Plasticity in Human Life History Strategy

Implications for Contemporary Human Variation and the Evolution of Genus *Homo*

by Christopher W. Kuzawa and Jared M. Bragg

The life history of *Homo sapiens* is characterized by a lengthy period of juvenile dependence that requires extensive allocare, short interbirth intervals with concomitantly high fertility rates, and a life span much longer than that of other extant great apes. Although recognized as species-defining, the traits that make up human life history are also notable for their extensive within- and between-population variation, which appears to trace largely to phenotypic and developmental plasticity. In this review, we first discuss the adaptive origins of plasticity in life history strategy and its influence on traits such as growth rate, maturational tempo, reproductive scheduling, and life span in modern human populations. Second, we consider the likely contributions of this plasticity to evolutionary diversification and speciation within genus *Homo*. Contrary to traditional assumptions that plasticity slows the pace of genetic adaptation, current empirical work and theory point to the potential for plasticity-induced phenotypes to "lead the way" and accelerate subsequent genetic adaptation. Building from this work, we propose a "phenotype-first" model of the evolution of human life history in which novel phenotypes were first generated by behaviorally or environmentally driven plasticity and were later gradually stabilized into species-defining traits through genetic accommodation.

Introduction

Modern humans are characterized by a life history strategy with features that are distinct from other nonhuman primates, including slow childhood growth, early weaning followed by a long period of dependence, a shortened interbirth interval, and lengthy life spans (Hawkes et al. 1998; Hill 1993; Hill and Kaplan 1999; Robson, van Schaik, and Hawkes 2006). Extensive provisioning of dependents by kin and potentially unrelated individuals allows humans to "stack" offspring and spread the burden of provisioning across alloparents, thus facilitating relatively high fertility despite the intensity of investment and high survival of each offspring (Bogin 1999; Hawkes et al. 1998; Hill and Hurtado 1996; Kaplan et al. 2000). Current interest in reconstructing the evolutionary emergence of these characteristics in modern humans and the life histories, behaviors, and biology of fossil hominins represents a key intersection between human biology and paleoanthropology.

Recent fossil discoveries have revealed extensive variation in the fossil signatures of life history variation in *Homo erectus*, calling into question previous assumptions regarding the evolution of size and shape in *Homo* and bringing variation per se to the fore as an important focus of analysis (e.g., Antón 2003; Antón et al. 2007). These findings raise questions about the evolutionary origins of this diversity, which might reflect distinct species or locally adapted variants of the same species (Antón 2003). While the magnitude of this regional variation may complicate taxonomic distinctions, it is also notable for its similarities to the variation observed across contemporary human populations, among which there is extensive variation in life history traits such as growth rate, body size, reproductive scheduling, and even life span. Biological anthropologists and others who study the life histories of contemporary humans have shown that much of this variation can be explained as the outcome of phenotypic or developmental plasticity triggered in response to social, nutritional, demographic, and other environmental conditions (Chisholm 1993; Ellis et al. 2009; Kuzawa and Pike 2005; Walker et al. 2006*a*). If plasticity is an important contributor to contemporary human variation, it follows that it has likely also been important as an influence on the life histories of human ancestors. As such, considering the mechanisms and adaptive significance of phenotypic plasticity in modern human life histories provides insights into the origin and function of similar variation in early *Homo*.

We have several interrelated goals in this review. We first

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discuss the role of phenotypic and developmental plasticity as a primary means of population adjustment to environmental changes occurring on generational or multigenerational timescales. This background discussion culminates in a review of evidence for environmental influences on human life history variation, which highlights the effects that nutrition and cues of mortality risk have on growth rate, maturational tempo, and adult size. A review of the literature on modern humans confirms that developmental plasticity is the primary source of much human life history variation across contemporary populations and points to the likely importance of similar processes as contributors to regional and temporal variation in the fossil record.

Because only those genotypes that are expressed phenotypically are subjected to selection, environment-driven phenotypic variation in human populations may also influence the process, pace, and direction of evolutionary change (West-Eberhard 2003). From the perspective of the emerging synthesis of developmental and evolutionary biology, the generation of regional variation through plasticity likely provided the raw phenotypic variation that was then selectively retained or pruned, leading the way for more gradual adaptation via natural selection. We conclude by speculating that environment-driven developmental plasticity may not only provide insights into the origins of variation in contemporary and fossil hominin populations but also may have played a fundamental role in the evolution of our species by facilitating phenotypic adaptation that preceded more durable genetic change and speciation.

The Unusual Human Life History

Organisms vary remarkably in the size and pace of life, which is reflected in body size, growth rate, fertility rate, and life span. These traits help define a species' life history, which may be viewed as a life-cycle strategy that optimizes expenditures in service of reproductive success (Stearns 1992). Classically, organisms are viewed as being constrained by finite energetic resources that must be partitioned to growth, reproduction, and maintenance functions (Gadgil and Bossert 1970). Growth builds a larger body that is less prone to predation and has an absolutely greater capacity to invest in reproduction. After growth ceases at maturity, energy previously allocated to growth is then shunted into reproduction. In females this involves support of offspring growth in utero and via breast milk, and in males the building and maintenance of sexually dimorphic, energetically costly traits and behaviors (Charnov 1993; Kuzawa 2007). Consequently, organisms face a fundamental trade-off in deciding how much of their time and energy budget to invest in growth or reproduction in the present or to processes that minimize or repair cellular or tissue damage to extend life span. For instance, because resources are finite, it is assumed that organisms may invest heavily in productivity—growth and reproduction—or have long life spans, but not both (Stearns 1989; Williams 1966).

As the reproductive benefits from investing in a durable, long-lived body may only be realized in the future, the utility of allocating scarce resources to maintenance activities is linked to the risk of unavoidable extrinsic mortality (e.g., predation) that members of a population or species face, which is viewed as a primary driver of life history diversification and evolution (Charnov 1993; Promislow and Harvey 1990). Species living in ecological contexts characterized by high mortality risk are less likely to live into the future and thus are predicted to allocate a larger fraction of their energy budget to current reproduction with comparably little devoted to maintenance (Kirkwood and Rose 1991). As a result, these "fast" life history species typically have shorter life spans and give birth to many lower-quality offspring with relatively low survival prospects. Conversely, when mortality risk is low, theory predicts a "slow" life history characterized by reduced expenditure on growth or reproduction and greater investment in life span–extending maintenance. Slow life history species tend to give birth to fewer offspring, but they invest more intensively in each, enhancing survival (Charnov and Berrigan 1993).

The human life history strategy is unusual in that it is clearly a slow and investment-oriented strategy in most respects while also characterized by some of the demographic benefits typically associated with fast life histories. Low mortality rates enable slow childhood growth and delayed onset of physical maturity, nutritional independence, and reproduction well beyond what is seen in other great apes (Hawkes et al. 1998; Kaplan et al. 2000; Walker et al. 2006*b*). Also consistent with a slow strategy, humans invest extensive amounts of time and energy in offspring during this protracted period of dependence (Gurven and Walker 2006). Yet despite this, contemporary foraging populations manage to reproduce nearly twice as fast as other great apes and have higher completed fertility (Walker et al. 2008).

This unusual life history capacity to invest heavily in more offspring has been traced to the distinctively human practice of weaning offspring early and the consequent shortening of the interbirth interval relative to other great apes (Galdikas and Wood 1990; Humphrey 2010; Knott 2001; Sellen 2006, 2007). Early weaning is accompanied by a long transitional period of providing weanlings specially prepared foods that may be acquired and provisioned flexibly within human social units (Bogin 1999; Bogin and Smith 1996; Hawkes et al. 1998; Kaplan et al. 2000; Lee 1996). This allows alloparents, such as grandmothers or older siblings, to provide a substantial fraction of the energetic needs of each offspring, thus freeing maternal metabolism to initiate new pregnancies. It is increasingly recognized that the remarkable demographic success and geographic range of our species hinges on the flexibility of these alloparental transfers, which allow us to "have our cake and eat it too," producing many high-quality and

low-mortality offspring (Kramer 2010; Lee 2003; Wells and Stock 2007).

The Timescales of Human Adaptation and the Role of Developmental Plasticity

While this strategy is characteristic of the human life history generally, nutritional and mortality conditions can vary widely across environments and through time. Although genetic adaptation by natural selection helps explain the durability of species-level characteristics that differentiate us from other great apes, the transience of many of the ecological challenges that populations confront may not be dealt with effectively by changes in gene frequencies, which require many generations and hundreds if not thousands of years to accrue in the gene pool. As such, population variation in life history parameters is largely traceable to developmental plasticity. West-Eberhard (2003:33) defines "phenotypic plasticity" as "the ability of an organism to react to an internal or external environmental input with a change in form, state, movement or rate of activity." "Developmental plasticity" refers to that subset of plastic phenotypic responses that involve irreversible modifications to growth and development. Plasticity is understood as being undergirded by a genetic architecture that allows context-dependent trait expression in response to varying environmental experiences and behaviors (McIntyre and Kacerosky 2011; Stearns 1992; Stearns and Koella 1986).

