The struggle for existence. How the notion of carrying capacity, K, obscures the links between demography, Darwinian evolution and speciation

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The struggle for existence: how the notion of carrying capacity, *K*, obscures the links between demography, Darwinian evolution, and speciation

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ABSTRACT

Question: Population ecology and population genetics are treated separately in most textbooks. However, Darwin's term the 'struggle for existence' included both natural selection and ecological competition. Using the simplest possible mathematical models, this paper searches for historical reasons for the lack of unity in ecological and evolutionary thought.

Assumptions and methods: Logistic density-dependent population growth and Lotka-Volterra competition models are used throughout. Derivations of the logistic from first principles of resource use, competition for space, and births and deaths of individuals are documented. A full range of possible kinds of natural selection, including constant selection, density- and frequency-dependent selection, as well as hard and soft selection, can emerge cleanly as natural outcomes from the simplest-imaginable haploid models derived from Lotka-Volterra competition. Extensions to incorporate more realism, including non-linear per capita density dependence, Allee effects, complex life histories, discrete generations, diploid Mendelian genetics, sexual populations, and speciation are briefly discussed.

Conclusions: Widespread use of *r*-*K* ('carrying capacity') models of population growth appears to have catalysed fundamental discords in ecology, and between ecology and evolution. Verhulst's original polynomial form of the logistic, here termed the *r*-α model, is both more natural in theory, and accords better with empirical data. The *r*-α formulation explains apparent paradoxes involving the *r*-*K* logistic, including controversial aspects of *r*- and *K*-selection. Adoption of first-principles birth–death or *r*-α modelling clarifies natural selection in density-regulated populations, and leads to an improved understanding of Darwinian evolution and speciation.

Keywords: adaptive dynamics, density-dependent selection, eco-evolutionary dynamics, history of ecology, population genetics, theoretical ecology.

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INTRODUCTION

The theoretical core of evolutionary biology – evolutionary genetics – often ignores the original impetus of its creation, population ecology. Both Darwin and Wallace independently happened upon the idea of natural selection after reading Malthus' treatise on population growth and human suffering (Malthus, 1826). Some of 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 population ecologists focused only on population densities of competing individuals and species. Meanwhile, the originators of population genetics, Ronald A. Fisher, J.B.S. Haldane, and Sewall Wright, based theories of natural selection on demography, but generalized their evolutionary models almost exclusively in terms of gene frequencies within species. As a result, today's textbooks treat population ecology and evolution by natural selection as almost entirely separate topics.

What is the importance of density dependence in population growth? What is 'fitness' in evolution? These questions may seem somewhat trivial to a mathematical biologist, but both have been subject to extensive discussion over the last century or so. Related debates were about '*r*-' versus '*K*-selection', and its relevance for life-history strategies in what is broadly considered part of 'ecology', as well as frequency- and density-dependent selection, and speciation, topics considered part of 'evolutionary genetics'.

In this article, I advance a novel thesis: that these debates have been particularly intractable due to a tendency for logistic and related models of population growth to deceive their users. The problem is not with demographic theory itself, but with our interpretation of particular formulations that include a 'carrying capacity' parameter, *K*. It is possible to claim even more: that these problems began with a historical accident, and then led to a major rift between evolutionary genetics and population ecology.

My attempt to explain this impasse is predicated on the Darwinian idea that natural selection is isomorphic with ecological competition. Therefore, demography, natural selection, and speciation should be formulated using the same underlying models. In contrast, models of natural selection are still largely separate from those of ecological competition (Lewontin, 2004), and neither map closely onto models of adaptive speciation.

For example, Schluter's treatment of adaptive radiation (Schluter, 2000) argues that the ecological theory of adaptive radiation consists of 'three main processes . . . The first is phenotypic divergence . . . driven by natural selection between environments. The second is phenotypic divergence mediated by competition for resources . . . The third [is] "ecological speciation", whereby new species arise by . . . divergent selection stemming from environments and resource competition' (Schluter, 2000, pp. 65–66). But demographic competition is the source of natural selection as well as the cause of speciation, and both are also forms of reproductive isolation. Competitive – or ecological – speciation can therefore result from a single, unified process in density-limited populations (Rosenzweig, 1978; Dieckmann and Doebeli, 1999). Both natural selection and resource competition depend entirely on differences among populations (or species) in per capita population growth: competition and natural selection are therefore identical. If we can sort out these issues in the simplest cases of natural selection, and if speciation falls in line (Metz, 2011), perhaps the mists will part to allow the reconstruction of a unified Darwinian theory of ecology and evolution, including competition, natural selection, and speciation.

LOGISTIC MODEL OF POPULATION GROWTH

The normal form of the logistic equation found in ecology textbooks is the 'carrying capacity' or *r*-*K* formulation:

$$\frac{1}{N}\frac{dN}{dt} = R = r\left(1 - \frac{N}{K}\right). \quad (1)$$

Note that the equation is expressed here in terms of per capita growth rate, R, Fisher's (1930) 'Malthusian parameter' of Darwinian fitness. N is the population size, t is time, r is the intrinsic rate of increase – the rate of increase when the population has very low size – and K is the equilibrium population size, or carrying capacity. This version of the logistic is ubiquitous in today's ecology textbooks. The *r*-*K* model is related to many other carrying capacity-based formulations of density regulation and interspecific competition, including the theta-logistic models (Ayala *et al.*, 1973), and the Ricker (Ricker, 1954) and similar discrete-time analogues of the logistic. Carrying capacity formulations have come to seem intuitive, and also underlie many recent theoretical and practical applications in ecology. The logistic and its extensions into competition have been used in many attempts to incorporate these ideas into evolutionary models, particularly in relation to density-dependent selection and *r*- and *K*-selection (see discussion below).

It is not generally realized that the use of a carrying capacity (K) parameter required a change of variables compared with the original equation for logistic growth (Verhulst, 1838; Pearl and Reed, 1920; Lotka, 1925; Volterra, 1927; Kostitzin, 1939). The form of the Malthusian parameter in these was:

$$\frac{1}{N}\frac{dN}{dt} = R = r - \alpha N. \quad (2)$$

Here r apparently has a similar meaning of intrinsic growth rate when population size is low, as in equation (1) (but see below for some differences), while α is the density-dependent crowding effect, or intraspecific competition coefficient. As shorthand, I shall call equation (2) the *r*-α model, in contrast to equation (1), the *r*-*K* model. This simpler, polynomial form of the logistic was justified by its original authors because it represents the simplest useful Taylor approximation to the true non-linear model (Verhulst, 1838; Lotka, 1925; Hutchinson, 1978). The two are of course inter-convertible formulations of the same underlying logistic, since the *r*-α model has equilibrium $\hat{N} = r/\alpha$, equivalent to K in equation (1). The r/α equilibrium seems intuitively reasonable as a balance between density-independent growth and its density-dependent regulation; in contrast, K, seen as a carrying capacity parameter, seems to confuse a fixed resource limit with equilibrium density. Given that both these formulations represent the same underlying model, it is obviously immaterial which we use. However, as I argue below, the *r*-*K* model has nonetheless misled generations of ecologists and evolutionary biologists in a way that the *r*-α model would not have done.

Trawling the tangled history, I have encountered publications that raise almost all the problems I discuss here (Schoener, 1973; Jensen, 1975; Vandermeer, 1975; Kozlowski, 1980; Hallam and Clark, 1981; Clark, 1983; Kuno, 1991; Christiansen, 2004; Lewontin, 2004; Gabriel *et al.*, 2005; Pastor, 2008). Although these treatments have in some cases long been available, their potential readership has largely ignored them. Textbooks continue to teach ecology and evolution as separate topics having rather different-looking basic theory. My treatment will in addition therefore develop a historical and explanatory hypothesis of how this situation came about. Few appear to have

put evolutionary problems together with the ecology to come up with a synthesis of the whole area (but see Kostitzin, 1939; Crow and Kimura, 1970; Leigh, 1971; Smouse, 1976; Prout, 1980; Meszéna and Pásztor, 1990; Christiansen, 2004; Meszéna *et al.*, 2005; Barton *et al.*, 2007; Metz, 2011). This I attempt here, for the simplest case of instantaneous logistic population growth, Lotka-Volterra competition, and natural selection in asexual haploids. Extensions of this model to diploidy, discrete generations, complex life history, and quantitative traits are discussed but glossed over briefly because they can involve much greater complexity; however, these extensions are readily obtained in principle, and have already appeared in various forms elsewhere (Kostitzin, 1936, 1939; Charlesworth, 1971, 1980; Roughgarden, 1971; Smouse, 1976; Levin, 1978; Prout, 1980).

I begin with a brief defence of the logistic and its extension to competition among populations, while also pointing to its real deficiencies. This prepares for the main part of the paper, which outlines how other apparent deficiencies and paradoxes of the logistic are illusory. I document how a historical accident led to the substitution of the original, more natural r-α logistic by today's widely used r-K formulation. I review how the r-α logistic can be derived 'microscopically' from first principles of births and deaths in density-limited populations, referring in passing to much more detailed theory showing that similar forms can be derived from considerations of the flow of energy and chemicals. Then I describe how natural selection in density-dependent populations becomes clarified in the r-α logistic, and how the r-K form has led to a number of controversies, focusing in particular on density-dependent selection, as well as r- and K-selection. I describe how many of these problems can be resolved using more natural, process-based r-α forms. Finally, I discuss how these findings are important for understanding speciation.

WHY USE THE LOGISTIC? IN DEFENCE OF THE SIMPLEST MODEL

A number of mathematical treatments successfully incorporate per capita population growth, Fisher's Malthusian fitness parameter, into very general models of natural selection and evolution (Fisher, 1922, 1930; Haldane, 1924; Charlesworth, 1971; Levin, 1978; Metz *et al.*, 1992; Barton *et al.*, 2007; Metz, 2011). My aims here are much more modest: to show how the most widely known model of density-dependent population growth, the logistic equation and its extension into Lotka-Volterra competition, can lead simply to a rich variety of behaviours under natural selection. Even this limited synthesis has received previous attention (Kostitzin, 1936; Smouse, 1976), but the results have been largely unrecognized: they are absent from textbooks of evolution and evolutionary genetics (Barton *et al.*, 2007; Hartl and Clark, 2007; Futuyma, 2009; Charlesworth and Charlesworth, 2010), ecology (Ricklefs and Miller, 2000; Begon *et al.*, 2006; Gotelli, 2008; Krebs, 2009), or combined textbooks (Wilson and Bossert, 1971; Roughgarden, 1979; Charlesworth, 1980; Ricklefs, 2008).

The logistic equation is unrealistic. It ignores separate sexes and life history, and makes the simplest possible assumption about density dependence – a linear decline of per capita growth rate via an instantaneous response to current density. The 'production curves' of natural populations, graphs of the rate of population growth with density, are known to be often skewed, rather than yielding the expected logistic parabola (Schoener, 1973; Roughgarden, 1997). It is clear that logistic growth can rarely be observed, except approximately in some unicellular organisms (Gause, 1934; Leslie, 1957; Vandermeer, 1969). The logistic equation is in continuous time, and all its processes are supposed to occur instantaneously, and so it does not adequately model populations with discrete generations, populations with time delays in the response to density, or those with complex life histories. These introduce lags, produce chaotic fluctuations, and other complications (Charlesworth, 1971; Metz *et al.*, 1992).

Nonetheless, if population growth rates per generation are small, and especially if densities are near equilibrium, it is clear that the continuous time logistic will hold as a useful first approximation for population growth (Verhulst, 1838; Lotka, 1925; MacArthur, 1972; Hutchinson, 1978), as well as in competition and natural selection (León and Charlesworth, 1978). It is a particularly useful first step in understanding population regulation in the simplest organisms or cases, and many general conclusions from logistic models should be true also for more realistic situations. Furthermore, the Lotka-Volterra extension of the logistic to competition is also unrealistic for the same reasons as is the logistic, and will provide a close fit to data only rarely. However, like the logistic, Lotka-Volterra competition is a useful first approximation. It also holds well enough in some cases (Gause, 1934; Leslie, 1957; Vandermeer, 1969) to gain important understanding of the process of competition among real populations, as well as evolution by natural selection.

An important group of non-linear models of population growth have 'Allee effects': the tendency for populations to decline at very low densities. The logistic assumes that maximal growth is achieved at the lowest possible density, but in nature members of a population can facilitate one another's survival, for example in group defence against predators, or because mate-finding becomes difficult at low densities. I do not treat this complication here, although the theory has been dealt with a number of times since the dawn of population ecology (Kostitzin, 1940; Asmussen, 1979; Jacobs, 1984). Logistic assumptions again hold approximately, in spite of Allee effects, provided the population is near equilibrium.

Perhaps the most important reason for clarifying the logistic equation and its relation to natural selection is its great familiarity due to a long tradition in ecology texts. Extending the logistic to cover natural selection is justified in part because the greater complexity of adding realism obscures the simple connection between ecology and evolution. Greater realism compounds still further the additional muddle in students' minds engendered by the notion of carrying capacity, the problem that forms the major focus of this article. Nonetheless, more advanced assumptions can readily be incorporated onto a logistic backbone, including non-linear density dependence, extended life histories, and delays in density dependence such as those introduced via discrete-time models. Thus, the logistic is a useful starter toolkit for modelling real systems, as well as a simple, clear didactic tool.

