

Appendix 2. Relationship of gene frequency evolution to population growth

by James Mallet and Wei-Chung Liu

Evolution is defined as a change in the fractions of alleles, p_1, p_2, in a population of size N (where $p_1 = n_1/N$ and $p_2 = 1 - p_1 = n_2/N$). How do changes in the numbers of each allele n_1, n_2, affect the the fractions of alleles in the population over time? Here, we use methods developed earlier (Volterra 1927; Crow and Kimura 1970; Leigh 1971; Smouse 1976), but apply them to the most general case. First, note that $\frac{d \log_e (n_1 / n_2)}{dt} = \frac{d \log_e n_1}{dt} - \frac{d \log_e n_2}{dt}$.

Given that $\frac{d \log_e x}{dx} = \frac{1}{x}$, and $\frac{d[f(x)]}{dt} = \frac{d[f(x)]}{dx}\frac{dx}{dt}$, this can be rewritten as:

$$\frac{d \log_e (n_1 / n_2)}{dt} = \frac{1}{n_1}\frac{dn_1}{dt} - \frac{1}{n_2}\frac{dn_2}{dt} \qquad \text{(A2.1)}$$

Note also that:

$$\frac{d \log_e (p_1 / p_2)}{dt} = \frac{d \log_e p_1}{dt} - \frac{d \log_e (1 - p_1)}{dt}$$

$$= \frac{1}{p_1}\frac{dp_1}{dt} + \frac{1}{(1 - p_1)}\frac{dp_1}{dt} = \frac{1}{p_1(1 - p_1)}\frac{dp_1}{dt} \qquad \text{(A2.2)}$$

Since $\frac{d \log_e (p_1 / p_2)}{dt} = \frac{d \log_e (n_1 / n_2)}{dt}$, we can put A2.1 and A2.2 together:

$$\frac{1}{p_1(1 - p_1)}\frac{dp_1}{dt} = \frac{1}{n_1}\frac{dn_1}{dt} - \frac{1}{n_2}\frac{dn_2}{dt} \qquad \text{(A2.3)}$$

Substituting in the general formulation for allelic density growth (equations 7a,b, A1.3) into A2.3 gives:

$$\frac{1}{p_1(1-p_1)}\frac{dp_1}{dt} = \left[B_1\left(n_1,n_2\right) - D_1\left(n_1,n_2\right)\right] - \left[B_2\left(n_1,n_2\right) - D_2\left(n_1,n_2\right)\right] \qquad \text{(A2.4)}$$

For logistic demography, we substitute appropriate analogues of Lotka-Volterra competition among alleles (equation A1.4) into equation A2.4:

$$\frac{1}{p_1(1-p_1)}\frac{dp_1}{dt} = \left[r_1 - c_1(n_1 + \alpha_{12}n_2)\right] - \left[r_2 - c_2(n_2 + \alpha_{21}n_1)\right] \qquad \text{(A2.5a)}$$

Or, expressed in rigid *K* format:

$$\frac{1}{p_1(1-p_1)}\frac{dp_1}{dt} = \frac{r_1}{K_1}(K_1 - n_1 - \alpha_{12}n_2) - \frac{r_2}{K_2}(K_2 - n_2 - \alpha_{21}n_1) \qquad \text{(A2.5b)}$$

Setting $p_1 = n_1/N$ in A2.5a, where $N = n_1 + n_2$:

$$\frac{1}{p_1(1-p_1)}\frac{dp_1}{dt} = r_1 - r_2 - N\left[\left(c_1 - \alpha_{21}c_2\right)p_1 - \left(c_2 - \alpha_{12}c_1\right)(1-p_1)\right] \qquad \text{(A2.6a)}$$

Or in rigid *K* format:

$$\frac{1}{p_1(1-p_1)}\frac{dp_1}{dt} = r_1 - r_2 - N\left[\left(\frac{r_1}{K_1} - \alpha_{21}\frac{r_2}{K_2}\right)p_1 - \left(\frac{r_2}{K_2} - \alpha_{12}\frac{r_1}{K_1}\right)(1-p_1)\right] \qquad \text{(A2.6b)}$$

Appendix 3. Equivalence of Fisherian and Wrightian fitness

Discrete per-generation 'Wrightian' fitness (e.g. for allele 1, the fitness W_1= 1+S; for allele 2, W_2= 1), where S is a selection coefficient, is perhaps more often used than Fisherian infinitesimal relative fitness, $S = (R_1 - R_2)$, in

population genetics (equation 8). Here, absolute Wrightian fitnesses measured over a single time unit (generation) are $W_1 = e^{R_1}$ and $W_2 = e^{R_2}$. Wrightian relative fitness, which determines the rate of evolution, will be

$$W_1 / W_2 = e^{R_1 - R_2} = e^S \approx 1 + S \qquad \text{(A3.1)}$$

provided S is small. Wrightian and Fisherian measures of selection S are thus equivalent when per generation selection is weak. This is true in the simple situation where S is a constant, but applies also when S depends on allelic densities n_1 and n_2.

Appendix 4. The Kirkpatrick and Barton model of range size evolution

The Kirkpatrick-Barton quantitative genetic theory of geographic range evolution (Kirkpatrick and Barton 1997) seems, in one formulation, to employ rigid K population regulation. Here I show that the model does in fact have an elastic density equilibrium. Ignoring influence from other sites, density at a single site changes as follows (their equation 7):

$$\frac{1}{N}\frac{dN}{dt} = \bar{R} = \bar{R}_e + \bar{R}_g \qquad \text{(A4.1)}$$

The means are taken across multilocus genotypes. Population regulation is effected by an ecological component, e, determined independently of genotype, by:

$$\bar{R}_e = r_{\max}\left(1 - \frac{N}{K'}\right) \qquad \text{(A4.2)}$$

where r_{max} and K' are spatially and genetically invariant and at first sight seem to have the usual meanings of 'intrinsic maximal rate of increase' and

'carrying capacity' (Kirkpatrick & Barton 1997: pp. 7-8). The genetic component, *g*, of population growth, meanwhile, is given by:

$$\bar{R}_g = -\frac{\left(\theta(x) - \bar{z}\right)^2}{2V_S} - \frac{V_P}{2V_S} \qquad \text{(A4.3)}$$

Equation A4.3 contains only density-independent terms, but is variable according to geographical location and genotypic constitution of the population. The *V* parameters represent phenotypic variance (*P*) and the strength of stabilizing selection (*S*) around the optimum, while the term $\left(\theta(x) - \bar{z}\right)^2$ measures the deviation of the quantitative phenotype $\bar{z}$ from the optimum $\theta(x)$ at a particular spatial location *x*.