Biological anthropologists have long emphasized nongenetic means of adapting to environmental challenges, and this work has highlighted the importance of developmental plasticity as a key mode of human adaptation (fig. 1; Frisancho 1977; Kuzawa 2005, 2008; Lasker 1969). The body copes with the most rapid ecological fluctuations using rapid, self-correcting, and reversible homeostatic systems that respond to changes or perturbations in a way that offsets, minimizes, or corrects deviations from an initial state. Homeostatic systems can be viewed as buffering the effects of environmental fluctuations to maintain approximate internal stability. A subset of homeostatic processes (Sterling 2004) also have an anticipatory component and a capacity for gradual resetting of target state and regulatory set points described as "allostasis."

When environmental trends are too chronic to be efficiently buffered by homeostasis or allostasis and yet not chronic enough for substantial genetic change to consolidate around, the flexible capacities of such systems may be overrun. It is easy to see how a sustained environmental change might overload a homeostatic system. As an example, consider an individual who moves to high altitude where oxygen saturation is low. Initial physiologic responses will include an elevated heart rate, which increases the volume of blood and thus the number of oxygen-binding red blood cells that pass through the lungs. By engaging a homeostatic system—heart rate—the body has found a temporary fix. However, if heart rate is already high under resting conditions, there is less leeway to deal with new challenges that might require a further increase in heart rate, such as running from a predator. Homeostatic changes may work as short-term solutions, but they are a poor means of coping with a condition such as high-altitude hypoxia if this is the new baseline environmental state.

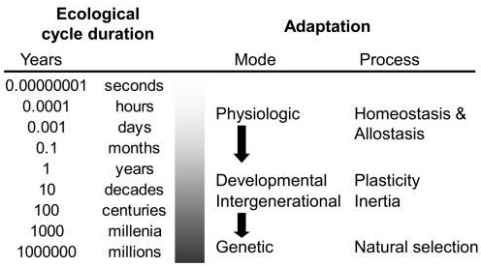

Figure 1. Timescales of human adaptability (modified after Kuzawa 2005, 2008). Light gray = more rapidly responsive/less durable; black = slowest to respond/most durable. Black arrows indicate the order with which novel phenotypes appear. Under most circumstances, novel environments or behaviors first induce novel phenotypes via reversible homeostatic processes that may be replaced by more durably accommodated phenotypes via developmental and intergenerational plasticity. If the changed conditions are stable for sufficient generations, natural selection may gradually fix the phenotype or reduce the costs associated with producing it. In this way, highly plastic traits can "lead the way" and accelerate the pace of genetic change.

This is where the value of developmental plasticity becomes clear. Individuals raised at high altitude have a more efficient strategy for coping with low oxygen availability, for they simply grow larger lungs during childhood (Frisancho 1977). This is an example of how developmental plasticity allows organisms to adjust biological structure on timescales too rapid to be dealt with through genetic change but too chronic to be efficiently buffered by homeostasis. Other classic examples of experience-driven plasticity include the development of the skeletal system (Pearson and Lieberman 2004), the central nervous system (Edelman 1993), and the immune system (McDade 2003).

Intergenerational Phenotypic Inertia

There is a growing list of biological systems that are not modified in response to the environment itself but to hormonal or nutrient signals or cues of past environments as experienced by ancestors, most typically the mother (Bateson 2001; Gluckman and Hanson 2004; Kuzawa 2001, 2005; Wells 2007; Worthman 1999). Brief "critical" or "sensitive" periods in early development often overlap with ages of direct nutrient, hormone, or behavioral dependence on the mother (e.g., via placenta, breast milk, or emotional attachment), which facilitate the transfer of integrated cues of past maternal or matrilineal experience (Kuzawa and Quinn 2009). These newer examples of early life developmental plasticity are thus distinct from conventional plasticity in the time depth and

stability of information to which the developing body responds.

The tendency for plasticity to respond to parental nutrient, hormonal, or behavioral cues that integrate past environmental experience has been defined as "phenotypic inertia" (Kuzawa 2005, 2008). One possible explanation for the utility of such effects is that offspring are calibrating growth and nutrient expenditure to the mother's capacity and willingness to invest in the present offspring, which reflect her own phenotypically embodied nutritional history (Wells 2003, 2007). From an alternative perspective, these intergenerational effects could calibrate offspring growth, metabolism, and physiological settings to a stable "running average" index of conditions experienced by recent matrilineal ancestors that serve as a "best guess" of conditions likely to be experienced in the future (fig. 2; Kuzawa 2005). By this reasoning, intergenerational effects of matrilineal experience (and possibly also patrilineal experience via germ-line epigenetic inheritance; see Eisenberg, Hayes, and Kuzawa 2012; Pembrey 2010) could extend the utility of developmental plasticity as a mode of adaptation by allowing organisms to track more integrated and stable trends occurring across a multigenerational timescale (for more, see Kuzawa 2005, 2008; Kuzawa and Quinn 2009; Kuzawa and Thayer 2011).

Thus, the type of organismal response to ecological challenge or novelty depends not only on the type of stressor or experience but also its stability and duration. Selection has shaped biology to respond to such experiences in a myriad of ways; as such, adaptation does not simply occur through evolution by natural selection and alteration of gene frequencies but also through homeostasis, allostasis, phenotypic/developmental plasticity, and phenotypic inertia across generations (fig. 1). As life history characteristics take many forms, from behavioral to physiological to molecular (Hill and Hurtado 1996), we should expect a similar breadth of mechanisms and timescales underlying life history evolution and adaptation. As one example, mounting evidence suggests that many key life history traits are among those most strongly shaped by early life experiences and that they may respond to signals conveyed by the mother across the placenta or via breast milk (Kuzawa 2007; Kuzawa and Pike 2005; Kuzawa and Quinn 2009; Wells 2003). Continuing in this vein, we argue that adaptive phenotypic plasticity has a central place in explaining variation in life history traits among contemporary human populations.

Variation in Modern Human Life Histories Traces Primarily to Environment-Driven Plasticity

We have reviewed the key derived characteristics of the human life history strategy. We have also considered the need for a capacity to modify priorities "on the fly" as an important dimension of life history strategy for most organisms in light of changing nutritional and demographic/mortality conditions. Consistent with this expectation, most human life history traits exhibit extensive sensitivity to ecological context (see table 1). Here we summarize the role of nutrition and cues of environmental risk as influences on the key life history parameters of growth trajectory, adult size, and reproductive strategy.

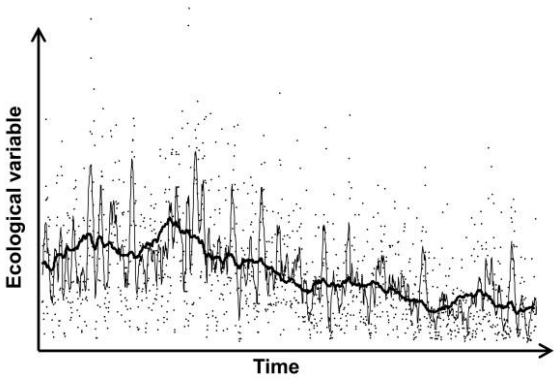

Figure 2. Value of intergenerational averaging as a way to identify a trend in a noisy signal, in this case representing availability of a hypothetical ecological resource. The two lines are running averages calculated across 10 time units (thin line) and 100 time units (dark line). As the window of averaging increases, an underlying long-term trend is uncovered. Transgenerational influences of maternal and grandmaternal experience on fetal and infant biology (inertia) may help achieve a similar feat. (From Kuzawa and Quinn 2009, with permission.)

Somatic Growth and Adult Body Size

Growth during the postnatal period can be divided into several periods of distinct hormonal regulation that vary in sensitivity to environmental influence and that ultimately determine age-specific contributions to adult size and sexual dimorphism (Karlberg 1989). Roughly the first two postnatal years reflect a continuation of a growth regime begun in utero. At this age, production of insulin-like growth factors that stimulate skeletal and somatic growth is insulin dependent, tying growth rate directly to nutritional intake and nutritional sufficiency. Prenatal evidence for this effect comes from a recent large study showing that the level of a woman's fasting glucose during pregnancy is a robust predictor of the size of her offspring at birth, illustrating that fetal glucose supply drives fetal growth (Metzger et al. 2009). After birth, exclusive breast-feeding sustains the infant's nutritional requirements for about the first 6 months of life, after which complementary foods must be introduced to avoid growth faltering (Sellen 2006). While breast-fed, infants obtain balanced nutritional resources to support growth and passive maternal immune protection, which minimizes the burden of energetically costly infections. The weaning transition often introduces nutritional stress as these resources are replaced with less balanced and less sterile complementary foods. As such, infancy is often

Table 1. Range of variation for key life history characteristics in modern human populations

