Hum Nat (2009) 20:204–268
DOI 10.1007/s12110-009-9063-7

Fundamental Dimensions of Environmental Risk

The Impact of Harsh versus Unpredictable Environments on the Evolution and Development of Life History Strategies

Bruce J. Ellis · Aurelio José Figueredo · Barbara H. Brumbach · Gabriel L. Schlomer

Published online: 22 April 2009

Abstract The current paper synthesizes theory and data from the field of life history (LH) evolution to advance a new developmental theory of variation in human LH strategies. The theory posits that clusters of correlated LH traits (e.g., timing of puberty, age at sexual debut and first birth, parental investment strategies) lie on a slow-to-fast continuum; that harshness (externally caused levels of morbidity-mortality) and unpredictability (spatial-temporal variation in harshness) are the most fundamental environmental influences on the evolution and development of LH strategies; and that these influences depend on population densities and related levels of intraspecific competition and resource scarcity, on age schedules of mortality, on the sensitivity of morbidity-mortality to the organism's resource-allocation decisions, and on the extent to which environmental fluctuations affect individuals versus populations over short versus long timescales. These interrelated factors operate at evolutionary *and* developmental levels and should be distinguished because they exert distinctive effects on LH traits and are hierarchically operative in terms of primacy of influence. Although converging lines of evidence support core assumptions of the theory, many questions remain unanswered. This review demonstrates the value of applying a multilevel evolutionary-developmental approach to the analysis of a central feature of human phenotypic variation: LH strategy.

B. J. Ellis (✉) · G. L. Schlomer
John and Doris Norton School of Family and Consumer Sciences, University of Arizona, McClelland Park, 650 North Park Ave, Tucson, AZ 85721-0078, USA
e-mail: bjellis@email.arizona.edu

A. J. Figueredo
Department of Psychology, University of Arizona, Tucson, AZ, USA

B. H. Brumbach
Department of Psychology, Northern Arizona University, Flagstaff, AZ, USA

Keywords Life history theory · Reproductive strategies · Puberty · Sexual maturation · Sexual behavior · Parenting · Evolutionary psychology · Human development · Bet-hedging · Adaptive individual differences · Extrinsic mortality · Animal behavior

The integration of reproductive life history (LH) theory into the study of human behavioral development is becoming increasingly important and pervasive in the evolutionary and developmental psychology literatures (e.g., Belsky et al. 1991; Chisholm 1993; Davis and Were 2008; Del Giudice 2009; Ellis 2004; Figueredo et al. 2006b; Quinlan 2007; Walker et al. 2006b). This integration has been hampered, however, by patchy, inconsistent, and sometimes confusing usage of LH concepts in psychological research. Indeed, applications of LH theory to human development have regularly conflated the most fundamental dimensions of environmental variation and influence: harshness and unpredictability. The current paper synthesizes theory and data from the field of LH evolution (Charnov 1993; Roff 1992; Stearns 1992) to advance a new integrative framework for explaining variation in the development of human LH strategies. This framework is the first to articulate how environmental harshness and environmental unpredictability, concomitantly, have shaped the evolution and development of LH strategies.

Central to the study of human development are questions about variation in sexual and reproductive behavior: What leads some teenagers to initiate sexual activity before others and become pregnant at a young age? Why do some people engage in serial or multi-partner relationships while others form and maintain enduring pairbonds? Why do some parents deeply invest in their children's health and well-being while others neglect and abuse them? Of course, answers to these questions are of great interest to policymakers as well. Less widely recognized is that the topics just raised, as well as related ones (e.g., timing of puberty, family size), are also of central interest to evolutionary biologists, both those interested in differences between species and those concerned with variation within species. For these scientists, it is LH theory that serves as the touchstone for understanding, and one purpose of this paper is to highlight the significance of this framework for explaining contemporary human development.

As it turns out, substantial progress has been made in this regard across the past two decades, perhaps beginning with Draper and Harpending's (1988) provocative recasting of Freudian notions of early father influence on female sexuality, followed swiftly by Belsky et al.'s (1991) evolutionary theory of socialization, which recast much of contemporary developmental thinking about environmental influences on development in evolutionary-biological and specifically LH terms. A corpus of subsequent theoretical and empirical work (e.g., Bereczkei and Csanaky 2001; Chisholm 1993, 1999; Del Giudice 2009; Ellis 2004; Ellis and Essex 2007; Figueredo et al. 2006b; Vigil et al. 2005) has tested and extended these early models.

In much of this work, just as in much developmental inquiry not informed by an evolutionary perspective, a critical concern has been how environmental conditions judged to be "negative" or "problematic" affect important developmental processes and outcomes, such as early puberty, precocious sexual activity, and adolescent pregnancy and childbirth. Despite the indisputable contributions of this extant body of work, the central premise of the current paper is that greater insight and empirical

achievement can be realized by unpacking environmental conditions in a manner that is explicitly informed by LH theory. This involves (1) delineating the nature of harsh versus unpredictable environmental conditions, (2) articulating the principles that govern how LH traits (e.g., timing of puberty, age at sexual debut and first birth, mating and parenting strategies) evolve in response to environmental harshness and unpredictability, and (3) using these principles to model the effects of childhood harshness and unpredictability on the development of human LH traits. Given the complexity of this task, we proceed through the following intermediary steps:

1. *LH Trade-Offs and Strategies*. This first section provides an overview of LH theory. At one level, LH strategies are species-typical adaptations to past ecological conditions (i.e., natural selection shapes species-typical LH strategies in response to recurring selection pressures over evolutionary time); at another level, LH strategies vary within species as adaptations to variable ecological and developmental conditions. Thus, species-typical LH strategies often represent modal rather than fixed patterns of development, around which individuals vary.

    A. We review major resource allocation trade-offs faced by individuals over the life course and explain how the nature of these trade-offs depends on the opportunities and constraints that individuals face in their environments.
    B. We discuss the generalized patterns that have been observed in the coordinated directionality of these trade-offs—patterns that result in clusters of correlated LH traits that lie on a slow-to-fast continuum and underlie the coherence of LH strategies.
    C. We briefly summarize the evidence for systematic within-species variation in LH strategy across a diverse array of animal taxa. Thus, we extend LH theory to the study of individual differences, recognizing that adaptive variation in LH strategy, both between and within species, is generated by a combination of evolved genetic diversity and phenotypic plasticity in response to environmental influences.
2. *The Impact of Harsh versus Unpredictable Environments on the Evolution of LH Strategies*. The second section develops and evaluates adaptationist hypotheses about why some LH features have been maintained by natural selection instead of others. Employing both between- and within-species comparisons, this analysis focuses on delineating functional responses to both harsh and unpredictable environmental conditions. We employ animal data to establish principles, and then apply these principles to explain the evolution of the modal human LH strategy as well as adaptive variation around that mode. These principles form the foundation for subsequent hypotheses about the development of human LH strategies.

    A. We define environmental harshness as the rates at which external factors cause disability and death at each age in a population. LH theory ascribes primary importance to the effects of extrinsic morbidity-mortality (external sources of disability and death that are relatively insensitive to the adaptive decisions or strategies of the organism) on LH evolution. Density-dependent effects, such as resource scarcity, are considered in relation to extrinsic morbidity-mortality. We describe how these effects differ systematically depending on age schedules of mortality.

   B. We define environmental unpredictability as the rates at which environmental harshness varies over time and space. Environmental unpredictability favors the evolution of various bet-hedging strategies. We describe how the effects of environmental unpredictability on LH evolution differ depending on age schedules of mortality and the extent to which environmental fluctuations affect populations versus individuals.
   C. Based on West-Eberhard's (2003) model of developmental plasticity and evolution, we argue that the principles governing the effects of harsh versus unpredictable environments on the *evolution* of LH strategies afford hypotheses about the effects of harsh versus unpredictable environments on the *development* of LH strategies. This connection inextricably links the field of LH evolution to developmental science.

3. *The Impact of Harsh versus Unpredictable Environments on the Development of Human LH Strategies*. The third section develops and evaluates hypotheses about how human LH strategies develop over the individual life span and what forces shape their expression. This involves examining the effects of a broad range of rearing experiences and ecological contexts (e.g., parent-child relationships, peer relationships, neighborhood quality, socioeconomic status, parental transitions, residential mobility) on LH traits and related variables (e.g., growth rates, timing of puberty and sexual debut, number of sexual partners, adolescent pregnancy, family size, parenting styles).

   A. The principles guiding the effects of harsh environments on the evolution of LH strategies are applied to human development. We argue that energetic conditions and levels of extrinsic morbidity-mortality affect LH development in a hierarchical manner, with energetics forming a baseline for LH development and levels of extrinsic morbidity-mortality moving individuals around that baseline in a predictable manner.
   B. The evolutionary logic of bet-hedging is applied to human LH development under unpredictable environmental conditions. Special attention is paid to the effects of harsh versus unpredictable environments on patterns of parental investment.

4. *Summary and Conclusion*. The final section provides a summary of the major arguments presented in this paper. We conclude by highlighting the importance of integrating evolutionary and developmental approaches in the analysis of major environmental influences on LH strategies.

LH Trade-Offs and Strategies

Resource-Allocation Decisions

From an evolutionary perspective, the most fundamental task faced by all organisms is the successful utilization of resources—matter and energy harvested from the environment—in the service of survival and reproduction. Natural selection favors *resource allocation strategies* that optimize this utilization over the life course and

across varying ecological conditions. The goal of LH theory is to explain the evolution and development of these strategies.

The key units of analysis in LH theory are LH traits: characteristics that determine rates of reproduction and associated patterns of growth, aging, mating behavior, and parental investment. Age at sexual maturity, gestation period, birth weight, litter size, postnatal growth rates, breastfeeding duration, birth spacing, length of juvenile dependence (provisioning), level of parental investment per child, adult body size, and longevity are all LH traits. According to LH theory (Charnov 1993; Roff 1992; Stearns 1992), variation in LH traits results from trade-offs in distribution of resources to competing life functions: bodily maintenance, growth, and reproduction. Owing to structural and resource limitations, organisms cannot simultaneously maximize all components of fitness and instead are selected to make trade-offs that prioritize resource expenditures, so that greater investment in one domain occurs at the expense of investment in other competing domains. For example, resources spent on growth and development (e.g., later age at sexual maturity, larger adult body size) cannot be spent on current reproduction because producing offspring reduces somatic growth; thus, the benefits of a prolonged maturational period are traded off against the costs of delayed reproduction. Each trade-off constitutes a decision node in allocation of resources, and each decision node influences the next decision node (opening up some options, foreclosing others) in an unending chain over the life course. This chain of resource-allocation decisions—expressed in the development of a coherent, integrated suite of LH traits—constitutes the individual's LH strategy.

Because the costs and benefits of different LH trade-offs vary as a function of individual characteristics and local circumstances, optimal LH strategies vary across individuals within and between populations. These individual and population differences develop through a combination of genetic variation and phenotypic plasticity in response to environmental conditions. Natural selection favors mechanisms of phenotypic plasticity that enable organisms, within their species-typical range, to adjust LH strategies within their own lifetimes. These developmental mechanisms actually make LH trade-offs by selecting between or "making decisions" about alternative ways of distributing resources (Chisholm 1999). Selection favors mechanisms that, in response to socioecological conditions, trade off resources between growth, maintenance, and reproduction in ways that recurrently enhanced inclusive fitness during a species' evolutionary history. In this manner, individuals adapt LH strategies to local conditions. At the same time, however, many allelic variations are maintained within populations, biasing development toward different sets of LH trade-offs and increasing phenotypic diversity.

Trade-Offs between Maintenance and Growth

A central resource-allocation trade-off made in childhood, beginning in the prenatal period, is between maintenance and growth (for an extensive review, see Bogin et al. 2007). A baseline level of energy expenditure is needed to stay alive and maintain basic functioning (e.g., brain metabolism, digestion, immune function, cellular/DNA repair, pathogen and predator defenses). Maintenance involves all energy allocated to allaying mortality, and the quality and quantity of investments in maintenance determine age-specific mortality schedules (probabilities of death; Worthman 2003).

Above baseline investments in maintenance, children can allocate resources to growth (and eventually reproduction). Growth encompasses developmental processes and activities that increase physical size and sociocompetitive competencies. Growth thus includes development of information-processing capacities and acquisition of skills and knowledge as well as increases in body mass. Growth functions to increase energy capture rates per unit of time devoted to food acquisition or production (increasing energy availability for reproduction over the lifespan) and increases success in intrasexual competition for mates (Hill and Kaplan 1999).

The main focus of LH analysis in this area has been on physical growth: development of lean body mass, energy storage (fat), and physical activity during the growing years. An early decision node in the human life course is how much energy to devote to physical growth. As reviewed by Kuzawa (2005), faster growth means earlier maturation, larger adult size, and greater capacity in the future to produce large and resilient offspring. Fast growth has costs, however, as bigger individuals have higher total energy requirements (maintenance costs) and are thus more vulnerable to malnutrition and impairment of reproductive function during periods of negative energy balance (Kuzawa 2005). In response to energetic conditions experienced in utero (e.g., Worthman and Kuzara 2005) and in early childhood (e.g., Ellis 2004), organisms make trade-offs between maintenance and growth. These trade-offs involve setting growth trajectories that will determine maintenance costs over the life course. In humans, for example, poor maternal nutritional status and resource restriction in utero lead to diversion of resources away from growth toward maintenance of basic functioning. The resulting fetal growth restriction fosters development of a more energy-sparing (famine-resistant) phenotype that economizes body maintenance costs, leading to low energy availability, slower growth, delayed sexual maturation, low gonadal steroid production, small adult body size, and low fecundity (reviewed in Kuzawa 2005, 2008; see also Arendt 1997; Walker et al. 2006b). Thus, trade-offs made in the prenatal period between the demands of maintenance and growth shape developmental trajectories that strongly influence subsequent LH trade-offs and strategies.

Trade-Offs between Current and Future Reproduction

Trade-offs between maintenance and growth in childhood set the stage for the most fundamental LH trade-off: the trade-off between current and future reproduction. Effort put into reproducing now will use energy or resources that cannot be used or saved for future reproduction. The organism faces the risk of crossing an investment threshold, above which resources consumed in support of current reproduction would have produced better fitness returns if they had instead been allocated to future reproduction (Chisholm 1999). The costs of current reproduction may be paid in terms of reduced number, quality, or survival of future offspring, as well as reduced growth and survival of the parent.

Trade-Offs between Growth and Current Reproduction Central to the general LH problem of allocating resources between current and future reproduction is the trade-off between continued growth and onset of reproduction. Metabolically, an organism will need to choose between investing energy in the production of offspring versus in its own growth. Reproductive effort has many costs, including finding, attracting, and

retaining mates; producing offspring (sexual activity, production of gametes, gestation, parturition); and sustaining offspring and enhancing their quality (e.g., lactation, provisioning, parental care, protection, teaching, socialization). At a comparative species level, the human life course—characterized by lengthy infancy and juvenile periods prior to sexual maturation—constitutes an extreme example of an evolved trade-off favoring prolonged growth over early reproduction. The implicit assumption is that the benefits of large body size and accumulated skills and knowledge compensate for the reproductive opportunities lost through prolonged growth.

Trade-offs between growth and current reproduction are well-documented in research on adolescent childbearing. Adolescent mothers have a smaller pool of energetic resources to devote to production of offspring. Such mothers tend to be smaller and convert less of their weight gain during pregnancy to fetal weight gain than do adult mothers (Garn et al. 1986), experience higher rates of antenatal complications and mortality than do adult mothers, and their offspring are at increased risk of stillbirths, congenital abnormalities, prematurity, low birthweight, and retardation (Black and DeBlassie 1985; Furstenberg et al. 1989; Luster and Mittelstaedt 1993). At the same time, however, adolescent childbearing reduces the probability of death prior to first reproduction (shorter exposure to all sources of mortality), increases the total reproductive output of lineages through shorter generation times, and results in longer reproductive lifespans (Ellis 2004).

Trade-Offs between Survival and Current Reproduction Another core element of the current-future trade-off is allocation decisions between survival and current reproduction. An organism putting effort into its own survival (or into body maintenance or existing offspring) will necessarily be putting less into current reproduction, and vice versa. When salmon swim upstream to spawn and then die, they represent the ultimate trade-off of survival for current reproduction. In this case, so much energy is diverted away from body repair and maintenance toward reproduction that the organism simply dies. Although mammals do not employ this extreme strategy, there are relevant analogs. If resources are insufficient to both maintain fat reserves and lactate, then nursing mothers will divert energy away from fat deposition (an adaptation for winter survival) toward milk production. Consequently, among red deer, nursing mothers have significantly higher mortality rates in the winter than do non-nursing mothers (Clutton-Brock et al. 1982). The human equivalent, documented in historic upper-class British families, is a positive correlation between number of births and late-life mortality (Doblhammer and Oeppen 2003), after adjusting for variation in women's health and mortality during their childbearing years.[1] Trade-offs go the opposite direction as well, however, as

[1] Although LH theory unequivocally states that there is a trade-off between current reproduction and survival, the trade-offs may be difficult to detect in comparisons between individuals who differ in physical condition, access to resources, social support, and related factors. This is because a person who is in good physical condition and has ready access to food, shelter, and a supportive kin network may be able to grow up faster, achieve larger adult size, have more children, and produce higher-quality offspring than another person who is in poor condition and has meager resources and little kin support. These disparities often generate positive correlations between people in LH traits that are in fact negatively correlated within persons (e.g., number of births versus longevity). Consequently, unless women's health and socioeconomic conditions are controlled for, correlations between female life expectancy and offspring number in natural fertility populations do not reliably emerge (see Hurt et al. 2006).

stressful conditions often cause individuals to divert energy to survival at the expense of current reproduction. Elevated rates of early miscarriage among human mothers with high cortisol levels (Nepomnaschy et al. 2006) is evidence of such a trade-off, as is suppression of ovarian hormonal functioning among women experiencing negative energy balance (e.g., Ellison 2001).

Trade-Offs between Offspring Quality and Quantity

Closely related to current-future trade-offs are quality-quantity trade-offs. Energetic constraints, both developmentally and over evolutionary time, bias organisms toward investing in either a relatively small number of "high-quality" offspring or a relatively large number of "low-quality" offspring (Stearns 1992). Higher-quality offspring receive more investment per child than do lower-quality offspring and thus tend to have better health, developmental, and survival outcomes. The quality-quantity trade-off is pervasive in preindustrial societies, where larger family size has been linked to poorer growth and survival outcomes. Among the Shuar of Ecuador, for example, more children per household is associated with decreased childhood height, weight, and body fat (Hagen et al. 2006). Further, across many societies, higher numbers of offspring are associated with higher child mortality rates (Cristescu 1975; Crognier 1998; Kunstadter et al. 1992; Strassmann and Gillespie 2002; Syamala 2001). Higher offspring number, however, does not always translate into lower offspring survival (reviewed in Hagen et al. 2006; see also note 1), and the offspring quality-quantity trade-off in the lifetime reproductive success of humans has only been found to reliably occur under poor socioeconomic conditions (Borgerhoff Mulder 2000; Gillespie et al. 2008).