When $N << K'$ in equation A4.1,

$$\bar{r}_0 = r_{\max} + \bar{R}_g \qquad \text{(A4.4)}$$

Therefore it is clear that the actual local intrinsic rate of increase, $\bar{r}_0$, involved in population regulation depends on the average genotypic constitution of the population as well as the constant term within equation A4.2. The equilibrium population size $\hat{N}$ is obtained when $\bar{R} = 0$:

$$\hat{N} = \frac{\bar{r}_0}{c} \qquad \text{(A4.5)}$$

where $c = r_{\max} / K'$ is a constant. It is evident that the equilibrium population size is not the same as K'; it depends both on a density-independent intrinsic rate of increase ($\bar{r}_0$, A4.4), which is locally and temporarily a constant although spatially and genetically variable, and a crowding coefficient, *c*, as in equation 5, that is the same everywhere. Thus there is complete separation of

density-independent, $\bar{r}_0$, and density-dependent, c, parameters; population regulation is exactly equivalent to the elastic K model of equation 5, and the model is a multilocus form of r selection in regulated populations.

Table 1. Conditions for coexistence and exclusion in two-species Lotka-Volterra models of competition (equation 4)

No.	Condition*	... implies+:	Interior equilibria
1	$1/\alpha_{21} < K_1/K_2 < \alpha_{12}$	$\alpha_{12}\alpha_{21} > 1$	one, unstable
2	$1/\alpha_{21} < K_1/K_2 > \alpha_{12}$		none, 1 replaces 2
3	$1/\alpha_{21} > K_1/K_2 < \alpha_{12}$		none, 2 replaces 1
4	$1/\alpha_{21} > K_1/K_2 > \alpha_{12}$	$\alpha_{12}\alpha_{21} < 1$	one, stable

* These conditions are expressed in classical rigid K format. The equivalent elastic K conditions for equation 6 are readily found via the conversion $K_i = r_i/c_i$.

+ If $\alpha_{ij}\alpha_{ji} < 1$ (condition 1), two species can partition the environment, giving exploitation competition that allows stable coexistence; if $\alpha_{ij}\alpha_{ji} > 1$ (condition 4), species show interference competition, with an unstable equilibrium that prevents coexistence.

Table 2. Types of haploid selection emerging from the Lotka-Volterra model

Model type	$S(...)^*$	Parameter-dependence of S	Density-…	Frequency-…	Hard vs. soft$^+$ selection	Possibility of interior equilibria
r selection	-	r_1, r_2	independent	independent	hard	-
c selection	N	c_1, c_2	dependent	independent	hard	-
K selection§	N	r_1, r_2, c_1, c_2	dependent	independent	hard	-
pure α selection¶	(N), p_1	c, α_{12}, α_{21}	effectively independent	dependent	~ soft	+
α selection & general	N, p_1	r_1, r_2, c_1, c_2, α_{12}, α_{21}	dependent	dependent	hard	+

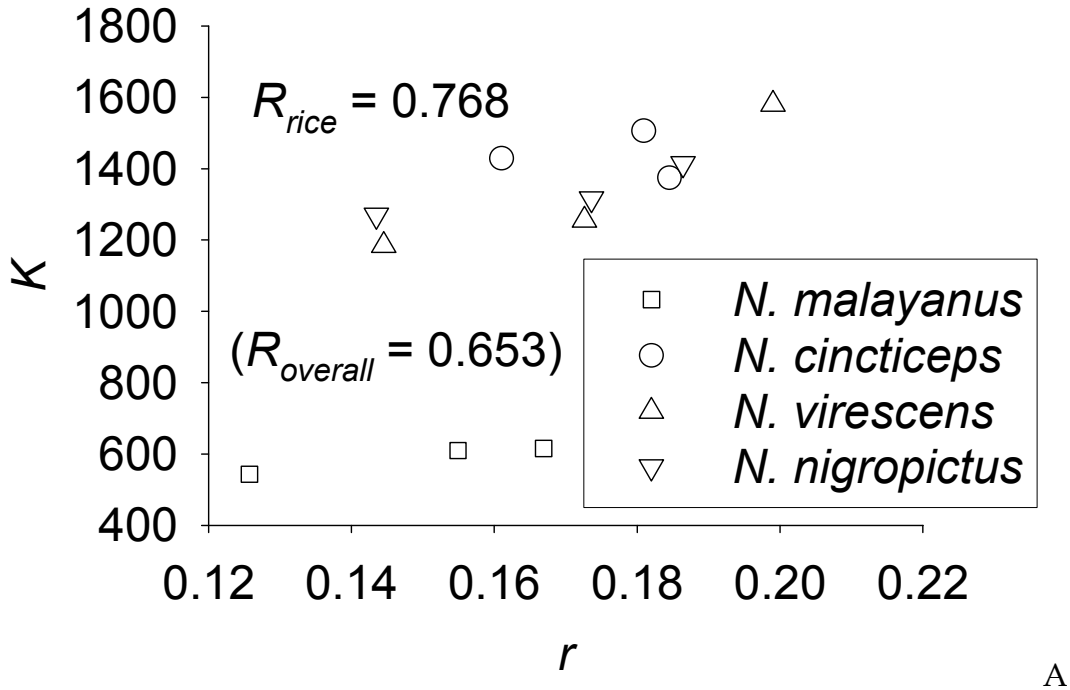
Notes

* variables of which the Fisherian selection term, S, is a function

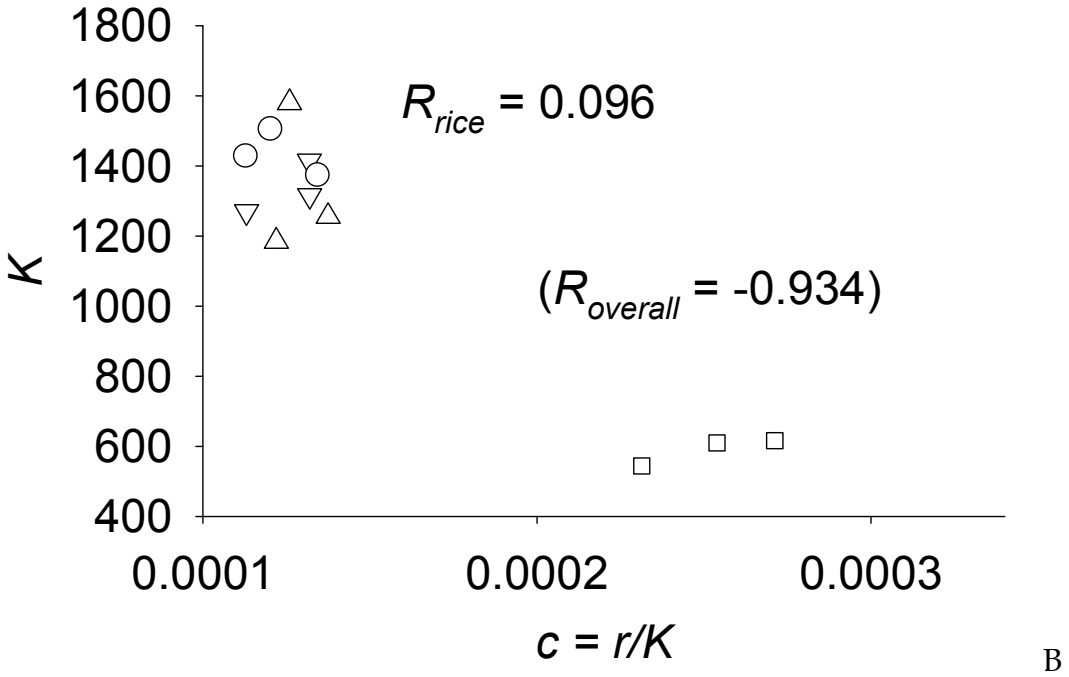
\+ *sensu* Christiansen (1975)

