Life History Theory and Human Development

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INTRODUCTION

The field of life history evolution studies how the entire life cycle from conception to death is designed by natural selection to ensure reproductive success despite problems posed by the environment in the forms of mortality and scarce resources. The design occurs within a framework of constraints and trade-offs shaped by past evolution working on the materials out of which organisms are built and the developmental and physiological mechanisms organisms have inherited from ancestors.

The field focuses on these traits: age and size at maturity, number and size of offspring, investment in offspring, sex-specific growth and mortality rates of offspring, interval between births, number of births per lifetime, length of the reproductive portion of the lifetime, and length and function of the postreproductive period, if any. Humans differ from their primate relatives in several ways: They have slow physical development during which their brains grow and mental capacities are gradually acquired, hence an extended childhood prior to the juvenile period shared with other mammals; they have relatively short intervals between births, hence a reproductive rate higher than that of their closest relatives of similar size; although they usually bear one offspring, they can bear twins; females have a postreproductive period—menopause; and they have a relatively long life span. In the following section, we discuss how natural selection shaped these features of the human life cycle as well as others.

Why should an evolutionary psychologist care about life history evolution? Because this is the framework within which our mental processes develop, mature, support our behavior, and then senesce and diminish. Developmental state at birth and length of childhood combine with learning
from particular environmental experiences to determine brain capacity and content at adulthood. Our stage in life influences the costs and benefits of many serious decisions, including when to mate, with whom to mate, and how much risk to take in producing offspring. Every generation must learn a great deal to live a successful life; life history evolution has given humans 15 to 20 years in which to do that. Every individual must attempt to produce offspring that survive to produce grandchildren; life history evolution has given humans about 25 years in which to do that. And the evolution of senescence, in the human case, has limited the fully rational period of our life span to about 60 years; we do not have any longer than that in which to acquire and exercise understanding and wisdom. Thus, life history evolution created the framework within which psychology is expressed and by which psychology is constrained.

THE COMPARATIVE EVIDENCE: HUMANS AMONG PRIMATES

Compared to their closest relatives, the bonobos, chimpanzees, and gorillas, humans are average in some life history traits and unusual in others (Table 3.1). We are about the same size as chimpanzees and bonobos, much smaller than gorillas, and about as sexually dimorphic as bonobos, less so than chimpanzees and gorillas. Our gestation length is slightly longer than that of bonobos and gorillas and significantly longer than that of chimpanzees. Our offspring are nearly twice as heavy at birth as bonobo and chimpanzee offspring and one and one-half times heavier than those of gorillas, despite our lighter body weight. Although our offspring develop more slowly than those of other apes do, we wean them 2 years earlier than do bonobos, chimpanzees, and gorillas. The length of our estrus cycle is the same as that of gorillas and a bit shorter than that of bonobos and chimpanzees, and the age at which human females first give birth is nearly twice that of these three close relatives. Our interbirth intervals are one and one-half years shorter than those of chimpanzees and bonobos, and half a year shorter than those of gorillas. Our average maximum life span is 20 years longer than bonobos, 25 years longer than chimpanzees, and 30 years longer than that of gorillas.

Table 3.1 Comparison of the Life Histories of Humans and Their Closest Relatives

<table>
<thead>
<tr>
<th></th>
<th>Humans Homo sapiens</th>
<th>Bonobos Pongo pygmaeus</th>
<th>Chimpanzees Pan troglodytes</th>
<th>Gorillas Gorilla gorilla</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female weight (kg)</td>
<td>40.1</td>
<td>37.0</td>
<td>31.1</td>
<td>93.0</td>
</tr>
<tr>
<td>Male weight (kg)</td>
<td>47.9</td>
<td>42.5</td>
<td>41.6</td>
<td>160.0</td>
</tr>
<tr>
<td>Ratio male/female wt</td>
<td>1.19</td>
<td>1.15</td>
<td>1.34</td>
<td>1.72</td>
</tr>
<tr>
<td>Gestation length (d)</td>
<td>267</td>
<td>260</td>
<td>228</td>
<td>256</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>3.30</td>
<td>1.73</td>
<td>1.76</td>
<td>2.11</td>
</tr>
<tr>
<td>Age at weaning (y)</td>
<td>2.0</td>
<td>3.9</td>
<td>4.0</td>
<td>4.3</td>
</tr>
<tr>
<td>Estrus cycle length (d)</td>
<td>28</td>
<td>30</td>
<td>36</td>
<td>28</td>
</tr>
<tr>
<td>Female age at first breeding (y)</td>
<td>19.3</td>
<td>10.7</td>
<td>11.5</td>
<td>9.9</td>
</tr>
<tr>
<td>Mean age of reproduction</td>
<td>28.2</td>
<td>—</td>
<td>22.4</td>
<td>—</td>
</tr>
<tr>
<td>Average maximum life span (y)</td>
<td>70</td>
<td>50</td>
<td>45</td>
<td>39</td>
</tr>
<tr>
<td>Rate of increase of senescent mortality with age</td>
<td>0.095</td>
<td>—</td>
<td>0.147</td>
<td>—</td>
</tr>
<tr>
<td>Interbirth interval (y)</td>
<td>3.5</td>
<td>4.8</td>
<td>5.0</td>
<td>4.0</td>
</tr>
</tbody>
</table>

Much intraspecific variation (e.g., see Ruff, 2002) is buried in the averages reported in Table 3.1, but this much is clear: Humans give birth to larger, less developed offspring that grow and mature more slowly (Aiello & Wells, 2002) and need parental care for a longer period of time than do the offspring of other apes, but we manage to wean them earlier and to give birth at shorter intervals than do all three of our closest relatives. We mature a decade later and live 2 to 3 decades longer, and our females therefore have a longer period of postreproductive life than they do. We do so because we experience lower adult mortality rates, especially after the age of 50, when the mortality rates of chimpanzees in particular rise dramatically (Gage, 1998).

**HOW TO CONSTRUCT AN EVOLUTIONARY EXPLANATION OF A LIFE HISTORY**

The study of evolution is divided into two subdisciplines, microevolution and macroevolution. Microevolution deals with short-term evolutionary dynamics occurring within populations and species, and macroevolution deals with deep time, broad relationships, and big patterns. Microevolution thus occurs within a framework of constraints created by macroevolution.

*The Historical Explanations of Macroevolution*

The type of explanation provided by macroevolution is historical: Things are so because they had a particular history whose consequences have been inherited. To understand that history, evolutionary biologists can use one or both of two approaches—paleontology and the comparative method. Because life histories do not fossilize, those taking a macroevolutionary approach to life histories have concentrated on the comparative method. To use that method, one must reliably reconstruct the phylogenetic tree expressing the relationships of the species of interest with their closest relatives. In recent decades, phylogenetic reconstruction has been made more rigorous and reliable by improvements to logic and to data collection. In combination, those methods applied to such data yield the most reliable hypotheses of relationships currently available. Given a phylogenetic tree, one can then use comparative methods to infer some of the sequence of events that resulted in the observed life history. By mapping the states of the traits in each species onto the tips of the branches of the tree, one can infer ancestral states and search for correlated changes among traits (e.g., Felsenstein, 1985; Pagel, 1994).

