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Mortality plateaus and directionality theory

Lloyd Demetrius

Department of Organismic and Evolutionary Biology, Harvard University, Cambridge, MA 02138, USA (ldemetr@oeb.harvard.edu)

Recent large scale studies of senescence in animals and humans have revealed mortality rates that levelled off at advanced ages. These empirical findings are now known to be inconsistent with evolutionary theories of senescence based on the Malthusian parameter as a measure of fitness. This article analyses the incidence of mortality plateaus in terms of directionality theory, a new class of models based on evolutionary entropy as a measure of fitness. We show that the intensity of selection, in the context of directionality theory, is a convex function of age, and we invoke this property to predict that in populations evolving under bounded growth constraints, evolutionarily stable mortality patterns will be described by rates which abate with age at extreme ages. The explanatory power of directionality theory, in contrast with the limitations of the Malthusian model, accords with the claim that evolutionary entropy, rather than the Malthusian parameter, constitutes the operationally valid measure of Darwinian fitness.

Keywords: directionality theory; Darwinian fitness; Malthusian parameter; entropy; senescence

1. INTRODUCTION

Recent studies of mortality rates at extreme ages based on a wide variety of species have shown that the Gompertzian model, a condition which describes a death rate which increases exponentially with age, is not a universal descriptor of mortality patterns. Analysis of the survivorship curves of large populations of medflies, nematodes and humans, now indicates a large diversity in life-history patterns, in particular, mortality rates which abate with age at extreme ages, giving rise to what is now called a mortality plateau (Carey *et al.* 1992; Brooks *et al.* 1994; Smith 1994). These observations have generated considerable interest among demographers because they are inconsistent with theories of the evolution of senescence proposed by Medawar (1952), Williams (1957) and Hamilton (1966), among others. This paper delineates the basis for this inconsistency, and furthermore shows that the observed mortality plateaus can be explained in terms of a new class of evolutionary models, called directionality theory (Demetrius 1997).

Classical studies of the senescence process are based on an evolutionary model, the Malthusian theory, which essentially goes back to Fisher (1930). The Malthusian theory asserts that the evolutionarily stable states of a population are described by fecundity and mortality distributions which maximize the population growth rate. In the context of this theory, the intensity of selection, i.e. the ability to discriminate between alternative genotypes, will be measured by the sensitivity of the Malthusian parameter to changes in the age-specific fecundity and mortality schedule (Hamilton 1966). Now, in exponentially increasing populations, the sensitivity of the population growth rate to changes in the age-specific net-fecundity variables will decrease with age. Accordingly, this index of selective intensity declines with age (Partridge & Barton 1993; Rose & Mueller 2000). This condition entails that mortality rates will be an exponentially increasing function of age—a property which is inconsistent with the observed deceleration of mortality rates at extreme ages.

In a recent effort to reconcile the existence of mortality plateaus with the Malthusian theory, Mueller & Rose (1996) have introduced several stochastic processes within the context of the Malthusian model to describe long-term changes in demographic rates. These studies, which are based on antagonistic pleiotropy and mutation accumulation as the driving genetic mechanisms, claim that the mortality patterns generated will be described by an exponential increase at early ages and a levelling off at later ages. These claims have recently been contested. Wachter (1999) has developed a general class of Markovian models which includes the Mueller–Rose systems, and has shown that although the transient states visited early by the stochastic processes do display mortality plateaus, the limiting states do not. Computational studies by Charlesworth & Partridge (1997) and Pletcher & Curtsinger (1998), also conflict with the claims advanced by Mueller & Rose (1996).

The notion that the Malthusian parameter, denoted r , characterizes fitness, i.e. the contribution of a type to the ancestry of successive generations, drives most current models of the evolution of life history. This characterization of fitness derives from analytical studies of the invasion process for mutant alleles in structured populations (Charlesworth & Williamson 1975; Pollack 1976), and is expressed in terms of the Malthusian principle: *the establishment of a mutant allele in a resident population is a deterministic process given by relative values of the Malthusian parameter.* We have:

- (i) if $\Delta r > 0$, the mutant invades;
- (ii) if $\Delta r < 0$, the mutant becomes extinct.

Studies based on the ergodic theory of dynamical systems (Demetrius 1997; Demetrius & Gundlach 1999) show that the Malthusian principle only holds in the limiting case when the population is exponentially increasing, and size is effectively infinite. When population size is finite—the typical condition—it was shown that Darwinian fitness is characterized by evolutionary entropy. This demographic parameter describes the variability in the age of reproducing individuals in the population. The significance of

entropy in an evolutionary context resides in the following property: it characterizes demographic stability, the rate of decay of fluctuations in population numbers due to small variations in the age-specific birth and death rates.

