

Life History Theory and Evolutionary Anthropology

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Life-history theory has been developed in biology to explain the variation in timing of fertility, growth, developmental rates, and death of living organisms, as well as events directly tied to these parameters. The theory is useful in explaining variations in age-specific human fertility and mortality patterns, as well as understanding how the human life course evolved to patterns so divergent from those that characterize our close primate relatives. Surprisingly, this same theory can also be used to explain why people often ignore the long-term consequences of behaviors that produce short-term gain.

WHY STUDY LIFE HISTORIES?

Medical professionals and public health officials are waging a losing battle to convince Americans to do what is best for them. Indeed, recent news reports suggest that doctors do not even heed their own advice about diet, exercise, work patterns, and a variety of other topics. The reason is simple. People are not designed to maximize their life span or to place their own longevity above all else when they must make decisions. Instead, we, like all other organisms with separate somatic and germ-cell lines, are designed by natural selection to die. In biological terms, people should be quite willing to sacrifice survival for important reproductive gains because we, like all other organisms, face tradeoffs between fertility, mortality, and the mortality of our offspring. Particular solutions to these tradeoffs will result in a higher contribution to the gene pool through time. Thus, the human biological machinery assigns priorities in a way that does not emphasize maximum longev-

ity, a fact that has not been appreciated by the human biologists, public health specialists, social scientists, and members of the medical establishment who are in the business of telling us what is best for us. Instead, these specialists have concentrated ever more closely on the molecular and physiological mechanisms that lead to death and on environmental conditions that constitute hazards. These emphases have been based on the belief that documenting the factors that decrease longevity will be sufficient to motivate people to avoid them, thereby decreasing mortality rates. The problem with this logic is that people expose themselves to mortality and morbidity hazards every day in the course of doing what they are designed to do—propagate their genes. Identifying health hazards alone, without studying the priorities that govern human decisions, will not affect the mortality rate of individuals who willingly and knowingly incur such risks.

Imagine giving advice to parents faced with the decision of whether or not to enter a burning building to save their own children. Even though saving their children would entail the risk of death, the parents would be likely to take that risk, ignoring the advice of anyone who told them to think only of their own survival. In an analogous situation, public health officials confidently advise Americans to change their diets and lifestyles, armed only with the knowledge that particular be-

haviors are associated with shorter life spans. These same health officials are at a complete loss to understand why the public so often ignores their advice. Whether there are short-term benefits associated with high salt and lipid diets, low fiber intake, smoking and drug use, unsafe sex, or type A personalities and lifestyles, etc., that are sufficient to outweigh the long term cost of such behaviors to individuals who adopt them has apparently never been considered by the public health establishment let alone been measured and evaluated carefully. We know that people with type A personalities who work hard generally receive economic benefit despite the fact that they may die young. Smokers claim that they perform better under stress; coffee drinkers believe they have more pep at the beginning and end of the day. High-salt diets may turn out to improve physical or mental performance. Recent data suggest that cholesterol may help combat depression.¹ The realization that people may ignore health advice because they are willing to sacrifice long-term health in order to gain short-term advantage has led to the search for a theory about such tradeoffs. Such a theory now exists. It comes from observations about life cycles of living organisms and their variation.

Among different species, the timing of life-course events such as giving birth and dying can seem curious and strange, almost arbitrary. This is bothersome to biologists, who recognize that phenotypic traits generally have specific functions and that life-course events such as survival and reproduction, being direct components of fitness, should be subject to strong natural selection. Comparisons both across and within species seem puzzling. Consider our own nearest phylogenetic relative, the chimpanzee. Generally, human hunter-gatherers

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are only slightly larger than wild chimpanzees, yet have an adult lifespan from maturity to death that is almost four times as long. For example, the average *adult* lifespan beyond sexual maturity for Ache hunter-gatherers is about 42 years, as compared to about 14 years for *Pan troglodytes*.^{2,3} Because survival should be strongly favored by natural selection and body size is generally associated with longevity, why do humans have such a long lifespan compared to other mammals of their size? Even more strangely, despite the fact that humans live longer, human females in natural fertility populations reproduce at a much faster rate than wild chimpanzees do. The average interbirth interval between surviving offspring among hunter-gatherers ranges from about 30 to 45 months, considerably less than the 60-month interbirth interval of wild chimpanzees.

Other comparisons with our closest phylogenetic relative are also puzzling. Chimpanzees and traditional humans grow at almost exactly the same rates during childhood, but chimpanzees stop growing and start reproducing about four years earlier than humans. As a result, they are slightly smaller as adults. Human females spend about half their adult lives in a post-reproductive phase, whereas chimpanzees and other primates generally reproduce throughout their entire lives, with, perhaps, a short period of secondary sterility during advanced old age. How could such an enormous divergence of life cycle take place during the short evolutionary period that has separated the two genera and, more importantly, what are the ultimate causes of these differences?

Comparisons within the human species across populations are equally intriguing. Recent improvements in nutrition in developed countries have led to changes in timing of the life course. Girls in well-nourished populations grow faster and reach sexual maturity at an earlier age, but also achieve a larger adult body size than do girls raised under poor nutritional conditions. In the same populations, however, there is little evidence, if any, of a change in the age of menopause. Although medical advances, along

with improvements in nutrition and sanitation, have drastically reduced infant, child, and adult mortality rates in modern societies, the absolute lifespan has not significantly increased. In the past, some individuals lived to be a hundred years old; today, few people survive much beyond a century.

Important differences in life-course trajectory can be seen within single societies such as our own. In the United

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States, women living in poor areas, inner city neighborhoods, rural counties, and Native American reservations generally begin reproduction in their mid-teen years. Women in the upper-middle class reach menarche at an early age, but choose to delay childbirth, often until they are well into their third decade, after more than half of their reproductive span has passed.

All these observations may seem unrelated. After all, what can the maturation rate of apes and other primates tell us about why modern college-educated women are using birth control to delay their reproduction for two decades after they have reached sexual maturation? What can the maturation rates tell us about why Americans continue to eat high-salt, high-fat diets against the advice of their doctors?

What unites these and many other apparently unrelated observations is that all have to do with the timing of important biological events. Thus, they fall within the range of a new, rapidly developing, and comprehensive biological theory about the timing and significance of life-course events. This theory attempts to explain why, despite initially puzzling variations, certain patterns or sets of traits can be seen as evolving in systematic and predictable ways within and between species. These evolved life histories may ultimately provide the key to understanding why humans often fail to avoid behaviors that can have long-term negative consequences.