*The Selection Explanations of Microevolution*

Two processes in microevolution affect the current state of the life history of a species or population: natural selection and genetic drift. Drift results in random differences among lineages; it can be used as the null hypothesis in testing other explanations. Deviations from random expectation are analyzed with approaches that assume that natural selection has designed the phenotype to improve reproductive success; these include optimization (Roff, 2001; Stearns, 1992), game theory, and adaptive dynamics, often incorporating risk reduction (Stearns, 2000) and conflicts among relatives mediated by kin selection (Griffin & West, 2002). The object explained is sometimes a single phenotypic state and sometimes, more powerfully, a reaction norm that expresses the range of phenotypes that a single genotype can express when confronted with a range of environments. For example, one can predict how age and size at maturity should evolve to change across a range of growth conditions in a specific manner described as the norm of reaction to that change in conditions (Berrigan & Koella, 1994; Kawecki & Stearns, 1993; Stearns & Koella, 1986).

Because the strength of natural selection is limited by the amount of variation in reproductive success in a population, selection explanations are couched in terms of the means and variances of mortality rates and birth rates acting on populations whose responses are constrained by trade-offs. Trade-offs occur when an evolutionary change in one trait that increases fitness is linked...
through physiological or developmental mechanisms to changes in other traits that decrease fitness (Stearns, 1989; van Noordwijk & de Jong, 1986). Often added insight is gained by analyzing the proximate mechanisms of development and physiology that cause trade-offs and, therefore, constrain the response to selection (Drent & Daan, 1980).

**The Mechanistic Explanations of Development and Physiology**

Proximate mechanisms are important because they determine the set of possible responses, a set that may include responses unanticipated by any theory. For life history evolution, proximate mechanisms are particularly important when they determine the possible rates and types of resources acquired and how those resources are allocated to growth, reproduction, and maintenance: when they mediate trade-offs among those functions. Our phylogenetic history has given us particular proximate mechanisms. We are mammals with internal fertilization and a 9-month pregnancy, giving birth to helpless young that require intense parental care for many years before they have good chances of surviving to reproduce. We are subject to infectious disease, which we counter with an adaptive but costly immune system. Our females store fat prior to and during pregnancy to finance the costs of pregnancy and nursing. Because we have determinate growth and stop increasing in height at maturation, our allocations switch at that point from growth to reproduction, always also involving fat storage, disease resistance, and other types of maintenance. Hormones produced in the brain and in other endocrine glands control the mechanisms that mediate those allocations. They determine how genetic variation will be expressed in the population and how trade-offs are realized in the whole organisms that we can observe and measure.

**Evolutionary Explanations of the Major Life History Traits**

Four types of approaches have been taken in explaining the evolution of life history traits: (a) optimization (including invariants), (b) adaptive dynamics (including its precursor, game theory), (c) genetic transmission of quantitative traits, and (d) comparative methods based on phylogenetics. Each makes its own assumptions in representing the process and history of evolution. The data used to test these explanations are estimated with the methods of demography.

Optimality models assume that natural selection has shaped life history traits within a framework of trade-offs to maximize some fitness measure, usually reproductive success per time unit or per lifetime. They ignore genetic details, assume that the optimal state is somehow attainable, look for equilibrium solutions, neglect dynamics (Stearns, 1992), and thus, assume that evolution has gone on in large populations that encounter stable ecological conditions for long periods. In such populations, their predictions are consistent with those of quantitative genetics (Charlesworth, 1990). Authors using optimality models to understand human life histories include Hill and Hurtado (1996), Voland (1998), Hill and Kaplan (1999), Mace (2000), Strassman and Gillespie (2002), and Ellison (2003a).

Life history invariants are the ratios (usually log-log) of life history traits that broadly characterize clades, such as birds, fish, or mammals (Charnov, 1993; e.g., the ratio of the log of age at maturity to the log of life span). The derivation of invariants usually assumes stationary populations in which lifetime reproductive success measures fitness. They are useful in posing both a puzzle—why these values and why the differences among groups?—and an expectation—why does this species deviate from the value for the clade?

Adaptive dynamics compares the performance of alternative phenotypes competing over many generations. The evolving population is modeled by studying the invasion of alternative strategies. The issues include the following: What strategies are stable against invasion by all conceivable alternatives? What strategies are not only stable but also actually attainable? How does the dynamic itself change selection? The method, particularly appropriate when frequency- and density-dependent
effects are strong (e.g., De Mazancourt & Dieckmann, 2004; Dercole, Ferriere, & Rinaldi, 2002), predicts a diversity of evolutionary outcomes not perceived by other approaches.

Evolutionary quantitative genetics deals with traits that vary continuously, such as weights at birth and reproductive investments, in contrast to traits that fall clearly into distinct classes. It makes a plausible assumption, convenient for statistical analysis, about the genetics of traits influenced by many genes. To infer the genetic and environmental contributions to variation, it measures the phenotype, the final product of the assumed causes, not the mechanisms—the physiology and development—that produce the phenotype. Its fundamental measures are the heritabilities of and genetic covariances among traits and the gradient of selection pressures operating on traits, all estimated empirically. By multiplying the heritabilities and genetic covariances by the selection gradient, one can predict evolutionary change from one generation to the next (Roff, 1997, 2001). Quantitative genetics is a useful framework in which to examine generation-to-generation microevolutionary change (e.g., Kääriä, Jokela, Helle, & Kojola, 1996).

Phylogenetic methods are described previously. As an example of the insights they can yield, Holden and Mace (1999) used a cultural phylogeny based on language to understand how the division of labor between males and females affects sexual dimorphism in human body size. They found that in cultures where women do more work to acquire food, there is less difference in stature between the sexes.

We now step through the human life history from gamete production and conception to aging and death.

CRITICAL EARLY EVENTS INVOLVING GAMETES AND EMBRYOS

Selection Arenas, Oocytic Atresia, and Menopause

A selection arena is a process based on the principle of natural selection that occurs inside an entity, such as a reproductive female, that has been designed by natural selection at a higher level. Oocytic atresia is a selection arena operating in humans and other mammals. Atresia means degeneration or loss. All oocytes are lost by menopause in humans (Finch & Sapolsky, 1999). The process starts in the third month of pregnancy, when about 7 million oocytes are present in the newly formed ovaries. By the time the child is born, that number has fallen to about a million, by menarche to less than 1,000, and by menopause to near zero. Such dramatic destruction of a key resource demands an explanation.