HISTORICAL ACCIDENTS LED TO ADOPTION OF THE *r-K* FORM OF THE LOGISTIC

As already mentioned, Darwin employed the term 'struggle for existence' as a synonym for both natural selection and ecological competition, and Fisher's Malthusian parameter (R in equations 1 and 2) was his formalization of Darwinian fitness. 'The Struggle for Existence' later formed the title for Georgii Gause's well-known book. Gause viewed his work as an experimental investigation of the mechanism of Darwinian natural selection rather than merely about ecological competition (Gause, 1934). His isocline graphical method for analysing competitive equilibria (Gause and Witt, 1935), since then widely used in generations of ecology courses, was explicitly titled as a paper on natural selection, not population ecology. Today, Gause is most closely associated with ecology, but before the Second World War it is clear that ecological competition and natural selection were synonyms.

Educators and theoreticians alike have perpetuated the idea that the equilibrium population size, K, is a measure of 'carrying capacity' and resource abundance. For example: '[The parameter K] has a ready biological interpretation as the carrying capacity

of the environment. *K* represents the maximum population size that can be supported' (Gotelli, 2008, p. 28; see also Gause, 1934, p. 34). Intuitively the idea seems to apply particularly well to some forms of population control, for example birds with limited nesting sites. Gotelli continues: 'As an analogy, think of the carrying capacity as a square frame that will hold a limited number of flat tiles. If the population should ever exceed the carrying capacity, there would be more tiles than could fit in the frame. The unused portion of the carrying capacity is the percentage of the area of the frame that is empty' (Gotelli, 2008, p. 28). [Gotelli here cites the 1985 edition of C.J. Krebs' (2009) ecology textbook. As we shall see, it is actually easier to derive a logistic model from an argument where the equilibrium population density is not equal to the number of spaces for tiles (see the nest box model below).] However, today's usage of *K* as a carrying capacity in this sense dates only since the mid-twentieth century (Sayre, 2008).

Raymond Pearl was the first to promote the *r*-*K* formulation, most influentially (as it turned out) in a textbook of medical biometry (Pearl, 1930). Pearl, together with Lowell Reed, had rediscovered the logistic growth model (Pearl and Reed, 1920), in ignorance at that time of Verhulst's (1838) much earlier paper. Pearl wished to estimate limits to human population growth, and famously predicted, incorrectly as it turned out, that the human population of the USA should level off at $K = 197$ million on the basis of a fit to the logistic. Pearl believed that he had discovered a physical law of population growth. The predicted equilibrium limits of human populations were, for Pearl, among his most important findings (Kingsland, 1982, 1985).

Gause wrote his influential book while studying with W.W. Alpatov in Moscow. Alpatov himself had worked with Pearl in the USA before returning to Russia. Gause and Alpatov adopted Pearl's (1930) method to fit their experimental results. The *r*-*K* model appears to have been used mainly because it helped Pearl, Alpatov, and Gause to use a double-pass method to fit logistic parameters to data on population growth: first determine the stable density *K*, and then use a linear fit of log-transformed population growth data to estimate *r* (Pearl, 1930). Interestingly, even this use of the *r*-*K* model for ease of fit was unnecessary: they could have used a simpler linear fitting procedure from which *r* and α, or *r* and *K*, could have been estimated in a single pass (Yule, 1925; Rhodes, 1940; Crossner, 1977).

Gause's highly original and influential work on experimental population ecology, and his subsequent collaboration with the mathematician Witt to develop Lotka-Volterra isocline analysis (Gause and Witt, 1935), led to a virtual fixation on the *r*-*K* formulation after the mid-twentieth century (Kingsland, 1982; Christiansen, 2004, p. 142). Gause's work and the *r*-*K* logistic was followed up and strongly promoted in the first major post-war textbook of ecology (Allee *et al.*, 1949). In this work, the term 'carrying capacity' was introduced to the general ecology literature, but away from where the logistic was outlined, and used in its original range management sense of the amount of range that could support a given population. Discussing the deer population of the Kaibab plateau of Arizona before predators were hunted out, the authors opined: 'pumas and wolves seem to have kept the number of deer well below the carrying capacity of the range' (Allee *et al.*, 1949, p. 706). It was a few years later that E.P. Odum made the first link between carrying capacity and the logistic equilibrium *K*: 'the upper *asymptote* of the *S*-shaped curve . . . has aptly been called the "carrying capacity" or the saturation level' (Odum, 1953, p. 122). This highly influential textbook went through many editions, and almost certainly led to today's synonymy of *K* and carrying capacity (Kingsland, 1982, 1985; Sayre, 2008). Today, the *r*-*K* model, and the use of *K* as a synonym for carrying capacity, is virtually universal in textbooks, and has been taught to generations

of undergraduates, particularly since the work of MacArthur. The predominant use of the *r*-*K* formulation seems clearly due to these historical accidents (Christiansen, 2004), although very likely helped along by the apparent 'aptness' of *K* as the resource limit or carrying capacity of the environment.

DERIVATIONS OF THE LOGISTIC FROM FUNDAMENTAL PROCESSES

A number of approaches have been used to derive demographic models from individual processes that underlie the population dynamics. These derivations might therefore give some clue as to which of the *r*-*K* (equation 1) and *r*-α (equation 2) models is the more 'natural' form (in so far as we can describe one formulation of the same equation as more natural than another).

The 'nest box' model: competition for space

Many people believe that the *r*-*K* model is particularly reasonable in one case: when there is a conflict among individuals for limited space (see, for example, Gotelli's argument above). This form of population control is known to exist in nature, including in certain hole-nesting birds. Provision of nest boxes can increase the population of great tits (*Parus major*), for example. The number of sites appears to represent the carrying capacity, *K*, in such a visualization.

However, it is not easy to construct a mechanistic model where all sites are occupied at equilibrium. The simplest such model would consider continuous births and deaths in an environment with limited space. I call this the 'nest box' model (Fig. 1), although the earliest version I have found proposes it as a model of trees growing towards a limited forest canopy (Schaffer and Leigh, 1976).

There are *T* nest boxes, or sites. Suppose each site can support one and only one female. Assume that the birth rate of individuals occurs at a constant rate b_0, and deaths occur spontaneously at a rate d_0. Deaths free up unoccupied sites. To make the problem simple, we add the further assumption that the parent stays, while newborn individuals must disperse to another site selected at random from all possible sites, and that they survive only if the new site is not already occupied (Fig. 1).

Suppose *N* is the population size, b_0 is the per capita birth rate, while the per capita death rate is d_0 plus additional deaths due to failure of newborns to find an unoccupied site. The probability that each newborn survives due to random dispersal to an unoccupied site is

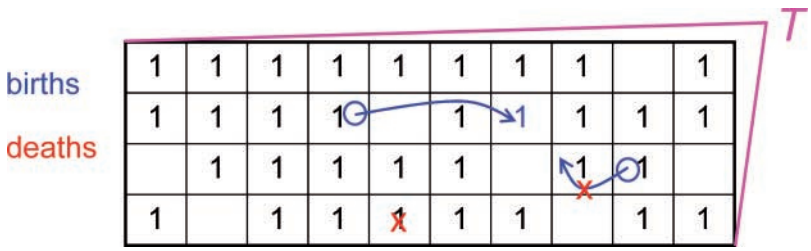

Fig. 1. Deriving the logistic via competition for space: the nest box model. Births and dispersal are represented by the circles and arrows. Deaths are represented by crosses.

$(1 - N/T)$. Then, the overall per capita growth rate of the population (Schaffer and Leigh, 1976; Kozlowski, 1980) is:

$$\frac{1}{N}\frac{dN}{dt} = b_0 - d_0 - b_0\frac{N}{T}. \tag{3}$$

Setting $b_0 - d_0 = r$, and the density-dependent death rate term $b_0/T = \alpha$, we again have per capita growth rate of $r - \alpha N$, i.e. equation (2). Thus space limitation can be used to derive a logistic growth equation, but the so-called carrying capacity, K, is less than the total number of available spaces, T, by a factor $(b_0 - d_0)/b_0$ (see also Clark, 1983).

Although in equation (3) a maximum of only one individual is present in every site, this simple model has a close relationship to the Levins metapopulation model (Levins, 1969), in which births are analogous to colonization, and deaths to subpopulation extinction. This metapopulation model itself generates a logistic function, as is well known (Hanski and Gilpin, 1991; Roughgarden, 1998). The model can also be extended to multiple species, allowing coexistence due to differences of colonization and extinction functions, in a process analogous to Lotka-Volterra stability, which Roughgarden called the 'logistic weed' model (Tilman, 1994; Roughgarden, 1998).

Kostitzin's birth–death model

More generally, one can derive models of population growth from simple enumerations of births and deaths, without precisely specifying the mechanistic causes. This was the approach of Kostitzin (1939) and Gabriel *et al.* (2005), which has been reproduced recently in a number of textbooks (Gotelli, 2008; Pastor, 2008).

The per capita growth rate (R) is given by the difference between births (B) and deaths (D) at any instant. Taking a Taylor approximation, density dependence acting on per capita birth and death rates might be linear, $B = b_0 - \beta N$ and $D = d_0 + \delta N$. Then, the per capita growth rate is given by:

$$\frac{1}{N}\frac{dN}{dt} = B - D = (b_0 - d_0) - (\beta + \delta)N. \tag{4}$$

Clearly, this is an r-α logistic, with $r = b_0 - d_0$ and $\alpha = \beta + \delta$, and the resultant equilibrium $K = (b_0 - d_0)/(\beta + \delta)$ is directly proportional to r, rather than appearing as an independent parameter.

Partitioning density-dependent and density-independent population growth parameters

The parameter r in the r-K model is usually called 'the intrinsic rate of increase', which seems to imply its effect on growth rate is independent of density. However, r in equation (1) is a density-dependent growth parameter in the sense that altering it affects the overall per capita growth rate, R, in a manner dependent on density. It is simplest to show this by taking partial derivatives. For the r-K model (equation 1), $\partial R/\partial r = 1 - N/K$, and $\partial R/\partial K = rN/K^2$. Thus both r and K are density-dependent growth parameters in equation (1) (Schoener, 1973).

In equation (2), however, there is a clean separation between the density-independent r and the density-dependent α parameters of population growth; the equivalent partial

derivatives are $\partial R/\partial r = 1$ and $\partial R/\partial \alpha = -N$. Although the numerical values of r may be the same in equations (1) and (2), leading to the confusing idea that r in each case is the same parameter, the effect of r is here shown to behave curiously differently when densities are non-zero. This difference between equations (1) and (2) is especially important when we are interested in the parameters as genetically determined traits in models of natural selection. As I point out below, this has led to considerable confusion in the *r*- and *K*-selection debate.

We cannot easily infer from these purely mathematical considerations which parameterization is more 'natural' – the classic version of equation (1), or the simpler polynomial equation (2). However, it does seem reasonable that density-independent effects of birth and death should in nature be separate from the effects of crowding, as in the r-α logistic (equation 2), rather than being convoluted with them as in the r-K logistic (equation 1).

Energy flow, chemical flow, and other considerations

Equations for population growth are closely related to those for chemical reaction kinetics (Pearl and Reed, 1920; Lotka, 1925). This is unsurprising, as organismal growth and reproduction depend directly on chemical reactions. Changes in sizes or numbers of organisms depend on energy flow and processing of chemicals into different states. Thus, we should be able to derive models for population growth explicitly from an understanding of energy and/or chemical flux. Several such derivations have been performed.

Roughgarden (1971, pp. 465–467) developed a discrete-time model of population growth based on ecological energetics, in which the interest was to 'indicate how an organism participates in the overall energy through his population'. The model assumes conservation of energy, and therefore that the energy dissipated by each individual must be equal to the energy input. Roughgarden assumed that dissipation is liable to increase and that input is liable to decrease with density. Assuming linear density dependence of dissipation and input, Roughgarden derived an energy balance equation that is a discrete-time analogue of equation (4), where births are equated with inputs and deaths are equated with dissipation. Schoener (1973) introduced a related continuous-time model based on energy flow and density-dependent energetic interactions among individuals, where the density dependence was linear. In both cases, the equilibrium density, K, represents a balance between density-independent growth, r, and density-dependent crowding effects, α, as in equation (2). As expected, a higher input (or lower dissipation) of any energy available, independent of density, tends to increase the equilibrium population size. Schoener (1973) further generalized his model to non-logistic functions, and to competition among multiple species.