LIFE HISTORY THEORY

A developmental biologist, J.T. Bonner,⁴ noted that the ultimate description of an organism is not just a description of its adult phase, but that of its life cycle. Life history consists of age-specific schedules of mortality and fecundity, as well as traits that are either directly the result of these schedules (such as life span and age at first reproduction) or that are directly connected to them (such as growth, body size, and developmental trajectory). Thus, the life history of an organism can be thought of as a complete description of that organism. The study of life-history evolution is the analysis of the evolution of fitness components. With such a grand view, it is not surprising that life-history theory is complex and still underdeveloped as compared to areas of evolutionary biology such as foraging theory or sex allocation theory. Nevertheless, a considerable amount is known about the way that life-history traits correlate with each other and are constrained into possible sets of relationships that are repeatedly observed regardless of an organism's phylogenetic affiliation. The conditions that lead to the tremendous diversity of observed life histories have been investigated in increasing detail during the past ten to fifteen years.⁵⁻⁷

The basic tenet of life-history theory, which logically flows from the laws of thermodynamics, is the principle of allocation, which states that energy used for one purpose cannot be used for another. As organisms pro-

ceed through their life cycle, they harvest energy from the environment and invest it in various life functions. Decisions about how to invest energy are made at the molecular, physiological, and behavioral levels. In general, we should expect natural selection to result in optimal allocation patterns, given relevant constraints. These patterns result in the life history of an organism. Natural selection can be expected to result in genetically evolved life histories that, subject to genetic and ontogenetic constraints, lead to higher fitness than would other feasible alternatives. Selection can also result in the ability to adjust life history traits facultatively in ways that are adaptive to a wide variety of circumstances. Both types of life-history predictions have been extensively examined in organisms ranging from plants and invertebrates to mammals.

Energy harvested during the life cycle can be used for maintenance and repair of the soma, growth, storage, or reproduction. Because energy used for one purpose cannot be used for another purpose, living organisms face a series of tradeoffs through time. The two most fundamental tradeoffs, which are at the center of all life-history theory, are those between current and future reproduction and between the number and fitness of offspring. Tradeoffs in life-history traits may be the result of facultative decisions or may have a strong genetic basis. Indeed, gene expression can result in a structure that takes energy not only to build, but also to maintain, and may preclude the efficient expression of other structural or behavioral traits later in life. Often, organisms pay high energy costs in order to escape the constraints of this "genetically expressed pleiotropy" through the evolution of complex life cycles that include one or more metamorphic events.⁸ In those events, structures are resorbed, after which they no longer require energy and constrain developmental expression. In theory, most, if not all, genetic tradeoffs could be avoided if the benefits of somatic reorganization, including organic resorption and resynthesis, outweighed the costs. In practice, however, genetic tradeoffs are likely to be common and may often result in a series of detrimental life-

history traits later in life.

The tradeoff between current and future reproduction generally has been referred to as the cost of reproduction.⁹ Experimental manipulation and genetic studies have shown a reduction in survival or fertility following higher reproductive effort among many animals.^{5,6} Such costs are virtu-

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ally impossible to measure using natural variation in a population because of phenotypic correlations. Such correlations are expected because individuals who are in better condition will often show both higher early reproductive rates and higher subsequent survival or fertility through time. This means that good measures of the cost of reproduction require sophisticated experimental or genetic manipulation.¹⁰

A good estimate of the reproductive cost function is critical for modeling optimal life-history trajectories for any organism under any set of conditions. This raises serious problems for human biologists. The cost of human reproduction is difficult to assess because of the inability to breed selectively for particular traits or to carry out experimental manipulations of reproductive effort. Nevertheless, some natural experiments can provide useful information when subjects do not self-select into high and low reproductive effort groups. Sophisticated multivariate analyses may also allow us to use comparative samples and partial out the effects of phenotypic correlation in order to obtain estimates of the cost of reproduction.

A useful tool to investigate life-history traits is the concept of reproduc-

tive value developed by Fisher.¹¹ The reproductive value of an individual of age x is its expected number of future offspring, measured in units of currently existing offspring. In a growing population, delaying reproduction reduces fitness because each delayed future offspring will represent a smaller proportion of the future population (Box 1). Thus, each future offspring in a growing population represents a smaller proportional contribution to the gene pool as compared to each currently existing offspring. Even in a stationary population, delaying reproduction is not advantageous if there is a large probability that the individual will not survive to reproduce at a later age. Thus, it seems that natural selection should never favor delayed reproduction. However, energy not spent in reproduction at a given age can be used to increase future reproductive potential (for example, by growth) or to increase the likelihood of survival for subsequent reproduction (e.g., by storing extra fat). As long as the energy saved by not reproducing at age x increases future reproductive value by more than the loss in current fecundity, it pays to delay reproduction (Box 1).

The second tradeoff implied by the principle of allocation is that between the number and fitness of offspring produced. With the same reproductive effort, an individual can produce many cheap offspring who have a low chance of surviving and finding a mate or can produce fewer but higher-quality offspring having a higher probability of surviving and higher potential fertility. This tradeoff, first explored by Lack,¹⁴ was quantitatively described by Smith and Fretwell.¹⁵ I extend Smith and Fretwell's logic to include the effects of parental fertility on the reproductive value, as well as survivorship of offspring. We can simplify the fertility decision by assuming that all investment in one reproductive event is terminated before investing in the next. If we assume that an individual is committed to a particular level of reproductive investment (I_x) at time x and expends i units of investment per offspring, then age-specific fertility (m_x) is equal to the total investment allocated to reproduction divided by investment

Box 1

The discrete-time definition of reproductive value generally employed in studies with real data is:

$$V_x = \sum_{y=x}^{\infty} \frac{1}{l_x} \cdot m_y \cdot e^{-r(y-x+1)} \quad (1)$$

where V_x is reproductive value at age x , m_x is fertility at age x , l_x is survival to age x , and r is the instantaneous growth rate of the population. Offspring born at some future year y rather than at time x are subject to two forms of discounting. First, the parent has only probability $1/l_x$ of surviving from age x to age y ; thus, the expected contribution at time x of each offspring actually born at time y is correspondingly less than that for each offspring born at time x . Second, if $r > 0$, the total population size at time y will be greater than that at time x by the factor $e^{r(y-x)}$, thereby diluting a parent's reproductive contribution by that amount relative to producing the same number of offspring at time x . The exponent also includes a +1 term so that reproductive value will match the counting convention of the Leslie Matrix¹² and the Lotka equation¹³ in which the sum starts at the first age class, and $V_0 = 1$.

The right-hand side of the first equation (1) can be broken down into two components representing present and future reproduction⁹:

$$V_x = m_x \cdot e^{-r} + \sum_{y=x+1}^{\infty} \frac{1}{l_x} \cdot m_y \cdot e^{-r(y-x+1)} \quad (2)$$

It is readily apparent that if increasing current fertility (m_x) entails a decrease in survival ($1/l_x$) or future fertility (m_y), then the optimal value of m_x depends on the exact quantitative relationship between current fertility and other components in (2). For instance, if $r = 0$ (a stationary population), then a decision between increasing m_x by an absolute amount g and decreasing $\sum m_y 1/l_x$ by an absolute amount c should depend on whether $(g-c)$ is positive (decide to increase current reproduction) or negative (decide to delay reproduction to age y).

When $r > 0$, the costs to future reproduction are devalued relative to the gains to current reproduction by the same degree that future offspring are devalued relative to offspring produced now. Thus, in the previous example, if the population size at time y was twice that at time x , a decision to delay reproduction to time y would require the more stringent condition that $g - (c/2) < 0$. Thus, natural selection may favor investment in immediate fitness gains (e.g., early reproduction) at the expense of later reproductive success either because mortality rates are high or because population growth rates are high.

per offspring, or Ix/i . In a population that is not growing, fitness obtained with I units of reproductive investment will be equal to the product of the number of offspring produced times their survival to adulthood times their reproductive value at adulthood. For any given level of reproductive investment, the more offspring produced, the lower their quality must be. Because low-quality offspring tend to survive less well and to have lower fertility, each contributes less to the parent's long-term genetic representation than would high-quality offspring. Hence, the product of these terms, which is the parent's expected genetic contribution, usually is maximized by producing offspring of intermediate quality (Box 2). It is important to note that the optimal investment per offspring does not depend on the parent's level of reproductive investment although, of course, the number of offspring actually produced does (see Box 2).

