Evolutionary Perspectives on Parenting

Chapter · September 2021

DOI: 10.1093/oxfordhb/9780190674687.013.1

2 authors:

Tomás Cabeza De Baca
National Institutes of Health

Bruce J Ellis
University of Utah

Evolutionary Perspectives on Parenting

Tomás Cabeza de Baca and Bruce J. Ellis

The Oxford Handbook of Evolutionary Psychology and Parenting
Edited by Viviana A. Weekes-Shackelford and Todd K. Shackelford

Print Publication Date: Oct 2021
Subject: Psychology, Social Psychology, Developmental Psychology
Online Publication Date: Sep 2021 DOI: 10.1093/oxfordhb/9780190674687.013.1

Abstract and Keywords

This chapter highlights parental behavior and household dynamics as one key set of factors that play a vital role in the regulation of development and behavior in children. It shows the benefits of using an evolutionary-developmental model and discusses fundamental points to consider when applying such a model to research. The application of Darwinian principles to domains of parenting and development provides a benefit to researchers by integrating the results of proximate-level research into a unified and interconnected framework. It also reorients within- and between-household differences in parenting as strategies molded by natural selection to maximize survival and reproduction under varied ecological circumstances. The chapter begins with a review of the theoretical foundations of evolutionary developmental psychology, followed by a brief explanation of methodological approaches used by developmentalists. It then looks at current research and future directions.

Keywords: parental behavior, household dynamics, evolutionary-developmental model, Darwinian principles, parenting, natural selection, evolutionary developmental psychology, developmentalists

For many organisms, the evolutionary criteria for success are survival, reproduction, and raising reproductively viable offspring. *Homo sapiens*, currently and over the course of our evolutionary history, has encountered a range of social, technological, political, and geographical milieu that we have had to navigate successfully to achieve reproductive success. Modern-day humans are a testament to the fact that our ancestors have been adept at surviving the social, political, and geographical contexts that they have encountered. Our adaptations, however, must also include the modes in which we raise our children. Natural selection will have played a major role in shaping parental behavior (Geary & Flinn, 2001). Although the environments we encounter today have changed from those encountered by our ancestors, the mechanisms that shaped their development and parenting strategies continue to operate today. Parental behavior—even today—should be attuned to and a reflection of our social and ecological environments.

Children's development and behavior are influenced by a multitude of nested and intertwining factors. The present chapter highlights parental behavior and household (p. 4) dynamics as one key set of factors that play a vital role in the regulation of development and behavior in children. This chapter also showcases the benefits of using an evolutionary-developmental model and discusses fundamental points to consider when applying such a model to research. The application of Darwinian principles to domains of parenting and development provides a benefit to researchers by integrating the results of proximate-level research into a unified and interconnected framework. It also reorients within- and between-household differences in parenting as strategies molded by natural selection to maximize survival and reproduction under varied ecological circumstances. Traditional developmental models of parenting typically rely on the modus operandi that aspects of "good" parenting, broadly defined, correspond with "good" child outcomes (Cabeza de Baca et al., 2012). Based on this premise, these traditional developmental models mainly focus on the impact of the immediate environment, known as *proximate effects.* Although these are important, we argue that it is important to also consider *functional* or *ultimate* interpretations of behavior that are key components of an evolutionary-developmental framework. Thus, functional or ultimate interpretations of behavior reframe parental behavior and child development as *adaptive responses* to features and experiences of the present ecology. Interpreting behaviors as functional responses to ecological constraints implies that models interpreting parenting and child outcomes as "good" or "bad" (often informed by prevailing views within Western, educated, industrialized, rich, and democratic [WEIRD] societies; Henrich et al., 2010) may not be appropriate. We argue that parental behavior should be viewed as being both ecologically relative and contingent on the household and neighborhood in which the child and his or her parents reside.

Individual differences in parental behavior include between- and within-family differences. An evolutionary approach to parenting examines parental behavior and developmental outcomes such that individual- and family-level variation are adaptive outcomes to ecological constraints. The purpose of this chapter is to provide a theoretical and methodological foundation to family and developmental researchers to integrate evolutionary and life history approaches into their respective research interests. We specifically showcase the application of evolutionary theory to parental behavior and family dynamics, highlight the integrative nature of evolution on family and developmental psychology, and emphasize the generation of hypotheses unique to an evolutionary perspective. We particularly focus on the progress and the present state of evolutionary developmental psychology. Our review is divided into three main sections: (a) First, we provide a brief review of the theoretical foundations of evolutionary developmental psychology, followed by a (b) brief explanation of methodological approaches used by developmentalists. We conclude by discussing (c) current research and future directions.

Theoretical Foundations of Evolutionary Developmental Psychology

The central premise of evolutionary psychology is to apply key tenets of ecology and evolutionary biology toward research questions and problems broadly encountered in the field (p. 5) of psychology. As such, evolutionary developmental psychology is a subfield that integrates existing developmental theory, literature, and knowledge with evolutionary models and hypotheses. The field broadly posits that humans have recurrently encountered a delimited set of reproductive and survival problems over our evolutionary history, and that our developmental systems—as instantiated in morphology, physiology, and behavior—have been shaped by natural selection to solve these problems, especially through conditional adaptations to varying environmental conditions. Although the integration of these two fields that emphasize different sets of life courses—developmental and phylogenetic—appears intuitive, evolutionary developmental psychology has been met with some resistance from traditional developmentalists, who view evolutionary theory as incongruent with their personal and academic agendas of alleviating suffering across vulnerable populations such as children, women, and ethnic minorities (Charlesworth, 1992; King & Cabeza de Baca, 2011). Other critics object to evolutionary approaches because they believe that they promote genetically deterministic views (i.e., all traits, behaviors, and outcomes are the result of genetic forces rather than bidirectional gene × environment interactions at all levels of analysis; Hagen, 2005).

The concerns put forth by traditional developmentalists about adopting an evolutionary framework are sincere but misguided. Evolutionary developmental psychology is not incongruent with melioristic viewpoints of alleviating suffering and inequality among vulnerable populations. In fact, think-tanks (e.g., www.evolution-institute.org) and proposed translational interventions informed by evolutionary-developmental approaches (Ellis et al., 2012a, 2017a, 2017b) are in line with the goals of combating systemic inequality. The field of evolutionary developmental psychology is also not genetically deterministic. The field, as a whole, acknowledges the role of heritable variation in shaping the development and expression of phenotypic traits; however, we assert that the expression of traits is highly plastic and contingent on the ecological input the organism receives early in development (Fawcett & Frankenhuis, 2015; West-Eberhard, 2003). Sophisticated advancements since Darwin continue to support natural selection, but they have, notably, also extended its impact by incorporating genomic and physiological mechanisms not known during Darwin's time, notably epigenetics (Jablonka & Raz, 2009) and gene × environment transactions (Meaney, 2010).

