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Theoretical Population Biology 72 (2007) 323–345
Theoretical Population Biology
www.elsevier.com/locate/tpb

Darwinian fitness

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Received 30 March 2006
Available online 5 July 2007

Abstract

The term Darwinian fitness refers to the capacity of a variant type to invade and displace the resident population in competition for available resources. Classical models of this dynamical process claim that competitive outcome is a *deterministic* event which is regulated by the population growth rate, called the Malthusian parameter. Recent analytic studies of the dynamics of competition in terms of diffusion processes show that growth rate predicts invasion success only in populations of infinite size. In populations of finite size, competitive outcome is a *stochastic* process—contingent on resource constraints—which is determined by the rate at which a population returns to its steady state condition after a random perturbation in the individual birth and death rates. This return rate, a measure of robustness or population stability, is analytically characterized by the demographic parameter, evolutionary entropy, a measure of the uncertainty in the age of the mother of a randomly chosen newborn. This article appeals to computational and numerical methods to contrast the predictive power of the Malthusian and the entropic principles. The computational analysis rejects the Malthusian model and is consistent with of the entropic principle. These studies thus provide support for the general claim that entropy is the appropriate measure of Darwinian fitness and constitutes an evolutionary parameter with broad predictive and explanatory powers.

Keywords: Darwinian fitness; Malthusian parameter; Evolutionary entropy; Finite size; Directionality theory

> The race is not to the swift nor the battle to the strong but chance and entropy, Nature's high arbiters, govern all.

1. Introduction

Evolutionary ecology in its broadest sense involves the integration of two distinct disciplines, with quite different mathematical cultures. One is population genetics, whose problematic is the dynamics of genetic change within a population due to mutation and natural selection. The second is population ecology which deals with changes in age distribution and changes in population size within a population as a consequence of the interaction of the organisms with the environment. One of the central concepts which has emerged in the rapprochement of these two disciplines is Darwinian fitness: the capacity of a variant type to displace the resident genotype in competition for the available resources.

Fitness, according to Darwin, means the capacity to survive and reproduce. This property includes a variety of behavioral factors—elements which are highly contingent on the environmental conditions that the organism experiences. In situations where competition involves the location of resources, fitness can be described by foraging ability; when the relative ability to evade predators is at issue, fitness may now involve visual acuity; in problems involving competition for mates, the capacity to intimidate rivals may now become the dominant trait. These behavioral features are highly qualitative predictors of net reproductive success. Consequently, they do not

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doi:10.1016/j.tpb.2007.05.004

provide a basis for understanding, in quantitative terms, the dynamics of invasion as new variants compete with the resident population for the available resources.

The problem of transforming Darwin's qualitative description of selective advantage into quantitative measures of fitness was first addressed by Fisher (1930). Fisher recognized that any index of competitive ability must incorporate demographic components—age-specific fecundity and mortality variables—as the operational units. A measurable macroscopic aggregate of individual fecundity and mortality variables is the rate of increase in population numbers. Accordingly, Fisher proposed this quantity as an index of Darwinian fitness and called it the Malthusian parameter, in honor of Malthus' contribution to the study of population dynamics. Fisher's proposition took hold and became the cornerstone of studies in evolutionary genetics and ecology (Roff, 1992; Stearns, 1992; Charlesworth, 1994). Although the Malthusian parameter continues to drive most theoretical and empirical studies of invasion dynamics in evolution and ecology, certain questions regarding its predictive and explanatory power have become prominent. We point to two issues—the first based on empirical, the second on theoretical considerations—that have been brought to bear on the pertinence of the Malthusian measure as an index of fitness.

The empirical issue concerns an extensive study of the invasion process in vertebrates and invertebrates in Britain (Lawton and Brown, 1986). The data indicate that the amplitude of population fluctuations, but not the population's intrinsic rate of increase, is the main determinant of invasion success. The empirical observations of Lawton and Brown are consistent with the claim that invasion is a highly stochastic process, and that the probability of establishment of an invader is correlated with body size. The studies furthermore indicate that the relation between invasion success and body size is dependent on the taxonomic status of the species. In large vertebrates, the probability of establishment of an invader increases with body size. However, the studies of insect orders show the opposite trend, with probability of establishment decreasing with increasing body size.

The theoretical issue concerns the analysis of the intensity of selection, a property measured by the sensitivity of demographic measures of Darwinian fitness to changes in the age-specific fecundity and mortality schedule (Hamilton, 1966). Perturbation studies using continuous models (Hamilton, 1966), have shown that the sensitivity of the Malthusian parameter to changes in the life-history variables is a decreasing function of age. This analytical fact has important implications for studies of the evolution of aging. The sensitivity property entails that, in the case of Malthusian models, evolution by natural selection will result in mortality rates that increase exponentially with age (Partridge and Barton, 1996; Rose and Mueller, 2000). This condition, however, is known to be inconsistent with empirical data for human and several laboratory populations (Carey and Judge, 2000). In human populations, for example, a simple exponential curve—the Gompertzian distribution—provides a good fit for the mortality data for most populations from age 35 to 95. However, after age 95, mortality rates decelerate or abate with age, defining what is called a mortality plateau.

The problems raised by these empirical and theoretical issues were addressed in a series of articles which pointed to the anomalies which result when the Malthusian parameter was used as a measure of Darwinian fitness. This critique of the Malthusian principle led to the study of a new class of population models which exploited the methods of statistical mechanics to generate new macroscopic descriptors of age-structured populations. We appealed to these parameters to analyze the invasion dynamics of variant types introduced in a resident population. This analysis, in sharp contrast to the classical models of competition (see, for example, Charlesworth and Williamson, 1975; Pollak, 1976), showed that the probability of establishment of a variant type is contingent on the prevailing ecological conditions and is determined by the rate at which the population returns to its original size after a random perturbation in the individual birth and death rates. This return rate, called the fluctuation decay rate, characterizes the robustness of the population, that is, the capacity of the population to maintain its steady state condition in the face of random perturbations in the life-history variables. Robustness can be analytically described by the demographic parameter, evolutionary entropy, a measure of the uncertainty in the age of the mother of a randomly chosen newborn (Demetrius, 1974; Demetrius et al., 2004).

Our analysis of the invasion process distinguishes between (i) *equilibrium* species, typically large vertebrates and perennial plants, which refer to populations which are either stationary or fluctuating around some constant size, (ii) *opportunistic* species, typically insects, small vertebrates and annual plants, which pertain to populations which undergo large irregular fluctuations in size (cf. Pianka, 2000; Emlen, 2004).

Studies based on diffusion processes showed that invasion success is modulated by the condition—equilibrium or opportunistic—that defines the population, and can be qualitatively described in terms of the following rules (Demetrius, 1997; Demetrius and Gundlach, 1999):

A(i) *Equilibrium species*: Variants with *increased* entropy will almost always invade, variants with *decreased* entropy will almost always become extinct.

A(ii) *Opportunistic species*:

(a) *Large population size*: Variants with *decreased* entropy will almost always invade; variants with *increased* entropy will almost always become extinct.

(b) *Small population size*: Variants with *decreased* entropy will invade with a probability that increases with population size, variants with *increased* entropy will become extinct with a probability that increases with population size.

The invasion criteria pertain to *local* changes in entropy. These criteria are the cornerstone of directionality theory, an analytic evolutionary model which studies *global*, that is, long run changes in entropy. Directionality theory considers evolution as a dual process. The first phase consists of the production of genetic variability through mutation and the invasion of these mutants in the resident population. The second phase consists of the ordering of this genetic variability through natural selection under various types of ecological constraints. The integration of these two phases leads to the replacement over time of one population type by another, with concomitant changes in life-history.

Directionality theory predicts the following relations between the equilibrium-opportunistic condition and global changes in entropy as the system evolves from one steady state to the next.

B(i) *Equilibrium species*: A uni-directional increase in entropy.
B(ii) *Opportunistic species*: A uni-directional decrease in entropy, when population size is large; and random, non-directional change in entropy when population size is small.

Directionality theory has been shown to have a wide explanatory and predictive power, as indicated by the computational studies reported in Kowald and Demetrius (2005), and the empirical studies using plant populations described in Ziehe and Demetrius (2005), and human populations analyzed in Demongeot and Demetrius (1989), Demetrius and Ziehe (1984). This evolutionary theory is the crux of a new class of models which deals with the origin and evolution of senescence (Demetrius, 2003) with important empirical implications (see, Olshansky and Rattan, 2005; Braeckman et al., 2006; Spinney, 2006).

The invasion criteria, as qualitatively expressed by (A), elucidate the observation, due to Lawton and Brown, that body size is a critical determinant of invasion success. The rationale for this observation rests on the analytical and empirical fact that entropy is related to body size (Demetrius (2000)). This relation entails that in equilibrium species, the probability of establishment of an invader will increase with body size, whereas in opportunistic species, the probability of establishment will decrease with body size.

The directionality principles for evolutionary entropy, as qualitatively summarized in (B), account for certain life-history patterns observed in studies of longevity. The principles explain the empirical observation that the mortality rates in human populations are not described by a Gompertzian distribution—as predicted by the Malthusian invasion condition—but by a so-called mortality plateau. The argument is based on the following fact: *the sensitivity of entropy to changes in the age-specific fecundity and mortality variables is a convex function of age* (Demetrius, 2001). The convexity property entails that the response of entropy to changes in the net-fecundity distribution will be relatively strong during the earlier and later stages of the reproductive phase, but relatively weak during the intermediate stages. Since evolution under mutation and natural selection will result in increased entropy in equilibrium species, and, *typically*, decreased entropy in opportunistic species, we can predict that: (i) in equilibrium species (humans and certain laboratory organisms, for example) mortality rates will abate with age at advanced ages, thus inducing a mortality plateau, and (ii) in opportunistic species (small vertebrates in the wild and insects) morality rates will increase exponentially with age, thus defining a Gompertzian distribution.

The entropic criteria for invasion success is a generalization of the Fisherian models, which deals with invasion dynamics in structured populations of infinite size, and the Wright–Kimura models, which pertain to invasion processes in non-structured populations of finite size.

The synthesis of the Fisher and the Wright–Kimura models has been shown to invoke new population concepts such as entropy, reproductive potential, and demographic variance, etc. These concepts have their mathematical origin in ergodic theory and statistical thermodynamics. Their emergence in a population context derives from the recognition, articulated in Demetrius (1974, 1975), that the steady state dynamics of structured populations—in which individuals of different ages or size "interact" according to birth and death processes—and the equilibrium dynamics of material aggregates (solids, liquids or gases)—in which the individual molecules "interact" according to the laws of classical mechanics—can be analyzed in terms of a similar mathematical formalism. The analytical basis of this formalism has been extensively developed in articles addressed primarily to mathematicians (see Demetrius, 1983; Arnold et al., 1994; Demetrius and Gundlach, 1999). This article aims to complement certain aspects of these mathematical studies with a computational analysis of the invasion process.

Our objectives are two-fold.

(1) To exploit the computational methods to explain the interdependence of the various demographic parameters which determine invasion success.
(2) To exhibit in numerical terms the relation between the entropic invasion criteria and the Malthusian condition.

We should point out at this juncture that certain deficiencies of the Malthusian parameter as a measure of invasion success has been recognized by various research groups (see, for example, Metz et al., 1996; Champagnat et al., 2001; Ferrière et al., 2004). The arguments reviewed in Metz et al. (1996), and Ferrière et al. (2004), for example, aim to integrate the effects of environmental conditions with different forms of density-dependence to provide general criteria for invasion success that go beyond

the Malthusian condition. These analyses are in large measure developed in terms of the same analytical and conceptual structure as the classical Malthusian models. Hence, they are not directly pertinent to the problems of invasion dynamics addressed in this article.

This paper is organized as follows. The origin of the Malthusian parameter and its significance as an invasion exponent are described in Section 2. The origin of the entropy concept, its conceptual and empirical basis, and its relation with the Malthusian parameter are reviewed in Section 3. The ideas in this section have been developed more extensively in earlier publications and are here included to make the paper self-contained and more accessible to empiricists. The characterization of entropy as an invasion exponent is developed in Sections 4 and 5. In Section 6 we compare the diffusion equations which arise in structured models with analogous equations derived in the Wright–Kimura models. The computational studies of the entropic principle and its relation to the Malthusian principle are developed in Sections 7–9. Section 10 summarizes the main results.