Another approach was to use a conservation of mass argument to examine exploitation of a resource by a primary producer or consumer (Williams, 1972). Williams' consumption model follows a model close to the Lotka-Volterra predator/prey model, while ensuring that the mass of the resource and the consumer in a closed (or open) system remains the same before and after consumption and population changes. Without going into details, systems both open and closed to matter flux lead to logistic growth of the consumer with equilibrium density, K, proportional to density-independent growth rate, r, as in equation (2) [see Pastor (2008) for further details and a clear explanation]. Similar considerations apply more generally to competition among a pair of species for a resource. Pastor (2008, p. 207) concludes: 'We have therefore recovered all the consequences of predator–prey models with stable limit cycles as well as the possibility of coexistence equilibrium between two competitors, but we have done it without the use of K at any level of the

food web . . . Therefore, the mass balance constraints of the flow of materials through a food web, required by conservation of matter, ultimately underlies the stabilities of populations, communities, and ecosystems'.

An alternative was to bolt classical, density-independent population-genetic fitnesses onto a purpose-built demographic model of life history, births, and deaths for a particular organism, *Drosophila* fruitflies, with a discrete-generation life-cycle (Prout, 1980). When genotypes at a single locus that differ in fitness were treated under this approach, it was again shown that equilibrium population density was proportional to the density-independent growth parameter, r, as in the r-α equation (2).

In all of these various models, where births and deaths are treated in some realistic manner as a result of individual birth–death, energy flow, or chemical flow processes, the resultant model can be reduced to a form of equation (4): each, therefore, has the underlying form of the r-α model (equation 2). Each of these derivations therefore results in an equilibrium population density approximately proportional to the rate of increase, r, in addition to its control by density-dependent parameters.

HOW THE r-α MODEL ALLEVIATES PROBLEMS WITH THE LOGISTIC

The controversy over density-dependent versus density-independent population regulation

From about 1950 until circa 1980, a major debate raged over the importance of density dependence in regulating natural populations. This controversy is largely over today, with most ecologists now in favour of the universality of density dependence (Turchin, 1995). The debate was largely stirred up by Herbert Andrewartha and Louis Charles Birch, who had argued that population levels were determined largely by climate (Andrewartha and Birch, 1954). While I do not condone the common interpretation of their argument, it is evident that their assertions about density-independent population determination were greatly misunderstood. Andrewartha and Birch argued particularly strongly against the extreme balance-of-nature ideas of Alexander John Nicholson among others, who argued that 'competition [i.e. density-dependence] always tends to cause animals to reach, and to maintain, their steady densities' (Nicholson, 1933, p. 176).

In a little-read passage, Andrewartha and Birch (1954, pp. 347–396) developed the r-α logistic model (equation 2) for population density regulation, and then showed that density-independent factors like climate could, in a sense, determine the equilibrium density $K = r/\alpha$ via death rates (d_0 in equation 4) within the parameter r. They outlined the 'practical weakness' introduced by Pearl's (1930) r-K formulation of the logistic (equation 1) – in particular when $r < 0$ (see below under 'Levins' Paradox'). Much of their critique was directed against terms like 'regulation', 'balance', and 'control' when applied to density; they did not deny the existence and importance of density-dependent factors. Their argument that densities were largely determined by density-independent factors is true in the sense that equilibrium densities might be very variable, due largely to climate-induced mortality affecting the parameter r, as is likely in the Australian desert grasshoppers and other organisms they studied. Similarly, a farmer's regular treatment of insecticides is a successful and useful method to reduce equilibrium densities of insect pests. This results from the toxin's density-independent effect on death rates and therefore r, and thence to K also (see also Fig. 3 below) (Andrewartha and Birch, 1954, p. 662), at least until resistance to the

insecticide evolves. Their opponents failed to appreciate this alternative view of the logistic, and it seems clear in retrospect that this misunderstanding considerably extended a fruitless debate.

Levins' Paradox, and other simple misunderstandings of the logistic

As we have seen, equation (1) has often been claimed to be unrealistic, and indeed it can behave very oddly indeed. Exponential growth ($dN/Ndt = r$) can be negative as well as positive; thus we have no problem imagining that, at some times or places, populations may decline, i.e. that $r < 0$. However, when we add density-dependent regulation to obtain equation (1), strange results can ensue. The best-known case occurs when $r < 0$, and $N_0 > K$ as a starting condition: equation (1) predicts a singularity so that population growth accelerates until $dN/dt \to +\infty$ after a finite time $t = \frac{\log\{N_0/(N_0 - K)\}}{|r|}$ (Kuno, 1991). This is the 'Levins' Paradox' of George Evelyn Hutchinson (Hutchinson, 1978; Gabriel *et al.*, 2005). If $r < 0$ with $N_0 > K$, we might expect the population to decline to extinction rather rapidly: exceeding the carrying capacity should reduce population growth still further below zero. Instead, a product of two negatives in equation (1) becomes positive and the population grows to infinity. The statistician Edwin Bidwell Wilson used this paradox to mock Raymond Pearl's law of population growth: when Pearl's equation was fit to Canadian data, an infinite human population was predicted in Canada by the year 2012 (Wilson, 1925) – thankfully, another unfulfilled prediction.

To avoid this problem, one is usually advised to deal only with populations having positive r; otherwise, the logistic model is argued to be pushed beyond its applicable limits. However, in theories of the evolution of geographic range limits of species, for example, we expect $r < 0$ in unfavourable regions (Kirkpatrick and Barton, 1997; Polechová *et al.*, 2009). Similarly, a stochastically fluctuating local population should sometimes exceed K (Lande *et al.*, 2009); alternatively, it may achieve this condition in a deterministic limit cycle or chaotic fluctuation due to the existence of time lags in the density dependence. Levins' Paradox lends apparent support to the idea that the logistic is an inconsistent model of population growth.

A number of other such paradoxes associated with equation (1) are known (Kuno, 1991); for instance, when $r = 0$, the population neither grows nor declines no matter how large N is, even if larger than K. This seems just as strange (Kuno, 1991).

However, Levins' Paradox and the other 'paradoxes' identified by Kuno are all simply resolved on reversion to the r-α model (equation 2). If the logistic equation is ever to produce a stable equilibrium, the crowding parameter α must be positive, so that the effect on per capita population growth, $-\alpha N$ in equation (2), is negative. Then $r \leq 0$ in Kuno's paradox will always lead to negative population growth overall, because the (now unattainable) equilibrium r/α is forced to be negative. Thus the paradox for the r-K model is caused by the intuitive idea under the carrying capacity or resource limit interpretation that one can have a positive K when there is a negative r. When $r = 0$ in equation (2), the population will decline at a rate $dN/dt = -\alpha N^2$, as expected. When r is negative, the decline will take place even faster, and the speed of decline will always increase with N, as it should.

Ginzburg's paradox

Ginzburg (1992) raised a different issue with the logistic; his note led to a flurry of replies and comments by other ecologists in *Trends in Ecology and Evolution* in the early 1990s. Supposing there is standard logistic growth, except that an additional fraction μ of the population is culled; this modifies equation (1) as follows:

$$\frac{1}{N}\frac{dN}{dt} = r\left(1 - \frac{N}{K}\right) - \mu. \tag{5}$$

'Are the final equilibrium abundances different [between equation 1 and equation 5]? Most ecologists will answer that the equilibrium values should be the same and that the higher rate of reproduction [in equation 1] just means that the population will "get there faster", but reach the same level nevertheless' (Ginzburg, 1992, p. 133). However, contrary to the ideas of 'most ecologists', the equilibrium changes from K (equation 1) to $K(r - \mu)/r$ in equation (5). Ginzburg continues: 'There are two possible conclusions. Either our intuition is wrong or the equation is wrong. I tend to side with intuition'. Therefore, 'in my opinion, the logistic equation, particularly in its common parameterization [i.e. equation 1] is one of the greatest disservices to theoretical ecology', and is 'fundamentally unable to serve as a basis for evolutionary conclusions'.

However, the equilibrium population density is more readily interpreted in an explicit model of births and deaths (equation 4 above) as a dynamic balance between density-independent growth and its control by crowding (Olson *et al.*, 1992; Gabriel *et al.*, 2005). If so, a reduction in r via an extra density-independent mortality is indeed expected to cause a reduction in the equilibrium. This is more transparently shown under form 2 of the logistic, where r is always clearly involved in the equilibrium density. The logistic may not represent all aspects of density-dependent growth in real populations, but the problem is not a fundamental one with the logistic: it is the particular formulation 1 and one's interpretation of that formulation that is at fault. The problem of intuition occurs because we automatically assume that the carrying capacity, K, should be a stand-alone parameter that can be altered independently of r.

The competition paradox: lack of involvement of *r* at equilibrium

Another frequently mentioned paradox occurs with the Lotka-Volterra extension of the logistic equation to a pair of species:

$$\frac{1}{N_1}\frac{dN_1}{dt} = r_1[1 - (N_1 - \gamma_{12}N_2)/K_1] \tag{6a}$$

$$\frac{1}{N_2}\frac{dN_2}{dt} = r_2[1 - (N_2 - \gamma_{21}N_1)/K_2]. \tag{6b}$$

The competition coefficients γ_{12} represent the effect of species 2 on species 1 relative to the effect of its own species, and vice versa for γ_{21}.

The problem may be simplest to explain via a personal anecdote. We had investigated a narrow contact zone between two closely related species of butterfly in Ecuador, *Heliconius himera* and *H. erato*. The species hybridize at a low rate, but behave mostly like separate species (Jiggins *et al.*, 1996). They are nearly identical in size and overall ecology. Indeed, they do not differ in host plant preference, and major host plants, which control butterfly

population density, occur throughout the contact zone (Jiggins *et al.*, 1997). These findings tend to persuade one that $\gamma_{12} \approx \gamma_{21} \approx 1$, and that the host-plant-related carrying capacities in any site along the zone should also be approximately equal (i.e. $K_1 \approx K_2$), yet *H. himera* is restricted to gallery forest in savannah conditions in drier, higher altitude sites, while *H. erato* occurs only in humid rainforest to the north and west. The narrow (5 km wide) contact zone occurs in a zone of transition between forest types: the two species appear to exclude each other across this aridity gradient. I had initially imagined that intrinsic (i.e. density-independent) rates of increase r were higher for *H. himera* in the drier upland forest, while *H. erato* has higher r in the wet lowlands. This is similar to the assumptions used in models of range limits in the absence of competition among species (Kirkpatrick and Barton, 1997; Polechová *et al.*, 2009).

Yet, as is well known from the Gause-Witt analysis of the isoclines of equations (6), where $dN_1/dt = 0$ and $dN_2/dt = 0$, depend only on the relative values of K and γ, and not on values of r. Geographic variation in r_1 and r_2 should, according to this model, be able to explain neither the competitive exclusion of each species, nor the narrowness of the contact zone if each species is at equilibrium densities. The approximate equality of K between the species with $\gamma_{ij} = 1$ should lead to approximate neutral stability. Any differential specialization by each species would lead to $\gamma_{ij} < 1$, and so to stable coexistence and a broad zone of stable contact, rather than a narrow zone of replacement as found.

Many others have puzzled over this 'particular shortcoming' of the Lotka-Volterra competition model (e.g. Begon *et al.*, 2006, p. 237). The intrinsic growth rate r seems an excellent candidate for a component of fitness in evolutionary biology, and is used in this way in classical population genetics incorporating constant selection, such as would occur in exponentially growing populations. How, then, can it not affect the joint equilibria between species or populations that are density regulated? This seems to pose a major problem in generalizing between natural selection within species and the population dynamics of competition between species.

Before dealing with the problem, we should note that r does have other effects in equation (6). For instance, the rate that equilibrium is attained depends on r (Roughgarden, 1971), and so does the stability of equilibria in models with ≥3 species (Strobeck, 1973). Furthermore, outcomes of competition in equation (6) under regular or stochastic environmental fluctuations also depend on r (Roughgarden, 1971; Lande *et al.*, 2009). However, the lack of involvement of r in isoclines (single species equilibria) under deterministic competition remains puzzling (Maynard Smith, 1998, p. 19). So what is wrong? Maynard Smith concluded simply that, 'this is an unfortunate feature of the logistic', and without resolving it, developed an alternative ecological model of density-dependent natural selection in which the equilibria depended on r.

Maynard Smith's reaction is not unusual (or, of course, in any way wrong). However, he apparently did not realize that such ideas depend on one's interpretation of formulation 1 of the logistic. Instead of equation (6), if we extend the original r-α Verhulst formulation 2 to Lotka-Volterra competition, we obtain:

$$\frac{1}{N_1}\frac{dN_1}{dt} = r_1 - \alpha_{11}N_1 - \alpha_{12}N_2 \tag{7a}$$

$$\frac{1}{N_2}\frac{dN_2}{dt} = r_2 - \alpha_{22}N_2 - \alpha_{21}N_1. \tag{7b}$$

Equation (7) is now in the original form of the first ecological competition models (Lotka, 1925; Volterra, 1927), and in Kostitzin's case (Kostitzin, 1939), of his model of natural selection in density-regulated populations.

After adopting Pearl's *r-K* logistic for curve fitting, Gause (1934) and Gause and Witt (1935) were apparently also the original promoters of equation (6) rather than equation (7). As Gause (1934) was the source of most post-war scientists' understanding of competition, equation (6) rather than equation (7) is the form now virtually universal in textbooks. [*Note*: Although an unimportant detail for our purposes here, my rendering of equation (7) here parameterizes interspecific competition (as well as intraspecific competition) in a slightly different way from that usually adopted in today's *r-K* models (equation 6), in that $\gamma_{12} = \alpha_{12}/\alpha_{11}$ and $\gamma_{21} = \alpha_{21}/\alpha_{22}$.]