Animal experiments have shown that this tradeoff does, in fact, characterize parental investment decisions.^{5,6} However, natural variation in parental investment per offspring (offspring size, clutch size, and interbirth

interval) cannot be used for direct estimation of the relationship between investment and offspring fitness. Parents who have more available resources are likely to produce both more and higher-quality offspring simultaneously, giving rise, again, to the problem of phenotypic correlations. However, when parental quality has been carefully controlled through manipulation of existing brood size or controlled feeding, the tradeoff between offspring number and fitness has easily been demonstrated.¹⁶

Thus, in summary, two important tradeoffs exist in the lifecourse: that between current reproductive effort versus future reproduction and that between the quantity and quality of offspring. The disaggregated form of the reproductive value equation (equation 2) directly shows the cost of reproduction tradeoff (m_x vs. l_y and m_y) and includes the offspring number versus fitness tradeoff embedded in it as the optimal fertility solution $m_x = m_x^*$.

Time Preference (or Cost-benefit Discounting)

The first tradeoff in life-history theory, that between current and future

reproduction, leads to an interesting set of biological problems that in many ways are analogous to discounting in the economics of interest rate theory.¹⁷ Should an organism take a small gain now or wait for a larger gain in the future? Should an organism take a small cost now to avoid paying a larger cost later? Intuitively, we know that this depends on how much greater the benefit or cost would be at a later time and on the probability that the benefit or cost will be realized.

In life-history theory, tradeoffs between current and future gain are central to understanding all aspects of phenotypic variation. The principle of allocation implies that all current investment in reproduction will entail a future cost in survival or fertility. However, the true impact of future costs is determined by the extrinsic mortality curve and the growth rate of a population. When survival is low, future costs are unlikely to be paid by most individuals, and thus should be discounted accordingly (Box 1). When population growth is rapid, future reproduction will contribute a lower proportion to the gene pool, whereas currently produced offspring will themselves contribute to the growing

Box 2

If total reproductive investment at time x , I_x is fixed, then $m_x = I_x/i$, where i is the investment per individual offspring. When survival to maturation, S_a , and reproductive value at maturation, V_a , are functions of i , then the optimal fecundity at age x , $m^*x = I_x/i^*$, where i^* , the optimal offspring investment, is found by solving (eqn. 4):

$$\frac{d \left[\frac{I}{i} \cdot S_a(i) \cdot V_a(i) \right]}{di} = 0 \quad (4)$$

subject to the condition that the second derivative of this

equation is negative. The solution to equation 4 is (eqn. 5):

$$i^* = \frac{S_a(i) \cdot V_a(i)}{S_a(i) \cdot V_a'(i) + S_a'(i) \cdot V_a(i)} \quad (5)$$

Note that the optimal investment per offspring is independent of how much parents have to invest (since I does not enter into the solution for solving i^*). If I_x/i^* is not an integer (as real numbers of offspring must be), the parent may either produce a slightly larger clutch of slightly sub-optimal size offspring, or a slightly smaller clutch of optimally sized offspring and store the extra energy for later survival or reproduction. Which option is taken depends on the time-discounting of present versus future reproduction (see Box 1).

gene pool. Thus, current reproduction is generally worth more in fitness units than is future reproduction (Box 1).

Most social scientists have not fully appreciated the importance of this point with regard to human behavior outside the economic arena. Its implications for medicine, public health, and public policy are enormous, yet have barely begun to be explored.^{17,18} People may be quite willing to engage in behaviors that provide small immediate benefits even if they are likely to impose high costs later in their lives (smoking, drinking, high-salt or high-fat diets, crime, etc.). Similarly they may not be willing to incur small costs in the present in order to avoid much higher costs later (exercise, educational investment, conservation of resources, saving, etc.). As biologists, we can expect natural selection to design a central nervous system that makes such decisions. The public policy implications may be depressing, but should be taken into account. Life-history theory and the principle of time preference can often make sense of what seem to be irrational decisions that involve linked costs and benefits spread over long periods.

Senescence and the Maximum Life Span

Senescence is a progressive increase in age specific mortality even when conditions for survival are ideal. Why organisms senesce is an important biological question. Recent exploration of this question using life-history theory has emphasized that the molecular mechanisms of aging are part of the question rather than

the answer to it.^{19,20} Rates of senescence among mammals vary considerably. Among humans, for example, the adult mortality rate doubles every eight years, whereas among mice it doubles every 120 days.²⁰ I previously suggested that large costs may be incurred late in life in exchange for small fitness gains early in life. This, in essence, is one likely explanation of why organisms grow old and deteriorate near the end of the normal adult life span.²¹ With a constant mortality rate, fewer and fewer individuals will live to a particular age. Deleterious effects that become manifest at ages when few individuals are likely to be alive simply are not under strong negative selection.²² If the same gene that produces a small gain in early fitness leads to the complete collapse of a major organ system late in life it still may be favored by selection. Thus antagonistic pleiotropy may lead to physiological deterioration and senescence.

One particularly interesting form of the "antagonistic pleiotropy" theory of senescence is called the disposable soma theory.²³ The proponents of the disposable soma theory point out that a certain amount of energy must be dedicated to somatic maintenance and repair in order for an organism to survive indefinitely. Without sufficient repair, molecular and physiological systems will deteriorate and ultimately become nonfunctional. Kirkwood and Rose have developed a mathematical model of the tradeoff of energy between maintenance and reproduction.²³ They conclude that under conditions common to living organisms, natural selection will favor

allocating less energy to maintenance than is necessary for indefinite survival.²³ Thus, many organisms can be expected to "reproduce themselves to death" through low somatic maintenance.

These theories suggest that rates of senescence are directly related to the extrinsic adult mortality rate, or the rate of death from causes that cannot be avoided by behavioral or physiological counter-strategies. This means that organisms having too high rates of mortality resulting from predation, accidents, or unavoidable environmental factors should experience rapid physiological senescence. This prediction is supported by one recent study on opossums living on islands (without predators) and on the mainland in the southeastern United States.²⁴

Current theories of senescence also invoke a tradeoff between reproduction and life span as the major reason why life spans are not infinite. Energy that could be used to extend the life span is instead used in reproduction. This notion, which is consistent with the cost-of-reproduction tradeoff discussed earlier, is also supported by studies showing that selection for longevity leads to decreased reproductive function.^{25,26} In addition, numerous studies have shown that selection for increased fertility results in a reduced life span.¹⁰ Indeed, there are cases in which the act of mating is itself sufficient to decrease survival even when no offspring are produced.²⁷ It is particularly interesting to note that sterilized individuals from species as diverse as fruit flies,²⁸ to cats, and hu-

mans^{29,31} have longer life spans than do animals with intact reproductive function. Thus, the data support the view that in order to propagate our genes at maximal rates we essentially are designed to die as a result of our insufficient investment in mechanisms to increase longevity.