The nature of all of these LH trade-offs depends on the opportunities and constraints that an organism faces in its environment. These opportunities and constraints are primarily shaped by the ecology. Food supply, intrasexual competition, and extrinsic mortality hazards, together with the extent to which parental investment in offspring quality affects each of these factors (energy capture rates, success in intrasexual competition, mortality regimes), greatly influence the costs and benefits of different LH trade-offs. Mortality schedules are especially important because they determine the probability that an individual will survive to realize time-delayed benefits or suffer time-delayed costs (Worthman and Kuzara 2005).

In total, LH theory attempts to explain the evolution and development of the overarching resource-allocation trade-offs made by individuals over the life course: growth versus maintenance, current versus future reproduction, and offspring quality versus quantity. These trade-offs are expressed in integrated sets of LH traits that constitute the individual's LH strategy. The core assumption of LH theory is that, over evolutionary and developmental time, individuals and lineages systematically adjust LH strategies in response to the specific risks and opportunities afforded by the environment, and that these adjustments recurrently enhanced inclusive fitness over evolutionary history.

The Slow-to-Fast LH Continuum

A complete description of the particular combination of LH trade-offs made by an organism will lead to the characterization of its overall LH *strategy*. On the one

hand, species-typical LH strategies encompass the suite of modal LH trade-offs that a given species has converged on over its natural selective history; on the other hand, adaptive individual differences in these strategies encompass both genetic diversity and evolved mechanisms of phenotypic plasticity that allow individuals to conditionally adjust LH strategies in response to more local environmental opportunities and constraints. Ultimately, what makes one pattern of LH trade-offs more advantageous than another is largely based on differences in ecology or the environment.

In addition to these external constraints, however, there are also internal constraints involving the interrelations among different LH trade-offs. This is because many LH trade-offs are not functionally independent of one another. The selection of certain LH options over others in one domain affects the optimality of similar trade-offs in other domains. Consequently, LH traits tend to be correlated through optimized patterns of trade-offs that jointly contribute to the increased fitness of the organism. Therefore, an interdependent chain of LH "decision-nodes" over the life course determines allocation of resources to competing demands of growth, maintenance, and reproduction

For example, one coordinated LH strategy might be characterized by behaviors that reflect long-term planning, parental investment, and social investment in kin and non-kin (Figueredo et al. 2006b). If one added a LH trait such as short lifespan to the above suite of allocations, the strategy described would no longer be functionally coherent. All of the energy invested in the future would be wasted if the expected lifespan of the organism were shortened. The resulting mismatched strategy would *not* optimize the organism's inclusive fitness. A synchronized optimization of trade-offs is therefore at the heart of what constitutes a coherent and coordinated LH strategy.

Therefore, in addition to the trade-offs made at the level of specific LH traits, generalized patterns exist in the coordinated directionality of these trade-offs. These patterns give rise to clusters of correlated LH traits that lie on a continuum that can be described as "slow" to "fast."[2] As Kaplan and Gangestad (2005:73) have stated, "mammalian species on the fast end exhibit short gestation times, early reproduction, small body size, large litters, and high mortality rates, whereas species on the slow end have the opposite features." Slow-fast LH continua have been documented across diverse animal taxa, ranging from mammals (Oli 2004; Promislow and Harvey 1990) to birds (Saether and Bakke 2000), reptiles (Clobert et al. 1998), and insects (Blackburn 1991).

The presence of LH continua, however, does not imply that all LH traits can be arrayed on a single slow-fast dimension. Indeed, some species show a mixture of slow and fast traits (e.g., Kraus et al. 2005). In a comparative analysis of 267 mammalian species, Bielby et al. (2007) found that most of the variance in LH traits could be explained by two factors. The first factor represents differences between

[2] This slow-fast continuum has also sometimes been referred to in the literature as a quality-quantity continuum or the *r*-*K* continuum (e.g., Belsky et al. 1991; Rushton 1985).

mammalian species in trade-offs between current and future reproduction (i.e., reproductive timing): "At one end are species that, for their body size, mature quickly, give birth frequently, and wean their offspring early, while species at the other end have the opposite suite of traits" (2007:751). The second factor represents differences between species in trade-offs between offspring quality and quantity, "ranging from species that (for their size) give birth to large litters of small neonates after short gestations to species producing (for their size) small litters of large neonates after a long gestation" (2007:751).

Comparative analysis of primate LH strategies reveals a marked slow-fast continuum (Ross 1988), though it is unclear whether this continuum is best represented as one or two factors (see Bielby et al. 2007: Table 1). Whereas small prosimians achieve sexual maturity after less than a year of rapid growth and then produce litters of multiple young once or twice a year, large great apes achieve sexual maturity at 7–16 years of age and have singleton births that are spaced 4–8 years apart (Kappeler et al. 2003). As one of the great apes, humans are on the slow end of the slow-fast continuum, with a prolonged period of juvenile dependency, late age at onset of reproduction, and greater longevity than any other terrestrial mammal (for an extensive review, see Hawkes 2006). Some features of human LH deviate from the slow pattern, however, including relatively early age at weaning and short interbirth intervals. Determining why humans have this combination of slow and fast LH traits has been an important focus of LH analysis (e.g., Hawkes et al. 2003; Kaplan et al. 2000).

Systematic Within-Species Variation in LH Strategies

Although LH theory was originally proposed to explain systematic differences between species in patterns of development and reproduction, significant within-species variation in LH strategy is pervasive within many diverse taxa. This documented variation has led to increasing applications of LH theory to the study of human personality and individual differences (e.g., Belsky et al. 1991; Chisholm 1993; Del Giudice 2009; Ellis 2004; Figueredo et al. 2006b). As reviewed in the following sections, through a combination of evolutionary and developmental processes, individual differences in LH strategies become adaptively coordinated with levels of harshness and unpredictability in local environments. In this section we introduce different forms and causes of within-species variation in LH strategy, both within and between populations.

Between-Population Variation in LH Strategy

Between-population variation in LH strategy can result from spatially or temporally separated populations experiencing different developmental conditions, different selection regimes, or various combinations thereof. There are numerous examples of divergent LH strategies between spatially separated populations resulting from exposure to different selection pressures. The clearest illustration of this phenomenon is found in comparisons between mainland and island populations that have been exposed to different levels of predation and food availability (see extended discussion below, "*r* Selection: The Case of Large

Herbivores on Islands"; "*K* Selection: The Island Syndrome in Rodents and Possums").

Variation between populations in LH traits can also arise from different developmental exposures to environmental conditions. In different populations of beavers, for example, age and size at sexual maturation and reproduction are associated with different degrees of exploitation by humans, with higher human predation favoring faster development and reproduction (Boyce 1981). Given the relatively short time period of human exploitation, the shift toward faster LH strategies among exploited beavers is most parsimoniously explained as a phenotypically plastic response to heightened levels of mortality.

Age at menarche in different human populations is the most well-documented example of variation across time and space in a LH trait. Median menarcheal age varies from about 12.0 years in some urban postindustrial societies to 18.5 years in rural highland Papua New Guinea or high-elevation Nepali groups (Parent et al. 2003; Worthman 1999). Dramatic reductions in age at menarche among underprivileged girls from Third World countries who are adopted into affluent Western families, compared with their peers who are not adopted (see Mul et al. 2002; Teilmann et al. 2006), indicate that differences between human populations in age at menarche are substantially driven by differences in local physical conditions, such as nutrition, disease loads, and elevation (Ellis 2004; Parent et al. 2003). General improvements in health and nutrition are also responsible for the worldwide secular trend (beginning at least 170 years ago in England) toward earlier onset of pubertal development (Eveleth and Tanner 1990; Tanner 1990).

In total, extant research supports the role of environmental conditions in regulating within-species variation in LH strategy in different populations. Whereas some of this variation arises from systematic differences in the developmental conditions encountered by members of each population, other observed differences in LH strategy are substantially based in population-level differences in gene frequencies that have resulted from exposure to different selection regimes. Developmental and evolutionary effects are not independent, however, and normally reinforce and regulate each other (see discussion below, "From Evolution to Development").

Condition-Dependent Within-Population Variation in LH Strategy

Systematic individual differences in LH strategies within populations have also been well documented, and much of this variation has been shown to be condition-dependent. Condition-dependent variation in LH strategy is contingent on the competitive ability or state of individuals in a population, which is typically regulated by a combination of genetic and environmental factors. Condition-dependent shifts in development of LH strategies are outputs of adaptations that track cues to organismic condition, such as age, size, and health. For example, a substantial body of research has examined the effects of body size and associated maturational characteristics on variation in reproductive strategies. Size is a product of both allelic variations and metabolic condition (resource availability) and influences not only the physical and social niches that an organism can inhabit, but also the total levels of energetic resources available for reproduction. Research on alternative mating behaviors in male swordtail fish (*Xiphiphorus nigrensis*)

provides a good example of the causes and consequences of size in relation to LH strategy. In this species, three alleles at the *P* locus on the Y chromosome correspond to three modes in size distribution of mature males (small, intermediate, and large; Ryan et al. 1992). Although all three genotypes perform the range of species-typical mating strategies, they do so at different, size-related frequencies. Specifically, small, intermediate, and large males generally sneak, sneak and court, and court females, respectively. Size is the primary mediating mechanism in this species through which allelic variations influence mating strategies, and the effects of allelic variations on size operate through regulation of pubertal timing (with later puberty resulting in larger size; Rhen and Crews 2002).

In addition to these genetic effects, timing of puberty is also sensitive to a number of environmental factors, such as food supply, temperature, and agonistic interactions with other males (Borowsky 1987a, 1987b). These environmental influences can result in genotypically small males that are larger than genotypically intermediate males, and alternative mating strategies correlate more strongly with size than with genotype (Ryan and Causey 1989). Finally, reflecting a combination of allelic variations, metabolic conditions, and such external factors as habitat quality and intrasexual competition, size-linked individual differences in LH strategies occur not only in swordtail fish, but across a wide range of nonhuman species (e.g., Australian broad-shelled river turtles [Booth 1998], caridean shrimp [Clarke 1993], lacertid lizards [den Bosch and Bout 1998], tabanid flies [Leprince and Foil 1993], crickets [Carriere and Roff 1995], and greater white-toothed shrews [Genoud and Perrin 1994]). For extensive reviews of condition-dependent regulation of LH traits, see Gross (1996) and West-Eberhard (2003).

Socially Contingent Within-Population Variation in LH Strategy

In addition to size- and energy-dependent variation in LH strategies, there is also systematic variation in LH strategies in response to social conditions. In species as diverse as swordtail fish (Borowksy 1987b), coral reef fish (Warner 1984), and orangutans (Tainaka et al. 2007), male size, timing of puberty, and associated mating strategies are regulated by severity of intrasexual competition. Quality of parental investment may also bias animals toward different LH strategies. In rats, for example, variation in maternal behavior—licking, grooming, arched-back nursing—triggers different regulatory switches in pups that, in a developmental cascade, affect transcription of the pup's stress-responsive genetic material, the reactivity of its neural and neuroendocrine circuits, its timing of puberty, and its individual profile of defensive responses and reproductive behavior (Cameron et al. 2005). Social regulation of human LH strategies is discussed extensively below (see "Environmental Harshness: Effects on the Development of Human LH Strategies"; "Environmental Unpredictability: Effects on the Development of Human LH Strategies").

Summary

There is substantial evidence for systematic variation in LH strategy across a diverse array of animal species, both *between* and *within* populations. Different populations and different individuals within populations make different resource allocation trade-

offs over the life course. These trade-offs are embodied in individual differences in LH strategies. Through evolutionary and developmental processes, these individual differences tend to be adaptively coordinated with environmental conditions.[3] The following sections address the fundamental dimensions of the environment—varying levels of harshness and unpredictability—that have been identified as guiding both the evolution and development of LH strategies.

Impact of Harsh versus Unpredictable Environments on the Evolution of LH Strategies

What are the fundamental dimensions of environmental variation that guide the evolution of LH strategies? This second section develops and evaluates adaptive hypotheses about why some LH strategies have been maintained by natural selection instead of others. The answer to this question, we contend, depends on the harshness and unpredictability of local environments.

LH Evolution: The Early Density-Dependent Models

Variation in LH strategy was originally thought to be primarily attributable to density-dependent selection (MacArthur and Wilson 1967). Density-dependent selection occurs when the prevailing selective pressures are a function of population density. Thus, conditions of relatively low population density would favor a sufficiently high reproductive rate to rapidly fill the environment with one's offspring and maximally exploit the abundant food supply without needing to invest in competitive abilities (production of "high-quality" offspring). The assumption is that even "low-quality" offspring can survive and thrive in resource-rich environments. This is referred to as "*r* selection," after the mathematical symbol (*r*) for the biotic potential or maximal reproductive rate of a species. Conversely, conditions of relatively high population density would instead favor sufficiently high competitive ability in offspring (e.g., efficient resource utilization) so as to monopolize the limited resources in saturated environments. This involves limiting one's reproductive rate to a level that is sustainable under those constrained circumstances. This is referred to as "*K* selection," after the mathematical symbol (*K*) for the carrying capacity of the environment, or the maximal number of individuals of any given species that it can support. In 1970, Pianka applied the logic of *r* and *K* selection to the evolution of LH strategies by explicitly linking environmental conditions to constellations of LH traits. Specifically, Pianka (1970) predicted that exposure to more resource-rich *r* environments would select for traits that maximize speed of reproduction and offspring number (e.g., earlier maturity, smaller body size, higher fecundity), whereas exposure to more resource-limited *K* environments would select for traits that facilitate production and maintenance of a small number of highly fit

[3] The present review is by no means exhaustive, and additional reviews of different portions of this theoretical and empirical literature can be found in Ware (1982), Abrams and Rowe (1996), Korpimaki and Krebs (1996), and Shanley and Kirkwood (2000). More comprehensive, book-length reviews of this literature include Stearns (1992) and Roff (2002).

offspring (e.g., later maturity, greater investment per offspring, longer lifespan). Some of the first attempts to apply LH theory to human variation employed the *r-K* framework (e.g., Rushton 1985; L. Ellis 1988).

A substantial body of research has supported MacArthur and Wilson's (1967) original contention that fitness is associated with different traits in low-density versus high-density environments, and, more specifically, that high-density environments (*K* selection) favors the evolution of competitive ability (Adler and Levins 1994; Allen et al. 2008; Boyce 1984; Kawecki 1993; Mueller 1997; Reznick et al. 2002). There is no firm support, however, for Pianka's (1970) application of *r-K* selection theory to the evolution of LH strategies (e.g., Promislow and Harvey 1990; Reznick et al. 2002; Roff 2002). Nonetheless, some animals do show characteristics of hypothetical *r* selection and are now generally referred to as displaying a fast LH strategy, whereas other animals show characteristics of hypothetical *K* selection and are now generally referred to as displaying a slow LH strategy. The criticism of Pianka's (1970) model is not that density-dependent selection is irrelevant to LH variation, but rather that other selective pressures, such as age-specificity of mortality and environmental variability, play a more fundamental role in structuring the evolution of LH strategies. Consequently, the primary importance ascribed to density-dependence effects has waned over time.

Environmental Harshness: Effects on the Evolution of LH Strategies

Environmental harshness indexes the rates at which external factors cause disability and death at each age in a population. Although traditional LH theory focuses on mortality and does not explicitly address the issue of morbidity, morbidity is relevant as well because non-lethal injuries, disease, and other forms of stress can affect reproductive success and, therefore, optimal allocation of resources to growth, survival, and reproduction. In the U.S., for example, the health and fertility of underclass women (largely ethnic minorities) deteriorate more rapidly over the life course than do those of middle-class women; indeed, maternal fertility and infant survival peaks almost 5 years earlier in underclass than in middle-class women (Geronimus 1987), leading to different optimal LH strategies in these two groups (see Geronimus 1987, 1992). Given the importance of morbidity in human reproductive outcomes, the traditional conceptualization of extrinsic mortality needs to be expanded to include morbidity; thus, we will employ the more inclusive phrase *morbidity-mortality.*

Whereas environmental harshness encompasses all external sources of morbidity and mortality, extrinsic morbidity-mortality only encompasses external sources that are relatively insensitive to the adaptive decisions or strategies of the organism (Stearns 1992:182); that is, extrinsic morbidity-mortality remains even when organisms optimally allocate resources between growth, survival, and reproduction. When harsh environments cause high levels of extrinsic morbidity-mortality, even prime-age adults suffer relatively high levels of disability and death. By contrast, when levels of extrinsic morbidity-mortality are low, environmentally imposed causes of disability and death affect individuals differently depending on their age, health, size, competitive abilities, metabolism, immune functioning, and related competencies.

As shown in Fig. 1, when high levels of extrinsic morbidity-mortality either increase total mortality (largely independent of condition or age) or disproportionately influence adult mortality, organisms tend to evolve faster LH strategies (Charlesworth 1980; Promislow and Harvey 1990). Fitness is enhanced in this context by trading off growth (and the benefits of reproducing at a larger size) for earlier maturation and reproduction. This faster LH strategy reduces the risk of mortality prior to reproduction, increasing the chance of successfully contributing offspring to the next generation. These same conditions select for trade-offs favoring offspring quantity over quality: the more offspring an organism produces, the higher the probability that some will survive into adulthood long enough to reproduce. In this context, the benefits of producing a small number of high-quality offspring are outweighed by the costs of relatively high and unavoidable adult mortality. The fundamental prediction from LH theory that higher levels of extrinsic morbidity-mortality select for earlier reproduction has been confirmed by extensive comparative data: greater longevity strongly correlates with (*a*) later ages at first reproduction both across and within primate clades (Walker et al. 2006a) and (*b*) later ages at reproduction and lower offspring number across mammalian species (Holliday 1995; Stearns 1992: Figure 5.10 [based on data from Harvey and Zammuto 1985]).

