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Fetal Programming: Adaptive Life-History Tactics or Making the Best of a Bad Start?

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ABSTRACT Fetal programming is an ontogenetic phenomenon of increasing interest to human biologists. Because the downstream consequences of fetal programming have clear impacts on specific life-history traits (e.g., age at first reproduction and the general age-pattern of reproductive investments), a number of authors have raised the question of the adaptive significance of fetal programming. In this paper, I review in some detail several classical models in life-history theory and discuss their relative merits and weaknesses for human biology. I suggest that an adequate model of human life-history evolution must account for the highly structured nature of the human life cycle, with its late age at first reproduction, large degree of iteroparity, highly overlapping generations, and extensive, post-weaning parental investment. I further suggest that an understanding of stochastic demography is essential for answering the question of the adaptive significance of fetal programming, and specifically the finding of low birth weight on smaller adult body size and earlier age at first reproduction. Using a stage-structured stochastic population model, I show that the downstream consequences of early deprivation may be "making the best of a bad start" rather than an adaptation per se. When a high-investment strategy entails survival costs, the alternate strategy of early reproduction with relatively low investment may have higher fitness than trying to play the high-investment strategy and failing. Am. J. Hum. Biol. 17:22–33, 2005.

Mounting evidence suggests that poor nutrition, and physiological insult more generally, in utero has downstream effects on later adult health. While the list of adult maladies that have been linked to early physiological insult is continually increasing, some particularly significant results include (1) increased risk for developing impaired glucose tolerance and reduced insulin sensitivity as adults (Phillips, 1998), (2) impaired immune function as adults (McDade et al., 2001), (3) increased coronary heart disease risk (Robinson and Barker, 2002), and (4) an overall increased rate of senescence (Sayer et al., 1998).

A number of authors have suggested that fetal programming might be adaptive (e.g., Bateson et al., 2004; Gluckman and Hanson, 2004). For example, Bateson et al. (2004: p 420) suggest "a functional and evolutionary approach derived from the rest of biology suggests that the pregnant woman in poor nutritional condition may unwittingly signal to her unborn baby that it is about to enter a harsh world. If so, this 'weather forecast' from the mother's body may result in her baby being born with characteristics, such as small body and a modified metabolism, that help it to cope with a shortage of food." The primary question motivating the present paper is whether a functional explanation of developmental programming, and its life-history sequellae, adds to our understanding of this ontogenetic phenomenon. Specifically, could the suite of developmental features observed in children who experience early adverse environments represent a facultative, strategic adaptation on the part of the developing child?

An excellent starting point for thinking about functional explanations for early life events is the work of Chisholm on mortality, attachment, and reproductive strategies in humans (Chisholm, 1993; Coall, and Chisholm 2003). Chisholm (1993) argues that the observation that girls who experience

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parental loss before menarche experience accelerated sexual maturation because the parental death provides evidence for a harsh environment. Employing bet-hedging theory (e.g., Seger and Brockerman, 1987), Chisholm suggests that such putative high-mortality environments favor life-history tactics characterized by increased reproductive effort—early age at first reproduction, reduced offspring investment, etc. Chisholm suggests that information on local mortality rates is communicated to children through the intermediary channel of attachment processes (Bowlby, 1969). High local mortality leads to parental stress and the subsequent insecure attachment of children. Insecure attachment leads to a developmental cascade resulting in a bio-behavioral phenotype adapted for "mating effort." In contrast, children growing up in low local mortality environments have less stressed parents, more secure attachment, and an adult phenotype adapted for "parenting effort." The logic linking mortality experience and reproductive tactics is derived from the classic theory of bet-hedging, which I will review in section 1.1.

Two key components of Chisholm's functional explanation for the effects of parental loss on children's life histories are:

1. Information from early environments must reliably communicate information regarding optimal life history tactics.
2. Life-history responses to early environments must yield higher fitness than alternative phenotypes.

For the first part of this paper, I will focus on point 2. Understanding the fitness consequences of alternate life histories requires a theoretical framework that allow fitnesses to be calculated. Because much of the intuition underlying adaptive explanations of alternate human life-history strategies derives from the classic bet-hedging model of Schaffer (1974), I will review Schaffer's model in some detail. Schaffer's model applies to "scalar" life histories—i.e., those lacking age- (or stage-) structure. As such, it turns out to be a special case, characterized by a set of restrictive assumptions, of a more general theory of stochastic demography for structured populations which developed in the decade following Schaffer's pioneering work. In section 1.2, I outline a basic framework for the stochastic demography of age-structured populations as developed by Tuljapurkar, Orzack, Lande, and others since 1980 (Tuljapurkar and Orzack, 1980; Tuljapurkar, 1982, 1990; Lande and Orzack, 1988).

