

Original Articles

The force of selection on the human life cycle

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Abstract

In this article, I present evidence for a robust and quite general force of selection on the human life cycle. The force of selection acts in remarkably invariant ways on human life histories, despite a great abundance of demographic diversity. Human life histories are highly structured, with mortality and fertility changing substantially through the life cycle. This structure necessitates the use of structured population models to understand human life history evolution. Using such structured models, I find that the vital rates to which fitness is most sensitive are prereproductive survival probabilities, particularly the survival of children ages 0 to 4 years. The fact that the preponderance of selection falls on transitions related to recruitment combined with the late age at first reproduction characteristic of the human life cycle creates a fitness bottleneck out of recruitment. Because of this, antagonistic pleiotropy with any trait that detracts from the constituent transitions to recruitment is expected. I explore the predictors of variation in the force of selection on early survival. High fertility increases the selective premium placed on early survivorship, whereas high life expectancy at birth decreases it.

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1. Introduction

Understanding variation in the life cycle is a fundamental goal of modern evolutionary biology. Species differ in the schedules at which they develop, reproduce, and die, and the fact that these features of an organism's life cycle are so proximate to fitness suggests functional value to the observed variation (Cole, 1954; Gadgil & Bossert, 1970). The human life cycle is characterized by a great deal of variation from one population to another. For example, populations have recorded total fertility rates (TFRs) (the number of offspring born to a woman who survives the entirety of her reproductive span) ranging from under two in the low fertility countries of contemporary Europe and Japan to more than 14 for historical agrarian populations (Kohler, Billari, & Ortega, 2006; Livi-Bacci, 1997; Wood, 1994). Similarly, life expectancy at birth ranges from little over 20 years to an excess of 80 years (Livi-Bacci, 1997; Keyfitz & Flieger, 1990). Even in hunter–gatherer populations, there

is considerable variation in both life expectancy at birth and total fertility (Early & Peters, 1990; Gurven & Kaplan, 2007; Hill & Hurtado, 1996; Howell, 1979). This variation is presumably due to the varying historical, social, and technological circumstances of different populations.

In this article, I attempt to answer the question of how such variation in the basic inputs to fitness affects the force of selection on the human life cycle. For example, does selection push in a fundamentally different direction in a population with an expectation of life at birth of 40 and a TFR of 10 compared with a population with a life expectancy of 70 and a TFR of 2? Any attempt to make general statements about the functional value of human behavior clearly depends on the answer to this question. This question is important also because of the existence of major demographic transitions in human history (Caldwell, 1982; Coale & Atkins, 1986; Notestein, 1953). Does the force of selection on the human life cycle fundamentally change after demographic transitions?

There is controversy in human behavioral biology regarding the relevance of either contemporary phenotypic variation or selective pressures for understanding how selection has shaped human behavior (Perusse, 1993;

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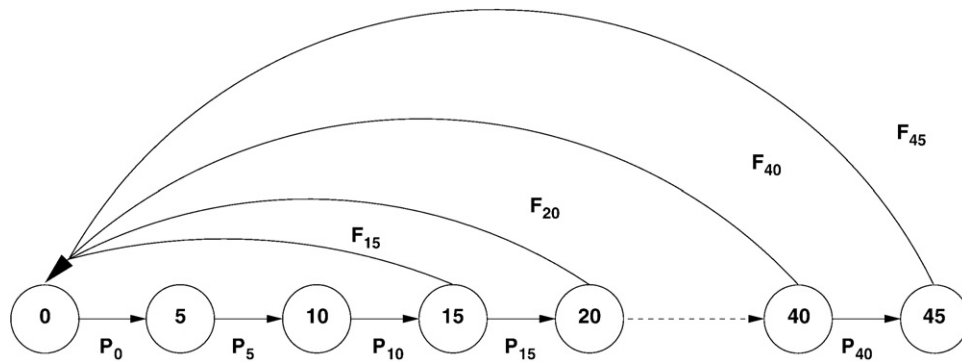


Fig. 1. Life cycle graph for an age-structured human population. Circles represent nonoverlapping 5-year age classes and arrows connecting them the survival probabilities, P_i , between stages. Loops back to the first class represent age-specific fertilities, F_i .

Tooby & Cosmides, 1990; Vining, 1986). However, two arguments suggest that understanding how selection changes with contemporary phenotypic variation is important. First, there is considerable variation in the demography of small-scale, face-to-face societies like those that characterized the entirety of human existence throughout the Pleistocene (Gurven & Kaplan, 2007). Second, the type of long-term evolutionary stasis argued for by Tooby and Cosmides (1990), for example, is itself dependent upon stabilizing selection. Thus, to understand evolutionary constancy and universality, we still need measures of selection on the life cycle.

The human life cycle has a number of peculiarities that make it challenging to understand how it has been and continues to be shaped by selection. First, the human life cycle is highly structured. In a demographic sense, structure means that mortality and fertility rates vary with age and do so in predictable ways across ensembles of people. This structure suggests that models that treat human populations as homogeneous—as does the Pearl–Verhulst logistic model, for example—will fail to capture the essential features of selection on human life histories. Fig. 1 depicts a schematic representation of the human life cycle divided into nonoverlapping 5-year age classes. This figure is known as the life cycle graph and provides a great deal of important information for understanding the dynamics of a structured life cycle (Caswell, 2001).