Characteristic	Males		Females		Environmental influences
	Range	Degree of plasticity	Range	Degree of plasticity	
Height (cm):					
Birth (length)	49.0[a]–51.9[b]	+	48.1[a]–52.2[b]	+	Mother's nutrition before pregnancy increases; stress, infection decrease
Weaning (3 years)	82.0[a]–99.0[b]	++	81.0[a]–98.6[b]	++	Exclusive breast-feeding until 6 months protects; introduction of complementary foods, and infection lead to faltering
Midchildhood (6 years)	97.0[b]–120.8[b]	++	96.8[a]–120.2[b]	++	Deficits from early growth and poor environmental or nutritional milieu reduce stature
Adult	144.4[b]–181.6[a]	++	135.8[a]–168.2[b]	+	Low birth weight and poor nutrition/growth before 3 years of age reduce stature
Weight (kg):					
Birth	2.4[a]–3.57[a]	++	2.50[b]–3.5[a]	++	
Weaning (3 years)	10.3[a]–16.1[b]	++	10.1[a]–16.1[a]	++	
Midchildhood (6 years)	14.5[b]–23.3[b]	++	13.8[b]–23.4[b]	++	
Adult	40.0[a]–88.1[b]	++	37.0[a]–87.4[b]	++	
Age at maturity (years):					
Menarcheal age			12.1[b]–18.4[a]	+++	Poor growth/nutrition delay; prenatal stress/childhood psychosocial stress and abundant nutrition accelerate
Age at peak height velocity[c]	12.0–17.0	++			Fast postnatal/infancy growth accelerates
Age at first birth[c]	20.9–37.0		16.2–25.0		Stress or cues of extrinsic mortality accelerates
Age at menopause[d]			48.2–52.6	++	Higher parity, longer cycles delay menopause; some evidence that smoking, small size at birth, poor early growth lead to earlier menopause
Life span:[e]					
e_0	21–37		21–37		Higher infant mortality and rates of infection and violence/accidents decrease
e_{15}	28.6–42.5		28.6–42.5		Reduced exposure to environmental and health stressors increases
e_{45}	13.7–24.2		13.7–24.2		

[a] Eveleth and Tanner 1976.
[b] Eveleth and Tanner 1990.
[c] Walker et al. 2006*a*.
[d] Leidy Sievert 2006.
[e] Gurven and Kaplan 2007; e_x is life expectancy at age x.

an age of nutritional stress with a high-mortality burden. This, combined with the need to buffer an unusually large and inflexible cerebral energy need, may help explain the heavy human investment in deposition of protective fat stores before birth and during the first 6 months of postnatal life (Kuzawa 1998).

Because this difficult transition coincides with the age of insulin-dependent, nutrition-driven growth, weaning-related growth deficits can carry into adulthood to influence final stature and body weight. Indeed, the magnitude of adult height deficits relative to healthy reference data has been shown to trace largely to growth faltering already present at 2 or 3 years of age (Billewicz and McGregor 1981; Martorell 1995), and much of the contemporary population variation in adult standing height is believed to reflect the effect of nutrition and hygiene during infancy and early childhood (Eveleth and Tanner 1976, 1990; Habicht et al. 1974; Victora et al. 2008).

Although growth rate remains sensitive to nutrition during the entire period of growth and development, long-term effects of nutrition on adult size diminish after infancy and early childhood as insulin-dependent growth is gradually replaced by a growth-hormone-regulated growth regime (Karlberg 1989). During childhood and puberty, nutritional deficits primarily slow the pace of maturity without affecting final stature or body size. During the pubertal growth spurt (see below), onset of gonadal production of sex steroids increases growth rate, especially in males. However, as with childhood growth, there is little evidence for lasting effects of nutrition during adolescence on final adult size. Generally, individuals who are better nourished or have more abundant fat stores during childhood enter puberty earlier, experience a more intense but briefer period of heightened pubertal growth, and attain maturity at a younger age (Tanner 1962). As nutritional conditions deteriorate, onset of pubertal growth is delayed, and the spurt is also protracted such that growth velocities are slower but spread across a longer period. Collectively, these findings show that nutrition primarily influences adult size by influencing growth attainment during fetal life, infancy, and early childhood, when nutritional resources are derived primarily from the mother's body, thus linking adult size in the present generation with matrilineal nutritional history (Kuzawa 2005, 2007; Kuzawa and Quinn 2009; Wells 2007).

Although nutrition is clearly a powerful influence on human variation in growth rate and adult size, it is worth noting that important genetic contributions to population variation in stature are especially likely at the extremes. For instance, the shortest populations in the world are "Pygmy" populations such as the Efe or Mbuti, whose atypically short stature likely has at least a partial genetic component that reflects convergent genetic selection in response to ecological or mortality conditions common in rainforest environments (Perry and Dominy 2009; Pickrell et al. 2009; Walker et al. 2006*a*). Similarly, the tall mean stature of the tallest human groups, such as the Rift Valley pastoralist populations, may have a partial genetic explanation (Gray, Wiebusch, and Akol 2004). However, these extremes aside, environmental factors, especially differences in nutrition and pathogen burden during the first 2–3 years of life, are recognized as the primary driver of variation in mean adult size across populations (Eveleth and Tanner 1976, 1990).

Nutrition as an Influence on Age at Reproductive Maturity

Age at maturity represents an important life history transition because it marks the age at which the body shunts energy previously allocated to somatic growth into reproduction (Charnov 1993; Kuzawa 2007). Although large adult size carries reproductive benefits, the ability to sustain the nutritional requirements of fast growth and the risk of preadult mortality that determines how long it is prudent to delay maturing are both variable. Thus, it is expected that maturational tempo will follow a gradient of developmental plasticity (i.e., a reaction norm) that is sensitive to availability of nutritional resources and also to cues reflecting the level of mortality risk (Coall and Chisholm 2003; Ellis et al. 2009; Stearns and Koella 1986; Walker et al. 2006*a*). Consistent with this expectation, maturational tempo is among the most variable of human life history characteristics and exhibits sensitivity to both nutritional and psychosocial stressors.

The role of nutrition as a driver of childhood and adolescent growth helps explain the extreme environmental sensitivity of pubertal timing. Population means for menarcheal age range from about 12 to 18 years (table 1). Rapid multigenerational secular trends clearly show that this variability largely reflects environmentally driven plasticity in maturational tempo. As noted above, in some Western European and Scandinavian countries, menarcheal age declined from around 17 to 18 years in the mid-nineteenth century to present population means of 12 or 13 years (Eveleth and Tanner 1976, 1990). More recent studies in non-European populations also demonstrate rapid declines in menarcheal age. For instance, in South Korea, age at menarche declined from 17 years in 1920 to 12–14 years in 1985, representing a rate of decline of 0.68 years/decade (Cho et al. 2009), while age at menarche declined at a similar rate of 0.65 years/decade from 1989 to 2008 in a rural Gambian population (Prentice et al. 2010).

Striking evidence for the sensitivity of menarcheal timing to environmental influence is illustrated in growth studies of girls adopted from orphanages in India or Bangladesh into high-income Scandinavian households. These studies document relatively high rates of precocious puberty with adoptees entering puberty as early as 7 years of age (Proos, Hofvander, and Tuvemo 1991; Teilmann et al. 2006). Intriguingly, the degree to which maturation was sped up in these girls depended on their age of adoption: girls adopted at older ages, who therefore spent more time in less favorable conditions, entered puberty earliest upon environmental improvement (Proos, Hofvander, and Tuvemo 1991). Such findings suggest that an individual's developmental response to environmental

factors such as nutrition may itself be contingent upon prior developmental conditions experienced during early life. Additional evidence for such "programming" effects of early experiences comes from the finding that being born small—often a result of fetal nutritional deficit or maternal stress during or before pregnancy—predicts earlier maturity especially when small birth size is followed by rapid catch-up growth after birth (Adair 2001; Ibáñez et al. 2000; Karaolis-Danckert et al. 2009; Ong et al. 2009).

The lack of an easily measured maturational marker in males comparable to the onset of menses has constrained understanding of both the extent of variability in male pubertal timing and sensitivities of male maturational tempo to environmental change. In the populations for which data are available from Walker et al.'s (2006*a*) tabulation of growth rates in small-scale societies, age at peak height velocity (a proxy for pubertal timing) ranges from 12 to 17 years (table 1). However, few studies have investigated the environmental or nutritional factors that predict variability in male maturational tempo. A recent study conducted in Germany found that in accordance with the effect of birth weight and early growth among females, males who were born small and gained weight rapidly from birth to 2 years experienced peak height velocity earlier (Karaolis-Danckert et al. 2009). Similarly, a recent study in a longitudinal birth cohort in the Philippines reported that males who experienced rapid weight gain immediately after birth reached puberty earlier (Kuzawa et al. 2010).

Nutrition, Developmental Plasticity, and the Origins of Sexual Dimorphism

In species marked by sexual size dimorphism, the greater size of males leads to correspondingly higher nutritional requirements. This is believed to help explain why males tend to exhibit greater responses, both positive and negative, to changes in nutritional conditions (Stinson 1985). As a result of this differential sensitivity, the magnitude of sexual dimorphism in traits such as body size will tend to shift as prevailing nutritional conditions change. For instance, baboons that self-provisioned off of trash dumps were found to weigh 50% more than wild-fed baboons (Altmann et al. 1993). The magnitude of the weight gain was much greater in the males than in the females, leading to an increase in adult size dimorphism (Altmann and Alberts 2005). In human populations, adult size in males has similarly been shown to be more sensitive to changes in socioeconomic condition or nutritional abundance (Stinson 1985).