It is important to note that any given source of morbidity-mortality (e.g., predation, famine, disease) may or may not be extrinsic. Indeed, extrinsic morbidity-

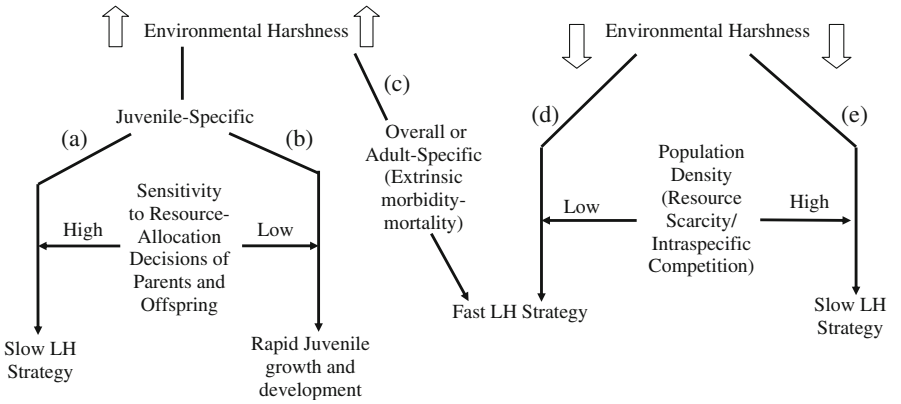

Fig. 1 Environmental harshness: Effects on the evolution of LH strategies. When high levels of extrinsic morbidity-mortality increase total mortality or disproportionately influence adult mortality, natural selection favors faster LH strategies (**c**). When juveniles, but not adults, suffer relatively high morbidity-mortality rates, then the evolution of LH strategies depends on the sensitivity of these rates to the resource-allocation decisions of parents and offspring. If incremental changes in parental investment/offspring quality significantly reduce juvenile morbidity-mortality, then natural selection favors slower LH strategies (**a**). But if juvenile disability and death are relatively insensitive to such changes in parental investment/offspring quality, and refuge is obtained by achieving adult size or status, natural selection tends to favor rapid juvenile growth and development (**b**). As environmental harshness decreases, more diffuse patterns of LH evolution occur and density-dependent effects become a major agent of selection. Low rates of environmental harshness combined with more resource-rich environments select for faster LH strategies (greater reproductive effort and productivity) (**d**). But as population density increases to approach the carrying capacity of the environment, intraspecific competition is heightened and slower LH strategies are favored by natural selection (**e**)

mortality is not defined by the source of death or disability but rather by which members of the population are affected. For example, in long-lived species, endemic disease exposures that primarily affect the young and weak can be expected to select for slow LH strategies, increasing investment in body maintenance (i.e., inflammatory host response) to ensure survival through the reproductive years. By contrast, disease exposures that cause substantial death and disability among prime-age adults (extrinsic morbidity-mortality) should select for faster LH strategies.

In species in which juveniles, but not adults, suffer relatively high levels of morbidity-mortality, the evolution of LH strategies should depend on the sensitivity of juvenile disability and death to the resource-allocation decisions of parents and offspring. If incremental changes in parental investment/offspring quality increase resistance to relevant environmental stressors, thus reducing juvenile morbidity-mortality, then natural selection should favor slower LH strategies (e.g., more parental care, larger body size, increased allocation of effort to predator-defense by juveniles; see Gosselin and Rehak 2007). However, in contexts in which juvenile disability and death are relatively insensitive to such changes in parental investment/offspring quality, and refuge is obtained by achieving adult size or status, natural selection should favor rapid juvenile growth and development (see Fig. 1). Fast growth and development in this context function to reduce the amount of time that individuals spend in the vulnerable juvenile stage (Arendt 1997; Case 1978). This morbidity-mortality profile does not favor uniformly fast LH strategies, however, because the juxtaposition of high juvenile and low adult morbidity-mortality selects for restraints on adult reproductive effort at any given time point (see bet-hedging discussion below, "Environmental Unpredictability: Effects on the Evolution of LH Strategies").

Despite this age- and stage-specific logic of mortality, juvenile and adult mortality rates are strongly correlated across mammalian species (r=0.93; partial r=0.79, after removing the effects of adult body weight [Promislow and Harvey 1990]). Distinctions between juvenile and adult mortality, therefore, may not be very meaningful in mammals; indeed, juvenile and adult mortality rates have comparable—and remarkably powerful—utility in predicting variation in LH traits across mammalian species (Promislow and Harvey 1990) and small-scale human societies (Walker et al. 2006b).

As environmental harshness decreases, enabling expansion of populations, more diffuse patterns of LH evolution occur. We propose that in this context density-dependent effects become a major agent of selection and predictions largely follow Pianka's (1970) original model. Density-dependent effects are regulated through such factors as food limitation, availability of territories, disease exposure, and conspecific violence. As shown in Fig. 1, given low rates of environmental harshness, more resource-rich, r-selecting environments (where resources are abundant relative to population size) select for faster LH strategies (greater reproductive effort and productivity; see especially Brown and Sibly 2006)

As population density increases to approach the carrying capacity of the environment (the population size that can be physically supported given natural resource limitations), intraspecific competition is heightened. This intraspecific competition may take different forms, however (Hassell 1975; Nicholson 1954; Rogers 1992). The first is called *scramble* or *exploitation* competition, in which all

conspecifics have equal access to the resources and seek to exploit them (convert them into energy for growth and reproduction) as quickly as they can before they are depleted by others. This form of competition has also been called *unadapted* or *incidental* because it results from the accidental and indirect interaction between individuals consuming the same resources, given that resources used by one are unavailable to others, and that these individuals may not even come into direct contact. In contrast, a second form of intraspecific competition is called *contest* or *interference* competition, in which all conspecifics do not have equal access to the resources owing to active interference (as by aggressive interactions, dominance hierarchies, or territoriality). This form of competition has also been called *adapted* or *programmed* because it is the result of specifically evolved competitive adaptations on the part of the individuals involved for obtaining disproportionate portions of the available resources. Because contest-competition clearly characterizes more *K*-selected species, such as humans, we focus on its implications for the evolution of life histories.

In contest competition, every individual does not suffer equally in terms of growth and reproduction as resources are depleted, and some individuals may not suffer at all owing to the existence of specifically evolved competitive adaptations. The more competitively successful individuals (the "winners") are able to monopolize mates, harvest a disproportionate share of the resources, and continue to survive and reproduce, whereas the less competitively successful individuals (the "losers") are effectively deprived of resources and mates, which curtails their survival and reproduction. Furthermore, contest competition has been called *compensatory* in that the population rarely exceeds carrying capacity as a result of direct and active interference between conspecifics. Thus, contest competition promotes the stability of population densities in relation to the carrying capacity of the environment.

In Pianka's (1970) version of density-dependent *r-K* selection theory, this pattern of population regulation and stability was associated with a *K*-selected or slow LH strategy (see Fig. 1). The assumption is that a slower LH strategy will be favored when allocation of resources to growth and competitive ability produces a sufficient gain in survival or future fertility to compensate for the decrement in current reproduction. In addition to direct advantages in contest competition, large body size and associated slow LH traits potentially enhance fitness under conditions of high population density through anatomical and/or physiological innovations that make new food resources available (e.g., evolution of a larger gut and microbial symbionts), cognitive adaptations that increase social competitiveness and resource acquisition (e.g., neocortical expansion), greater physiological homeostasis (e.g., increased resistance to cold stress and/or starvation), and reduced predation pressure (see Brown and Sibly 2006).

Effects of Harsh Environments on the Evolution of LH Strategies: The Integrative Guppy Example

The effects of extrinsic morbidity-mortality and population density on the evolution of LH strategies have been extensively studied in an unusually large corpus of research on Trinidadian guppies (*Poecilia reticulate).* Natural populations of guppies

are distributed across high- and low-predation sites in the mountains of Trinidad. High-predation sites are found in relatively downstream locations where guppies co-occur with several larger species of fish. Low-predation sites are found in more upstream locations, above rapids or waterfalls that exclude larger fish. At the high-predation sites, guppies of all ages are regularly preyed upon, population densities are low, and bioenergetic resources needed for growth and reproduction are plentiful. By contrast, at the low-predation sites, only juvenile guppies are eaten by predators, population densities are about four times higher than in high-predation sites, and bioenergetic resources are in short supply.

Reznick and colleagues collected guppies from both populations and assessed their life histories, in part by examining dissected specimens (reviewed in Reznick et al. 2002; Reznick and Ghalambor 2005). The researchers were able to draw several conclusions about the LH strategies associated with low- versus high-predation sites. Guppies from high-predation sites displayed faster LH strategies: lower birth weight, faster growth, earlier age at sexual maturation, smaller size at first birth, shorter intervals between litters, larger litter size, and greater reproductive effort per pregnancy (i.e., a higher percentage of consumed resources was devoted to production of young [as opposed to growth or maintenance]). For example, female guppies from the high-predation sites produced two to three as many offspring as equal-sized females from the low-predation sites (high offspring quantity), but the average dry mass of individual offspring in high-predation localities was only about 60% of their counterparts in low-predation areas (low offspring quality).

Although guppy LH strategies display substantial plasticity in response to changes in predation cues and physical and social environments (Bashey 2006; Dzikowski et al. 2004; Rodd et al. 1997), laboratory studies have shown that the divergent LH strategies associated with low- versus high-predation sites have a partially genetic basis (Reznick 1982; Breden et al. 1987), which was subject to natural selection over time. To test for the effects of these different habitats, a variety of experiments were conducted in which wild guppies were moved from their natural high-predation sites to low-predation sites farther upstream. When the descendants of the transplanted guppies were evaluated after 11 years (30 generations later), their life histories had shifted in the predicted direction, displaying slower LH strategies that were matched to the low-predation environment: later age at maturity, larger size at maturity, fewer offspring per litter, and greater offspring size (Reznick and Shaw 1997; Reznick et al. 1996). Furthermore, the researchers were able to induce the opposite effects—fostering the evolution of faster LH strategies—by either transplanting wild guppies from low- to high-predation sites or introducing predatory fish into previously low-predation environments (reviewed in Reznick and Ghalambor 2005).

In sum, different selection regimes arising from different environmental conditions, in concert with adaptive phenotypic plasticity, led to the development and evolution of variable LH strategies within the same species. The combination of high levels of extrinsic morbidity-mortality and high resource availability favored the development and evolution of faster LH strategies marked by early reproduction and high offspring number. Conversely, the combination of low predation, high population density, and limited resources favored the development and evolution of slower LH strategies marked by later reproduction and higher offspring quality.

Similar patterns of LH development and evolution have been documented in other poeciliid fish as well (Jennions and Telford 2002; Johnson and Belk 2001).

Age- or Stage-Specific Mortality Effects

Although the guppy data provide support for the hypothesis that (given adequate bioenergetic resources for growth and reproduction) high overall levels of extrinsic morbidity-mortality select for faster LH strategies, the data do not address age- or stage-specific mortality effects. This is because analysis of the stomach content of predators from high-predation sites revealed that guppies of all sizes were eaten at about the same rates.

A more clear-cut case of selective predation on adults is harvesting of commercial fish. Fishing equipment is typically designed to be size-selective, such as through the use of minimum mesh sizes that target larger individuals. A substantial body of research has documented changes in growth rates, maturational timing, and adult body size in commercial fish stocks during the twentieth century (reviewed in Gårdmark et al. 2003; Law 2000). This research suggests that high mortality rates induced by commercial fishing are one of the major environmental factors contributing to evolutionary change in exploited populations. As predicted by LH theory, increased adult mortality has apparently favored the evolution of faster LH strategies. Specifically, decreasing age- and size-at-maturation has been documented in a number of exploited fish stocks (e.g., halibut, Pacific salmon, North Sea plaice, Northeast Arctic cod, Baltic cod, Atlantic cod; Gårdmark et al. 2003; Law 2000). Although in most cases the necessary research has not yet been conducted to determine definitively whether these changes are underpinned by genetic evolution, the shift toward faster LH strategies is clearly predicted by LH models. Further, similar shifts toward faster LH strategies have begun to occur in populations of ungulates that have experienced generations of selective killing of prime-age adults through sport hunting (Coltman et al. 2003; Festa-Bianchet 2002). Finally, experimental work with fruitflies (*Drosophilia melanogaster*) has demonstrated under controlled laboratory conditions that high adult mortality rates cause the evolution of faster LH strategies (Gasser et al. 2000).

What about the effects of selective predation on juveniles? As stated above, LH theory predicts that when harsh environments impose high levels of mortality on juveniles, and levels of juvenile mortality are insensitive to the resource-allocation strategies of either the parents or juveniles, selection will favor rapid growth and development. This hypothesis has been tested in an analysis of 115 species of North American passerine birds that vary widely in nest predation rates (Remeš and Martin 2002). Higher nest predation was strongly correlated with faster growth rates of nestlings (even after controlling for adult body size), with shorter amounts of time that chicks remained in the nest (even after controlling for pure growth rates), and with lower body mass at fledging relative to adult body mass (i.e., leaving the nest at earlier stages of development). These data support the hypothesis that, when environmental conditions impose higher levels of juvenile disability and death, selection favors more rapid juvenile growth and development. The assumption is that incremental changes in parental investment/offspring quality could not effectively shield offspring against nest predation.

The effects of different sources of juvenile mortality on LH development have been studied in the marine snail *Nucella ostrina* (Gosselin and Rehak 2007). Different populations of these snails differ both in the physical harshness of their environments (wave exposure) and in predation pressure. Whereas populations exposed to greater wave action had larger hatching sizes, variation in predation pressure did not correlate with juvenile size. Consistent with LH theory, the snail populations exposed to more intense wave action (or other factors covarying with wave action) counteracted this stress by allocating more resources to juvenile growth/offspring quality. By contrast, predation rates were presumably not sensitive to incremental changes in the resource-allocation strategies of parents and offspring, and thus predation rates did not exert a directional selection pressure on juvenile size.

Density-Dependent Effects

The observational and experimental research conducted on guppies suggests that population density and predation are two sides of the same coin. In environments with low predation pressure, population densities increase, resources become scarce, and animals evolve slower LH strategies. The opposite occurs in high-predation environments. The challenge presented by this natural pattern of covariation is that it is difficult to untangle the relative effects of population density and predation on LH evolution. We have proposed above that high population density and associated scarcity of resources cause the evolution of slower LH strategies in populations characterized by low environmental harshness. The guppy data, however, are also potentially consistent with an alternative explanation: that slower LH strategies simply derive from low rates of externally imposed morbidity-mortality, regardless of population density and resource availability. This alternative is unlikely, however, because organisms should always benefit from accelerating LH strategies if there are no costs to doing so (e.g., if there is low extrinsic morbidity-mortality and an absence of density-dependent regulation; see Brown and Sibly 2006).

A decade-long research program on LH variation in the side-blotched lizard (*Uta stansburiana*) has demonstrated the importance of density-dependent effects in a population characterized by relatively low extrinsic morbidity-mortality. Offspring survival rates in natural populations of California side-blotched lizards oscillate in 2-year density-dependent cycles (Sinervo et al. 2000). This recurring boom-bust cycle has selected for two contrasting female LH strategies (morphs): A "fast" orange-throated morph that produces a large number of small progeny and a "slow" yellow-throated morph that produces a small number of large progeny. Consistent with *r-K* selection theory, Sinervo et al. (2000) demonstrated that the strength of selection on the two morphs varied as a function of population density. The fast, orange-throated females were favored at low density because they produced a high quantity of offspring. This caused a predictable overshooting of carrying capacity within a year and subsequent population crash. The slow, yellow-throated females were favored at high density because they produced high-quality offspring that were better adapted to surviving the crash cycles. These data indicate that cyclical, density-dependent processes in nature select for systematic variation in LH strategy (in both fast and slow directions), as specified by the current model.

r Selection: The Case of Large Herbivores on Islands

According to the current model, faster LH strategies evolve when low extrinsic morbidity-mortality occurs in the absence of density-dependent regulation of a population. Consider the case of large herbivores on islands. Large herbivores, such as deer or elephants, arrive on islands through propagules that initially subsist on a small portion of the island's total biomass. The combination of reduced predation by mainland predators (low extrinsic morbidity-mortality), reduced competition from other species with overlapping diets (high resource availability), and low population density has favored the evolution of faster LH strategies in large insular herbivores (Raia et al. 2003; Raia and Meiri 2006). This shift toward faster strategies is underpinned by reallocation of effort away from antipredator behavior and interspecific competition (e.g., reduced investment in growth and maintenance) toward reproduction. The result is earlier maturation, smaller body size, and production of relatively high numbers of low-quality offspring (Raia et al. 2003; Raia and Meiri 2006). This movement toward faster LH strategies (*r* selection) can be expected to continue until offspring mortality equals offspring production (population equilibrium), at which point selection for a faster LH strategy should cease.

As population densities increase, there could even be *K* selection for more competitive offspring. However, *K* selection in large mammals is highly constrained on islands because larger animals necessarily consume more resources and occur in smaller numbers, which makes them more vulnerable to extinction. Indeed, extinction risk is especially high in ecologically restricted environments such as islands. Because islands are geographically enclosed, afford smaller feeding niches owing to reduced island biodiversity, and are especially susceptible to climatic fluctuations and events (e.g., hurricanes), island populations experience relatively frequent population crashes (see MacArthur and Wilson 1967). In total, although high levels of extrinsic morbidity-mortality cause the evolution of faster LH strategies, it does not follow that low levels of extrinsic morbidity-mortality cause the evolution of slower LH strategies. When low rates of externally imposed morbidity-mortality are combined with adequate availability of resources, as has often been the case with large herbivores on islands, movement toward faster LH strategies can be expected to occur.

K Selection: The Island Syndrome in Rodents and Possums

Smaller mammals do not face the high level of extinction risk on islands that larger mammals do, and insular populations of rodents tend to live at higher population densities than do mainland populations (Adler and Levins 1994; Gliwicz 1980), though there are exceptions to this rule (such as on islands with low habitat quality or large areas that resemble mainlands). High population densities in insular rodents result from a combination of isolation (limiting dispersal), reduced interspecific competition for food and territories, reduced predation pressure, and reduced habitat diversity (Adler and Levins 1994; Williamson 1981). These island conditions constitute a confluence of low rates of externally imposed morbidity-mortality and high population density and should thus favor slow LH strategies (*K* selection).