Equation (7) is as suitable as equation (6) for textbooks of ecology, coupled with Gause and Witt isocline analysis, providing we overcome our educational training in late twentieth-century carrying capacity ideas. After trying this out for the first time at Harvard (spring 2012), my teaching assistants and I believe that equation (7) is even somewhat simpler and more intuitive for teaching. For instance, a revised Gause and Witt graphical isocline analysis is shown in Fig. 2.

As can be seen from Fig. 2, the isocline method is unchanged, except that the isocline for each species now crosses axes at heuristically reasonable values of population size determined by a balance between the intrinsic rates of growth for that species and the locally important competition/crowding coefficient, depending on which form is most abundant.

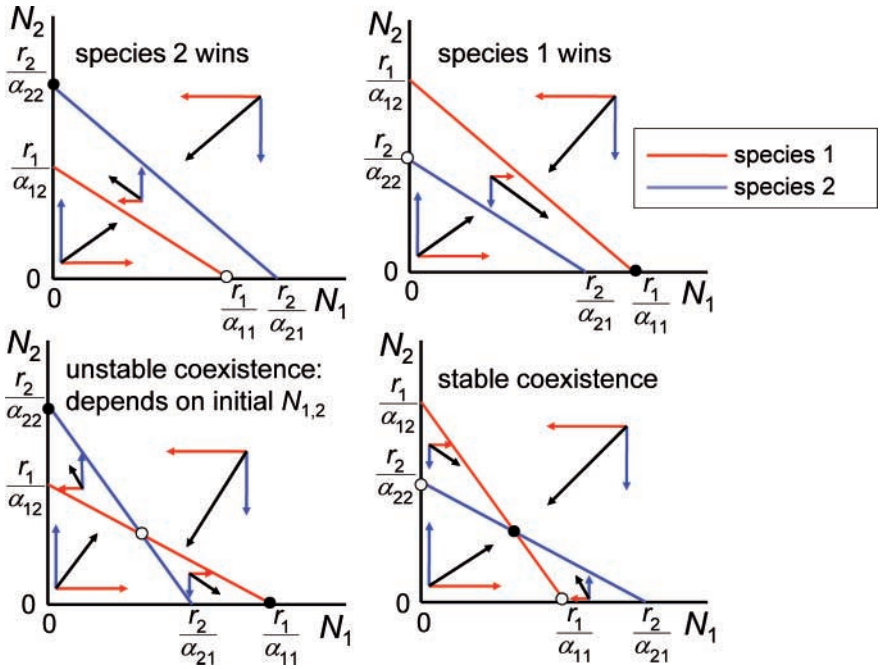

Fig. 2. Gause-Witt isocline analysis of Lotka-Volterra competition, using equation (7). The isocline for each species i is the line on the N_1/N_2 phase plane where $dN_i/dt = 0$. Joint equilibria are shown as solid circles (stable) and open circles (unstable).

Recent theory has used microscopically derived birth–death models (similar to equation 4) to investigate multi-species coexistence conditions in individual-based models of spatially structured populations (Law *et al.*, 2003; Murrell, 2010); these models will naturally result in local demography similar to an *r*-α Lotka-Volterra model, and they therefore avoid the problems of the *r*-*K* model.

EMPIRICAL EVIDENCE FOR THE PROPORTIONALITY OF *K* TO *r*

So far, we have seen that the use of the carrying capacity, *K*, as a parameter in the logistic can be misleading, and that it can avoid confusion to view the equilibrium density as a balance between opposing density-independent (*r*) and density-dependent (α) parameters ($K = r/\alpha$). In this section, we explore empirical evidence for a relationship between *K* and *r*. If *r* and α are in a sense the 'real' parameters, whereas *K* is a compound parameter derived from these, equilibrium density should sometimes change in direct proportion to density-independent factors that affect *r*. If, on the other hand, *K* is a 'real' parameter in its own right, we might expect no effect on *K* of changing *r* experimentally, as in Ginzburg's intuition (above). Interestingly, Andrewartha and Birch (1954, p. 392) attempted to search for experimental evidence for proportionality of *r* and *K* in support of their *r*-α model, but were unable to find any at that time. Today, considerably more data are available.

Kuno (1991) cites data on three rice-feeding leafhoppers of the genus *Nephotettix* reared at different temperatures (Valle *et al.*, 1989). These treatments affected both the initial population growth (*r*) and the carrying capacity (*K*). When log *r* was plotted against log *K*, a slope of 0.935 was achieved explaining 59% of the variance; however, when the effect of *r* was removed by plotting $r/K = 1/\alpha$ against *K*, there was a non-significant slope of 0.065 explaining only 0.7% of the variance. Thus we might conclude that the main effect of the treatment was to alter *r* rather than the crowding effect α, and through this density-independent effect, the carrying capacity, *K*.

Another example is from the environmental toxicology literature. When low doses of toxic chemicals and pollutants are added to water bodies (a purely density-independent effect), the equilibrium densities of many species are reduced along with their intrinsic rate of population growth, *r*. Hendriks *et al.* (2005) performed a meta-analysis on many such data for different toxic compounds (both organic and inorganic pollutants), and on different species ($n = 128$ studies overall). They plotted the fraction *K*(treated)/*K*(untreated) against the fraction *r*(treated)/*r*(untreated) for each experiment. Again, there was a strong correlation explaining 48% of the variance between these relative measures *K* and *r*, with a slope of 0.94 (Fig. 3). Adopting these principles could have important conservation implications for understanding the population densities of toxin-sensitive species in polluted environments (Hendriks and Enserink, 1996).

In both the studies of Kuno (1991) and Hendriks *et al.* (2005), the slopes were not far from unity. Given that *r* represents the difference between the density-independent birth and death rates, we expect a reduction in *r* and a corresponding reduction in $K = r/\alpha$ when the death rate is increased or birth rate is decreased through toxin treatment, under equation (4), by the same factor, giving a slope of 1 when log *K* is plotted against log *r* (Kuno, 1991), or where the relative values of *K* are plotted against the relative values of *r* (Hendriks *et al.*, 2005).

Similar experiments with strains of bacteria or protists also gave strong positive correlations between *r* and *K* (Luckinbill, 1978, 1979; Fitzsimmons *et al.*, 2010). The close match of all these experiments with the expectation from the *r*-α model argues strongly that equation (2)

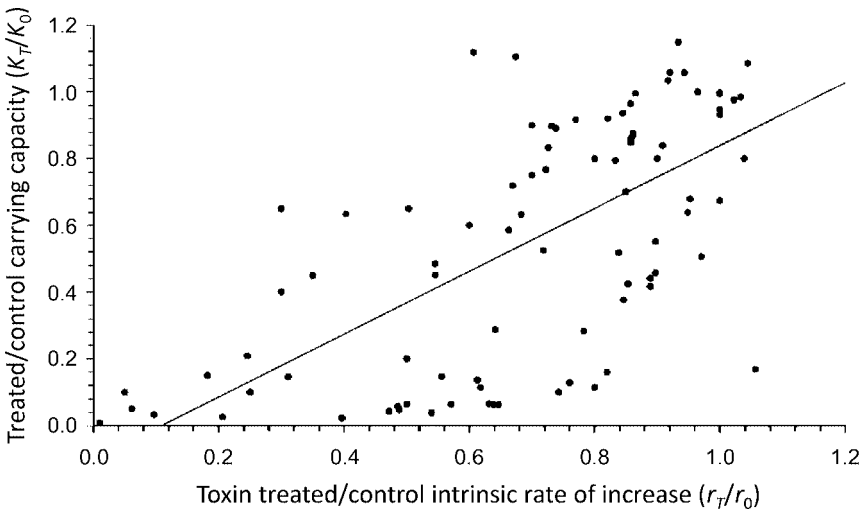

Fig. 3. Effect of toxin treatment on population growth parameters of aquatic species (Hendriks *et al.*, 2005). Reproduced with permission from John Wiley and Sons and the authors.

encapsulates the real mechanistic structure of the logistic better than equation (1), where K was assumed independent of r. (Note that this was in spite of likely trade-offs; see discussion of r- and K-selection below.)

NATURAL SELECTION AND LOTKA-VOLTERRA COMPETITION

Crow and Kimura (1970) made an important attempt to unify a theory of natural selection with Lotka-Volterra competition. Their main interest was to vindicate the constant-selection population genetic models of Fisher in continuous time, and those of Wright in discrete time. Both Fisher (1930) and Haldane (1924) had originally justified their selection models in demographic terms, but their treatments were sufficiently sketchy to lead to an assumption that both were treating natural selection only under the simplest assumption of exponentially growing populations. Fisher's treatment was certainly interpreted in this way by MacArthur (1962). However, it is clear that Fisher's fitness measure, his 'Malthusian parameter' (denoted R in this paper) included density-dependent per capita growth, and was not merely a constant r parameter (Fisher, 1930, pp. 42–46).

The simplest case of natural selection supposes that populations of haploid alleles, haplotypes, or clonal genotypes compete by growing at different rates. The goal here is to understand how changes in the frequency p of a focal haplotype or allele 1 emerges from population growth. By analogy with equation (4), we compare the per capita growth in density, n_1, of the focal haplotype 1 with that of all other haplotypes, n_2, in a population of total density $N = n_1 + n_2$:

$$\frac{1}{n_1}\frac{dn_1}{dt} = R_1 = B_1 - D_1 \tag{8a}$$

$$\frac{1}{n_2}\frac{dn_2}{dt} = R_2 = B_2 - D_2. \tag{8b}$$

Meanwhile, the entire population grows according to:

$$\frac{dN}{dt} = R_1 n_1 + R_2 n_2. \tag{8c}$$

The birth and death rates, 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 as in equation (4). Generalizing a result from Crow and Kimura (1970), who themselves acknowledged the methods of Leigh (1971), we find that the rate of change of haplotype frequency, p, is as follows (from Appendix 1, equation A1.4):

$$\frac{dp}{dt} = \{R_1 - R_2\} p(1-p). \tag{9}$$

$S = \{R_1 - R_2\}$ measures the strength of Fisherian natural selection. The values of R measure haplotype fitnesses, which depend, in general, on haplotype density because of their basis in density-dependent viability and fertility. If S depends on N but not p, we consider that the selection is density- but not frequency-dependent. If S varies with p, selection is frequency-dependent.

If $R_1 = r_1$ and $R_2 = r_2$ are constants as in the case of exponential population growth, then their difference is also a constant, so that selection, S, is by definition density-independent and frequency-independent. Interestingly, the haplotype frequency under this constant selection model grows as a logistic (Crow and Kimura, 1970) even when density is growing exponentially. Because equation (9) with constant r_1 and r_2 is itself a logistic equation, a logit transformation linearizes haplotype frequency evolution (Fig. 4A, C). However, constant selection can also occur when there is density-dependent regulation.

This is readily shown by substitution of Lotka-Volterra population dynamics into equation (9) (from equation A1.6 in Appendix 1), first shown by Smouse (1976):

$$\frac{dp}{dt} = \{r_1 - r_2 - [(\alpha_{11} - \alpha_{21})p - (\alpha_{22} - \alpha_{12})(1-p)]N\} p(1-p). \tag{10}$$

Classical density-independent and frequency-independent selection ('constant selection'; Fig. 4B, D) can result in density-regulated populations if the per capita density-dependent effects of each species on the other are the same as on its own species (i.e. if $\alpha_{11} = \alpha_{21}$ and $\alpha_{22} = \alpha_{12}$) (Appendix 2, case 2). The gene frequency trajectory is identical to that under exponential growth (cf. Fig. 4C, D), whether the population is near to or far from equilibrium (Fig. 4B). More generally, these conditions will not pertain, and natural selection will be both frequency-dependent and density-dependent (Smouse, 1976). However, the density-independent effects of r_1 and r_2 are in essence the 'main effects', and the effects of the α parameters are second-order, 'interaction effects' between haplotypes; it can be envisaged that differences in α values will tend to be smaller between haplotypes than the differences due to intrinsic birth and death rates that are components of r-values. If so, most evolution by natural selection within species may indeed be approximately independent of density and frequency.