In studies of the invasion process (Demetrius & Gundlach 1999; Demetrius 2000), we have assumed that population size is finite, and we have distinguished between the different modes of growth a population may experience as a consequence of ecological constraints: *bounded growth* refers to populations which spend the greater part of their life history in the stationary phase, or with a population size which fluctuates around some constant number, whereas *unbounded growth* pertains to populations which spend the greater part of their life history in the exponential phase. Our analysis of the invasion process showed that the outcome of selection between mutant and incumbent is contingent on this class of ecological constraint, and determined by entropy. The selective outcome can be expressed in terms of the entropy principle: *the establishment of a mutant allele in a resident population is a stochastic process determined by relative values of entropy*. The correspondence between ecological constraints and invasion conditions can be described as follows.

- (i) Bounded growth constraints:
 - (a) $\Delta H > 0$, mutant invades almost surely (a.s.);
 - (b) $\Delta H < 0$, mutant becomes extinct (a.s.).
- (ii) Unbounded growth constraints, large population size:
 - (a) $\Delta H < 0$, mutant invades (a.s.),
 - (b) $\Delta H > 0$, mutant becomes extinct (a.s.).
- (iii) Unbounded growth constraints, small population size:
 - (a) $\Delta H < 0$, mutant invades with a probability that increases with population size;
 - (b) $\Delta H > 0$, mutant becomes extinct with a probability that increases with population size.

As observed in Demetrius (2000) (see Appendix A), the two invasion criteria are related: the Malthusian principle is the limit ($M \rightarrow \infty$, M being population size) of the entropy principle. Accordingly, directionality theory, the dynamic theory of evolution which revolves around entropy as a measure of Darwinian fitness, subsumes the Malthusian theory and provides a general framework for understanding the dynamics of invasion in structured populations.

This paper outlines the evolutionary model of mutation and selection that generates the tenets embodied in the entropy principle. We will then show that the intensity of selection, as measured by the sensitivity of entropy to changes in the age-specific fecundity and mortality variables, is a convex function of age. 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. As we observed, entropy determines demographic stability. The convexity condition of the entropy sensitivity function thus reflects the fact that the response of demographic stability to changes in the net-fecundity variables will be strong in the early and later age classes where the net-fecundity is relatively small, but weak at intermediate

ages where the net-fecundity is relatively large. This representation of selection intensity in terms of entropy will be invoked to study evolutionary trends in the life-history variables. In our analysis, we will assume that genes have pleiotropic effects. We will then exploit the directionality principles for evolutionary entropy (Demetrius 1997) to predict the following correlations between the ecological constraints that a population experiences and its evolutionarily stable mortality patterns. We have:

- (i) populations which evolve under bounded growth constraints—*mortality rates which abate with age at advanced ages*;
- (ii) populations which evolve under unbounded growth constraints—*mortality rates which increase exponentially with age*.

Most natural populations will be subject to ecological conditions which correspond to bounded growth constraints. In view of this condition, directionality theory predicts that for most natural populations mortality patterns will be characterized by rates which decelerate at advanced ages. This prediction, which is consistent with the observed empirical data, stands in sharp contrast to the corresponding tenet of the Malthusian theory, which asserts that mortality rates will increase exponentially with age. The explanatory power of directionality theory, in contrast to the limitations of the Malthusian theory, provides strong support to the general claim that evolutionary entropy, rather than the Malthusian parameter, represents the operationally valid measure of Darwinian fitness.

The contrast between the main elements of the Malthusian theory and directionality theory are summarized in table 1.

2. DIRECTIONALITY THEORY

Directionality theory studies the evolutionary dynamics of populations structured by age and described by the following parameters: $l(x)$, the probability that an individual survives to age x ; $m(x)$, the mean number of offspring produced by an individual of age x . The product $l(x)m(x)$ is called the net-reproductive function and is denoted $V(x)$.

We will assume that $V(x)$ is a concave function with a turning point, as in figure 1. This property describes most natural populations.

The growth rate of a population, called the Malthusian parameter and denoted r , is the unique real root of the equation

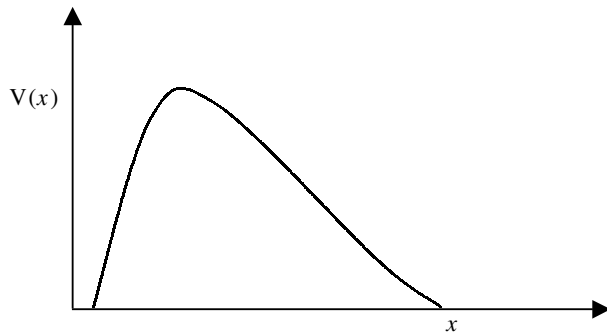
$$1 = \int_0^{\infty} \exp(-rx) V(x) dx. \quad (2.1)$$

The function $p(x) = \exp(-rx) V(x)$ is a probability density function that describes the age of the mother of a randomly chosen newborn. The generation time T , the mean age of mothers at the birth of their offspring, is given by $T = \int_0^{\infty} xp(x) dx$. The population growth rate r can be expressed as the sum of two functions, evolutionary entropy H and the reproductive potential Φ ;

$$r = H + \Phi. \quad (2.2)$$

Table 1. Malthusian theory and directionality theory: a contrast.

evolutionary property	Malthusian theory	directionality theory
measure of fitness	population growth rate	entropy
intensity of selection	decreasing function of age	convex function of age
invasion criteria	<i>deterministic</i> : relative values of the Malthusian parameter	<i>stochastic</i> : relative values of entropy
evolutionarily stable states	maximal values of the Malthusian parameter	extremal (maximal and minimal) values of entropy

Figure 1. The net-fecundity function $V(x)$ versus age x .