Evolutionary developmental psychology typically employs *life history theory* to examine and explain variation in parenting effort and behavior as conditional adaptations to levels of ecological predictability and resource availability (Chisholm et al., 1999; Ellis et al., 2009). This framework systematically links early childhood experiences to psychological/behavioral development, sexual and reproductive development, and, ultimately, health and longevity. In order for our discussion of life history theory to make sense, we discuss the metatheoretical foundations that guide and inform life history theory and other middle-level theories of evolution.

(p. 6) Natural Selection

The field of evolutionary developmental psychology uses the theory of natural selection as an overarching *metatheory* to organize and explain the function and ultimate causes of behavior and to integrate across the fields of psychology with those in the life sciences (e.g., Ellis & Ketelaar, 2000). Evolution by natural selection is built upon four premises (Barrett et al., 2002): First, individual differences (i.e., variation) exist between organisms of the same species across a variety of phenotypic traits. Second, some of this variation is heritable (and thus can be transmitted from parents to offspring through biological mechanisms). Third, organisms will produce more offspring than can be sustained by the environment or that will successfully reproduce. As such, intense competition exists between organisms for access to resources. Finally, organisms with heritable variants that enable them to compete successfully and adapt to local environmental conditions have more descendants than those with heritable variations that are less suited to the environment. This process of differential survival and reproduction results in small incremental modifications in existing phenotypes, leading to an accumulation of characteristics that are organized to enhance reproductive success. These characteristics that are produced by natural selection are termed *adaptations*.

Life History Theory

Whereas evolution by natural selection emphasizes species-typical development in function and morphology across time and context (e.g., the Environment of Evolutionary Adaptedness; Tooby & Cosmides, 1990), life history theory (e.g., Del Giudice et al., 2015; Ellis et al., 2009) considers the adaptive role of individual differences by focusing on context-dependent developmental plasticity across the life course of the individual. Individuals must strategically allocate time and bioenergetic resources toward different facets of development in ways that sufficiently maximize their survival and reproduction. The major conundrum an organism faces is that time and energy are finite. This means that individuals, consciously and unconsciously, must make developmental trade-offs and prioritize how to devote their time, effort, and bioenergetics resources. The fundamental trade-off is between *reproductive effort* (e.g., time and resources allocated toward mating and parenting, broadly) and *somatic effort* (e.g., time and resources allocated toward the growth, maintenance, and health). Trade-offs within reproductive effort are further broken down between *parental/nepotistic effort*, whereby individuals invest time and resources in raising and caring for their offspring and the offspring of closely related kin, and *mating effort*, time, energy, and behavior oriented toward increasing mating opportunities (Del Giudice et al., 2015). Thus, the parental strategies that an individual pursues should be a product of unconscious and conscious decision-making in response to context-relevant information.

(p. 7) Psychosocial Acceleration Theory

Psychosocial acceleration theory, advanced by Belsky and colleagues (1991), sought to integrate social learning and attachment theory, theories central to developmental psychology, with life history theory. Their intent was to advance an evolutionarily informed model of child socialization that incorporated physical and reproductive development with traditional psychosocial domains often explored in traditional psychology (see Figure 1.1). The first 5 to 7 years of a child's life is a sensitive period for shaping developmental trajectories across the domains purported in their model. These years are theorized to be vital because caregiver attachment is at its most salient and impactful and, thus, provides the developing child with key information about the predictability and availability of resources, degree of interpersonal cooperation, and the strength of romantic pair-bonds. If a child experiences a household inundated with romantic/family conflict and increased psychosocial stress, the child's development will be more likely to (p. 8) be shaped in a manner that orients a child toward a fast life history strategy (left column of Figure 1.1). A fast life history strategy prioritizes high reproductive output, which begins by a shortening of childhood through early menarche (Ellis, 2004).

Over the course of the child's life, these developmental trade-offs are instantiated across a coordinated suite of behaviors and traits, collectively known and organized as *life history strategies*. Regulation across these developmental trade-offs is steered by a combination of environmental (Del Giudice, 2014a) and heritable factors (Figueredo & Rushton, 2009). Life history strategies reside on a slow–fast continuum in which different "strategies" emphasize different facets of development. For instance, slow life history strategists are oriented toward investing in long-term social and romantic relationships. When they have children, they often have them at an older age and limit the number of children they have (see Figure 1.1). The household milieu in which they raise their children is characterized by warmth, support, and responsiveness.

The current chapter focuses on how early family and household factors shape appraisals of the environment and cognition, regulate the peripheral neuroendocrine systems, and impact sexual development. We conclude by introducing the emerging integration of health and development.

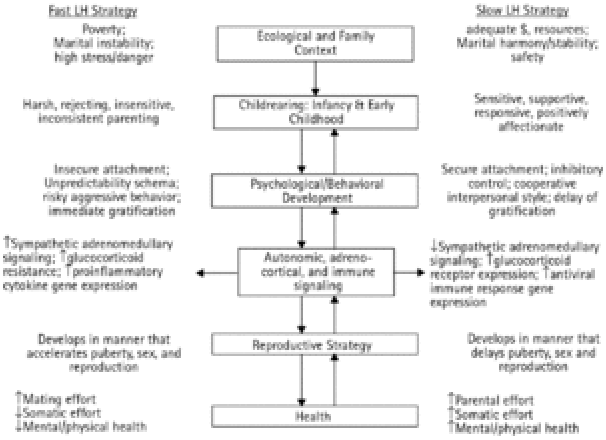

Figure 1.1 This is a schematic figure first proposed by Belsky et al. (1991) and modified by Cabeza de Baca and Ellis (2017). Life history models of development propose that the first 5–7 years of life is a vital time for the calibration of life history strategies. The dynamics of early ecological and family context and the quality of parenting impact the development of the child across key developmental domains that range from psychological to health. As noted by the columns, life history strategies range on a continuum from fast to slow. The different life history strategies emphasize different trade-offs: Slower life history strategies favor investment in high-quality parenting and health; faster life history strategists will invest time and bioenergetic resources toward greater mating effort, reducing investment in health. It is important to note that there is a bidirectional and transactional relationship between the facets of development. Different facets proposed can mutually impact other facets. The horizontal arrows propose that physiological systems may modulate and regulate the pathways and facets.

Evolutionary Approaches to Parenting: Methodological Foundations

Examining the impact of parenting on child behavior has been investigated with many methodologies across many different academic disciplines. This makes sense because evolutionary frameworks can be applied to research in many different fields. The purpose of this section is to describe briefly a selection of methodological approaches that are used by evolutionary developmental scientists to investigate parenting. Here we present four methodological approaches: (1) Comparative/Nonhuman, (2) Experimental, (3) Ethological/Observational, and (4) Sibling/Behavioral Genetic. It is important to note that these four approaches are not distinct and often overlap with each other.