Second, as with many other vertebrates, generations overlap. In a natural fertility population, it is quite possible that a woman’s grandchildren can be older than her youngest children. Cant and Johnstone (2008) have argued that human generations are more separated than expected compared with other primates. Demographically, this does not matter, as the pioneering work of Thompson (1931) showed. The key point is that generations are not completely discrete. Although this observation seems, at first glance, obvious and perhaps trivial, its implications for the types of models used to represent the human life cycle are profound. In particular, because generations overlap, the timing of reproduction can matter critically for fitness, particularly if the population is not assumed a priori to be demographically stationary. In

many evolutionary studies of human behavior, the fitness measure adopted is lifetime reproductive success (Chagnon, 1988; Turke & Betzig, 1985; Volland, 1990). However, this measure only applies to discrete generation populations that are demographically stationary. In the more general case of nonstationarity and where generations overlap, the proper measure of fitness is the rate of increase given by the Euler–Lotka equation (Charlesworth, 1994), written here for a population with discrete nonoverlapping age classes,¹

$$1 = \sum_{i=\alpha}^{\beta} \lambda^{-i} \left(\prod_{j=1}^{i-1} P_j \right) F_i. \tag{1}$$

Here, α is the age at first reproduction (AFR), β is the age at last reproduction, the P_j is the age-specific interval survival probability, and F_i is the fertility rate at i . Eq. (1) has a unique real root. Fitness is given by this root, the multiplicative intrinsic rate of increase λ , which is defined implicitly by Eq. (1). The intrinsic rate of increase r is the natural logarithm of λ , divided by the length of the projection interval (which is also the width of the age classes).

The human life cycle is characterized by an extremely late AFR. Given the potential importance of timing of reproduction for fitness, this observation is puzzling. The traditional explanation for this late AFR focuses on the development of competitive competence in populations that remain predictably in the vicinity of their carrying capacity (MacArthur & Wilson, 1967; Pianka, 1970). Although this explanation certainly remains a viable contending hypothesis (Kaplan &

¹ I have written the Euler–Lotka equation in a manner consistent with the matrix model I use in subsequent sections. The form typically seen in textbooks is written in continuous time as $1 = \int_{\alpha}^{\beta} e^{-rx} l(x) m(x) dx$, where α is age at first reproduction, β is age at last reproduction, r is the rate of increase, x is age, $l(x)$ is the probability of surviving to exact age x , and $m(x)$ is the fertility rate (in daughters) of women age x . Note that $l_i = \prod_{j=1}^{i-1} P_j$. That is the cumulative probability of survival is the product of all previous interval probabilities.

Robson, 2002), other hypotheses may better explain this phenomenon. In addition to the late AFR, human reproductive patterns are characterized by a marked degree of iteroparity. That is, reproduction is spread out over a long reproductive span, lasting 25 years or more. Not only are the bouts of reproduction themselves drawn out over a parent’s lifetime, but so is parental investment. Successful human reproduction, leading to the recruitment of offspring into the breeding pool, requires extensive obligate parental investment (Hill, Kaplan, Lancaster, & Hurtado, 2000; Lee & Kramer, 2002; Robinson, Lee, & Kramer, 2008). A notable feature of patterns of reproductive investment is that humans frequently invest heavily in two or more offspring simultaneously. A weaned child remains almost completely dependent upon his or her mother for years after weaning, whereas the weaned chimpanzee is economically independent of its mother (Goodall, 1986; Hill et al., 2000).

The facts that the human life history is so strongly structured and that it is characterized by overlapping generations have implications for the type of models that need to be used to understand how selection acts to yield a late-breeding highly iteroparous organism with high obligate parental investment in multiple simultaneously dependent offspring.

For this analysis, we would ideally like a probability sample of the human demographic experience. However, such a sample from the diversity of human demographic experience is essentially impossible. The great majority of human history has been characterized by life in small face-to-face societies, subject to natural fertility and relatively high mortality (particularly among infants and juveniles). Nonetheless, the great majority of reliable data on age-specific demographic schedules comes from contracepting nation-states that use vital event registration. The solution to this problem that I propose is to construct a space that defines the limits of human demographic variation and to explore the shape of selection around the limits of this space. This strategy of identifying points that define specific regions of a parameter space has been extremely effective in understanding past population dynamics from molecular phylogenies (Nee, Holmes, Rambaut, & Harvey, 1995, 1996). Thus, the first goal of this article is to define the demographic space that the human life cycle occupies. This is the launching point for more extensive analyses.

2. Human demographic space

Livi-Bacci (1997) suggests that the diversity of human demographic schedules can usefully be reduced to a low-dimensional space defined by two summary measures: (1) life expectancy at birth e_0 and (2) TFR, the sum of a woman’s age-specific fertility rates across her reproductive lifespan.