As reviewed by Adler and Levins (1994), established rodent populations living under island conditions that promote relatively high population density tend to experience directional selection for increased body size, delayed sexual maturation, reduced reproductive output, and reduced aggression. A central component of this LH strategy is high competitive ability in offspring so as to monopolize limited resources in a saturated environment. A good example of this phenomenon is provided by a comparison of two populations of Virginia possums (*Didelphis virginiana*) that have been physically separated for the past 4,000–5,000 years with one living on the mainland under high-predation/low-density conditions and the other living on an island under low-predation/high-density conditions. Austad (1993) found that the island possums had evolved slower LH strategies—later ages at first reproduction, smaller litters, slower growth in offspring, slower senescence (i.e., less progressive physiological deterioration with age, as indicated by collagen aging of tail tendons), and longer lifespan—than did the mainland possums. These differences were not accounted for by variation in body mass index (BMI, a common measure of animal leanness), blood glucose levels (an indicator of metabolic state), or disease loads; indeed, none of these factors differed between the two populations. Rather, the populations differed dramatically in population density (by a factor of 4 to 1) and rates of extrinsic morbidity-mortality (predation), and these appeared to be the relevant causal factors (as was the case with guppies; see above).

Although the island possums shifted toward a slower LH strategy and production of more competitive offspring, this shift did not encompass body size. The evolution of body size was presumably constrained by intraspecific competition for food resources on a densely populated island (see Palkovacs 2003). The equality of body size between mainland and island possums is consistent with a larger body of data in mammals indicating that suites of correlated LH traits can co-evolve, independent of body size (Bielby et al. 2007; Oli 2004). In sum, the combination of low extrinsic morbidity-mortality and high population density can be expected to favor slower LH strategies, but whether that slow strategy includes larger body size depends on resource constraints.

Hard versus Soft Selection Pressures

Harshness as resource scarcity refers to the general depletion of organismic resources, including internal physiological resources as well as external material resources. Wallace (1975, 1981) distinguished between soft and hard selection. Soft selection is density- or frequency-dependent; i.e., the strength of a selection pressure depends on the density or frequency of conspecifics in the population (as in classic *r* and *K* selection). Hard selection pressures occur independently of local densities. On the one hand, high population density can produce harshness as resource scarcity in a given locality, resulting in population-specific shortages of food supply, nesting sites, territories, and so forth. On the other hand, density-independent factors such as harsh climates or meager habitats can cause harshness as resource scarcity at a metapopulation level. Harshness as resource scarcity, therefore, can act as either a soft or hard agent of selection on LH strategy.

The research reviewed above indicates that harshness as resource scarcity can act as an agent of soft selection on LH strategy. In addition, there are many examples in

nature of harshness as resource scarcity acting as an agent of hard selection on LH strategy. In birds, for example, it has been shown that predictably poor habitat quality is associated with small clutch sizes (Stearns 1992). Similarly, across fish species, colder aquatic environments tend to select for slower LH strategies (Fonseca and Cabral 2007). A good illustration of the LH consequences of an environment that is harsh due to food scarcity is the case of tubernose birds, and more specifically the wandering albatross. These birds live in a harsh environment that particularly affects increased risk of juvenile mortality. For an adult, a single foraging trip takes the animal approximately 33 days and can be as long as 15,000 km. During the foraging trip made by the mother, the young must go up to a month without food. In order to adapt to this harsh environment, a suite of slow LH traits have evolved: large body size (the wandering albatross is the largest species in the order), restriction of clutch size to one, individual lifespans ranging from about 50 years to 60 years, and wide birth spacing (total number of eggs laid in a lifetime ranges from 20 to 25; Stearns 1992). In total, recurrent harshness due to resource scarcity, whether resulting from soft or hard selection pressures, generally favors the evolution of slower LH strategies.

Implications for the Evolution of Human LH Strategies

Although humans have one of the highest rates of child survivorship in the animal world, only about 60% of children in hunting-and-gathering societies survive to reproductive age (Kaplan and Lancaster 2003). The mortality in hunting-and-gathering societies is especially high in the first 5 years of life (particularly in infancy). Once individuals reach age 15, however, mortality rates level off at about 0.5% per year until age 40, when they begin to increase, gradually at first and then sharply in the sixties and seventies. This age-specific mortality schedule shows strong similarities across hunting-and-gathering societies, suggesting that it is an evolved LH characteristic of our species (Kaplan and Lancaster 2003; cf. Hill et al. 2007, who emphasize greater variability in hunter-gatherer mortality schedules). The extraordinarily low rates of human mortality, particularly in reproductive-age adults, have been a major evolutionary impetus toward slow LH strategies in humans.

The special features of human LH have been summarized by Kaplan and Lancaster (2003:179):

> Compared to other primates, there are at least four distinctive features of human life histories: (1) an exceptionally long lifespan, (2) an extended period of juvenile dependence, resulting in families with multiple dependent children of different ages, (3) multigenerational resource flows and support of reproduction by older postreproductive individuals, and (4) male support of reproduction through the provisioning of females and their offspring. The brain and its functional abilities are also extreme among humans.

As conceptualized by Kaplan and colleagues (Kaplan and Lancaster 2003; Kaplan et al. 2000), these distinctive characteristics constitute a coevolved suite of traits. Specifically, the long juvenile period in humans coevolved with other LH characteristics that allay juvenile mortality (e.g., multigenerational resource flow, grandparental investment, male provisioning and protection). This coevolution

enabled the extended prereproductive period necessary to produce the exceptionally powerful human brain (Deaner et al. 2003; MacDonald and Hershberger 2005).

An important factor in the evolution of the distinctive human LH strategy was the "extreme commitment to learning-intensive foraging strategies and a dietary shift toward high-quality, nutrient-dense, difficult-to-acquire food resources" (Kaplan and Lancaster 2003:179). Kaplan and colleagues, along with others (e.g., Clutton-Brock and Harvey 1980; Harvey and Krebs 1990; Parker and McKinney 1999), have emphasized that the selection pressures associated with occupying a skill-intensive foraging niche played an important role in the evolution of intelligence. By contrast, other theorists have emphasized selection pressures associated with negotiating social interactions, and particularly the need to use and manipulate social information within large-group settings, as a key factor in the evolution of higher cortical functions (e.g., Alexander 1989; Byrne and Whiten 1988; Dunbar 1998; Flinn et al. 2005; Geary 2005).

Ecological and social selection pressures may be connected, however, in the evolution of human LH strategies. In addition to providing the energy-rich diet necessary to support the developing human brain through a long childhood, the improvement over hominid evolution in the ability to extract and process bioenergetic resources (Kaplan and Robson 2002) enabled larger group sizes and higher population densities. Higher population densities are associated with higher mortality rates in hunting-and-gathering societies (Walker and Hamilton 2008), presumably because of increased competition for resources, higher levels of conspecific violence, and more exposure to disease. Taken together, the combination of low extrinsic morbidity-mortality (e.g., low predation pressures, with humans generally positioned as the top predator; see Geary 2005), high population densities resulting from a combination of low extrinsic morbidity-mortality and efficient hunting and gathering practices, and high levels of competition for limited resources within and between increasingly complex social groups favored the notably slow human LH strategy and large human brain (i.e., more socially, cognitively, and physically competitive offspring). This hypothesis is consistent with comparative primate data indicating that, across species, group size (i.e., social complexity and competition) is strongly associated with indices of neocortical expansion and higher executive-to-brainstem ratios, after controlling for variation in body size, longevity, and home range (Walker et al. 2006b; see also Dunbar 2003).

Although the slow human LH strategy—prolonged childhood, long lifespan, slow growth rates, low fertility, high parental investment, large absolute and relative brain size—encompasses the suite of modal LH trade-offs that our species has converged on over its natural selective history, there is important within-species variation in LH strategy, both within and between populations (see above, "Systematic Within-Species Variation in LH Strategies"). For example, across small-scale human societies (hunter-gatherers and subsistence-based horticulturalists), age at menarche and age at first birth each occur about 1 year earlier for every 10% decline in child survivorship to age 15 (after controlling for adult body size as a proxy measure of nutrition; Walker et al. 2006b; see also Walker and Hamilton 2008). Likewise, across human societies, small body size (pygmy stature) and early fertility peaks are associated with high overall mortality rates, independent of nutritional factors (Migliano et al. 2007). Although the extent to which this cross-cultural variation

arises from phenotypically plastic responses to different developmental conditions versus genetic changes resulting from exposure to different selection regimes is unknown, the observed covariation between mortality rates, growth rates, body size, and timing of reproductive development and fertility are predicted by LH theory. Specifically, higher levels of extrinsic morbidity-mortality select for faster LH strategies.

Variation between and within human populations in LH strategies has also been linked to measured genetic variation. For example, the modal slow human LH strategy may be supported by the common 4R variant of the human dopamine receptor D4 (DRD4) gene. DRD4 regulates dopamine receptors in the brain, and variants of this gene have been linked to individual differences in such personality traits as extraversion and novelty-seeking (Ebstein 2006). The 4R allele was apparently the most common form of the DRD4 gene throughout human prehistory (Wang et al. 2004). Under conditions of environmental harshness and resource limitation, which are common in pre-agricultural foraging societies, biparental investment in offspring, durable pairbonds, and strong family ties and cooperation (i.e., slower LH strategies) are generally needed to survive and reproduce successfully (see Draper and Harpending 1988; Geary 2000; Rodseth and Novak 2000). Harpending and Cochran (2002) suggest that these ancestral conditions helped to maintain the 4R allele, which is associated with more risk-averse mating and social behavior.

Whereas the DRD4 4R allele appears to have emerged around a half-million years ago and is common in most geographical locations, the DRD4 7R allele, which is associated with more impulsive and risk-prone behavior, appears to have been selected for during the past 40,000–50,000 years and has a widely variable and non-random global distribution (Chen et al. 1999; Wang et al. 2004). Based on an analysis of this distribution, Chen et al. (1999) have argued that the 7R allele promotes migratory behavior, with bearers of 7R more likely to lead populations far from their ancient lands of origin (e.g., South American Indians, Pacific Islanders). An alternative explanation, however, proposed by Harpending and Cochran (2002), is that the 7R allele is favored by selection under conditions of surplus resources. In such luxuriant contexts, where offspring can be successful without intensive biparental investment (as is common in many agricultural and modern societies), higher levels of energetic, impulsive, and noncompliant behavior characteristic of male bearers of the 7R allele may facilitate fast sexual behavior and success in intrasexual competition (Harpending and Cochran 2002; Penke et al. 2007). Recent increases in the frequency of the 7R allele (Ding et al. 2002) are consistent with this hypothesis. In total, 7R bearers may not only be more likely to become propagules colonizing new environments (generating between-group variation in LH strategies) but may also employ faster LH strategies than 4R bearers in well-resourced, multi-niche environments (supporting within-group variation in LH strategy).

In sum, there is much variation in LH strategies between different human populations (e.g., Rushton 2004; Walker et al. 2006b; Walker and Hamilton 2008). On the one hand, genetic polymorphisms, such as those at the DRD4 locus, are potentially relevant because they may account for meaningful cultural and individual variation in LH strategies. On the other hand, comparative data from small-scale human societies suggest that differences between populations in LH strategies are

responsive to mortality rates. Much more work is needed, however, to delineate the potential evolutionary and developmental bases of such differences and their coordination with environmental conditions.

Environmental Unpredictability: Effects on the Evolution of LH Strategies

Many animals inhabit environments in which resources and mortality rates vary unpredictably over time or space; that is, these factors "vary but in a manner that cannot be predicted other than in terms of a probability distribution" (Roff 2002:287). One source of unpredictability is the behavior of predators. For example, the great blue heron and great egret colonies at the Bolinas Lagoon Preserve (north of San Francisco) have been monitored since 1967. In unpredictable intervals—1975, 1983, and 1989—the colonies were decimated by predators (raccoons or golden eagles; Pratt 1993). Another source of unpredictability is weather. Great tit populations in the Netherlands, for example, experience unpredictable climate changes (stochastic variation in the severity of winters), which greatly impact food supply (presence versus absence of mast seeding beeches), which in turn strongly influences survival rates, physical condition at fledging, population density, and intrasexual competition for territories and mates (Dingemanse et al. 2004). Sources of environmental unpredictability can be arrayed along two dimensions: temporal and spatial. Whereas temporal variability occurs when the rates at which external factors cause disability and death are unpredictable over time (e.g., good years versus bad years), spatial variability occurs when these rates are geographically unpredictable (e.g., heterogeneous foraging patches).

As shown in Fig. 2, stochastic conditions that result in widely varying levels of juvenile mortality favor the evolution of bet-hedging strategies that reduce variance in offspring fitness, whereas stochastic conditions that cause high variation in adult mortality favor the evolution of relatively fast LH strategies (Murphy 1968; Roff 2002). Both high absolute levels of adult mortality (harshness) and high variation in adult mortality (unpredictability), therefore, select for fast LH strategies. This equivalency makes logical sense: both harshness and unpredictability present adult organisms with morbidity-mortality risks that are largely insensitive to their adaptive decisions or strategies (i.e., these risks are largely unavoidable). Nonetheless, harsh environments can be predictable (i.e., short life expectancy with low variation around the mean) or unpredictable (i.e., high variation around the mean).

Unpredictable environments limit the fitness of any single phenotype, given that one strategy cannot be optimally adapted to all potentially occurring conditions. Bet-hedging theory proposes that, under certain conditions, unpredictable environments select for strategies that reduce temporal variance in fitness, even at the cost of reduced arithmetic mean fitness (Philippi and Seger 1989; Hopper 1999). These bet-hedging strategies increase the probability of achieving some reproductive success every generation while limiting success in good conditions and shielding against total failure in bad.

Einum and Fleming (2004) distinguish between two types of bet-hedging: conservative and diversified. Conservative bet-hedging corresponds to pursuing a relatively slow LH strategy, in which individuals sacrifice offspring quantity for

quality by producing a smaller number of offspring than would be optimal over a reproductive lifetime in a stable environment of the same average quality. The conservative strategy involves producing offspring that are reasonably well equipped to handle the range of fluctuating conditions encountered over the organism's evolutionary history. When such offspring perform fairly well across this range, and/or when environmental changes affect an entire population on the timescale of a generation (e.g., years of drought) and thus cannot be handled through niche selection, natural selection tends to favor conservative bet-hedging (Donaldson-Matasci et al. 2008; see Fig. 2).

By contrast, diversified bet-hedging involves "spreading the risk" by increasing phenotypic variation among offspring, and thus increasing the probability that at least some offspring will be suited to whatever environmental conditions occur in the next generation. Diversified bet-hedging can be achieved through maintenance of genetic polymorphisms or through variable expression of phenotypes arising from a monomorphic genetic structure. When any single phenotype performs poorly across the range of changing conditions encountered over evolution (i.e., when generalist strategies fail), and/or when environments vary substantially across individuals in a single generation (enabling diverse organisms to evaluate and select niches that match their phenotypes), selection tends to favor diversified bet-hedging (Donaldson-Matasci et al. 2008; see Fig. 2).

Conservative and diversified bet-hedging are not mutually exclusive, and the same species may display both. As described above, great tits (*Parus major*) inhabit

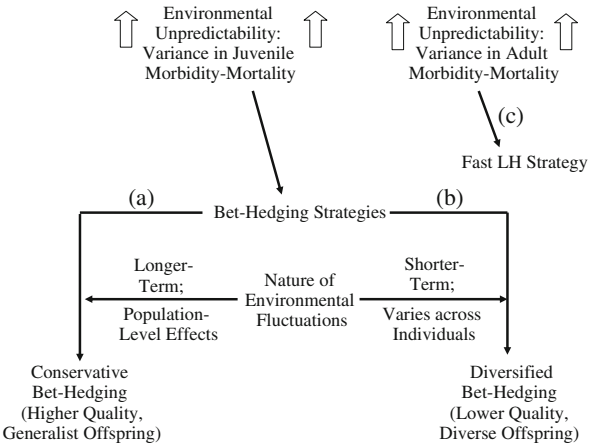

Fig. 2 Environmental unpredictability: Effects on the evolution of LH strategies. Unpredictable environmental conditions that cause high variation in adult mortality favor the evolution of relatively fast LH strategies (**c**). In contrast, stochastic conditions that result in widely varying levels of juvenile mortality favor the evolution of bet-hedging strategies that reduce variance in offspring fitness. When offspring perform fairly well across the range of fluctuating conditions encountered over evolutionary history, and/or when environmental changes affect an entire population on the timescale of a generation (e.g., years of drought) and thus cannot be handled through niche selection, natural selection tends to favor conservative bet-hedging (**a**). By contrast, diversified bet-hedging involves "spreading the risk" by increasing phenotypic variation among offspring; it is favored when environments vary substantially across individuals in a single generation and any single phenotype performs poorly across this range of changing conditions (**b**)

environments characterized by substantial temporal unpredictability. One adaptation shown by them is conservative bet-hedging: Average clutch size (8.53) is below the optimal size (12), given the long-term average quality of their habitat (Boyce and Perrins 1987). This smaller clutch size has apparently been selected for because, in bad years, individuals laying smaller clutches experience substantially better nesting success. This *bad-years effect* "reduces the mean and increases the variance in fitness for individuals laying large clutches more than it does for individuals laying smaller clutches" (Boyce and Perrins 1987:142). Although these conditions have given rise to conservative bet-hedging, the unpredictability of the great tit's environment has also favored diversified bet-hedging: adaptive genetic variation in personality, which can be characterized along the Hawk-Dove dimension. As reviewed by Ellis et al. (2006), unpredictable variation in climate cycles strongly affects food supplies and intrasexual competition among great tits, resulting in density-dependent selection for Hawks and Doves, but in opposite directions in good and bad years and in males and females. This covariation between the Hawk-Dove dimension of personality in great tits and fitness in fluctuating environments (Dingemanse et al. 2004) provides an empirical basis for the maintenance of adaptive genetic variation as a diversified bet-hedging strategy. In general, maintenance of genetic variation is linked with environmental heterogeneity (Ellis et al. 2006; Futuyma and Moreno 1988; Hedrick 1986; MacDonald 1995; Wilson 1994).

In sexually reproducing species, perhaps the simplest form of diversified bet-hedging is high offspring number (Simons 2007): As offspring number increases, so does total variance in offspring genotypes and phenotypes. Another form of diversified bet-hedging is extended age schedule of reproduction (Roff 2002). Just as parents can hedge their bets by producing offspring with different phenotypes, parents can reduce variance in offspring fitness by spacing births to ensure that offspring are of varying ages (as a buffer against age-specific forms of mortality) and by producing offspring over many different years (as protection against large-scale environmental fluctuations that create long time periods unfavorable to reproduction). Turtle LH is the classic example of this bet-hedging strategy: low and highly variable survival of eggs and first-year juveniles, high rates of adult survival, long reproductive lifespan, low annual reproductive effort, and a high degree of iteroparity (e.g., 95 reproductive episodes over an average 105-year lifespan in Michigan snapping turtles; Cunnington and Brooks 1996). The resource-allocation strategies of turtles, therefore, involve trade-offs of current reproduction and offspring quality for survival, long life, iterated reproductive episodes, and offspring quantity.