Here,

$$H = -\frac{\int_0^{\infty} p(x) \log p(x) dx}{\int_0^{\infty} xp(x) dx}; \quad \Phi = \frac{\int_0^{\infty} p(x) \log V(x) dx}{\int_0^{\infty} xp(x) dx}. \quad (2.3)$$

The numerator in the expression for H is the variability in the age of reproducing individuals in the population, whereas for Φ , it is the total net-offspring production averaged over the different age classes. We observe from (2.2) that the following implications hold:

$$\Phi < 0 \Rightarrow r < H; \quad \Phi > 0 \Rightarrow r > H. \quad (2.4)$$

Accordingly, the function Φ can be used to classify populations in terms of their growth rate: $\Phi < 0$ (bounded growth) describes a population whose growth rate is bounded by entropy—a condition which evidently includes populations with stationary size; $\Phi > 0$ (unbounded growth) represents a population whose growth rate always exceeds entropy. The condition $\Phi < 0$ characterizes populations which spend the greater part of their evolutionary history with size fluctuating around some constant value; the constraint $\Phi > 0$ describes populations which spend the greater part of their evolutionary history in the exponential growth phase.

According to directionality theory, evolution is a concatenation of two processes, each unfolding on distinct time-scales. The first process, which unfolds on a short time-scale, involves the introduction of new types in the population by mutation. Competition between the mutant (initially rare) and the ancestral type will result in either the extinction of the mutant or its increase in frequency. In the event that the mutant increases in frequency, new types will be generated by mating between the mutant and ancestral types, creating a

population consisting of diverse genotypes. The second process, which now unravels on a longer time-scale, involves the selective dynamics of the competing genotypes under the existing ecological constraints, bounded or unbounded growth conditions.

The mathematical model of this process focuses on the shift in entropy which occurs as the composition of the population changes under the mutation–selection regime. The model considers a population at demographic equilibrium, described in terms of a stable age distribution and an entropy H . Mutation occurs in a small subset of the population. This process induces changes in the fecundity and mortality of individuals in the subpopulation, which is now described by a new demographic state with entropy that we will denote H^* .

Mutations are assumed to have pleiotropic effects, i.e. the phenotypic response to allelic changes is not localized at some distinct age but will be expressed with varying intensity over the complete life cycle of the organism. The pleiotropic condition entails that post-reproductives may be beneficiaries of the effects of natural selection, a force which acts only on individuals in the reproductive phase. (For a discussion of the empirical support and evolutionary significance of pleiotropy, see Rose (1991).)

In view of the pleiotropic assumption, a mutation that affects survivorship will cause a change in the distribution $l(x)$, thus generating a new distribution $l^*(x)$. Changes that affect fecundity will be described similarly. The independent changes in the survivorship and fecundity will have an effect on the net-fecundity distribution $V(x)$. Our model assumes that these changes are characterized by the form $V^*(x) = V^{1+\delta(x)}(x)$, where $\delta(x)$ is monotonic in x ; see figure 2.

The monotonicity condition, which we impose uniquely on the net-fecundity function instead of the fecundity and the mortality distributions, (see Demetrius 1992), is prescribed to exclude atypical allelic changes; namely, variations which, for example, exert positive effects on the net-fecundity function at early and late reproductive ages but have negative effects at intermediate ages.

The interaction of the mutant subpopulation (entropy H^*) with the incumbent (entropy H), in the event that the mutant increases in frequency, will result in driving the composite population to a new state of demographic equilibrium, defined by the entropy H' . The directionality theorems for entropy annotate the changes in entropy, from the initial state H to the new state H' .