I exploit the regularity of human mortality schedules to plot fitness isoclines for combinations of e_0 and TFR. The

definition of generation time, T , the time it takes a population growing at instantaneous rate r to increase by a factor of R_0 , is

$$R_0 = e^{rT}. \tag{2}$$

R_0 is the net reproduction ratio, the sum across the life cycle of the product of age-specific survival (P_i) and fertility (F_i),

$$R_0 = \sum_i \left(\prod_{j=1}^{i-1} P_j \right) F_i. \tag{3}$$

Here, I have written the probability of survival to age i as the product of all preceding age-specific survival probabilities (P_j) starting from the first age class. Livi-Bacci notes (1) that T is remarkably constant across human populations, and (2) R_0 can be well approximated by the product of TFR and the fraction of women who survive to reproductive age, $S(\alpha)$. In Fig. 2, I plot the fitness isoclines using relationships between e_0 and $S(\alpha)$ from the Coale–Demeny West model life table family, assuming $T=30$. That is, for a given model table (which is indexed by e_0), I look up the corresponding value of $S(\alpha)$, which I then use in the calculation.

Fig. 2 can be divided into four quadrants defined by high vs. low e_0 and high vs. low TFR. I require that the populations must have a deterministic rate of annual increase $r>0$. Any population with an expected growth rate less than zero is destined to extinction, so analyzing its asymptotic behavior is not rewarding. Although no population can sustain 2% annual growth indefinitely either, stochastic variation in vital rates typically reduces the long run rate of

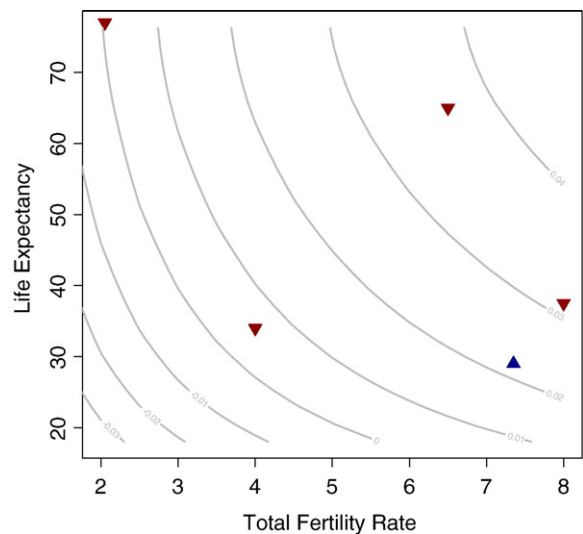


Fig. 2. Fitness isoclines for combinations of life expectancy at birth (e_0) and TFR. The four populations, indicated by triangles, mark the four quadrants of the space (conditional on $r>0$) corresponding to high vs. low e_0 and TFR. Included in the plot is Taiwan from 1906 (upward-pointing triangle), which Hamilton used in his classic (1966) perturbation analysis. See text for a discussion of the growth rates.

Table 1
Summary statistics and data sources for the four human populations analyzed

Population	Year	e_0	TFR	r (%)	Citation
USA	2002	77	2.05	0.8	Martin et al. (2005), Wilmoth (2007)
Venezuela	1965	65	6.5	3.8	Keyfitz & Flieger (1990)
Ache	Forest period	37.5	8.0	2.7	Hill & Hurtado (1996)
!Kung	Pre-1974	34	4.0	0.7	Howell (1979)

increase well below the growth rate of the average life history (Tuljapurkar, 1990).

Superimposed on the r isoclines are points for four populations that fall into each of these quadrants: !Kung (Howell, 1979), Ache (Hill & Hurtado, 1996), Venezuela in 1965 (Keyfitz & Flieger, 1990), and the contemporary United States (Martin et al., 2005; Wilmoth, 2007).² The first two populations are characterized by low life expectancy, whereas the !Kung has a low TFR and the Ache has a high TFR. The latter two populations have high life expectancy with Venezuela having high TFR and the United States having low TFR. Summary statistics of the four populations are provided in Table 1.

I will use these four populations to mark corners in Livi-Bacci's human growth space. How does selection act upon populations in different quadrants in the space? As will become clear in later sections, the specific demographic details of the population matter remarkably little for understanding the overall patterns of selection on the human life history. In a subsequent section, I will simulate life histories using model schedules of age-specific vital events to explore more fully human demographic space and understand the way the force of selection on the human life cycle is shaped by demographic inputs.

3. Fitness sensitivities: measuring the force of selection

Increasing the probability of survival at any age before the age of last reproduction while holding everything else constant will increase fitness. Similarly, increasing fertility at any age, in the absence of trade-offs, will increase fitness. However, not all changes to age-specific schedules of survival and fertility will have equivalent impacts on fitness. The rate at which fitness changes with a small change to the demographic schedules that define fitness via Eq. (1) is a measure of the force of selection on the human life cycle. In this section, I ask the following questions: How does the force of selection change across the human

life cycle? How does variation in demographic schedules affect the force of selection?

Caswell (2001) has demonstrated the power of matrix approaches to studying structured populations, and I will use a number of his tools in subsequent sections. The second edition of his book (Caswell, 2001) provides a comprehensive overview of matrix methods for demography, evolutionary and otherwise. For all the analysis in this and subsequent sections, I use the demogR library for the R statistical programming language (R Development Core Team, 2008), which was designed specifically for the construction and analysis of age-structured population models (Jones, 2007).