§ *sensu* MacArthur and Wilson (1967)

¶ general model, except $K_1 = K_2$ and $r_1 = r_2$, and assuming replacement while $N \approx K$ during evolution

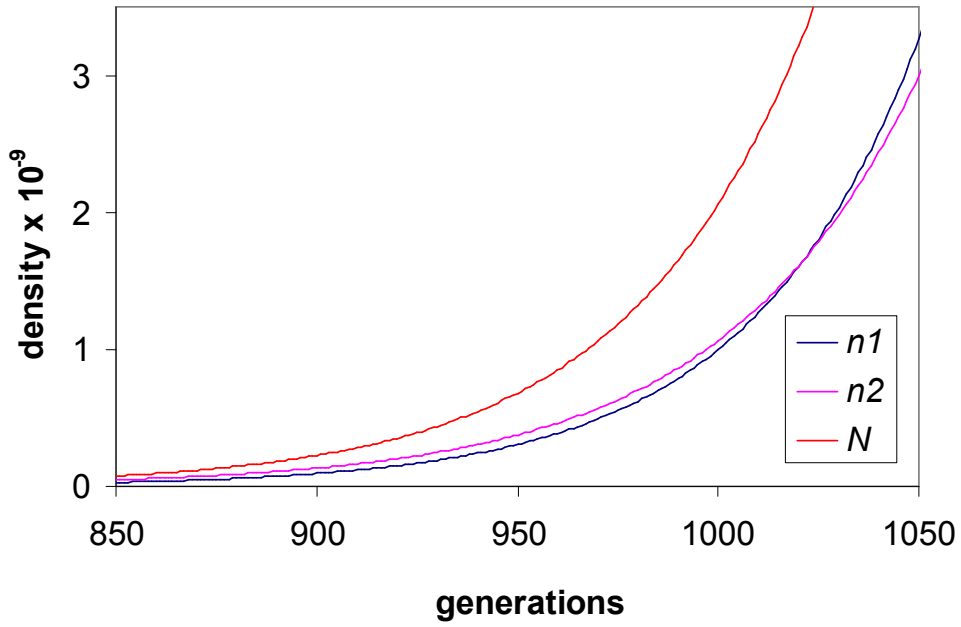


A

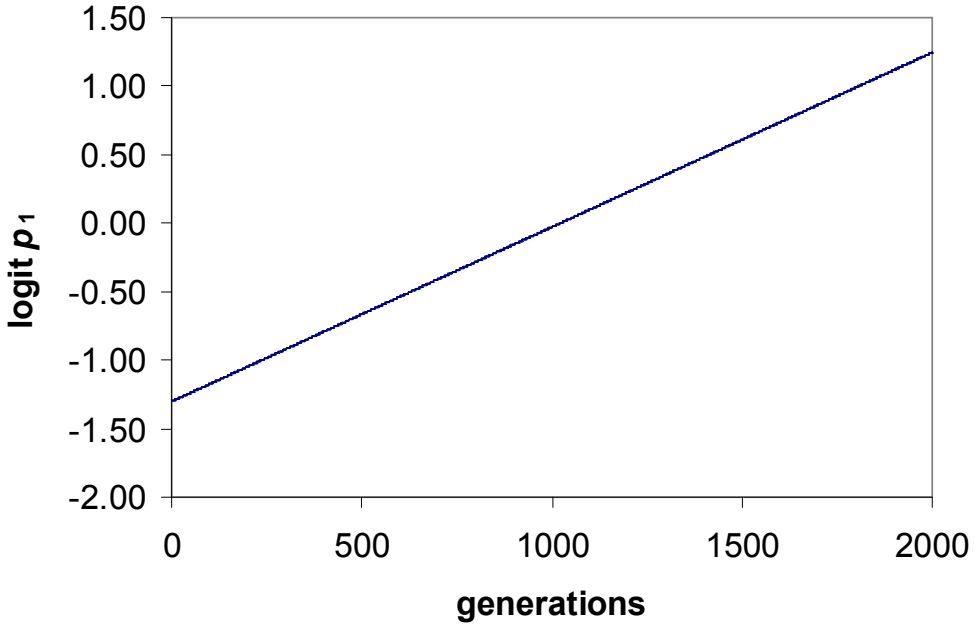


B

Fig. 1. Fitted logistic parameters for *Nephotettix* leafhoppers on rice at three different temperatures. A. Dependence of *K* on *r*. B. Dependence of *K* on *c*. Product-moment correlation coefficients, *R*, are shown for the rice species (i.e. excluding *N. malayanus*, which is not normally on rice) and overall (Valle et al. 1989).