The explanation appears to be that atresia eliminates oocytes that contain genetic defects in either the nuclear or the mitochondrial genome. The rate of mutation in the nuclear genome is between 0.1 and 100 mutations in each genome per generation. Mitochondria are a special issue because they usually reproduce asexually, pass regularly through population bottlenecks, and therefore cannot avoid accumulating deleterious mutations (Muller, 1964). This problem is solved if only a small number of mitochondria are introduced into each of many oocytes and if the oocytes with defective mitochondria advertise that fact in their biochemical profile, giving maternal tissue capable of action a signal used to decide from which oocytes nourishment should be withdrawn (Jansen & de Boer, 1998; Krakauer & Mira, 1999). There is some support for this idea. The probability of oocyte destruction is affected by the state of the mitochondrial genome in rodents (Perez, Trbovich, Gosden, & Tilly, 2000), and the number of mitochondria in a primordial oocyte is small, less than 10 (Jansen & de Boer, 1998). The nature of the signal that elicits destruction is yet unknown.

Sperm Selection

Human males have a higher mutation rate than human females because there are many more cell divisions between zygote and sperm than between zygote and egg (Crow, 1993). That children with new dominant mutations tend to have older fathers (Haldane, 1947) suggests that older males have
a higher proportion of genetically defective sperm than younger males do. To the extent that genetic defects erode the performance of sperm in finding oocytes in a female reproductive tract designed to detect and eliminate defective sperm, sperm also pass through a selection arena that improves zygote quality. While sperm selection is well documented in other species, selection against genetically defective sperm is not yet well demonstrated in humans. Doctors working in reproductive medicine assume that it occurs and are concerned about the potential consequences of bypassing it when they perform in vitro fertilizations.

Spontaneous Abortion

Spontaneous abortions occur more frequently than is often assumed. The rate is difficult to determine precisely, for most aborted zygotes and embryos pass out with the next menses. Estimates of the proportion of pregnancies that end in early, unrecognized abortion range from 30% to 75% (Haig, 1998). Clinically recognized pregnancies miscarry in 10–20% of cases, most of which have chromosomal abnormalities. Twins are another situation in which spontaneous abortion is frequent: Up to 71% of gestations diagnosed as twins were singletons when delivered (Levi, 1976). This suggests that spontaneous abortion functions both to eliminate defective embryos and to halve the reproductive investment implied by twins.

Evidence for one important reason for recurrent spontaneous abortions comes from Ober, Elias, Kostyu, and Hauck’s (1992) work on Hutterite communities in South Dakota. The Hutterites, who moved to North America from Switzerland in the 19th century, are a small community that has become relatively inbred. Some Hutterite women suffer from recurrent spontaneous abortions. Ober et al. discovered that women whose husbands had similar combinations of immune genes were more likely to suffer spontaneous abortions than women who had married men with combinations different from their own. The vertebrate immune system relies on diversity in the immune genes to generate through recombination the broad spectrum of antibodies needed to fight novel infections. Offspring unable to generate that broad spectrum would have been likely to die young of infectious diseases in premodern societies. Ober’s work suggests that the female human reproductive tract evolved to detect and discard immunologically deficient embryos early in development before they cost very much in either time or energy, giving the mother another chance to conceive a healthy embryo, a chance that would be better with a different father.

The screening system that eliminates genetically deficient embryos probably suffers from the general decline in performance that accompanies aging. If it does, that would explain the increased incidence of birth defects in children born to older women, notably Down’s syndrome (Forbes, 1997). At the other end of life, the strikingly higher rate of spontaneous abortions in women who reach menarche at age 12 or less, as compared with those who do so at age 14 or more (Liestøl, 1980), might be the result of less previous screening through oocytic atresia in the younger group, which would allow more defective genomes to be conceived and thus elicit compensatory screening through spontaneous abortion at the zygote stage.

Conflicts over Intrauterine Growth

Once a zygote has survived the gauntlets of atresia and spontaneous abortion and has settled into the uterus to grow, it does not relax into a supportive environment, for its interest in nutrition can exceed what its mother has been selected to transfer (Haig, 1993; Trivers, 1974). The fetus can win part of this conflict by remodeling the endometrium to gain direct access to the maternal blood supply with vessels that do not constrict, rendering the mother incapable of limiting nutrition to the fetus without also limiting it to her own tissues, and by turning the placenta into an endocrine gland with direct access to maternal blood. Placental hormones manipulate maternal physiology for fetal benefit and are countered by increased maternal insulin production; if this countermeasure is not sufficient, gestational diabetes results in the mother. If the fetus is poorly nourished, it may increase
its blood supply by increasing the resistance of its mother’s peripheral circulation, resulting in pre-eclampsia (dangerously high maternal blood pressure).

Conflicts also exist within fetal cells among genes that are only expressed when derived from the mother and genes that are expressed only when derived from the father. Such differential gene expression is programmed in the germ lines of the parents through selective methylation, or imprinting, of specific genes; imprinted genes are silenced during the development of the embryo. Most genes imprinted in humans affect embryonic growth through their control of insulin-like grown factors (IGFs). The genes that are turned off in the paternal germ line are those that would restrict fetal growth; those turned off in the maternal germ line are those that would accelerate fetal growth. Thus, the father’s imprinting acts to remove more resources from the mother than she has been selected to provide, and the mother’s imprinting acts to counter the paternal effect. The effects of imprinting are revealed by mutations in humans and genetic manipulations in mice that prevent imprinting from occurring in one sex or the other. When the father’s genes are not imprinted, the maternal genes have the upper hand, and the embryo is about 10% lighter at birth. When the mother’s genes are not imprinted, paternal genes have the upper hand, and the embryo is about 10% heavier at birth (Haig, 1992).

The duration of pregnancy and the weight of the child at birth are thus determined both by the mutual interests of mother, father, and child in the health of the child and by subtle conflicts between child and mother and mother and father over the level of investment actually given.

Size at Birth

Human birth weight correlates well with human age at maturity (Harvey & Clutton Brock, 1985), but as age at maturity is rather late in humans compared with other primates, babies are heavier than would be expected based on the weights of their parents alone. The extra weight is due to larger brain size and more adipose tissue than in other primate offspring. In fact, brain size should be even bigger at birth when compared to the brain size of adult humans (Leakey, 1994), but human pelvises, which have to remain narrow for our upright gait, limit the size of a baby’s head. Human babies are thus particularly altricial, with brain development continuing outside the womb, leaving more scope for environmental interactions than in other primates. Why human babies are so much fatter than their primate relatives is not yet clear: Arguments range from “pretty baby” runaway selection processes (with mothers favoring their plumper children) to the fact that humans may be the only primates with enough surplus food (thanks to cooperative feeding) to produce fat babies (Haig, 1998; Pond, 1997).