2. The Malthusian parameter

The Malthusian parameter derives from Lotka's (1925) model of the population dynamics of age-structured populations. He showed that a population with fecundity and mortality variables which are continuous functions of age will ultimately attain a growth rate, r, which is the real root of the equation,

$$1 = \int_0^\infty \exp(-rx) V(x)\, dx. \tag{1}$$

Here $V(x)$ is the net reproductive function which is given by the product, $l(x)$, the probability that an individual survives to age x, and $m(x)$, the mean number of offspring produced in the age interval x to $x + dx$.

The analysis of competition between mutant types and a resident population in terms of the Lotka model was pioneered by Fisher (1930). Elaborations of this model by Charlesworth and Williamson (1975), Pollak (1976) have led ultimately to the claim that the invasion dynamics of a mutant allele is a deterministic process whose outcome is predicted by the Malthusian parameter. Hence, the selective advantage, s, is given by

$$s = \Delta r. \tag{2}$$

Table 1
Invasion criteria: Δr

Demographic condition	Selective outcome
$\Delta r > 0$	Mutant invades
$\Delta r < 0$	Mutant becomes extinct

Here Δr denote the difference in the Malthusian parameter between the variant and the resident type. The invasion criterion can be expressed in terms of Table 1.

The Malthusian principle has been extended to more general situations involving populations evolving in stochastic environments (see, for example, Ferrière and Gatto, 1995). These extensions essentially assert that the invasion exponent is described by some measure of growth rate in population numbers. This measure is given by the dominant Lyapunov exponent of a matrix sequence, a quantity which is analogous to the dominant eigenvalue of the Leslie matrix in classical population models. Accordingly, these developments can be subsumed under the criteria given in Table 1.

The Malthusian principle is based on models which implicitly assume that populations have infinite size. This constraint on size entails that random variations in the individual birth and death rate—demographic stochasticity—will have a negligible effect on the population dynamics. Hence, the establishment of a rare mutant will be a *deterministic* process regulated by the population growth rate.

This situation no longer prevails when size is finite. In this case, demographic stochasticity will induce irregular fluctuations in population numbers. The number of descendants left by an individual will now become a random variable. Accordingly, the establishment of a rare mutant now becomes a *stochastic* process regulated by several parameters: the population growth rate, the population size, and the demographic variance, that is the variance in the net-reproductive function (Demetrius, 1997; Demetrius and Gundlach, 1999).

3. Entropy and the reproductive potential

The Malthusian parameter derives from models which parametrize structured populations in terms of their age distribution and analyses changes in the age-distribution over time. It is the rate of increase of total population numbers when the population attains the stable age-distribution (Lotka, 1925; Leslie, 1945). A new class of macroscopic descriptors of structured populations was derived by considering models in which populations are parametrized in terms of their "genealogies" (Demetrius, 1974). This term refers to a recording of successive ancestors of a particular individual which at time zero is in a particular age-class. A central result of this new theory is the following analytical fact: *The population growth rate satisfies a variational principle which is formally analogous to the extremal principle for the free energy in equilibrium statistical mechanics* (Demetrius, 1974; Arnold et al., 1994). This variational principle implies that the population growth rate r, as defined in Eq. (1), can be expressed as the sum of two macroscopic variables, namely,

$$r = H + \Phi. \tag{3}$$

The quantities H and Φ are given by

$$H = -\frac{\int_0^\infty p(x)\log p(x)\,dx}{\int_0^\infty xp(x)\,dx} \equiv \frac{S}{T};$$
$$\Phi = \frac{\int_0^\infty p(x)\log V(x)\,dx}{\int_0^\infty xp(x)\,dx} \equiv \frac{E}{T}, \quad (4)$$

where $p(x) = \exp(-rx)V(x)$.

The expression $T = \int_0^\infty xp(x)\,dx$ is the generation time, the mean age of mothers at the birth of their offspring.

Hence Eq. (3) can also be expressed in terms of the identity

$$rT = S + E.$$

We will now describe and illustrate with empirical data the main properties of the demographic parameters defined in Eq. (4).

3.1. Demographic entropy

The quantity $S = -\int_0^\infty p(x)\log p(x)\,dx$ is a measure of the uncertainty in the age of the mother of a randomly chosen newborn. It is called demographic entropy and describes the degree of iteroparity of the population. Large values of S correspond to the following life-history properties: late age of sexual maturity, small net progeny sets, broad reproductive span. Small values of S define: early age of sexual maturity, large net progeny sets, narrow reproductive span (Demetrius, 2003).

A property which has played an important role in studies of life-history patterns is the so-called fast–slow continuum (cf. Promislow and Harvey, 1990). Species that mature early have large reproductive rates, and short generation times are said to occupy the "fast" end of the continuum. Species with the opposite suite of traits occupy the "slow" end of the continuum. The following analytical fact derives from perturbation studies of the life-history variables: *Demographic entropy quantifies the position of a population along the fast–slow life-history continuum.*

Small mammals and herbs in disturbed habitats (low entropy species) will occupy the fast end, whereas large mammals and trees (high entropy species) will occupy the slow end (see Ziehe and Demetrius, 2005).

Table 2 illustrates the entropic parametrization of the fast–slow continuum by considering the life-history pattern of a large mammal (high entropy), a medium sized mammal (intermediate entropy), and a small mammal (low entropy).

3.2. Evolutionary entropy

The quantity $H = S/T$ has the dimension of inverse time. It is called evolutionary entropy or entropy rate to distinguish it from S, the demographic entropy. The entropy rate H provides an analytic characterization of the robustness of a population. Robustness, in its broadest sense, describes the capacity of a population to maintain its phenotypic characteristic in the face of random perturbations in the genotypic states, a notion whose general biological significance was emphasized by Waddington (1959). In the demographic context, we use the term to refer to the capacity of a population to maintain its predicted steady state condition in the face of random perturbations in the age-specific birth and death rates. The perturbation in this model derives from demographic stochasticity and is thus distinct from variations in population observables induced by environmental factors.

Analytically, the notion robustness, denoted R, is described by Demetrius et al. (2004):

$$R = \lim_{n\to\infty}\left(-\frac{1}{n}\log\, Q_n(\varepsilon)\right).$$

Here $Q_n(\varepsilon)$ is the probability that the sample mean of some population observable, defined at instant n, differs from the asymptotic mean by more than ε.

By appealing to the theory of large deviations, we showed (Demetrius et al., 2004) that

$$\Delta H \Delta R > 0. \quad (5)$$

The quantities ΔH and ΔR denote changes in the macroscopic parameters H and R induced by a change in the age-specific fecundity and mortality variables.

R describes the rate of convergence to the steady state condition after a random perturbation. It is negatively correlated with the intensity of fluctuations, a property which can be measured by the coefficient of variation in population size. In view of the fluctuation-stability theorem, as expressed by Eq. (5), the entropy rate H, will also be negatively correlated with the coefficient of variation in population size: the larger the entropy rate, the smaller the coefficient of variation. Since changes in demographic entropy S, and changes in the entropy rate H are positively correlated (Demetrius, 2001), we can infer that demographic entropy and the coefficient of variation in population size will be described by a similar property. Empirical support for the correlation between

Table 2
Relation between life-history properties and demographic entropy

Species	Body size (kg)	Age of sexual maturity (years)	Litter size	Age of last reproduction (years)	Demographic entropy	Reference
Spermophilus armatus	0.35	1	5	4	0.870	Slade and Balph (1974)
Ovid canadensis	55	2	1	8	1.835	Deevey (1947)
Connochaetes taurinus	170	3	1	20	2.315	Watson (1970)

demographic entropy and the coefficient of variation in population size was described in Ziehe and Demetrius (2005) using data from plant populations.

3.3. The reproductive potential

The quantity E, and likewise the parameter Φ, can assume both positive and negative values. E, called the net-reproductive index, describes the net-offspring production $\log V(x)$, averaged over all age classes. The quantity $\Phi = E/T$ has the dimension of inverse time and is called the reproductive potential. We now provide arguments to support the following characterization of the reproductive potential. *The reproductive potential* Φ *quantifies the position of a population along the equilibrium-opportunistic axis.*

We observe from Eq. (3), that

$$\Phi = r - H. \tag{6}$$

Hence,

$$\Phi < 0 \implies r < H; \quad \Phi > 0 \implies r > H. \tag{7}$$

Equilibrium and opportunistic populations are qualitative notions which originate from the so-called $r-K$ theory of life-history evolution (Pianka, 2000). Equilibrium species, we recall, are defined as populations whose growth rate is either stationary or slowly growing (r small). These populations are described by small fluctuations in population numbers, and rapid return to the steady state condition after a random perturbation in the age-specific birth and death rate (H large). In view of Eq. (7), we conclude that equilibrium species will be described by the condition $\Phi < 0$.

Opportunistic species are defined by populations which are subject to relatively long episodes of rapid population growth (r large) followed by an abrupt decline in population numbers. In these systems, populations undergo large irregular fluctuations in numbers due to the effects of demographic stochasticity (H small). Accordingly, we have from Eq. (7) that $\Phi > 0$.

We give in Table 3 values for r, H, and Φ for selected examples of equilibrium species—three large mammals and a large bird; and opportunistic species—a small mammal, two insects and a small bird.

We should point out that the values of the demographic variables given in Table 3 are regulated by the scaling units—weeks, months or years—which are invoked in generating the various life tables. However, the sign of Φ does not vary with the scaling, which is a necessary requirement for the significance of Φ as a measure of the equilibrium—opportunistic distinction.

The significance of the equilibrium—opportunistic distinction can be underscored by appealing to empirical studies of demographic changes in human populations over a relatively long time period—Sweden (1778–1965), France (1851–1965) (see Demetrius and Ziehe, 1984; Demongeot and Demetrius, 1989). Table 4 gives the values for r, H, and Φ for Sweden and France at selected points over a 200 year and 100 year period, respectively.

The values of the demographic variables described in Table 4 indicate that although the growth rate r showed a large variation—with negative and positive values—the reproductive potential remained negative. Hence, over a period of two centuries, gradual genetic and cultural changes, together with the punctuated changes in demographic patterns induced by two world wars, have resulted in no variation in the sign of the reproductive potential. Humans are a typical example of an equilibrium species. The hunter-gathering phase represents 99% of human evolutionary history. During this period, population

Table 4
Trends in demographic variables for human populations: Sweden (1778–1965), France (1851–1965)

Year	r	H	Φ
For Sweden			
1778	0.0062	0.0493	−0.0431
1828	0.0093	0.0481	−0.0385
1878	0.0107	0.0485	−0.0370
1928	−0.060	0.0520	−0.0580
1965	0.0048	0.0528	−0.0480
For France			
1851	−0.002	0.0502	−0.0504
1871	−0.0087	0.0516	−0.0603
1909	−0.0017	0.0524	−0.0540
1945	0.0049	0.0504	−0.0456
1965	0.0096	0.0506	−0.04810

Table 3
Demographic variables for equilibrium and opportunistic species

Taxon	Species	Body size (g)	r	H	Φ	References
Large mammal	*Equus burchelli*	250,000	0.05	0.0343	−0.288	Spinage (1970)
Large mammal	*Cervus elaphus*	175,000	0.058	0.44	−0.38	Lowe (1969)
Large mammal	*Ovis canadensis*	57,900	0.088	0.45	−0.362	Geist (1968)
Large bird	*Gyps fulvis*	9000	0.087	0.257	−0.168	Niel and Lebreton (2005)
Small mammal	*Tamias striatus*	100	0.962	0.602	0.360	Spinage (1970)
Small bird	*Petrona petrona*	35	0.765	0.652	0.113	Niel and Lebreton (2005)
Insect	*Calandra oryzae*	0.01	0.843	0.01	0.61	Birch (1948)
Insect	*Tribolium castaneum*	0.01	1.26	0.27	0.01	Leslie and Park (1949)

growth rate has ranged between 0.007 and 0.0015 per thousand per year. The large increases in growth rate (0.36 per 1000), at the advent of agriculture 10,000 years ago, and (0.56 per 1000) since 1750, represent only 1% of human evolutionary history (Coale, 1974). The negative values of Φ for Sweden and France, given in Table 4, are typical for modern human populations. Computational studies of the life tables of agricultural and hunter-gatherer populations also yield negative values for Φ. These observations indicate the robustness of the reproductive potential as a quantitative measure of the distinction between equilibrium and opportunistic species.