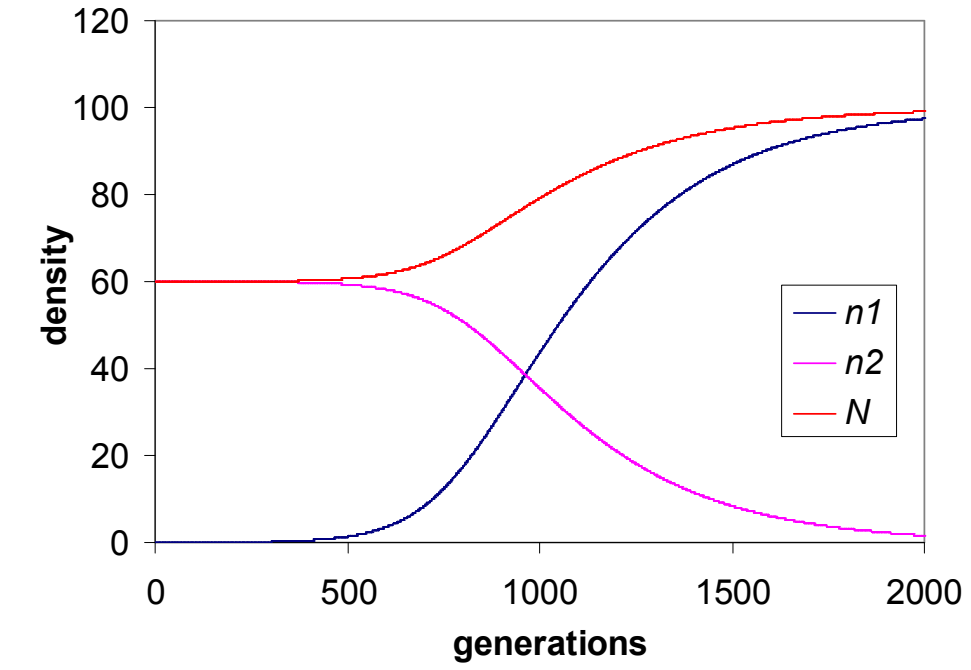


A

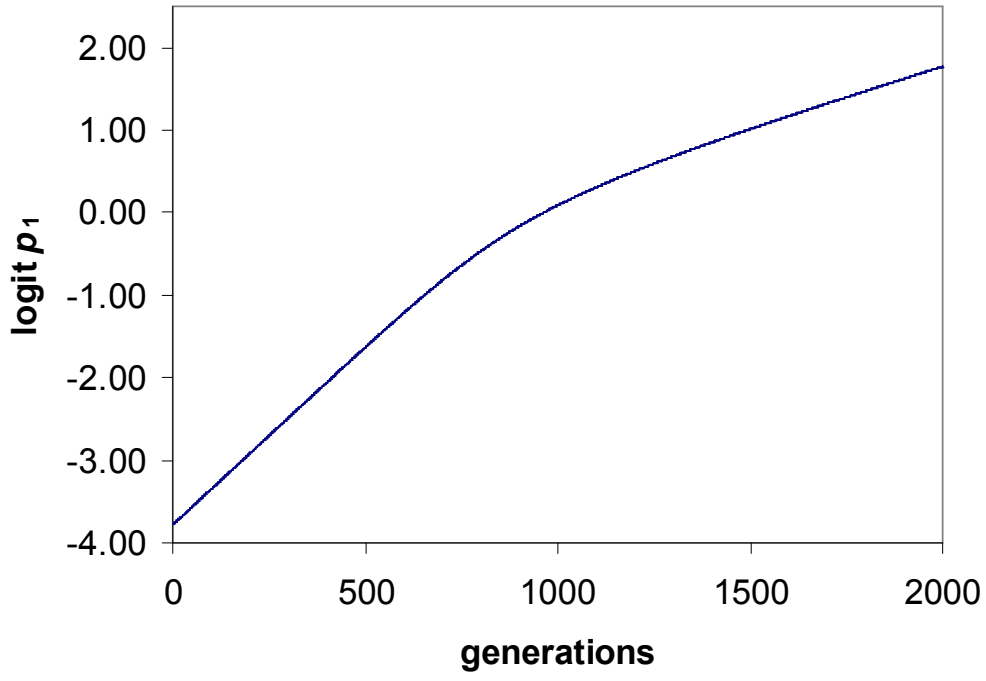


B

Fig. 2. Replacement of one allele by another under exponential growth (equations 9,10). Here $s = r_1 - r_2 = 0.024 - 0.021 = 0.003$. Allele 1 is initially at low density ($n_1 = 0.05$, $n_2 = 1.0$, when $t = 0$ generations), but eventually replaces allele 2. (A). A segment of the density trajectory, showing exponential population growth of both alleles where allele 1 begins to overtake allele 2. (B). The replacement of alleles on a logit scale, $\log_{10}[p_1/(1-p_1)]$, over a long time course. The constant slope is $s / \log_e(10) \approx 0.0013$, as expected from equation 10.