The analyses for constant environment models (Demetrius 1992) and for random matrix models (Arnold *et al.* 1994; Demetrius & Gundlach 1999) show that these changes are contingent on the ecological constraints the

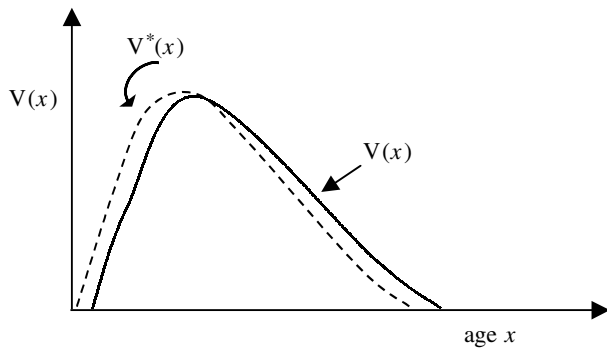


Figure 2. Mutational changes in the net-fecundity function $V(x)$.

population endures. The relation between ecological constraints and directional trends in entropy which are predicted by the theory are delineated as follows:

- (i) populations with bounded growth—a *unidirectional increase in entropy*;
- (ii) populations with unbounded growth and large size—a *unidirectional decrease in entropy*;
- (iii) populations with unbounded growth and small size—*random non-directional changes in entropy*.

The directionality theorems expressed by items (i)–(iii) refer to global changes in entropy over evolutionary time. An immediate consequence of the theorems is a set of correlations between the ecological conditions which impinge on the population and determine its growth rate, and its age-specific fecundity and mortality distribution at evolutionary equilibrium. These correlations are described as follows:

- (iv) in the case of populations evolving under bounded growth constraints, the evolutionarily stable life history is characterized by fecundity–mortality distributions which maximize entropy;
- (v) in populations subject to unbounded growth constraints, the evolutionarily stable life history is characterized by distributions which minimize entropy.

The directionality principles, as expressed by (i)–(iii) and the optimality principle for entropy, as described by (iv) and (v) will provide a basis for explaining from an evolutionary perspective the different mortality patterns observed in natural populations.

3. INTENSITY OF SELECTION

The directionality principles indicate that entropy parameterizes the direction of evolutionary change by natural selection. Accordingly, the intensity of natural selection will be determined by the sensitivity of entropy to changes in the age-specific fecundity and mortality functions. We now derive analytic expressions for the sensitivity relations using the method of functional differentiation (see Luenberger (1969) for the underlying theory). We refer to Demetrius *et al.* (2001) for a similar analysis in terms of the Leslie model.

(a) Sensitivity analysis

Functional differentiation of the equation for the growth rate r given by (2.1) can be applied to determine the corresponding point derivatives, which we denote by $\partial r/\partial V_x$, $\partial r/\partial l_x$ and $\partial r/\partial m_x$. We obtain the well known relation

$$\frac{\partial r}{\partial V_x} = \frac{\exp(-rx)}{T}. \quad (3.1)$$

We infer from (3.1) that

$$\frac{\partial r}{\partial l_x} = \frac{\exp(-rx)m(x)}{T}; \quad \frac{\partial r}{\partial m_x} = \frac{\exp(-rx)l(x)}{T}. \quad (3.2)$$

We shall now apply functional differentiation to the entropy function H to obtain the analogue of equation (3.1), namely

$$\frac{\partial H}{\partial V_x} = -\frac{\exp(-rx)}{T}S(x), \quad (3.3)$$

and infer the corresponding derivatives

$$\frac{\partial H}{\partial l_x} = -\frac{\exp(-rx)m(x)}{T}S(x); \quad \frac{\partial H}{\partial m_x} = -\frac{\exp(-rx)l(x)}{T}S(x), \quad (3.4)$$

where

$$S(x) = W(x) - U, \quad (3.5)$$

and

$$W(x) = -x\Phi + \log V(x); \quad U = \frac{1}{T} \int_0^\infty xp(x)W(x)dx. \quad (3.6)$$

To evaluate the sensitivity of H with respect to point changes in $V(x)$, we introduce the new function S defined by $S = HT$. We have

$$S = - \int_0^\infty p(x)q(x)dx,$$

where $q(x) = \log p(x)$. Functional differentiation of the function S and the generation time T yields

$$\frac{\partial S}{\partial V_x} = -\frac{p(x)q(x)}{V(x)} + \frac{1}{T} \frac{p(x)}{V(x)} \int_0^\infty xp(x)q(x)dx \quad (3.7)$$

and

$$\frac{\partial T}{\partial V_x} = \frac{xp(x)}{V(x)} - \left(\frac{1}{T}\right) \frac{p(x)}{V(x)} \int_0^\infty x^2p(x)dx. \quad (3.8)$$

From equations (3.7) and (3.8) and by appealing to the expression $S = HT$ we obtain

$$T \frac{\partial H}{\partial V_x} = -\frac{p(x)}{V(x)} [xH + q(x)] + \left(\frac{p(x)}{V(x)}\right) \left(\frac{1}{T}\right) \int_0^\infty (xH + q(x))xp(x)dx.$$

Now in view of the identity $r = H + \Phi$, we have

$$xH + q(x) = -x\Phi + \log V(x).$$

Table 2. Life-table and sensitivity functions for Sweden in 1835.