Human birth length ranges from 40 to 57 cm and birth weight from 1 to 4.5 kg (Tanner, 1990), with extreme values for viable infants having increased with advances in modern medicine. Average birth weight is consistently 0.5 kg lower than the weight that would produce optimal survival of newborn babies (Karn & Penrose, 1952). Blurton Jones (1978) suggested that this is due to parent-offspring conflict. Because the range of birth weights at which babies can survive is broader under modern medical care than in the past, stabilizing selection for optimal birth weights is now relaxed (Ulizzi & Manzotti, 1988; Ulizzi & Terrenato, 1992).

The 50th percentile healthy baby in the United States measures 51 cm and weighs 3.2 kg. Birth weight averages range from 2.4 kg in poor regions to 3.6 kg in affluent ones (Eveleth & Tanner, 1976). Low birth weight can have several causes, each with differing consequences: Low birth weight due to mild prematurity with adequate weight for age may have few long-term consequences if adequate care is provided. However, stunted babies, who are small for their gestational age, usually suffer more long-term consequences, which vary depending on shape. Light babies may either be normal and short or long and thin, depending on the timing of food restriction during pregnancy. Overall, low birth weight is associated with increased infant mortality and morbidity and adversely affects long-term physical growth, immune response, and mental development (Hokkenkoelaga et al., 1995; McCormick, 1997). It can also affect the timing of maturation (Adair, 2001): Long,
thin baby girls achieve menarche 6 months earlier than do normal short ones. But weight is not the whole story: For various reasons, and despite lower birth weights, females and firstborns suffer less mortality than males and late born offspring (Cogswell & Yip, 1995).

**GROWTH AND MATURATION**

**Human Growth Patterns**

Population means for height range from 136 to 152 cm for adult females and from 164 to 183 cm for adult males (Eveleth & Tanner, 1976). Population means for weight range from 38 kg to 72 kg for females and from 46 kg to 76 kg for males. Secular trends have been influencing these means for decades, with humans in developed countries gaining 1–2 cm in height per decade. In addition, worldwide obesity is increasing and accelerating. In the United States in particular, height has reached a plateau but weight still is growing (Bogin, 1999; Harrison, Tanner, Pilbeam, & Baker, 1977; Malina, Zavaleta, & Little, 1987; Tanner, 1990), while in Mexico and India obesity and diabetes are spreading at a striking rate. The energy in food places stronger restrictions on human growth than does the limitation of specific nutrients (Calow, 1979). And stress interacts with available energy to affect growth and age at menarche in girls, including stress caused by father absence: Girls whose fathers are absent mature earlier, for reasons still under debate (cf. Hulanicka, Gronkiewicz, & Koniarek, 2001; Kanazawa, 2001; Macintyre, 1992; Maclure, Travis, Willett, & MacMahon, 1991; Quinlan, 2003).

Following years studying children whose catch-up growth rates vary to compensate for periods of food restriction or other stress, Tanner (1963) portrayed human growth as target seeking and self-stabilizing. But as Bogin (1999) points out, such a descriptive approach does nothing to explain why human growth has this pattern, nor how it differs from our primate relatives. Human growth can be divided into five periods: (a) **infancy** (from birth to weaning, traditionally around age 2), (b) **childhood** (from weaning to full brain growth, around age 7—Bogin argues this is a uniquely human stage), (c) the **juvenile** period (from age 7 to the beginning of puberty, around age 10 in girls and age 12 in boys), (d) **adolescence** (from the beginning of puberty to full sexual maturity around age 14 in girls and 16 in boys—Bogin argues that the length of adolescence is special in humans), and (e) **adulthood**, when growth has normally stopped, although in cases of stunting catch-up growth can be observed until age 25 in both sexes (but not if the young women are pregnant).

Bogin (1999) sees childhood as a feeding adaptation: Human babies can be weaned earlier than expected, freeing the mother to get pregnant again, because child food can be provided by group members other than the mother (grandmother, father, older sister, or neighbor depending on social arrangements). This unique adaptation enables humans to rear large costly offspring much more rapidly than can our primate relatives. Children still depend on adult care during this period, being unable to survive on their own or to gather their own food to any significant degree. In contrast, juveniles have some ability to fend for themselves and might survive if orphaned or abandoned, despite their lack of adult size, skills, and experience. Juveniles learn to be independent while benefitting from group support without the stress of competition for adult status and reproductive opportunities. Adolescence follows and is especially marked in humans. Other species put on weight at adolescence, but the human height growth spurt is unique in magnitude (Watts, 1986). It appears advantageous to shorten the transition from protected childhood to exposed adulthood, possibly to avoid sexual competition before one is ready to mate. Because girls usually appear to be adult and even start menses before they are completely fertile, they can start to engage in adult roles without immediately becoming pregnant. In males, the opposite is true: Boys start to produce functional sperm before growing tall and exhibiting adult male characteristics. Some have speculated that this could be to enable covert paternities while avoiding aggression from competing adult males. Both patterns reduce the initial costs of adulthood.
Growth yields adult women smaller than it yields adult men—human sexual dimorphism. Why should women be smaller than men? One reason might be that there is greater sexual dimorphism in polygynous species than there is in monogamous species, mostly due to increased male-male competition in a polygynous environment. Compared to their primate relatives, human males are only slightly larger than human females, implying mild polygyny (cf. Diamond, 1991; Trivers, 1985). Nettle (2002) confirmed in Britain that tall men generally had high reproductive success but found that both very short and very tall women had lower reproductive success: In that environment, selection favors a stable sex dimorphism. However, in a natural-fertility/natural-mortality environment, Sear, Allal, Mace, and McGregor (2004) found that tall Gambian women enjoyed a significant reproductive advantage via lower offspring mortality.

Women also differ from men in storing more fat, especially in the buttocks, thighs, and breasts. Men tend to be more muscular. This difference is thought to reflect women’s greater need for fat reserves for reproduction and men’s greater need for muscular tissue for competition. The exact patterning of tissue in the body, in particular the waist-to-hip ratio in women and the obviousness of their breasts, is probably the result of sexual selection (Bailey, 1982; Bailey & Katch, 1981; Bogin, 1999; Lieberman, 1982; Pond, 1997; Singh, 1993; Stini, 1969).

**Optimal Age and Size at Maturity**

Optimal age and size at maturity is determined by the trade-off between maturing early at a smaller size and later at a larger size. The advantages of early maturity are mainly short generation lengths, whose benefits are compounded across generations, and the security of having reproduced before random mortality may strike. The advantages of later maturity are increased size, knowledge, experience, and acquisition of goods or shelter, which may all contribute to more successful reproduction in the longer term, particularly to the survival and reproductive success of offspring.

The timing of puberty is hormonally controlled from the brain (Bogin, 1999; Cameron, 1991, 1996; Knobil, 1990), but hormone production is influenced by environmental cues (e.g., nutrition and paternal presence). Walker et al. (2006) review 22 hunter-gatherer life histories and provide average age and size at first birth for 16 populations. Reproductive maturity ranges from 16 to 20 years in females, with a mean of 19.2 years. Adult height ranges from 136 to 166 cm, with a mean of 149 cm. Males mature later at larger size.