A key aspect of my argument is that an adequate functional explanation for variation in human life histories should account for the salient features of the human life history: (1) a tremendous degree of age-structuring, (2) highly overlapping generations, (3) strong, obligate parental investment, (4) delayed age at first reproduction, and (5) a high degree of iteroparity. Another notable feature of the human life cycle that may or may not have relevance to this particular developmental phenomenon is (6) substantial post-reproductive survival. I will not address this point directly in this paper. Points 1–5 are central to the development of a life-history theory grounded in stochastic demography of structured populations.

Throughout the paper, I maintain a distinction between life-history strategies and life-history tactics. A "strategy" is generally construed to mean "a plan for successful action based on the rationality and interdependence of the moves of the opposing participants" (*Oxford English Dictionary*, 2nd edition, 1989). Tactics, in contrast, are the specific means employed to achieve the goal of a strategy. While the distinction in biology is somewhat artificial, I feel that the focus on tactics as the means through which a desired strategy is executed, conditional on physiological state, environmental exigences, etc., helps to focus the discussion on interactions between function and ontogeny fundamental to the problem of the fetal origins of adult disease.

In section 2, I present a simple stochastic demographic model for a structured population. In this model, survival trade-offs are experienced in a state-dependent manner. I show that the adult phenotype of the child who experienced physiological insult can be seen as "making the best of a bad start." While the fitness of this tactic is lower than the unperturbed ("standard") life history, it fares better than a tactic of attempting to achieve the standard life history and failing with high probability.

In the Discussion (section 3), I briefly take up the question of the reliability of early information for shaping life-history tactics, suggest some future avenues for research, and conclude with some generalizations about the evolution of human life histories that emerge from analysis of structured population models.

1.1. Life-history theory in variable environments: bet-hedging and beyond

Chisholm's functional model for the life history consequences of early life experiences is based on some classic work in life history theory showing that the optimal strategy for an organism with a scalar life history in a variable environment maximizes the geometric mean of fitness (Cohen, 1966; Murphy, 1968; Schaffer, 1974). The geometric mean of a random sample of n items from a random variable X is the nth root of the product of the x_i's. It is also equivalent to the antilog of the arithmetic mean of the logarithm of the sample from X. Linear analysis of the geometric mean shows that, for a given arithmetic mean, the geometric mean is negatively related to variance. In a variable environment—where zero fitness is possible—minimizing the variance in intertemporal fitness (i.e., between-generation in the discrete-generation framework) is one means of maximizing the geometric mean. Under variable environments, the geometric mean of fitness is the relevant measure for scalar life histories, as fitnesses are multiplicative across generations, and any generation with zero fitness ends the lineage. A particularly coherent treatment of this problem comes from the classic analysis of Schaffer (1974).

In Schaffer's seminal work on adaptation in variable environments, he writes "here I relax these assumptions [of constant vital rates] for the special case in which fecundity and mortality do not vary with age." (p 783) He goes on to note that his work suggests that "generalizations common in the literature—namely that fluctuating environments *always* select for small litters and increased longevity—are not deducible from theory." (p 783, emphasis in original).

Because the life history that Schaffer analyzes in scalar, that is, it has no age structure, the fitness measure is the annual contribution of individuals to the population. Let λ refer to darwinian fitness, B refer to annual production of daughters, and P be the annual post-breeding survival probability of adults. Note that B is in fact a composite parameter, reflecting both the mother's fertility and the survival of offspring to the census. It is clear that in this simple life history,

$$\lambda = B + P. \tag{1}$$

λ is the growth rate of the scalar population: $N(t+1) = \lambda N(t)$. Gadgil and Bossert (1970) suggest that both B and P are most reasonably seen as functions of reproductive effort, denoted E. Effective fecundity increases with effort, whereas post-breeding survival decreases with effort. For concave (down) $P(E)$ and $B(E)$ (i.e., for diminishing returns to effort), the optimal life history lies at some intermediate point $0 \leq E \leq 1$, and the optimal reproductive effort, and hence life history, satisfies the relation

$$\frac{dB}{dE} = -\frac{dP}{dE}. \tag{2}$$

In words, the optimal effort $\hat{E}$ occurs where the marginal fertility benefit of increased effort exactly balances the marginal survival cost of increased effort. It is for such concave life histories that iteroparity will be optimal. Where P and B are convex (i.e., concave up), the optimal life history is semelparous (i.e., characterized by a single, suicidal breeding event).

When the environment varies, the values of $B(E)$, $P(E)$, and consequently λ will vary from year to year. The long run growth of the population is given by

$$N(t) = \lambda(t-1)\lambda(t-2)\ldots\lambda(0)N(0).$$

The expected value of λ is defined as:

$$\bar{\lambda} = \mathrm{IE}\lambda(t) = \frac{1}{t}\lambda(t-1)\lambda(t-2)\ldots\lambda(0).$$

The population size at time t is then given by the following simplified relationship:

$$N(t) = \bar{\lambda}^t N(0).$$

In this case, fitness is maximized by maximizing $\bar{\lambda}$. Schaffer's analysis focused on a specific case of an environment with two randomly distributed states, "good" and "bad," with associated fitnesses λ_g and λ_b. Thus, $\bar{\lambda} = \sqrt{\lambda_g \lambda_b}$. In Schaffer's original paper, the fitness measure analyzed is, in fact, $\bar{\lambda}^2$, because it is easier to work with under the special assumptions of randomly (and equally) distributed environments.