Comparative/Nonhuman Research

Evolutionary biology and ecology have a rich history of utilizing comparative research with nonhuman animals. The heart of evolutionary theory began with Charles Darwin's phenotypic comparisons of beak size and morphology between different species of finch in the Galapagos (Darwin, 1859). More than 150 years later, researchers continue to use sophisticatedly designed animal models to test evolutionary hypotheses and inform parental research (see Bales, 2017, for a brief review of animal literature and parenting). Use of animal models in parental research facilitates and informs human research in many ways. Foremost, because animals have shorter life spans, longitudinal or intergenerational effects documented over several cohorts can be tracked over considerable time. Relatedly, manipulation of certain conditions (e.g., housing, food, stress exposure) that are deemed unethical in humans can be performed in animal models. Foundational animal models, (p. 9) which have used a "cross-fostering" paradigm to control for genetic and environmental confounds (reviewed in Meaney, 2010) have shown that high-quality parenting (indexed by high levels of licking and grooming in rodents) down-regulates autonomic and adrenocortical stress reactivity. This calibration of stress responsivity presumably prepares the developing pup for a relatively safe, stable environment. This calibration is instantiated mechanistically through methylation of glucocorticoid receptors in hippocampal neurons (Liu et al., 1997; Weaver et al., 2004). Comparative or cross-species research also attempts to delineate evolutionary history by comparing and contrasting behavior and/or morphology across diverse taxa. This work involves comparison of neural circuitry (Panksepp et al., 2002) as well as variation in levels of parental investment within and across species (Clutton-Brock, 1991).

Experimental Approaches

Experimental design is a powerful tool that allows for the control of extraneous variables, the manipulation of key variables of interest, random assignment of participants, and replication across different samples. The ultimate purpose of experimental approaches is to show, with some level of certainty, that certain variables, when manipulated, exert an effect or outcome. Previous evolutionary theory (Draper & Harpending, 1982; Ellis, 2004) has denoted the importance of paternal presence on the sexual development and behavior of girls. Briefly, these theories contend that presence or absence of a father within the household indicates the availability and stability of resources accessible to the developing child. Further, the presence or absence of a father is also purported to be a salient marker for the mating dynamics present in the child's local ecology. Paternal absence denotes a local mating ecology characterized by transient relationships, unstable pair-bonds, and increased sociosexuality. Most of the research examining this evolutionary question has been correlational in nature, since manipulation of paternal presence in households would be unethical and impossible to perform in humans (see section on "Sibling Design/ Behavioral Genetics"). Del Priore and Hill (2013), however, used an experimental social psychological method to test hypotheses derived from *paternal investment theory* (Ellis, 2004). Paternal presence or absence was manipulated in young women using a priming paradigm to evoke feelings of psychological or physical abandonment (e.g., "Take a few seconds to think back to a time when your biological father was absent for an important life event when you really needed him. Describe in detail how your father's lack of support—or his physical or psychological absence—made you feel"; Del Priore & Hill, 2013, p. 236). Across five studies, results revealed that young women primed by paternal disengagement were more likely to endorse permissive sexual attitudes, have an increased number of desired sexual partners, have increased sexualized thoughts, and have greater negativity toward condom use than other groups (Del Priore & Hill, 2013). The use of experimental design facilitated testing of alternative explanations by accounting for extraneous variables. Based on the results, increased risky sexual (p. 10) attitudes and behavior were specific to women, were not accounted for by other forms of disengagement or abandonment (e.g., friends and mother), and were not the result of negative affect from the experimental prime. Taken together, this set of studies eloquently used the power of experimental design to provide robust support for evolutionary developmental hypotheses that have been mostly supported by correlational designs.

Ethology/Observational Studies

The most common observational approach in developmental psychology is to invite children and their parents to a laboratory, record their interactions during a standardized task, and use multiple raters to code and classify their behaviors (e.g., Barnett et al., 2008). Most animal and early cross-cultural research utilized ethological methods to observe, code, and describe behaviors. Like developmental laboratory methods, these observational methods were grounded in a taxonomy of behaviors coded by trained raters in "real time" and within naturalistic settings. Observing organisms in their natural element, ethologists argued, would showcase constellations of behaviors that were adaptive to local conditions. Flinn (2006), an evolutionary anthropologist, has used ethological methods to observe and record behaviors of family members residing in the Caribbean island of Dominica and investigate the impact of these behaviors on child development and health. One prospective naturalistic study conducted from 1988 to 1994 involved tracking the composition and dynamics of 82 Dominican households, using a combination of interview and observational methods. Additionally, cortisol derived from the saliva of the focal child was collected twice daily (Flinn & England, 1995). The work was notable for advancing an evolutionary developmental understanding of the impact of household dynamics and parenting. This research indicated that the stress response system, captured by cortisol levels, is sensitive to the dynamics and composition of the family. Children residing in households with stepfathers, half-siblings, or in single-mother households without alloparental support had the highest levels of cortisol. The combination of having nonrelated or distantly related kin and low social support increased the frequency of conflict witnessed or experienced by the child. The release of cortisol following conflict or stressful experiences may have helped overcome the stressors in the short term, but, in the long term, it presumably calibrated the stress response system and the life history trajectory of the child.

Sibling Design/Behavioral Genetics

Behavioral genetic approaches examine the extent of environmental and heritable influence on traits and characteristics by using sibling, adoption, or twin designs (Segal, 1993). The use of monozygotic and dizygotic twins allows the conceptual disaggregation of genetic and environmental sources of variation in individual differences through the use of bivariate correlations (e.g., Falconer Formula; Roff, 2012) to complex biometric models through structural equation modeling (Rijsdijk & Sham, 2002). Use of these (p. 11) research designs has been helpful for addressing evolutionary questions that are of interest to developmentalists (Segal, 1993; see Schlomer & Ellis, 2016, for a basic summary). One key example has been the use of the sibling design to examine the impact of household influences on reproductive and sexual development in girls (DelPriore et al., 2017; Ellis et al., 2012b; Tither & Ellis, 2008). As described earlier, paternal investment theory (Ellis, 2004) posits that father absence and/or exposure to paternal dysfunction accelerates sexual development by orienting young girls toward early pubertal timing and early sexual behavior. Research supporting this hypothesis has largely been correlational, representing a major limitation. Critics of this literature have argued that (1) the effects of environment and heritability on pubertal timing should be disentangled to avoid confounds (Rowe, 1994) and (2) the constellation of sexual development and behavior are largely heritable (Rowe, 2002). The incorporation of the sibling design to test paternal investment theory suggests that pubertal timing and sexual behavior may have a conditionally adaptive component (Ellis et al., 2012b; Tither & Ellis, 2008), though not all behavior genetic analyses have supported a causal effect of fathers (reviewed in Barbaro et al., 2017).