The first formal models of optimal age and size at first birth focused on maternal weight gain: Fatter women are more fertile and later maturity allows more time to accumulate fat before starting a long cycle of pregnancies and breastfeeding. Stearns and Koella (1986) drew on historical data to illustrate this trade-off, and Hill and Hurtado (1996) developed a detailed population-level model for Aché hunter-gatherers on the same assumptions. Both models predicted optimal age at first birth accurately and were consistent with observations that puberty is completed earlier under better nutritional conditions and with increasing obesity (Herman-Giddens et al., 1997). In general, age at puberty is positively correlated with age at first reproduction (Udry, 1979).

The growing discrepancy between biological puberty, which arrives earlier and earlier as nutrition improves, and psychosocial maturation and socially accepted age at first birth, increasingly delayed in Western societies, affects the evolutionary psychology of adolescents, for their bodies and their cultures are sending them conflicting messages concerning appropriate behavior (Gluckman & Hanson, 2006).

**THE PATTERNING OF REPRODUCTIVE INVESTMENT AFTER PUBERTY**

Distinguishing the proximate and ultimate determinants of human reproductive rate and parental investment is helpful. Demographers are brought up on Bongaart’s (1980) proximate determinants of fertility: He argued that marriage patterns, postpartum subfecundity, contraceptive use, and
venereal disease explain most of the variation in birth rates among populations. An evolutionary perspective shifts the emphasis toward explaining both population level and individual differences. Evolutionary ecology and life history theory draw attention to factors affecting fertility not in Bongaart’s list by considering the ultimate causes of that variation. In particular evolutionary demographers have investigated how access to resources influences individual variation in marriage patterns (e.g., Josephson, 2002) and—given the unusual life history pattern of humans compared to other apes—how grandmother presence (e.g., Sear, Mace, & McGregor, 2000) and father absence (e.g., Waynforth, 2002) affect reproductive scheduling. In general one expects a combination of cultural, social, and economic conditions affecting marriage patterns, hormone levels mediating postpartum subfecundity (Bribiescas, 2001; Ellison, 1994), and conscious decisions about birth rate to explain how population and individual variation in fertility maximize reproductive success in given environments, while sexually transmitted infections may lead to maladaptive outcomes.

**Interbirth Intervals**

There is a trade-off between infant survival and mother’s fertility, for children compete for her investment. Several studies show that short birth intervals can endanger the life of the children that open and close that interval (Alam, 1995; Bøhler & Bergström, 1995; Hobcraft, McDonald, & Rutstein, 1985). It appears that large families also impose costs on 2- to 4-year-old children (LeGrand & Phillips, 1996). Mothers usually maximize their lifetime reproductive success by having more children than would maximize offspring survival, a central tenet of parent-offspring conflict (Trivers, 1985).

Blurton Jones (1986) showed that hunter-gatherer Kung mothers with shorter interbirth intervals experienced higher infant mortality, with the optimal balance between the birth interval and infant survival at around 4 year intervals. He argued that this interval, which is longer than the average in developed countries, results from hunter-gatherers having to carry both infant and food supply: More closely spaced offspring would be impossible to transport. It should be noted, however, that another forager group, the Aché, who also carry their young everywhere, manage a three-year interbirth interval (Hill & Hurtado, 1996). Some attribute the long Kung interbirth intervals to the prevalence of sexually transmitted infections in that population.

In a natural fertility population of farmers in Mali, Strassmann & Gillespie (2002) found that most mothers in fact achieved reproductive success far below the population maximum. The reason is that individuals vary in condition and experience: Some mothers are better than others at producing viable offspring, and mothers reproduce according to their individual optima.

**Single Versus Multiple Births**

Most human births are singleton, but enough births (around 4%) are twins for this to be more than a developmental accident. Twinning provides a clear example of the “supermum” effect. Mothers of twins experience higher lifetime reproductive success and longer reproductive spans in some environments but not others (Sear, Shanley, McGregor, & Mace, 2001). Some studies of natural fertility societies have shown that if both twins are girls the twins can be successful (Lummaa, Jokela, & Haukioja, 2001). Overall, there is much evidence that twinning may be costly for the twins themselves. In the Gambia, twin mortality is double that of singleton mortality, suggesting that the process is inefficient to the point of maladaptation (Sear et al., 2001). Twinning could be a by-product of polyovulation that allows high quality women to maintain short interbirth intervals; the down side is that twinning sometimes occurs and then infant mortality is high. Infanticide is more frequent in twin than in singleton births in societies like the Aché and may be a facultative option exercised in times of food stress (Hill & Hurtado, 1996).
**Sex Allocation**

There is rather mixed evidence that human mothers influence the sex ratio of their offspring at birth in direct response to body condition, as Trivers and Willard (1973) predicted for polygynous species. Many studies have failed to find any effect, but some studies have found local effects. For example, Ethiopian women with higher body mass indices were more likely to have sons after a drought year, suggesting that male fetuses were less likely to be carried to term in more food-stressed women (Gibson & Mace, 2003). However, humans are not seasonal breeders. We have the option of altering the birth interval in response to the state of nutrition or workload (e.g., Gibson & Mace, 2006; Jasienska & Ellison, 1998; Wood, 1994), and this may well be a more common response to environmental conditions than is the sex ratio manipulation seen in more seasonal breeders like red deer.

Far more common in humans is sex biasing of parental investment after the child is born. Parental investment in humans is not just about nutrition. Intergenerational transfers of resources, such as territory, skills, or wealth, are key to reproductive success in many social species, including humans. In wealth-inheriting societies, parents may have to show the color of their money, in the form of bride-price or dowry, to marry off their children. Bride-price is a payment from the groom or his family to the parents of the bride and is typically found in polygynous societies (Hartung, 1982), where males use resources to monopolize several females if they can afford to. Poorer males in such societies are unable to acquire mates. Dowry, where money is paid from the family of the bride to the newlyweds or their family, occurs when it is females that are in competition for mates (Gaulin & Boster, 1990). Such female-female competition is most likely to arise in societies with socially imposed monogamy. In contrast to polygynous societies, in which the benefits of wealth are likely to be diluted among many wives, in monogamous societies a woman who marries a wealthy man has sole access to his wealth for the benefit of her offspring alone, and hence, female-female competition for wealthy men becomes intense. The costs of bride-price and dowry influence parental reproductive scheduling. A father who already has sons does not want too many more in bride-price societies where the groom or his family provide the main costs of marriage and setting up home (Mace, 1996), whereas in dowry societies, female infants with a large number of elder sisters can be at increased risk of infanticide for the same reason (Das Gupta, 1987).