Here I use age-structured demographic projection matrices to examine how we expect natural selection to act upon the human life history. Projection matrices carry with them a host of powerful tools for mathematical analysis. They also have the attractive feature of mapping directly onto the life cycle graph presented in Fig. 1. A Leslie matrix \mathbf{A} is a sparse square $k \times k$ matrix, where k represents the number of age classes. The subdiagonal of the matrix contains the probabilities of survival from one age class to the next (P_i), whereas the first row contains age-specific fertility rates (F_i). All other matrix entries are zero. There are two other important features of projection matrices for age-structured human populations. First, the human life cycle is structured by age (and not by developmental stage, size, etc.). This insures that all paths through the life cycle must pass through the first age class (i.e., infancy). Although this may seem obvious, it is not always the case in organisms with complex life cycles (e.g., those with both sexual and asexual reproduction). Two properties insure that the population will converge to a stable age distribution: irreducibility (or connectedness) and primitivity. If we consider only those age classes before the termination of reproduction ($i < 50$, say), this insures that the life cycle graph is strongly connected (all nodes can reach each other in the life cycle graph) and the resulting projection matrix is said to be irreducible. Second, as noted in the Introduction, the human life cycle is characterized by a strong degree of iteroparity. As long as the age classes are not too wide, the age-structured life cycle graph is bound to have two consecutive age classes with nonzero fertility. This condition is sufficient to insure that the resulting matrix is primitive and will converge to a stable age distribution. For a more complete (and technical) discussion of these issues, see Keyfitz (1977) or Caswell (2001).

For the age-structured population modeled as a Leslie matrix, fitness is given by the dominant eigenvalue λ of the matrix \mathbf{A} . A matrix with k rows and columns will have k eigenvalues (indexed in decreasing absolute value from 1 to k), and if the conditions of irreducibility and primitivity are met, each of these k will be distinct. The Perron–Frobenius theorem insures that one of these k eigenvalues will be positive, real, and strictly greater than all other eigenvalues. Whenever the meaning is unambiguous, I drop the subscript

² Note that the growth rates calculated in Fig. 2 differ slightly from both the observed growth rates and those calculated later in the paper. This difference arises because of the simplifying assumptions of Eq. (2), where we use the approximation $R_0 \approx S(\alpha) \cdot TFR$.

on the dominant eigenvalue λ_1 , denoting it simply as λ . This eigenvalue λ is the same as the real solution to Eq. (1). The dominant eigenvalue λ has associated right and left eigenvectors \mathbf{u} and \mathbf{v} , respectively (again I drop the subscript for notational simplicity). The $k \times 1$ right eigenvector of a Leslie matrix corresponds to the stable age distribution of the population, whereas the $k \times 1$ left eigenvector corresponds to the age-specific reproductive values. Using these quantities, Caswell (1978) derived a method to calculate the partial derivative of fitness with respect to a small perturbation in some life cycle transition. The sensitivity of λ to a perturbation in the ij th element of \mathbf{A} is given by:

$$s_{ij} = \frac{\partial \lambda}{\partial a_{ij}} = v_i u_j, \tag{4}$$

where v_i is the i th element of \mathbf{v} and u_j is the j th element of \mathbf{u} , and it is assumed that the vectors have been scaled so that $\langle \mathbf{v}, \mathbf{u} \rangle = 1$. The sensitivity of fitness is thus the product of the reproductive value of the receiving stage and the stable proportion of the donor stage.

Fitness sensitivities tell us how small changes in age-specific survival and fertility will be translated into fitness. Improvements to life cycle transitions with high sensitivities

will change fitness more than improvements to less sensitive transitions.

Fig. 3C, D clearly reveals that the life cycle transitions to which fitness is most sensitive are those of early survival and the survival of 0- to 4-year olds in particular. This, of course, is part of the classic result of Hamilton (1966). However, although Hamilton’s analysis is frequently evoked to explain senescence, the fact that the force of selection is expected to be highest on infant survival (not that of higher reproductive value young adults) is rarely mentioned.

3.1. Fitness elasticities

A useful extension of the fitness sensitivity is the fitness elasticity. Elasticities are proportional sensitivities, measuring the corresponding increase in λ of a given increase in a_{ij} . For example, an elasticity measures by what percentage λ will change given a 1% change in the survival of 0- to 5-year olds. The elasticity of the ij th matrix element is:

$$e_{ij} = \frac{\partial \log \lambda}{\partial \log a_{ij}} = \frac{a_{ij}}{\lambda} \frac{\partial \lambda}{\partial a_{ij}}. \tag{5}$$

Elasticities sum to unity and can be conceived, in a limited way, as representing the fraction of total selection