Although equation (10) is relatively simple, a total of six parameters control population regulation and the strength of natural selection for two haplotypes, so the behaviour is correspondingly rich. Even this most basic, haploid model of natural selection in density-regulated populations can produce anything from constant selection (Fig. 4B, D), to frequency- and density-dependent natural selection, as well as 'hard' (Fig. 5) and,

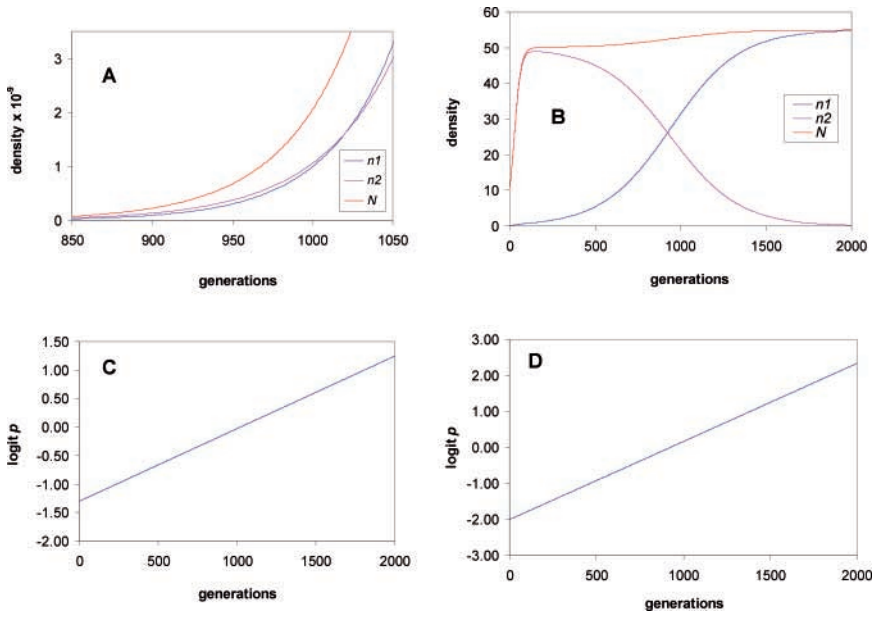

Fig. 4. Constant selection in exponentially growing and density-regulated populations. (A) Exponentially growing population: $S = r_1 - r_2 = 0.024 - 0.021 = 0.003$. Haplotype 1 is initially at low density, but eventually replaces haplotype 2. A segment of the trajectory where haplotype 1 begins to overtake haplotype 2 is shown. (B) Natural selection under generalized haploid Lotka-Volterra competition (equation 10), showing constant, density-independent selection: $r_1 = 0.055$, $r_2 = 0.05$, $\alpha_{11} = \alpha_{22} = \alpha_{12} = \alpha_{21} = 0.001$. (C) The replacement of haplotypes in the exponentially growing population (see A), on a logit frequency scale, $\log_{10}[p/(1-p)]$. The constant slope is $S/\log_e 10 = 0.0013$. (D) Replacement of haplotypes in a density-regulated population (see B), on a logit scale. Density-independent replacement continues throughout the trajectory, in spite of the switch between non-equilibrium to equilibrium population dynamics at approximately generation 100 (see B). The constant slope is $S/\log_e 10 = 0.0022$.

approximately also, 'soft' selection (Fig. 6) (Table 1, Appendix 2). Selection may increase or decrease equilibrium density (Fig. 5) or leave it relatively unaffected (Fig. 6), depending on parameter values.

Non-linear evolution of logit frequency (Figs. 5, 6) provides a useful definition of frequency-dependent selection. Also, I follow Christiansen (1975) in referring to soft selection as selection that does not alter population density; hard selection is selection that alters population density. Internal equilibria may exist, but stable polymorphism in a haploid model requires frequency-dependent selection; neither density-independent selection, nor density-dependent selection alone are sufficient to allow stable polymorphisms (Table 1, Appendix 2).

DIPLOIDY AND NATURAL SELECTION IN DENSITY-DEPENDENT POPULATIONS

The above results apply only to the simplest clonal organisms. In sexual diploids, there can be genic selection equivalent to haploid selection provided demographic parameters of heterozygotes are intermediate (Kimura, 1978). More generally, selection involves competition

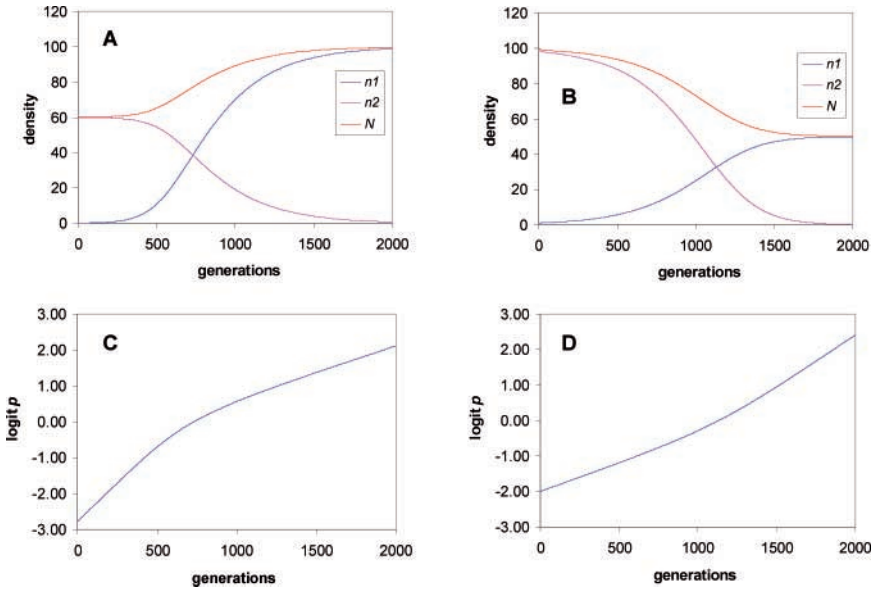

Fig. 5. Natural selection in density-regulated populations (equation 10). (A) Haplotype density where selection increases density: $r_1 = 0.10$, $\alpha_{11} = 0.001$, $r_2 = 0.03$, $\alpha_{22} = 0.0005$, and $\gamma_{12} = 1/\gamma_{21} = 1.5$. In Figs. 5 and 6, inversely related γ are used to give parallel isoclines, ensuring directional selection. (B) Haplotype density where selection decreases density: $r_1 = 0.03$, $\alpha_{11} = 0.0006$, $r_2 = 0.05$, $\alpha_{22} = 0.0005$, and $\gamma_{12} = 1/\gamma_{21} = 0.44$. (C) Replacement of haplotype frequency in (A). Initial rate of replacement is $(r_1 - \alpha_{12}r_2/\alpha_{22})/\log_e 10 \approx 0.0043$; final slope is $(r_2 - \alpha_{21}r_1/\alpha_{11})/\log_e 10 \approx 0.0014$ (see equation A2.5). (D) Replacement of haplotype frequency in (B).

among sexual diploids: at its simplest among three genotypes at a biallelic locus, A_1A_1, A_1A_2, and A_2A_2. In haploids, the only interactions were α parameters among haplotypes or alleles within populations. Diploidy introduces new potential interactions among mating individuals, as well as intra-genotype interactions due to dominance and heterosis, and variation among progenies of each genotype (Smouse, 1976). Births and deaths occur at different times during the life cycle, and can be affected by density dependence differently. All of these combine in complex ways with the purely competitive interactions discussed above for haploids (Appendix 3).

The difficulty of analysing diploid models has been exacerbated still further by opaqueness introduced by the *r-K* logistic formulation, and in many cases by additional complexity due to discrete generations. To save space, I briefly discuss earlier results for sexual diploids but do not deal with the mathematics. Selection among diploids has been explored both in continuous-time models (Kostitzin, 1939; MacArthur, 1962; Smouse, 1976; Desharnais and Costantino, 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 additional paradoxes and chaotic behaviour introduced by discrete time (Cook, 1965; Charlesworth, 1971; May, 1974; 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).

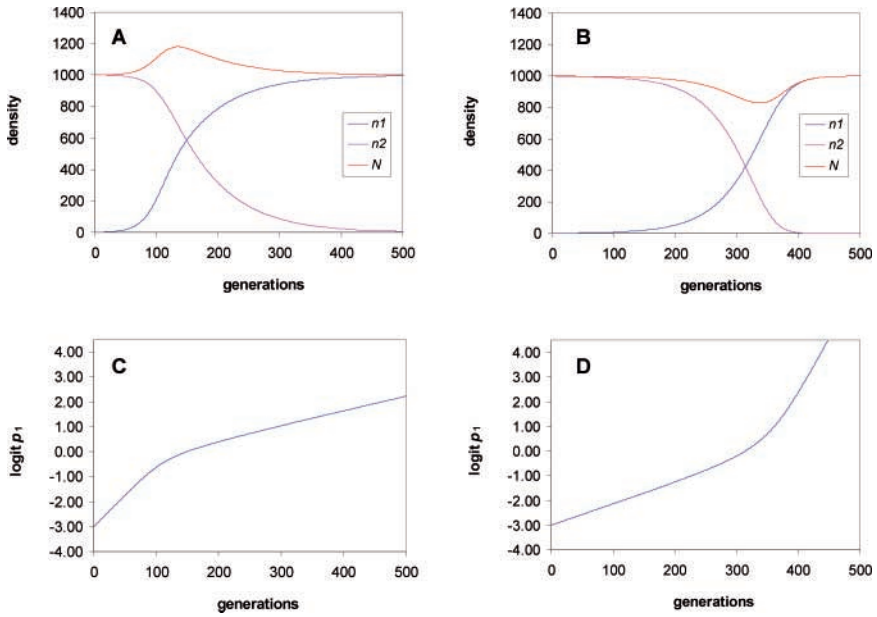

Fig. 6. Approximately pure frequency-dependent selection (equations 10 and A2.4). (A) No change in equilibrium density among haplotypes, but a temporary rise in population density occurs during replacement: $r_1 = 0.15$, $\alpha_{11} = 0.00015$, $r_2 = 0.02$, $\alpha_{22} = 0.00002$, and $\gamma_{12} = 1/\gamma_{21} = 0.6$. (B) No change in equilibrium density among haplotypes, but a temporary fall in population density occurs during replacement: $r_1 = 0.05$, $\alpha_{11} = 0.00005$, $r_2 = 0.15$, $\alpha_{22} = 0.00015$, and $\gamma_{12} = 1/\gamma_{21} = 0.6$. (C, D) Replacement of haplotype frequency in (A) and (B) respectively.

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 a Lotka-Volterra formulation to the assumption that $\gamma_{ij,kl} = 1 \; \forall \; ij \neq kl$ (where ij and kl each represent one of the diploid genotypes 11, 12, 22 in a diploid version of equation 6; see Appendix 3). This is purely density-dependent selection, equivalent to MacArthur and Wilson's K-selection (MacArthur and Wilson, 1967). Unlike the equivalent haploid/asexual model (Appendix 2, case 3), polymorphic equilibria are possible with selection on diploids. If heterozygotes are intermediate, $K_{11} \geq K_{12} > K_{22}$, then haplotype 1 replaces haplotype 2 (Fig. 7A, C), and vice versa for opposite signs. If there is heterosis (over-dominance, or higher fitness of heterozygotes) for K, i.e. $K_{11} < K_{12} > K_{22}$, stable polymorphisms result at equilibrium (Fig. 7B, D; the reverse inequality, under-dominance, gives an unstable polymorphic equilibrium) (Kostitzin, 1936, 1939; MacArthur, 1962). Equivalent heterosis under the r-α model, the diploid equivalent of equation (7), can be obtained by noting that $K_{ij} = r_{ij}/\alpha_{ij,ij}$ (Appendix 3). Purely competitive equilibria or declines in population size are not possible when $\gamma_{ij,kl} = 1 \; \forall \; ij \neq kl$ as in MacArthur's analysis (Appendix 2, case 3 for haploids), and in most other treatments of diploid density-dependent dynamics (Anderson, 1971; Roughgarden, 1971; Asmussen, 1983b). On relaxing the pure density-dependent assumption, much richer behaviour emerges, with combinations such as heterozygous advantage accompanied by equilibrium population decline possible (Fig. 7B, D). In her analysis of a discrete-time analogue, Asmussen (1983b) found up to four possible

Table 1. Types of selection possible in the haploid Lotka-Volterra model (equation 10)

Model type	Parameter dependence of S	Density (N)[a]- . . .	Frequency (p)[a]- . . .	Hard vs. soft[b] selection	Interior equilibria
'Constant selection', r-selection	r_1, r_2	-independent	-independent	hard	–
'Pure density-dependent selection'	$r_1 = r_2$ $\alpha_{11} = \alpha_{12}$ $\alpha_{22} = \alpha_{21}$	-dependent	-independent	hard	–
r- and K-selection[c]	$r_1 = r_2$ $\alpha_{11} = \alpha_{12}$ $\alpha_{22} = \alpha_{21}$	-dependent	-independent	hard	–
'Pure frequency-dependent selection'[d]	$r_1/\alpha_{11} =$ r_2/α_{22} α_{12}, α_{21}	-dependent (especially if far from equilibrium density)	-dependent	~soft	+
General model	$r_1, r_2, \alpha_{11},$ $\alpha_{22}, \alpha_{12}, \alpha_{21}$	-dependent	-dependent	hard	+

[a] Variables, N or p, of which the Fisherian selection term, S, is a function.
[b] Hard selection causes an alteration in N, whereas soft selection does not (Christiansen, 1975). Earlier definitions of these terms (Wallace, 1968; Saccheri and Hanski, 2006) are difficult to apply; Christiansen's distinction is particularly important where multiple populations interact, for instance in clines, because population density will influence fractional migration rates in and out of populations.
[c] *Sensu* MacArthur and Wilson (1967).
[d] General model, except that replacement occurs while $N \approx K_1 = K_2$ during evolution.

interior equilibria, of which two could be stable; however, complete analytical results were impossible to obtain.