Environmental experiences early in the life cycle may be key to the establishment of these differences. Before birth and during the first 6 months of postnatal life, testosterone production is temporarily high in males, which has long-term "organizational" effects on male reproductive biology, behavior, and body growth (Jost 1961; Phoenix et al. 1959). Recent evidence points to these early periods of hormonally driven organizational effects as potential sources of plasticity in the pattern and degree of biological and behavioral difference between males and females. In a well-characterized birth cohort in the Philippines, males who grew rapidly during the age of high postnatal testosterone but not at other early ages gained weight and height faster, matured earlier, and were taller and more muscular as adults (Kuzawa et al. 2010). These relationships were greatly reduced or not present for most outcomes among same-aged females. Men who grew rapidly after birth also reported an earlier age at first sex, more lifetime sex partners, and more recent sexual activity. Because these men also showed evidence for greater adult testicular sensitivity to luteinizing hormone and higher testosterone levels, the authors speculated that nutritional experiences during the early postnatal critical period might have lasting effects on the magnitude of physical and behavioral differences between males and females. It is notable that male body size and related energetic costs were reduced in response to early life cues reflecting reduced nutrition, perhaps indicating a capacity to calibrate life history and energetic expenditure as prevailing nutritional conditions change. Sex-specific sensitivities of growth rate and developmental processes suggest that any environmental changes that influence food availability could have differential effects on males and females that could thereby influence the pattern of sexual dimorphism within and between populations.

Psychosocial Stress, Maturational Tempo, and Reproductive Scheduling

Although the public health and growth and development literatures have traditionally focused on the role of nutrition and hygiene as influences on growth and adult size, more recent work is showing that psychosocial stress can also influence growth rate and maturational tempo. One link between stress and growth stems from the energy burden of the stress response itself, which may compete with growth, leading to a reduced growth rate (Nyberg et al., forthcoming).

In addition to such direct resource trade-offs, stress may also serve as a barometer of nonnutritional risks, such as unavoidable mortality, which is recognized as shaping the optimal timing of maturity and reproductive scheduling in models of mammalian life history evolution (Charnov 1993; Promislow and Harvey 1990). Building from this premise, a long-standing research tradition in developmental psychology and anthropology has developed evolutionary explanations for the sensitivity of maturational tempo to parental or other social cues (Belsky, Steinberg, and Draper 1991; Chisholm 1993; Draper and Harpending 1982). In these studies, the observation that girls from harsh or unstable family environments tend to mature earlier is interpreted as evidence that human maturational tempo is responsive to cues of extrinsic mortality risk as reflected in attachment quality and parental investment (Chisholm 1993; Ellis 2004; Ellis et al. 2009). Numerous studies have tested this and related predictions (Ellis

and Garber 2000; Hulanicka, Gronkiewicz, and Koniarek 2001; Pesonen et al. 2008; Tither and Ellis 2008). For instance, Chisholm et al. (2005) found that retrospectively reported total life stress explained about 11% of the variance in menarcheal age in a sample of college-aged women, with higher stress levels predicting earlier maturity. Quinlan (2003) similarly found that women whose parents separated before they were 6 years old matured earlier than girls whose parents did not separate. The effect size in these studies is often on the order of 1–2 months (e.g., Belsky et al. 2010), which is quite small, especially when compared with the large multigenerational trends in menarcheal age documented in association with nutritional improvements.

Cues of extrinsic mortality may better explain variation in reproductive scheduling and the intensity of parental investment. Age at first reproduction varies widely in the data compiled by Walker et al. (2006*a*; table 1), and there is a large and growing literature showing that children exposed to high extrinsic mortality or low parental investment during early life not only mature earlier but also start reproducing at a younger age (Burton 1990; Chisholm et al. 2005; Low et al. 2008; Nettle, Coall, and Dickins 2010). Nettle (2010) found that across neighborhoods in England, women in the most socioeconomically deprived communities gave birth for the first time an average of 8 years earlier than women in the most affluent communities, paralleling Wilson and Daly's (1997) finding of earlier and more intensive reproductive scheduling in Chicago neighborhoods with the highest homicide rates. In another study, Nettle, Coall, and Dickins (2011) found that prolonged maternal absence, low paternal investment, and many residential moves during childhood were independent predictors of earlier age at first birth. Each of these stressors lowered the age at first reproduction by about half a year, and their effects were additive.

There is also evidence that individuals who experience stressful environments during childhood invest less in their own offspring (Ellis et al. 2009; Hurtado et al. 2006). For instance, parental investment is reduced under conditions of harsh, unavoidable stressors such as pathogen loads, famine, or warfare (Quinlan 2007). Such reduced investment need not be solely behavioral: associations between low socioeconomic status and birth weight have been documented and interpreted in terms of reduced investment in offspring (Coall and Chisholm 2003, 2010; Nettle 2010). Conversely, in high-opportunity/low-mortality populations, age at first reproduction is typically delayed as a response to lowered mortality rates and higher costs and future payoffs of investing in offspring (Low et al. 2008). Indeed, in many European countries, age at first birth now routinely occurs in the 30s (ESHRE Capri Workshop Group 2010) even as age at biological maturity has declined because of nutritional and energetic improvements.

Anthropologists have criticized the focus on the nuclear family as the presumed unit of human child rearing in much of this work (Chisholm et al. 2005; Hrdy 2009). Instead, humans can be described as a cooperatively breeding species in which reproducing females rely on flexible patterns of alloparental care as a fundamental component of their reproductive strategy (Hill and Hurtado 2009; Hrdy 2005, 2009; Kramer and Ellison 2010). Moreover, complicating the traditional emphasis on the presumed primary role of men as hunters and providers of calories, recent work highlights evidence for derived neuroendocrine adaptations specific to the human lineage that encourage direct male care of offspring (Gettler et al. 2011). In light of these revelations, it seems likely that the developmental capacities for facultative adjustment of life history have been shaped to be sensitive to a much broader range of social cues than previously theorized. For instance, while transplacental nutrients or maternal breast milk are important sources of maternal ecological information before birth and during infancy (Kuzawa and Quinn 2009), after 6 months of age, an increasing proportion of energetic needs are met by complementary feeding of specially prepared foods that may be provided by various relatives or group members (Bogin and Smith 1996; Sellen 2006, 2007). To the extent that nutrition during infancy helps calibrate growth and reproductive expenditure, in humans these trajectories may be as much reflective of availability of alloparental care as any direct maternal metabolic investment. This example illustrates how theories concerning the role of stress and social cues in life history scheduling and resource allocation need to address the fact that human children are often highly reliant on investment from individuals other than biological parents.

Summary: Phenotypic Responses to Changing Ecological Conditions

In figure 3, we summarize the pattern of life history traits that theory predicts in environments with different combinations of nutritional sufficiency and unavoidable mortality risk. At one extreme are the most-favorable environments in which nutritional sufficiency is high, allowing fast growth and the attainment of a large adult size despite relatively early maturity (fig. 3*A*). In this example, early maturity is secondary to favorable nutrition and a consequent fast growth rate. When populations experience energy sufficiency but cues of high extrinsic risk, they are expected to grow quickly but also mature early and thus are predicted to be slightly smaller as adults (fig. 3*B*). In contrast, low-nutrition and low-risk environments are expected to lead to growth that is sufficiently slow that despite a compensatory delay in maturity, individuals still attain a shorter adult size (fig. 3*C*). Finally, another theoretical extreme is represented by a combination of low nutritional resources and high extrinsic risk, which should lead to relatively early maturation at a small size (fig. 3*D*).

Mortality risk influences optimism about surviving into the future to reproduce, and as such there are predicted to be tandem shifts in relative allocation between maintenance and reproduction and also in the level of investment in each offspring. Theory predicts that as unavoidable mortality in-

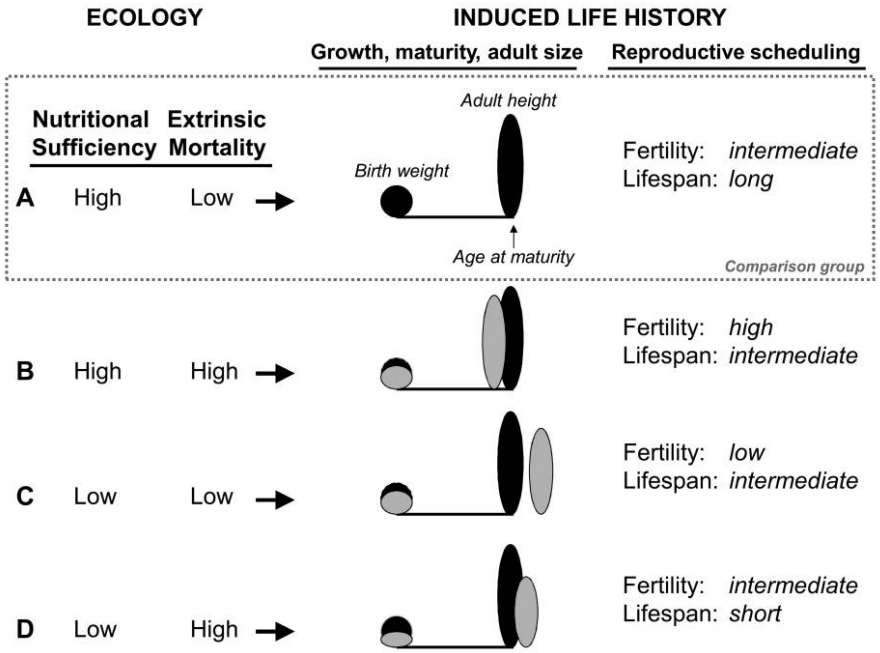

Figure 3. Summary of developmentally plastic life history changes predicted by different combinations of nutritional sufficiency and cues of threat or extrinsic mortality risk. Comparison group for depiction of birth weight, maturational tempo, and adult stature = high nutrition/low extrinsic mortality risk group (see text for discussion). Not drawn to scale.

creases, not only is maturity sped up but also age at first birth, which is accompanied by an increase in fertility rate related in part to a reduction in the interbirth interval and reductions in offspring investment, size, and survival (Belsky, Steinberg, and Draper 1991; Chisholm 1993; Ellis et al. 2009; Nettle, Coall, and Dickins 2011). This plasticity in both the rate of metabolic expenditure and in the scheduling of developmental and reproductive events helps explain much of the variation in growth rate, body size, and reproduction across contemporary human populations.