Age at Maturity and Adult Body Size

Models of optimal age at sexual maturity have been among the most successful life history models for predicting actual observed values of a trait in natural populations.³²⁻³⁷ An elegant but simple model is that of Charnov,³⁵ who assumed zero population growth and simplified reproductive value at birth in two components—the probability of surviving to age of first reproduction and reproductive value at the age of first reproduction—the product of which defines lifetime reproductive success. If reproductive value at the age of the first reproduction increases with age, an organism is faced with a trade-off between beginning reproduction at a lower reproductive value but higher probability of survival, or waiting until the probability of survival will be lower but the reproductive value will be higher.

Thus, organisms face a trade-off between reproductive value and survivorship and should be selected to begin reproduction at an age that will maximize the product of juvenile survivorship times their reproductive value at maturity. The optimal age of maturity is when the proportional change in reproductive value (which increases with age) exactly equals the proportional change in juvenile survivorship (which decreases with age). If reproductive value is proportional to body mass and the minimum (adult) mortality rate is reached prior to maturity,^{5,6} this condition is the same as saying that the weight gain per unit time divided by body mass at maturity should be equal to the adult mortality rate. Using allometric relationships between growth rate and body mass, Charnov^{7,34} shows that age at maturity and other related traits such as adult life span and fecundity can be predicted accurately across mammals. More complicated models that

measure fitness as the intrinsic growth rate (r) as a function of age at maturity, and that use the empirical relationship between body size and reproductive value have also successfully predicted age of maturity in a variety of plants and animals.^{5,6}

Although all organisms have a maximum body size that is determined by the metabolic expenditure and the energy-harvest functions of body size, the theory predicts that many organisms should stop growth well before their theoretical asymptote. Moreover, the growth halt may be quite abrupt if the growth rate is steep at sexual maturity. Knowing the time of first reproduction and the growth function with respect to time also allows prediction of observed adult body size. Thus, a single model not only explains age at maturity, but also mean adult body size, if feeding niche and growth rates are taken as givens.

There is strong empirical support for the idea that age at maturity represents the optimal solution to a tradeoff between increasing reproductive value and decreasing survival. Temporarily increased mortality rates around the age of maturity have been shown to lead to earlier sexual maturity in organisms ranging from invertebrates to mammals.^{38,39} Most interestingly, several experimental studies show that animals can react to indirect cues indicating hazards to adult mortality by accelerating their maturation rates.⁴⁰ For many aquatic animals, the mere presence of chemical substances associated with predators or predatory action is enough to produce earlier sexual maturity.⁴¹ These findings have obvious implications for the interpretation of data suggesting that human females may reach sexual maturity at younger ages under a variety of stressful conditions.⁴² The theory can also be used to predict facultative shifts toward early and late reproduction in modern societies under conditions of varying adult mortality. Interestingly, some sociologists⁴³ have used this same logic (but not based in life-history theory) to explain high rates of teenage pregnancy in American inner-city neighborhoods.

The models for age at maturity can also be used to examine the secular

trend in age at menarche for modern human populations. This phenomenon clearly represents phenotypic plasticity to a variable environment. We can determine the extent to which the observed trend can be understood as an adaptive "reaction norm". One study using the assumption that better nutrition leads to both higher growth rates and higher juvenile survivorship correctly predicted both the character of the observed secular trend (i.e., that well-nourished women reach menarche earlier, but at a larger body size than women who are poorly nourished) and the actual ages and body sizes of women at menarche in European societies of the nineteenth and twentieth centuries.⁴⁴

It should be emphasized that any parameter that affects reproductive value through time can be substituted for the growth function in order to predict optimal time of first reproduction. In humans, for example, much energy is stored extra-somatically. Factors such as education, which influence income throughout the life span may affect $V(\alpha)$ and may be more appropriate than body size for predicting the optimal age at the commencement of reproduction. Indeed, this life history framework easily accommodates many American women's use of contraception to delay first reproduction so that they can pursue educational opportunities that will increase their adult income. Such a model is consistent with women's own statements about delaying reproduction in order to finish their education and, presumably, attain a higher income level. Body size, however, may be a good indicator of proportional increases in reproductive value in many traditional societies. In fact, a model that uses the empirically observed relationship between body size and fertility can accurately predict the age at first reproduction for the Ache foragers of Paraguay.²

Optimal Litter Size, and Inter-birth Interval

Lack¹³ was the first biologist who explicitly modeled the tradeoff between the number and fitness of offspring. There is a tendency, often referred to by evolutionary biologists as the Lack effect, for selection to fa-

vor an intermediate litter size because it leads to the highest number of surviving offspring. With a given energy budget for reproduction through time, parents are forced to partition resources in ways that affect the survival and future fertility of their offspring. This tradeoff has also been studied by modeling optimal offspring size,¹⁵ which defines the optimal litter size when reproductive effort during a single bout of reproduction is controlled. Animal studies have provided strong confirmation that a tradeoff between the quality and quantity of offspring exists when total parental effort is carefully controlled.^{5,6} Subsequent theoretical exploration suggests that this tradeoff is often complicated by parent-offspring conflict,⁴⁵ which can lead to a litter size far from the predicted parental optimum. Optimal litter size is also likely to be affected by the cost of the litter to parent's remaining reproductive potential,⁴⁶ as well as the effects of stochastic variation on litter size and the fitness gain curve.⁴⁷ Thus, there are many reasons not to expect an exact fit between the predicted optimum litter size (or interbirth interval) and the observed values in real populations when models are based only on the tradeoff between litter size and offspring survival.

Despite these complications of the theory, one human study has reported support for the simple version of the Lack tradeoff between offspring number and fitness. Blurton Jones⁴⁸ measured success rates at raising children to adulthood for different interbirth intervals among the !Kung. He reported that the interbirth interval that led to the highest number of surviving offspring for !Kung parents was also the most common interbirth interval in the population, approximately 48 months. This study, the first to apply evolutionary logic and an optimization approach to human fertility, thus makes an important theoretical contribution. The results must be evaluated cautiously, however, for several reasons.

First, there is no a priori reason to expect that !Kung women who had modal interbirth intervals should have the highest fitness. The likelihood of phenotypic correlation in this situation instead suggests that women

with the shortest inter-birth intervals should have the highest fitness.⁴⁹ Simply put, if there were variation in the condition of !Kung females, those in better condition would be likely to have slightly higher fertility than others and equal or higher offspring survival rates. Second, there is a series of methodological problems with the study, including the fact that the success rate of pairs of children who span an interbirth interval, was measured, not the child mortality rate. Children who survived were counted as having failed if their prior or following sibling died before adulthood, whereas two deaths were not necessarily counted when both siblings died. Third, interbirth intervals were not directly observed, but were estimated by a variety of techniques by an observer who may have been biased by a belief that dead infants should have been born after shorter interbirth intervals.⁵⁰ Finally, the raw data on the interbirth interval success rate do not support the Lack model. Instead, the mortality data were statistically fitted to a "backload model" and the expected mortality from that model was then used to support the Lack interpretation. Although such a procedure is theoretically satisfying, it can lead to flawed estimates of mortality if the model is incorrect. If the raw data are taken at face value, the interbirth interval that maximizes the rate of producing surviving offspring is 72 months, with 84 months being the second best interbirth interval. This is far from the most commonly observed interval.