HISTORY OF EQUATION (10)

The history of these discoveries is somewhat obscure, and is not found in textbooks. When writing their influential population genetics textbook, Crow and Kimura (1970) were concerned to justify classical population genetical constant-selection models in terms of density-dependent demography (Kimura and Crow, 1969; Crow and Kimura, 1970). The problem with doing this was that r-K Lotka-Volterra dynamics apparently excluded constant selection at density equilibrium (because r is not involved in equation 6; the competition paradox above). Thus it seemed that the intrinsic rate of increase, r, could not be involved universally in natural selection, and that all selection in density-regulated populations near equilibrium must therefore be density dependent.

MacArthur (1962, p. 1897) had earlier argued for the same reason that 'the carrying capacity of the environment, K, replaces fitness [i.e. Fisher's Malthusian parameter, r, which MacArthur interpreted as a constant] as the agent controlling the action of natural selection'. Crow and Kimura nonetheless argued for their most general case 4 (equation A2.2b) that the selection 'is in many cases . . . changing slowly': even though selection does change with density and gene frequency, it might be approximately constant for a while

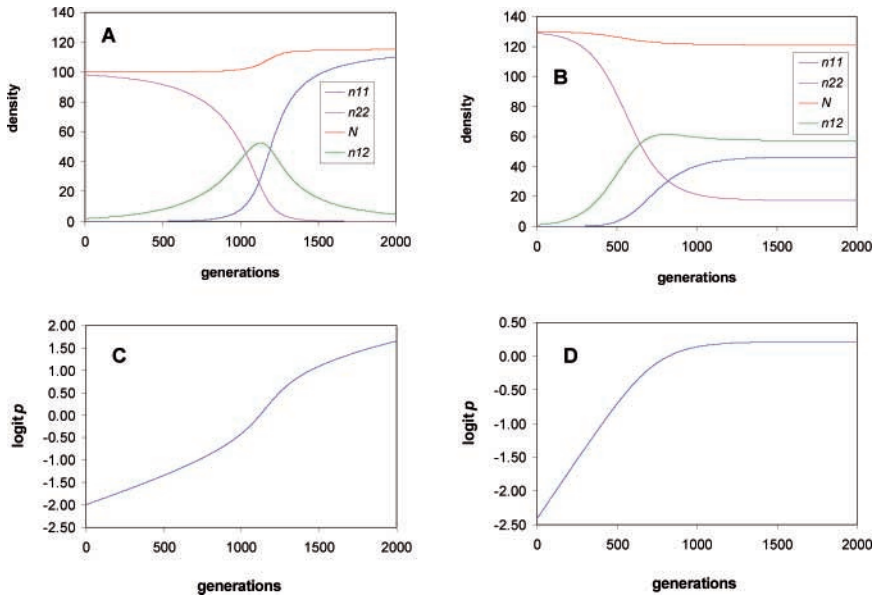

Fig. 7. Replacement of diploid alleles under selection, showing three-phased evolution of genotypes 11, 12, and 22 (equation A3.1). (A) Genotypic densities with classical density-dependent selection: $r_{11} = 0.02$, $r_{12} = 0.05$, $r_{22} = 0.20$, $\alpha_{11,11} = 0.00087$, $\alpha_{12,12} = 0.00027$, $\alpha_{22,22} = 0.00200$, and $\gamma_{ij,kl} = \alpha_{ij,kl}/\alpha_{ij,ij} = 1$ for all other i, j, k, l. (B) Genotype densities with generalized diploid selection, including frequency- and density-dependent selection: $r_{11} = 0.10$, $r_{12} = 0.03$, $r_{22} = 0.20$, $\alpha_{11,11} = 0.0002$, $\alpha_{12,12} = 0.00037$, $\alpha_{22,22} = 0.00154$, $\gamma_{11,12} = \gamma_{11,22} = \gamma_{12,11} = \gamma_{12,22} = 1.15$, and $\gamma_{22,11} = \gamma_{22,12} = 1/1.15$. (C, D) Haplotype frequency evolution of (A) and (B) respectively. In (C), approximate three-phase evolution is evident as each of the three genotypes replaces the previous one successively (see A).

[León and Charlesworth (1978) also made a similar argument]. Crow and Kimura were also able to find another special case (their case 2) of equations (6) where differences in r alone generated constant selection, but this required that density-dependent control was the same on each of the two haplotypes (in spite of differences in r). Because regulation of density involves parameters of both r and K, this would imply a peculiar form of density regulation in which the rate of increase of the second species, rather than just the density of that species, plays a role in regulating the density of the first species, and vice versa. This seems a rather artificial form of density control.

Furthermore, Crow and Kimura used a version of equation (7) (i.e. a simplified r-α model, their case 3) to show that mere differences in r would give rise to constant selection. However, Crow and Kimura regarded this Verhulstian logistic as 'weaker population control', and supposed that 'this situation is probably quite unusual in nature' compared with the r-K model, even though equation (7) merely represents a change of variables compared with equations (6).

In fact, Kostitzin (1936) had analysed a diploid r-α model of competition much earlier (equivalent to equation 10), and had briefly stated some major results, including that constant selection and heterozygous advantage were possible outcomes. However, Smouse (1976) was the first – and until this paper the only – person to state equation (10) explicitly, as well as to generalize this result to diploids and investigate its behaviour in detail. Most

population genetics texts, in contrast to that of Crow and Kimura (1970), completely ignore the problem that selection will generally occur in density-regulated populations, and that it will in general lead to frequency- and density-dependent selection. They instead appear to view density- and frequency-dependent selection as rather specialized areas of interest: for example, density-dependent selection (e.g. Hartl and Clark, 2007, p. 231) and frequency-dependent selection (Hartl and Clark, 2007, p. 230). Models of frequency-dependent selection are normally developed without any explicit demographic rationale, a tradition I myself have previously followed (Mallet, 1986; Mallet and Barton, 1989).

The puzzle of why Wrightian mean fitness is not maximized under frequency-dependent selection in such non-demographic models (Hartl and Clark, 2007) is related to the finding that densities can decline under frequency- and density-dependent selection (Table 1, Fig. 5). Under all scenarios, including these special cases, evolution climbs a Malthusian fitness 'hill' towards a state with the highest local instantaneous growth, R_i, at any one time. However, the fitness landscape itself changes during evolution and population growth, so that the end result is always $R = 0$ at equilibrium (Fisher, 1930; Metz, 2011).

An exception to general avoidance of the topic of density-dependent populations by recent population genetics texts is found in Barton *et al.* (2007, p. 468, their figure 17.15D, C). Like Maynard Smith and Crow and Kimura, Barton *et al.* were concerned to show how a constant-selection model could result in a demographic context. A graphical presentation was used to show that density-independent selection could readily result within density-regulated populations [in this case, the model demonstrated the case using a simplified discrete-time logistic growth equation (Ricker, 1954)]. Models of selection for host evolution of resistance to pathogens provide another exception (Koslow and DeAngelis, 2006). This model incorporates both host and pathogen population dynamics and results in an r-α model giving frequency-dependent selection, but this general approach seems not to have become a mainstream method in population biology.

Clearly, many mathematical population biologists fully appreciate the relationship of population dynamics to population genetics (Fisher, 1930; Kostitzin, 1939; Metz *et al.*, 1992; Christiansen, 2004; Meszéna *et al.*, 2005; Barton *et al.*, 2007). Nonetheless, I have yet to see these findings laid out clearly in any mainstream population genetics or population ecology text.

THE CONTROVERSY OVER DENSITY-DEPENDENT SELECTION: *r*- AND *K*-SELECTION

In 1967, MacArthur and Wilson, following on from MacArthur (1962), proposed that natural selection could be of two forms, '*r*-selection' and '*K*-selection', on the basis of an *r*-*K* model similar to equations (6) (p. 149):

> We have now replaced the classical population genetics of expanding populations, where fitness was *r*, as measured in an uncrowded environment, by an analogous population genetics of crowded populations where fitness is *K* . . .
> 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 favors 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 favors efficiency of conversion of food into offspring – there must be no waste.

See also MacArthur (1972, pp. 226–230) for elucidation. These inferences are approximately correct for selection-based derivations of Appendix 2, case 3, equation A2.3b. MacArthur's (1962) implicit assumption was that $\gamma_{12} = \gamma_{21} = 1$ in equation (6) (as in equation A2.3b), so that the crowding effect on each form by its competitor was the same as by its own species, giving what became the usual purely density-dependent selection assumption for diploids (Table 1, Appendices 2, 3). Intrinsic rates of increase, r, are apparently unimportant near equilibrium density, and the superior competitor is the form with the higher K. However, if densities are much lower than the equilibrium, as in newly arrived colonizer species, then the situation is similar to a pair of species competing under pure r-selection during exponential growth, and the form with the higher r will be superior.

However, under the r-α models of equations (7), (10), and (A2.3a), constant selection is possible regardless of density, with populations differing only in r and not in crowding parameters, α (Fig. 4; Appendix 2, case 1). Here, because the equilibrium $K = r/\alpha$, differences in either r or α control fitness as well as K (equation 9). The apparent replacement of Fisherian fitness, R, by the equilibrium density, K (MacArthur, 1962; MacArthur and Wilson, 1967), can be interpreted as due to this involvement of r in K as well as to the density-dependent control parameters, α.

The MacArthur-Wilson r- vs. K-selection ideas initiated a strong debate among ecologists and evolutionary geneticists after its publication. A postdoctoral associate of MacArthur's expanded the idea and argued that species could be classified as to whether they fell into r- or K-selected ecological syndromes (Pianka, 1970). MacArthur and Wilson's idea was based on a logistic-like model with instantaneous life history. They assumed a trade-off between r and K: selection for a high intrinsic growth rate r to cope with non-equilibrium conditions would reduce carrying capacity K; selection for competitive ability near equilibrium density would impinge negatively on reproductive rate. With Pianka's extension of the idea, r- and K-selected species became viewed as having alternative life-history strategies: r-selected species were those that bred early, had many, small offspring, and tended to be short-lived colonizing forms, generalists, and often semelparous; K-selected species bred late, were iteroparous, long-lived, had few, large offspring, dispersed little, and were characteristic of stable environments. Pianka's work was particularly influential via many editions of his evolutionary ecology textbook (Pianka, 1974).

The ideas of MacArthur and Wilson (1967) and extensions by Pianka (1970) had been presented as largely verbal arguments. Sewall Wright's discrete-generation population genetic models were then in vogue among population geneticists. The mathematical treatments of MacArthur and Wilson's ideas by Roughgarden (1971) and Anderson (1971) therefore employed discrete-generation Wrightian fitness: logistic growth Lotka-Volterra equations were formulated as per generation r-K difference formulations. In both cases, density was regulated by the total population density, leading to purely density-dependent fitnesses (as in equation A2.2). Both r and K could vary among genotypes. These assumptions are equivalent to setting $\gamma_{12} = \gamma_{21} = 1$ in a discrete-generation form of equation (6), or that $\alpha_{11} = \alpha_{12}$ and $\alpha_{22} = \alpha_{21}$ in equation (10). These investigations confirmed the basic findings of MacArthur and Wilson: in a stable environment, the allele specifying the highest value of K would prevail. In a harsh or seasonal environment that resulted frequently in a reduction of population size below carrying capacity, the allele with the higher r would dominate.

Experimental tests for trade-offs between r- and K-selection were often not successful. In Luckinbill's experiments that selected for higher growth rate under both low-density (r) and

high-density (*K*) conditions, there was no evidence for the expected trade-offs. In *Escherichia coli*, selection largely at density equilibrium (i.e. *K*-selection) led to the fitter strains overall, which also had higher *r* than populations selected during continued rapid growth maintained by dilution (*r*-selection) (Luckinbill, 1978). In *Paramecium*, selection for growth rate at low density (*r*-selection) led to populations with higher equilibrium densities (*K*) (Luckinbill, 1979). These results are of course expected if the carrying capacity, *K*, is a direct function of *r*, as expected from the *r*-α model of equation (2). This effect very likely explains why comparisons of strains within *Paramecium primaurelia*, as well as of species of ciliates in the genera *Paramecium*, *Tetrahymena*, and *Colpidium* all showed strong positive correlations between *r* and *K* (Luckinbill, 1979). Antibiotic-resistant mutant strains of *Pseudomonas fluorescens* also showed tight positive correlations between *r* and *K* (Fitzsimmons *et al.*, 2010). These experiments and comparative analyses all suggest strong, direct linkage between intrinsic rate of increase and equilibrium density that masks any interaction that may result from real trade-offs between *r* and α.