The frequency of good versus bad years is defined by a parameter s, which describes the annual deviation from the constant arithmetic mean fitness.

Case 1: Variation affects effective fecundity. Assume first that environmental fluctuations only affect the annual production of daughters. This could come about through reduced fertility of mothers or through reduced survival of immatures. In this case, eq. (1) for the two states becomes

$$\lambda_g = B(1+s) + P, \tag{3}$$

$$\lambda_b = B(1-s) + P. \tag{4}$$

When it is assumed that both $B(E)$ and $P(E)$ are concave (down) functions (i.e., both effective fecundity and survival show diminishing returns to effort), then the optimal effort $\hat{E}$ satisfies the following relationship:

$$\left(1 - \frac{s^2 B}{B+P}\right)\frac{dB}{dE} = -\frac{dP}{dE}. \tag{5}$$

The first term on the left-hand side of this equation is less than one. Thus, relative to the result for a constant population (eq. (2)), variation in fertility induces the optimal $\hat{E}$ to be inversely proportional to s. *More variable environments favor reduced reproductive effort*.

Case 2: Variation affects adult survival. Now assume that environmental variability affects only post-breeding survival of adults, P.

$$\lambda_g = B + P(1+s), \tag{6}$$

$$\lambda_b = B + P(1-s). \tag{7}$$

The optimal reproductive effort thus satisfies the relation

$$\frac{dB}{dE} = -\left(1 - \frac{s^2 P}{B+P}\right)\frac{dP}{dE}. \tag{8}$$

Since the first term on the right-hand side of the equation is less than 1, the marginal fertility benefit to increased effort that balances the marginal survival cost is relatively lower. For variability affecting adult survival, *more variable environments favor greater reproductive effort*.

It is these two results which form the basis of "bet-hedging" theory, which itself lies at the heart of a number of models in evolutionary anthropology (e.g., Chisholm, 1993; Madsen et al., 1999). For example, Chisholm (1993) suggests that high-mortality environments favor increased mating effort at the expense of parenting effort. The theoretical justification for this prediction is founded in eq. (8) above.

Schaffer's paper, like that of Gadgil and Bossert (1970) shortly before it, was a watershed in life-history theory. The elegance of the analysis and simplicity of the results presented in this paper have made it an important source of ideas for the evolution of human life histories. It is testament to this elegance and simplicity that its ideas have permeated evolutionary anthropology to the extent they have (e.g., Chisholm, 1993; Madsen et al., 1999). Unfortunately, there are a number of complications inherent in the human life history that make Schaffer's particular analysis inapplicable.

The model Schaffer (1974) presents is founded upon four key assumptions:

1. Scalar life histories (i.e., no age structure)
2. Constant fertility and mortality
3. Discrete (i.e., non-overlapping) generations
4. Random—and uniform—environmental fluctuations (e.g., no temporal autocorrelation)

The first two of these points are related. Structured life histories are structured, in part, because the vital rates change systematically with age. Point 3 is key since it dramatically changes the manner in which selection operates on an organism's life history. The last point is important for understanding the response of populations to variable environments, and determines the form of the expected growth rate $\bar{\lambda}$.

As noted originally by Thompson (1931), a population that exists in an environment in which continuous breeding is possible (or that the interval between breeding and nonbreeding seasons is short) will be characterized by increasingly overlapping generation times. There are some important consequences of this fact. First, these is no natural point at which to enumerate an individual's offspring. Second, as the population continues to change throughout an individual's reproductive span, the relative contribution to the total population of any single offspring will change as time passes and the individual ages.

In a population with overlapping generations, a fraction of offspring will begin breeding before their parental generation has

ceased breeding. While the definition of "generation" varies, one natural way to think about a generation is the mean age of mothers in a stable equivalent population. For humans, this value varies remarkably little between 25 and 29 years (Livi-Bacci, 1997). In a natural fertility population, the variance around this mean can be quite high, and it is not particularly extraordinary for a woman in a natural fertility population to simultaneously be a grandmother and mother of a newborn.

The evolutionary implication of these observations is that timing of reproduction matters critically for natural selection. This is the basis for Fisher's (1958) concept of reproductive value. The contribution of a woman to the total population at some point in the future will vary according to her age and the rate of increase of the population. As a woman approaches her first reproduction, her expected contribution increases because there is less of a remaining interval in which she could die before reproducing. Similarly, as a woman ages, the population grows (or contracts), making her later offspring less (more) valuable for fitness.