age x	$l(x)$	$m(x)$	$\partial H/\partial m_x$	$\partial H/\partial l_x$	$\partial H/\partial V_x$
1	1.00000	0.00000			
2	0.69283	0.00000			
3	0.94844	0.00000			
4	0.62904	0.04165	0.22830	0.15116	0.36293
5	0.61064	0.25623	0.06290	0.02639	0.10301
6	0.58855	0.50361	-0.00180	-0.00154	-0.00306
7	0.56362	0.57977	-0.02230	-0.02294	-0.03957
8	0.53488	0.49613	-0.02100	-0.01948	-0.03926
9	0.50710	0.26653	0.00630	0.00331	0.01242
10	0.47528	0.06674	0.06820	0.00958	0.14349
11	0.44587	0.00000			
12	0.41132	0.00000			
13	0.37164	0.00000			
14	0.31420	0.00000			
15	0.22778	0.00000			
16	0.14411	0.00000			
17	0.07832	0.00000			
18	0.03328	0.00000			

Hence

$$T \frac{\partial H}{\partial V_x} = -\frac{\rho(x)}{V(x)} (-x\Phi + \log V(x)) + \frac{\rho(x)}{V(x)} \frac{1}{T} \int_0^\infty (-x\Phi + \log V(x)) x \rho(x) dx.$$

Substituting the expressions for $S(x)$ given in equation (3.5) and $W(x)$, and U given in equation (3.6), we immediately obtain equation (3.3).

(b) Convexity properties

We first observe that since $V(x)$ is concave, we have $V''(x) < 0$. By computing the second derivative of $W(x)$, we observe that $W''(x) < 0$; hence $S(x)$ is also a concave function of age. We will now show that the convexity properties of the function $S(x)$ are inherited by the sensitivity functions given by equations (3.3) and (3.4). We shall distinguish between stationary ($r = 0$), and exponentially growing populations ($r > 0$).

- (i) *Stationary growth constraints.* We observe from equation (3.3) that $\partial H/\partial V_x = -S(x)/T$, and conclude that $\partial H/\partial V_x$ is a convex function of age. In view of the shape of the net-fecundity distribution $V(x)$, which the function $S(x)$ necessarily inherits, the function $\partial H/\partial V_x$ will first decrease with age until it attains a certain minimum value, and then increase with age.
- (ii) *Exponentially growing populations.* The analytical results derived for the case $r = 0$ will not necessarily be inherited by the sensitivity functions when $r > 0$. This derives from the fact that the convexity property of $S(x)$ is not necessarily preserved by the product $\exp(-rx)S(x)$. However, studies of a large family of life tables indicate that the convexity properties derived in the case $r = 0$ persist also in the case $r > 0$. These numerical studies have also been invoked to investigate the geometric properties of the sensitivity functions $\partial H/\partial l_x$, and $\partial H/\partial m_x$ as given in equations (3.4). These studies indicate that

the patterns characterizing $\partial H/\partial l_x$ and $\partial H/\partial m_x$ are similar to the patterns which define the function $\partial H/\partial V_x$.

In table 2 we give the $l(x)$ and $m(x)$ schedules for Sweden in 1835 and present numerical values for the sensitivity functions. The convexity patterns described by $\partial H/\partial V_x$, $\partial H/\partial l_x$ and $\partial H/\partial m_x$ are similar. In each case, three distinct phases of the age classes can be identified:

- phase I: $\partial H/\partial V_x > 0$ and decreases sharply with age;
- phase II: $\partial H/\partial V_x < 0$;
- phase III: $\partial H/\partial V_x > 0$ and increases sharply with age.

We can now invoke the changing patterns which the intensity of selection, as described by $\partial H/\partial l_x$ and $\partial H/\partial m_x$, to predict the evolution of the fecundity and mortality distributions. The directionality principles, as described in §2 by (i)–(iii), indicate that the evolution of the life-history patterns will be modulated by the ecological constraints the population experiences. We therefore distinguish between the two modes of demographic response—bounded and unbounded growth.

(i) Bounded growth

When this ecological condition prevails, evolution by mutation and natural selection will result in changes in the functions $m(x)$ and $l(x)$ that *increase* H . Because $\partial H/\partial m_x$ and $\partial H/\partial l_x$ are both convex functions of age, the changes generated in both $m(x)$ and $l(x)$ will be described by an increase in the early and later age-classes. In the case of the function $m(x)$, this will result in a more uniform distribution (figure 3a); in the case of $l(x)$ we obtain a survivorship curve with an extended tail (figure 3b). The corresponding mortality curve will be described by a plateau at extreme ages, reflecting a mortality rate which abates with age at extreme ages.