Parental condition undoubtedly varies significantly among most human groups. The problems of phenotypic correlation lead us to realize that natural variation in interbirth interval should not provide an accurate assessment of the costs of shorter interbirth intervals in any population. Only those parents who make a strategic error (in evolutionary terms) should have higher mortality associated with shorter interbirth intervals. Studies on birds suggest that it should often not be possible to measure a Lack effect without experimentally manipulating brood size.¹⁶ Thus, we should carefully examine any study that claims to show either a positive relationship between interbirth interval and survival

in natural populations or a relationship strong enough to suggest higher fitness for women with modal interbirth intervals. Many studies have failed to find such an effect.^{2,53-55}

Menopause

The fact that human women cease to reproduce when they still have a high probability of surviving for many years is a rare pattern among mammals and one that probably is absent among all other primates.^{56,57} Among the Ache hunter-gatherers, for example, menopausal women who are 45 years old can expect, on average, to live another 21 years. The fact that 45-year-old women have virtually no remaining reproductive function presents an interesting challenge to life-history theory. Several evolutionary biologists have suggested that this pattern could be favored by kin selection if older females were able to use energy more efficiently to increase the reproductive success of their close relatives than they could by attempting to produce additional offspring of their own.^{21,58-62} A theoretical exploration suggests that menopause could indeed evolve by kin selection under certain conditions.⁶³ However, a recent empirical test of this hypothesis⁵⁶ failed to find evidence that menopause could be maintained through kin selection in one group of hunter-gatherers. Current evidence does not provide good support for the idea that kin selection explains the maintenance of menopause in current human populations. (Whether or not it explains the origin of menopause is another issue.) Instead, a tradeoff between early and late reproductive function seems more likely to explain why human and some other mammalian females lose reproductive function long before the end of their life span. Specifically, Peter Ellison (personal communication) has proposed that the human female trait of producing all viable oocytes before birth and storing them in arrested metaphase throughout the life span (semelgameogenesis) may be a mechanism to reduce the mutational accumulation that takes place with cell division. This same mechanism, however, leads to the ultimate depletion of viable oocytes. Presumably, the gains in fitness

early in life outweigh the later pleiotropic costs of reproductive senescence.

Interestingly, baleen whales and elephants appear to be the only mammals that reproduce at ages greater than 50 years, and both are large enough to contain, at birth, ovaries with a higher number of viable oocytes. Some species of whales, but not elephants, also have lifespans similar to that of humans. Whether or not all mammalian females are characterized by semelgametogenesis is unknown. But because humans have an exceptionally long life span relative to their body size, it would not be surprising if human females are one of only a few species that literally run out of oocytes during adulthood. On the other hand, good data also suggest that female toothed whales of several species undergo reproductive senescence at about the age of 40 years even though, having a normal life span of more than 80 years, their mortality hazard is still low when fertility terminates.^{64,65} Thus, a coherent evolutionary model of menopause remains a serious challenge for life-history theory.

Reproductive Value and Biased Kin Investment

Parental investment theory suggests that parents should invest in offspring according to the likely fitness returns of such investment. This realization is the basis of modern sex ratio theory,⁶⁶ but also forms the basis for understanding a variety of other phenomena related to parental investment patterns. If offspring are at risk of death, parents should take action proportional to the reproductive value of those offspring if they survive the immediate risk. Thus, when individuals can expend energy to affect the well-being of alternative offspring or other relatives, kin will be given priority according to their reproductive value (devalued by their genetic coefficient of relatedness).⁶⁷ This rule of investment should be applied to parental care decisions and in patterns of altruistic behavior between close kin. Moreover, it introduces an important modification to Hamilton's Rule of kin selection.⁶³

In many societies, small children who face high mortality rates before

adulthood and the first possibility of reproduction will have low reproductive value. Parents should devalue such children relative to older children or children in societies characterized by higher survivorship. Infanticide, child homicide, and child

...human females, despite the fact that they have longer reproductive spans, generally reproduce at higher rates than chimpanzee females. Because reproduction takes considerable energy above metabolic demands, this either means that human females are more efficient at harvesting energy than are chimpanzee females or that human females acquire extra energy from other sources...

neglect patterns should all reflect these differences. Available data on human societies ranging from hunter-gatherers to modern Americans indeed suggest that children are most often the victims of homicide, particularly when the killing is done by a relative.^{2,68} Similarly, elderly people who produce no resources and are unlikely or unable to reproduce may be of relatively low value to close kin (and themselves). The tendency for elderly individuals to be killed, selectively allowed to die, or even to terminate their own lives, also makes sense in light of Fisher's reproductive value equation and the expenditure of resources to promote genetic contribution. The connection between reproductive value and biological worth has even

led some evolutionary anthropologists to suggest that across societies, different patterns of mourning for individuals who die at different ages can be partly explained by age-specific reproductive value.^{69,70}

Humans, Chimpanzees, and Hominids

Anthropologists have long realized that the life-history trajectory of humans is very different from those of our nearest phylogenetic relatives. Humans live longer, are larger, develop more slowly during childhood, and begin reproduction at later ages than chimpanzees. Many of these traits can probably be best understood as the consequences of radically different adult mortality rates. Other traits, however, are more complicated and are undoubtedly related to differences in parental investment patterns that keep human juveniles dependent on adults for their survival almost until they reach sexual maturity. Life-history theory can be used to examine these patterns and to make informed guesses about when hominid life histories began to diverge from those of pongids. Some of these predictions undoubtedly can be tested by examining patterns of dental eruption and skeletal maturation in fossil hominids. Work in this area has already suggested that the life histories of some early hominids may have been much more similar to that of chimpanzees than that of modern humans.^{71,72}

Human adults have exceptionally low mortality rates compared to our nearest primate relatives, as well as mammals of our body size. A simple regression model of log survivorship by weight suggests early adult mortality rates on the order of 0.14 to 0.2 per year for mammals of human body size.⁷³ Chimpanzee data³ allow a crude estimate of early adult mortality at around 0.07 to 0.1 per year. On the other hand, current data on human hunter-gatherers and technologically primitive groups suggest that human early adult mortality during recent evolutionary history has been around 0.01 to 0.015 per year.^{2,50,74,75} The fact that humans have a five to ten-fold decrease in adult mortality rate as compared to chimpanzees has important

ramifications for understanding hominid life-history divergence. First, humans and chimpanzees grow at about the same rate throughout childhood.^{76,77} Thus, using the models for the optimal age at first reproduction, it is readily apparent that chimpanzees should begin reproduction at earlier ages than humans and, thus, at smaller sizes. This should change the entire developmental trajectory to ensure that juvenile development is complete before the optimal age of maturity.