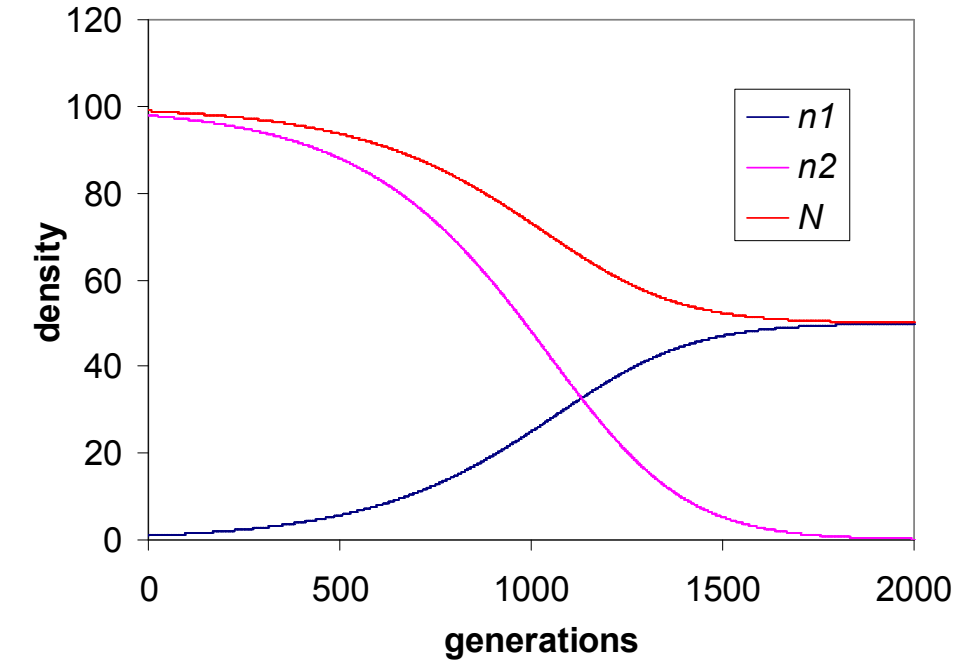


A

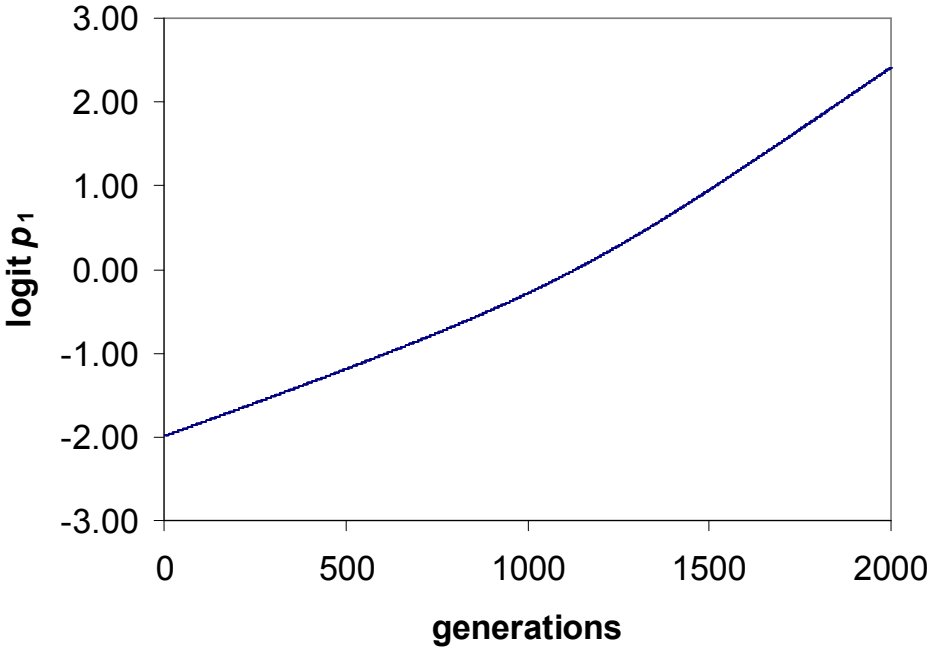


B

Fig. 3. Natural selection under weak generalized Lotka-Volterra competition (equations 11, 12), showing increase in equilibrium population size. Parameters: $r_1 = 0.10$, $K_1 = 100$, $r_2 = 0.03$, $K_2 = 60$, and $\alpha_{12} = 1/\alpha_{21} = 1.5$. As in Figs. 4-6, inversely related α are here used to give parallel isoclines, ensuring directional selection. A. Allelic density. B. Replacement of allele frequency on a logit scale, $\log_{10}[p_1/(1-p_1)]$, over time. The initial slope is $[r_1(K_1 - \alpha_{12}K_2)/K_1]/\log_e 10 \approx 0.0043$, and the final slope is $[r_2(K_2 - \alpha_{21}K_1)/K_2]/\log_e 10 \approx 0.0014$ as expected (see equations 18).

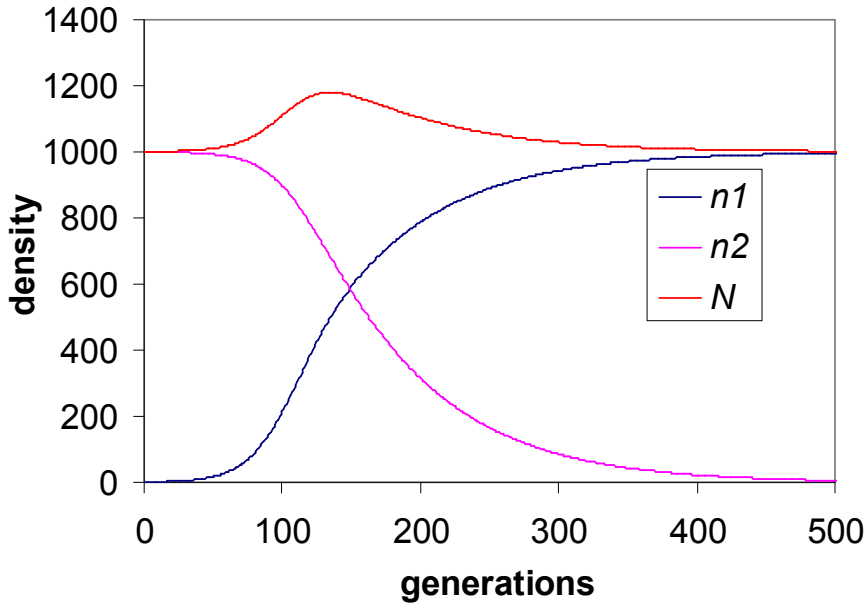


A

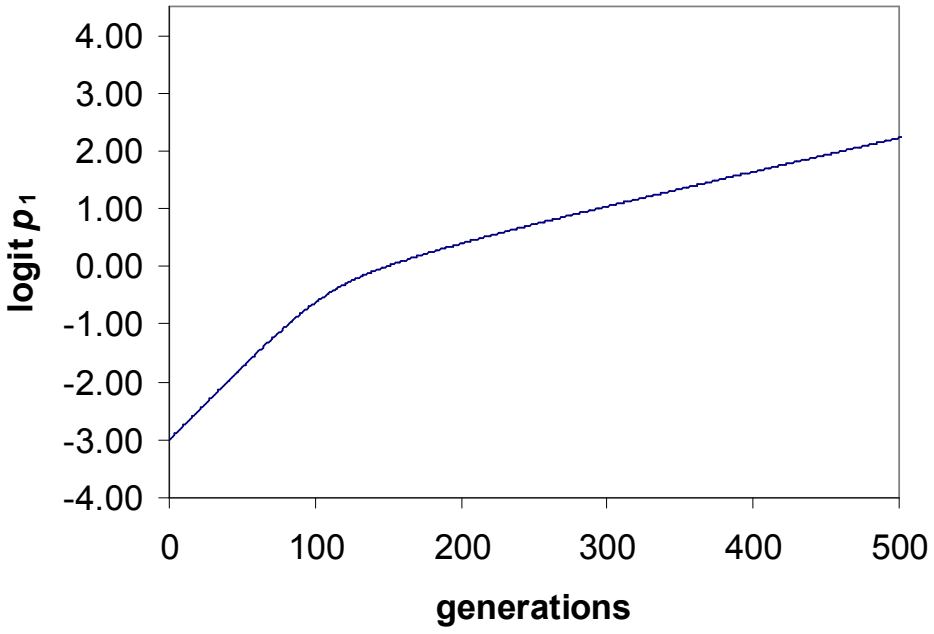


B

Fig. 4. Natural selection under weak generalized Lotka-Volterra competition (equations 11, 12), showing reduction in equilibrium population size during allelic replacement. Parameters: $r_1 = 0.03$, $K_1 = 50$, $r_2 = 0.05$, $K_2 = 50$, and $\alpha_{12} = 1/\alpha_{21} = 0.44$. A. Allelic density. B. Replacement of allele frequency on a logit scale, $\log_{10}[p_1/(1-p_1)]$, over time.