(ii) Unbounded growth

Under these demographic constraints, evolution by mutation and natural selection will result in changes in $m(x)$ and $l(x)$ that *decrease* H (we assume that population sizes are large). In view of the convexity of $\partial H/\partial m_x$ and $\partial H/\partial l_x$ we infer that the changes generated in both $m(x)$ and $l(x)$ will now be a decrease in the early and later age-classes. The distribution $m(x)$ will become less uniform, whereas the distribution $l(x)$ will be characterized by a survivorship curve with a restricted tail (see figure 3c,d). The corresponding mortality curve will be represented by a mortality rate which increases exponentially with age.

(c) Fecundity–mortality patterns

Our analysis thus provides a quantitative explanation of the diversity of fecundity–mortality distributions observed in natural populations. Our study shows that these distributions are contingent on the ecological conditions that the populations experience during their evolutionary history. Mortality rates which abate with age at extreme ages, and fecundity distributions which are spread out over several age-classes are correlated—these patterns are generated under conditions where growth is bounded. Mortality rates which increase exponentially with age, and fecundity distributions which are restricted to a compact age range are also correlated—these

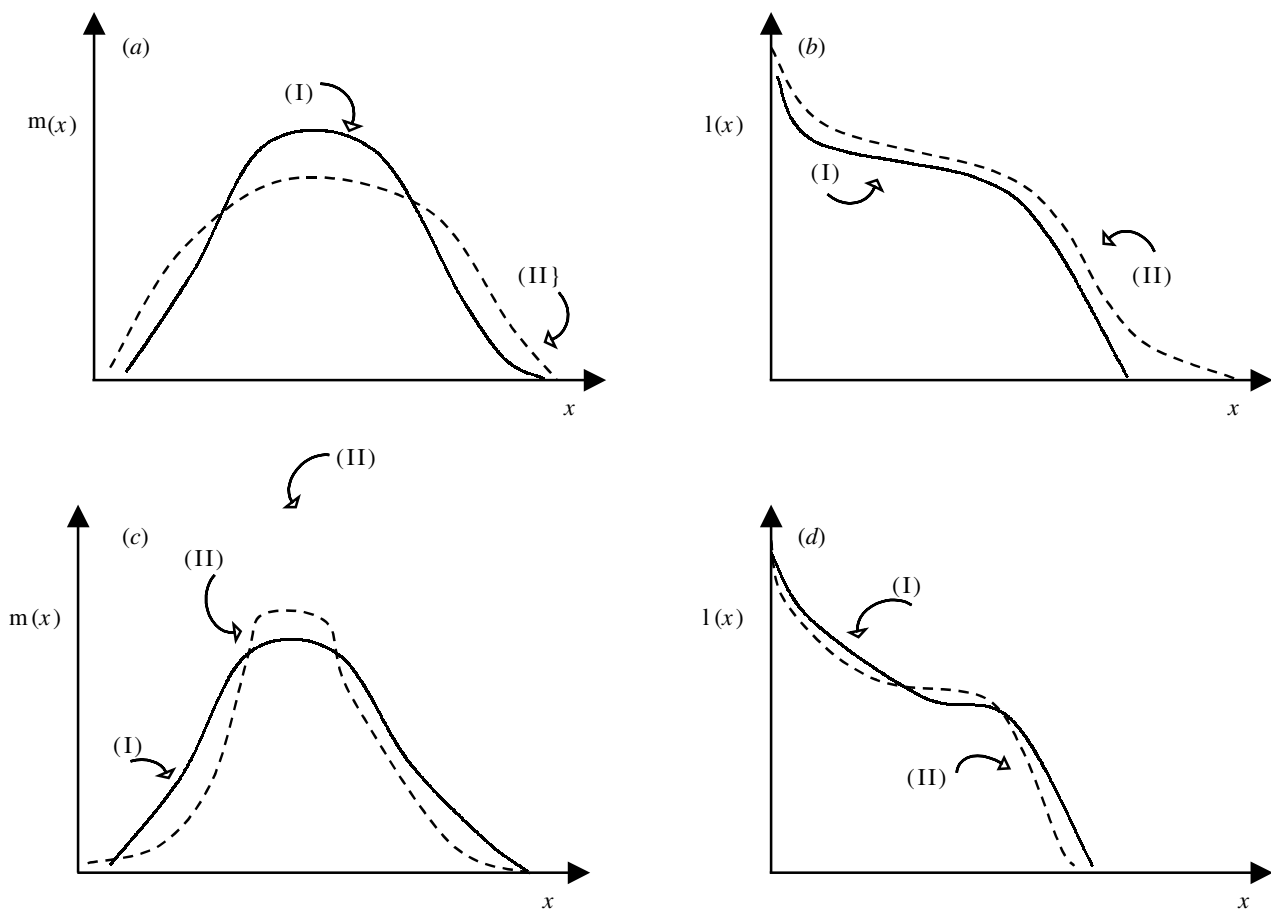


Figure 3. Effect of growth constraints on the evolution of life-history patterns: (a) changes in $m(x)$ and (b) changes in $l(x)$ under bounded conditions; (c) changes in $m(x)$ and (d) changes in $l(x)$ under unbounded conditions. (I) initial state; (II) final state.

distributions are induced by evolution under conditions of unbounded growth.