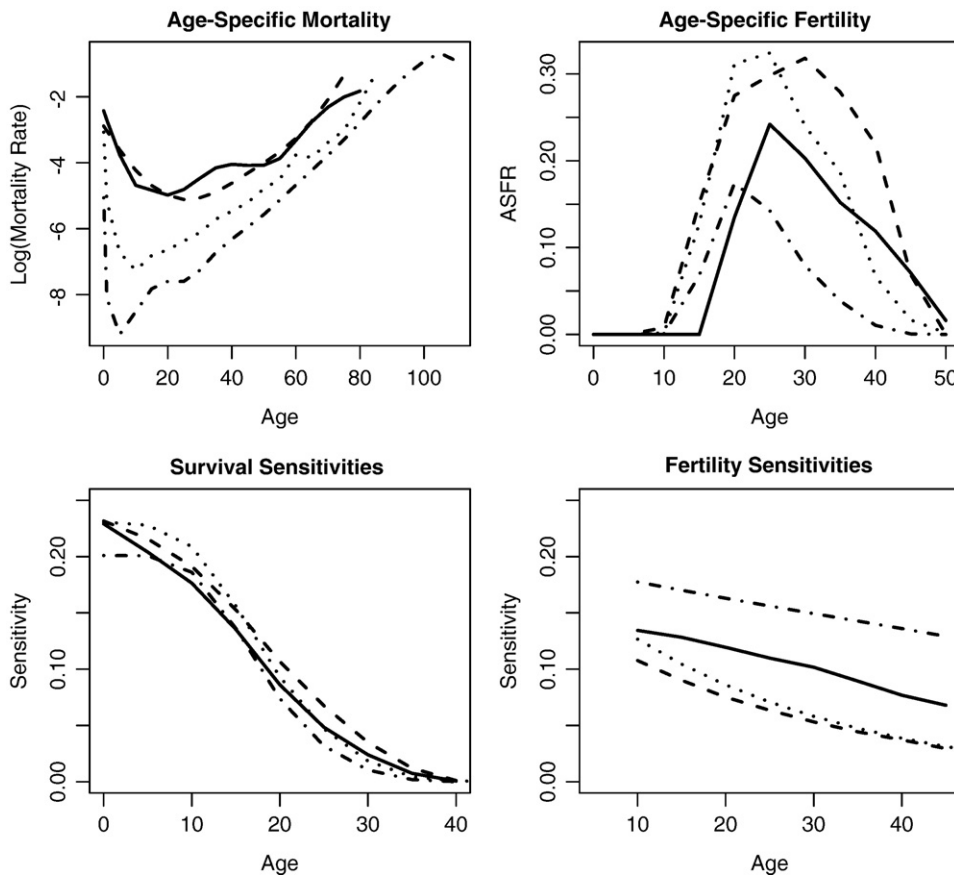


Fig. 3. Demographic rates and fitness sensitivities of the four populations: (A) natural logarithms of the central death rates (${}_tM_x$), (B) age-specific fertility rates, (C) sensitivities of λ to perturbations in age-specific survival, (D) sensitivities of λ to perturbations in age-specific fertility. In all plots, !Kung=solid line, Ache=long dashed line, USA=alternating long–short dashed line, Venezuela=short dashed line.

accounted for by individual transitions. The degree to which elasticities measure the apportionment of selection to the elements of the life cycle is limited, resulting from the fact that elasticities themselves are contingent. In particular, they measure the proportional change in fitness given a small change in a vital rate while holding everything else constant. First, everything is very rarely held constant. Second, because selection acts on a life history, the vital rates, by definition, will change. Because the vital rates change, so too will the elasticities. Nonetheless, elasticities provide a useful snapshot of the current force of selection on the life cycle conceived statically.

The fitness elasticities of the four populations show a similar pattern to the sensitivities (Fig. 4). The highest elasticities are those of prereproductive survival. The elasticities of fertility—particularly early and late in the reproductive period when the absolute fertility is low—are relatively smaller than the corresponding fitness sensitivities.

A second structural property of fitness elasticities is that the sum of the elasticities of all outgoing transitions in a life cycle must equal the sum of the elasticities of all incoming transitions (van Groenendael, De Kroon, Kalisz, & Tuljapurkar, 1994). In terms of the matrix model, this means that the sum of elasticities for column i will be equal to the sum of elasticities for row i . In a life cycle that is structured by age, there will only be two nonzero entries per column. In contrast, there will be multiple nonzero entries along the first row of the Leslie matrix as long as the organism is iteroparous. This observation leads to the following conjecture: age-structured life cycles characterized by strong iteroparity will experience the strongest selection on survival in the first age class.

Another way of approaching this observation is to note that the human life cycle—like the life cycle of any age-structured population—can be conceptually simplified to those events that transpire before the onset of reproduction and those that happen after its onset. The theorem of van Groenendael et al. (1994) then indicates that half of the total selection on the human life cycle falls before AFR. The summed transitions leading to AFR can be called “recruit-

ment.” Similarly, all life cycle transitions occurring after AFR can be called “reproduction.” Note that this includes the survival of reproductive age adults. This is similar to Charnov’s decomposition of the mammalian life cycle into recruitment and adult reproductive value (Charnov, 1997). There are typically fewer transitions before AFR than there are after it in the human life cycle. Consequently, the total recruitment elasticity is more concentrated in those transitions. Furthermore, the reproduction elasticity is split between both fertility and adult survival. Survival to breeding age and subsequent recruitment into the reproductive population therefore represents the largest single contribution to human fitness. This, coupled with the extremely late AFR characteristic of the human life cycle, creates a major fitness bottleneck in juvenile recruitment.

This observation focuses attention on what selective regime favored such highly iteroparous reproduction in the human life cycle. Iteroparity is traditionally seen as a risk-spreading adaptation in variable environments (Murphy, 1968; Seger & Brockerman, 1987; Schaffer, 1974). However, the impact of environmental variability on the optimal life history in age-structured populations is more complex (Jones, 2005; Tuljapurkar, 1990). Tuljapurkar (1990) has shown that iteroparity is still the most likely outcome of selection in a variable environment for an age-structured population if the magnitude of variability is sufficiently high. At low and intermediate levels of environmental variability, more semelparous life histories can be favored.