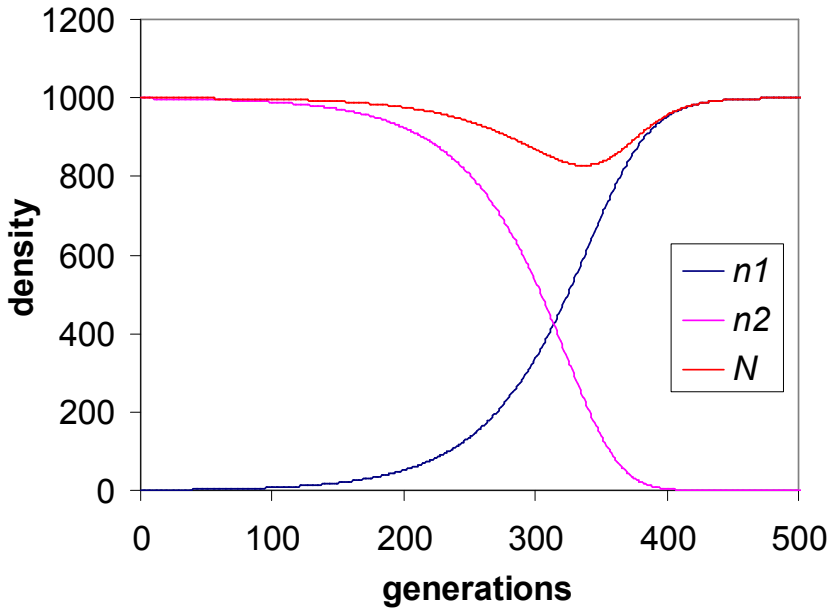


A

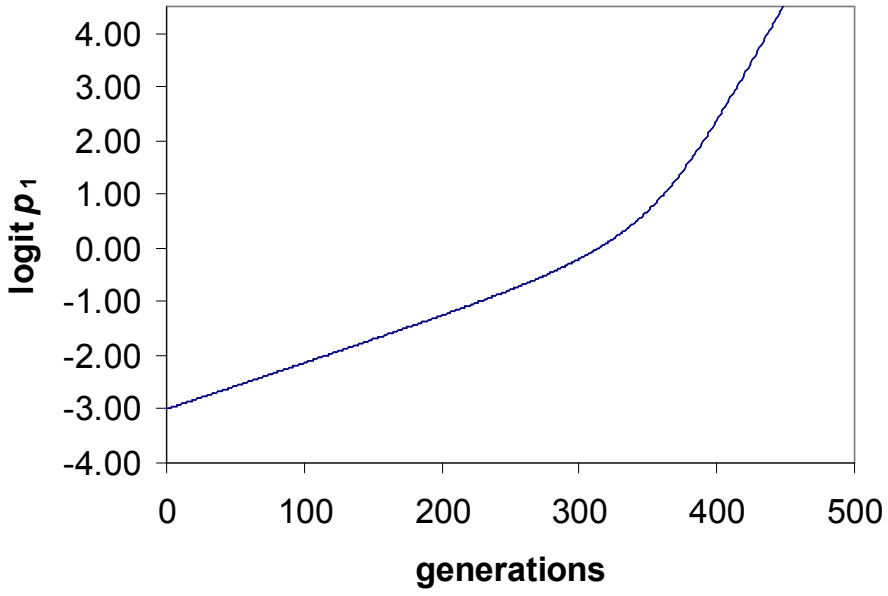


B

Fig. 5. Natural selection under strong generalized Lotka-Volterra competition (equations 11, 12), showing pure α selection with no difference in K among alleles, but a temporary rise in population size during replacement. Parameters: $r_1 = 0.15$, $K_1 = 1000$, $r_2 = 0.02$, $K_2 = 1000$, and $\alpha_{12} = 1/\alpha_{21} = 0.6$. A. Allelic density. B. Replacement of allele frequency on a logit scale, $\log_{10}[p_1/(1-p_1)]$, over time.

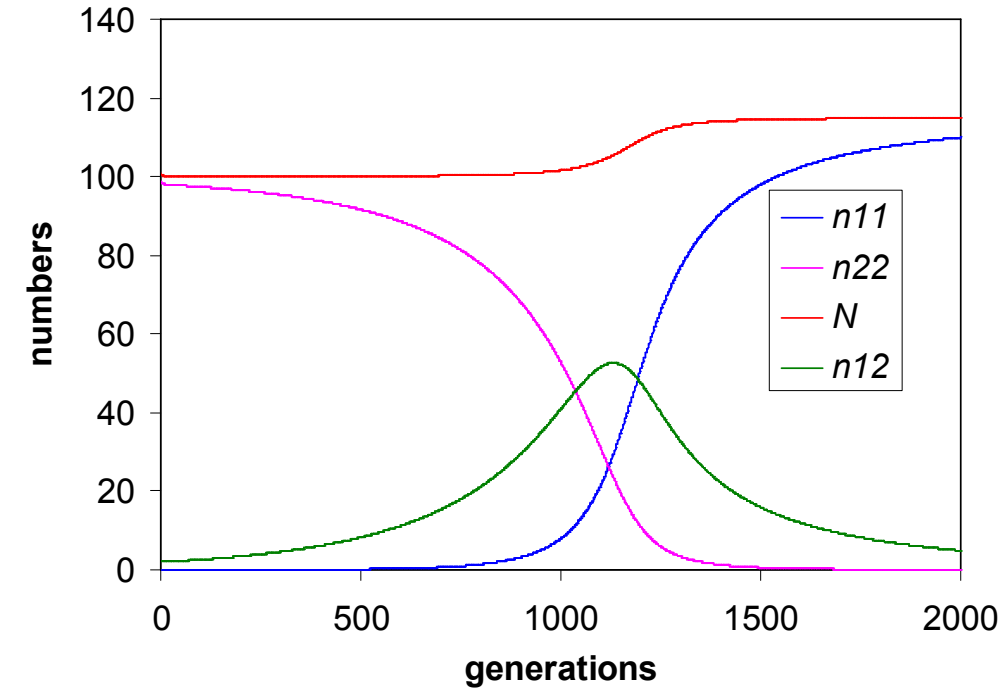


A

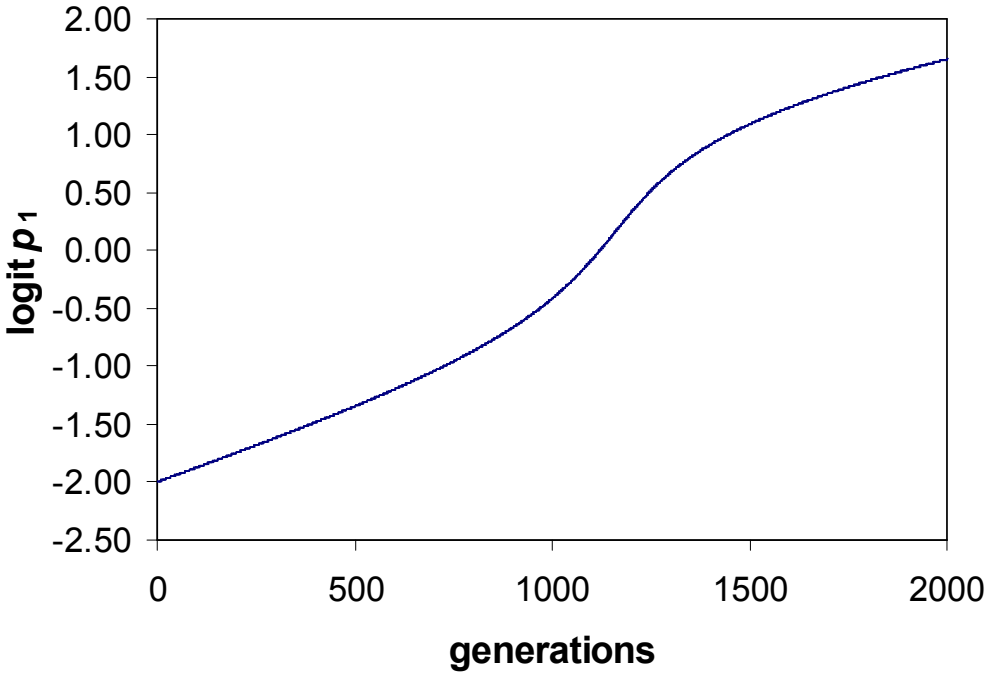


B

Fig. 6. Natural selection under strong generalized Lotka-Volterra competition (equations 11, 12), showing pure α selection with no difference in K among alleles, but a temporary dip in population size during replacement. Parameters: $r_1 = 0.05$, $K_1 = 1000$, $r_2 = 0.15$, $K_2 = 1000$, and $\alpha_{12} = 1/\alpha_{21} = 0.6$. A. Allelic density. B. Replacement of allele frequency on a logit scale, $\log_{10}[p_1/(1-p_1)]$, over time.