Although this strategy presumably involves restricting investment in young to a level that does not undermine adult survival, research on the LH strategies of long-lived birds breeding in stochastic environments indicates that parents adjust reproductive effort according to their own physical condition and the likelihood that chicks will survive (Erikstad et al. 1998). These adjustments can involve trade-offs of survival for current reproduction. Thus, the bet-hedging LH strategy of many long-lived birds, which involves extended age-scheduling of reproduction as insurance against stochastic variation in environmental conditions between years, is mitigated by phenotypically plastic responses that enable matching of reproductive effort to local conditions within years.

Because age-scheduling of reproduction provides a viable solution to the unpredictability problem, more extended age-scheduling reduces selection pressures for phenotypic/genotypic diversity of offspring (Schultz 1989). Nonetheless, there is extensive evidence of both developmental and evolutionary shifts toward greater offspring diversity in unpredictable environments. The maintenance of adaptive genetic variation in response to fluctuating environmental conditions (see examples above: swordtail fish, side-blotched lizards, great tits) is consistent with bet-hedging theory. In general, traits exposed to fluctuating selection pressures (i.e., environmental change) display relatively high heritabilities (Boag 1983; Burger et al. 1989).

Further, increased offspring diversity as a bet-hedging strategy has been supported by animal data showing that females increase intra-clutch variation in egg or hatchling size in more stochastic environments (Koops et al. 2003; Lips 2001; Philippi and Seger 1989). For example, both within and between populations of female brook trout, mean variability in egg size increases as a function of increasing environmental unpredictability (Koops et al. 2003). Increases in offspring diversity can also be achieved through multiple mating. In fluctuating environments, reproducing with different sexual partners reduces variance in offspring fitness by increasing the probability that at least some offspring will be well-suited to the niche they inhabit (Fox and Rauter 2003; Yasui 2001).

Finally, research has begun to illuminate mechanisms underlying increased phenotypic diversity under conditions of stress and uncertainty. Specifically, mechanisms favoring the generation of phenotypic diversity have even been found in *Escherichia coli* bacteria (Rocha et al. 2002). Stress response genes were examined for the presence of elements known to increase variability during the transfer of genetic information during both replication and gene expression. A significantly higher number of genetic elements (short close repeats) capable of inducing phenotypic variability (by slipped-mispair during DNA, RNA, or protein synthesis) were found in the stress response genes as compared with the rest of the genome. These results suggest that evolved genetic mechanisms exist in these bacteria for the generation of phenotypic diversity as a response to environmental stress, where the intensity, duration, and nature of the stress is highly variable and the optimal response to it is unpredictable. In addition, increased morphological variability has been shown to result from experimentally induced environmental stress in different species of shrew (Badyaev and Foresman 2004), and this has been interpreted as a "bet-hedging" adaptation for increasing phenotypic diversity in response to the natural variability of habitats (e.g., increased food competition, extensive mortality, changes in population distribution).

Implications for the Evolution of Human LH Strategies

Across mammalian species, variation in cranial capacity is highly correlated with variation in LH traits (Rushton 2004) and thus can be used to make inferences about LH strategy. Many researchers have noted that the gradual increase in cranial capacity that started during the evolution of *Australopithecus* and continued throughout the evolution of *Homo* in the Pleistocene coincided with the onset of global climate change approximately 3.5 mya (Calvin 2002; Elton 2008; Gribbin and Gribbin 1990; Potts 1998). Two general climatic hypotheses have been proposed.

The first is that the overall Pleistocene drop in temperature drove this evolutionary change, because colder environments were *harsh* and therefore selected against individuals with smaller brains (Lynn 1991; Rushton 1995). This hypothesis is consistent with the position presented above that resource scarcity and related harsh ecological conditions select for slower LH strategies. The second hypothesis is that Pleistocene climactic variability drove this evolutionary change, because repeated glacial and interglacial cycles selected against individuals who could not adapt over developmental time to rapidly changing and *unpredictable* environments (e.g., Calvin 2002; Chiappe and MacDonald 2005; Potts 1998). This hypothesis is consistent with conservative bet-hedging in response to environmental unpredictability occurring on a multigenerational timescale.

Recent analyses have used the human paleontological record to discriminate between these competing hypotheses. Specifically, published paleoanthropological measurements of fossil human cranial capacities (DeMiguel and Henneburg 2001) were correlated with published paleoclimatological measures of global temperatures, based on the Deuterium ($‰^{2}H$) content trapped in stratigraphic sections of ice sheets in Antarctica (Petit et al. 1999) and the Oxygen-18 ($‰^{18}O$) content trapped in stratigraphic sections of the Greenland ice sheet,[4] for the period between 10 kya and 205 kya (Wolf and Figueredo 2008).[5] When both the linear and quadratic effects of time on human cranial capacity were statistically controlled, *mean* temperature during the millennium preceding each fossil cranium (which has been estimated as the minimum amount of time necessary for human evolutionary change; see Lumsden and Wilson 1981) showed no significant incremental effect on cranial capacity, but the *variance* in temperature over the same period had a significant incremental effect.

This finding is consistent with Brunswikian evolutionary developmental theory (Figueredo et al. 2006a), which predicts that *variance* in ecological conditions over evolutionary time should select for developmental plasticity as a buffer against environmental change, whereas *mean* ecological conditions should only select for phenotypes that are matched to the "average" environment. Thus, the recurrent ice ages did not select for individuals who were permanently adapted to chronic cold; instead, the repeated glacial and interglacial cycles selected for bigger brains that enabled individuals to rapidly adapt to alternating periods of hot and cold.

Consistent with the emphasis of Kaplan and colleagues on selection pressures related to foraging being linked to the evolution of intelligence (Kaplan and Lancaster 2003; Kaplan et al. 2000), improved foraging ability (e.g., planning, inventing new techniques) would be especially important for being able to adapt to rapidly changing climates. However, it is also likely that these climatic changes repeatedly exacerbated the social competition for diminished resources during climatically harsher periods of suddenly reduced environmental carrying capacity, following climatically milder periods fostering population growth. This theory and

[4] The Greenland Summit Ice Cores CD-ROM (1997), available from the National Snow and Ice Data Center, University of Colorado at Boulder, and the World Data Center-A for Paleoclimatology, National Geophysical Data Center, Boulder, Colorado. (See http://nsidc.org/).

[5] Wolf and Figueredo, *Human cranial capacity and global climate change over the past 205,000 years*. Manuscript submitted for publication, 2008.

data complement social-brain models of the evolution of intelligence (e.g., Alexander 1990; Flinn et al. 2005; Geary 2005), which posit that population expansions and contractions exacerbated social competition, and that the disproportionately large size of the human brain evolved primarily in response to these social competitive pressures. In total, consistent with a conservative bet-hedging perspective, the disproportionately large and adaptable human brain may be a product of exposures to the widely varying physical (e.g., climatic), biological (e.g., nutritional), and social (e.g., competitive) selection pressures that were encountered during our recent evolutionary history.

Low offspring number in traditional human societies may also reflect conservative bet-hedging. Demographic studies of hunter-gatherers such as the !Kung of Botswana and the Ache of Paraguay indicate that observed fertility levels are lower than would be optimal over a reproductive lifetime (see Hill and Hurtado 1996; Hill and Kaplan 1999), given the long-term average carrying capacities of their environments. This species-typical human trade-off favoring offspring quality over quantity was presumably selected for because, given variable ecological conditions and associated high levels of social competition, individuals producing a smaller number of higher-quality offspring had greater reproductive success in bad years (i.e., these individuals experienced smaller declines in mean fitness during periods of physical or social adversity) and lower variance in fitness over time. Thus, the slow human LH strategy may partly reflect a conservative bet-hedging strategy.

At the same time, however, human LH strategies also display evidence of diversified bet-hedging. Specifically, genetic variation underlying individual differences in LH strategies has been maintained in the face of natural selection for modal LH adaptations. Genetic research, both behavioral and molecular, has documented substantial genetic influences on a wide range of human LH traits: age at menarche, age at first birth, interbirth interval, fecundity, age at last reproduction, and adult longevity (Kirk et al. 2001; Pettay et al. 2005; Rodgers et al. 2001b; for reviews, see Rodgers et al. 2001a; Ellis 2004). Importantly, the patterning of genetic influences on these LH traits is not random. Both historical Finnish data (Pettay et al. 2005) and modern US data (Rowe 2002) indicate substantial genetic correlations among female LH traits (e.g., age of menarche, age at first sexual intercourse, age at first reproduction, longevity). Likewise, behavioral genetic analyses have provided evidence of genetic covariation in cognitive and behavioral indicators of human LH strategy (Figueredo et al. 2004).

In sum, there is substantial genetic diversity within human populations influencing LH strategy (as well as developmental plasticity in response to relevant environmental cues; see succeeding discussion of development). Further, the substantial degree of genetic correlation among different LH traits suggests that this genetic variation does not merely reflect residual "noise" left over by incomplete stabilizing selection but may itself be adaptively coordinated. From a bet-hedging perspective, either temporally or spatially heterogeneous environments could maintain this systematic genetic variation (see Gillespie 1973; Leimar 2005; Sasaki and Ellner 1995). Analogous with the research discussed above on the LH strategies of long-lived birds breeding in stochastic environments, the pattern of partial heritability and partial environmentality of human LH traits suggests that the genetic diversity of LH strategies (consistent with diversified bet-hedging) is tempered by

phenotypic plasticity (e.g., big brains that enable us to respond flexibly to environmental variation).

From Evolution to Development

Evolutionary change is dependent on developmental change (i.e., phenotypes must develop to be selected); thus, developmental and evolutionary changes often proceed hand-in-hand (Jablonka and Lamb 2005; West-Eberhard 2003). The principles governing the effects of harsh versus unpredictable environments on the evolution of LH strategies, therefore, afford hypotheses about the effects of harsh versus unpredictable environments on the development of LH strategies.

To illustrate this point, we further elaborate on the island syndrome in rodents, following West-Eberhard's (2003) model of developmental plasticity and evolution. Organisms are responsive to alterations in the conditions of their lives, whether those alterations originate from mutations or persistent changes in their environment. This response normally involves modifications of the phenotype; however, these modifications must build on preexisting phenotypic characteristics and occur within the context of extant developmental systems. When these modifications are induced by environmental change, most or all members of a population can be affected. Given that such phenotypic changes (*a*) are normally constrained by the organism's natural selective history of environmental exposures to related conditions, (*b*) are induced by and thus correlated with the environment that the organism currently inhabits, and (*c*) capitalize on preexisting regulatory mechanisms that are condition-sensitive, these changes will often be adaptive.

Whether genetically or environmentally induced, significant phenotypic modifications generally involve a developmental reorganization (*phenotypic accommodation*), whereby coordinated changes in morphology, physiology, and behavior occur in response to the phenotypic modification (e.g., a goat born without forelegs that develops behavioral and morphological specializations that resemble those of kangaroos; see West-Eberhard 2003). Adaptive phenotypic accommodation enables the modified individual to maintain function (or at least reduce the amount of functional disruption) under the new conditions. These modifications can then be transmitted across generations through various mechanisms (e.g., parental behavior, transfer of physical substances, hormonal effects that influence gene expression in subsequent generations, epigenetic inheritance of environmentally induced variation and structures; Badyaev 2005; Jablonka and Lamb 2005; West-Eberhard 2003). This allows time for natural selection to operate. Further quantitative genetic variations are then selected that improve the functional response or reduce its detrimental side-effects (*genetic accommodation*). According to the West-Eberhard (2003) model, phenotypic accommodation often occurs first and then is followed, reinforced, and extended by genetic accommodation.

These processes are demonstrated by changes that occur in founder populations of rodents on islands. As discussed by Adler and Levins (1994; Levins and Adler 1993), newly established island rodent populations generally experience a number of environmental changes, such as changes in climate, competition with other species, and predation. Adler and Levins (1994) suggest that the most important environmental change, however, is population density. Indeed, the phenotypically

plastic changes that occur in insular rodents are all well-established correlates of density. Some of these changes are induced responses to the new conditions (e.g., increased survival as a result of reduced exposure to predation agents, reduced dispersal in response to geographic constraints). These changes inevitably involve phenotypic accommodation—adjustments of the organisms as a whole—to the new insular conditions (e.g., reduced dispersal and longer survival result in more stable social structures, greater neighbor familiarity, and reduced conspecific aggression; higher density results in reduced reproductive effort and larger body size; Adler and Levins 1994). However, because of differences between members of the founder population in genotypes, epigenetic factors, ontogenetic histories, and interactions therein, individuals will differ in their ability to adaptively respond to the new conditions. According to the West-Eberhard (2003) model, selection favors individuals who respond in a plastic and functional manner to the new conditions, and who preserve these changes through cross-generational transmission. These transmissible changes in developmental systems are then extended and adjusted by natural selection acting on the genetically heritable components of the systems.

In total, selection pressures in the environment tend to move evolution and development in the same direction in a mutually reinforcing manner. Recurring developmental variants, whether genotypically or environmentally induced, are the starting points for the evolution of novel adaptive traits (West-Eberhard 2003). We propose, therefore, that organized developmental responses to levels of harshness and unpredictability in childhood environments parallel evolutionary responses to these dimensions, as outlined above. By this logic, organized developmental responses precede and ultimately enable systematic (adaptive) evolutionary changes; hence, the principles that govern the effects of harsh versus unpredictable environments on the evolution of LH strategies can be used as a guide to the formation of hypotheses about the development of LH strategies.

Impact of Harsh versus Unpredictable Environments on Development of Human LH Strategies

Environmental Harshness: Effects on the Development of Human LH Strategies

In considering developmental questions, a central issue is the matching of LH strategies to the environmental conditions in which they are expressed. This can occur (1) through phenotypic plasticity, where individuals facultatively adjust development to match environmental conditions (i.e., environmental information captured during ontogeny instructs development); (2) through active gene-environment correlations, where individuals seek out environmental niches that match their genotypes; (3) through passive gene-environment correlations, where individuals are born into environments that correspond to their genotypes; and/or (4) through genetic changes resulting from exposure to different environmental selection regimes (such as in divergent evolution in mainland versus island populations).

These processes almost certainly work simultaneously and interactively to produce observed correlations between LH strategies and local conditions (see above, "Systematic Within-Species Variation in LH Strategies"). In the following

sections we examine relations between specific environmental factors specified by the theory (i.e., indices of environmental harshness and unpredictability) and expression of human LH strategies. Most of the relevant research does not permit deconstruction of the complex genetic and environmental influences and interactions that underpin these relations. Rather, our goal is to assess the degree of correspondence between the major dimensions of environmental variation specified by LH theory and predicted variation in human LH strategies on the slow-to-fast continuum. Based on the balance of evidence, we will attempt to distinguish, as possible, between the varying causal processes specified above.

Impact of Health and Energetic Conditions

As discussed above, the combination of low extrinsic morbidity-mortality, high population densities owing to efficient hunting and gathering practices, and high levels of competition for limited resources within and between increasingly complex social groups favored the notably slow human LH strategy and large human brain. With humans generally positioned as the top predator, the most prevalent sources of morbidity and mortality became density-dependent: malnutrition, infectious and parasitic diseases, and conflict with other humans (e.g., Hill et al. 2007). Indeed, mortality rates increase with increasing population densities among hunter-gatherers (Walker and Hamilton 2008). In the context of low extrinsic morbidity-mortality, high population density and related factors (e.g., limited energetic resources, high intraspecific competition) tend to favor the evolution of slower LH strategies (see Fig. 1).

The convergent developmental hypothesis is that harsh conditions arising from high population densities and related energetic limitations also favor the development of slower LH strategies (Fig. 3). Because successful conversion of energy harvested from the environment into reproduction is the central task faced by all organisms, obtaining an adequate supply of food is and always has been a fundamental adaptive problem. Consequently, energetic conditions—caloric intake, energy expenditures, and related health conditions—set a baseline for many developmental processes, including development of LH strategies. Ancestral human populations underwent periods of feast (when food supplies were abundant) interspersed with periods of famine (e.g., during drought conditions, or overpopulation), resulting in feast-famine cycles (Chakravarthy and Booth 2004). Drawing on LH theory, various evolutionary biologists and psychologists (e.g., Ellison 2001; MacDonald 1999; Surbey 1998) have argued that famine-type conditions (i.e., malnutrition, low energy intake, negative energy balance, and associated internal stressors such as disease) cause the developing person to shift toward a slower LH strategy.

The nature of these slow strategies differs, however, in evolutionary and developmental contexts. Although both evolutionary and developmental responses to resource scarcity shift individuals toward more extended maturational periods and lower offspring number, evolutionary but not developmental changes generally result in larger body size and bigger offspring. As discussed above (see "From Evolution to Development"), developmental changes precede and may later be modified by evolution. Developmental movement toward a slower LH strategy translates into development of a more energy-sparing phenotype (Fig. 3): slower

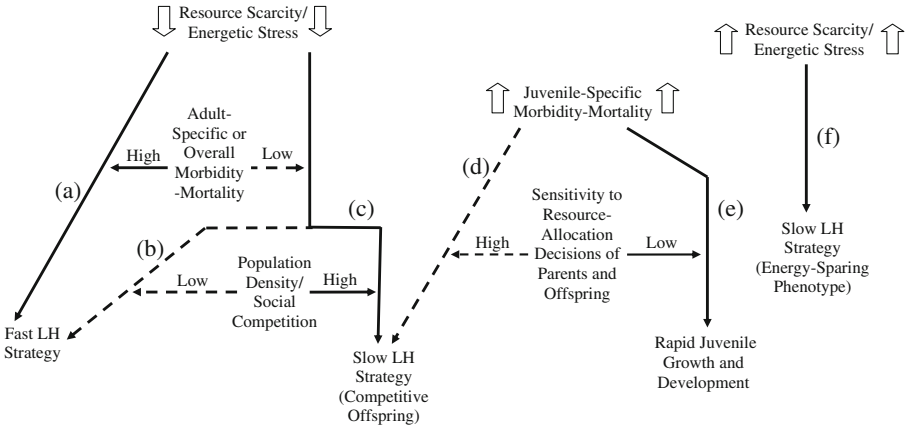

Fig. 3 Environmental harshness: Effects on the development of human LH strategies. Solid lines depict developmental hypotheses that have been empirically supported (see text). The dashed lines depict developmental hypotheses that remain to be tested. Both paths to the development of a fast LH strategy depend on there being adequate bioenergetic resources to support growth and development (**a**, **b**). If bioenergetic resources are in short supply, the resulting movement toward a slower LH strategy translates into development of a more energy-sparing phenotype (**f**). But if resources are sufficient, then environmental cues signaling high overall or adult-specific levels of morbidity-mortality should move development toward a relatively fast LH strategy (**a**). The impact of high juvenile-specific rates of morbidity-mortality depends on the sensitivity of these rates to the resource allocation strategies of parents and offspring. If such strategies significantly reduce juvenile morbidity-mortality, then parents and offspring should shift development toward slower LH strategies (**d**). But if juvenile disability and death are relatively insensitive to parent and child resource-allocation strategies, and refuge is obtained by achieving adult size or status, then juveniles should accelerate growth and development (**e**). Finally, the co-occurrence of low levels of resource scarcity/energetic stress, low rates of extrinsic morbidity-mortality, and high levels of population density/social competition favor the development of slow LH strategies (**c**). But the combination of low levels of resource scarcity/energetic stress, low rates of extrinsic morbidity-mortality, and low levels of population density/social competition should promote fast LH strategies (**b**)

growth, delayed sexual maturation, low gonadal steroid production, small adult body size, and low fecundity (see discussion above, "LH Trade-Offs and Strategies"). Initial responses to energetic stress, therefore, include trade-offs favoring maintenance over growth, future over current reproduction (late age at first birth), and offspring quality over quantity (low offspring number).[6] If such energetic constraints persist over evolutionarily significant time periods (as is normally the case when *K*-selected species reach carrying capacity), then selection can be expected to further move individuals toward an integrated slow LH strategy. Central to this integrated slow strategy is production of more competitive offspring, often with larger brains and bodies, to promote success at contest competition and more efficient utilization of resources.