Fisher (1958: p 31) maddeningly writes "...the preset value of the future offspring of persons aged [a] is easily seen [in the following relationship]":

$$v(a) = \frac{e^{ra}}{l(a)} \int_a^{\omega} e^{-rx} l(x) m(x) dx,$$

where r is the intrinsic rate of increase, $l(x)$ is the survivorship to age x, $m(x)$ is the age-specific fertility (in daughters), and ω is the age of reproductive cessation. In a growing population (i.e., $r > 0$), late-born offspring are heavily discounted, as e^{-rx} will approach zero as x increases. This results simply from late-born offspring representing a smaller fraction of the total in a growing population. In a high-mortality population, $v(a)$ rises steeply as the age at first birth approaches.

While this relationship may have only been *easily* seen by Fisher himself, it does fall naturally into standard stable population theory as first articulated, for example, by Lotka (1907). Reproductive value is simply a summing of the births to mothers of all ages starting at a in the stable population and then weighted by terms that correct for the fact that the mother has already survived to a and in which time the population has increased by a factor e^{ra}.

It is this discounting—both the exponential discounting induced by the fact that the population is growing and the discounting stemming from the fact that an individual may not survive to reproduce—that is essential for understanding the dynamics of a structured population in both a deterministic and stochastic framework. In particular, the way that selection works on a structured population in a stochastic environment depends critically on the age-specific force of selection on the mean population.

Force of selection on age-specific demographic traits. Hamilton (1966) first devised a method of calculating the force of selection on age-specific demographic traits for a population characterized by overlapping generations. Caswell (1978) translated Hamilton's perturbation analysis into matrix terms, greatly facilitating calculation of the force of selection on age-specific traits.

Consider a one-sex population[1] composed of k age classes. The dynamics of this population can be described by a system of linear equations that depict the population recurrence through survival from one age class to the next and from each reproductive age to the infant class. This system can be represented in compact form by a Leslie matrix, a square $k \times k$ matrix with the age-specific fertility rates along the first row and survival probabilities along the subdiagonal. Denote this matrix $\mathbf{A}$ and its ijth element as a_{ij}. The population described by $\mathbf{A}$ grows asymptotically at rate λ_1 and is characterized by a stable age-structure $\mathbf{w}$ and reproductive value vector $\mathbf{v}$ (reviewed in Caswell, 2000). Assume that $\mathbf{w}$ and $\mathbf{v}$ have been normalized so that $(\mathbf{w}, \mathbf{v}) = 1$. Caswell (1978) showed that, for a small perturbation in matrix element a_{ij}, the change in λ_1 is given by

$$\frac{\partial \lambda_1}{\partial a_{ij}} = v_i w_j. \tag{9}$$

The partial derivatives of fitness with respect to individual projection matrix

[1]Or a population characterized by female demographic dominance, a plausible situation in which female reproduction is limiting.

elements are generally known as "fitness sensitivities" (Caswell, 2000). In words, the sensitivity of λ_1 to a change in trait a_{ij} is simply the product of the reproductive value of stage i and the normalized fraction of the stable population in stage j. That is, the sensitivity is the expected contribution to future population size at stage i weighted by the fraction of the population in stage j. The role of reproductive value is straightforward. The weighting by age-structure accounts for the fact that a contribution from a stage that compromises a negligible fraction of the population—even if that stage has high reproductive value—will have a small impact on the growth of that population.

In Fig. 1, I plot demographic schedules of three small-scale human populations: the !Kung (Howell, 1979), the Ache (Hill and Hurtado, 1996), and the Yanomama (Early and Peters, 1990). The top panels plot the survivorship curves and age-specific fertility curves. The bottom panels plot the fitness sensitivities of eq. (9). The left-hand panel plots the sensitivities with respect to survival, while the right-hand panel plots the sensitivities with respect to fertility. The sensitivities for early (i.e., pre-reproductive) survival are highest—nearly twice as high as the highest fertility sensitivity. Improving early survival will have the greatest impact improving fitness. This is characteristic of the human life history. In general, higher mortality populations, regardless of where the mortality strikes, will have relatively higher early-survival sensitivities.