4. Invasion dynamics: the entropic condition

Diffusion equations were applied to analyze models of competition between a resident population and a mutant (Demetrius and Gundlach, 1999). The resident population is described by the parameters r, H, and Φ, the mutant by r^*, H^*, and Φ^*. The analytical studies show that the outcome is determined by ΔH, where $\Delta H = H^* - H$. However, whether an increase or a decrease in entropy confers a selective advantage, depends on the condition, equilibrium ($\Phi < 0$), or opportunistic ($\Phi > 0$) which describes the population.

The invasion criteria summarized in Table 5 are derived from analytical studies which integrate the ergodic theory of dynamical systems with diffusion processes (Demetrius, 1997; Demetrius and Gundlach, 1999). The conditions described in Table 5 can be explained by a qualitative and heuristic argument. The thrust of the argument revolves around two items:

(i) The characterization of entropy as a measure of robustness, that is, the capacity of a population to maintain a steady state condition in the face of random perturbations in the birth and death rates.
(ii) The representation of the equilibrium—opportunistic condition in terms of constraints on the disposition of the resources. The equilibrium state defines species subject to limited but constant resources, whereas the opportunistic state refers to species subject to abundant but variable resource conditions.

Table 5
Invasion criteria: ΔH

Demographic constraints	Invasion condition	Selective outcome
$\Phi < 0$ (equilibrium species)	$\Delta H > 0$	Invasion occurs almost surely (a.s.)
	$\Delta H < 0$	Extinction occurs a.s.
$\Phi > 0$ (opportunistic species)		
Large population size	$\Delta H < 0$	Invasion occurs a.s.
	$\Delta H > 0$	Extinction occurs a.s.
Small population size	$\Delta H < 0$	Invasion with a probability increasing in population size
	$\Delta H > 0$	Extinction with a probability increasing in population size

Our qualitative argument is based on the observation, first noted in Lotka (1922) (see also Watt, 1986; Brown et al., 1993; Parsons, 2005), that, in competition for the available resources, selective advantage accrues to organisms who are most efficient in acquiring resources and converting the metabolic energy into viable offspring.

Efficiency in resource acquisition and resource conversion have differential effects on the invasion success of a mutant. When resources are limited but constant, the rate at which the organism acquires resources becomes the limiting factor with regards to the efficient direction of the available energy into net-reproductive power. However, when resources are abundant but variable, the rate of conversion of the resources into net-offspring production now becomes limiting (see, for example, Brown et al., 1993).

Now in the case of equilibrium species, resources are limited but constant, and population size will remain relatively stable. Selective outcome under these conditions will be determined by efficiency in resource acquisition. This property will be positively correlated with the robustness of the population. Mutants with *increased* robustness, and consequently increased entropy, will acquire resources at a faster rate than the ancestral type. Such mutants will have a selective advantage and hence increase in frequency.

In the case of opportunistic species, resources are abundant but variable, and populations will undergo large variations in numbers. Selective outcome will now be determined by the rate at which the organism converts the available resources to net-offspring production— a property which will be negatively correlated with robustness: mutants with *decreased* robustness, and consequently decreased entropy, will convert resources into viable offspring at a faster rate than the ancestral type. When population size is large, the outcome of competition between the mutant and ancestral type will be predictable: mutants with lower entropy will have a selective advantage. However, when population size is small, stochastic effects will dominate and competitive outcome will now be a random process.

The invasion criteria are given in Table 5 in terms of evolutionary entropy H. This quantity describes a rate, namely the rate of increase of an effective population size (Demetrius, 1983), and determines robustness, the rate at which the population returns to its steady state condition after a random perturbation in the age-specific variables (Demetrius et al., 2004).

Demographic entropy, S, is an analog of the Gibbs–Boltzmann entropy in statistical mechanics. S is the product of the entropy rate, H and the generation time, T. Now perturbation studies (Demetrius, 2000) show that evolutionary changes in the parameters H and S are positively correlated. We write

$$\Delta H \Delta S > 0. \tag{8}$$

In view of (8), the invasion condition described in terms of ΔH can be reformulated in terms of ΔS.

Since S quantifies the position of the population along the fast–slow life-history continuum, the reformulation of the invasion criteria in terms of demographic entropy S provides a complementary characterization of invasion success which can be roughly described as follows: late age of sexual maturity, small litter size and broad reproductive span confers a selective advantage in *equilibrium* species ($\Phi<0$); early age of sexual maturity, large litter size, and narrow reproductive span bestows a selective advantage in *opportunistic* species ($\Phi>0$).

5. The Malthusian and entropic principles: a contrast

In this section we will contrast the Malthusian principle, as given in Table 1, with the entropic principle, as summarized in Table 5. In order elucidate the relation between the two invasion criteria, we will give a brief summary of the mathematical basis for the entropic principle. A more extended development is given in Appendix A.

The derivation of the entropic invasion criteria rests on a mathematical model which considers the resident population as defined by the net-reproductive function $V(x)$. Consequently, the resident will be described in terms of the demographic parameters, r, H, σ^2, which as shown in Eqs. (1) and (4), are all functions of $V(x)$.

The quantities r and H are given by Eqs. (1) and (4), respectively. The quantity σ^2, called the demographic variance, is given by

$$\sigma^2 = \frac{\int_0^\infty p(x)W^2(x)\,dx}{T}, \tag{9}$$

where $W(x) = -x\Phi + \log\, V(x)$.

The analysis assumes throughout that $\Phi \neq 0$. This constraint represents a generic condition for age-structured populations.

The demographic parameters r, H, Φ, and σ^2 are all functions of $V(x)$, the net-reproductive function. Mutants are here defined by a perturbation of the net-reproductive function: $V^*(x) = V(x)^{1+\delta(x)}$.

The change in the net-reproductive function is assumed to be the result of changes in the activity of the enzymes that regulate the metabolic reactions which determine the individual's life-history. Since enzyme activity typically increases or decreases as the individual ages, we will assume that $\delta(x)$ is a monotonic function of age (Demetrius, 1992).

The demographic parameters which describe the mutant population are given by r^*, H^*, and σ^{*2}—which are all functions of $V^*(x)$. We write

$$\Delta r = r^* - r; \quad \Delta H = H^* - H; \quad \Delta\sigma^2 = \sigma^{*2} - \sigma^2.$$

The condition for the increase in frequency and ultimate fixation of the invading population is evaluated by considering the stochastic dynamics of the frequency of the invader. For this purpose a continuous time diffusion approximation is used in order to analyze the frequency $p(t)$, given by

$$p(t) = \frac{N^*(t)}{N^*(t) + N(t)}. \tag{10}$$

Here $N(t)$ denote the population size of the resident population and $N^*(t)$ the population size of the invader, where $N^*(t) \ll N(t)$.

Let $\psi(p,t)$ denote the probability density function of the stochastic process which describes the change in frequency, denoted p, of the invading genotype. We have, see Appendix A, the Fokker–Planck equation,

$$\frac{\partial\psi}{\partial t} = -\frac{\partial[\alpha(p)\psi]}{\partial p} + \frac{1}{2}\frac{\partial^2[\beta(p)\psi]}{\partial p^2}, \tag{11}$$

where

$$\alpha(p) = p(1-p)\left[\Delta r - \frac{1}{M}\Delta\sigma^2\right] \tag{12a}$$

and

$$\beta(p) = \frac{p(1-p)}{M}[\sigma^2 p + (\sigma^2 + \Delta\sigma^2)(1-p)]. \tag{12b}$$

The quantity M is the total population size. The parameter r is the population growth rate, defined in Eq. (1), whereas σ^2 is the demographic variance given by Eq. (9).

The Fokker–Planck equation, Eq. (11) can be analyzed to yield the probability $P(y)$ that a mutant, with initial frequency y, invades the population. This is given by

$$P(y) = \frac{1 - \left(1 - \frac{\Delta\sigma^2}{\sigma^2+\Delta\sigma^2}y\right)^{(2Ms/\Delta\sigma^2)+1}}{1 - \left(1 - \frac{\Delta\sigma^2}{\sigma^2+\Delta\sigma^2}\right)^{(2Ms/\Delta\sigma^2)+1}}, \tag{13}$$

where

$$s = \Delta r - \frac{1}{M}\Delta\sigma^2 \tag{14}$$

Now, the geometry of the function $P(y)$ is determined by the sign of s. This is expressed in terms of the implications:

$$s>0 \implies P(y) \text{ convex}; \quad s<0 \implies P(y) \text{ concave}.$$

The above fact, together with certain analytic properties of $P(y)$ was exploited to express the conditions for invasion in terms of the parameters: $\Delta r, \Delta\sigma^2$, and M. This is given in Table 6.

The criteria given in Tables 5 and 6 are equivalent. This equivalence is a result of the following series of perturbation relations (Demetrius, 1992; Arnold et al., 1994):

$$\Delta r = \Phi\delta_1; \quad \Delta H = -\sigma^2\delta_2; \tag{15}$$

$$\Delta\sigma^2 = \gamma\delta_3. \tag{16}$$

The magnitude of the mutation effects $\delta_1, \delta_2, \delta_3$ satisfy $\delta_1\delta_2 > 0, \delta_2\delta_3 > 0$.

Table 6
Invasion criteria: $\Delta r, \Delta\sigma^2, M$

Demographic constraint	Selective outcome
$\Delta r>0, \Delta\sigma^2<0$	Invasion occurs a.s.
$\Delta r<0, \Delta\sigma^2>0$	Extinction occurs a. s.
$\Delta r>0, \Delta\sigma^2>0$	
$M>\Delta\sigma^2/\Delta r$	Invasion occurs a.s.
$M<\Delta\sigma^2/\Delta r$	Extinction with a prob., decreasing in M
$\Delta r<0, \Delta\sigma^2<0$	
$M<\Delta\sigma^2/\Delta r$	Invasion with a prob. decreasing in M
$M>\Delta\sigma^2/\Delta r$	Extinction a.s.

The quantity γ is given by

$$\gamma = 2\sigma^2 - \frac{3\sigma^2}{T}\int_0^\infty xp(x)W(x)\,dx + \frac{1}{T}\int_0^\infty p(x)W^3(x)\,dx. \tag{17}$$

Numerical and analytical studies of typical net-reproductive functions show that $\gamma>0$.

The perturbation relations for $\Delta r, \Delta H$, and $\Delta\sigma^2$ given by Eqs. (15) and (16), and the positivity of σ^2 and γ entails the following relations:

$$\Phi<0 \implies \Delta r\Delta H>0; \quad \Phi>0 \implies \Delta r\Delta H<0, \tag{18a}$$

$$\Delta H\Delta\sigma^2<0. \tag{18b}$$

Eq. (18a) asserts that in the case of *equilibrium species* ($\Phi<0$), mutational changes in the growth rate and entropy are positively correlated, whereas in the case of *opportunistic species* ($\Phi>0$), mutational changes in the growth rate and entropy are negatively correlated. The relation (18b) states that mutational changes in entropy and the demographic variance are negatively correlated.

The relations given by (18a) and (18b) are sufficient to establish the equivalence between the invasion criteria based on entropy, given in Table 5, and the criteria based on the Malthusian parameter and the demographic variance as expressed is Table 6.

The condition $M>\Delta\sigma^2/\Delta r$, $M<\Delta\sigma^2/\Delta r$, in Table 6 correspond to the distinction, large and small population size, described in Table 5.