Because a strong degree of iteroparity is clearly fundamental to the human life cycle, this suggests that the selective regime that shaped the human life history was characterized by high levels of variability. This observation suggests that renewed effort should be placed into measuring the impact of environmental variability (both past and present) on human life history decisions and outcomes.

3.2. Life cycle contributions to infant survival sensitivities

The fitness sensitivities of early survivorship are always the highest for the age-structured human life cycle. There is,

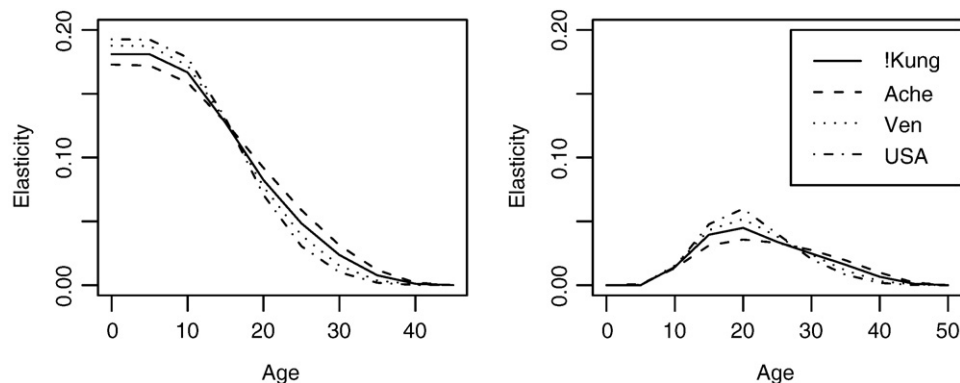


Fig. 4. Elasticities of fitness (λ) to perturbations in (A) age-specific survival and (B) age-specific fertility.

Table 2
Summary of the regression model of s_{21} against the logarithms of life expectancy and TFRs and their interaction

	Estimate	SE	t value	$p(> t)$
(Intercept)	0.2216	0.0056	39.56	<.0001
Log(TFR)	0.0518	0.0035	14.70	<.0001
Log(e_0)	-0.0094	0.0014	-6.65	<.0001
Log(TFR):log(e_0)	0.0033	0.0009	3.56	.0004

nonetheless, variation in the value of these early fitness sensitivities across different demographic schedules. To better understand this variation, I simulated a wide range of life histories using model schedules of age-specific mortality and fertility, calculated sensitivities, and evaluated the contribution of differences in indices of survival and fertility on the value of s_{21} , the sensitivity of survival from age 0 to 5 years. I used the four regional families of Coale–Demeny model life tables (Coale, Demeny, & Vaughn, 1983) combined with a wide range of Coale–Trussell model fertility schedules (Coale & Trussell, 1974, 1978) to generate simulated life histories. Coale–Demeny regional model life tables come in 25 levels of overall mortality, indexed by life

expectancy at the age of 10 years, in four distinct patterns—or shapes—of age-specific mortality. The Coale–Trussell model fertility schedules allow both the level and shape of age-specific fertility to be varied systematically. I simulated a total of 25,000 life histories and discarded those that yielded an annual rate of increase less than zero and greater than 5%, yielding a total of 12,198 simulated life histories.

I regressed the value of s_{21} on the logarithm of TFR and e_0 (and their interaction). I log transformed both TFR and e_0 because of their skewed distributions. Table 2 summarizes the model. Although s_{21} always has the greatest fitness sensitivity in the life cycle, its value varied from 0.173 to 0.363. Both TFR and e_0 have modest but highly significant effects on the value of s_{21} , with TFR increasing and e_0 decreasing the value of s_{21} . In addition, there was a strong interaction between e_0 and TFR. This interaction can be seen in a plot of the s_{21} against both e_0 and TFR (Fig. 5). Particular combinations of life expectancy and TFR have very similar sensitivity values. Note that in these simulations, e_0 varied in 2.5-year increments (Coale et al., 1983), whereas TFR effectively varied continuously. This is why the scatterplots exhibit the banding particularly visible in the s_{21}/e_0 subplots.