Discussion: Summary and Implications of Life History Plasticity

Organisms must manage the costs of building a body and the bodies of offspring while calibrating the scheduling of developmental and reproductive events in response to nutritional resources, the risk of mortality, and other changing or unpredictable ecological conditions (Charnov 1993; Promislow and Harvey 1990; Stearns 1992). It is this environmental variability that necessitates plasticity in growth, development, reproduction, and other components of a species' life history strategy. Consistent with this perspective, our review highlights the overwhelming importance of environmentally driven developmental plasticity as a source of contemporary human life history variation.

Extrapolating from these findings in modern human populations, developmental plasticity has likely been an important influence on the phenotypes of earlier members of the hominin lineage. As the ancestors of contemporary human populations spread across Africa and eventually Eurasia, the local conditions that they faced would have varied in energetic and demographic conditions. Regional or population differences in such parameters as growth rate, maturational tempo, body size, and sexual dimorphism would have likely arisen via plasticity as local or regional populations were confronted by these ecological differences. While at present it is impossible to know whether the same range of plasticity in life history responses was present in ancestral hominins, it is likely that plastic responses were at least similar in kind if not degree. For instance, it is probable that hominin populations that experienced nutritional abundance grew faster and matured earlier at larger body sizes. Similarly, populations in contexts with relatively easy-to-exploit transitional foods may have weaned earlier than other groups, leading to the achievement of higher fertility rates. It is also interesting to consider that males were likely more strongly influenced by improvements in energetic conditions than were females, which could increase sexual size dimorphism in such environments. Conversely, in energetically marginal circumstances, maturation was likely delayed, dimorphism reduced, and body size and growth rates diminished. Building on this theme, we conclude by considering a final question with central importance to attempts to reconstruct hominin evolution and to interpret

the fossil record: what is the relationship between a trait's plasticity and its genetic evolution via natural selection?

Did Plasticity Lead the Way in Human Evolution?

While biological anthropologists have long considered the role of phenotypic and developmental plasticity as a means of adaptation and a source of within-species variation, recent theoretical and empirical work in evolutionary biology has emphasized the role of plasticity as an influence on the pace of evolutionary change and speciation (Kuzawa 2012; West-Eberhard 2003). Classically, it was often assumed that plasticity decouples genotypes from the specifics of phenotypes, which could reduce the strength of selection on underlying genetic variants (see Ghalambor et al. 2007). Contrary to this perspective, studies have shown that at least in some instances, the most phenotypically and developmentally plastic traits can evolve most rapidly (Stearns 1983; Wund et al. 2008). For example, after it was introduced to Hawaii, evolution of the mosquito fish has been most rapid for traits that exhibited the greatest plasticity (Stearns 1983). There is similar evidence that the diversification of fish species in the Canadian lakes created after the retreat of the Laurentide Ice Sheet was facilitated by plasticity (Robinson and Parsons 2002). Recent evolutionary theory is providing insights into how plasticity can accelerate rather than dampen the pace of genetic adaptation (West-Eberhard 2003): when novel environments first induce phenotypes via developmental plasticity, plasticity serves as the source of raw phenotypic variability on which natural selection then acts to shape subsequent genetic adaptation.

For phenotypic plasticity to "lead the way" and facilitate genetic evolution, several steps must occur, with a typical scenario involving the following: (1) an organism or population moves into a novel environment or experiences a change in an existing environment, (2) plasticity facilitates accommodation to the novel conditions by improving the "fit" between phenotype and environment, (3) the genetic architecture of this newly expressed phenotypic variation is then modified by natural selection to improve on the initially plastic phenotype or to increase the efficiency with which the phenotype is produced.

A growing list of studies provides evidence that plasticity can serve as an important source of phenotypic alternatives that are then filtered by natural selection and stabilized through genetic change. In an experiment designed to assess the degree of developmental plasticity of jaw morphology (Aubret and Shine 2009), tiger snakes from populations recently introduced to island environments (where larger jaw sizes are favored because of larger prey) showed a greater capacity for plasticity to match head growth to prey size. Snake populations with longer histories on the island grew larger head sizes regardless of the size of prey consumed during growth and development, showing that the initial capacity for plasticity was replaced by trait fixation after many generations of consistent selection for larger jaws. Other studies illustrate how developmental mechanisms underlying such adaptively plastic phenotypes can provide the substrate for later species divergence following longer periods of niche specialization. Among species of spadefoot toads, between-species diversity largely traces to ancestral larval plasticity in response to climactic conditions (Gomez-Mestre and Buchholz 2006). Similarly, a study of developmental morphology among three-spined sticklebacks found that the phenotypes of divergent freshwater species mirrored patterns of environmentally induced developmental variation in the marine species, an extant representative of the ancestor to the freshwater species, suggesting that plasticity provided the developmental alternatives from which the various freshwater species diverged as they moved into novel environments with different prey types (Wund et al. 2008).

Each of these examples illustrates how species diversity can originate from ancestrally shared patterns of plasticity among populations exposed to distinct environmental conditions. This likely reflects gradual genetic improvement of the induced phenotype via natural selection, which can take the form of fixation (loss of plasticity), as a shift in the underlying genetic architecture of the reaction norm (Ghalambor et al. 2007; Price, Qvarnström, and Irwin 2003), or as a compensation for some of the physiologic or other costs associated with the induced phenotype (Storz, Scott, and Cheviron 2010).

By this reasoning, the various modes of adaptation (fig. 1) do not simply cover different timescales of variability but may also represent a sequence of evolutionary change with less durable nongenomic modes of biological adaptation allowing appropriate phenotypic adjustments to novel conditions which are eventually superseded by more durable and efficient genetic accommodation (arrows, fig. 1; West-Eberhard 2003). Indeed, this idea that plasticity-induced phenotypic variants allow organisms to cope with environmental and behavioral novelty and that more durable genetic change might only later and more gradually follow has a long history (Baldwin 1896; Waddington 1953) and has recently been the focus of rekindled attention among evolutionary biologists (Sarkar 1999; West-Eberhard 2003).

Some examples of plasticity "leading the way" are intuitive. For instance, the biomechanical sensitivity of skeletal development shows that behavioral change is accompanied by changes in trabecular alignment and diaphyseal robustness (Pearson and Lieberman 2004). The capacity for more dramatic plasticity-induced realignment of musculoskeletal elements is illustrated by examples of quadrupedal animals born without forelegs that facultatively adopt bipedal locomotion (West-Eberhard 2003:51–54). Similarly, Japanese macaques trained to perform by walking upright exhibit a humanlike gait (Hirasaki et al. 2004), which is facilitated in part by similar changes in bone morphology (Nakatsukasa and Hayama 1991, cited in Hirasaki et al. 2004). In light of this work, it seems

likely that the gradual adoption of locomotor changes among early hominins was first a behavioral innovation that led to plasticity-based developmental changes in skeletal morphology (Hirasaki et al. 2004) that were gradually and incrementally fixed by genetic evolution (West-Eberhard 2005).

We noted that developmental adaptation in lung volume allows populations raised at high altitude to cope with hypoxia without having to mobilize homeostatic responses. While developmental adaptation of this type is well documented (Frisancho 1977; Moore, Niermeyer, and Zamudio 1998), populations with long histories living at high altitude show evidence for genetic adaptation to hypoxia (Beall 2007). Some of these adjustments appear to compensate for some of the costs associated with plasticity-induced phenotypes (Storz, Scott, and Cheviron 2010), demonstrating how developmental adaptation is not only engaged before genetic change but may lead the way for gradual fixation of more durable genetic adaptations.

Extrapolating from these cases, we speculate that similar principles may apply to the evolution of human life history traits. As one example, take the human Pygmy phenotype found in populations inhabiting environments characterized by low nutritional sufficiency (Shea and Bailey 1996) and high unavoidable mortality (Migliano and Guillon 2012; Migliano, Vinicius, and Lahr 2007; Walker et al. 2006*a*). Because a slow growth rate is a well-described response to low nutritional availability (Eveleth and Tanner 1990) while early maturity may be driven by cues signaling high unavoidable mortality risk (Ellis et al. 2009), features of this phenotype were likely induced first by developmental plasticity within individuals who moved into these ecologies. Evidence for genetic contributions to short stature in these populations (Pickrell et al. 2009) suggests that phenotypes induced by plasticity were eventually accommodated by selection operating on novel growth-regulating mutations.

If some plastic traits have the potential to evolve most rapidly under selection pressure (Stearns 1983; West-Eberhard 2003), it is interesting to consider the role of development in the evolution of the derived plastic features of the human life history. Take, for instance, human longevity (Gurven and Kaplan 2007; Hawkes et al. 1998; Kaplan et al. 2000). The delay in senescent processes that extend the human life span by several decades beyond that of other extant great apes likely required increasing maintenance expenditures (Hawkes 2003; Kaplan, Lancaster, and Robson 2003; Kaplan et al. 2000) that almost certainly required reduced expenditure in other domains, such as reproduction (Hawkes et al. 1998; Kirkwood and Rose 1991). In humans, any reduction in maternal reproductive effort is likely facilitated by early weaning of dependent infants from maternal metabolic investment (breast milk) and the early introduction of specially prepared foods often provided by alloparents (Sellen 2006, 2007).