The economic framework of reproductive decisions is also important in Western societies, as exemplified in the following three studies. First, in babies born in Philadelphia over a 6-month period with parental investment measured by the amount of breast feeding and the length of the subsequent birth interval, families with incomes over $60,000 per year invested more heavily in sons than in daughters, whereas the reverse was true in families earning less than $10,000 per year (Gaulin & Robbins, 1991). Second, whereas contemporary Hungarian gypsies invest significantly more heavily in daughters than in sons, as measured by the duration of breast-feeding, the length of the subsequent birth interval, and the length of secondary education, the relatively wealthier native Hungarians exhibit the opposite pattern. The investment patterns closely matched the relative numbers of grandchildren gained through each sex of offspring (Bereczkei & Dunbar, 1997). Third, such reproductive decisions may change with the economic circumstances of the parents from one generation to the next. In six north German peasant communities in the mid-19th century, the preference for sons over daughters as measured by their respective mortality rates during the first year of life varied with economic circumstances. When populations were able to expand into virgin land, sons were preferred because they could acquire farms, but when populations were at saturation levels and there was little opportunity to acquire new land, daughters were preferred because they could still marry into higher socioeconomic classes. There appeared to be about a 30-year, or one generation, lag between the environmental stimulus and the corresponding behavioral response (Voland, Dunbar, Engel, & Stephan, 1997).

Biased investment by parents does not necessarily involve deliberate killing, although it is clear that sometimes it does. Often the children die from neglect, cryptic physical abuse, or the
consequences of psychological or economic discrimination. Whatever the mechanisms, it is striking that humans in both traditional and contemporary societies sometimes display precisely the kinds of differential investment in offspring of the two sexes that have been so creatively predicted and strikingly confirmed in other species.

**Bet-Hedging and Risk Minimization**

Humans reduce the risk of reproductive failure in several ways. Some females have the opportunity to mate several times; the resulting higher level of genetic variation among their half-sib offspring increases the probability that some of their offspring will resist disease and be more attractive to potential mates. Whether these theoretical benefits of multiple paternity outweigh the potential costs is not clear. An increase in offspring number is itself a method of spreading the risk of reproductive failure, an effect reflected in the royal reproductive strategy “an heir and a spare.” In The Gambia mothers are explicit about wanting many children in the hope that one will be lucky and successful (Allal, personal communication, 2006).

**Infanticide**

Deliberate infanticide by mothers may have been common in hunter-gatherer and other traditional populations. Child abandonment (tantamount to infanticide) appears to have reached epidemic proportions in some parts of historical Europe, particularly during urbanization or when natural mortality was declining, birth rates were high, and contraceptive practices were not well developed (Hrdy, 2000). In hunter-gatherer societies, female infanticide appears to be more common than male infanticide, possibly because males have additional value to families as hunters and warriors.

There is more female infanticide in the Inuit at more northerly latitudes (E. A. Smith & S. A. Smith, 1994): This group is highly reliant on food hunted by males, who must use dangerous hunting strategies, and polygyny is hardly possible for males, for the costs of supporting even one wife are great in this harsh environment (where mothers may be so constrained by the need to keep young children out of the cold that they may not be able to leave their houses for much of the year).

In other groups, such as the Aché, the fitness benefits, if any, of female-biased infanticide is not clear (Hill & Hurtado, 1996). However children who lost one of their parents were at high risk of infanticide—few Aché were prepared to pay the high costs of raising any child that is not their own. In farming societies, the chances of surviving orphanhood appear to be much higher (e.g., Mace & Sear, 2005; Pavard, Gagnon, Desjardins, & Heyer, 2005). But there is clear evidence of a small but measurable mortality risk associated with a mother’s remarriage to a new male in most societies in which this has been investigated, including contemporary settings such as Canada (Daly & Wilson, 1988). The adverse effect of human stepparents is certainly not equivalent to the normative infanticide practiced by incoming males in such species as langurs or lions. A recent study of accidental child deaths in Australia (Tooley, Karakis, Stokes, & Ozanne-Smith, 2006) found a significant increased mortality risk in stepparent households in cases where no foul play was suspected, suggesting that it is the distraction of a mother’s efforts from parental investment in existing children to investment in a new mate that is likely to be a major effect in the fitness costs experienced by stepchildren.

**Menopause**

The existence of several postreproductive decades in human females has triggered much debate about how such a long section of women’s lives could persist without immediate Darwinian benefits. Two questions focus this debate. First, is menopause unique in human females, or does it exist in other mammals, perhaps to a lesser extent? Second, have the existence and duration of menopause been the direct objects of selection, or are they the neutral or deleterious by-products of selection of some other trait? Menopause may also have originated as a by-product that then experienced mild positive selection.
Menopause around age 50 is universal in human populations (Wood, 1994), whereas in the wild it has been observed only in pilot whales and in captivity it has been reported only in few very old primate females (one bonobo, one pigtail macaque) both very near death (Austad, 1994; Pavelka & Fedigan, 1991). Although the captive animals showed similar hormonal profiles and oocyte depletion as do humans, their “menopause” was in step with their general senescence and not decades earlier as in women. Note that even with the relatively low life expectancies found in natural mortality populations, survival rates after cessation of childbearing are good, with many women reaching 60 or 70 years of age (Hill & Hurtado, 1991).

One proximate reason for the sudden cessation of reproduction is oocyte depletion: Menopause may be a by-product of atresia (Cresswell et al., 1997; Faddy & Gosden, 1996; Gosden et al., 1998). Under this view, selection is seen as having adjusted the stringency of the oocytic atresia filter in human ancestors to the level currently found in chimpanzees and bonobos, where females run out of oocytes at about the time they have their last offspring. Changes in other traits—in humans many of them social—then lead to improved survival late in life. Because selection pressures late in life are not strong, they cannot rapidly readjust the stringency of the atresia filter, whose advantage is strong and comes early in life. Both females and males live longer, but females have run out of oocytes. Menopause is then not a selected adaptation but a by-product of selection on the stringency of the atresia filter. A significant period of postreproductive survival would be expected in species in which there has been a relatively recent drop in late life mortality. In the end, weak selection for further reproduction will reduce the stringency of the atresia filter and lengthen reproductive life span. This atresia by-product hypothesis for menopause is certainly not yet established, but it does have one attractive feature not shared by the grandmother hypothesis: It explains the striking variation in age of onset of menopause (45–54 years; cf. Faddy & Gosden, 1996, figure 2) as the by-product of slight random variations in the long atresia process. If menopause were a primary adaptation rather than a by-product of something else, it should be much less variable in age of onset than it in fact is. This explanation neatly connects processes at the beginning and the end of life.

While the atresia hypothesis is good at explaining why female reproduction must decline in an abrupt manner when compared with males, there remain two aspects of menopause that need further discussion:

1. Women often stop having children a decade before menopause takes place, even accounting for declining fecundity rates. This suggests that optimal family size may often be reached before atresia is completed (Bogin, 1999). There are at least two reasons why this might be the case. First, the rate of death of mothers in childbirth increases with maternal age and number of prior pregnancies and is probably linked to lower muscle tone and decreased immunological performance (Bergsjo, 1997; Fikree, Midhet, Sadruddin, & Berendes, 1997). Second, as families grow larger, the costs linked to sibling rivalry and divided inheritances rise. For at least these reasons, the optimal number of children each woman should have may be selected to be fewer than the maximum possible. There would then be no selection pressure for reproduction after a certain age.