The differential sibling-exposure design (Tither & Ellis, 2008) is both genetically controlled (i.e., participants are biological sisters, and genetic differences between sisters vary randomly across birth order) and environmentally controlled (participants lived in the same household). The sibling design additionally facilitated a “natural experiment” approach to father absence. As age of menarche has a significant heritable component (e.g., $h^2 \sim .50$; Rowe, 2002), any systematic deviation in pubertal timing between the sisters as a function of birth order would be explained by early differential household experiences. Sisters of discrepant ages from divorced households experienced varying levels of exposure to paternal absence, with younger sisters receiving a greater “dosage” of paternal absence. As hypothesized, younger sisters in divorced households had earlier menarche than their older siblings; the effect was moderated by paternal social deviance such that young sisters in divorced households experienced even earlier puberty if they were exposed to greater paternal social deviance (Tither & Ellis, 2008).

Early Attachment and Childhood Experiences Shape Socioemotional and Cognitive Tools

> Hush now baby, baby, don’t you cry. Mother’s gonna make all your nightmares come true. Mother’s gonna put all her fears into you.

—*"Mother" by Pink Floyd*

Developmental models of life history (e.g., Belsky et al., 1991; Chisholm et al., 1993; Ellis et al., 2009) emphasize the saliency of ecological cues of adult *extrinsic morbidity-mortality*—external sources of disability and death that are relatively insensitive to the adaptive decisions or strategies of the organism—on the development and orientation of faster life history strategies (Ellis et al., 2009; Promislow & Harvey, 1990). Psychosocial (p. 12) acceleration theory proposed by Belsky and colleagues (1991) discusses the pathways by which household dynamics and parental behavior may convey important information to the child about the state of the "outside world's" level of extrinsic morbidity-mortality. Households marked by unpredictability and conflict, compounded by low parental effort (e.g., low levels of sensitivity/monitoring, high levels of abuse/neglect), convey to the developing child that the immediate world outside their household will be harsh, conflictual, and unpredictable (Dunkel et al., 2015; Figueredo et al., 2015).

Similar to attachment theory (Bowlby, 1969), life history models propose that repeated and pervasive interactions with caregivers calibrate "inner working models" to attachment figures; however, evolutionary developmental scientists take the predictions by Bowlby further and posit that attachment will not only shape attachment survival strategies in childhood but also will impact attachment with romantic partners in adulthood and alter reproductive development and strategies through neuroendocrine pathways (Belsky et al., 1991; Del Giudice, 2009, 2011). Accordingly, stress-adapted individuals may develop sexual strategies *in addition* to the socioemotional and cognitive capacities that fit their perceived social and romantic world (Figure 1.1: Psychological/Behavioral Development).

The brain is a sensitive "social tool" used to actively monitor and respond to social challenges presented to the developing child (Flinn, 2006). Any environmentally relevant appraisal captured by the brain should emerge across different physiological and behavioral systems—such as in socioemotional and cognitive functioning (Cabeza de Baca et al., 2016b). The socioemotional and cognitive tools needed to navigate in a controllable, predictable, and cooperative environment should vary compared to uncontrollable, unpredictable, and hostile environments (Frankenhuis et al., 2016). Much like Bowlby's inner working models of attachment applied to the world, generally, evolutionary theorists propose *unpredictability schemas*, or worldviews and appraisals that life may be stressful and short and that the world and others are unreliable, threatening, and unpredictable (see Figure 1.1: Psychological/Behavioral Development; Belsky et al., 1991; Ross & Hill, 2002).

The majority of the work investigating unpredictability has focused on family or household-level indicators of unpredictability, including lack of routines, nurturance, or discipline, changes in caregivers or parental relationships, and residential or school moves (Belsky et al., 2012; Matheny et al., 1995; Ross & Hill, 2000; Simpson et al., 2012). This work has found that individuals who report living in low-quality households characterized by poor family routines or reduced maternal investment had developed higher levels of unpredictability schema (Cabeza de Baca et al., 2016a; Ross et al., 2016a, 2016b). In college students, a retrospective report of family-level unpredictability (denoted by inconsistent nurturance, discipline, and family meals) was linked to an increased unpredictability schema (Ross et al., 2016a). The report also suggested that sustained levels of appraised unpredictability may lead to mental distress. Individual reports of appraised unpredictability schema mediated the association between family-level unpredictability (p. 13) and depressive-anxious symptoms (Ross et al., 2016a). Thus, although these schemas may facilitate navigation in deleterious environments, sustained hypervigilance from these perceptions may come at mental health costs.

Parental resources and condition is a component of household context for the developing child, shaping the quality of investment received by the child and, ultimately, altering life history trajectories (Belsky et al., 1991; Schlomer et al., 2011). Unpredictable conditions experienced in childhood can be transmitted across generations, decreasing men's use of supportive parental approaches (Szepsenwol et al., 2015). Early harshness (operationalized by lower socioeconomic status) and unpredictability (operationalized by greater residential or caregiver changes) predicts increased maternal depression in longitudinal models. Maternal depression, in turn, is associated with increased household instability, greater parental hassles, and increased harsh parenting in childhood—and this predicted earlier onset of sexual activity (Belsky et al., 2012; Shelleby et al., 2014). This brief review of early parental factors illustrates that household experiences alter worldviews about others and of one's environment and can impact reproductive strategies.

In modern society, poverty is a robust indicator of extrinsic morbidity-mortality (Adler & Ostrove, 1999; Ellis et al., 2009; Williams & Collins, 1995). In general, individuals with lower socioeconomic status believe they will live shorter lives (Mirowsky & Ross, 2000). In addition to these appraisals, ethnic minorities are exposed to more family deaths (e.g., parents, spouses, and children) at an earlier age in their lives than Whites (Umberson et al., 2017), shaping their reproductive behaviors in conscious ways (Burton, 1990; Geronimus, 1996). The unique experiences of vulnerable or minority populations residing in resource-scarce and harsh environments include cues that signal brief rather than lengthy lives, impacting assessments of life expectancy (Cabeza de Baca et al., 2016b). As stated by Ellis et al. (2009, p. 245):

> 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.… 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.

Chronic exposure to environmental cues of extrinsic morbidity-mortality alters an individual's "death perceptions," known as subjective life expectancy. Research indicates that early direct and indirect exposure to violence modulates one's perceived life expectancy (Warner & Swisher, 2014). If one's appraisal of the environment is unpredictable, time and death perceptions should be oriented to fit a shorter, transient existence. Orientation toward a shorter life encourages divestment in the future, with an emphasis on the (p. 14) present (Daly & Wilson, 2005). What does this mean from an evolutionary perspective? Perceptions of a shorter life are expected to orient individuals toward faster life history strategies, primarily diverting time and bioenergetic resources away from parenting and health and toward mating and reproduction (Ellis et al., 2009). This life history trade-off is instantiated across a wide range of behavioral and socioemotional outcomes, including increased risk-taking (emphasis on deviant or risky sexual behavior; e.g., Borowsky et al., 2009; Piquero, 2016) and decreased engagement in health-maintaining behaviors (e.g., McDade et al., 2011; Scott-Sheldon et al., 2010).