A



B

Fig. 7. Replacement of one diploid allele by another under strong generalized density-dependent selection, showing three-phased evolution of genotypes 11, 12, and 22. A. Genotypic densities. B. Allelic fraction. Here r_{11} = 2.00, r_{12} = 0.10, r_{22} = 0.03, K_{11} = 100, K_{12} = 110, K_{22} = 115, and ($\alpha_{ij,kl}$ = 1 $\forall$ i,j,k,l).

Literature Cited

Anderson, W. W. 1971. Genetic equilibrium and population growth under density-dependent selection. American Naturalist 105:489-499.

Anderson, W. W. and J. Arnold. 1983. Density-regulated selection with genotypic interactions. American Naturalist 121:649-655.

Asmussen, M. A. 1979. Density-dependent selection II. The Allee effect. The American Naturalist 114:796-809.

Asmussen, M. A. 1983a. Density dependent selection incorporating intraspecific competition. I. A haploid model. Journal of Theoretical Biology 101:113-127.

Asmussen, M. A. 1983b. Density-dependent selection incorporating intraspecific competition. II. A diploid model. Genetics 103:335-350.

Asmussen, M. A. and M. W. Feldman. 1977. Density dependent selection I: a stable feasible equilibrium may not be attainable. Journal of Theoretical Biology 64:603-618.

Ayala, F. J., M. E. Gilpin, and J. G. Ehrenfeld. 1973. Competition between species: theoretical models and experimental tests. Theoretical Population Biology 4:331-356.

Barton, N. H. 1979. The dynamics of hybrid zones. Heredity 43:341-359.

Barton, N. H. 1983. Multilocus clines. Evolution 37:454-471.

Barton, N. H., D. E. G. Briggs, J. A. Eisen, D. B. Goldstein, and N. H. Patel. 2007. Evolution. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, New York.

Barton, N. H. and K. S. Gale. 1993. Genetic analysis of hybrid zones. Pages 13-45 *in* R. G. Harrison, ed. Hybrid Zones and the Evolutionary Process. Oxford University Press, New York.

Barton, N. H. and G. M. Hewitt. 1989. Adaptation, speciation and hybrid zones. Nature (London) 341:497-503.

Barton, N. H. and L. Partridge. 2000. Limits to natural selection. BioEssays 22:1075-1084.

Begon, M., J. L. Harper, and C. R. Townsend. 1996. Ecology. Individuals, Populations and Communities. Blackwell Science, Oxford.

Bell, G. 1997. The Basics of Selection. Chapman & Hall, New York.

Bolnick, D. I. and P. Nosil. 2007. Natural selection in populations subject to a migration load. Evolution 61:2229-2243.

Boyce, M. S. 1984. Restitution of *r*- and *K*-selection as a model of density-dependent natural selection. Annual Review of Ecology and Systematics 15:427-447.

Charlesworth, B. 1971. Selection in density-regulated populations. Ecology 52:469-474.

Charlesworth, B. 1980. Evolution in Age-Structured Populations. Cambridge Univ. Press.

Christiansen, F. B. 1975. Hard and soft selection in a subdivided population. American Naturalist 109:11-16.

Christiansen, F. B. 2004. Density-dependent selection. Pages 139-157 *in* R. S. Singh and M. K. Uyenoyama, eds. The Evolution of Population Biology. Cambridge University Press, Cambridge.

Clarke, B. 1962. Balanced polymorphism and the diversity of sympatric species. Pages 47-70 *in* D. Nichols, ed. Taxonomy and Geography. Systematics Association, Oxford.

Crow, J. F. and M. Kimura. 1970. An Introduction to Population Genetics Theory. Burgess Publishing Co, Minneapolis, Minn.

Desharnais, R. A. and L. M. Constantino. 1983. Natural selection and density-dependent population growth. Genetics 105:1029-1040.

Fisher, R. A. 1918. The correlation between relatives on the supposition of Mendelian inheritance. Transactions of the Royal Society of Edinburgh 52:399-433.

Fisher, R. A. 1922. On the dominance ratio. Proceedings of the Royal Society of Edinburgh 42:321-341.

Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford.

Gabriel, J.-P., F. Saucy, and L.-F. Bersier. 2005. Paradoxes in the logistic equation? Ecological Modelling 185:147-151.

Gause, G. F. 1934. The Struggle for Existence. Williams and Wilkins Co., Baltimore, Maryland.

Gause, G. F. and A. A. Witt. 1935. Behavior of mixed populations and the problem of natural selection. The American Naturalist 69:596-609.

Gill, D. E. 1974. Intrinsic rates of increase, saturation densities, and competitive ability. I. The evolution of competitive ability. American Naturalist 108:103-116.

Ginzburg, L. R. 1992. Evolutionary consequences of basic growth equations. Trends in Ecology & Evolution 7:133.

Gotelli, N. J. 2008. A Primer of Ecology. Sinauer Associates, Sunderland, Mass.

Hairston, N. G., D. W. Tinkle, and H. M. Wilbur. 1970. Natural selection and the parameters of population growth. Journal of Wildlife Management 34:681-690.

Haldane, J. B. S. 1924. A mathematical theory of natural and artificial selection. Transactions of the Cambridge Philosophical Society 23:19-40.

Haldane, J. B. S. 1957. The cost of natural selection. Journal of Genetics 55:511-524.

Hanski, I. and M. Gilpin. 1991. Metapopulation dynamics: brief history and conceptual domain. Biological Journal of the Linnean Society 42:3-15.

Hendriks, A. J., J. L. M. Maas-Diepeveen, E. H. W. Heugens, and N. M. van Straalen. 2005. Meta-analysis of intrinsic rates of increase and carrying capacity of populations affected by toxic and other stressors. Environmental Toxicology and Chemistry 24:2267-2277.

Holt, R. D. 2009. Bringing the Hutchinsonian niche into the 21st century: ecological and evolutionary perspectives. Proceedings of the National Academy of Sciences, USA 106:19659-19665.

Hutchinson, G. E. 1978. An Introduction to Population Ecology. Yale University Press, New Haven, Conn.

Joshi, A., N. G. Prasad, and M. Shakarad. 2001. *K*-selection, α-selection, effectiveness, and tolerance in competition: density-dependent selection revisited. Journal of Genetics 80:63-75.