It is evident that as $M\to\infty$, the selective advantage as expressed by Eq. (14) reduces to Eq. (2). Accordingly, the invasion criteria described in Table 1 are the limiting case of the conditions enunciated in Table 5. In view of the equivalence between Tables 5 and 6, we conclude that the Malthusian principle is the limit ($M\to\infty, M$ denotes population size) of the entropic criteria.

The contrast between the main properties of the Malthusian model and the entropic principle is summarized in Table 7.

Table 7
The Malthusian model and the entropy principle: a contrast

Evolutionary property	Malthusian model	Entropic model
Measure of fitness	Population growth rate	Entropy
Intensity of selection	Decreasing function of age	Convex function of age
Invasion criteria	*Deterministic*: Relative values of population growth rate	*Stochastic*: Relative values of entropy

6. Diffusion equations in population genetics: models with and without age structure

The fundamental quantity which is used in population genetics to characterize the genetic composition of a Mendelian population is the gene frequency. When population size is finite, the sampling effects of gametes introduce an element of chance into the change of gene frequencies so that the dynamical changes in gene frequency must be treated as a stochastic process.

Wright (1931, 1942, 1945) was the first to recognize that these stochastic processes can be treated by the application of diffusion equations and produced an extensive theory which investigated in detail changes in gene frequency under various assumptions of random genetic drift, mutation, migration and selection.

Diffusion processes, as an analytic theory of stochastic phenomena in population genetics, were later systematized by Kimura (1955, 1957, 1962), with special reference to the invasion dynamics of a mutant allele and the probability of gene fixation.

The arguments by Wright and Kimura, though based on the assumption that population size is finite, postulate an infinite gametic pool. In view of the law of large numbers, this postulate excluded the action of stochastic effects due to variance in offspring number.

The importance of the variance in offspring number in determining the outcome of selection was recognized by Gillespie (1974, 1975), who extended the Wright–Kimura models by assuming that the gametic pool is essentially the same size as the final population. This important generalization of the Wright–Kimura models has been the basis for extensions to include various classes of migration effects (Proulx, 2000; Shpak, 2005; Shpak and Proulx, 2006).

The Gillespie models are concerned with the effects of within-generation variance in offspring number on the fixation probabilities of mutant alleles. This class of models, as in the case of Wright–Kimura systems, pertains to the dynamics of competition within a population whose individuals do not vary in terms of age or size. In these models differences in generation time and demographic variability are not considered in the analysis of the evolutionary dynamics.

The stringency of these assumptions was recognized by Demetrius and Gundlach (1999), who integrated the

methods of ergodic theory and the theory of diffusion processes to study, under more general conditions, the invasion dynamics of mutant alleles. The diffusion equations which were derived from the study of this new class of models are generalizations of the equations derived from models concerned with selection within populations defined by the absence of age structure. We will now specify the nature of these generalizations by describing the assumptions and the structure of the various models and the relations between them.

6.1. Models without age structure

6.1.1. The Wright–Kimura model

The model assumes that the gametic pool is infinite. This condition entails that variance effects due to sampling from the gametic pool can be neglected, hence the selective value can be described completely by the mean number of offspring, denoted μ.

Since the populations are not structured by age, size or any such characteristic, a mutation can be described directly in terms of changes $\Delta\mu$, in the mean offspring number.

The selective advantage, s, is given by

$$s = \Delta\mu. \tag{19}$$

The probability density function, $\psi(p,t)$, which describes the change in frequency, denoted p, of a genotype is given by Eq. (11), where the drift and diffusion terms are

$$\alpha(p) = p(1-p)\Delta\mu, \tag{20a}$$

$$\beta(p) = \frac{p(1-p)}{M}. \tag{20b}$$

6.1.2. Models based on variance in offspring number

This class of models differs from the Wright–Kimura systems by postulating a finite gametic pool of the same size as the population (Gillespie, 1974). The selective value is now described by the mean offspring number, μ, and the variance in offspring number, $\tilde{\sigma}^2$. A mutation is described in terms of its direct effect on the parameters μ and $\tilde{\sigma}^2$.

The analysis of the dynamics between mutant and resident type shows that the selective advantage now becomes

$$s = \Delta\mu - \frac{1}{M}\Delta\tilde{\sigma}^2. \tag{21}$$

The probability density function $\psi(p,t)$ also satisfies the diffusion equation, Eq. (11), with the drift and diffusion terms given by

$$\alpha(p) = p(1-p)\left(\Delta\mu - \frac{1}{M}\Delta\tilde{\sigma}^2\right), \tag{22a}$$

$$\beta(p) = \frac{p(1-p)}{M}[\tilde{\sigma}^2 p + (\tilde{\sigma}^2 + \Delta\tilde{\sigma}^2)(1-p)]. \tag{22b}$$

As shown in Gillespie (1975), when $\alpha \to \infty$, where α denotes the size of the gametic pool, we have $\tilde{\sigma}^2 \to 1$, hence $\Delta\tilde{\sigma}^2 \to 0$. Hence, the Wright–Kimura model is a limiting case, as the gametic pool size tends to infinity, of the Gillespie model.

6.1.3. Models based on growth rate and demographic variance

This model, in contrast to the structures described in Sections 6.1.1 and 6.1.2, is concerned with the analysis of competition between populations, each population being defined by a particular genotype (Demetrius and Gundlach, 2000). Formally, each genotype is described by a triple (Ω, μ, φ), where

(i) Ω is a set of strategies, $\Omega = (x_1, \ldots, x_d)$.
(ii) $\varphi : \Omega \to \mathbb{R}$, a function on Ω which assigns to each $x_i \varepsilon \Omega$, a non-negative number $\varphi(x_i)$, the net-offspring production associated with the strategy x_i.
(iii) $\mu = (\mu_i)$ is a probability distribution. It refers to the preference of certain strategies in the population.

The evolutionary dynamics of the population is now described in terms of the parameters:

(i) the population growth rate $r = \log Z(\varphi)$, where $Z(\varphi) = \sum_i \varphi(x)$,
(ii) the demographic variance $\sigma^2 = \int(\log\varphi)^2\, d\mu - (\int \log\varphi\, d\mu)^2$.

A mutation in this model is defined in terms of a change φ^* in the function φ defined by

$$\log\varphi^* = (1+\delta)\log\varphi,$$

where δ, a real number, is assumed small.

The analysis of the dynamics of selection between mutant and resident types shows that the selective value, s, is now given by

$$s = \Delta r - \frac{1}{M}\Delta\sigma^2. \tag{23}$$

The probability density function $\psi(p,t)$, which describes the change in frequency of the mutant population, also satisfies Eq. (11). The drift and diffusion terms now become

$$\alpha(p) = p(1-p)\left(\Delta r - \frac{1}{M}\Delta\sigma^2\right), \tag{24a}$$

$$\beta(p) = \frac{p(1-p)}{M}[\sigma^2 p + (\sigma^2 + \Delta\sigma^2)(1-p)]. \tag{24b}$$

Eqs. (23), (24a) and (24b), are formally identical to the equations for selective value given by Eq. (21), and the drift and diffusion term given by Eqs. (22a) and (22b), respectively. From appropriate scaling of the demographic parameters we can infer that the model described by 6.1.2, which refers to selection within a population, is a limiting case of the model described in Section 6.1.3, which pertains to selection between populations.

6.2. Age-structured models

The demographic model (Demetrius, 1997; Demetrius and Gundlach, 1999) is a generalization of the model described in Section 6.1.3, to populations where the fecundity and mortality variables are functions of age. Each genotype is now defined by a Leslie matrix, or, in the case of a continuous model, by a net-reproductive function $V(x)$. The selection dynamics is now described in terms of the population variables: (i) the population growth rate, as defined by Eq. (1), (ii) the demographic variance, given by Eq. (9).

A genotype is now defined in terms of a population of individuals of the same genetic constitution but differing in terms of their age. Mutations result in a change in genotypic composition.

In view of the demographic heterogeneity of the population, a mutation cannot be modeled as a simple change in the fecundity or survivorship variables, analogous to the changes described in the Wright–Kimura systems. A mutation is now considered as a small perturbation in the net-reproductive function $V(x)$ that determines the genotype. These perturbations will induce changes Δr, $\Delta\sigma^2$, in the demographic variables, r and σ^2, respectively.

For the mutant $V^*(x)$, we have imposed the condition

$$V^*(x) = V(x)^{1+\delta(x)},$$

where $\delta(x)$ is monotonic in x (see Demetrius, 1992).

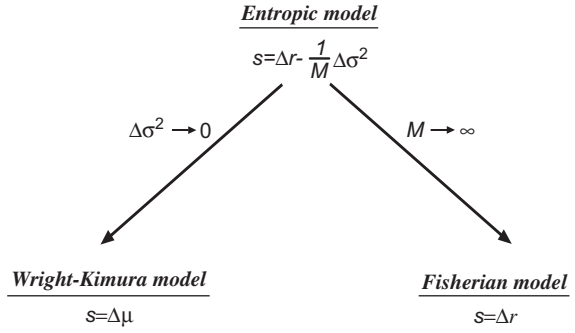

Fig. 1. The relation between the selective advantage parameters of the entropic model (age structure and finite size), the Wright–Kimura (no age structure and finite size), and the Fisherian model (age structure and infinite size).

In view of the constraints on the perturbations in $V(x)$, the changes, Δr, $\Delta\sigma^2$, are not arbitrary. They will be correlated, as they are both specific functions of the individual demographic variables, as is shown by Eqs. (15) and (16).

The selective value in structured populations now assumes the form

$$s = \Delta r - \frac{1}{M}\Delta\sigma^2, \tag{25}$$

where the growth rate r is given by Eq. (1), and the demographic variance σ^2 by Eq. (9).

The probability density function $\psi(p,t)$ which describes the change in frequency of the mutant population is also given by Eq. (11), where the drift and diffusion terms are formal analogies of Eqs. (24a) and (24b).

The above characterization of structured population models, and the relation we have delineated between the Wright–Kimura models and the models described in Sections 6.1.2 and 6.1.3 indicate that structured population models, which we call entropic models, are natural and meaningful generalizations of the Wright–Kimura systems. These systems are the limit, $\Delta\sigma^2 \to 0$, of the entropic models. We also observe from Eq. (25), that as $M \to \infty$, the selective value for the entropic models now reduces to the equation $s = \Delta r$, the selective value for the Fisherian models with demographic structure. Hence the Fisherian models are also limiting states of the entropic model.

Our representation of the entropic model (age structure and finite size), as generalizations of the Wright–Kimura model (no age structure and finite size), and the Fisherian model (age structure and infinite size) is described in Fig. 1.

The correspondence between the three classes of models, (a) age structure and infinite population size (Fisher), (b) no age structure and finite size (Wright–Kimura), (c) age structure and finite size (the entropic model), is described in Table 8.

7. Computational studies: model description

To evaluate and contrast the predictions of the Malthusian and entropic principles we performed computer simulations of direct competition between a population of wild type genotypes and a mutant genotype that is initially present at low frequency. We will draw extensively from the computational studies described in Kowald and

Table 8
Relations between the three classes of models

Population properties	Fisherian models	Wright–Kimura models	Entropic models
Demographic constraints	Infinite size, age structure	Finite size, no age structure	Finite size, age structure
Darwinian fitness	Malthusian parameter, r	Mean number of offspring, μ	Entropy, H
Selective advantage	$s = \Delta r$	$s = \Delta\mu$	$s = \Delta r - \frac{1}{M}\Delta\sigma^2$

Demetrius (2005). We refer to this article for the details of the model on which the computation is based.

Our analysis of this numerical experiment is based on a C++ program that simulates a population of genotypes based on the Leslie model, a discrete analog of the continuous process described by Lotka. In this framework the population consists of a number of discrete age classes. Each year individuals in the age classes either die with a certain probability or move into the next higher age class. Furthermore, in each age class an individual generates on average a certain number of offspring. The genotype of an individual is therefore completely determined by two vectors of numbers: one for the age specific mortality and the other for age specific fertility. From these data the Malthusian parameter r can be calculated according to Eq. (1), and the evolutionary entropy H and the reproductive potential Φ can be calculated, using Eq. (4).