Beyond shifts in worldviews and appraisals (i.e., the unpredictability schema), stress-adapted individuals have shifts in cognitive abilities across an assortment of tasks that may reflect neural reorganization between stress-adapted and non-stress-adapted individuals (Ellis et al., 2017a; Frankenhuis et al., 2016). Models informed by life history theory propose two key sets of hypotheses regarding cognitive abilities: the *specialization hypothesis* and the *sensitization hypothesis* (Ellis et al., 2017a). The specialization hypothesis advanced by evolutionary developmental psychologists proposes that developmental exposure to stress does not so much impair cognition (as per traditional deficit models) as direct or regulate it toward development of skills and abilities that are adaptive under high-stress conditions (e.g., harshness, unpredictability). The sensitization hypothesis argues that brain and cognitive specializations formed from growing up in harsh and unpredictable environments will emerge during current stressful conditions. Research supporting these two hypotheses should manifest effects at two levels of organization: (1) brain circuitry and neural architecture and (2) individual differences in cognitive abilities.

Exposures to deprived or stressful environments associated with poverty in childhood produces major neural and brain reorganization (Gianaros & Hackman, 2013; Gianaros & Wager, 2015). Allostatic load models of development interpret these outcomes as brain injuries resulting from sustained exposure to neurotoxic stress hormones (McEwen & Gianaros, 2010). At the proximate level, this is correct, but we must ask, why? Why would the brain be designed to be susceptible to these stress hormones? If life history theory's focus is on the allocation of finite resources across different facets of development to maximize survival and reproduction, then brain reorganization and neural pruning can be interpreted as prioritization to maintain brain circuitry that best adapts the individual to his or her environment (Figueredo et al., 2006). Because brains are calorically costly (Flinn, 2006), pruning may serve to conserve costly resources in addition to maintaining quicker access to neural circuitry. Research examining the overall structure of the brain finds that higher household income and a decreased neighborhood disadvantage (i.e., harshness) was associated with an increase in white brain matter (Gianaros et al., 2013). White matter in the brain is comprised of myelinated neural tissue and accounts for over half of the brain (Fields, 2010). Other structural changes have been documented. Animal models and human research have documented structural changes in areas of the brain associated with learning and memory and emotion processing and (p. 15) decision-making (reviewed in Hanson et al., 2015). Selective pruning of these areas (e.g., the amygdala and hippocampus) will result in an emergence of behaviors and cognitions oriented toward impulsivity, aggression, and risk-taking (Figueredo et al., 2006). A study examining the association between cavum septum pellucidum (CSP) volume (an indicator of brain abnormality) and the *K*-Factor, a psychometric assessment of slow life history, found that individuals with greater CSP volume had faster life history strategies (Dunkel et al., 2017). Greater CSP volume has been linked to a range of clinical disorders and subclinical behavior, including increased impulsivity and callousness (White et al., 2013).

Executive functioning tasks designed to capture prefrontal functioning (lower impulsivity, aggression, and planning/insight/control) have identified noted differences in executive functioning in individuals residing in adverse conditions. Fine-grained analyses that disentangle the different facets of executive function report differences that are relevant from a life history perspective. For instance, individuals who grew up in households that were harsh and unpredictable and were exposed to a lab stressor exposing them to economic uncertainty showed enhanced performance on attention-shifting tasks (Mittal et al., 2015). Enhancement on attention-shifting tasks may facilitate greater vigilance needed to navigate an unsafe environment with unpredictable threats.

Early Environmental Context Regulates Physiological and Reproductive Systems

Psychosocial acceleration theory (Belsky et al., 1991) has been used to generate the hypothesis that household and environmental conditions regulate the development of life history trajectories, including sexual development. It specifically posited that family dynamics and household quality (including father absence/presence; Ellis, 2004) influence pubertal timing—an important decision node regulating the timing and emergence of sexual and reproductive strategies. Since putting forth their hypotheses, many scholars have built upon Belsky et al.'s work, suggesting the need for integration with physiology (Ricklefs & Wikelski, 2002) and articulating the physiological pathways that extend Belsky et al.'s hypothesis. Here we discuss the impact of early life stress on physiological systems. We then follow with a discussion of how physiological systems—mainly those tasked with stress response—impact sexual development.

The Adaptive Calibration Model (ACM; Del Giudice et al., 2011; Ellis & Del Giudice, 2014) proposes that physiological systems, including the immune, metabolic, neuroendocrine, and autonomic systems, orchestrate the development of life history trajectories that best fit the individual's immediate environment (see Autonomic, Adrenocortical, and Immune signaling in Figure 1.1). Repeated and chronic activation of these physiological systems in early childhood provides statistical summaries about the state of the environment, including the safety of the environment, the number of potential threats, and the severity of those threats. For instance, early childhood conditions have been shown to impact epigenetic markers within the hypothalamic-pituitary-adrenal (HPA) axis (p. 16) associated with regulation of the stress response system, such as the glucocorticoid receptor exon 1F in humans (reviewed in Turecki, & Meaney, 2016). This suggests that the HPA axis tracks different relevant stressors, including dangerous environments/situations, uncertain/unpredictable contexts, and social evaluative stress (Slavich et al., 2010). Short-term activation of the HPA facilitates overcoming “unpredictable, uncontrollable events that require alert readiness and mental anticipation” (Flinn & England, 1995, p. 855). Sustained activation of this system provides feedback to other physiological systems that potential threats may be present in the environment and may perpetuate regulatory alterations across physiological, developmental, and behavioral domains. For instance, mothers put into physiologically reactive situations in laboratory tests transmit their physiological reactivity toward their infants when reunited (Waters et al., 2014). They further found that the reactive synchrony between mother and child increased over time, following the task. They hypothesize that the underlying mechanism for physiological transmission has been attributed to maternal touch, transmitting, nonverbally, the condition of the social milieu the mother is experiencing. If this is so, mothers may be providing feedback about the environment to their young children and shaping their stress response systems through touch (see Meaney, 2010, for an analogous animal model).