One might also predict that chimpanzees would have higher fertility rates than humans throughout the life span. This is not the case. Instead, human females, despite the fact that they have longer reproductive spans, generally reproduce at higher rates than chimpanzee females. Because reproduction takes considerable energy above metabolic demands, this either means that human females are more efficient at harvesting energy than are chimpanzee females or that human females acquire extra energy from other sources (i.e., human males). Given their higher fertility and longer reproductive spans, one might also expect that humans would have higher juvenile mortality rates than chimpanzees (indeed, this would have to be true if both populations were stationary). But recent studies of hunter-gatherers indicate that this is not the case, and thus lead to the question of how human populations maintained zero growth rates during their first 90,000 years of history. Either currently available data do not represent typical demographic patterns of our species through history or frequent population crashes have been an important feature of that history. Humans also have exceptionally long periods of juvenile dependence that are partially subsidized by male economic contributions. This allows offspring to develop slowly and remain relatively helpless almost until they have reached full adult size. Again, one might expect this to slow human reproductive rates relative to those of chimpanzees, but the opposite has been observed.

This brief comparison suggests that events associated with a reduction in hominid adult mortality rates and the origin of male provisioning patterns are critical to understanding most dif-

ferences between the life-histories of humans and chimpanzees. Because the only good data on adult mortality among chimpanzees in the wild come from a site without human or other predators we are not sure exactly why chimpanzee mortality rates are so high. It is likely that, during the past several million years, chimpanzee populations have faced significant predation from carnivores and hominids. (If it were otherwise, they should senesce at slower rates.) One might speculate that predation rates became lower among hominids than chimpanzees as soon as hominids began to forage socially, especially given the fact that female chimpanzees spend much of their time in solitary foraging. It is also possible that predation on hominids further decreased when they began to use weapons. Male provisioning of females and adolescents seems closely tied to the transition of hominids from frugivores to carnivores and omnivores. Thus, social foraging, the use of weapons, and male provisioning may have been key points in hominid evolution that led to the currently observed divergence between the life-histories of humans and apes. If these points can be defined in the archeological record, it may be possible to test life-history predictions using fossil hominid remains.

A GENERAL MODEL OF LIFE-HISTORY

A small but growing consensus is beginning to emerge among life-history theorists that extrinsic adult mortality rates may be the most important initial variable in limiting the feasible set of life-history parameters that maximize the fitness of an organism.^{78,79} Extrinsic adult mortality rates are the result of predation, environmental hazards, and other factors not related to the reproductive effort of the organism. Within stationary populations and among organisms for which reproductive value is a function of body size, the adult mortality rate and the growth law will allow prediction of optimal age and size at maturity. This, in combination with rates of energy harvest and expenditure as functions of body size, will predict optimal reproductive effort. The offspring fitness parental investment

function can be used to predict the body size of offspring at birth and the fertility rates that maximize fitness with a given reproductive expenditure. In populations that are not growing, birth and death rates must balance. Consequently, juvenile mortality rates are determined by the difference between fertility and the adult mortality function. Thus, the adult mortality hazard, in combination with growth rates and a series of functions that relate investment patterns to fitness, can, in theory, be used to predict most life-history parameters for any organism. This is shown schematically as a relatively simple causal chain in Figure 1.

If we drastically simplify life-history for organisms having a reproductive value at maturity that is proportional to their body size at maturity, we need only investigate the growth law and the optimal parental investment patterns to arrive at a rough prediction of age-specific fertility and mortality schedules from the adult mortality function. The expected fitness of offspring as a function of parental investment, which should be influenced by a variety of socio-environmental factors, is difficult to predict theoretically. However, it can be measured through appropriate experimental manipulation of parental investment.

Not all major life-history issues have been resolved. First, the scheme in Figure 1 does not easily address the tradeoff between current reproduction and future survival. Indeed, according to the model I have described, energy allocated to reproductive effort is assumed to be determined by the difference between energy harvest rates and metabolic costs at the optimal adult body size. The model thus assumes that the estimation of optimal metabolic costs is straightforward. In reality however, metabolic costs are not fixed, but vary as a function of investment in maintenance and repair. Hence, the energy expended on current reproduction is excess energy that is available, *given a particular allowed senescence rate*. The optimal partitioning of excess energy into current reproduction, storage, somatic repair, or maintenance is therefore a complicated process, the outcome of which should be partially determined by the extrinsic adult mortality rate.²³

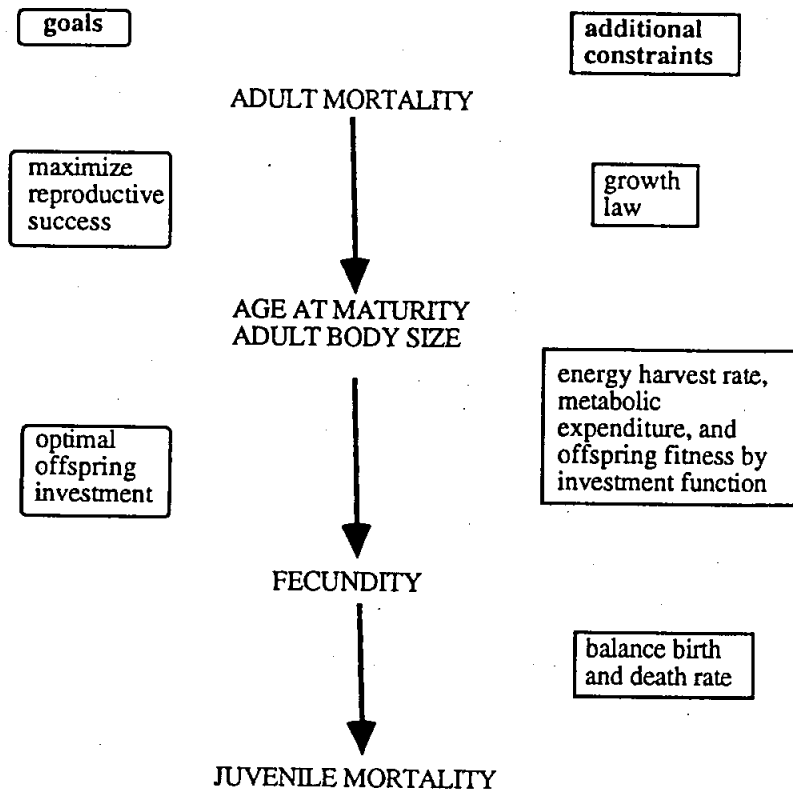


Figure 1. A causal chain of life history variables among female mammals (adapted from Harvey and Nee⁷⁹ after Charnov⁷⁸).