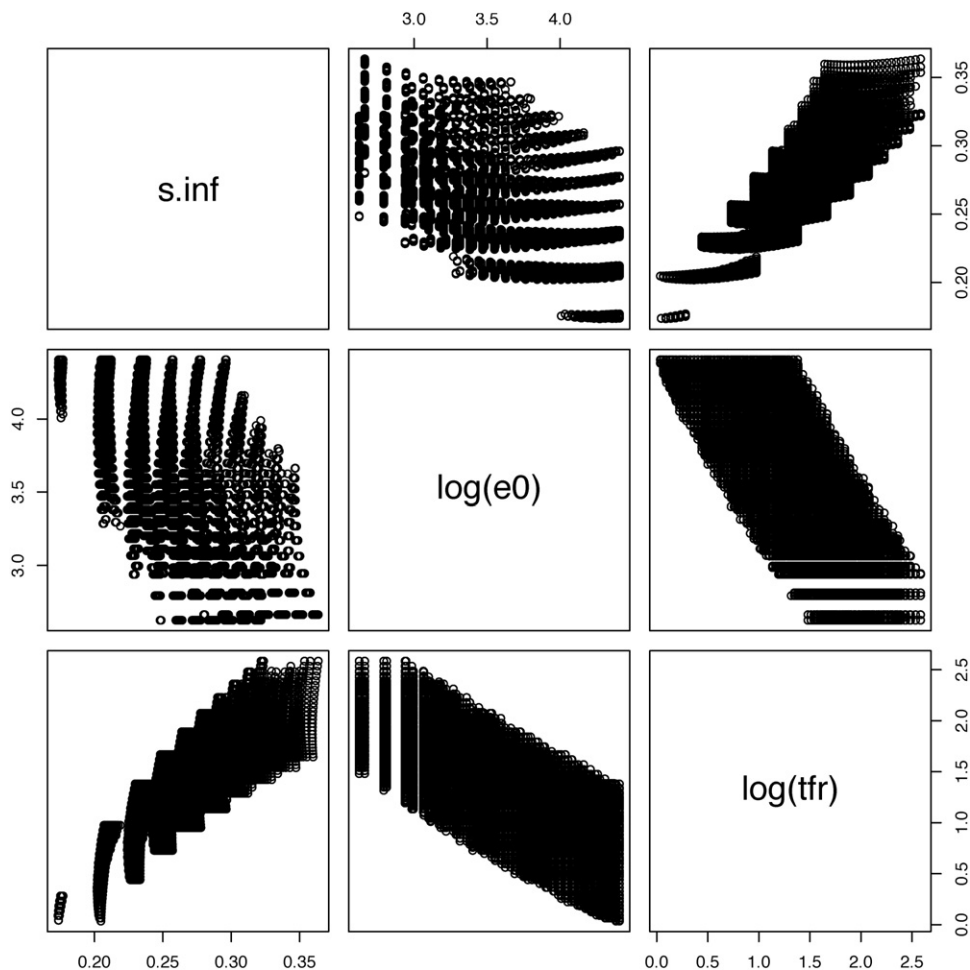


Fig. 5. Scatterplot matrix showing the correlations between s_{21} , TFR, and e_0 .

4. Discussion

Human populations are characterized by a tremendous degree of economic, social, and demographic diversity. Nonetheless, the results I have presented here indicate that the force of selection on the human life cycle is robust and remarkably consistent across the possible range of demographic variation. These observations make it clear that (1) the observed demographic diversity in humans is not itself likely to result from natural selection, and (2) that the force of selection points in largely the same direction on the adaptive landscape for all human populations.

In his classic article, Hamilton (1966) first performed a perturbation analysis of the characteristic equation (Eq. [1]), showing that the force of selection on age-specific survival declines monotonically with age. Although his analysis was very similar to mine, there are two ways in which the present analysis extends Hamilton's work. First, Hamilton (1966) used a single population to illustrate his calculations: Taiwan during the Japanese colonial period, a population characterized by both quite high fertility and mortality. I have expanded his analysis to consider a much broader range of the human demographic space (Fig. 2). Second, although Hamilton's survival perturbation was essentially an elasticity, his fertility perturbation was $\partial r/\partial F_i$, where $r = \log(\lambda)$. This is neither a sensitivity nor an elasticity according to the definitions I use, which follow (Caswell, 2001). Furthermore, it means that the fitness sensitivities with respect to survival and fertility have subtly different interpretations (Baudisch, 2005). Subsequent work in demography and life history theory uses either sensitivities or elasticities sensu Caswell (Benton & Grant, 1996; Lande, 1982; Tuljapurkar, 1990). I use sensitivity and elasticity measures that are consistent with the demographic developments subsequent to Hamilton's work, making them more comparable with common use of perturbation analysis in evolutionary biology.

In this analysis, the preponderance of selection, as measured by fitness sensitivities and elasticities, falls upon the transitions leading to recruitment. A structural feature of elasticities is instructive in this respect. The summed elasticities of incoming and outgoing transitions in a life cycle stage must be equal (van Groenendaal et al., 1994). In an age-structured population, the more iteroparous the life cycle is, the more transitions will lead to the first life cycle stage. The implication of this is that the force of selection will increase on survival from this stage. Humans are highly iteroparous, and consequently, a large fraction of the total fitness elasticity is accounted for by survival out of the first age class. It is interesting to note that as fertility becomes more concentrated with the aid of modern contraception, the relative elasticity of early survival is reduced. This can be seen in the differences between elasticities of the !Kung and Ache on the one hand and the USA on the other.

Although the sensitivity of λ to first period survival (i.e., s_{21}) is always the greatest in the age-structured human life cycle, its value varies considerably for different combina-

tions of age-specific schedules of survivorship and fertility. I found that the value of s_{21} increases as TFR increases and declines as e_0 increases (Fig. 5). Somewhat paradoxically, having more babies places a premium on their survival. The intuition behind this is that in high-fertility populations, the age structure is more heavily weighted toward the youngest age classes. Since fitness sensitivities are the product of the receiving stage's reproductive value and the sending stage's stable age contribution, populations with higher fractions of 0- to 4-year olds will have greater values of s_{21} , ceteris paribus. Understanding the dynamic interplay between the force of selection on early childhood survival, which increases with TFR, and fertility, which presumably trades off against childhood survival, remains a major challenge in the formulation of an adequate theory of human life history evolution.