Kimura, M. 1968. Evolutionary rate at the molecular level. Nature (London) 217:624-626.

Kimura, M. 1978. Change of gene frequencies by natural selection under population number regulation. Proceedings of the National Academy of Sciences, USA 75:1934-1937.

Kirkpatrick, M. and N. Barton. 1997. Evolution of a species' range. American Naturalist 150:1-23.

Kostitzin, V. A. 1936. Sur les équations différentielles du problème de la sélection mendelienne. Comptes Rendus de l'Académie des Sciences, Paris 203:156-157.

Kostitzin, V. A. 1937. Biologie Mathématique. Armand Colin, Paris.

Kostitzin, V. A. 1939. Mathematical Biology. G.G. Harrapp & Co., London.

Kostitzin, V. A. 1940. Sur la loi logistique et ses généralisations. Acta Biotheoretica 5:155-159.

Kuno, E. 1991. Some strange properties of the logistic equation defined with *r* and *K*: inherent defects or artifacts? Researches on Population Ecology 33:33-39.

Leigh, E. G. 1971. Adaptation and Diversity. Natural History and the Mathematics of Evolution. Freeman, San Francisco.

León, J. A. and B. Charlesworth. 1978. Ecological versions of Fisher's fundamental theorem of natural selection. Ecology 59:457-464.

Leslie, P. H. 1957. An analysis of the data for some experiments carried out by Gause with populations of the protozoa, *Paramecium aurelia* and *Paramecium caudatum*. Biometrika 44:314-327.

Lewontin, R. C. 2004. Building a science of population biology. Pages 7-20 *in* R. S. Singh and M. K. Uyenoyama, eds. The Evolution of Population Biology. Cambridge University Press, Cambridge.

Lotka, A. J. 1925. Elements of Physical Biology. Williams & Wilkins, Baltimore, Maryland.

Lotka, A. J. 1932. The growth of mixed populations: two species competing for a common food supply. Journal of the Washington Academy of Sciences 21:461-469.

Luckinbill, L. S. 1979. Selection and the *r*/*K* continuum in experimental populations of protozoa. American Naturalist 113:427-437.

Lynch, M., J. Conery, and R. Bürger. 1995. Mutation accumulation and the extinction of small populations. American Naturalist 146:489-518.

MacArthur, R. H. 1962. Some generalized theorems of natural selection. Proceedings of the National Academy of Sciences, USA 48:1893-1897.

MacArthur, R. H. and E. O. Wilson. 1967. The Theory of Island Biogeography. Princeton University Press, Princeton, New Jersey.

Mallet, J. and M. Joron. 1999. The evolution of diversity in warning colour and mimicry: polymorphisms, shifting balance, and speciation. Annual Review of Ecology and Systematics 30:201-233.

Malthus, T. R. 1826. An Essay on the Principle of Population. Or a View of its Past and Present Effects on Human Happiness; with an Inquiry into our Prospects Respecting the Future Removal or Mitigation of the Evils which it Contains. John Murray, London.

May, R. M., J. A. Endler, and R. E. McMurtrie. 1975. Gene frequency clines in the presence of selection opposed by gene flow. American Naturalist 109:650-676.

Maynard Smith, J. 1974. Models in Ecology. Cambridge University Press, Cambridge.

Maynard Smith, J. 1989. Evolutionary Genetics. Oxford University Press, Oxford.

Mueller, L. D. 1997. Theoretical and empirical examination of density-dependent selection. Annual Review of Ecology and Systematics 28:269-288.

Mueller, L. D. and F. J. Ayala. 1981. Trade-off between *r*-selection and *K*-selection in *Drosophila* populations. Proceedings of the National Academy of Sciences, USA 78:1303-1305.

Parry, G. D. 1981. The meanings of *r*- and *K*-selection. Oecologia 48:260-264.

Pastor, J. 2008. Mathematical Ecology of Populations and Ecosystems. Wiley-Blackwell, Chichester, UK.

Pianka, E. R. 1972. *r* and *K* selection or *b* and *d* selection? American Naturalist 106:581-588.

Polechová, J., N. Barton, and G. Marion. 2009. Species' range: adaptation in space and time. American Naturalist 174:E186-E204.

Poulsen, E. T. 1979. A model for population regulation with density- and frequency-dependent selection. Journal of Mathematical Biology 8:325-343.

Prout, T. 1980. Some relationships between density-independent selection and density-dependent population growth. Evolutionary Biology 13:1-68.

Roughgarden, J. 1971. Density-dependent natural selection. Ecology 52:453-468.

Roughgarden, J. 1979. Theory of Population Genetics and Evolutionary Ecology: an Introduction. Macmillan, New York.

Saccheri, I. and I. Hanski. 2006. Natural selection and population dynamics. Trends in Ecology & Evolution 21:341-347.

Scudo, F. M. and J. R. Ziegler. 1978. The Golden Age of Theoretical Ecology: 1923-1940. A collection of works by V. Volterra, V.A. Kostitzin, A.J. Lotka, and A.N. Kolmogoroff. Springer-Verlag, Berlin.

Slatkin, M. 1973. Gene flow and selection in a cline. Genetics 75:733-756.

Slatkin, M. 1979. The evolutionary response to frequency- and density-dependent interactions. American Naturalist 114:384-398.

Slatkin, M. and J. Maynard Smith. 1979. Models of coevolution. Quarterly Review of Biology 54:233-263.

Slobodkin, L. B. 1960. Ecological energy relationships at the population level. American Naturalist 94:213-236.

Smouse, P. E. 1976. The implications of density-dependent population growth for frequency- and density-dependent selection. American Naturalist 110:849-860.

Stearns, S. C. 1977. The evolution of life history traits: a critique of the theory and a review of the data. Annual Review of Ecology and Systematics 8:145-171.

Strobeck, C. 1973. *n*-species competition. Ecology 54:650-654.

Valle, R. R., E. Kuno, and F. Nakasuji. 1989. Competition between laboratory populations of green leafhopper, *Nephotettix* spp. (Homoptera: Cicadellidae). Researches on Population Ecology 31:53-72.

Verhulst, P.-F. 1838. Notice sur la loi que la population suit dans son accroissement. Correspondance Mathématique et Physique 10:113-121.

Volterra, V. 1927. Variazioni e fluttuazioni del numero d'individui in specie animali conviventi. Regio Comitato Talassografico Italiano, Memoria 131:1-142.

Wagner, G. P. 2010. The measurement theory of fitness. Evolution 64:1358-1376.

Williams, F. M. 1972. Mathematics of microbial populations, with emphasis on open systems. Transactions of the Connecticut Academy of Arts and Sciences 44:397-426.

Wilson, D. S. and M. Turelli. 1986. Stable underdominance and the evolutionary invasion of empty niches. American Naturalist 127:835-850.

Wilson, E. O. and W. H. Bossert. 1971. A Primer of Population Biology. Sinauer Associates, Sunderland, Massachussetts.