7.1. Mutations

The resident population is described by a net-fecundity function $V(x)$, which we assume is a concave function of age x. We assume that the mutant $V^*(x)$ is a perturbation of the function $V(x)$ defined by $V^*(x) = V(x)^{1+\delta(x)}$ where $\delta(x)$ is monotonic in x. A consequence of the monotonicity condition is that the relation between $V(x)$ and $V^*(x)$ can be categorized in terms of the following patterns (see Kowald and Demetrius, 2005).

(i) A translation of the function $V(x)$, which corresponds to a change in the age of sexual maturity.
(ii) A rescaling of the function $V(x)$, which characterizes an increase or a decrease in the net-fecundity function.

The genotypes of mutant and wild type are constructed in such a way, that their net reproductive rate exceeds one, which entails a growing population. To simulate the competition between individuals additional growth constraints have to be applied.

Equilibrium species live under conditions where the population size remains roughly constant. If the total population size (wild type plus mutant) exceeds the carrying capacity, an additional extrinsic mortality probability is applied to each individual (regardless of genotype), so that the population is reduced to the allowed maximum population size. Because external mortality is a probabilistic process, the total population size fluctuates around the carrying capacity. The frequencies of wild type and mutant, however, are free to change and the simulation is continued until only one genotype (wild type or mutant) is left in the population. The competition experiments are repeated 200 times to assess the invasion probability of the mutant.

Opportunistic species are characterized by a different life-history. Their population size grows exponentially for 12 time periods after which a probabilistic external mortality is applied that reduces the population size to its initial value.

8. A computational study: the entropic principle—the constraints on the reproductive potential

The entropic principle, as summarized in Table 5, asserts that the invasion dynamics is a stochastic process which is determined by ΔH and is contingent on the parameter Φ.

The simulations we now describe are based on numerical studies which investigate the selective outcome between wild type and mutant under different ecological constraints described in terms of the parameter Φ.

8.1. $\Phi < 0$ *(equilibrium species)*, $\Delta H > 0$

The analysis asserts that for equilibrium species, genotypes that have a larger entropy should almost always invade and increase in frequency.

Fig. 2 shows typical simulation results for a large population of an equilibrium species. Initially the population consists of 9500 wild-type and 500 mutant individuals (5% mutants) and develops according to the rules outlined under Section 7. ΔH, the difference of entropy between mutant and wild type, is 0.045. Since the computer simulations reflect the stochastic nature of the invasion process, the competition between mutant and wild type was repeated 200 times to obtain a statistical measure of the invasion probability. Under the described conditions the mutant was able to invade in 84% of the cases. The development of mutant and wild-type population numbers is shown in Fig. 2 for three successful invasion events.

8.2. $\Phi < 0$ *(equilibrium species)*, $\Delta H < 0$

The situation is completely different if the demographic entropy of the mutant is lower than that of the wild type. Initially the mutant is again present at 5%, but now ΔH is negative (-0.033). During the 200 repetitions the mutant was never able to invade. As can be seen in Fig. 3, the mutant finally dies out. Although it might temporarily increase in frequency, it never reaches fixation.

8.3. $\Phi > 0$ *(opportunistic species, large size)*, $\Delta H > 0$

The entropic principle asserts that in large populations that undergo episodes of rapid growth followed by a catastrophic breakdown (opportunistic species), genotypes with a higher entropy will almost always become extinct. Again we tested this by computer simulation. As before the initial mutant frequency was 5%, but this time the population was free to grow unconstrained for 12 time steps (years), after which the total population was reduced back to the carrying capacity of 10 000 individuals, resulting in an oscillating behavior. We studied the situation with the mutant having a higher entropy than the wild type ($\Delta H = 0.00023$). As expected, the mutant

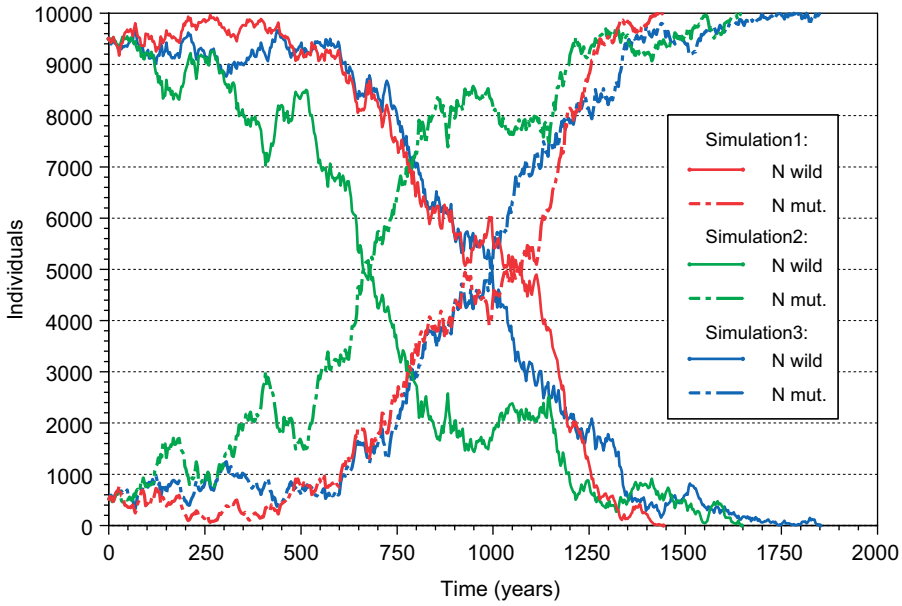

Fig. 2. Development of the number of wild type and mutant individuals over time for a large equilibrium species. The simulation was performed as explained in Section 7. The total population size consisted of 9500 wild type and 500 mutant individuals. In these simulations $H_{mut} > H_{wt}$, leading to 84% invasion success of the mutant. The diagram shows population trajectories of three typical simulation runs.

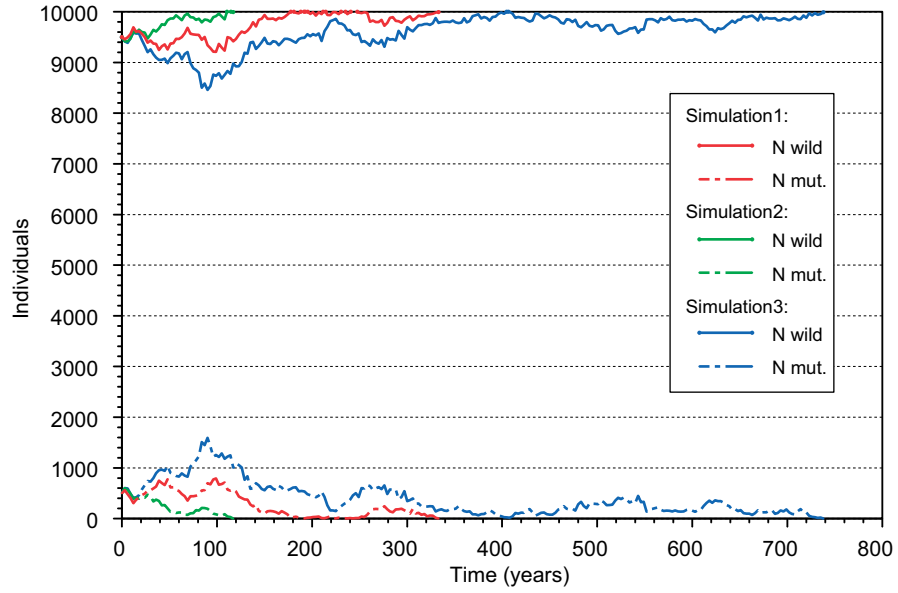

Fig. 3. Development of the number of wild type and mutant individuals over time for a large equilibrium species. The simulation was performed as explained in Section 7. The total population size consisted of 9500 wild type and 500 mutant individuals. In these simulations $H_{mut} > H_{wt}$, leading to no successful invasion events within 1000 simulations. The diagram shows population trajectories of three simulation runs.

could only invade in 6% of the encounters and Fig. 4 shows an example of the more likely case described by success of the wild type.

8.4. $\Phi > 0$ *(opportunistic species, large size)*, $\Delta H < 0$

The invasion behavior is different, as predicted, if the mutant has a smaller entropy than the wild type (Fig. 5). If $\Delta H = -0.03$, the invasion success, calculated from the simulations, is 88%. The diagram shows a successful invasion process that is completed after approximately 3500 years.

8.5. $\Phi > 0$ *(opportunistic species, small size)*

The simulations described in Sections 8.3 and 8.4 investigated the behavior of *large* populations of opportunistic species. However, the entropic principle

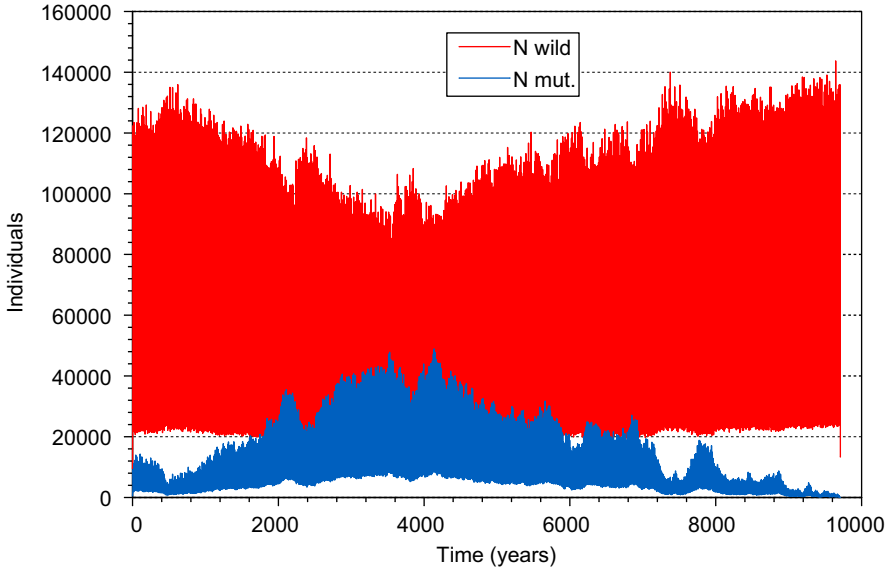

Fig. 4. Development of the number of wild type and mutant individuals over time for a large opportunistic species. The simulation was performed as explained in the Section 7. The initial population size consisted of 9500 wild type and 500 mutant individuals. In these simulations $H_{mut} > H_{wt}$, leading to only 6% invasion success of the mutant. The diagram shows population trajectories for a typical simulation run in which the mutant could not invade the wild type. Because of the opportunistic life style the population sizes oscillate strongly.

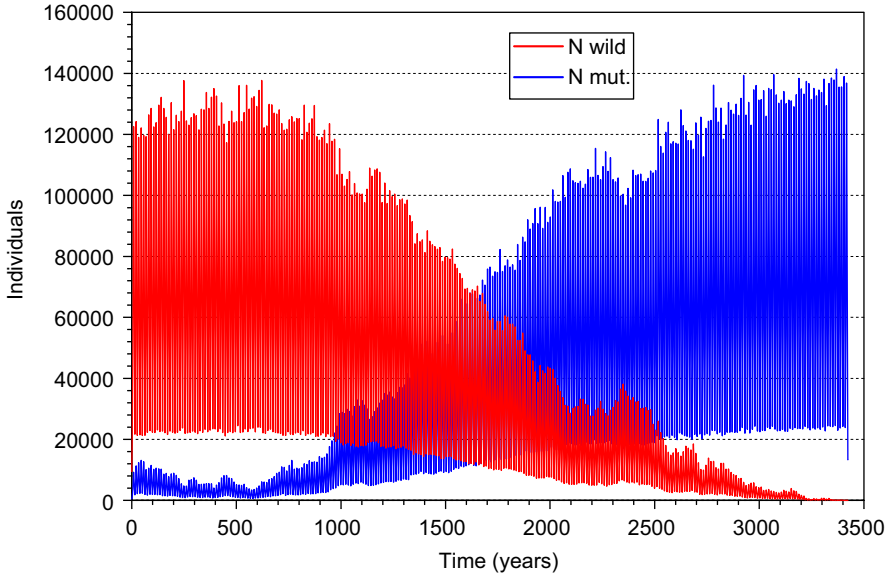

Fig. 5. Development of the number of wild type and mutant individuals over time for a large opportunistic species. The simulation was performed as explained in Section 7. The initial population size consisted of 9500 wild type and 500 mutant individuals. In these simulations $H_{mut} > H_{wt}$, leading to 88% invasion success of the mutant. The diagram shows population trajectories for a typical simulation run in which the mutant successfully invaded the wild type.

asserts that in *small* populations of opportunistic species stochastic events will dominate, irrespective of the constraint on the entropy. Accordingly, the invasion success should be a random event. This prediction is investigated for mutants first with smaller then with larger entropy. As usual the initial frequency of mutants was 5%, but in these simulations the total population only consists of 60 individuals. Although the mutant genotype had smaller entropy than the wild type ($\Delta H = -0.03$), it only reached fixation in 6.5% of the simulations. As the three examples described in Fig. 6 demonstrate, when population size is small, the mutant often dies out very quickly. Simulations with different positive values for ΔH and different initial mutant frequencies showed that the invasion probability is largely independent of the value of ΔH, but is instead proportional to the initial mutant frequency. This observation is consistent with Eq. (11) which predicts that the invasion probability $P(y)$ will be proportional to the initial frequency when N is small.