Just as the impetus to adopt bipedal locomotion was almost surely behavioral before genetic adaptation could gradually occur, so too was early weaning likely first a behavioral decision. Early weaning facilitated by complementary feeding likely "freed up" energy for increased maintenance expenditures, allowing investment in more durable and long-lived somas. Consistent with this perspective, the longest human life spans tend to accompany reduced reproductive expenditure as reflected in lower fertility rate or completed family size (e.g., Doblhammer and Oeppen 2003; Gagnon et al. 2009; Jasienska 2009; but see Le Bourg 2007). By analogy, we speculate that behaviorally driven decreases in reproductive effort facilitated the initial expression of longer-lived phenotypes, made possible through increased maintenance effort, that were later genetically accommodated by natural selection as a species-defining trait over a longer, evolutionary time frame. This sequence of changes contrasts from scenarios posited previously in discussions of the adaptive evolution of human longevity, which often implicitly or explicitly assume that genetic changes that favor increased longevity would have initiated this life history pattern (e.g., Hawkes et al. 1998).

The extensive developmental plasticity in human life history traits reviewed here gains new importance in light of evidence that plasticity can influence the direction and pace of evolutionary change. Many of the traits that differentiate the human life history from that of other primates and great apes—including slow growth rate, early weaning, delayed maturity, high fertility, and perhaps even long life span—demonstrate phenotypic variation that traces to developmentally or behaviorally mediated plasticity in response to environmental factors such as nutrition and cues of unavoidable mortality. We have sketched some of the observations that lead us to hypothesize that this environmentally induced phenotypic variation likely preceded and ultimately facilitated genetic adaptations that gradually stabilized the life history characteristics that help define our species. We hope that this review helps stimulate interest in the broader insights that developmental plasticity may provide into the diversification and evolution of genus *Homo*, including the lineage that led eventually to modern *Homo sapiens*.

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References Cited

Adair, Linda S. 2001. Size at birth predicts age at menarche. *Pediatrics* 107: e59. http://pediatrics.aappublications.org/content/107/4/e59.

Altmann, Jeanne, and Susan C. Alberts. 2005. Growth rates in a wild primate population: ecological influences and maternal effects. *Behavioral Ecology and Sociobiology* 57:490–501.

Altmann, Jeanne, Dale Schoeller, Stuart A. Altmann, Philip Muruthi, and Robert M. Sapolsky. 1993. Body size and fatness of free living baboons reflect food availability and activity levels. *American Journal of Primatology* 30:149–161.

Antón, Susan C. 2003. Natural history of *Homo erectus*. *American Journal of Physical Anthropology* 122:126–170.

Antón, Susan C., Fred Spoor, Connie D. Fellmann, and Carl C. Swisher III. 2007. Defining *Homo erectus*: size considered. In *Handbook of paleoanthropology*, vol. 3. Winfried Henke, Ian Tattersall, and Thorolf Hardt, eds. Pp. 1655–1693. New York: Springer.

Aubret, Fabien, and Richard Shine. 2009. Genetic assimilation and the post-colonization erosion of phenotypic plasticity in island tiger snakes. *Current Biology* 19:1932–1936.

Baldwin, James M. 1896. A new factor in evolution. *American Naturalist* 30: 441–451.

Bateson, Patrick. 2001. Fetal experience and good adult design. *International Journal of Epidemiology* 30:928–934.

Beall, Cynthia M. 2007. Two routes to functional adaptation: Tibetan and Andean high-altitude natives. *Proceedings of the National Academy of Sciences of the USA* 104:8655–8660.

Belsky, Jay, Laurence Steinberg, and Patricia Draper. 1991. Childhood experience, interpersonal development, and reproductive strategy: an evolutionary theory of socialization. *Child Development* 62:647–670.

Belsky, Jay, Laurence Steinberg, Renate M. Houts, and Bonnie L. Halpern-Felscher. 2010. The development of reproductive strategy in females. *Developmental Psychology* 46:120–128.

Billewicz, W. Z., and Ian A. McGregor. 1981. The demography of two West African (Gambian) villages, 1951–75. *Journal of Biosocial Science* 13:219–240.

Bogin, Barry. 1999. Evolutionary perspective on human growth. *Annual Review of Anthropology* 28:109–153.

Bogin, Barry, and B. Holly Smith. 1996. Evolution of the human life cycle. *American Journal of Human Biology* 8:703–716.

Burton, Linda. 1990. Teenage childbearing as an alternative life-course strategy in multigeneration black families. *Human Nature* 1:123–143.

Charnov, Eric L. 1993. *Life history invariants: some explanations of symmetry in evolutionary ecology*. Oxford: Oxford University Press.

Charnov, Eric L., and David Berrigan. 1993. Why do female primates have such long lifespans and so few babies? or life in the slow lane. *Evolutionary Anthropology: Issues, News, and Reviews* 1:191–194.

Chisholm, James S. 1993. Death, hope, and sex: life-history theory and the development of reproductive strategies. *Current Anthropology* 34:1–12.

Chisholm, James S., Julie Quinlivan, Rodney Petersen, and David Coall. 2005. Early stress predicts age at menarche and first birth, adult attachment, and expected lifespan. *Human Nature* 16:233–265.

Cho, Geum Joon, Hyun Tae Park, Jung Ho Shin, Jun Young Hur, Young Tae Kim, Sun Haeng Kim, Kyu Wan Lee, and Tak Kim. 2009. Age at menarche in a Korean population: secular trends and influencing factors. *European Journal of Pediatrics* 169:89–94.

Coall, David A., and James S. Chisholm. 2003. Evolutionary perspectives on pregnancy: maternal age at menarche and infant birth weight. *Social Science and Medicine* 57:1771–1781.

———. 2010. Reproductive development and parental investment during pregnancy: moderating influence of mother's early environment. *American Journal of Human Biology* 22:143–153.

Doblhammer, Gabriele, and Jim Oeppen. 2003. Reproduction and longevity among the British peerage: the effect of frailty and health selection. *Proceedings of the Royal Society B* 270:1541–1547.

Draper, Patricia, and Henry Harpending. 1982. Father absence and reproductive strategy: an evolutionary perspective. *Journal of Anthropological Research* 38:255–273.

Edelman, Gerald M. 1993. Neural Darwinism: selection and reentrant signaling in higher brain function. *Neuron* 10:115–125.

Eisenberg, Dan, M. Geoffrey Hayes, and Christopher W. Kuzawa. 2012. Delayed paternal age of reproduction in humans is associated with longer telomeres across two generations of descendants. *Proceedings of the National Academy of Sciences of the USA* 109:10251–10256.

Ellis, Bruce J. 2004. Timing of pubertal maturation in girls: an integrated life history approach. *Psychological Bulletin* 130:920–958.

Ellis, Bruce J., Aurelio Figueredo, Barbara Brumbach, and Gabriel Schlomer. 2009. Fundamental dimensions of environmental risk. *Human Nature* 20: 204–268.

Ellis, Bruce J., and Judy Garber. 2000. Psychosocial antecedents of variation in girls' pubertal timing: maternal depression, stepfather presence, and marital and family stress. *Child Development* 71:485–501.

ESHRE Capri Workshop Group. 2010. Europe the continent with the lowest fertility. *Human Reproduction Update* 16:590–602.

Eveleth, Phyllis B., and James M. Tanner. 1976. *Worldwide variation in human growth*. New York: Cambridge University Press.

———. 1990. *Worldwide variation in human growth*. 2nd edition. New York: Cambridge University Press.

Frisancho, A. Roberto. 1977. Developmental adaptation to high altitude hypoxia. *International Journal of Biometeorology* 21:135–146.

Gadgil, Madhav, and William H. Bossert. 1970. Life historical consequences of natural selection. *American Naturalist* 104:1–24.

Gagnon, Alan, Ken R. Smith, Marc Tremblay, Hélène Vézina, Paul-Philippe Paré, and Bertrand Desjardins. 2009. Is there a trade-off between fertility and longevity? a comparative study of women from three large historical databases accounting for mortality selection. *American Journal of Human Biology* 21:533–540.

Galdikas, Biruté M. F., and James W. Wood. 1990. Birth spacing patterns in humans and apes. *American Journal of Physical Anthropology* 83:185–191.

Gettler, Lee T., Thomas W. McDade, Alan B. Feranil, and Christopher W. Kuzawa. 2011. Longitudinal evidence that fatherhood decreases testosterone in human males. *Proceedings of the National Academy of Sciences of the USA* 108:16194–16199.

Ghalambor, Cameron K., John K. McKay, Scott P. Carroll, and David N. Reznick. 2007. Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Functional Ecology* 21:394–407.

Gluckman, Peter, and Mark A. Hanson. 2004. Living with the past: evolution, development, and patterns of disease. *Science* 305:1733–1736.

Gomez-Mestre, Ivan, and Daniel R. Buchholz. 2006. Developmental plasticity mirrors differences among taxa in spadefoot toads linking plasticity and diversity. *Proceedings of the National Academy of Sciences of the USA* 103: 19021–19026.

Gray, Sandra J., Brandi Wiebusch, and Helen A. Akol. 2004. Cross-sectional growth of pastoralist Karimojong and Turkana children. *American Journal of Physical Anthropology* 125:193–202.