The immune system is similarly equipped to track key facets of ecological threats and social support (McDade et al., 2016). For instance, observed and coded parent–child conflict was negatively associated with GR-α and β2-AR receptors linked to regulation of anti-inflammatory signaling in adolescents (Ehlrich et al., 2015). More conflict predicted a down-regulation of the products of these alleles, potentially signaling an overactivation of the immune system. The patterns of findings are similar for older adults. In a subsample of the MIDUS (Midlife in the United States) dataset, adverse childhood experience was directly associated with a composited biological measure of inflammation (Hostinar et al., 2015). The association could also be the result of sustained activation of the immune system from recent life events. The results also revealed a direct association between stressful life events experienced within the last 5 years (e.g., fired from a job, death of a child) and increased inflammation. Over the course of the individual’s lifetime, these physiological systems adaptively calibrate by setting the physiological parameters and reactivity patterns. Research finds a link between early adversity and increased activation of the immune system, indexed by higher levels of inflammatory markers in adulthood (Baumeister et al., 2016).

Although the parameters of these physiological systems—including the stress response system—are highly plastic and reside on a slow–fast life history continuum, Del Giudice and colleagues (2011) have suggested that four physiological patterns exist across varying levels of physiological responsivity and across developmental contexts (see figure 2 in Del Guidice et al., 2011, p. 1577): sensitive, buffered, vigilant, and unemotional. Research specifically investigating the existence of the four ACM patterns in Dutch adolescent males (TRAILS dataset; Tracking Adolescents’ Individual Lives Survey) identified the (p. 17) four-pattern ACM classification through latent profile analysis of the sympathetic and parasympathetic nervous systems and the HPA axis (Ellis et al., 2017b). The longitudinal model tracked the participants between the ages of 11 and 16 years, securing data about household and environmental dynamics and socioemotional and cognitive functioning. As hypothesized by the ACM, the model identified key household and environmental antecedents across the different groups. Males classified as vigilant, denoted by high reactivity, were found to have the highest levels of childhood stress and prebirth risk factors; in comparison, the sensitive group, males denoted by high reactivity but purported to reside in safe environments, had reports of residing in high-quality households and low levels of childhood stress (Ellis et al., 2017b). Behavioral reports also varied across the groups: The vigilant group had the highest level of depressive symptomology and withdrawn behavior, as predicted by the ACM; conversely, the sensitive subgroup had the lowest level of aggressive behavior but had slightly elevated levels of depression symptomology and withdrawn behavior.

As noted, physiological systems within the individual are coordinated in a manner such that each system provides a transactional role with the other systems. Of importance, the stress response system assists in regulation of sexual development and reproduction along with the hypothalamic-pituitary-adrenal-gonadal axis (HPG; Del Guidice et al., 2011; Hoyt & Falconi, 2015; Marceau et al., 2015). In conjunction, hormones from these systems during middle childhood (Del Giudice, 2014b) and into puberty produce neural reorganization in the brain (Juraska & Willing, 2017), possibly in anticipation for the sociosexual environment the child will face in adulthood (Figueredo et al., 2006). Prior to reproduction, individuals need to mature sexually. In adverse conditions inundated with harshness and instability, sustained activation of stress response systems may signal to reproductive systems (e.g., HPG) to accelerate sexual maturation, shortening childhood and taking advantage of reproduction opportunities (see Figure 1.1: Reproductive Strategy). A large body of research has documented that early adversity and psychosocial stress predict early puberty and earlier engagement in sexual and reproductive behavior (reviewed in Ellis, 2004; Amir et al., 2016; Belsky et al., 2015; Bleil et al., 2013; James et al., 2012; Sung et al., 2016). Parent–child relationships regulate the trajectory of life history strategies. Early secure attachment to mothers buffered young girls against harshness experienced in the first 5 years of life (operationalized by income-to-needs ratio). Girls insecurely attached to their mother who also resided in harsher environments (e.g., more poverty) experienced early menarche. This association was not found in girls who were securely attached to their mothers (Sung et al., 2016).

Future Directions of Evolutionary Psychology and Parenting: Health

The progression of advanced phenotyping methods of physiological systems, availability of extensive longitudinal datasets, and advanced statistics to model these data has (p. 18) produced an academic milieu exciting to evolutionary developmental psychologists and for developmental psychology in general. In parallel, traditional models of development have integrated across a variety of disciplines, including health psychology, developmental psychopathology, medicine, and epidemiology. This convergence has led to a paradigm shift whereby chronic degenerative diseases are reinterpreted as endpoints from systemic and chronic stress experienced across the life course—notably early childhood (Shonkoff et al., 2009, 2012). More and more fields are starting to take these variables into account and are examining the impact of early psychosocial stress on a range of biological indices health and disease (e.g., Miller et al., 2011; Repetti et al., 2002). Broadly, these early childhood models of stress and health are rooted in allostatic load models (McEwen, 1998; McEwen & Wingfield, 2003) that posit that sustained exposure to "toxic stress" activates coordinated responses across varied physiological systems to adapt to stress in the short term. Over time, these "adaptations" produce dysregulation across the immune systems, HPA, and other systems, causing early morbidity and mortality (see Ellis & Del Giudice, 2014, for a discussion of the ACM and allostatic load).

The research generated by testing allostatic load models of development are of interest to evolutionary developmental psychologists. Evolutionary models of development, such as psychosocial acceleration theory (Belsky et al., 1991; Figure 1.1: Health), emphasize that life history trade-offs, such as between somatic effort and reproductive effort, should result in marked changes in health and longevity. Ellis and Del Giudice (2014, p. 1) note that allostatic load models have a major limitation that may be remedied by an evolutionary framework:

> The key limitation of such models is that they do not provide a theory of adaptive individual differences in physiological mediators and related patterns of social and physical development. In the absence of such a theory, models of toxic stress and allostatic load have operated on the assumption that there is an optimal level of biological responsivity to social and environmental challenges, and that overly heightened or dampened responsivity is dysfunctional and tends to undermine mental and physical health
>
> (e.g., Evans & English, 2002; Juster et al., 2011; Lupien et al., 2006).

In contrast to allostatic load, evolutionary frameworks emphasize that health is the result of developmental trade-offs that are instantiated across key decision nodes over the life course that maximize survival and reproduction. The important next step researchers must consider is *how* early childhood experiences get "under our skin" and emerge as health and longevity outcomes. In other words, how do we examine the trade-off between reproductive effort and somatic effort?

The impact of reproductive factors on cardiovascular risk is a fertile research area for testing evolutionary models of health. As briefly reviewed earlier, evolutionary models of development suggest and empirical evidence supports the link between early psychosocial (p. 19) adversity and early menarche (reviewed in Ellis, 2004). Research on cardiovascular health has noted that early menarche has been linked to a variety of cardiometabolic risk factors (Day et al., 2015) and to cardiovascular disease events (Canoy et al., 2015). What is not clear is how early and accelerated reproductive effort leads to decreased health. We suggest that somatic effort can emerge across different mediating pathways, including health-maintaining behavior (e.g., diet, exercise) or endogenous changes within the individual (e.g., loss in cellular integrity or structural damage).