Long-term work by Laurence Mueller and Francisco Ayala did find some of the expected trade-offs in *Drosophila*, but these were not always as expected. In an early experiment, the growth rates of isofemale lines reared at low densities were positively correlated with their performances at higher densities, including at and above carrying capacity for the rearing conditions used (Mueller and Ayala, 1981). This provides no evidence of trade-offs between fitnesses at high and low density. However, with populations selected to grow at low and high densities, there did appear to be a slight trade-off with populations accustomed to high densities (*K*-selected strains) performing slightly better at high density than populations accustomed to low densities (*r*-selected strains), and vice versa (Mueller and Ayala, 1981). The existence of strong positive correlations in low- and high-density fitness across all strains are almost certainly due to the positive effect of *r* on *K* and on overall density-dependent fitness expected from equation (10); the evidence for differential fitness found for low- and high-density adaptation does point to the existence of some trade-off between *r* and α parameters in equation (10). Three decades of laboratory experimental work on selection in density-dependent populations and its relation to theory have been reviewed recently (Mueller, 1997, 2009).

Today, the ideas of *r*- and *K*-selection are still discussed in a few textbooks, but many now omit the topic altogether (Ricklefs, 2008) or disparage it (Gotelli, 2008). The recent volume celebrating the 40th anniversary of the publication of MacArthur and Wilson's *The Theory of Island Biogeography* (Losos and Ricklefs, 2010) makes no mention of *r*- and *K*-selection. This is astonishing: at the time of their first publication, *r*- and *K*-selection were viewed as among the most seminal and widely celebrated ideas to emerge from population ecology. Today, the testability of the idea seems limited, especially when extended into life-history theory (Stearns, 1977; Boyce, 1984; Mueller, 1997; Gotelli, 2008). A particularly clear account points out that the majority of life-history theory, including trade-offs among parameters at different life stages, can be developed focusing purely on *r*, with little account taken of density-dependent regulation (Reznick *et al.*, 2002). This is more valid under *r*-α models than it appears to be under *r*-*K* models of selection (e.g. equation A2.3b) because differences in *r* have strong effects on fitness whether populations are near or far from equilibrium density in equations (10) or (A2.3a) – indeed, it is identical to that in exponentially growing populations (Fig. 4).

DENSITY-REGULATED POPULATIONS AND ADAPTIVE SPECIATION

Two major principles underpinned Darwin's theory for the origin of species. The first was 'the struggle for existence', or ecological competition. The second was 'the principle of divergence', today called ecological character displacement. Darwin pointed out that divergence of character of a pair of species was necessary for coexistence, and his character displacement idea was the first theory of ecological – or adaptive – speciation. Darwin did not rule out other reasons for divergence (e.g. geographic isolation), but was mostly interested in explaining local biodiversity, which requires multiplication of species that can subsequently coexist in a single place and time.

It is a reasonable argument that the simplest haploid logistic models discussed here have very little applicability to the topic of speciation, at least in multicellular, sexual eukaryotes. For instance, one can have no 'reproductive isolation' with asexuals. Nonetheless, the principles outlined here underlie much more complex models for the evolution of ecologically divergent entities that differ at many loci and are able to coexist. This can lead to a form of speciation where divergent populations are able to overlap in space and time (Rosenzweig, 1978).

As with classical Lotka-Volterra competition (equation 7) or Lotka-Volterra haplotype frequency evolution among asexual haploids in a single panmictic population (equation 10), coexistence of two forms is stable only if haplotypes compete less strongly with each other than with their own type, so that $r_1/\alpha_{12} > r_2/\alpha_{22}$ and $r_2/\alpha_{21} > r_1/\alpha_{11}$ (see Fig. 2). This is equivalent to specifying that some frequency-dependent selection (Appendix 2, see Table 1) must exist for a new form to arise. Pure density-dependent selection or classical MacArthurian *r*- and *K*-selection (Appendix 2, see Table 1) cannot allow stable coexistence by a pair of species or haplotypes (except via heterozygote advantage in diploids). How near each form can be in terms of parameters can be investigated by means of models of 'robust coexistence' (Meszéna *et al.*, 2006). As well as applying to existing species, or haploid asexuals, these models apply in broad outline also to diploid sexual populations in the process of speciation, as in Dieckmann and Doebeli's (1999) and related 'adaptive dynamics', or 'evolutionary branching' models of speciation (Weissing *et al.*, 2011). These models assume that invasion of unoccupied niches allows greater opportunities for population growth, as in Darwin's principle of divergence, and require frequency-dependent and density-dependent selection (Table 1) for diversification to occur.

The adaptive landscape on which populations move as they change in genetic constitution will be variable. It can be shown that a single population evolves to a zone of minimal fitness, given that it exploits the optimal resources, and reduces these at equilibrium so that the Malthusian fitness, $R = 0$ (Metz, 2011). If evolutionary branching occurs, it will be due to peaks of fitness (unoccupied ecological niches) either side of this single population equilibrium, which create disruptive selection and each attract a part of the population (Metz, 2011; Weissing *et al.*, 2011). However, evolutionary branching models of speciation often incorporate few details of reproductive isolation and instead focus on ecological divergence as a means to achieve branching and coexistence of different forms. Thus they have been considered more applicable to the maintenance of polymorphism in asexual haploids than as models of speciation *per se*.

In contrast, classical population genetic models of ecological speciation rarely incorporate density regulation explicitly (Turelli *et al.*, 2001; Kirkpatrick and Ravigné, 2002; Gavrilets, 2004). Instead, they focus on evolution of reproductive barriers between emerging species, and

implicitly assume that only gene or genotype frequencies, rather than population density, need to be modelled. However, it has been known for a long time that even a crude implementation having constant disruptive selection in density-regulated populations (e.g. where a fixed total number emerge after a selection event in a particular ecological patch) can promote coexistence and polymorphisms of divergent ecologically adapted forms, or forms which choose particular ecological patches. The reason for this, in spite of constant selection within each patch, is that each form has a greater advantage when rare if it can occupy an unexploited niche, i.e. there is frequency-dependent selection overall (Levene, 1953; Rausher, 1984; Maynard Smith, 1998, pp. 70–74).

Similarly, one version of the resource distribution used in models of evolutionary branching has a fixed distribution of carrying capacity, K, along an ecological axis (Dieckmann and Doebeli, 1999). This seems to imply that density-dependent selection is involved. However, given that the intrinsic rate of increase is directly proportional to the equilibrium density, $K = r/\alpha$ at any point along the ecological axis, one might consider these carrying capacities to be due to differences in r, i.e. due to density-independent selection within any patch. This approach of considering local density-dependent regulation is also adopted in spatial models of the evolution of range size (Kirkpatrick and Barton, 1997; Polechová *et al.*, 2009). However, as in Levene's model, mixing of the offspring from multiple different ecological patches or localities renders the selection frequency-dependent overall, and allows branching to occur (Dieckmann and Doebeli, 1999; Weissing *et al.*, 2011).

Invasion of a new ecological patch and coexistence of divergently adapted phenotypes is clearly greatly favoured when one recognizes how strong disruptive selection in an invading form might be under density-dependent regulation, such as Lotka-Volterra competition. For example, the intrinsic Wrightian fitness differential of many species, proportional to e^{R1}/e^{R2}, may be very high, so that if a mutant (type 1) with adaptations to a hitherto unexploited ecological niche arose and invaded an empty ecological patch, it could easily have a $\gg 10\times$ advantage in per capita fitness. For example, with $n_1 \approx 0$:

$$\frac{1}{n_1}\frac{dn_1}{dt} = R_1 = r_1 - \alpha_{11}n_1 \approx r_1$$

compared with fitnesses of the parent population, where

$$\frac{1}{n_2}\frac{dn_2}{dt} = R_2 = r_2 - \alpha_{22}n_2 \approx 0,$$

since $\hat{n}_2 \approx r_2/\alpha_{22}$ at density equilibrium.

While this potentially huge fitness differential does not guarantee invasion, the counter-effects of population mixing may need to be extreme to prevent evolution of a new form. Clearly, the bias in fitness between the two forms is greatly amplified when density dependence is considered. In addition, in Dieckmann and Doebeli's (1999) model, the resource spectrum has fixed K. However, given the results in the current paper, each form i in essence creates its own local carrying capacity distribution, $K_i = r_i/\alpha_{ii}$, as adaptive alleles are recruited. Thus recruitment of alleles permitting higher r_i will be even more strongly favoured than would appear to be the case even under the model of Dieckmann and Doebeli.

Invasion possibilities will be enhanced still further if any reduction of mixing among patches occurs as an indirect result of this local adaptation (i.e. if the adaptation acts as

a 'magic trait') – for example, if patches are coarse-scaled relative to dispersal (Mallet, 2008). It is possible to imagine this process continuing further, recruiting more and more genes, until gene flow is reduced considerably, and the daughters form distinct clusters of genotypes, as in ecological races or incipient species (Drès and Mallet, 2002). Habitat choice (Rausher, 1984) and various forms of sexual selection or reinforcement (Kirkpatrick and Ravigné, 2002; Weissing *et al.*, 2011; Lenormand, 2012) may strengthen the differences and reduce mixing still further. In all these cases, it becomes clearer how ecological adaptation acts as an important early driver of speciation once a fuller understanding of the importance of density regulation in natural selection is reached.

Ecologists have made many advances in understanding ecological coexistence by basing their ideas on fixed species that cannot evolve, while incorporating simplified but useful models of density regulation such as Lotka-Volterra models. They have been interested in biodiversity, especially in the maintenance of local diversity (although these ideas are today extended geographically into global ideas of biodiversity and macroecology). However, ecological models rarely address the question of how new species form. In contrast, evolutionary biologists investigating speciation have tended to model natural selection while considering only gene frequencies within populations. Population genetics has often ignored density dependence, and ecological theories of coexistence. Ecological traits are often assumed to be based on constant selection differences (density- and frequency-independent selection). In models of speciation, the major focus of attention has been to understand reproductive isolation, or a complete splitting of two populations, rather than the evolution of ecological specialization that may lead to coexistence, with reduced mixing as a by-product. Today, there are signs of a change. There is a return to more Darwinian ideas, and a growing number of ecologists and evolutionary biologists view natural selection, ecological competition, and demography as a more unitary process (Rosenzweig, 1978; Metz *et al.*, 1992; Dieckmann and Doebeli, 1999; Schluter, 2000; Vellend, 2010; Metz, 2011; Schoener, 2011; Nosil, 2012). This is clearly the most fruitful approach for understanding adaptive speciation.

CONCLUSIONS

This article enquires to what extent misinterpretations and controversies might have been avoided if Verhulst's original polynomial formulation of the logistic equation, $dN/dt = rN - \alpha N^2$ (where α represents a 'crowding coefficient'), had instead been more widely appreciated. In this formulation, the equilibrium population density $K = r/\alpha$ becomes easily understood as a dynamic balance between density-independent and density-dependent parameters, rather than as a separate fixed parameter in its own right.

The polynomial r-α logistic is mathematically equivalent to the r-K logistic normally found in today's textbooks, but its simpler interpretation can clarify many issues. Some of the apparently paradoxical consequences of r-K formulations are readily and intuitively avoided. Furthermore, the r-α model can be derived readily from approximations to fundamental processes of energy and chemical flow. It also simplifies the relationship between interspecific competition and natural selection, and resolves one important difficulty with the MacArthur-Wilson formulation of r- and K-selection. A correct understanding of the compound nature of 'carrying capacity' as a population equilibrium can thus lead to a better understanding of the unity of ecology and evolution with speciation theory.

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APPENDIX 1: RELATIONSHIP OF GENE FREQUENCY EVOLUTION TO POPULATION GROWTH

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Evolution in a haploid population is defined as a change in the fractions of haplotypes, p, q, in a haploid population of size N (where $N = n_1 + n_2$, $p = n_1/N$, and $q = 1 - p = n_2/N$). How do changes in the numbers of each haplotype n_1, n_2, affect the fractions of haplotypes in the population over time? Here, we use methods developed earlier (Volterra, 1927; Crow and Kimura, 1970; Leigh, 1971; Smouse, 1976), and apply them to the most general case of Lotka-Volterra competition. 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

$$\frac{d\log_e x}{dx} = \frac{1}{x} \qquad \text{and} \qquad \frac{d[f(x)]}{dt} = \frac{d[f(x)]}{dx}\frac{dx}{dt},$$

this can be rewritten as:

$$\frac{d\log_e(n_1/n_2)}{dt} = \frac{1}{n_1}\frac{dn_1}{dt} - \frac{1}{n_2}\frac{dn_2}{dt} \tag{A1.1}$$

We are interested in evolution of the predominance of n_1 in a population.

$$\frac{d\log_e(p/q)}{dt} = \frac{d\log_e p}{dt} - \frac{d\log_e(1-p)}{dt}$$

$$= \frac{1}{p}\frac{dp}{dt} + \frac{1}{(1-p)}\frac{dp}{dt} = \frac{1}{p(1-p)}\frac{dp}{dt} \tag{A1.2}$$

Since

$$\frac{d\log_e(p/q)}{dt} = \frac{d\log_e(n_1/n_2)}{dt},$$

we can put (A1.1) and (A1.2) together:

$$\frac{1}{p(1-p)}\frac{dp}{dt} = \frac{1}{n_1}\frac{dn_1}{dt} - \frac{1}{n_2}\frac{dn_2}{dt} \tag{A1.3}$$

Substituting in the general formulation for allelic density growth (equations 8a,b) into (A1.3) gives:

$$\frac{1}{p(1-p)}\frac{dp}{dt} = [B_1(n_1,n_2) - D_1(n_1,n_2)] - [B_2(n_1,n_2) - D_2(n_1,n_2)] \tag{A1.4}$$

For logistic demography, we assume Lotka-Volterra competition among haplotypes (equation 7) in equation (A1.4):

$$\frac{1}{p(1-p)}\frac{dp}{dt} = r_1 - \alpha_{11}n_1 - \alpha_{12}n_2 - r_2 + \alpha_{22}n_2 + \alpha_{21}n_1 \tag{A1.5}$$

Substituting $p = n_1/N$ in (A1.5):

$$\frac{1}{p(1-p)}\frac{dp}{dt} = r_1 - r_2 - [(\alpha_{11} - \alpha_{21})p - (\alpha_{22} - \alpha_{12})(1-p)]N \tag{A1.6}$$

In contrast, using the *r*-*K* formulation we have:

$$\frac{1}{p(1-p)}\frac{dp}{dt} = r_1[1 - (n_1 - \gamma_{12}n_2)/K_1] - r_2[1 - (n_2 - \gamma_{21}n_1)/K_2] \tag{A1.7}$$

As with the logistic itself (see pp. 634–635), equation (A1.7) cannot readily be separated into density-dependent and density-independent parts, unlike equation (A1.6).