Gurven, Michael, and Hillard Kaplan. 2007. Longevity among hunter-gatherers: a cross-cultural examination. *Population and Development Review* 33: 321–365.

Gurven, Michael, and Robert Walker. 2006. Energetic demand of multiple dependents and the evolution of slow human growth. *Proceedings of the Royal Society B* 273:835–841.

Habicht, Jean-Pierre, Charles Yarbrough, Reynaldo Martorell, Robert M. Malina, and Robert E. Klein. 1974. Height and weight standards for preschool children: how relevant are ethnic differences in growth potential? *Lancet* 303:611–615.

Hawkes, Kristen. 2003. Grandmothers and the evolution of human longevity. *American Journal of Human Biology* 15:380–400.

Hawkes, Kristen, James F. O'Connell, Nicholas G. Blurton Jones, Helen Alvarez, and Eric L. Charnov. 1998. Grandmothering, menopause, and the evolution of human life histories. *Proceedings of the National Academy of Sciences of the USA* 95:1336–1339.

Hill, Kim. 1993. Life history theory and evolutionary anthropology. *Evolutionary Anthropology* 2:78–88.

Hill, Kim, and A. Magdalena Hurtado. 1996. *Ache life history: the ecology and demography of a foraging people*. Hawthorne, NY: Aldine de Gruyter.

———. 2009. Cooperative breeding in South American hunter-gatherers. *Proceedings of the Royal Society B* 276:3863–3870.

Hill, Kim, and Hillard Kaplan. 1999. Life history traits in humans: theory and empirical studies. *Annual Review of Anthropology* 28:397–430.

Hirasaki, Eishi, Naomichi Ogihara, Yuzuru Hamada, Hiroo Kumakura, and Masato Nakatsukasa. 2004. Do highly trained monkeys walk like humans? a kinematic study of bipedal locomotion in bipedally trained Japanese macaques. *Journal of Human Evolution* 46:739–750.

Hrdy, Sarah. 2005. Comes the child before the man: how cooperative breeding and prolonged postweaning dependence shaped human potentials. In *Hunter-gatherer childhoods: evolutionary, developmental, and cultural perspectives*. Barry S. Hewlett and Michael E. Lamb, eds. Pp. 65–91. New Brunswick, NJ: Aldine Transaction.

———. 2009. *Mothers and others: the evolutionary origins of mutual understanding*. Cambridge, MA: Harvard University Press.

Hulanicka, Barbara, Lidia Gronkiewicz, and Jan Koniarek. 2001. Effect of familial distress on growth and maturation of girls: a longitudinal study. *American Journal of Human Biology* 13:771–776.

Humphrey, Louise T. 2010. Weaning behaviour in human evolution. *Seminars in Cell and Developmental Biology* 21:453–461.

Hurtado, A. Magdalena, Carol A. Lambourne, Kim Hill, and Karen Kessler.

2006. The public health implications of maternal care trade-offs. *Human Nature* 17:129–154.

Ibáñez, Lourdes, Angela Ferrer, Marcia Victoria Marcos, Francisco Rodriguez Hierro, and Francis de Zegher. 2000. Early puberty: rapid progression and reduced final height in girls with low birth weight. *Pediatrics* 106:e72. http://pediatrics.aappublications.org/content/106/5/e72.full.

Jasienska, Grazyna. 2009. Reproduction and lifespan: trade-offs, overall energy budgets, intergenerational costs, and costs neglected by research. *American Journal of Human Biology* 21:524–532.

Jost, Alfred. 1961. The role of fetal hormones in prenatal development. *Harvey Lectures* 55:201–226.

Kaplan, Hillard, Kim Hill, Jane Lancaster, and A. Magdalena Hurtado. 2000. A theory of human life history evolution: diet, intelligence, and longevity. *Evolutionary Anthropology: Issues, News, and Reviews* 9:156–185.

Kaplan, Hillard, Jane Lancaster, and Arthur Robson. 2003. Embodied capital and the evolutionary economics of the human life span. *Population and Development Review* 29:152–182.

Karaolis-Danckert, Nadina, Anette E. Buyken, Antje Sonntag, and Anja Kroke. 2009. Birth and early life influences on the timing of puberty onset: results from the DONALD (Dortmund nutritional and anthropometric longitudinally designed) study. *American Journal of Clinical Nutrition* 90:1559–1565.

Karlberg, Johan. 1989. A biologically-oriented mathematical model (ICP) for human growth. *Acta Paediatrica Scandinavica* 350(suppl.):70–94.

Kirkwood, Thomas B. L., and Michael R. Rose. 1991. Evolution of senescence: late survival sacrificed for reproduction. *Philosophical Transactions of the Royal Society B* 332:15–24.

Knott, Cheryl. 2001. Female reproductive ecology of the apes. In *Reproductive ecology and human evolution*. Peter T. Ellison, ed. Pp. 429–463. New Brunswick, NJ: Aldine Transaction.

Kramer, Karen L. 2010. Cooperative breeding and its significance to the demographic success of humans. *Annual Review of Anthropology* 39:417–436.

Kramer, Karen L., and Peter T. Ellison. 2010. Pooled energy budgets: resituating human energy-allocation trade-offs. *Evolutionary Anthropology: Issues, News, and Reviews* 19:136–147.

Kuzawa, Christopher W. 1998. Adipose tissue in human infancy and childhood: an evolutionary perspective. *American Journal of Physical Anthropology* 107:177–209.

———. 2001. Maternal nutrition, fetal growth, and cardiovascular risk in Filipino adolescents. PhD dissertation, Emory University, Atlanta.

———. 2005. Fetal origins of developmental plasticity: are fetal cues reliable predictors of future nutritional environments? *American Journal of Human Biology* 17:5–21.

———. 2007. Developmental origins of life history: growth, productivity, and reproduction. *American Journal of Human Biology* 19:654–661.

———. 2008. The developmental origins of adult health: intergenerational inertia in adaptation and disease. In *Evolution and health*. Wenda Trevathan, E. O. Smith, and James J. McKenna, eds. Pp. 325–349. Oxford: Oxford University Press.

———. 2012. Why evolution needs development, and medicine needs evolution. *International Journal of Epidemiology* 41:223–229.

Kuzawa, Christopher W., Thomas W. McDade, Linda S. Adair, and Nanette Lee. 2010. Rapid weight gain after birth predicts life history and reproductive strategy in Filipino males. *Proceedings of the National Academy of Sciences of the USA* 107:16800–16805.

Kuzawa, Christopher W., and Ivy L. Pike. 2005. Introduction. *American Journal of Human Biology* 17:1–4.

Kuzawa, Christopher W., and Elizabeth A. Quinn. 2009. Developmental origins of adult function and health: evolutionary hypotheses. *Annual Review of Anthropology* 38:131–147.

Kuzawa, Christopher W., and Zaneta M. Thayer. 2011. Timescales of human adaptation: the role of epigenetic processes. *Epigenomics* 3:221–234.

Lasker, Gabriel W. 1969. Human biological adaptability. *Science* 166:1480–1486.

Le Bourg, Éric. 2007. Does reproduction decrease longevity in human beings? *Ageing Research Reviews* 6:141–149.

Lee, Phyllis C. 1996. The meanings of weaning: growth, lactation, and life history. *Evolutionary Anthropology: Issues, News, and Reviews* 5:87–98.

Lee, Ronald D. 2003. Rethinking the evolutionary theory of aging: transfers, not births, shape senescence in social species. *Proceedings of the National Academy of Sciences of the USA* 100:9637–9642.

Leidy Sievert, Lynnette. 2006. *Menopause: a biocultural perspective*. New Brunswick, NJ: Rutgers University Press.

Low, Bobbi S., Ashley Hazel, Nicholas Parker, and Kathleen B. Welch. 2008. Influences on women's reproductive lives: unexpected ecological underpinnings. *Cross-Cultural Research* 42:201–219.

Martorell, Reynaldo. 1995. Results and implications of the INCAP follow-up study. *Journal of Nutrition* 125(suppl.):1127S–1138S.

McDade, Thomas W. 2003. Life history theory and the immune system: steps toward a human ecological immunology. *American Journal of Physical Anthropology* 122:100–125.

McIntyre, Matthew H., and Pamela M. Kacerosky. 2011. Age and size at maturity in women: a norm of reaction? *American Journal of Human Biology* 23:305–312.

Metzger, Boyd E., Lynn P. Lowe, Alan R. Dyer, Elisabeth R. Trimble, Brian Sheridan, Moshe Hod, Rony Chen, et al. 2009. Hyperglycemia and adverse pregnancy outcome (HAPO) study: associations with neonatal anthropometrics. *Diabetes* 58:453–459.

Migliano, Andrea Bamberg, and Myrtille Guillon. 2012. The effects of mortality, subsistence, and ecology on human adult height and implications for *Homo* evolution. *Current Anthropology* 53(suppl. 6):SXXX–SXXX.

Migliano, Andrea B., Lucio Vinicius, and Marta Mirazón Lahr. 2007. Life history trade-offs explain the evolution of human Pygmies. *Proceedings of the National Academy of Sciences of the USA* 104:20216–20219.

Moore, Lorna, Susan Niermeyer, and Stacy Zamudio. 1998. Human adaptation to high altitude: regional and life-cycle perspectives. *Yearbook of Physical Anthropology* 107:25–64.