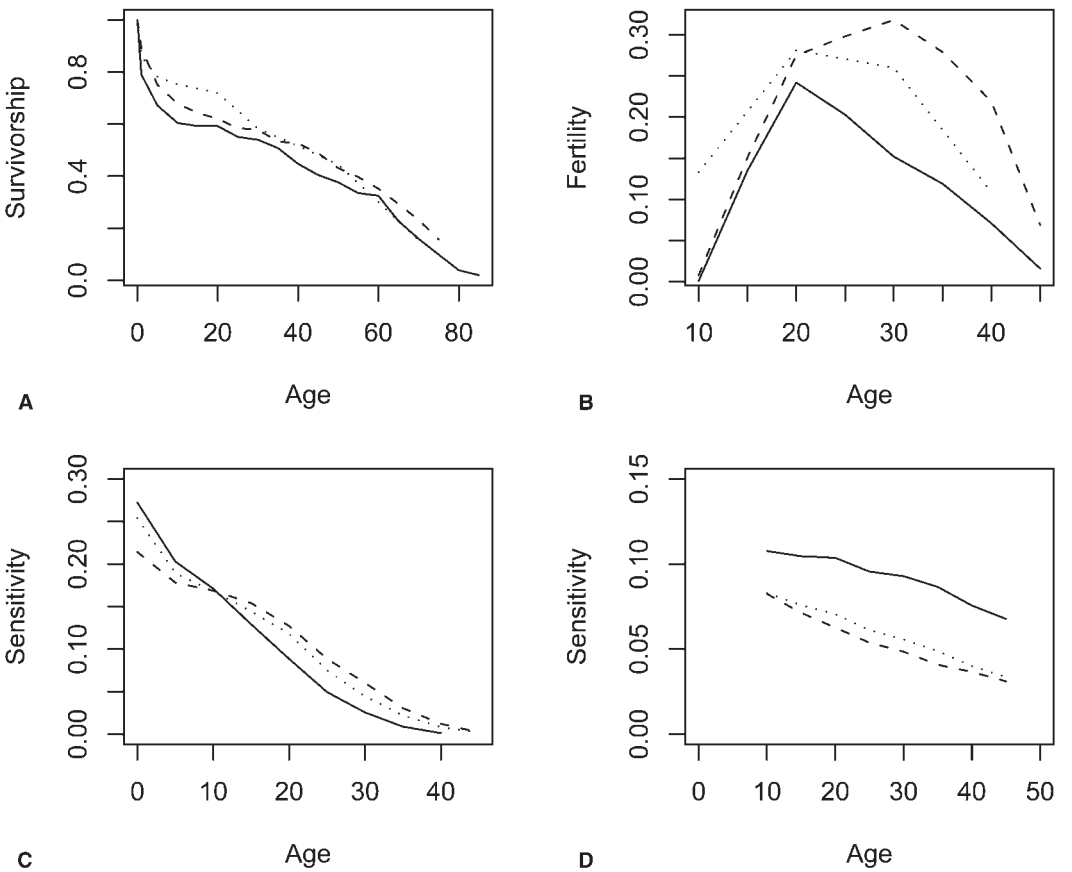

Fig. 1. Human life histories are structured. Clockwise from top right: **A**: survival curves for three populations; **B**: age-specific fertility curves; **C**: fitness sensitivities for age-specific survival; **D**: fitness sensitivities for age-specific fertility. Solid line, !Kung; dashed line, Ache; dotted line, Yanomama.

1.2. Dynamics of structured populations in variable environments: the devil is in the details

The theoretical results derived for scalar life histories do not readily generalize to structured populations. Schaffer's classic analysis of a scalar life history in a variable environment suggests that variability in effective fecundity should lead to decreased reproductive effort, whereas variability in adult survival should lead to increased reproductive effort. The results for structured life histories in variable environments are more ambiguous. For example, variability in effective fecundity can favor either increased or decreased reproductive effort (Tuljapurkar, 1990). The devil, it seems, is in the details in stochastic models for structured life histories.

Denote the logarithm of the stochastic growth rate as $\log \lambda_s$. Working with the logarithm leads to more compact and intuitively meaningful mathematical notation. Instead of a single projection matrix, we now consider an average of the matrices for the different environments $\bar{\mathbf{A}}$. Defining λ_1 as the rate of increase of this average projection matrix and $\mathbf{s}$ as a vector of sensitivities of the elements of the average projection matrix, Tuljapurkar's second-order approximation of the long-run stochastic growth rate is

$$\log \lambda_s \approx \log \lambda_1 - \frac{\tau^2}{2\lambda_1^2}, \tag{10}$$

where,

$$\tau^2 = \mathrm{s}^T\ \mathrm{C}\ \mathrm{s}, \tag{11}$$

and $\mathbf{C}$ is a matrix of covariances between the different elements of the life cycle. The term τ^2 represents the variance in λ_1.

Equations (10) and (11) make several points clear regarding the fitness of structured life histories in stochastic environments. First, greater variance in the growth rate (which, in turn, results from more variation in the elements that contribute to $\bar{\mathbf{A}}$) reduces stochastic fitness with respect to the fitness of the average life history. Variance will be increased in general by positive covariance in the matrix $\mathbf{C}$. Second, any trait that has a positive covariance with the high-sensitivity traits of early survival will reduce stochastic fitness by a large degree. Third, reductions of stochastic fitness due to high variance are attenuated if λ_1 is high enough.

All three of these points have clear applicability to understanding human life histories. First, the prolonged obligate parental investment characteristic of humans means that the human life cycle abounds with positive covariances: a bad year for adult survival will be a bad year for childhood survival and a bad year for fertility. While this may seem commonsense, it is not necessarily true for organisms with complex life cycles. These positive covariances will furthermore be amplified by the fact that the juvenile survivorship traits have the highest sensitivities of any life cycle elements. Finally, the value of λ_1 will be relatively low, even for the highest possible human survival and fertility schedules.