Based on an evolutionary history of feast-famine cycles within human lifetimes, under famine-type conditions, members of the human species should be primed to

[6] Such trade-offs, however, only have meaning at a given level of resource availability or "condition": A woman in poor condition may delay onset of reproduction and possess only the capacity to produce a small number offspring that are high enough quality to survive. Her offspring are not high-quality in an absolute sense (compared with the quality of offspring that a woman in good condition could produce), but they do represent a (within-person) trade-off of quantity for quality, given her condition.

delay maturation and suppress reproductive functioning until predictably better times (i.e., feast-type conditions). The core argument is that natural selection has favored physiological mechanisms that track variation in resource availability and adjust the maturation and functioning of the reproductive axis to match that variation. Consistently good conditions in early and middle childhood signal to the individual that accelerated pubertal development and early reproduction are sustainable. Conversely, conditions of resource scarcity cause the individual to reserve energy for maintenance and survival (rather than growth or reproduction). Food availability is critical because surplus metabolic energy—the extent to which energy production exceeds maintenance costs—can be harvested by animals and converted into growth and reproduction. The greater the surplus, the greater the capacity for both growth and reproduction.

Data from developing countries have consistently supported the hypothesis that poor nutrition slows sexual maturation. As reviewed by Ellis (2004), children who experience chronically poor nutritional environments, whether assessed indirectly through socioeconomic status (SES) or directly in dietary studies, tend to experience relatively late pubertal development. The necessary condition for delayed puberty, however, appears to be serious or sustained nutritional deprivation; the level of dietary variation found in modern Western societies does not appear to meet these conditions (with the possible exception of high-fiber diets; e.g., de Ridder et al. 1991; Meyer et al. 1990). These data are consistent with the secular trend (beginning at least 170 years ago in England) toward earlier onset of pubertal development, as well as faster tempo of pubertal development (de Muinck Keizer-Schrama and Mul 2001; Worthman 1999), in association with general improvements in health and nutrition accompanying modernization (Tanner 1990).

Poor energetic conditions—inadequate caloric intake, high energy expenditure—not only slow maturation of the reproductive axis, they also undermine its functioning in adult women (i.e., reducing the probability of pregnancy over a given time period). The effects of energetic conditions on ovarian functioning occur on a graded continuum (Ellison 2001): Whereas minor energetic stress elicits small changes in ovarian function (e.g., causing low luteal progesterone values that result in reduced likelihood of implantation), serious energetic stress provokes major disruptions of ovarian functioning (e.g., causing anovulatory menstrual cycles or even total suppression of cycling). These graded effects of energetic condition have been demonstrated in studies of the reproductive physiology of women in traditional societies (reviewed in Ellison 2001; see also Hurtado and Hill 1990; Prentice et al. 1987). For example, the subsistence ecology of the Lese of Congo's Iturbi Forest is characterized by substantial seasonal variation in food supply, resulting in both chronic and acute periods of energetic stress. During the hunger season, many (but not all) Lese women suffer caloric deficits and lose weight; women who lost more than two kilograms were found to have lower progesterone levels and ovulatory frequency than their peers who managed to maintain their weight. After the hunger season, when nutritional conditions improved, all of the women in the study experienced increased progesterone levels and ovulatory frequency (Ellison et al. 1989; Bailey et al. 1992). This temporal covariation between energetic conditions and suppression-activation of the reproductive system is consistent with the hypothesis that our reproductive physiology is adapted to feast-famine cycles.

In total, the data showing close links between energetic factors and both maturation and functioning of the reproductive axis, together with major advances in our understanding of the molecular signals and neuroendocrine mechanisms that mediate these links (see Cunningham et al. 2004; Ellison 2001; Fernandez-Fernandez et al. 2006; Gamba and Pralong 2006), underscore the primacy of resource scarcity/energetic stress in LH development: From adolescence into adulthood, individuals adaptively and predictably adjust reproductive development to match ecological conditions. Specifically, poor energetic conditions cause individuals to shift toward a slower LH strategy.

Impact of Morbidity-Mortality Rates: Theory

From a LH perspective, both energetic conditions and age-specific rates of morbidity-mortality can be expected to shape the development of LH strategies. Walker and colleagues (Walker et al. 2006b; Walker and Hamilton 2008) present comparative data from subsistence-based human populations showing that both nutritional status (as indicated by body size) and mortality rates (both juvenile and adult) account for unique variance in ages at menarche and first birth. We propose that energetic conditions—health and nutrition—form a baseline for LH development, and other environmental conditions (e.g., extrinsic morbidity-mortality, unpredictability) move individuals around that baseline (see also Coall and Chisholm 2003). Thus, as shown in Fig. 3, all paths to the development of fast LH strategies depend on there being adequate bioenergetic resources (low resource scarcity/energetic stress) to support growth and development.

According to LH theory, when high levels of extrinsic morbidity-mortality either increase total mortality (largely independent of condition or age) or disproportionately influence mortality among prime-age adults, organisms should evolve faster LH strategies (see above, "Environmental Harshness: Effects on the Evolution of LH Strategies"). Given the links between development and evolution, these same conditions should also promote the development of faster LH strategies. Faster strategies in this context—a context that devalues future reproduction—function to reduce the risk of disability or death prior to reproduction. At the same time, however, the ability of organisms to shift toward faster LH strategies in response to morbidity-mortality cues depends on there being adequate bioenergetic resources to support growth and development (Fig. 3).

Although in many animal taxa important distinctions have been established between the effects of juvenile and adult mortality on LH evolution (see above, "Environmental Harshness: Effects on the Evolution of LH Strategies"), these distinctions may be of less importance among mammals in general, and humans in particular, because juvenile and adult mortality rates are strongly correlated (mammals: Promislow and Harvey 1990; small-scale human societies: Walker et al. 2006b). This strong correlation suggests that variations in overall rates of morbidity-mortality, rather than juvenile- or adult-specific rates, may be the most relevant to explaining variation in human LH strategies.

Nonetheless, LH theory generates predictions about the effects of juvenile-specific mortality on the development of LH strategies, and research is needed to test these predictions in humans. In species in which juveniles, but not adults, suffer

relatively high levels of morbidity-mortality, the evolution of LH strategies should depend on the sensitivity of juvenile disability and death to the resource-allocation decisions of parents and offspring (see above, "Environmental Harshness: Effects on the Evolution of LH Strategies"). When incremental changes in parental investment/offspring quality significantly reduce (counteract) juvenile morbidity-mortality, natural selection can be expected to favor slower LH strategies. The concomitant developmental prediction is that this set of conditions should also favor the development of slower LH strategies (e.g., higher levels of parental care, lower offspring number, higher offspring quality; Fig. 3). Along these lines, monogamous marriage and father-present social systems are more likely to be found among hunter-gatherers inhabiting harsh physical environments where biparental care (male provisioning) is substantial and important for offspring survival and reproductive success (Draper and Harpending 1988; Geary 2000; Kaplan and Lancaster 2003: Table 7–1; Marlowe 2003). Conversely, when juvenile morbidity-mortality is relatively insensitive to variations in parental investment/offspring quality but refuge is obtained by achieving adult size or status, the prediction is that individuals will reduce the amount of time spent in the vulnerable juvenile stage by accelerating growth and development (given adequate bioenergetic resources to support this strategy; Fig. 3).

Many modern human populations are characterized by low rates of externally imposed morbidity-mortality (owing to our position as the top predator and the general advances in disease prevention and treatment), low levels of resource scarcity/energetic stress (owing to highly efficient food production), and high levels of population density/social competition (urbanization). As shown in Fig. 3, the co-occurrence of these three factors should favor the development of slow LH strategies, as in classic *K* selection. In this context, individuals may trade off current for future reproduction by delaying first birth or increasing birth intervals to accrue greater reproductive capacity (e.g., more resources and sociocompetitive competencies that can be converted into reproduction). Many people in Western societies, for example, delay reproduction to enhance their education, work skills, and socioeconomic status. This trade-off may benefit individuals reproductively by enabling them to produce more competitive offspring (see Low et al. 2002).

The other side of the coin is that the combination of low levels of resource scarcity/energetic stress, low rates of extrinsic morbidity-mortality, and low levels of population density/social competition should promote fast LH strategies, as in classic *r* selection (see Fig. 3). A case in point is the European expansion into suitable ecologies throughout the world ("Neo-Europes"), such as the Americas, South Africa, Australia, and New Zealand, in the early modern era (Crosby 2004). The demographic parameters of these populations had previously been limited by such factors as scarcity of arable land, social strife, and endemic diseases in Europe. The lifting of such constraints, however, resulted in movement toward faster LH strategies and greatly increased population growth rates in the new environments.

Impact of Morbidity-Mortality Rates: Data

To examine the range of hypotheses articulated above, empirical studies would need to separately assess juvenile-specific and adult-specific/overall morbidity-mortality

rates and test for their relative effects on the development of LH strategies. Unfortunately, very little extant research on human LH strategies has discriminated between these constructs or their proximal indicators. The current literature review, therefore, focuses primarily on the effects of overall mortality rates (e.g., life expectancy at birth) and cues to morbidity-mortality (i.e., observable environmental risks) in one's local environment. Although this strategy is not ideal, it is tenable given the strong correlation between juvenile and adult mortality.

Between-Population Variation in Mortality Rates A few studies have examined differences in mortality rates across neighborhoods, countries, or small-scale societies and then computed correlations with LH traits. Wilson and Daly (1997) conducted an analysis of life expectancy across Chicago neighborhoods in relation to reproductive timing. Life expectancy is an encompassing index that includes all causes of death, from disease to homicide, at all stages of life. Daly and Wilson (1997) posit that our minds are functionally designed to keep track of local death rates through observation of the fates of other relevant people (e.g., Were both of your grandfathers already dead before you were born? Have some of your primary school classmates already died?). Wilson and Daly (1997) found that women living in neighborhoods characterized by shorter life expectancies reproduced at earlier ages. Neighborhood conditions in Chicago also predicted variation in offspring number: average number of children born to ever-married women was 2.9 in high-quality neighborhoods, 3.7 in medium-quality neighborhoods, and 5.0 in low-quality (ghetto) neighborhoods (Hogan and Kitagawa 1985).

Low et al. (2008) examined the relation between life expectancy and age at first birth in 170 nations. The data were drawn from United Nations sources. Low et al. (2008) found that variation in life expectancy at birth accounted for 74% of the variation in age at first birth, with shorter life expectancy predicting earlier age at first birth. In addition, the data clearly indicated that women from poorer countries had earlier ages at first birth; thus, the correlation between life expectancy and age at first birth was not an artifact of women in poorer physical condition delaying reproduction. Israel constituted an outlier in the data, with a high and relatively stable life expectancy at birth (80 years) and a relatively early age at first birth (just under 22 years). This incongruous data point highlights the likely fact that individuals do not directly detect mortality rates but instead respond to proximate cues to levels of extrinsic morbidity-mortality, which may be quite prevalent in Israeli society. Although the very strong positive correlation between life expectancy (and its proximate correlates) and age at first birth cannot demonstrate causation, the correlation is clearly specified by LH theory and concurs with comparative primate (Walker et al. 2006b) and mammalian (Harvey and Zammuto 1985; Stearns 1992: Figure 5.10) data demonstrating very strong positive correlations between longevity and age at first reproduction. To more clearly establish causation, however, one would need to show that changes in age at first birth followed changes in life expectancy. Unfortunately, such historical, cross-national data are unavailable.

An analogous study of 22 small-scale human societies (hunter-gatherers and subsistence-based horticulturalists) was conducted by Walker and colleagues (Walker et al. 2006b; see also Walker and Hamilton 2008). Adult body size is so

closely linked to childhood nutrition that it can be used as a proxy measure for energy availability while growing up. Consistent with much past research (e.g., Ellis 2004; Ellison 2001), Walker, Gurven et al. demonstrated that societies with larger and taller adults displayed faster childhood growth rates and earlier ages at menarche and first birth in females. Most striking, however, after controlling for adult body size, higher rates of childhood mortality further predicted faster growth and earlier reproductive development. In these multivariate analyses, age at menarche and age at first birth each occurred about 1 year earlier for every 10% decline in survivorship to age 15. In sum, lower juvenile survivorship was uniquely and significantly associated with faster juvenile growth and earlier timing of puberty and reproduction.

If juveniles suffer high mortality, and incremental changes in the resource-allocation strategies of parents and offspring do not substantively shield juveniles against this mortality, then selection should favor evolutionary and developmental shifts toward rapid growth and sexual maturation (Fig. 3). These shifts function to reduce the amount of time spent in the vulnerable juvenile stage. Although this accelerated strategy provides refuge from some sources of morbidity and mortality, rapid growth and development favors investments in current over future reproduction, increases maintenance costs and provisioning demands on parents, and "may bring few benefits for pre-reproductive youngsters with underdeveloped cognitive capacities in complex foraging or social settings" (Walker et al. 2006b:306).

Alternatively, juvenile mortality rates per se may not be the key variable. Walker et al. (2006b) documented a 0.59 correlation between juvenile and adult mortality, indicating that juvenile mortality was a reliable predictor of adult mortality. This substantial correlation suggests that overall rates of mortality, rather than juvenile- or adult-specific rates, could be the most relevant factor accelerating LH strategies. Further research is needed to clarify this issue.

In total, across Chicago neighborhoods, modern nation-states, and small-scale preliterate societies, higher mortality rates are strongly associated with faster LH strategies. These findings conform to the predictions of LH theory and strongly parallel comparative primate and mammalian data.

These comparisons between human populations, however, do not directly address the degree to which observed LH variation arises from phenotypically plastic responses to different developmental conditions, from gene-environment correlations, or from genetic changes resulting from exposure to different selection regimes. Whereas variation in reproductive timing across Chicago neighborhoods could not plausibly reflect evolutionary divergences, this variation may be underpinned by developmental responses to mortality cues (as contended by Wilson and Daly 1997), gene-environment correlations (e.g., clustering of genes for fast LH strategies in underclass neighborhoods), or, most likely, a combination of these factors. Gene-environment correlations, however, probably have less relevance for explaining the cross-national data presented by Low et al. (2008). Given the heterogeneity of modern nation-states and the relatively rapid historical changes that have occurred in life expectancy and age at first birth, the observed pattern of age at first birth is more likely to constitute a developmental than an evolutionary response to varying levels of morbidity-mortality across the 170 countries. Whereas this developmental

interpretation was endorsed by the authors (Low et al. 2008), Walker and Hamilton (2008), in their study of mostly hunting-and-gathering societies, suggest that mean differences between populations in growth rates and reproductive timing have been shaped by the evolutionary effects of mortality. This evolutionary argument implies that, at the population level, organized developmental responses to varying levels of morbidity-mortality were extended and adjusted by natural selection (see above, "From Evolution to Development").

Within-Population Variation in Extrinsic Morbidity-Mortality Whereas between-population studies have focused on variation in mortality rates across groups, within-population studies have generally focused on individual differences in developmental exposures to proximal cues to morbidity-mortality. The psychobiological mechanisms that regulate LH strategies should have been designed by natural selection to detect and respond to environmental indicators of morbidity-mortality—observable cues that reliably covaried with morbidity-mortality risks during our evolutionary history (e.g., low social or economic status, exposures to violence, dangerous ecological conditions, harsh childrearing practices; see Chisholm 1993, 1999). Within a population, children who have greater exposure to cues indicating high levels of extrinsic morbidity-mortality should develop faster LH strategies (see Belsky et al. 1991; Bereczkei and Csanaky 2001; Chisholm 1999; Promislow and Harvey 1990; Wilson and Daly 1997), particularly if there are adequate bioenergetic resources for growth and reproduction (see Fig. 3).

What are reliable cues to extrinsic morbidity-mortality? One important cue is socioeconomic status (SES). In Western societies, lower levels of SES are linearly related to higher levels of virtually all forms of morbidity and mortality (e.g., Adler et al. 1993; Chen et al. 2002). From a LH perspective, therefore, lower levels of SES in Western societies should lead to faster LH strategies because people inhabiting low SES environments have systematically greater exposure to premature disability and death, on the one hand, while possessing adequate bioenergetic resources to support growth and reproduction, on the other. This prediction has been supported by a large body of research showing negative correlations between SES and various indicators of a fast LH strategy, including early sexual activity (e.g., Ellis et al. 2003; Kotchick et al. 2001), adolescent pregnancy and childbearing (e.g., Ellis et al. 2003; Miller et al. 2001), high offspring number (Vining 1986), and low levels of parental investment per child (e.g., Belsky et al. 1991; Ellis et al. 1999).