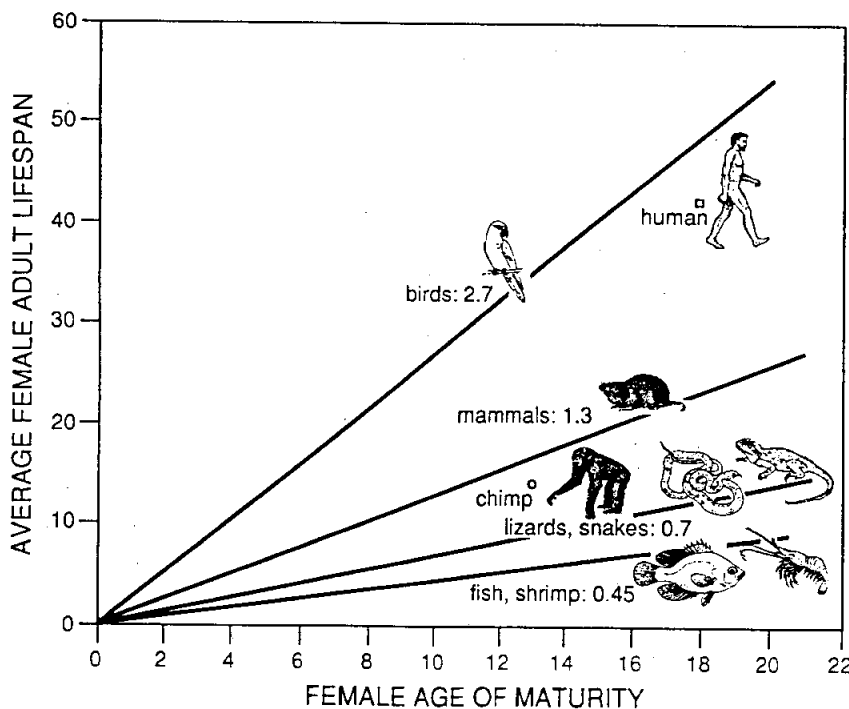


Figure 2. The relationship between age at maturity and adult lifespan for various phylogenetic groups (adapted from Charnov and Berrigan⁸⁰). Note that humans fit close to the bird curve whereas chimpanzees look like other mammals.

This scheme also does not fully explain phylogenetic regularities in life-history patterns, including a large number of strong correlations between life-history traits after the effects of body size are removed.⁷³ For example, a plot of $1/M$ (M is the adult mortality hazard) by α (the age at first reproduction) leads to constant slopes within birds, mammals, reptiles, and fish (Fig. 2), but the slope varies between these groups from 2.7 in birds to .45 in fish.^{80,81} The mammalian slope of 1.3 is predictable with a few simple assumptions, including a measure of the size-dependent growth exponent and the relative size at weaning versus adult body size.⁸² Nevertheless, other phylogenetic groups have very different $M \cdot \alpha$ values and must, therefore, be characterized by different functions relating reproductive value to age at maturity. It is important to note that humans do not fit anywhere near the predicted mammalian values for relationships between many life-history traits but, instead, look much more like birds (Fig. 2). This is true despite the fact that chimpanzees do fit close to the mammal curve. The bird-like life-history pattern of humans may be, in part, the result of extremely high levels of parental care, delayed juvenile independence (like birds), and exceptionally low adult mortality rates (also like birds). In any case, the investigation of these yet-to-be-discovered principles of symmetry in life-history traits⁸³ within and across phylogenetic groups is a major challenge for the future. Applying life-history to the demographic patterns of our species should allow us to explain such patterns rather than simply redescribe them with mathematical models.

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REFERENCES

- 1 Barret-Conner et al (1993) *Lancet*, Feb 9.
- 2 Hill K, Hurtado AM (1993) *Ache Life History: The Ecology and Demography of a Foraging*