As noted above, the results for structured population models do not necessarily correspond to the expected results derived from the formalism of Schaffer (1974). Variability in effective fecundity can favor either increased or decreased reproductive effort, depending on the correlation structure of the vital rates across environments. Tuljapurkar (1990) nonetheless identifies three generalizations for structured models in which variability is experienced in effective fecundity/juvenile survivorship. Measuring environmental variability as the coefficient of variation c of effective fecundity, he identifies three dynamical regimes:

1. Low variability: A small degrees of variation in effective fecundity generally favors early age at first reproduction and high reproductive effort.
2. Intermediate variability: While a degree of iteroparity is favored, a wide variety of life histories characterized by more or less reproductive effort have similar fitness. In the intermediate region, a measure of nonadaptive life-history polymorphism is expected.
3. High variability: For the highest degrees of variability in effective fecundity, the most iteroparous are favored. Moreover, if successive elements of the net reproduction schedule (i.e., $\psi(x) = l(x)m(x)$) are positively correlated (as they are in human life histories), delayed reproduction can be favored.

Thus, variation—even when restricted to effective fecundity/juvenile survivorship—

can yield a wide variety of optimal life histories. The outcome depends critically upon the correlation structure of the vitals rates and is sensitive to the possibility of nonadaptive invasion due to drift. It is only when the variation is highest that variation in effective fecundity unambiguously favors a large degree of iteroparity (i.e., a low reproductive effort strategy).

2. MODEL FOR THE COST OF REPRODUCTION IN A STOCHASTIC ENVIRONMENT

Assume an environment characterized by two states, good and bad. These states could reflect the productivity of the environment in a given year as a result of, say, rainfall, or they could reflect the average low temperature during the winter in a seasonal environment. For simplicity, assume these states are independently and identically distributed (i.i.d.). This model is depicted in Fig. 2. In a good year, the probability of remaining good for the following year is π_G and of becoming bad is $1 - \pi_G$. In a bad year, the probability of remaining bad for the following year is π_B and of becoming bad is $1 - \pi_B$. When $1 - \pi_G = \pi_B$, the environment is i.i.d. Note that in the Schaffer model, $1 - \pi_G = \pi_B = 0.5$ By changing the values of π_G and π_B so that $1 - \pi_G \neq \pi_B$, environmental autocorrelation is induced.

Compare three life histories: LH1, LH2, LH3. LH1 and LH2 are identical except for the poor-year survival of LH2, which is lower. This situation reflects a state in which the individual with LH2 is somehow physiologically compromised. When conditions are good, she is able to get by, but when conditions are bad, her lack of physiological buffering (e.g., through energy stores, throughput potential, utilization efficiencies, etc.) makes her more susceptible to mortality. This situation corresponds to a life history in which the cost of reproduction is paid in a state-dependent manner (Albon et al., 1987). LH3 is the same as LH2 except that it is characterized by an earlier age at first reproduction and higher mortality during the good years. Table 1 summarizes these schematic life histories. The first column indicates the relative age at first reproduction (AFR), while the second column indicates relative survival in the good-year environment. The final column indicates the bad-year survival relative to the individual life-history's good-year survival.

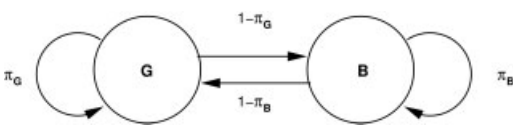

Fig. 2. Model of stochastic environment.

TABLE 1. Model life histories

Life history	AFR	Good-year survival	Bad-year survival
LH1	+	+	+
LH2	+	+	−
LH3	−	−	+

The environmental model is calibrated so that the limiting probability of being in the good state was $\pi_1 = 0.8$. Figure 3 plots the results of the model for the three model life histories. The stochastic growth rate of LH1 is substantially greater than the other two. The lowest stochastic growth rate—characterized by a decline of almost 3% annually—is LH2. LH3 is intermediate between the two. While it is not the optimal life history, it is substantially better than LH2.

Under the assumptions of this model, it is better to mature early and pay a cost in good-year survival than to play the standard tactic

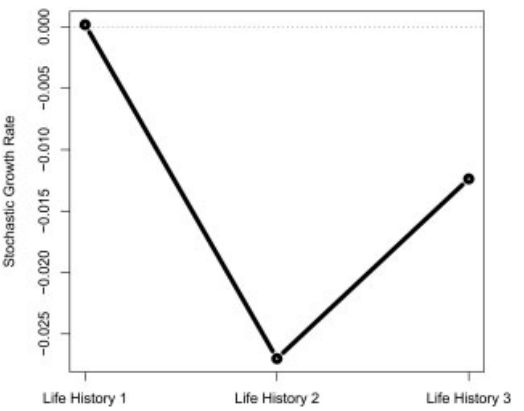

Fig. 3. Stochastic growth rates for the three model life histories. LH1 has the highest fitness, while LH2 has the lowest. LH3, while substantially lower than LH1, is much higher than LH2, illustrating the concept of making the best of a bad job. The line connects the three stochastic growth rates and is drawn simply to facilitate visual comparison.

and suffer severe bad-year mortality. However, it is better still to play the dominant tactic and get it right.