2. What thus needs to be explained are the selection pressures keeping women fit and functional two or more decades after the end of childbearing. Two important ideas are the “mother” and “grandmother” hypotheses, which argue that investment in current offspring or grand-offspring can bring enough inclusive fitness gains to compensate for losses in direct reproduction. The grandmother hypothesis has been repeatedly modeled and tested in several natural fertility populations, both historic and contemporary (Alvarez, 2000; Hawkes, O’Connell, Blurton Jones, Alvarez, & Charnov, 2000; Mace & Sear, 2005; Rogers, 1993; Sear et al., 2000; Shanley & Kirkwood, 2001; Voland & Engel, 1986). The benefits of grandmothering for the survival and reproductive success
of their children and grandchildren are usually significant but not often large enough to compensate for the lack of direct childbearing, if it is assumed that additional childbearing would have the same benefits in older as in younger women, which can be questioned by the arguments previously mentioned. The benefits of grandmothering have probably contributed to selection for longer female lives, despite earlier cessation of direct reproduction, in interaction with conditions imposed by atresia, childbearing risks that increase with age, and indirect costs of large families.

LIFE SPAN AND AGING

Age-specific selection pressures adjust the length of life to an intermediate optimum determined by the interaction of selection with trade-offs intrinsic to the organism. The conditions that select for longer life are those that decrease the value of offspring and increase the value of adults. These include lower adult mortality rates, higher juvenile mortality rates, increased variation in juvenile mortality rates from one reproductive event to the next, and increases in the ability of adults to transfer fitness-increasing support to the next generation (i.e., parental and group care of offspring). Superimposed on the adaptive pattern determined by optimal allocation of resources to maintenance and reproduction are the maladaptive effects of aging. The effects of aging increase mortality rates and decrease fecundity rates in late life beyond the levels predicted from optimal allocation.

Aging evolves because the strength of selection declines with age (Fisher, 1930; Haldane, 1941; Hamilton, 1966). Because there is always some mortality, as individuals age their continued survival contributes less and less to their reproductive success. This fact causes the strength of selection to decline with age and permits genes that have negative effects only late in life but positive (Williams, 1957) or neutral (Medawar, 1952) effects early in life to spread through the population to fixation. Aging follows the onset of reproduction with widespread, diffuse erosion of physiological and biochemical functions caused by many genes of small effect that produce aging as a by-product of selection for reproductive performance—including parental and group care—earlier in life. Aging is not itself an adaptation.

We age more slowly than do our closest relatives, living 2 to 3 decades longer than chimpanzees, bonobos, or gorillas. One reason for the evolution of our longer life is that we have encountered lower extrinsic adult mortality rates because of our cooperative social organization and effective group defense against predators and enemies. However, the drop in adult mortality rates is not itself sufficient to explain our longer life. As Kaplan and Robson (2002) and Lee (2003) have shown, the effects of intergenerational transfers—parental investment and cooperative child care—are needed to explain the long life, low fertility, and high investment in offspring that have evolved in humans and other species. Such effects appear to have been stronger in humans than in our closest relatives.

Evidence is mixed on whether intergenerational transfers impose costs on those who make them: whether a cost of reproduction exists in humans. Three studies illustrate the variety of effects found. In one, Friedlander (1996) documented significantly poorer survival in women who had children than in those who did not, and poorer maternal survival per child ever born, in women born 1880–1904 in Southern California. Those effects were not significant in the cohorts born 1905–1929. Her results suggest that having children increases women’s risk of mortality from late-life diseases, that such risks increase with maternal age, and that they have been decreased by modern hygiene and medicine. In a second study, Lycett, Dunbar, and Voland (2000) saw a different pattern in farmers in northwest Germany followed from 1720 to 1870. They found no relation between number of children and longevity in their total sample, but when they broke it down by economic class, they found an increasingly strong negative relation between number of children and longevity in poorer and poorer women. Thus, human females do appear to suffer a cost of reproduction whose
expression depends on socioeconomic environment. In the third, Doblhammer and Oeppen (2003) controlled for the effects of differences in health and of mortality occurring within the reproductive age classes in a genealogy of the British peerage and then found a significant trade-off between reproduction and longevity for females—but not for males. Whether or not humans experience a cost of reproduction thus appears to depend on their sex, nutritional status, economic well-being, and social class.

**DEVELOPMENT AND PHYSIOLOGY**

*Developmental Determinants of Adult Survival and Reproduction*

Barker (1998; Barker, Winter, Osmond, Margetts, & Simmonds, 1989) discovered that women who are nutritionally stressed during pregnancy give birth to underweight children who later in life are more likely to develop insulin resistance, obesity, high blood pressure, and cardiovascular disease. This observation stimulated a large literature reviewed in Lummaa (2003), Gluckman, Hanson, and Spencer (2005), and Kuzawa and Pike (2005). The pattern has been confirmed experimentally in rats (Desai & Hales, 1997) and in human populations in Europe and India, and the definition of the inducing stimulus has been broadened from events occurring during pregnancy to include postnatal stresses with long-term consequences. While the original observations were made on populations encountering severe famine, later work has shown that smaller babies run higher risks of late-life diabetes, hypertension, and cardiovascular disease whether their mothers were undernourished or not (Gluckman, Hanson, Spencer, & Bateson, 2005).

One interpretation is that the developing embryo perceives its undernutrition as a signal predicting the nutritional conditions it will encounter later in life and sets its metabolism to anticipate nutritional stress throughout life. If this prediction is wrong—if late-life nutrition is actually adequate—then the mismatch between physiology and environment produces a maladaptive response involving obesity, diabetes, and heart disease. Another interpretation is that the embryo’s development and physiology is simply irrevocably disrupted by undernutrition and that it is stuck for the rest of its life making the best of a bad job. Both interpretations remain plausible and the subjects of ongoing research.

This connection between early-life environments and late-life susceptibility to disease helps to explain the global epidemic of obesity, diabetes, and heart disease, especially in India, Mexico, and Africa, where the adult level of nutrition may be poor but improving and still better than that of Paleolithic environments, and where the stress of starvation falls especially heavily on pregnant women.

The early life events that induce late life responses extend from prenatal development into early childhood. Girls who are stunted by poor nutrition as children attain menarche about a year later than those with good nutrition (Khan, Schoeder, Martorell, Haas, & Rivera, 1996) and have reduced output of ovarian hormones throughout their adult life (Ellison, 1996).

These plastic life history responses have profound implications for medicine, epidemiology, and public health, especially in the Third World.