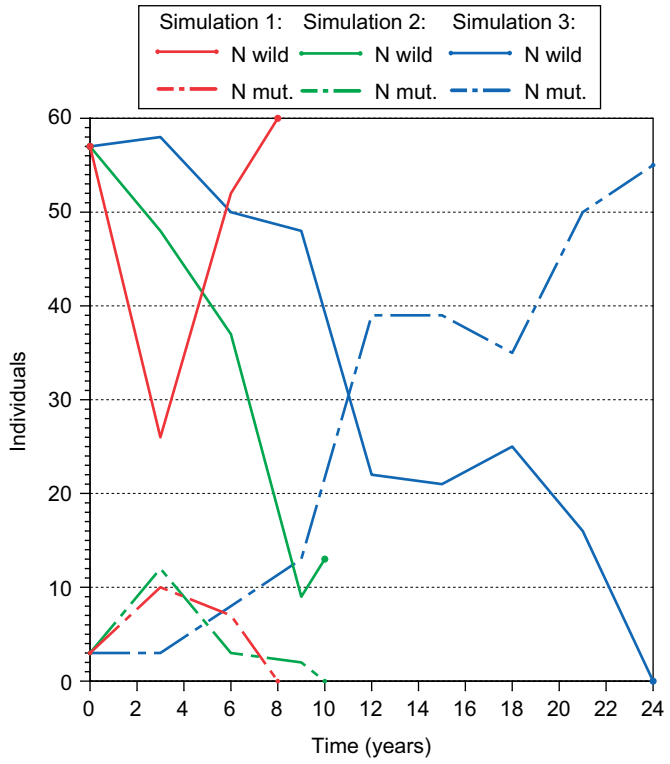

Fig. 6. Development of the number of wild type and mutant individuals over time for a small opportunistic species. The simulation was performed as explained in Section 8. The initial population size consisted of 57 wild type and three mutant individuals. In these simulations $H_{mut} > H_{wt}$. The diagram shows population trajectories for three typical simulation runs.

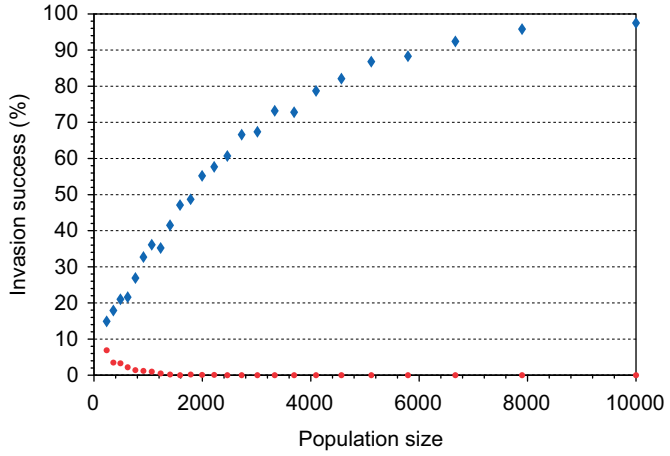

Fig. 7. Invasion success of a mutant with a Malthusian parameter, r, that is larger (diamonds) or smaller (circles) than the Malthusian parameter of the wild type, depending on the population size. For large population sizes r correctly predicts the outcome of the competition. For small population sizes, however, the invasion success becomes equal to the initial mutant frequency (10%) independent of r.

9. A computational study—constraints on growth rate, population size, and demographic variance

The probability of fixation $P(y)$ of a mutant allele is given, according to the entropic principle, in terms of population size, and the demographic parameters, growth rate and demographic variance. We will invoke the expression for $P(y)$, see Eq. (11), to contrast the predicted patterns of invasion success with the patterns generated by the simulation. The effect of population size on invasion success will be analyzed in Section 9.1. The computational studies reject the Malthusian tenet, namely, when $\Delta r > 0$ invasion always occurs. The numerical analysis indicates the stochastic nature of the invasion process and its dependence on population size. These effects are consistent with the predictions of the theory.

The effect of invasion success on the growth rate and demographic variance is analyzed in Section 9.2. These studies demonstrate the relation between predicted and observed patterns. Our analysis indicates that the discrepancy between the observed and predicted patterns decreases as the growth rate r decreases, and the variance σ^2 increases. These correlations are consistent with the predictions of the theory.

9.1. Invasion success: the dependence on population size

We investigate the relation between predicted pattern and simulation by studying the relation between invasion success, population size and initial frequency of mutant. We evaluate this dependence by performing computer simulations for competition between a wild type and a mutant for populations varying in size from 200 to 10,000. For each population size 1000 repetitions were performed from which the invasion probability was calculated. Two different types of simulations were performed, one with the mutant having a higher Malthusian parameter ($\Delta r = 0.074$) and a second with the genomes of wild type and mutant being exchanged ($\Delta r = -0.074$). The initial frequency of the mutant was in each case 10%. The diamonds in Fig. 7 show the results for $\Delta r = 0.074$ and the circles the outcome for $\Delta r = -0.074$.

We observe the following features:

Even if $\Delta r > 0$, the invasion success shows a strong dependence on population size. With a population size of 5000 the fixation probability is still below 90%, reaching 97.5% for 10 000 individuals.

A similar but opposite situation exists if $\Delta r < 0$. Although the mutant has a smaller Malthusian parameter than the wild type, in a population with 230 individuals it replaces the resident genotypes in 6.9% of the cases.

The study of the relation between invasion success and initial frequency gives rise to a general property: *Irrespective of the value of the selective advantage, for very small populations the invasion success becomes equal to the initial mutant frequency.*

Such a behavior is predicted by Eq. (11), which shows that for very small M, $P(y)$ is approximately the initial frequency y.

9.2. Invasion success: the dependence on growth rate and demographic variance

In this study we analyze the relation between predicted and observed patterns in terms of its dependence on the demographic variance, σ^2, for the wild type.

We consider the invasion success of several pairs of genotypes characterized by different values of r and σ^2.

The life tables used in the study are 4×4 Leslie matrices: each matrix has a fecundity, denoted a, which is independent of age, and a survivorship b, which is constant from one age class to the next.

$$A = \begin{bmatrix} a & a & a & a \\ b & 0 & 0 & 0 \\ 0 & b & 0 & 0 \\ 0 & 0 & b & 0 \end{bmatrix}.$$

We consider two classes of models, defined by fecundities $a = 1$ (Class I), $a = 2$ (Class II) for the resident type.

The genotypes are described by the net-reproductive function $[V_1, V_2, V_3, V_4]$. Here $V_1 = a$, $V_2 = ab$, $V_3 = ab^2$, $V_4 = ab^3$. These values and the corresponding demographic parameters are described in Table 9.

The initial frequency of the mutant in each case was 5%. In the computational study we first fix the carrying capacity and then run the experiment to determine the probability of invasion: to estimate invasion success several runs were done. The graphs in Fig. 8(a) contrast the invasion success for the two genotypes in Class (I). The graphs in 8(b) contrast the invasion success for the three pairs of genotypes in Class (II).

We observe the following features from the graphs:

(a) The predicted and observed patterns are qualitatively similar.
(b) The diffusion approximation overestimates the simulated extinction probabilities.
(c) The magnitude of the overestimation decreases with increasing values for the demographic variance. This is true in both class (I) types where we consider two models whose wild type is given by the variance 0.088 and 0.3612. In class (II) models, there are three pairs of genotypes: the demographic variance of the wild type varies from 0.2417 to 0.4356.
(d) The magnitude of the overestimation is small when the Malthusian parameter is small. A small deviation from predicted value is observed in the class (I) model with $r = 0.1692$, a larger variation is observed when $r = 0.3947$. Relatively larger deviations from predicted values are observed in the class (II) models. In these models the values for r are relatively large, ranging from 0.7064 to 0.783.

We will now comment on observations (b), (c) and (d) in order to explain the observed deviations of the predictive patterns from the simulations.

The reason for (b) is due to the fact that the sample paths in the diffusion process are continuous, whereas those in the simulations are discrete. The continuous and discrete nature of the two processes entails that there will be significant differences in the extinction time. Extinction in the diffusion approximation occurs when the population size first reaches one individual. However, as observed in Karlin and McGregor (1964), Lande and Orzack (1988), the extinction will not necessarily occur at an integer value of elapsed time, in sharp contrast to the extinction event in the discrete simulation. Accordingly, certain sample paths will be considered an extinction in the continuous model, but a non-extinction event in the discrete simulation. This condition will result in an overestimation of the extinction probability in the diffusion approximation.

The rationale for (c) derives from certain limiting arguments which are implicitly invoked in the approximation of the dynamics of discrete time stochastic processes

Table 9
Life table and demographic parameters

Type	Net reproductive function				Demographic parameters			
(b for wild)	V_1	V_2	V_3	V_4	r	H	Φ	σ^2
Class I models ($a = 1$)								
Wild (0.4965)	1	0.4965	0.2466	0.1224	0.3947	0.6127	−0.2180	0.0880
Mut.	1	0.4997	0.2497	0.1248	0.3967	0.6135	−0.2168	0.0865
Wild (0.1849)	1	0.1849	0.0342	0.0063	0.1692	0.4298	−0.2607	0.3612
Mut.	1	0.1877	0.0352	0.0066	0.1715	0.4331	−0.2616	0.3580
Class II models ($a = 2$)								
Wild (0.1880)	2	0.3761	0.0707	0.0133	0.7830	0.2926	0.4904	0.4356
Mut.	2.0139	0.3724	0.0689	0.0127	0.7879	0.2882	0.4997	0.4360
Wild (0.707)	2	0.1414	0.0100	0.0007	0.7279	0.1489	0.5790	0.3682
Mut.	2.0139	0.1387	0.0095	0.0007	0.7337	0.1452	0.5885	0.3641
Wild (0.0266)	2	0.0532	0.0014	0.00003	0.7064	0.0699	0.6365	0.2417
Mut.	2.0139	0.0516	0.0013	0.00003	0.7127	0.0675	0.6452	0.2364