- People. In press. Aldine de Gruyter: New York.
- 3 Goodall J (1986) *Chimpanzees of Gombe: Behavioral Patterns*, p 112. Cambridge MA: Harvard University Press.
 - 4 Bonner JT (1965) *Size and Cycle*. Princeton: Princeton University Press.
 - 5 Stearns S (1992) *The Evolution of Life Histories*. Oxford: Oxford University Press.
 - 6 Roff DA (1992) *The Evolution of Life Histories: Theory and Analyses*. New York: Chapman and Hall.
 - 7 Charnov EL (1993) *Life-History Invariants*. Oxford: Oxford University Press.
 - 8 Werner E (1988) Size, scaling, and the evolution of complex life cycles. In Ebenmann B and Persson L (eds), *Size Structured Populations*, pp 60-81. Berlin: Springer-Verlag.
 - 9 Williams GC (1966) *Adaptation and Natural Selection*. Princeton: Princeton University Press.
 - 10 Bell G, Koufopanou V (1986) The cost of reproduction. In Dawkins R, and Ridley M (eds), *Oxford Surveys in Evolutionary Biology*, pp. 83-131. Oxford: Oxford University Press.
 - 11 Fisher, RA (1930) *The Genetical Theory of Natural Selection*. Oxford: Clarendon Press.
 - 12 Leslie PH (1945) On the use of matrices in certain population mathematics. *Biometrika* 33:183-212.
 - 13 Lotka AJ (1907) Relation between birth rates and death rates. *Science* 26:21-33.
 - 14 Lack D (1947) The significance of clutch size. *Ibis* 89:302-352.
 - 15 Smith CC, Fretwell SD (1974) The optimal balance between size and number of offspring. *Am Naturalist* 108:499-506.
 - 16 Gustafsson L, Southerland WJ (1988) The costs of reproduction in the collared flycatcher *Ficedula albicollis*. *Nature* 335:813-815.
 - 17 Rogers A (1992) Evolution of Time Preference by Natural Selection, submitted to *Am Econ Rev*.
 - 18 Williams GC, Nesse R (1991) The dawn of Darwinian medicine. *Quart Rev of Biol* 66:1-22.
 - 19 Rose MR (1991) *The Evolution of Senescence*. Oxford: Oxford University Press.
 - 20 Austed SN (1992) Comparative aging and life histories. *Aging*, in press.
 - 21 Williams GC (1957) Pleiotropy, natural selection and the evolution of senescence. *Evolution* 11:398-411.
 - 22 Charlesworth B (1980) Evolution in age structured populations. Cambridge: Cambridge University Press.
 - 23 Kirkwood JK, Rose MR (1991) Evolution of senescence: Late survival sacrificed for reproduction. In Harvey P, Partridge L, Southwood (eds), *The Evolution of Reproductive Strategies*, pp 15-24. Cambridge: Cambridge University Press.
 - 24 Austed SN (1992) Retarded aging rate in an insular population of opossums. *J Zool*, in press.
 - 25 Rose MR (1984) Laboratory evolution of postponed senescence in *Drosophila melanogaster*. *Evolution* 38:1004-1010.
 - 26 Luckinbill L, Clare N, Krell W, Cirocco W, and Richards P (1987) Estimating the number of genetic elements that defer senescence in *Drosophila*. *Evol Ecol* 1:37-46.
 - 27 Fowler K, Partridge L (1989) A cost of mating in female fruitflies. *Nature* 338:760-761.
 - 28 Maynard-Smith J (1958) The effects of temperature and egg laying on the longevity of *Drosophila subobscura*. *J Exp Biol* 35:832-842.
 - 29 Bronson RT (1981) Age at death of necropsied intact and neutered cats. *Am J Vet Res* 42:1606-1608.
 - 30 Hamilton JB (1965) Relationship of castration, spaying, and sex to survival and duration of life in domestic cats. *J Gerontol* 20:96-104.
 - 31 Hamilton JB, Mestler GE (1969) Mortality and survival: A comparison of eunuchs with intact men and women in a mentally retarded population. *J Gerontol* 24:395-411.
 - 32 Stearns S, Crandall RE (1981) Quantitative predictions of delayed maturity. *Evolution* 35:455-463.
 - 33 Roff DA (1981) On being the right size. *Am Naturalist* 118:405-22.
 - 34 Roff DA (1984) The evolution of life history parameters in teleosts. *Can J Fisheries Aquatic Sci* 41:984-1000.
 - 35 Charnov EL (1989) Natural selection on the age of maturity in shrimp. *Evol Ecol* 3:236-239.
 - 36 Kozlowski J (1992) Optimal allocation of resources to growth and reproduction: Implications for age and size at maturity. *Trends Evol Ecol* 7:15-19.
 - 37 Taylor B, Gabriel W (1992) To grow or not to grow: Optimal resource allocation for *Daphnia*. *Am Naturalist* 139:248-266.
 - 38 Boyce MS (1981) Beaver life-history responses to exploitation. *J Appl Ecol* 18:749-753.
 - 39 Minchella DJ, Loverde PT (1981) A cost of increased early reproductive effort in the snail *Biomphalaria glabrata*. *Am Naturalist* 118:876-881.
 - 40 Skelly DK, Werner EE (1990) Behavioral and life-history responses of larval American toads to an odonate predator. *Ecol* 7:2313-2322.
 - 41 Crowl TA, Covich AP (1990) Predator-induced life history shifts in a freshwater snail. *Science* 247:949-951.
 - 42 Belsky J, Steinberg L, Draper P (1991) Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Dev* 62:647-670.
 - 43 Geronimus A (1987) On teenage childbearing and neonatal mortality in the United States. *Pop Dev Rev* 13:245-279.
 - 44 Stearns S, Koella J (1986) The evolution of phenotypic plasticity in life history traits: Predictions for norms of reaction for age- and size-at-maturity. *Evolution* 40:893-913.
 - 45 Godfray HC, Parker GA (1991) Clutch size, fecundity and parent-offspring conflict. *Phil Trans R Soc Lond B* 332:67-79.
 - 46 Charnov EL, Krebs JR (1974) On clutch size and fitness. *Ibis* 116:217-219.
 - 47 Godfray HC, Ives JR (1988) Stochasticity in invertebrate clutch size models. *Theor Popul Biol* 33:79-101.
 - 48 Blurton Jones N (1986) Bushman birth spacing: A test for optimal interbirth intervals. *Ethol Sociobiol* 7:91-105.
 - 49 Borgerhoff Mulder M (1992) Reproductive Decisions. In Smith E, Winterhalder B (eds), *Evolutionary Ecology and Human Behavior*, pp. 339-374. Hawthorne, NY: Aldine De Gruyter.
 - 50 Howell N (1979) Demography of the Dobe !Kung. New York: Academic Press.
 - 51 Harpending H (1993) The ecological !Kung, in press.
 - 52 Blurton Jones N (1993) The ecological !Kung: A response, in press.
 - 53 Wolfers D, Scrimshaw S (1975) Child survival and intervals between pregnancies in Guayaquil, Ecuador. *Popul Studies* 29:479-495.
 - 54 Palloni A, Millman S (1986) Effect of interbirth-intervals and breastfeeding on infant mortality and early childhood mortality. *Popul Studies* 40:215-236.
 - 55 Pennington R, Harpending H (1992) The structure of an African pastoralist community: Demography, history, and ecology of the Ngamiland Herero. Cambridge: Oxford University Press.
 - 56 Hill K, Hurtado AM (1991) The evolution of reproductive senescence and menopause in human females. *Hum Nature* 2:315-350.
 - 57 Paveleka M, Fedigan LM (1991) Menopause: A comparative life history perspective. *Yearbk of Phys Anthropol* 34:13-38.
 - 58 Alexander RD (1974) The evolution of social behavior. *Ann Rev of Ecol System* 5:325-383.
 - 59 Gaulin S (1980) Sexual dimorphism in the human post-reproductive lifespan: Possible causes. *Hum Evol* 9:227-232.
 - 60 Hamilton WD (1966) The moulding of senescence by natural selection. *J Theor Biol* 12:12-45.
 - 61 Hawkes K, O'Connell JF, Blurton Jones N (1989) Hardworking Hadza Grandmothers. In Standen V and Foley R, (eds), *Comparative Socioecology: The Behavioral Ecology of Humans and Other Mammals*, pp 341-366. London: Basil Blackwell.
 - 62 Trivers R (1972) Parental investment and sexual selection. In Campbell B (ed), *Sexual Selection and the Descent of Man*, pp 136-179 Chicago: Aldine.
 - 63 Rogers A (1993) Why menopause. *Evol Ecol*, in press.
 - 64 Marsh H, Kasuya T (1986) Evidence for reproductive senescence in female cetaceans. *Rep Intl Whaling Comm, Special Issue* 8:57-74.
 - 65 Olesiuk PF, Bigg MA, Ellis GM (1990) Life history and population dynamics of resident killer whales (*Orcinus orca*) in the coastal waters of British Columbia and Washington State. *Rep Intl Whaling Comm, Special Issue* 12:209-243.
 - 66 Charnov EL (1982) *The Theory of Sex Allocation*. Princeton: Princeton University Press.
 - 67 Milinski M (1978) Kin selection and reproductive value. *Z Tierpsycho* 47:328-9.
 - 68 Daly M, Wilson M (1988) *Homicide*. New York: Aldine de Gruyter.
 - 69 Thornhill R, Thornhill N (1989) The evolution of psychological pain. In Behl R, Behl N (eds), *Sociobiology and the Social Sciences*. Houston, Texas: Tech University Press.
 - 70 de Catanzaro D (1991) Evolutionary limits to self preservation. *Ethol Sociobiol* 12:13-28.
 - 71 Smith BH (1991) Dental development and the evolution of life history in Hominidae. *Am J Phys Anthropol* 86:157-174.
 - 72 Smith BH (1992) Life history and the evolution of human maturation. *Evol Anthropol* 1:134-42.
 - 73 Promislow DEL, Harvey PH (1990) Living fast and dying young: A comparative analyses of life history variation among mammals. *J Zool* 220:417-437.
 - 74 Melancon T (1982) Marriage and reproduction among the Yanomamo Indians of Venezuela. Unpublished Ph.D. thesis, Pennsylvania State University.
 - 75 Early J, Peters J (1990) *The Population Dynamics of the Mucajai Yanomamo*. New York: Academic Press.
 - 76 Gavan JA (1953) Growth and development of the chimpanzee: A longitudinal and comparative study. *Hum Biol* 25:93-143.
 - 77 Kirkwood JK (1985) Patterns of growth in primates. *J Zool Lond* 205:123-136.
 - 78 Charnov EL (1991) Evolution of life history variation among female mammals. *Proc Nat Acad Sci* 88:1134-1137.
 - 79 Harvey P, Nee S (1991) How to live like a mammal. *Nature* 350:23-24.
 - 80 Charnov EL, Berrigan D (1990) Dimensionless numbers and life history evolution: Age at maturity vs the adult lifespan. *Evol Ecol* 5:63-68.
 - 81 Charnov EL, Berrigan D (1991) Dimensionless numbers and the assembly rules for life histories. Harvey P, Partridge L, Southwood (eds) *The Evolution of Reproductive Strategies*, pp 41-48. Cambridge: Cambridge University Press.
 - 82 Charnov EL (1990) On the evolution of age of maturity and the adult lifespan. *J Evol Biol* 3:139-144.
 - 83 Charnov EL (1991) Pure numbers, invariants and symmetry in the evolution of life histories. *Evol Ecol* 5:339-342.