Nakatsukasa, Masato, and Shin-Ichi Hayama. 1991. Structural strength of the femur of bipedally trained macaque. *Journal of the Anthropological Society of Nippon* 99:289–296.

Nettle, Daniel. 2010. Dying young and living fast: variation in life history across English neighborhoods. *Behavioral Ecology* 21:387–395.

Nettle, Daniel, David A. Coall, and Thomas E. Dickins. 2010. Birthweight and paternal involvement predict early reproduction in British women: evidence from the National Child Development Study. *American Journal of Human Biology* 22:172–179.

———. 2011. Early-life conditions and age at first pregnancy in British women. *Proceedings of the Royal Society B* 278:1721–1727.

Nyberg, Colleen, William Leonard, Susan Tanner, Thomas McDade, Tomas Huanca, Ricardo Godoy, and TAPS Bolivia Study Team. Forthcoming. Diurnal cortisol rhythms and child growth: exploring the life history consequences of HPA activation among the Tsimané. *American Journal of Human Biology*.

Ong, Ken K., Pauline Emmett, Kate Northstone, Jean Golding, Imogen Rogers, Andrew R. Ness, Jonathan C. Wells, and David B. Dunger. 2009. Infancy weight gain predicts childhood body fat and age at menarche in girls. *Journal of Clinical Endocrinology and Metabolism* 94:1527–1532.

Pearson, Osbjorn M., and Daniel E. Lieberman. 2004. The aging of Wolff's "law": ontogeny and responses to mechanical loading in cortical bone. *American Journal of Physical Anthropology* 125:63–99.

Pembrey, Marcus E. 2010. Male-line transgenerational responses in humans. *Human Fertility* 13:268–271.

Perry, George H., and Nathaniel J. Dominy. 2009. Evolution of the human Pygmy phenotype. *Trends in Ecology & Evolution* 24:218–225.

Pesonen, Anu-Katrina, Katri Räikkönen, Kati Heinonen, Eero Kajantie, Tom Forsén, and Johan G. Eriksson. 2008. Reproductive traits following a parent-child separation trauma during childhood: a natural experiment during World War II. *American Journal of Human Biology* 20:345–351.

Phoenix, Charles, Robert Goy, Arnold Gerall, and William Young. 1959. Organizing action of prenatally administered testosterone propionate on the tissues mediating mating behavior in the female guinea pig. *Endocrinology* 65:369–382.

Pickrell, Joseph K., Graham Coop, John Novembre, Sridhar Kudaravalli, Jun Z. Li, Devin Absher, Balaji S. Srinivasan, et al. 2009. Signals of recent positive selection in a worldwide sample of human populations. *Genome Research* 19:826–837.

Prentice, Sarah, Antony J. Fulford, Landing M. A. Jarjou, Gail R. Goldberg, and Ann Prentice. 2010. Evidence for a downward secular trend in age of menarche in a rural Gambian population. *Annals of Human Biology* 37: 717–721.

Price, Trevor D., Anna Qvarnström, and Darren E. Irwin. 2003. The role of phenotypic plasticity in driving genetic evolution. *Proceedings of the Royal Society B* 270:1433–1440.

Promislow, Daniel E. L., and Paul H. Harvey. 1990. Living fast and dying young: a comparative analysis of life-history variation among mammals. *Journal of Zoology* 220:417–437.

Proos, Lemm A., Yngve Hofvander, and Torsten Tuvemo. 1991. Menarcheal age and growth pattern of Indian girls adopted in Sweden. 1. Menarcheal age. *Acta Paediatrica Scandinavica* 80:852–858.

Quinlan, Robert J. 2003. Father absence, parental care, and female reproductive development. *Evolution and Human Behavior* 24:376–390.

———. 2007. Human parental effort and environmental risk. *Proceedings of the Royal Society B* 274:121–125.

Robinson, Beren W., and Kevin J. Parsons. 2002. Changing times, spaces, and faces: tests and implications of adaptive morphological plasticity in the fishes of northern postglacial lakes. *Canadian Journal of Fisheries and Aquatic Sciences* 59:1819–1833.

Robson, Shannen L., Carel P. van Schaik, and Kristen Hawkes. 2006. The derived features of human life history. In *The evolution of human life history*. Kristen Hawkes and Richard R. Paine, eds. Pp. 17–44. Santa Fe, NM: School of American Research Press.

Sarkar, Sahotra. 1999. From the Reaktionsnorm to the adaptive norm: the norm of reaction, 1909–1960. *Biology and Philosophy* 14:235–252.

Sellen, Daniel W. 2006. Lactation, complementary feeding, and human life history. In *The evolution of human life history*. Kristen Hawkes and Richard R. Paine, eds. Pp. 155–196. Santa Fe, NM: School of American Research Press.

———. 2007. Evolution of infant and young child feeding: implications for contemporary public health. *Annual Review of Nutrition* 27:123–148.

Shea, Brian T., and Robert C. Bailey. 1996. Allometry and adaptation of body proportions and stature in African Pygmies. *American Journal of Physical Anthropology* 100:311–340.

Stearns, Stephen C. 1983. The evolution of life-history traits in mosquitofish since their introduction to Hawaii in 1905: rates of evolution, heritabilities, and developmental plasticity. *American Zoologist* 23:65–75.

———. 1989. Trade-offs in life-history evolution. *Functional Ecology* 3:259–268.

———. 1992. *The evolution of life histories*. New York: Oxford University Press.

Stearns, Stephen C., and Jacob C. Koella. 1986. The evolution of phenotypic plasticity in life-history traits: predictions of reaction norms for age and size at maturity. *Evolution* 40:893–913.

Sterling, Peter. 2004. Principles of allostasis. In *Allostasis, homeostasis, and the costs of adaptation*. Jay Shulkin, ed. Pp. 2–36. New York: Cambridge University Press.

Stinson, Sara. 1985. Sex differences in environmental sensitivity during growth and development. *American Journal of Physical Anthropology* 28:123–147.

Storz, Jay F., Graham R. Scott, and Zachary A. Cheviron. 2010. Phenotypic plasticity and genetic adaptation to high-altitude hypoxia in vertebrates. *Journal of Experimental Biology* 213:4125–4136.

Tanner, James Mourilyan. 1962. *Growth at adolescence, with a general consideration of the effects of hereditary and environmental factors upon growth and maturation from birth to maturity*. Oxford: Blackwell.

Teilmann, Grete, Carsten B. Pedersen, Niels E. Skakkebaek, and Tina K. Jensen. 2006. Increased risk of precocious puberty in internationally adopted children in Denmark. *Pediatrics* 118:391–399.

Tither, Jacqueline M., and Bruce J. Ellis. 2008. Impact of fathers on daughters' age at menarche: a genetically and environmentally controlled sibling study. *Developmental Psychology* 44:1409–1420.

Victora, Cesar G., Linda Adair, Caroline Fall, Pedro C. Hallal, Reynaldo Martorell, Linda Richter, and Harshpal Singh Sachdev. 2008. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 371:340–357.

Waddington, Conrad H. 1953. Genetic assimilation of an acquired character. *Evolution* 7:118–126.

Walker, Robert S., Michael Gurven, Oskar Burger, and Marcus J. Hamilton. 2008. The trade-off between number and size of offspring in humans and other primates. *Proceedings of the Royal Society B* 275:827–834.

Walker, Robert S., Michael Gurven, Kim Hill, Andrea Migliano, Napolean Chagnon, Roberta D. Souza, Gradimir Djurovic, et al. 2006*a*. Growth rates and life histories in twenty-two small-scale societies. *American Journal of Human Biology* 18:295–311.

Walker, Robert S., Kim Hill, Oskar Burger, and A. Magdalena Hurtado. 2006*b*. Life in the slow lane revisited: ontogenetic separation between chimpanzees and humans. *American Journal of Physical Anthropology* 129:577–583.

Wells, Jonathan C. K. 2003. The thrifty phenotype hypothesis: thrifty offspring or thrifty mother? *Journal of Theoretical Biology* 221:143–161.

———. 2007. The thrifty phenotype as an adaptive maternal effect. *Biological Reviews* 82:143–172.

Wells, Jonathan C. K., and Jay T. Stock. 2007. The biology of the colonizing ape. *Yearbook of Physical Anthropology* 134:191–222.

West-Eberhard, Mary Jane. 2003. *Developmental plasticity and evolution*. New York: Oxford University Press.

———. 2005. Developmental plasticity and the origin of species differences. *Proceedings of the National Academy of Sciences of the USA* 102:6543–6549.

Williams, George C. 1966. Natural selection, the costs of reproduction, and a refinement of Lack's principle. *American Naturalist* 100:687–690.

Wilson, Margo, and Martin Daly. 1997. Life expectancy, economic inequality, homicide, and reproductive timing in Chicago neighbourhoods. *British Medical Journal* 314:1271–1274.

Worthman, Carol M. 1999. Epidemiology of human development. In *Hormones, health, and behavior*. Catherine Panter-Brick and Carol M. Worthman, eds. Pp. 47–104. Cambridge: Cambridge University Press.

Wund, Matthew A., John A. Baker, Brendan Clancy, Justin L. Golub, and Susan A. Foster. 2008. A test of the "flexible stem" model of evolution: ancestral plasticity, genetic accommodation, and morphological divergence in the threespine stickleback radiation. *American Naturalist* 172:449–462.