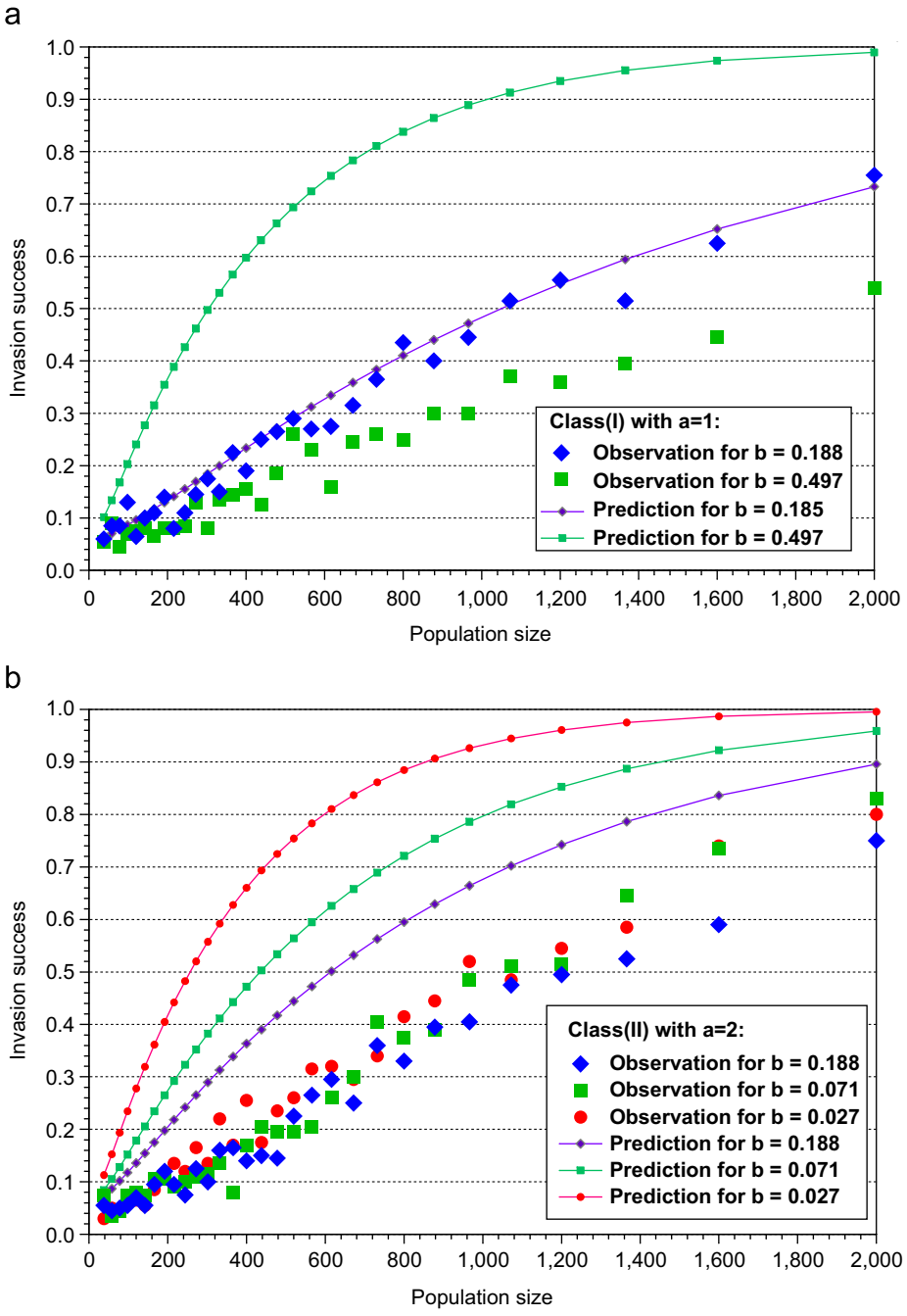

Fig. 8. Invasion success as a function of demographic variance. Comparison of the predicted and observed patterns of invasion success for three pairs of wild type and mutant with different values for the demographic variance. The divergence of predicted pattern from observed decreases with increasing demographic variance.

with continuous time diffusion processes. The limiting argument depends on the rate at which certain parameters of the model tend to zero. According to the central limit theorem for the abstract dynamical systems associated with structured populations, the rate will be determined by the demographic variance σ^2. In models where σ^2 is small, there will be weak agreement between the predicted values and the simulation. This is illustrated by comparing the two examples described in class (I) models. The discrepancy is small when $\sigma^2 = 0.3612$, but large when $\sigma^2 = 0.088$. We also observe that in class (II) models the discrepancy between the predicted patterns and the simulations reduces with increasing demographic variance.

The observations noted in (d) are due to the fact that the accuracy of the diffusion approximation also decreases with increasing values of the Malthusian parameter. This is evident from the nature of the assumptions invoked in deriving the diffusion equations. Accordingly, we expect the concordance between the predicted values and the numerical studies to be strong when r is small and weak when r is large. The patterns observed in the class (I) models with $r = 0.1692$ and 0.3947

and the class (II) models with $r = 0.7064$, 0.7279, and 0.783 are consistent with this argument.

10. Conclusion

The numerical studies we have described provide a computational support for the following analytical results.

1. Evolutionary entropy predicts the outcome of competition between a variant and a resident type. This outcome is a *stochastic* event which is contingent on the condition—equilibrium or opportunistic—of the species.
   (a) In the case of equilibrium species, population size remains relatively constant. When this condition obtains, mutants with increased entropy have increased demographic stability or robustness and will almost always prevail in competition with the resident for the available resources.
   (b) In the case of opportunistic species, population size undergoes large variations. Under such constraints, selective outcome is determined by both entropy and population size. When size is large, mutants with decreased entropy and consequently decreased demographic stability will almost always prevail. When size is small, the selective outcome is unpredictable.
2. The Malthusian parameter predicts the outcome of competition between variant and resident only when population size is infinite. In this case the selective outcome is a *deterministic* event.
3. The invasion criteria described by the Malthusian condition is the limit, as population size tends to infinity, of the criteria described by entropy.

Acknowledgements

We would like to thank A. Riemer for help regarding the development of the simulation program. We are also grateful to the referees for their careful study of numerous revisions of the manuscript and their advice in its organization.

Appendix A

The condition for the frequency and ultimate fixation of the invading population is evaluated by considering the stochastic dynamics of the frequency of the invader. The frequency $p(t)$ is given by

$$p(t) = \frac{N^*(t)}{N^*(t) + N(t)}.$$

Here $N(t)$ denotes the population size of the resident population and $N^*(t)$ the population size of the invader; where $N^*(t) \ll N(t)$.

Let $\psi(p, t)$ denote the probability density of populations with allele frequency p at time t. We will show that $\psi(p, t)$ satisfies the diffusion equation:

$$\frac{\partial \psi}{\partial t} = -\frac{\partial[\alpha(p)\psi]}{\partial p} + \frac{1}{2}\frac{\partial^2[\beta(p)\psi]}{\partial p^2}, \tag{A.0}$$

where $\alpha(p)$ and $\beta(p)$ are given by (9a) and (9b), respectively.

Eq. (A.0) was derived by integrating the methods and techniques of ergodic theory with the theory of diffusion processes (see Demetrius and Gundlach, 1999).

The derivation involves several elements.

(i) The application of a statistical mechanics formalism to generate the macroscopic parameters that appear in the diffusion equations.
(ii) The use of a central limit theorem for dynamical systems to provide a rationale for demographic parameters which are invoked in the diffusion equations.
(iii) The appeal to the Ito calculus in obtaining the diffusion approximation.

These elements are of a highly technical nature. The Appendix is intended to give a relatively non-technical guide to the main mathematical ideas involved.

We begin with certain preliminaries. This section describes the classical Leslie model based on considering the dynamics of age distributions. We then proceed to a statistical mechanics description, based on the notion of "genealogies".

The formalism outlined in this description is the cornerstone of the mathematical models we have developed to study the transient dynamics of structured populations. The analysis revolves around the concepts, reproductive potential, entropy, and demographic variance. These concepts play a central role in the models for the diffusion approximations.

A.1. Preliminaries

A.1.1. Population processes in terms of their age-distribution

We consider a simple time discrete model obtained by dividing a population into d age-classes. Hence, a vector $\bar{z}(n) = (z_1(n), z_2(n), \ldots, z_d(n))$ represents an age-distribution of the population at time n. Changes in the age-distribution are described by the discrete dynamical system given by

$$\bar{z}(n+1) = A\bar{z}(n), \tag{A.1}$$

where $A = (a_{ij})$, the so-called Leslie matrix is given by

$$A = \begin{bmatrix} m_1 & m_2 & \ldots & & m_d \\ b_1 & 0 & \ldots & & 0 \\ 0 & b_2 & \ldots & & 0 \\ . & & & & \\ . & & & & \\ . & & & & \\ 0 & 0 & & b_{d-1} & 0 \end{bmatrix},$$

where m_i is the mean number of offspring produced by individuals in the ith age-class, b_i the probability that an individual survives from age class (i) to age class $(i+1)$. Write

$$l_j = \begin{cases} 1, & j = 1, \\ b_1 b_2 \dots b_{j-1}, & j > 1, \end{cases}$$

and let $V_j = l_j m_j$.

We assume that the matrix A is primitive. By the Perron–Frobenius theorem, we conclude that

(i) A has a simple positive real dominant eigenvalue λ.
(ii) There exist positive unit vectors $\bar{u}, \bar{v} \in \mathbb{R}^d$ such that

$$A\bar{u} = \lambda\bar{u}, \quad \bar{v}A = \lambda\bar{v}. \tag{A.2}$$

The vector $\bar{u} = (u_i)$ corresponds to the stationary age-distribution. The vector $\bar{v} = (v_i)$ is a measure of the relative contribution made to the stationary population in the future by the individual age groups.

$$r = \log \lambda$$

is the population growth rate or the Malthusian parameter. It describes the growth rate of the population number $N(n) = \sum_{j=1}^{d} z_j(n)$, that is

$$r = \lim_{n\to\infty} \frac{1}{n} \log N(n). \tag{A.3}$$

The dominant eigenvalue λ is the unique positive real root of the equation

$$1 = \sum_{j=1}^{d} \frac{V_j}{\lambda^j},$$

from which we can deduce that the function

$$p_j = \frac{V_j}{\lambda^j} \tag{A.4}$$

defines a probability distribution. It is the probability that the mother of a randomly chosen newborn belongs to age class j.

A.1.2. Population processes in terms of genealogies

The description of the population process in terms of dynamical changes in the age distribution is the basis for generating the characterization of the population growth rate r. This macroscopic parameter is the cornerstone of the classical models of population dynamics. The statistical mechanics formalism we now describe is the framework for generating a new class of population observables, which include the reproductive potential, and the demographic variance (Demetrius, 1983).

The relevance of this mathematical model to population processes is based on the observation that the dynamics of the age-distribution given by Eq. (A.1) can be considered as a deterministic representation of an underlying stochastic process. This process is characterized by a probability measure on the space of "genealogies", a notion we will now describe.

This new formalism recognizes that the matrix $A = (a_{ij})$ can be represented as a directed graph, G. This graph, see Fig. 9, is obtained by joining nodes (i) to (j) if $a_{ij} > 0$.

A path in the graph G can be described by a sequence

$$x = (x_0, x_1, x_2, \dots),$$

where $x_i \epsilon \{1, \dots, d\}$ and $a_{x_i, x_{i+1}} > 0$.

A path is called a genealogy as it represents a recording of successive ancestral states of a particular individual which at time 0 is in the class x_0.

Let Ω denote the set of all infinite backward paths of the graph G. The genealogies generated by a single individual in age class (1) at time 0 will be characterized by a certain distribution which can be calculated from the Markov chain generated by the transition matrix P.

The Markov measure $\tilde{\mu}$ associated with the transition matrix $P = (p_{ij})$ has the representation

$$\tilde{\mu}\{x_n = i_n, \dots, _{n+k} = i_{n+k}\} = \pi_{i_n} p_{i_n i_{n+1}} \cdots p_{i_{n+k-1} i_{n+k}},$$

where

$$p_{ij} = \frac{a_{ji} v_j}{\lambda v_j}.$$

The vector $\tilde{\pi} = (\pi_i)$ is the stationary distribution of the Markov matrix P.

The statistical mechanics representation of the Leslie model described by (A.1) can be characterized by the triple $(\Omega, \tilde{\mu}, \varphi)$, where Ω denotes the space of genealogies, $\tilde{\mu}$ the probability measure on Ω, and $\varphi : \Omega \to \mathbb{R}$ defined by

$$\varphi(x) = \log a_{x_0 x_1}, \tag{A.5}$$

where a_{ij} denote the i,jth term of the Leslie matrix A.