3. DISCUSSION

An adequate life-history theory of early life events should be capable of incorporating (1) the structuring of the human life cycle, and the overlapping generations so entailed; (2) stochastic variation in environmental quality, for this is what drives risk-spreading strategies like developmental plasticity in the first place; and (3) the information content of early environmental signals to the developing fetus or child.

The length of the human generation and the degree to which generations overlap greatly complicates the way reliable information can be apprehended by the developing fetus. An individual is likely to experience multiple bad years in her lifetime. With a generation length ranging from 25 to 30 years, the typical human forager would have probably experienced several bad years. The flip side of this is that this typical forager probably also experienced several particularly bountiful years. Life-history adaptations for poor environments should not be expected to be sensitive to short periods of poor energetic intake, though shortfalls during critical points in ontogeny can certainly establish constraints. A more integrated measure of the environment—e.g., "nutrition" (Kuzawa, 2005)—is likely to be a far more reliable indicator of local environmental quality and basis for state-dependent adaptive decisions.

It is in this sense that the predictive value of early environments is key. Colwell (1974) decomposed the overall predictability of an environment into two orthogonal factors: constancy and contingency. Constancy measures the degree to which the environment varies overall. A constant environment is completely predictable. Contingency, on the other hand, measures the regularity of change. Seasonal rains, for example, are a highly contingent environmental feature. Highly contingent environments are predictable. Organisms can adapt specific tactics to deal with a variable environments when the environmental information provides predictive value for the optimal tactic at a future point (see Horton, 2005; Crespi and Denver, 2005). A fundamental question in the context of the adaptive significance of fetal programming is what type of information regarding environmental predictability is being communicated to the fetus?

Thus, the form that environmental variability takes is essential. In a thought-provoking paper, Richerson et al. (2001) suggest that agriculture would have been impossible during the Pleistocene because of the high-frequency environmental variability characteristic particularly of the upper Pleistocene. Temperature proxy data (Anklin et al., 1993; Dansgaard et al., 1993) for the Greenland Ice Core Project (GRIP) show striking, possibly decadal-scale, temperature variation during the late Pleistocene. The GRIP data indicate possible temperature swings of 16°C in Greenland over the course of decades (Lang et al., 1999). While the tempo and magnitude of change are only partially understood, the GRIP data indicate that substantial, high-power, high-frequency variability has been a feature of the environment throughout the recent evolution of the genus *Homo*.

Long reproductive span (and, hence, generation length), delayed age at first reproduction, and high levels of parental investment can themselves be seen as adaptations to unpredictable environments (Tuljapurkar, 1990; Tuljapurkar and Wiener, 2000). A highly iteroparous organism integrates reproductive investments across a wide range of temporal environments. In effect, the long-lived organism hopes to "get lucky" on a couple occasions. Note that in order to maintain approximate demographic stability, a woman living in a high-mortality population with, say, 50% of children reaching breeding age, needs only to produce two surviving offspring. Nonetheless, total fertility rates of natural fertility populations frequently exceed 6–8 (Wood, 1994). Recent work on intergenerational transfers of hunter–gatherers further suggests that human children are massively expensive to rear, requiring 20 years of net parental investment (Kaplan, 2000). Given this background, the conditions that would favor the high "mating-effort" strategies suggested by Chisholm (1993) and Coall and Chisholm (2003) should be quite restricted. Trading-off parenting effort jeopardizes offspring recruitment (i.e., offspring survival to age of first reproduction), a critical juncture in the human life cycle.

A consideration of the general features of the human life cycle leads to a similarly critical evaluation of the weather-forecast metaphor for fetal programming of Bateson et al.

(2004). Regardless of whether or not it experienced fetal insult, an organism that takes 20 years to mature, while living in a variable environment, is likely to experience multiple periods of extended energetic shortfall throughout its ontogeny. In the absence of developmental constraint, is the optimal life history of an organism whose gestation corresponded to a low-energy period really going to differ dramatically from one that happened to be born at a good time? Both will experience a series of periods of energetic shortfall throughout their development. The critical difference between the two is the initial conditions, where their own physiologies dictate their growth. The organism whose gestation occurred during a poor energetic environment starts small and remains so for reasons of developmental constraint.

The discussion of fetal programming fits into larger considerations of the evolution of phenotypic plasticity. Some results from stochastic demography have a strong bearing upon the evolution of plasticity. Orzack and Tuljapurkar (1989) analyze a special-case model of an age-structured population in which only fertility (or, equivalently, juvenile survivorship) varies. If variability affects all age-specific fertilities equally, eq. (10) simplifies to:

$$\log \lambda_s = \log \bar{\lambda} - \frac{c^2}{2T_0^2},$$

where c is the coefficient of variation of the fertilities, and T_0 is the generation length.