These SES-LH trait correlations need to be interpreted with caution, however, because they could substantially reflect passive and active gene-environment correlations, with genes for faster LH strategies clustering in lower socioeconomic groups. One study examined the effects of changes in SES, independent of possible gene-environment correlations, that resulted from a "natural experiment." In a rural community in North Carolina, SES was substantially altered by circumstances outside the control of the affected individuals: the introduction of a casino. As luck would have it, this introduction took place in the middle of an ongoing longitudinal study of child and adolescent health (Costello et al. 2003; n=1,420 families). One-fourth of the children in the study were Native Americans, and all of the Native American families received an income supplement from the casino. This income supplement moved 14% of the families in the study out of poverty, while 53%

remained poor and 32% were never poor. Although the study did not directly assess LH traits, the major outcome variable in the study—child and adolescent externalizing behavior problems (conduct disorder and oppositional defiant disorder)—reliably predicts development of faster LH strategies (i.e., early sexual debut, multiple sexual partners, adolescent pregnancy and childbearing; e.g., Fergusson and Woodward 2000; Serbin et al. 1991; Underwood et al. 1996). The persistently poor and ex-poor children displayed comparably high levels of externalizing behavior problems prior to the introduction of the casino (with both groups scoring significantly higher than the never-poor group). After the introduction of the casino, externalizing problems in the ex-poor group fell to the same low level as in the never-poor group, while externalizing problems in the persistently-poor group remained high (more than twice the levels displayed by either the ex-poor or never-poor children). In sum, this quasi-experimental design demonstrated that relief of poverty caused a large reduction in externalizing behavior problems—a known antecedent of a fast LH strategy. These data are consistent with the hypothesis that development of LH strategies is responsive to socioeconomic conditions.

What are other important cues to extrinsic morbidity-mortality? Personal knowledge of deaths among adolescents and young adults in one's local environment is probably the most powerful signal to accelerate LH strategies; such knowledge provides the most direct and salient information about local mortality rates and the individual's probability of premature death. In addition, exposures to violence should provide important cues to levels of extrinsic morbidity-mortality. Such exposures may include direct experiences with violence (as a perpetrator or victim), directly witnessing violence (e.g., seeing someone get stabbed or beat up), inhabiting an environment characterized by high levels of violence (e.g., living in a neighborhood with high rates of violent crime), or obtaining information from others regarding rates of violence in one's local environment. Further, growing up in family and neighborhood contexts characterized by short life expectancies or high rates of premature illness and physical disability should also shift individuals toward faster LH strategies. Finally, relevant cues to external morbidity and mortality risks may be conveyed to children by parents through harsh (abusive) or unsupportive (neglectful) childrearing practices.

Although it is well-established that residence in disadvantaged neighborhoods is associated with development of faster LH strategies (e.g., Cohen et al. 2000; Miller et al. 2001; Ramirez-Valles et al. 1998), less is known about the mechanisms through which neighborhood effects occur. One well-studied mechanism is poverty. As Ramirez-Valles et al. suggest, "Financial deprivation creates a set of values and norms, weak adult supervision, and a limited availability and involvement in prosocial activities, facilitating sexual risk behavior" (1998:239). From a LH perspective, however, the most salient feature of low SES environments should be exposure to cues indicating high risk of premature disability or death. A small number of studies have looked at the effects of such cues (neighborhood hazards) on sexual and reproductive behaviors, controlling for SES. Lauritsen (1994) examined the effects of "neighborhood disorder" (parents' reports of the extent to which vandalism, abandoned housing, presence of winos and junkies, assaults and muggings, burglary and thefts, and rundown or poor housing were problems in

their neighborhoods) on rates of sexual intercourse among adolescents, controlling for adolescents' age, family structure, and family income. Upchurch et al. (1999) investigated the effects of neighborhood hazards (adolescents' reports of personal threats, such as drive-by shootings; physical deterioration, such as rundown housing; and social threats, such as the presence of gangs) on rates of early sexual activity, controlling for neighborhood SES and race/ethnicity. Cohen et al. (2000) assessed the effects of neighborhood conditions (researchers' ratings of housing quality, abandoned cars, graffiti, trash, and public school deterioration) on rates of gonorrhea, controlling for neighborhood income levels, education levels, and unemployment rates. In each of these studies, more observed or perceived cues to neighborhood deterioration and danger were associated with earlier sexual debut or higher rates of risky sexual behavior in adolescence, independent of the effects of SES. In sum, these data link neighborhood cues to extrinsic morbidity-mortality to faster LH strategies, regardless of differences in financial conditions between neighborhood residents.

In addition to this research on neighborhood effects, a large developmental literature documents effects of involvement in violence (as perpetrator or victim) on sexual and reproductive behaviors. Violence in childhood and adolescence has been measured in various ways, including self-, peer-, teacher-, and parent-reports of involvement in antisocial or violent activities (e.g., Fergusson and Woodward 2000; Serbin et al. 1991; Underwood et al. 1996), association with violent or delinquent peers (e.g., Capaldi et al. 1996; Scaramella et al. 1998), and history of victimization (physical abuse, sexual abuse; Kotchick et al. 2001; Miller et al. 2001). Regardless of how violence was measured, involvement in violence—as a perpetrator or victim—was reliably associated across all of these studies with faster LH strategies (e.g., early sexual debut, multiple sexual partners, adolescent pregnancy and childbearing).

Finally, a substantial developmental literature has also documented relations between quality of parental investment and development of LH strategies. As proposed by LH theorists, quality of parental investment is a key mechanism through which young children receive information about levels of stress and support in their local environments, including levels of extrinsic morbidity-mortality (e.g., Belsky et al. 1991; Bereczkei 2007; Chisholm 1999; Ellis 2004). Indeed, the informational value of parental investment has been demonstrated in recent cross-cultural research. Based on analysis of mostly preindustrial societies in the Standard Cross-Cultural Sample, Quinlan (2007) found that low-quality parental investment tracked ecological stress, with mothers decreasing parental care and terminating breastfeeding at earlier ages under conditions of warfare, famine, and high pathogen stress. Quinlan posits that this diminution in parental investment occurs because parental care (above a basic threshold) does not shield children against such sources of morbidity and mortality. Children, in turn, respond to parental cues. Specifically, LH theorists have proposed that children are functionally designed to respond to variation in parental investment by adaptively adjusting LH strategies on the slow-fast continuum. Along these lines, harsh or neglectful parenting, low parent-child connectedness and support, and low parental monitoring are reliably associated with such fast LH traits as early puberty, early sexual debut, adolescent pregnancy and childbearing, and short life expectancies (e.g., Bereczkei and Csanaky 2001; Ellis 2004; Ellis et al. 2003; Foster et al. 2008; Kotchick et al. 2001; Miller et al. 2001). In

sum, low-investment parenting strategies provide reliable cues to extrinsic morbidity-mortality and may operate to accelerate LH development in offspring.

In total, a vast literature has documented reliable associations between cues to extrinsic morbidity-mortality—lower SES, low local life expectancies, exposures to violence, neighborhood hazards, low parental investment—and faster LH strategies. Although these data are clearly consistent with LH theory, they do not unambiguously support the theory because most extant research has not employed causally informative designs. On the one hand, LH theory clearly posits that individuals facultatively adjust LH strategies to match levels of extrinsic morbidity-mortality. On the other hand, the now-well-established links between indicators of morbidity-mortality and LH traits could reflect gene-environment correlations. Implementation of causally informative research that can discriminate between these competing explanations is still in its infancy (see initial studies by D'Onofrio et al. 2006; Ellis et al. 2009; Mendle et al. 2006; Tither and Ellis 2008). Nonetheless, the cross-national data presented by Low et al. (2008) are unlikely to be explained by gene-environment correlations. The most reasonable and parsimonious conclusion, we believe, is that shorter life expectancies/high mortality rates—as indicated by the conditions of people's lives that reliably forecast premature aging or death—facultatively accelerate LH strategies.

Environmental Unpredictability: Effects on the Development of Human LH Strategies

According to LH theory, stochastic environmental conditions that result in widely varying levels of juvenile mortality favor the evolution of bet-hedging strategies that reduce variance in offspring fitness, whereas stochastic conditions that cause high variation in adult mortality favor the evolution of relatively fast LH strategies (see Fig. 2*)*. A first developmental hypothesis that follows from this logic is that exposures to stochastic conditions (or reliable cues to environmental unpredictability) that signal widely varying levels of juvenile mortality should result in the development of bet-hedging strategies (diversified or conservative). Drawing on Donaldson-Matasci et al. (2008), this hypothesis can be further elaborated: Whereas longer-term environmental changes that affect entire populations of juveniles and can be handled by generalist strategies (e.g., investment in greater competitive ability in offspring) should promote conservative bet-hedging, shorter-term environmental fluctuations that vary across individuals in a single generation and cannot be handled by a single generalist phenotype should promote diversified bet-hedging (Fig. 4). Conservative bet-hedging involves producing a lower number of offspring than would be optimal over a reproductive lifetime in a stable environment of the same average quality; it depends on parental capacity to enhance the survival, competitiveness, and eventual reproductive success of offspring across the varying conditions. Diversified bet-hedging, by contrast, involves such behaviors as producing a high number of offspring and/or reproducing with multiple partners (to increase genotypic/phenotypic diversity), extending the age-scheduling of reproduction (to hedge against temporal fluctuations), and stress-induced increases in genotypic/phenotypic diversity; it is favored when parental care does not substantially shield children against the variable sources of morbidity-mortality

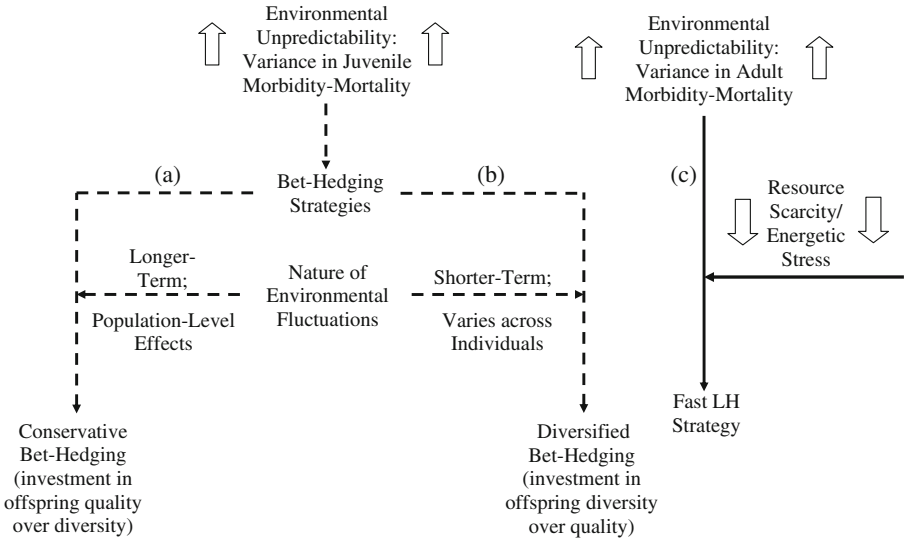

Fig. 4 Environmental unpredictability: Effects on the development of human LH strategies. Solid lines depict developmental hypotheses that have been empirically supported (see text). The dashed lines depict developmental hypotheses that remain to be tested. Given adequate resources, environmental cues indicating high variance in adult morbidity-mortality shift development toward relatively fast LH strategies (**c**). By contrast, exposures to stochastic conditions (or reliable cues to environmental unpredictability) that signal widely varying levels of juvenile morbidity-mortality should promote development of bet-hedging strategies (diversified or conservative) (**a**, **b**)

encountered in fluctuating environments. To our knowledge, no human research has tested whether relevant exposures to varying (stochastic) levels of juvenile mortality result in the specified parental bet-hedging strategies.

A second developmental hypothesis is that exposures to stochastic conditions that indicate widely varying levels of adult morbidity-mortality should result in the development of faster LH strategies (see Fig. 4). Although a fast LH strategy shares some elements with a diversified bet-hedging strategy, the former primarily concerns faster pace of reproduction (e.g., earlier sexual maturation, shorter birth intervals) whereas the latter fundamentally concerns offspring diversification. In contexts where environmental factors cause high absolute levels of mortality or high variability in mortality among prime-age adults (harshness or unpredictability, respectively), as could occur with introductions of HIV/AIDS into a population, the prediction is that individuals will shift toward faster tempo of reproduction, but not that individuals will specifically shift toward greater offspring diversification.

Although the preceding logic specifies differences between the effects of high variability in mortality rates in juveniles versus adults, we again emphasize that these distinctions may have limited relevance for mammals in general, or humans in particular, because juvenile and adult mortality rates are very strongly correlated. This strong correlation suggests that spatial or temporal variability in overall rates of extrinsic morbidity-mortality, rather than juvenile- or adult-specific rates, may be the most important facet of environmental unpredictability in relation to human LH strategies. Nonetheless, the effect of variation in adult versus juvenile mortality remains an important empirical question.

In the preceding section ("Environmental Harshness: Effects on the Development of LH Strategies"), we argued that relevant psychobiological mechanisms should have evolved to detect and respond to proximal cues to *levels* of extrinsic morbidity-mortality (harshness). Here we extend that logic by arguing that these mechanisms should also have been selected to detect and respond to proximal cues to *variability* in morbidity-mortality risk (unpredictability; e.g., stochastic changes in ecological context, geography, economic conditions, family composition, parental behavior). Because levels of and variability in extrinsic morbidity-mortality are distinct factors, developmental exposures to environmental indicators of harshness and unpredictability should each uniquely contribute to acceleration of LH strategies.

Ross and Hill (2002) propose that childhood unpredictability contributes to an *unpredictability schema*—"a pervasive belief that people are unpredictable and the world is chaotic" (p. 458)—which orients individuals toward the "here and now" and increases risk-taking behaviors (e.g., early sexual activity, risky sexual behavior, adolescent pregnancy and childbearing). Development of an unpredictability schema, therefore, may be an important mediating mechanism through which exposures to stochastic conditions shift individuals toward faster LH strategies (given adequate bioenergetic resources to support growth and reproduction).

Childhood Unpredictability

A small body of research has investigated the effects of unpredictability of childhood environments on LH traits. In an analysis of data from the National Longitudinal Study of Adolescent Health, Brumbach et al. (2009) assessed exposures to both harsh and unpredictable environmental conditions in adolescence. Harshness was operationalized as exposure to violence from conspecifics and unpredictability was measured by frequent changes or ongoing inconsistency in several dimensions of childhood environments. As predicted by the theory, experiences signaling harshness and unpredictability in adolescence each independently (uniquely) contributed to the development of faster LH strategies from adolescence through young adulthood.

Given the centrality of parents in children's developmental environments, perhaps the most salient measure of childhood unpredictability is number of parental changes. Indeed, children who experience changes in parental figures are exposed to a high level of environmental instability and unpredictably (Raley and Wildsmith 2004). Along these lines, several studies have examined the effects of number of parental transitions on LH traits (Albrecht and Teachman 2003; Capaldi et al. 1996; Woodward et al. 2001; Wu 1996; Wu and Martinson 1993). Each of these studies employed large representative national samples and/or prospectively studied community samples over the course of childhood. Parental transitions were operationalized as changes in adult household members resulting from such factors as separation/divorce, death, remarriage/cohabitation, reconciliation, adoption, or placement of the child in foster care, a group home, or a detention center. These studies all examined the unique effects of number of parental transitions after controlling for the effects of a variety of potential confounds, including multiple measures of environmental harshness. Wu and Martinson (1993; Wu 1996) controlled for religion, mother's age at first birth, number of siblings, father's SES, mother's and daughter's years of completed schooling, and daughter's

intelligence. Albrecht and Teachman (2003) controlled for religion and religiosity, mother's and father's education, mother's age at first birth, mother's work status, number of siblings, and daughter's age at menarche. Woodward et al. (2001) controlled for parent SES, marital conflict, physical and sexual abuse, mother's age at first birth, being born into a single-mother household, daughter's age at menarche, daughter's intelligence, and daughter's conduct problems. Capaldi et al. (1996) controlled for parents' SES, parental antisocial behavior, deviant peer affiliation, child antisocial/delinquent behavior, parental monitoring, and physical maturation. In each of these studies, number of parental transitions emerged as a central and substantively important predictor of accelerated LH strategy (i.e., earlier age at first sexual intercourse, higher rates of premarital intercourse, teenage pregnancy, and premarital birth), above and beyond the combined effects of all of the measures of environmental harshness and child characteristics.

Another relevant index of childhood unpredictability is frequency of residential change, which involves breaking current peer and community relationships and establishing new ones. A number of studies have examined relations between childhood residential changes and development of LH traits. This literature clearly indicates that frequent residential mobility in adolescence is associated with development of a faster LH strategy: earlier age at first sexual intercourse, multiple sex partners in adolescence, and higher rates of premarital sex, pregnancy, and childbearing (Baumer and South 2001; Crowder and Teachman 2004; Gibbs 1986; South et al. 2005; Stack 1994; Sucoff and Upchurch 1998). One explanation for this effect is that children who experience multiple residential changes often experience changes in schools and peer groups. More delinquent peer groups are more accepting of these newcomers than are other social groups, and matriculating children tend to adopt the delinquent behaviors of their new peers, which often includes sexual activity (South et al. 2005). Because families that frequently change residence constitute a low SES population, residential mobility measures may conflate environmental harshness and unpredictability. Much of the research on this topic, however, has demonstrated persistent effects of residential mobility while controlling for SES variables as well as surrounding neighborhood disadvantage (Baumer and South 2001; Crowder and Teachman 2004; South et al. 2005; Sucoff and Upchurch 1998). Thus, as is the case for parental transitions, frequent residential mobility uniquely predicts fast LH strategy, above and beyond the measured effects of environmental harshness.

Although the foregoing studies adjusted for many relevant covariates, this methodology necessarily relies on an arbitrary and incomplete set of control variables that the researchers measured; it cannot account for unmeasured environmental or genetic factors. This limitation highlights the need for causally informative research designs that assess the impact of unpredictable childhood environments.

Along these lines, there is an ongoing randomized controlled trial that intervenes to reduce levels of unpredictability in the lives of very high risk adolescents: girls in the juvenile justice system assigned to out-of-home care (Chamberlain et al. 2007; Leve et al. 2005). As described by Leve and Chamberlain (2004), these girls have very high genetic and environmental risk for delinquency, risky sexual behavior, and teenage pregnancy. Part of that environmental risk is a developmental history characterized by extraordinarily high levels of instability and change. Before these

girls become teenagers, they experience an average of eight parental transitions (changes in adult household members; Leve and Chamberlain 2004), often moving between different group care programs or between group care and residence with their birth families. These parental transitions generally involve substantial changes in rules, relationships, privileges, resources, safety levels, and routines. According to the present theory, this high level of instability and change should result in strong development of unpredictability schemas (Ross and Hill 2002) and very fast LH strategies.