For instance, the emergence of early puberty in young girls, in addition to psychosocial factors, has been implicated by increased access to heavily processed food that is part of a "supermarket diet." Diets characterized by inexpensive and calorically dense foods may trigger physiological switch points within the body that an adolescent girl has reached a level of body fat that can facilitate somatic conditions necessary to maintain a pregnancy (Ellis, 2004). Thus, weight gain may have short-term benefits by accelerating sexual maturity, facilitating pregnancy, and benefiting reproductive success; in the long term, however, increased weight and body mass may induce cardiovascular risk (e.g., antagonistic negative pleiotropy; Williams, 1957). Thus, the association between early menarche and cardiovascular risk may work through several mediating health-maintaining factors—such as diet, stress eating, or exercise—and emerge as increased body mass or higher waist circumference (Bleil et al., 2013).

Another consideration has recently been proposed. It is possible that the linkages between early adversity, accelerated reproduction, and decreased health may emerge through endogenous changes within the individual's cellular system. The two-hit model of accelerated aging in females (Belsky & Shalev, 2016; Shalev & Belsky, 2016) proposes that there are two detrimental "hits" that may impact accelerated aging and disease: early life and family adversity and early and accelerated reproduction. As has been discussed, early exposure to conflictual and unpredictable households and contexts (Belsky et al., 1991; Ellis et al., 2009) produces alterations of the stress response system (among others) that orient young girls toward faster life history strategies. At sexual maturity, stress-adapted women will incur additional physiological stress through mating and reproduction, at the cost of diminished health and longevity. Thus, the cumulative cost of both early adversity and reproduction will impact cellular markers of aging and deterioration. Although future research must incorporate early household stressors and reproductive events to fully test the "two-hit" model, preliminary evidence in cardiology suggests that accelerated and increased reproduction increases the likelihood of cardiovascular disease risk and events (Canoy et al., 2015; Parikh et al., 2010; Sanghavi et al., 2015). This should come as no surprise to life history scholars: Increased reproductive output, even in healthy women, should come at a cost to somatic effort. Future research should examine whether early household stressors that capture facets of harshness and unpredictability moderate the reproductive health–physical health relationship.

(p. 20) Conclusion

In this chapter, we discussed the merits of incorporating an evolutionary framework within the field of developmental psychology. Evolutionary theory—notably life history theory—is a powerful integrative tool to unify disparate research across fields and disciplines under the metatheory of natural selection. As such, evolutionary developmental psychology is a field of research that considers the impact proximate and ultimate forces play on child ontogeny. Following a brief introduction to evolutionary theory, we discussed the psychosocial acceleration model (Belsky et al., 1991) as one organizing theory used primarily by evolutionary researchers interested in parenting and household effects on physical, psychological, and behavioral development. The fundamental premise of this model is that early childhood experiences within the household provide important and salient cues to the child about the safety and predictability of the outside world. These repeated experiences—whether denoted by high conflict or high warmth—are regulated and filtered by the stress response systems that orient and organize a child's development to fit their world. Over time, life history strategies emerge through instantiated developmental decisions the child makes over the lifetime. Harsh and unpredictable environments will orient the child to pursue a faster life strategy that emphasizes investment in mating over parenting and somatic health; safe and predictable environments dictate to the child that the ecology may favor slower life history strategies that emphasize somatic health and parenting over mating. We next followed with evidence supporting hypotheses posited by evolutionary theorists. Specifically, we discussed research illustrating the impact household and family dynamics play on socioemotional and cognitive development and physiological and sexual development. The chapter was concluded by discussing the emerging field of evolutionary developmental health and the developing research within the realm of reproduction and cardiovascular health.

Although we and others have discussed the benefits of applying a Darwinian framework to parenting and family research, there is still resistance to its use. Despite claims of genetic determinism or social Darwinism, we assert that our framework and our aims are used to understand the underlying mechanisms by which family influences impact development and long-term health. The onus rests on evolutionarily minded researchers not only to educate others about evolutionary approaches to development but also to justify and persuade them on the utility of such models to improve the quality of life for vulnerable populations.

References

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: What we know and what we don't. *Annals of the New York Academy of Sciences*, *896*(1), 3–15.

Amir, D., Jordan, M. R., & Bribiescas, R. G. (2016). A longitudinal assessment of associations between adolescent environment, adversity perception, and economic status on fertility and age of menarche. *PloS One*, *11*(6), e0155883.

Bales, K. L. (2017). Parenting in animals. *Current Opinions in Psychology*, *15*, 93–98.

(p. 21) Barbaro, N., Boutwell, B. B., Barnes, J. C., & Shackelford, T. K. (2017). Genetic confounding of the relationship between father absence and age at menarche. *Evolution and Human Behavior*, *38*(3), 357–365.

Barnett, M. A., Deng, M., Mills-Koonce, W. R., Willoughby, M., & Cox, M. (2008). Interdependence of parenting of mothers and fathers of infants. *Journal of Family Psychology*, *22*(4), 561–573.

Barrett, L., Dunbar, R., & Lycett, J. (2002). *Human evolutionary psychology*. Princeton University Press.

Baumeister, D., Akhtar, R., Ciufolini, S., Pariante, C. M., & Mondelli, V. (2016). Childhood trauma and adulthood inflammation: A meta-analysis of peripheral C-reactive protein, interleukin-6 and tumour necrosis factor-α. *Molecular Psychiatry*, *21*(5), 642–649.

Belsky, J., Ruttle, P. L., Boyce, W. T., Armstrong, J. M., & Essex, M. J. (2015). Early adversity, elevated stress physiology, accelerated sexual maturation, and poor health in females. *Developmental Psychology*, *51*(6), 816–822.

Belsky, J., Schlomer, G. L., & Ellis, B. J. (2012). Beyond cumulative risk: Distinguishing harshness and unpredictability as determinants of parenting and early life history strategy. *Developmental Psychology*, *48*(3), 662–673.

Belsky, J., & Shalev, I. (2016). Contextual adversity, telomere erosion, pubertal development, and health: Two models of accelerated aging, or one? *Development and Psychopathology*, *28*, 1367–1383.

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, *62*(4), 647–670.

Bleil, M. E., Adler, N. E., Appelhans, B. M., Gregorich, S. E., Sternfeld, B., & Cedars, M. I. (2013). Childhood adversity and pubertal timing: Understanding the origins of adulthood cardiovascular risk. *Biological Psychology*, *93*(1), 213–219.

Borowsky, I. W., Ireland, M., & Resnick, M. D. (2009). Health status and behavioral outcomes for youth who anticipate a high likelihood of early death. *Pediatrics*, *124*(1), e81–e88.

Bowlby, J. (1969). *Attachment and loss* (Vol. 1. Attachment). Basic Books.

Burton, L. M. (1990). Teenage childbearing as an alternative life-course strategy in multigeneration black families. *Human Nature*, *1*(2), 123–143.