At the most general level, life history theory has as its constituent elements the traits that define the life cycle (e.g., age-specific schedules of reproductive investments) and the trade-offs that bind them together (e.g., reproductive effort vs. future or current survival, number vs. quality of offspring). These trade-offs are driven by both the quantitative genetics underlying the traits and the epigenetic processes that give rise to them. In this analysis, I have only analyzed the force of selection on demographic schedules and have not examined trade-offs explicitly. The results of this analysis are nonetheless informative regarding these trade-offs. In particular, we should expect any trait that is negatively correlated with juvenile recruitment to be opposed by selection.

The logic of this statement is made particularly lucid by considering Lande's equation (Lande, 1982) for the evolution of a quantitative multivariate trait z :

$$\lambda \Delta \bar{z} = G \nabla \lambda, \quad (6)$$

where $\Delta \bar{z}$ is a vector describing the change in the mean of the multivariate trait z —here taken to be the collection of life cycle transitions, G is the additive genetic covariance matrix, and $\nabla \lambda$ is the fitness gradient—a column vector of all the projection matrix sensitivities.

For concreteness sake, consider a hypothetical trade-off between some adult fertility rate and infant survival. A negative covariance between these traits (i.e., a trade-off) will be multiplied by the largest sensitivity in the life cycle. Given the magnitude of the differences in sensitivities of early survival to the sensitivities of all the fertility rates, the absolute value of the sum of these scaled covariances is likely to be greater than the scaled additive genetic variance in fertility. Thus, even if selection favored increased fertility in the absence of trade-offs (as, of course, it does), fertility is expected to decrease. This whole discussion is predicated on the existence of additive genetic variation driving change in the trait but illustrates the fundamental importance of such high fitness sensitivities for juvenile recruitment.

Elasticities are proportional sensitivities: For a percentage change in a vital rate, by what percentage will fitness change? Depending on the values of the vital rates, a percentage change may be easier for some transitions than for others. For example, survival is a probability. An increase of 1% in the 0→5 survival for the USA in 2002 would be impossible because it would mean that more individuals would enter the 5-year-old age class than leave the 0-year-old one! In contrast, among the Ache, the same proportional increase to the same transition probability would make infant survival go from 0.751 to 0.759—not an unimaginable increase. Similarly, increasing contemporary American fertility by 1% certainly seems within the realm of biological possibility, whereas increasing the fertility of 35-year-old Ache women (with their TFRs of 8) seems less biologically plausible.

I have not dealt explicitly with the problem of postreproductive survival. However, the analysis is nonetheless potentially instructive about its evolution. It is not difficult to imagine that postreproductive survival could be favored if it increased the survivorship of juveniles. Given the high fitness elasticities of juvenile survivorship (together with the seeming feasibility of making improvements under hunter-gatherer demographic schedules) and the relatively low elasticities of fertility—even at the peak of a woman's reproductive career—it seems more plausible that investments in postreproductive lifespan would have been favored more for its value in increasing grandchildren's survival rather than daughters' fertility, a point supported empirically by Sear and Mace (2008).

The model for the evolution of human life histories that has probably received the most attention of late is due to Charnov (1991). The fact that the fitness elasticities for prereproductive survival are the highest in the human life cycle—and are likely to be the highest among any iteroparous age-structured life cycle—calls into question the one fundamental assumption of Charnov's life history model. Charnov (1991) presented a model for mammalian life history evolution in which the causal pathway flows from adult mortality rates to AFR, by way of a growth law. Size at maturity determines adult fertility. Perfect population stationarity is assumed, and the mechanism by which such homeostasis is achieved is through juvenile mortality. Under this formulation, juvenile mortality is not free to vary independently (in contrast to Charnov's earlier work: Charnov & Schaffer, 1973) but instead is adjusted to insure population stationarity. In other words, juvenile mortality takes on the role of bookkeeping in this influential model, which has been used extensively in evolutionary anthropology in recent years (Hill, 1993; Hawkes, O'Connell, Jones, Alvarez, & Charnov, 1998; Kaplan, Hill, Lancaster, & Hurtado, 2000). The results presented here suggest that juvenile mortality should be expected to exert a strong selective force in itself. It remains an open research question whether a model that can account for the joint effects of juvenile and adult mortality while retaining the appealing reproductive power allometry of Charnov's model can be formulated.

In this article, I have shown that there is a universal pattern to the way selection acts on the age-specific patterns of survival and fertility in the human life cycle. Such universality suggests we should look for specific species-typical adaptations to parenting and the promotion of child survival. The existence of extensive intergenerational transfers with net downward flow (Lee, 2003) is clearly one avenue for the promotion of child survival and recruitment. The results that I have presented in this article also suggest that behavioral plasticity, allowing parents to capitalize on favorable environmental circumstances for the promotion of child survival on the one hand and to buffer child survival against deleterious environmental circumstances on the other, should be a hallmark of human parental care behavior. A recent empirical example of this type of phenomenon comes from Marlowe's study of male parental investment among the Hadza of Tanzania. Marlowe (2003) shows that while women typically subsidize men's hunting through their more reliable foraging among the Hadza (Hawkes, O'Connell, & Jones, 2001), men increase their provisioning when their wives are energetically stressed because of the presence of young nurslings. Note that although the population models I have analyzed are one-sex models, the same logic applies to two-sex models, a topic I take up in a related article. The survival of men's offspring will have the highest impact on their fitness, with the provision that men's paternal uncertainty will discount this benefit somewhat.

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