While their results defy simple generalization, there is one clear result regarding variability in juvenile survival and iteroparity. As discussed above, although a variety of more or less iteroparous life histories have approximately equal fitness at intermediate levels of variability, extreme variation favors highly iteroparous life histories. As Tuljapurkar (1990) notes, phenotypic plasticity is measured by c. If phenotypic plasticity were increased in a variable environment, strong iteroparity would be favored. This results from the fact that greater iteroparity will lead to a larger value of T_0, the generation time, which appears in the denominator of the term subtracted from $\log\bar{\lambda}$. There is thus the strong possibility that plasticity and iteroparity—two hallmarks of the human adaptive suite—are fundamentally related.

If the downstream phenotypic consequences of early-life programming are adaptive, they are likely to be adaptive for the mother and not necessarily the child. The existence of a suboptimal phenotype in a given offspring is simply part of a mother's diversified portfolio, whereas for the offspring herself, it is the basis for all her future life-history tactics. This observation raises to possibility that developmental programming could usefully be viewed within the context of parent–offspring conflict, as has been suggested for pregnancy and other developmental phenomena by Haig (1993), a point made recently by Wells (2003).

Why does the Schaffer model make neat predictions about the impact of variance on life-history traits while the general framework of the stochastic demography of structured populations does not? Essentially, structure induces correlations, which are absent for the scalar life histories of the Schaffer model. These correlations shape in a fundamental way the response of a given life history to environmental variability. The covariances of the human life history are likely to be extremely strong because of the strong obligate parental care exhibited by humans. Specific data on mortality correlations across temporal environments in human populations are rare. However, Scott and Duncan (1998) have shown that the cross-correlation between child and adult mortality had its maximal peaks at zero time lag in a historical British mortality series. This result indicates that peaks of adult and child mortality coincided in the time series, providing powerful evidence for strong correlations between adult and juvenile survival. Again, since the fitness sensitivities of the average projection matrix will be highest for juvenile survivorship, such correlations will have a strong impact on the stochastic growth rate, the appropriate measure of fitness in a random environment.

The model discussed in section 2 illustrates the qualitative features of stochastic demography. It is not intended to represent an actual case, because insufficient data currently exist to estimate such a model. It is, however, designed to capture an essential feature of the fetal origins story, namely, that physiologies promoting survival in good versus bad periods are likely to conflict. Thus, the price LH3 pays for higher bad-year survival relative to LH2 is lower good-year survival. To estimate the stochastic growth rates of actual human populations, data need to be collected on the distribution of

environmental states and the corresponding state-specific demographic schedules. Estimates of the correlations between demographic rates across environments (as presented, e.g., in Scott and Duncan (1998)) are similarly critical—although these correlations are implicit if the state-specific demographic rates can be estimated.

The alternative life history modeled (LH3) in section 2 is characterized by early age at first reproduction. In general, early age at menarche is predicted by rapid somatic growth (Ellison, 1981), as demonstrated by the secular trend in menarcheal age (Tanner, 1990). Early age at first reproduction is nonetheless a possibility for the downstream phenotype. There are two reasons for the theoretical expectation of early first reproduction in the alternate phenotype: (1) other early-life insults are associated with earlier first reproduction (reviewed in Coall and Chisholm, 2003) and low birthweight and early reproduction may be linked as a complex; (2) there is little possibility that an alternate life-history tactic characterized by relatively late first reproduction and high mortality could ever be adaptive. Evidence suggesting that adults who experienced early insults have later reproduction and higher mortality would be *prima facie* evidence against the functional interpretation of fetal programming.

Future evolutionary investigations into the phenomenon of fetal programming should focus on (1) age at first reproduction, (2) early fertility, and (3) adult survival during bad years. Most of the epidemiological effects of fetal programming show relatively late age-of-onset: coronary artery disease, NIDDM, rate of senescence. The most important negative (or positive) consequences will be experienced before the age corresponding to the generation length of the organism.

4. CONCLUDING THOUGHTS ON HUMAN LIFE HISTORIES

Humans are long-lived and are characterized by late age at first reproduction, long reproductive spans, and highly overlapping generations. Furthermore, the rates at which people die or give birth change, often dramatically, with age. These features of the human life history complicate the evaluation of the optimal life history in variable environments. The tools for analyzing the demography of structured populations—in both deterministic and stochastic environments—can be brought to bear on questions of human life-history adaptation and suggest two robust generalizations. First, the force of selection falls predominantly on early survival. Second, a fitness-maximizing strategy for a structured population in a stochastic environment minimizes the variance in the rate of increase of the average life history. Two possible strategies for minimizing this variance include reducing positive correlations between vital rates—particularly ones to which fitness is highly sensitive—and increasing mean generation length through delayed first reproduction and iteroparity.

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