The function φ can be interpreted as a potential function on the space of genealogies Ω.

The importance of this representation resides in the fact that it can be invoked to generate not only the population growth rate, defined by (A.3), but also a new class of macroscopic variables, analogous to the macroscopic parameters in statistical thermodynamics (see Demetrius, 1983).

The fundamental parameter in these derivations is the function

$$\begin{aligned} S_n(\varphi(x)) &= \log a_{x_0 x_1} + \log a_{x_1 x_2} + \cdots + \log a_{x_{n-1} x_n} \\ &= \log a_{x_0 x_1}\ a_{x_1 x_2} \cdots a_{x_{n-1} x_n}. \end{aligned} \tag{A.6}$$

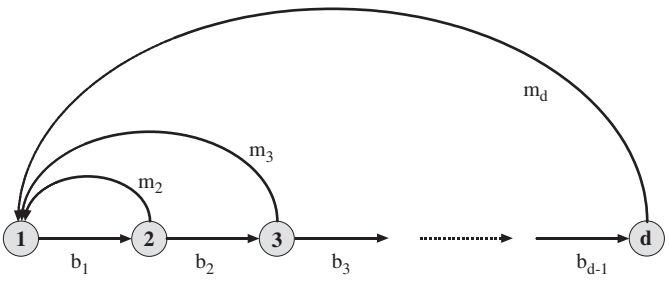

Fig. 9. Life cycle graph of a Leslie matrix.

The quantity $S_n(\varphi(x))$ represents the net-offspring production rate of the individuals that describe the genealogy.

The population growth rate $r = \log \lambda$, which is defined in terms of the Leslie model by Eq. (A.3), can be expressed in terms of the function $S_n(\varphi(x))$

We observe that

$$\exp S_n(\varphi) = a_{x_0x_1}a_{x_1x_2}\cdots a_{x_{n-1}x_n}.$$

Write

$$Z_n(\varphi) = \sum_{x_0x_1\ldots x_n} \exp\, S_n(\varphi) = \sum_{x_0x_1\ldots x_n} a_{x_0x_1}a_{x_1x_2}\cdots a_{x_{n-1}x_n}.$$

We now have (see, for example, Demetrius, 1983; Arnold et al., 1994)

$$\log \lambda = \lim_{n\to\infty}\frac{1}{n}\log Z_n(\varphi). \tag{A.7}$$

By evaluating the asymptotic mean and variance of the random variable X_n induced by $S_n(\varphi)$, we can derive the population variables: reproductive potential Φ, and the demographic variance σ^2. These quantities are aggregates of the age-specific birth and death rates, the "microstates" of the population process.

We have

$$\lim_{n\to\infty}\frac{1}{n}E(X_n) = \int \varphi d\tilde{\mu}, \tag{A.8}$$

where $\varphi = \log a_{x_0x_1}$,

$$\lim_{n\to\infty}\frac{1}{n}Var(X_n) = \sum_{k=0}^{\infty}\rho_k, \tag{A.9}$$

where

$$\rho_k = \int\left(\log a_{x_{k+1}x_k} - \int \log a_{x_{k+1}x_k}\, d\tilde{\mu}\right) \times\left(\log a_{x_1x_0} - \int \log a_{x_1x_0}\, d\tilde{\mu}\right) d\tilde{\mu}.$$

The expressions (A.8) and (A.9) can be explicitly computed for the potential function φ induced by the Leslie matrix defined by (A.1) (see Demetrius and Gundlach, 1999).

Thus, the mean value described by (A.8), called the reproductive potential, and denoted Φ, assumes the explicit form:

$$\Phi = \frac{\sum p_j \log V_j}{\sum jp_j}.$$

The variance term given by (A.9), and called the demographic variance and denoted σ^2, can also be expressed in terms of the age-specific demographic variables. We obtain

$$\sigma^2 = \frac{\sum p_j W_j^2}{\sum jp_j},$$

where $W_j = -j\Phi + \log V_j$.

These quantities are the discrete analogs of Eqs. (4) and (9).

A.1.3. Demographic variance and the central limit theorem

The demographic variance as described by Eq. (A.9) will play a fundamental role in the diffusion analysis. We now briefly describe the mathematical rationale for this role.

We first observe that, by the ergodic theorem (see, for example, Demetrius et al., 2004):

$$\lim_{n\to\infty}\frac{1}{n}S_n(\varphi(x)) = \int \varphi\, d\tilde{\mu}.$$

This equation asserts that if choose a genealogy $x = (x_0, x_1, x_2, \ldots)$ then with probability one the long term mean of its net-offspring production equals the normalized average Φ taken over all genealogies with respect to $\tilde{\mu}$.

Now write

$$P_n(\varphi) = \left|\frac{1}{n}S_n(\varphi(x)) - \Phi\right|.$$

The quantity $P_n(\varphi)$ represents the deviation of the sample mean $\frac{1}{n}S_n(\varphi(x))$ from the normalized average Φ. We can apply the central limit theorem for dynamical systems (see Demetrius et al., 2007) to study the fluctuations

$$\frac{1}{n}S_n(\varphi(x)) - \Phi. \tag{A.10}$$

The central limit theorem implies that asymptotically, the deviations of the sample path S_n from the mean for $n \to \infty$ can be approximated by Brownian motion with variance $\sigma^2 t$, if we consider a continuous time evolution S_t, $t\varepsilon\mathbb{R}$. This observation is the basis for invoking the variance described by Eq. (A.9) in the description of the diffusion equation.

A.2. Diffusion equation

The derivation of the diffusion equation draws from the observations described in *Preliminaries*, in particular A.1.3.

We will consider $N(t)$, the population size at time t, as the solution of a diffusion equation and hence we will derive the infinitesimal moments for this process:

$$\lim_{\Delta t\to 0}\left(\frac{1}{\Delta t}\right)E\{N(t+\Delta t) - Nt|N(t) = N\} = r(0)N,$$

$$\lim_{\Delta t\to 0}\left(\frac{1}{\Delta t}\right)E\{N(t+\Delta t) - N(t))^2|N(t) = N\} = N^2\lim_{\Delta t\to 0}\left(\frac{1}{\Delta t}\right)\frac{\sigma^2\Delta t}{N} = \sigma^2 N,$$

where we denote by E the expectation.

In determining the first infinitesimal moment we assume that the fluctuations described by (A.10) have mean zero.

In the determination of the second infinitesimal moment we invoke the observation that, if N becomes large, the influence of fluctuations becomes small and hence the process becomes deterministic. Thus, we make the assumption that the change in $\log N(t)$ in the time interval Δt due

to fluctuations is caused by Brownian motion with variance $\sigma^2\Delta t/N(t)$. We can therefore describe the evolution of the density $f(N,t)$ of the process $N(t)$ by the solution of the Fokker–Planck equation:

$$\frac{\partial f}{\partial t} = -r(0)\frac{\partial(fN)}{\partial N} + \frac{\sigma^2(0)}{2}\frac{\partial^2(fN)}{\partial N^2}.$$

Equivalently, we could characterize $N(t)$ as a solution of the stochastic differential equation:

$$dN = rN\,dt + \sigma\sqrt{N}\,dW_t. \tag{A.11}$$

We get respective representations for the process $N^*(t)$ and its density $f^*(N^*,t)$, namely,

$$\frac{\partial f^*}{\partial t} = -r(\delta)\frac{\partial(f^*N^*)}{\partial N^*} + \frac{\sigma^2(\delta)}{2}\frac{\partial^2(f^*N^*)}{\partial N^{*2}},$$

and

$$dN^* = r(\delta)N^*dt + \sigma(\delta)\sqrt{N^*}dW_t^*. \tag{A.12}$$

We assume that the processes $N(t)$ and $N^*(t)$ evolve simultaneously and stochastically independent, that is, W and W^* are independent Wiener processes.

Define $M(t) = N(t) + N^*(t)$ and $p(t) = N^*(t)/M(t) = N^*(t)/(N(t)+N^*(t)) = 1-(N(t)/(N(t)+N^*(t)))$.

Then, Eqs. (A.11) and (A.12) are equivalent to

$$dM = (r + p\Delta r)M\,dt + \sigma\sqrt{(1-p)M}\,dW_t + \sigma(\delta)\sqrt{pM}\,dW_t^*, \tag{A.13}$$

$$dp = p(1-p)\left(\Delta r - \frac{\Delta\sigma^2}{M}\right)dt - \sigma p\sqrt{\frac{1-p}{M}}dW_t + \sigma(\delta)(1-p)\sqrt{\frac{p}{M}}dW_t^*. \tag{A.14}$$

The derivation of (A.13) is immediate: we observe, using the equation for M that $rN + r(\delta)N^* = r(N+N^*) + \Delta rN^* = rM + \Delta rpM$.

Eq. (A.14) can be obtained by Ito's formula. From the relations

$$\frac{\partial p}{\partial N} = -\frac{N^*}{M^2},\quad \frac{\partial p}{\partial N^*} = \frac{N}{M^2},\quad \frac{\partial^2 p}{(\partial N)^2} = \frac{2N^*}{M^3},$$
$$\text{and}\quad \frac{\partial^2 p}{(\partial N^*)^2} = -\frac{2N}{M^3},$$

we have

$$dp = \left[rN\frac{\partial p}{\partial N} + r(\delta)N^*\frac{\partial p}{\partial N^*} + \frac{1}{2}\times\left((\sigma\sqrt{N})^2\frac{\partial^2 p}{(\partial N)^2} + (\sigma(\delta)\sqrt{N^*})^2\frac{\partial^2 p}{\partial N^{*2}}\right)\right]dt + \sigma\sqrt{N}\frac{\partial p}{\partial N}\,dW_t + \sigma(\delta)\sqrt{N^*}\frac{\partial p}{\partial N^*}\,dW_t^*$$
$$= \left[-rp(1-p) + r(\delta)p(1-p) + \frac{\sigma^2 p(1-p)}{M} - \frac{\sigma(\delta)^2 p(1-p)}{M}\right]dt - \frac{\sigma N^*\sqrt{N}}{M^2}\,dW_t + \frac{\sigma(\delta)N\sqrt{N^*}}{M^2}\,dW_t^*.$$

Hence,

$$dp = p(1-p)\left(\Delta r - \frac{\Delta\sigma^2}{M}\right)dt - \sigma p\sqrt{\frac{1-p}{M}}dW_t + \sigma(\delta)(1-p)\sqrt{\frac{p}{M}}dW_t^*.$$

We are mainly interested in the problem of invasion or extinction of the mutant type. We express the interaction between the two types by assuming that the total population size $M(t)$ is constant for $t<t_0$, where t_0 represents the instant at which resources or spatial constraints begin to operate. The invasion event is analyzed for small $N^* \ll M$ and N close to equilibrium. Consequently, the changes in N^* and N will be at the expense of each other. This implies that the changes in M will be negligibly small. In the sequel we will assume that M is constant.

We can now state the main result: let M be a constant. Eq. (A.14) generates a diffusion process with drift

$$\alpha(p) = p(1-p)\left(\Delta r - \frac{\Delta\sigma^2}{M}\right), \tag{A.15}$$

and variance

$$\beta(p) = \frac{p(1-p)}{M}(\sigma^2 p + \sigma(\delta)^2(1-p)). \tag{A.16}$$

The diffusion process has a density ψ solving the Fokker–Planck equation:

$$\frac{\partial\psi}{\partial t} = -\frac{\partial[(\alpha(p)\psi]}{\partial p} + \frac{1}{2}\frac{\partial^2[\beta(p)\psi]}{\partial p^2}. \tag{A.17}$$

The drift term in (A.17) is an immediate consequence of (A.14).

In order to obtain the variance term in (A.17) we observe that in (A.14) the Wiener processes W_t and W_t^* are independent, which yields a diffusion coefficient

$$\beta(p) = \left(\sigma p\sqrt{\frac{1-p}{M}}\right)^2 + \left(\sigma(\delta)(1-p)\sqrt{\frac{p}{M}}\right)^2 = \frac{p(1-p)}{M}(\sigma^2 p + \sigma(\delta)^2(1-p)).$$

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