We should observe that the analysis we have developed is based on the notion that entropy H determines Darwinian fitness; hence the intensity of selection will be given by $\partial H/\partial V_x$, $\partial H/\partial m_x$ and $\partial H/\partial l_x$. Now in the context of the Malthusian theory, the intensity of selection will be described by $\partial r/\partial V_x$, $\partial r/\partial m_x$ and $\partial r/\partial l_x$. It is evident from equations (3.1) and (3.2) that these expressions are decreasing functions of age. Now, according to the Malthusian theory, evolution tends to increase r ; consequently, mortality curves will be represented by a mortality rate which increases with age—a condition which conflicts with the existence of mortality plateaus at advanced ages.

4. DISCUSSION

Directionality theory is particularly pertinent in predicting old-age mortality rates in modern human populations. We first observe that, until 200 years ago, the size of the human population remained fairly stable because high birth rates were balanced by high death rates, with growth rate showing an increase only in more recent times. However, as the demographic studies of Demetrius & Ziehe (1984) and Demongeot & Demetrius (1989) indicate, although growth rates in recent times have increased exponentially, the rates are typically bounded by entropy. Human populations can therefore

be considered to have been subject to bounded growth constraints throughout their evolutionary history. We can therefore appeal to directionality theory to predict that if mortality is due primarily to senescent causes, as is becoming increasingly the case in economically advanced countries, the pace of longevity improvement at older ages will show a significant increase.

Empirical studies are consistent with these predictions. These studies show that during the second half of the twentieth century, age-specific death rates in the industrialized countries have shown a significant decline (Vaupel 1997; Wilmoth 1998). Assuming that the pace of mortality decline will continue in the future, Tuljapurkar *et al.* (2000) forecast that mean life span is likely to increase faster than predicted by official forecasts. The pertinence of this claim partly hinges on the validity of the constancy assumption of the pace of mortality decline. Directionality theory provides new perspectives on the strength of this assumption. In the context of this theory, the pace of mortality decline in industrialized countries will be constrained by the sensitivity of entropy to changes in the net-fecundity variables. In low entropy populations, this sensitivity will be relatively large; in high entropy populations, it will be relatively small. Industrialized countries vary both in terms of their entropy and the rate at which entropy changes over time (see Demetrius & Ziehe (1984) for a comparative study of trends in entropy for Sweden and France). In view of the temporal changes in entropy, the pace of mortality

decline may vary over time and the constancy assumption invoked in extrapolative forecasts may not hold. Directionality theory presents a formalism for determining the magnitude of deviations from constancy. This theory provides a conceptual framework for the extrapolative approach to prediction, and can be exploited to yield more reliable forecasts of life span in industrialized countries.

As Vaupel (1997) has emphasized, the pace of mortality decline at older ages has implications for social and health policy. Hence accurate predictions of the growth rate of the elderly population have now become of increasing importance. The operational significance of entropy in this context underscores the relevance of directionality theory, not only as an explanatory model, but also as an instrument which can provide quantitative predictions in an area with large implications for biomedical research.

5. CONCLUSIONS

This paper has invoked directionality theory, a class of evolutionary models based on entropy as a measure of Darwinian fitness, to explain the deceleration in mortality rates at extreme ages observed in many natural populations. Our analysis rests on two observations.

- (i) In populations subject to bounded growth constraints, evolution by mutation and natural selection results in a unidirectional increase in entropy.
- (ii) The intensity of selection, as measured by the sensitivity of entropy to changes in the fecundity and mortality distribution is a convex function of age, with relatively strong effects during the earlier and later reproductive phases, and weak effects at intermediate ages.

APPENDIX A: INVASION CONDITIONS IN STRUCTURED POPULATIONS

The invasion–extinction dynamics of a mutant allele in a structured population can be characterized by appealing to:

- (i) certain perturbation relations of the set of demographic variables that describe the population;
- (ii) models of competition between ancestral and mutant populations using the techniques of diffusion processes.

(a) Perturbation relations

Demographic studies (Demetrius 1992) show that starting with the function

$$p(x) = \exp(-rx)V(x),$$

where r denotes the population growth rate and $V(x)$ is the net-reproductive function, we can generate the following series of macroscopic variables which characterize the population dynamics. We have:

- (i) the entropy H , the reproductive potential Φ , and the generation time T ;
- (ii) the demographic variance σ^2 given by

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

- (iii) the correlation index γ given by

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

Let Δr , ΔH , $\Delta\sigma^2$ denote the changes in the demographic variables which result from a mutation that induces a small change in the net-fecundity function. Perturbation analysis (Arnold *et al.* 1994) of the net-fecundity distribution shows that the changes Δr , ΔH and $\Delta\sigma^2$ are given by

$$\Delta r = \Phi\delta; \quad \Delta H = -\sigma^2\delta; \quad \Delta\sigma^2 = \gamma\delta, \quad (\text{A1})$$

where δ can be interpreted as a measure of the intensity of the mutation.