APPENDIX 2: SPECIAL CASES OF EQUATION (10)

Logistic regulation special case 1: 'density-independent selection', 'pure *r*-selection', 'constant selection'

If heritable differences among haploids affect only r_1 and r_2, all other parameters being equal (i.e. $\alpha_{11} = \alpha_{22} = \alpha_{12} = \alpha_{21} = \alpha$), equation (10) reduces to:

$$\frac{dp}{dt} = \{r_1 - r_2\}\, p(1-p) \tag{A2.1}$$

. . . in other words, identical to equation (9), but with constant R_1 and R_2. The haplotype frequency dynamics are identical to those of selection in a population with exponential growth, even though the population is now regulated to the average equilibrium density $[r_1 p + r_2(1-p)]/\alpha$ (Smouse, 1976). The haplotype with the higher r also has the higher equilibrium density, and will predominate in evolution. Evolution proceeds at a rate independent of density, however far either haplotype is from its equilibrium density. This is demonstrated by linear change in logit-transformed haplotype frequency (Fig. 4B, D). The same density-independent result is also true somewhat more generally if the per capita density-dependent effects of each species on the other are the same as the per capita effect of its own species on itself (i.e. if $\alpha_{11} = \alpha_{21}$ and $\alpha_{22} = \alpha_{12}$).

This result (Volterra, 1927; Leslie, 1957; Smouse, 1976) seems to have gone largely unrecognized (MacArthur, 1962; Roughgarden, 1971; Desharnais and Costantino, 1983; Asmussen, 1983a), because r is convoluted with K in the r-K formulation. As we have seen, with $K_1 = K_2 = K$ and $\gamma_{12} = \gamma_{21} = 1$ in equation (6), values of r have no effect on outcomes: coexistence of haplotypes is neutrally stable. The situation is given by equation (A2.2b; below) with $K_1 = K_2$ so that $dp/dt = (r_1 - r_2)(1 - N/K)p\,(1-p)$. At population equilibrium, $N = K$ and no gene frequency evolution is possible, although when $N < K$, the population will evolve to maximize r. Curiously, therefore, only density-dependent selection is possible under conventional r-K regulation, apparently making the most basic, Fisherian population genetic model impossible.

Crow and Kimura found one apparent exception, but this depends on their ruse of forcing density regulation of each haplotype to depend in part on the value of r of the other haplotype as well as on overall density N [case 2 in Crow and Kimura (1970, p. 27)]; this hardly seems justified. Even when recognized, the r-α logistic was characterized as 'weaker population control', a situation that 'is probably quite unusual in nature' [case 3 in Crow and Kimura (1970, p. 28)]. A suitable name for the current case is 'pure r-selection', which has a different meaning to MacArthur and Wilson's term when $n_i \gg 0$ (see below). Because α is constant, equilibrium density K can vary, and is directly proportion to r.

As in exponential growth, this pure r-selection is independent of density or 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; Table 1). Population geneticists often assume soft selection implicitly, and ignore population density. This is clearly not a valid assumption even in this, the most basic form of Fisherian natural selection in both density-regulated and unregulated populations.

Logistic regulation special case 2: 'pure density-dependent selection'

If selection is primarily controlled by heritable differences in crowding effects α (or equivalently in equation A1.7, variable *K*), but all other effects are again identical (i.e. $\alpha_{11} = \alpha_{12} = \alpha_1$ and $\alpha_{22} = \alpha_{21} = \alpha_2$ and $r_2 = r_1 = r$), equation (10) reduces to:

$$\frac{dp}{dt} = \{(\alpha_2 - \alpha_1)N\}\, p(1-p) \tag{A2.2a}$$

This is similar, again, to the classical constant selection model of equation (11), 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 *r*-*K* logistic (equation A1.7) appears less elegant [case 4 in Crow and Kimura (1970: 29)]:

$$\frac{dp}{dt} = \left\{ r \left[\frac{K_1 - K_2}{K_1 K_2} \right] N \right\} p(1-p) \tag{A2.2b}$$

This type of selection might be termed 'pure density-dependent selection', because there is no density-independent selection (involving *r*) or frequency-dependent selection (involving differences in between- and within-species population regulation). Like *r*-selection, density-dependent selection is independent of gene frequency and hard – it always increases equilibrium population size.

Logistic regulation special case 3: '*r*- and *K*-selection' *sensu* MacArthur and Wilson

A logistic interpretation of the intent of MacArthur's pioneering paper (MacArthur, 1962) and of the '*r*- and *K*-selection' verbal models of MacArthur and Wilson (1967) would allow both *r* and *K* to vary among haplotypes, while crowding is still effected by all haplotypes identically (i.e. $\alpha_{11} = \alpha_{12} = \alpha_1$ and $\alpha_{22} = \alpha_{21} = \alpha_2$). This combines special cases 1 and 2 and gives the expected combination of equations (A2.1) and (A2.2):

$$\frac{dp}{dt} = \{(r_1 - r_2) + (\alpha_2 - \alpha_1)N\}\, p(1-p) \tag{A2.3a}$$

When expressed according to the *r*-*K* formulation, the same equation appears clumsier:

$$\frac{dp}{dt} = \{(r_1 - r_2) - (r_1/K_1 - r_2/K_2)N\}\, p(1-p) \tag{A2.3b}$$

As with equation (A2.2), 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 α. Interior equilibria are impossible under either density-independent or density-dependent selection or their combinations in these haploid models, because Lotka-Volterra conditions for equilibria cannot be met (Fig. 2). This is the classical model of 'density-dependent selection' (e.g. Hartl and Clark, 2007, p. 231). A discrete-generation version of equation (A2.3b) was the one first used to analyse dynamics of diploid populations (Anderson, 1971; Charlesworth, 1971; Roughgarden, 1971). Similar results for density-independent selection, pure density-dependent selection and their combination (MacArthurian *r*- and *K*-selection) are also obtained in a model related to the *r*-α model of equation (10), differing only in that per capita density dependence is logarithmic rather than linear (Kimura, 1978).

Logistic regulation special case 4: 'pure frequency-dependent selection'

Suppose selection does not alter equilibrium density, i.e. $K_1 = K_2 = K = r_1/\alpha_{11} = r_2/\alpha_{22}$, while α_{12} and α_{21} are free to vary. This case does not simplify, and the mathematics are therefore given by the general equation (10). Although equilibrium densities remain the same on fixation of either variant, the dynamics of gene frequencies are as complex as in the general case. This type of selection might be termed 'pure frequency-dependent selection', or alternatively, 'α-selection' (Gill, 1974; Joshi *et al.*, 2001). An interesting additional simplification occurs when all solitary allelic growth parameters are identical, $r_1 = r_2 = r$ and $\alpha_{11} = \alpha_{22} = \alpha = r/K$, so that haplotypes compete only via differing α_{12} and α_{21} interactions, and equilibrium population density K does not evolve:

$$\frac{dp}{dt} = \{[(\alpha - \alpha_{12})(1-p) - (\alpha - \alpha_{21})p]N\}\, p(1-p) \tag{A2.4}$$

Even when density remains the same during evolution, we see a two-phase evolutionary process on a logit scale (equation A2.2, Fig. 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 (Fig. 6). Pure frequency-dependent selection is both effectively density-independent and frequency-dependent. Furthermore, because equilibrium density does not change, selection can be approximately 'soft'. Frequency-dependent selection emerges from these simple demographic models solely via differences in α_{12} and α_{21} interaction among haplotypes.

Interior equilibria are possible, both stable and unstable, and are given by the same conditions as for Lotka-Volterra competition (Fig. 2). A special case of interior equilibrium in equation (A2.4) occurs if $\hat{p} = (\alpha - \alpha_{12})/[(\alpha - \alpha_{21}) + (\alpha - \alpha_{12})]$. 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 equilibria, even when K does not differ among haplotypes.

Logistic regulation: general case of haploid selection

For the most general kinds of selection, evolution under equation (10) will consist of two approximately logit-linear phases (Smouse and Kosuda, 1977); when haplotype 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:

$$\frac{dp}{dt} \approx \frac{r_1(K_1 - \gamma_{12}K_2)}{K_1}\, p(1-p) = \left(r_1 - \frac{r_2\alpha_{12}}{\alpha_{22}}\right) p(1-p) \tag{A2.5a}$$

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

$$\frac{dp}{dt} \approx -\frac{r_2(K_2 - \gamma_{21}K_1)}{K_2}\, p(1-p) = -\left(r_2 - \frac{r_1\alpha_{21}}{\alpha_{11}}\right) p(1-p) \tag{A2.5b}$$

Provided that density equilibration is rapid compared with the rate of evolution, we expect initial evolution of a rare advantageous haplotype at one constant rate, followed by a shift during evolution to a different constant rate mode when the haplotype becomes more common, when plotted on a logit scale as in Figs. 5 and 6. Even with slow replacement evolution (and, therefore, weak selection), significant differences in r and α among

haplotypes will mean that replacement evolution is frequency-dependent and is no longer linearized by a logit transformation (Figs. 5 and 6). In equations (10) and (A2.4), stable or unstable polymorphisms are possible, as in generalized Lotka-Volterra competition. The condition for Lotka-Volterra coexistence (bottom right of Fig. 3) must be met for a polymorphic equilibrium to be stable, and that for unstable coexistence (bottom left of Fig. 3) for an equilibrium to be unstable (see Table 1).

Equation (10) 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 1.

APPENDIX 3: SELECTION IN LOTKA-VOLTERRA DIPLOID POPULATIONS

Diploid dynamics of Lotka-Volterra competition among haplotypes has never been treated in the most general cases. Instead, such analyses have been carried out under classical ideas of density-dependent selection with *r*-*K* formulation Lotka-Volterra-type models, usually with discrete-time Wrightian fitnesses (Anderson, 1971; Roughgarden, 1971; Asmussen, 1983b). The usual assumption is that individuals of all genotypes are equal with respect to density regulation on each genotype, leading to pure density-dependent selection, and no polymorphic competitive equilibrium possible (although heterosis may lead to stable equilibrium).

But interactions in simple Lotka-Volterra populations are potentially more complex, requiring a plethora of parameters and subscripts. For instance, if $\alpha_{ij,kl}$ is the crowding effect of genotype *kl* on genotype *ij*, then the usual density-dependent assumption means that $\alpha_{ij,kl} = \alpha_{ij,mn}\ \forall\ k, l, m, n$. To render more general Lotka-Volterra dynamics, although still accounting only for pairwise interactions at two haplotypes or alleles, one could use up to 12 parameters for the demographic model alone as follows:

$$\frac{1}{N_{11}}\frac{dN_{11}}{dt} = r_{11} - \alpha_{11,11}N_{11} - \alpha_{11,12}N_{12} - \alpha_{11,22}N_{22} \tag{A3.1a}$$

$$\frac{1}{N_{12}}\frac{dN_{12}}{dt} = r_{12} - \alpha_{12,11}N_{11} - \alpha_{12,12}N_{12} - \alpha_{12,22}N_{22} \tag{A3.1b}$$

$$\frac{1}{N_{22}}\frac{dN_{22}}{dt} = r_{22} - \alpha_{22,11}N_{11} - \alpha_{22,12}N_{12} - \alpha_{22,22}N_{22} \tag{A3.1c}$$

Mating among genotypes within these populations ensures that individuals often produce offspring unlike their own kind, so that equations (A3.1a–c) are not complete for predicting the dynamics without mating parameters. It is much easier to iterate these equations than to solve them for haplotype frequency (see Fig. 7, for the case of random mating).