Kerr et al. (2009) randomly assigned 166 of these girls (ages 13–17) to either Multidimensional Treatment Foster Care (MTFC) or intervention services as usual (group care). MTFC involves individual placement in highly trained and supervised homes with state-certified foster parents. The goal of MTFC is to reduce behavioral problems, and the method employed to attain this goal involves creating maximally structured and predictable environments for the girls. The foster parents and other caregivers carry out an organized behavior management program at home, in school, and in the community that emphasizes fair and consistent limits and predictable consequences for rule breaking. For example, the foster parents use a point system to track and regulate the youths' behavior, where points are awarded for positive behaviors (e.g., attending classes, completing chores) and taken away for negative behaviors (e.g., not completing homework, disobeying an adult). Accumulated points translate into more freedom and privileges. Both the girls in the MTFC and group care conditions were followed up from baseline over a 2-year period. During that time, only 27% of the girls in MTFC became pregnant compared with 46% of the girls in group care (Kerr et al. 2009). Importantly, this difference remained statistically significant after controlling for baseline age, criminal referrals, pregnancy history, and sexual activity. This finding is especially notable given that the MTFC group had substantially more opportunities to interact with male peers and thus, presumably, more chances to get pregnant.

In total, it appears that increasing the structure and predictability of the rearing environments of these very high risk girls caused them to delay reproductive activities. These results extend the descriptive, longitudinal research summarized above indicating that unpredictable childhood environments (i.e., parental transitions, residential changes) predict faster LH strategies. Taken together, these data provide reasonable support for the hypothesis that childhood exposures to stochastic conditions accelerate LH strategies.

Effects of Harsh versus Unpredictable Environments: The Case of Parental Investment

According to LH theory, when juveniles, but not adults, suffer relatively high levels of morbidity-mortality, and incremental changes in parental investment/offspring quality can significantly reduce this morbidity-mortality, natural selection should favor the evolution of slower LH strategies (see Fig. 1). An analogous logic applies to development. In the early years of life, quality of parental investment is the main conduit through which young children receive information about risks and opportunities in their environments. When extrinsic morbidity-mortality is low, parents have substantial capacity to shape conditions in ways that enhance the

health, competitiveness, and eventual reproductive success of their offspring; it should be advantageous, therefore, for parents to pursue a relatively slow LH strategy, investing a lot of resources in a limited number of competitive offspring, even if developmental conditions are harsh (i.e., high juvenile mortality) or unpredictable (i.e., high variation in juvenile mortality). The latter condition should shift parents toward conservative bet-hedging rather than a slower LH strategy per se, as discussed above. Along these lines, monogamous marriage and father-present social systems are more likely to be found among hunter-gatherers inhabiting harsh environments where biparental care (male provisioning) is substantial and important for offspring survival and reproductive success (Draper and Harpending 1988; Geary 2000; Kaplan and Lancaster 2003: Table 7–1; Marlowe 2003).

By contrast, when extrinsic morbidity-mortality is high, increases in parental care and resources (above a basic level) do not enhance offspring fitness. Under such conditions, it should be advantageous for parents to pursue a relatively fast LH strategy, focusing on mating effort, high offspring number, and low investment per offspring. When stochastic conditions cause high variation in morbidity-mortality in offspring, and increased parental effort does not shield offspring against this variation, then parents should shift toward diversified bet-hedging.

A key issue is, how do parents extract information from their environment about levels of and variation in extrinsic morbidity-mortality? What are the salient environmental cues that indicate whether high-quality parental investment can buffer children against harsh or unpredictable developmental conditions? A set of experiments with bonnet macaques (*Macaca radiata*), in which mothers were exposed to harsh versus unpredictable foraging conditions, suggests that exposures to stochastic environmental conditions may be especially likely to bias mothers toward low parental investment.

Infant bonnet macaques, along with their mothers, were placed in one of three ecological settings: (1) low foraging demand (LFD), where food was available ad libitum; (2) high foraging demand (HFD), where food was more difficult to obtain and widely dispersed within their enclosure; and (3) variable foraging demand (VFD), where foraging schedules oscillated between LFD and HFD in 2-week intervals. Typical studies ran for about 16 weeks, and no cues were present for the macaques in the VFD condition that would indicate the transition between foraging schedules (Rosenblum and Paully 1984; Rosenblum and Andrews 1994). Mothers in VFD conditions were the most aggressive toward other adults and engaged in the least grooming behavior. The VFD mothers also appeared to be more anxious and less responsive to their infants than either LFD or HFD mothers: They more frequently broke contact with their infants and tended to maintain greater spatial distances between themselves and their offspring than did the other mothers. Infants tended to respond to these maternal distancing behaviors by increasing attempts to elicit parental investment. Indeed, mother-initiated separation and infant return-to-contact scores were significantly higher in the VFD group than in either the LFD or the HFD group. The VFD infants also displayed less attachment security (showing less willingness to separate themselves from their mothers and explore a novel laboratory environment; Andrews and Rosenblum 1994).

The impact of the different foraging conditions on attachment styles could be interpreted from a LH perspective. Belsky (1999; Belsky et al. 1991) and Chisholm

(1996, 1999) have conceptualized attachment styles as phenotypic mechanisms that embody information about local environmental risk and uncertainty. Both theorists posit that different types of insecure attachment embody information about distinct types of childhood stress and function to guide development of alternative survival and reproductive strategies that are matched to these distinct childhood contexts. Although the bonnet macaque research is well-positioned to test these functional hypotheses, and the researchers followed the offspring into adulthood, no LH or reproductive outcomes were examined. Instead, the researchers focused on behavioral and neuroendocrine indicators of fear and anxiety. They found, more than 4 years after the manipulation of maternal foraging conditions, that VFD offspring were more timid, less gregarious, and more subordinate than their peers raised under stable conditions. Moreover, VFD offspring displayed relatively strong behavioral reactions to anxiety-provoking pharmacologic agents (Rosenblum and Andrews 1994) and abnormalities in their adrenocortical profiles (i.e., heightened concentrations of cerebrospinal fluid [CSF] corticotropin-releasing factor and reduced CSF cortisol levels; Coplan et al. 1996, 2001).

In total, although both harsh (HFD) and unpredictable (VFD) conditions undermined the quality and quantity of parental investment, environmental unpredictability had a significantly greater impact on parental functioning and subsequent child outcomes. This raises questions about the nature of information conveyed by low parental investment. Bonnet macaque mothers and offspring largely adapted to chronically harsh conditions. Most importantly, harsh conditions do not imply that parental investment is expendable. In fact, the opposite may be true. As discussed above, cross-cultural analyses of preliterate human societies indicate that harsh ecologies are associated with father presence and comparatively high levels of biparental care of offspring, presumably because high levels of maternal and paternal investment are needed to ensure child survival in this context. In fact, predictably harsh conditions may promote harsh parenting practices (e.g., harsh discipline, authoritarian parenting style) not because such environments undermine parental effort, but because it is important for parents to firmly control their children's behavior in environments characterized by high morbidity and mortality threats from predictable sources. Further, harsh conditions arising from high population densities and related energetic limitations favor the development of slower LH strategies (see Fig. 3).

Rather than arising from predictably harsh ecological conditions, low-parental-investment strategies may be driven primarily by (*a*) environmental unpredictability (stochastic conditions) and (*b*) cues that reliably signal high extrinsic morbidity-mortality (e.g., repeatedly attending funerals of adolescents and prime-age adults). Both factors may uniquely indicate that parents have limited ability to affect the survival and long-term reproductive outcomes of their offspring (i.e., that child morbidity-mortality is largely uncontrollable). It is well-established in past research that familial and ecological stressors—low SES, residence in dangerous neighborhoods, father absence, warfare, famine, high pathogen loads—are associated with low parental investment (e.g., Belsky et al. 1991; Ellis et al. 1999, 2003; McLloyd 1988; Quinlan 2007). Even among families in Western societies that have adequate bioenergetic and material resources to support reproduction, familial and ecological stressors undermine the quality and extent of parental investment. We propose that

these effects occur not because of chronic adversity, but because significant familial and ecological stressors provide cues to extrinsic morbidity-mortality and/or impose a level of unpredictability on the lives of parents that undermines parental motivation. Either way, the probable result is a facultative shift toward faster LH strategies (i.e., more mating effort, higher offspring number, less investment per child). Low-investment parenting strategies, in turn, signal extrinsic morbidity-mortality to offspring and should thus accelerate LH development in children and adolescents.

Summary and Conclusion

The LH strategies of individuals become adapted to their environments through two fundamental processes: evolution and development. Whereas natural and sexual selection adapt LH strategies to recurring environmental conditions encountered over evolutionary time, developmental experiences capture information that enables individuals to match LH strategies to environmental conditions encountered in their own lifetime. Through a combination of evolutionary and developmental responses to environmental harshness and unpredictability, organisms make predictable resource allocation trade-offs, and these trade-offs result in adaptive coordination between LH strategies and environmental conditions.

Environmental harshness indexes the rates at which external factors cause disability and death at each age in a population; environmental unpredictability constitutes levels of variation across time and space in environmental harshness. These overarching environmental factors shape the evolution and development of LH strategies. The effects of environmental harshness and unpredictability depend on such factors as age schedules of mortality, the extent to which rates of morbidity and mortality are sensitive to the resource-allocation decisions of parents and offspring, population densities and associated levels of resource scarcity and intraspecific competition, and the extent to which fluctuating environmental risks affect individuals versus populations over short versus long timescales. These interrelated factors operate at evolutionary *and* developmental levels and should be distinguished because they exert distinctive effects, are hierarchically operative in terms of primacy of influence, and affect ancestral, aboriginal, and contemporary societies to differing degrees. The fact that environmental harshness and unpredictability, and their various moderating conditions, operate in an interrelated manner—meaning that just knowing one of these environmental dimensions does not afford accurate prediction of evolution or development—necessitates substantial consideration of each.

When high levels of extrinsic morbidity-mortality either increase total mortality or disproportionately influence adult mortality, natural selection favors faster LH strategies. However, in species in which juveniles, but not adults, suffer relatively high levels of morbidity-mortality, the selection pressures change. In this context, the evolution of LH strategies depends on the sensitivity of juvenile disability and death to the resource-allocation decisions of parents and offspring. If incremental changes in parental investment/offspring quality significantly reduce juvenile morbidity-mortality, then natural selection should favor slower LH strategies. But under conditions in which juvenile disability and death are relatively insensitive to such

changes in parental investment/offspring quality, and refuge is obtained by achieving adult size or status, natural selection tends to favor rapid juvenile growth and development.

As externally imposed rates of morbidity-mortality decrease in a population, more diffuse patterns of LH evolution occur and density-dependent effects become a major agent of selection. Low rates of environmental harshness combined with more resource-rich environments select for faster LH strategies (greater reproductive effort and productivity). But as population density increases to approach the carrying capacity of the environment, intraspecific competition is heightened and slower LH strategies are favored by natural selection.

Unpredictable environmental conditions that cause high variation in adult mortality favor the evolution of relatively fast LH strategies. In contrast, stochastic conditions that result in widely varying levels of juvenile mortality favor the evolution of bet-hedging strategies that reduce variance in offspring fitness. Conservative bet-hedging involves producing offspring that are reasonably well equipped to handle the range of fluctuating conditions encountered over the organism's evolutionary history. When such offspring perform fairly well across this range, and/or when environmental changes affect an entire population on the timescale of a generation (e.g., years of drought) and thus cannot be handled through niche selection, natural selection tends to favor conservative bet-hedging. By contrast, diversified bet-hedging involves "spreading the risk" by increasing phenotypic variation among offspring; it is favored when environments vary substantially across individuals in a single generation and any single phenotype performs poorly across this range of changing conditions. These bet-hedging strategies increase the probability of achieving some reproductive success every generation while limiting success in good conditions and shielding against total failure in bad.

As reviewed above in "Impact of Harsh versus Unpredictable Environments on the Evolution of LH Strategies," the principles governing the effects of harsh versus unpredictable environments on the evolution of LH strategies have proven useful in explaining a wide range of variation in LH traits, both across and within species, including evolution of the slow modal human LH strategy and variation around that mode. A confluence of related factors has favored the notably slow human strategy: low extrinsic morbidity-mortality, with humans generally positioned as the top predator; improvement over hominid evolution in the ability to extract and process bioenergetic resources, enabling larger group sizes and higher population densities; high levels of competition for limited resources within and between increasingly complex social groups; and conservative bet-hedging in response to recurrent glacial and interglacial cycles over the past 200,000 years. At the same time, however, variation in environmental harshness and unpredictability has maintained differences between and within human populations in LH strategy. For example, local variation in mortality rates predicts differences in LH strategies across small-scale human societies. Further, short-term environmental unpredictability and change has favored diversified bet-hedging within human populations, resulting in the maintenance of genetic variation underlying individual differences in LH strategy.

The current developmental theory, as summarized in Figs. 3 and 4, builds on well-established theory and data from the field of LH evolution. We synthesized concepts and knowledge from the field to derive guiding principles and then employed these

principles to generate a series of testable hypotheses about the effects of variation in environmental harshness and unpredictability on the development of human LH strategies. All paths to the development of a fast LH strategy depend on there being adequate bioenergetic resources (low resource scarcity/energetic stress) to support growth and development. Given sufficient resources, environmental cues indicating high levels of extrinsic morbidity-mortality shift development toward relatively fast LH strategies. But if bioenergetic resources are in short supply, the resulting movement toward a slower LH strategy translates into development of a more energy-sparing phenotype.

Many modern human populations are characterized by low levels of resource scarcity/energetic stress (owing to highly efficient food production), low rates of extrinsic morbidity-mortality (owing to our position as the top predator and the general advances in diseases prevention and treatment), and high levels of population density/social competition (urbanization). The cooccurrence of these three factors should favor the development of slow LH strategies, including high parental investment to maximize offspring quality. By contrast, the combination of low levels of resource scarcity/energetic stress, low rates of extrinsic morbidity-mortality, and low levels of population density/social competition should promote fast LH strategies, since organisms should always benefit from accelerating LH strategies if there are no costs to doing so.

Both high absolute levels of adult morbidity-mortality (harshness) and high variation in adult morbidity-mortality (unpredictability) promote the development of fast LH strategies. This equivalency makes logical sense: both harshness and unpredictability present adult organisms with morbidity-mortality risks that are largely insensitive to their adaptive decisions or strategies (i.e., these risks are largely uncontrollable). Because levels of and variability in extrinsic morbidity-mortality are distinct factors, developmental exposures to environmental indicators of harshness and unpredictability should each uniquely contribute to acceleration of LH strategies.

The evolutionary logic changes, however, when harsh or unpredictable conditions primarily affect juveniles. The impact of high (disproportionate) juvenile morbidity-mortality on the development of LH strategies should depend on the sensitivity of this morbidity-mortality to the resource allocation strategies of parents and offspring. Under predictably harsh conditions, where parents and offspring can predict and meaningfully counteract external threats to offspring survival, parents should increase allocation of resources to offspring quality while offspring should increase allocations to maintenance. This prioritization of resources results in a developmental shift toward a relatively slow LH strategy. But when parental care (above a basic threshold) does not shield children against morbidity-mortality risks, parents can be expected to restrain investment levels. LH theory posits that juveniles should accelerate growth and development in this context, if obtaining adult size or status provides refuge against high juvenile-specific rates of morbidity-mortality. In addition, exposures to stochastic conditions (or reliable cues to environmental unpredictability) that signal widely varying levels of juvenile mortality should result in the development of bet-hedging strategies (diversified or conservative).

Because previous applications of LH theory to human development have not distinguished between the effects of harsh and unpredictable environments, many of the current hypotheses are novel and constitute new extensions of LH theory. Our

ability to evaluate the empirical status of the present developmental theory and derivative hypotheses, however, was at once tantalizing and incomplete. On the one hand, many lines of evidence supported the developmental hypotheses advanced in this paper (see solid lines in Figs. 3 and 4). Indeed, as reviewed in "Impact of Harsh versus Unpredictable Environments on Development of Human LH Strategies" of this article, there is good evidence that exposures to both harsh and unpredictable environmental conditions facultatively accelerate human LH strategies. On the other hand, because extant research was generally not designed to test the current hypotheses, many questions remain unanswered.

The current theory thus sets an agenda for future research on LH strategy. This agenda highlights the need for developmental scientists to distinguish between the effects of environmental harshness and environmental unpredictability (and interactions between them; see Brumbach et al. 2009 for an initial investigation); to consider the extent to which fluctuations in harsh environmental conditions are distributed across individuals versus populations and short versus long time periods; to consider age-graded effects of harsh and unpredictable environmental conditions on morbidity and mortality; to consider the extent to which rates of morbidity-mortality are sensitive to the resource-allocation decisions of individuals; to consider relations between extrinsic morbidity-mortality and energetic factors; and to delineate proximal cues to environmental harshness and unpredictability that provide inputs to the psychobiological mechanisms that regulate LH development.

In conclusion, we have attempted to demonstrate the value of applying a multilevel evolutionary and developmental approach to the analysis of a central feature of phenotypic variation: LH strategy. Elucidating how different types of harsh environmental conditions, and how stochastic variation in these conditions across time and space, affect the evolution of LH strategies provides a solid basis for generation of hypotheses about the development of LH strategies. Indeed, LH theory provides a foundation for addressing fundamental questions about human development: What are the evolutionarily relevant environments of the child? *How* do developmental experiences and genetic diversity influence the connecting series of resource-allocation trade-offs that form the individual's LH strategy? And *why* do these trade-offs systematically occur in response to varying levels of environmental harshness and unpredictability? It is our hope that the current review moved us closer to answering these questions.

Acknowledgments We are indebted to Jay Belsky and Steven Gangestad, for detailed comments on multiple drafts of this manuscript, and to Marco Del Giudice, for his thoughtful input to this work.

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Bruce Ellis is a professor of Family Studies and Human Development and the John & Doris Norton Endowed Chair in Fathers, Parenting, and Families at the University of Arizona. He seeks to integrate evolutionary and developmental perspectives in his research on family environments, child stress reactivity, and sexual development.

Aurelio José Figueredo is a professor of psychology at the University of Arizona and serves as director of the Graduate Program in Ethology and Evolutionary Psychology. His major area of research interest is the evolutionary psychology and behavioral development of life history strategy, sex, and violence in human and nonhuman animals. He also studies quantitative ethology and social development of insects, birds, and primates.

Barbara Hagenah Brumbach is an assistant professor in the Department of Psychology at Northern Arizona University. Her research examines individual differences in life history strategy and ecological predictors of the development of life history strategy over the life course.

Gabriel L. Schlomer (M.S.) is a Ph.D. student in the Family Studies and Human Development program at the University of Arizona. His research interests include developmental antecedents to human life history strategies, parent–child conflict, and adolescence.