The demographic variance, by definition, is evidently always positive, whereas the correlation index γ may have positive or negative values. However, for typical life tables, i.e. concave net-fecundity distributions as in figure 1, numerical studies show that γ is positive. Hence, we obtain the following relations:

$$\Phi < 0 \Rightarrow \Delta r\Delta H > 0; \quad \Phi > 0 \Rightarrow \Delta r\Delta H < 0 \quad (\text{A2})$$

$$\Delta H\Delta\sigma^2 < 0. \quad (\text{A3})$$

(b) Diffusion processes and invasion dynamics

The condition for the ultimate establishment of a mutant in the population can be analysed in terms of diffusion processes. We refer to Demetrius & Gundlach (2000), for details. Important precursors to our study include the review by Feller (1951), the analyses of Wright (1945) and Kimura (1962), and their extensions due to Gillespie (1974). The analysis of these authors is based upon Wrightian models of population genetics—a class of models which ignores age-structure. The development given in Demetrius & Gundlach (1999) appeals to the ergodic theory of dynamical systems to extend the classical diffusion analysis to structured populations.

Let $p(t) = N^*(t)/[N^*(t) + N(t)]$, where $N^*(t)$ denotes the population size of the mutant and $N(t)$ the population size of the incumbent. Let $\psi(p, t)$ denote the probability density function of the process that describes the change in frequency p as a function of time. Assuming that the mutant is initially rare and that the total population size $M(t) = N^*(t) + N(t)$ remains constant during the invasion process, we showed that $\psi(p, t)$ satisfies the diffusion equation

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

where

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

and

Table A1. Invasion criteria in terms of $\Delta r, \Delta\sigma^2$ (finite size).

ecological constraint and demographic condition	selective outcome
$\Delta r > 0, \Delta\sigma^2 < 0$	invasion occurs a.s. ^a
$\Delta r < 0, \Delta\sigma^2 > 0$	extinction occurs a.s.
$\Delta r > 0, \Delta\sigma^2 > 0$	
$M > \gamma/\Phi$	invasion occurs a.s.
$M < \gamma/\Phi$	extinction with a probability, decreasing in M
$\Delta r < 0, \Delta\sigma^2 < 0$	
$M < \gamma/\Phi$	invasion with a probability, decreasing in M
$M > \gamma/\Phi$	extinction a.s.

^aa.s., Almost surely.

Table A2. Invasion criteria: ΔH (finite size).

ecological constraints	demographic condition	selective outcome
$\Phi < 0$ (bounded growth)	$\Delta H > 0$	invasion occurs a.s. ^a
	$\Delta H < 0$	extinction occurs a.s.
$\Phi > 0$ (unbounded growth)	$M > \gamma/\Phi$ (large size)	invasion occurs a.s.
	$M < \gamma/\Phi$ (small size)	extinction occurs a.s.
	$\Delta H < 0$	invasion with a probability, increasing in M
	$\Delta H > 0$	extinction with a probability, increasing in M

^aa.s., Almost surely.

Table A3. Invasion criteria: Δr (infinite size).

ecological constraint	demographic condition	selective outcome
$r > 0$ (exponential growth)	$\Delta r > 0$	invasion occurs
	$\Delta r < 0$	extinction occurs

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

Write

$$s = \Delta r - \frac{1}{M} \Delta\sigma^2. \quad (\text{A4})$$

The quantity s represents a measure of selective advantage.

Let $P(y)$ denote the probability that a mutant with initial frequency y invades the population. As shown in Demetrius & Gundlach (2000), $P(y)$ is given by

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

Moreover, the geometry of the function $P(y)$ is determined by the sign of s . We have

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

The above fact, together with certain analytic properties of $P(y)$, can be exploited to express the conditions for invasion in terms of the parameters Δr and $\Delta\sigma^2$. This is given in Table A1.

Now in view of the perturbation relations (A2) and (A3) we can reformulate the invasion criteria given in table A1 uniquely in terms of conditions on the entropy H . This is described in table A2. The expression a.s. (almost surely) in tables A1 and A2 means that the event occurs with a probability of approximately unity and is independent of population size.

We should observe at this point that when $M \rightarrow \infty$ the selective advantage s defined by equation (A4) reduces to

$$s = \Delta r. \quad (\text{A5})$$

Now, the invasion criteria given in table A2 and expressed in terms of ΔH are derived on the assumption that the population size M is finite. However, we now observe from A5 that, when $M \rightarrow \infty$, these criteria can be expressed uniquely in terms of Δr as shown in table A3. This observation is the basis for the claim that the Malthusian theory represents the limit ($M \rightarrow \infty$) of directionality theory.

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