*Sex Hormones: Trade-offs, Morphology, and Buffering*

Within an individual, trade-offs among traits are often mediated by energy allocations controlled by hormones (Calow, 1978, 1979; Ellison, 2003b; Wade, Schneider, & Li, 1996). Hormones elicit responses on the scale of seconds to hours and coordinate both the development and the activity of many tissues with a single signal. The difference in response of various tissues is often a function of the density of receptors on their cell surfaces, not of local variation in the concentration of the hormone, which is well mixed in the blood. Sex hormones play important roles in trade-offs between
reproduction, disease resistance, and fat storage. Testosterone and leptin are two of the hormones that mediate human life history trade-offs.

Testosterone is a steroid secreted primarily from testes and ovaries but also from the adrenal glands and placenta. Males produce about 20 times as much as females do and that difference in circulating testosterone concentration accounts for many of the differences between the sexes in morphology, physiology, and behavior. In most tissues, testosterone activates androgen receptors on the cell surface directly; it can also be converted to estradiol and activate estradiol receptors, primarily in bones and brain. In embryonic development, testosterone induces the formation of the male genitals and the development of the prostate gland and the seminal vesicles. At puberty, increasing testosterone levels accompany the appearance in both sexes of adult body odor, increased skin oil, acne, pubic and axillary hair, the adolescent growth spurt, bone maturation, and fine hair on the upper lip and sideburns. Late-puberty testosterone effects are normal only in males but may cause changes in females with hormone imbalance. They include penis enlargement, increased libido, growth of hair on face, chest, and thighs, decreased subcutaneous fat, increased muscle mass, increased aggression, spermatogenesis, remodeling of face, chest, and shoulders, and completion of bone growth. In adults of both sexes testosterone maintains muscle mass and strength, bone mass and strength, and mental and physical energy. It is thus a key hormone coordinating the expression of many traits throughout the life cycle.

Two traits thus coordinated are survival and reproductive effort; they are tied together through the effects of testosterone on the immune system and on secondary sexual characters and fat storage. Substantial evidence from other vertebrates, somewhat less from humans and nonhuman primates, suggests that testosterone and other androgens regulate the allocation of energy between reproduction and immune functions. During infections, energy is switched from maintaining skeletal muscle mass, red blood cells, and bone density to increasing the immune response, much more so in males than in females. In uninfected males, the normal maintenance of secondary sexual characters by androgens diverts energy away from immune function and increases susceptibility to disease (Muehlenbein & Bribiescas, 2005): The more macho the male, the greater his risk of infection.

In females another hormone, leptin, mediates the trade-offs between reproduction and survival. Leptin is a protein that regulates energy intake and expenditure through effects on appetite and metabolism. It is secreted by fat cells and its concentration in the blood reflects overall fat storage in the body. It binds to the part of the hypothalamus known as the "satiety center," where it signals to the brain that the body has eaten enough. It works by inhibiting neurons that stimulate eating and stimulating neurons that inhibit eating by giving the feeling of satiation. Insulin is the only other hormone functioning as a signal of adiposity; together with leptin, it regulates body fat levels. Obese people have high levels of leptin circulating in their blood, but their bodies are resistant to its effects, just as people with type 2 diabetes are resistant to the effects of insulin. Leptin also plays a role in angiogenesis, the growth of new blood vessels. Because angiogenesis must occur to feed growing cancers, leptin plays a role in regulating the conditions necessary for the growth of metastatic cancers. Therefore obese women experience two effects mediated by leptin that increase their risk of metastatic cancers. They cycle dependably, not experiencing stress-induced amenorrhea, and thus, undergo the frequent episodes of cell differentiation that make possible the mutations that lead to cancer (Strassmann, 1996); when cancer does occur, their bodies are ready to respond by growing the arteries needed to feed it.

The human ovary responds adaptively to stress and nutrition as signals of the probability of a positive reproductive outcome as affected by age, maturation, energy balance, and activity level (Ellison, 1990, 1996). The stress of agricultural work and of poor nutrition appear to be related through effects on ovarian function to seasonal variation in conceptions and spatial and temporal variation in lactation and in the ability of lactation to suppress reproductive function (Ellison, 1994). Even without changing fat stores, the stress of physical work can itself suppress female reproductive function (Jasienska & Ellison, 1998). When women come under nutritional stress, some reproductive
traits respond sensitively, and some do not. Ovarian function, duration of gestation, and final birth weight are sensitive to energy balance, but the rate at which energy is supplied to the embryo by the mother is less sensitive. Energy balance does not have strong effects on the volume of milk produced during lactation, but it does affect the duration of lactational amenorrhea (Ellison, 2003a). Thus pregnant and nursing mothers are strongly stressed by poor nutrition, which causes early births of underweight infants and increases the interbirth intervals of their mothers. Leptin is perhaps the most important signal mediating interactions of stress, fat stores, and female reproduction.

**DISCUSSION**

The causes of variation in life histories among and within human populations are diverse and hierarchical. Phylogenetic history creates one framework within which other effects are expressed: The life histories of the Dobe !Kung and Northern Europeans differ in part because they have been reproductively isolated from each other for roughly 100,000 years. Natural selection causes local adaptation: Life span decreases where externally imposed mortality rates are high, and it increases where such rates are low. Genes and culture probably coevolve to produce effects on life histories, but this has not yet been demonstrated conclusively. Nutrition plays a key role: Well-nourished humans mature earlier and have shorter interbirth intervals than nutritionally stressed humans. Conflicts within the family, between parents and offspring and among sibs, also affect performance through impacts on growth, mating opportunities, and other traits. The way in which genetic differences interact with the local environment to produce phenotypic differences is mediated by physiological mechanisms, many of them controlled by hormones. Thus understanding human life history evolution requires a broad view across many academic specialties and an ability to synthesize the effects of diverse causes.

Human evolution did not stop in the Pleistocene. Human life histories can still be evolving, for many human life history traits are heritable and there is abundant variation in reproductive success in contemporary human populations. The key issue is whether variation in life history traits currently correlates strongly with variation in reproductive success. In some environments, it probably does; in others, it probably does not; the issue thus remains open. If, as seems likely, human life histories are evolving, they are doing so at a rate that is very slow compared to the rate of cultural change. It is thus virtually certain that contemporary human life histories are increasingly becoming poorly matched to rapidly changing cultural environments.

The human life history constrains both cognition and cultural dynamics. It implies that it will take humans 15–20 years to complete the physiological development and cultural learning required for adult function. Because this must be done by every individual in every generation, the rate of cultural change is constrained by both the evolved rate of brain development and the rate at which culture can be acquired by learning. Every population consists of a mixture of the young, who are still involved in doing this, and the older, who might form a different culture if they were not both constrained and stimulated by the young. After 5–6 decades of functional adulthood, aging erodes cognitive capacity. Thus, the window available for fully realized cultural transmission is framed by the portion of the population roughly between 20 and 80 years of age. This is a fundamental constraint on cultural evolution.

**REFERENCES**