Cabeza de Baca, T., Barnett, M. A., & Ellis, B. J. (2016a). The development of the child unpredictability schema: Regulation through maternal life history trade-offs. *Evolutionary Behavioral Sciences*, *10*(1), 43–55.

Cabeza de Baca, T., Figueredo, A. J., & Ellis, B. J. (2012). An evolutionary analysis of variation in parental effort: Determinants and assessment. *Parenting*, *12*(2–3), 94–104.

Cabeza de Baca, T., Wahl, R. A., Barnett, M. A., Figueredo, A. J., & Ellis, B. J. (2016b). Adversity, adaptive calibration, and health: The case of disadvantaged families. *Adaptive Human Behavior and Physiology*, *2*(2), 93–115.

Canoy, D., Beral, V., Balkwill, A., Wright, F. L., Kroll, M. E., Reeves, G. K., Green, F. J., & Cairns, B. J. (2015). Age at menarche and risks of coronary heart and other vascular diseases in a large UK cohort. *Circulation*, *131*, 237–244.

Charlesworth, W. R. (1992). Darwin and developmental psychology: Past and present. *Developmental Psychology*, *28*(1), 5–16.

Chisholm, J. S. (1999). *Death, hope and sex: Steps to an evolutionary ecology of mind and morality*. Cambridge University Press.

Chisholm, J. S., Ellison, P. T., Evans, J., Lee, P. C., Lieberman, L. S., Pavlik, Z., Ryan, A. S., Salter, E. M., Stini, W. A., & Worthman, C. M. (1993). Death, hope, and sex: Life-history theory and the development of reproductive strategies. *Current Anthropology*, *34*(1), 1–24.

Clutton-Brock, T. H. (1991). *The evolution of parental care*. Princeton University Press.

Daly, M., & Wilson, M. (2005). Carpe diem: Adaptation and devaluing the future. *The Quarterly Review of Biology*, *80*(1), 55–60.

Darwin, C. (1859). *On the origin of species by means of natural selection, or the preservation of favored races in the struggle for life*. John Murray.

Day, F. R., Elks, C. E., Murray, A., Ong, K. K., & Perry, J. R. (2015). Puberty timing associated with diabetes, cardiovascular disease and also diverse health outcomes in men and women: The UK Biobank study. *Scientific Reports*, *5*, 11208.

Del Giudice, M. (2009). Sex, attachment, and the development of reproductive strategies. *Behavioral and Brain Sciences*, *32*(1), 1–21.

Del Giudice, M. (2014a). Life history plasticity in humans: The predictive value of early cues depends on the temporal structure of the environment. *Proceedings of the Royal Society of London B: Biological Sciences*, *281*(1780), 20132222.

(p. 22) Del Giudice, M. (2014b). Middle childhood: An evolutionary-developmental synthesis. *Child Development Perspectives*, *8*(4), 193–200.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews*, *35*(7), 1562–1592.

Del Giudice, M., Gangestad, S. W., & Kaplan, H. S. (2015). Life history theory and evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (2nd ed., pp. 88–114). Wiley and Sons. **http://dx.doi.org/10.1002/9781119125563.evpsych102**

Del Priore, D. J., & Hill, S. E. (2013). The effects of paternal disengagement on women's sexual decision making: An experimental approach. *Journal of Personality and Social Psychology*, *105*, 234–246.

Del Priore, D., J., Schlomer, G. L., & Ellis, B. J. (2017). Impact of fathers on parental monitoring of daughters and their affiliation with sexually promiscuous peers: A genetically and environmentally controlled sibling study. *Developmental Psychology*, *53*, 1330–1343.

Draper, P., & Harpending, H. (1982). Father absence and reproductive strategy: An evolutionary perspective. *Journal of Anthropological Research*, *38*(3), 255–273.

Dunkel, C. S., Cho, H. B., van der Linden, D., Woodley of Menie, M., & Platek, S. M. (2017). Cavum septum pellucidum volume and life history strategy. *Evolutionary Behavioral Sciences, 11*(3), 262–271. **http://dx.doi.org/10.1037/ebs0000090**.

Dunkel, C. S., Mathes, E. W., Kesselring, S. N., Decker, M. L., & Kelts, D. J. (2015). Parenting influence on the development of life history strategy. *Evolution and Human Behavior*, *36*(5), 374–378.

Ehrlich, K. B., Miller, G. E., & Chen, E. (2015). Harsh parent–child conflict is associated with decreased anti-inflammatory gene expression and increased symptom severity in children with asthma. *Development and Psychopathology*, *27*, 1547–1554.

Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin*, *130*(6), 920–958.

Ellis, B. J., Bianchi, J. M., Griskevicius, V., & Frankenhuis, W. E. (2017a). Beyond risk and protective factors: An adaptation-based approach to resilience. *Perspectives on Psychological Science*, *12*(4), 561–587.

Ellis, B. J., & Del Giudice, M. (2014). Beyond allostatic load: Rethinking the role of stress in regulating human development. *Development and Psychopathology*, *26*(1), 1–20.

Ellis, B. J., Del Giudice, M., Dishion, T. J., Figueredo, A. J., Gray, P., Griskevicius, V., Hawley, P. H., Jacobs, W. J., James, J., Volk, A. A., & Wilson, D. S. (2012a). The evolutionary basis of risky adolescent behavior: Implications for science, policy, and practice. *Developmental Psychology*, *48*(3), 598–623.

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature*, *20*(2), 204–268.

Ellis, B. J., & Ketelaar, T. (2000). On the natural selection of alternative models: Evaluation of explanations in evolutionary psychology. *Psychological Inquiry*, *11*(1), 56–68.

Ellis, B. J., Oldehinkel, A. J., & Nederhof, E. (2017b). The adaptive calibration model of stress responsivity: An empirical test in the Tracking Adolescents' Individual Lives Survey study. *Development and Psychopathology*, *29*(3), 1–21.

Ellis, B. J., Schlomer, G. L., Tilley, E. H., & Butler, E. A. (2012b). Impact of fathers on risky sexual behavior in daughters: A genetically and environmentally controlled sibling study. *Development and Psychopathology*, *24*(1), 317–332.

Ellis, B. J., Volk, A., Gonzalez, J. M., & Embry, D. D. (2016). The Meaningful Roles intervention: An evolutionary approach to reducing bullying and increasing prosocial behavior. *Journal of Research on Adolescence*, *26*, 622–637

Evans, G. W., & English, K. (2002). The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*, *73*, 1238–1248.

Fawcett, T. W., & Frankenhuis, W. E. (2015). Adaptive explanations for sensitive windows in development. *Frontiers in Zoology*, *12*(1), S3.

Fields, R. D. (2010). Change in the brain's white matter. *Science*, *330*(6005), 768–769.

Figueredo, A. J., Patch, E. A., & Ceballos, C. E. G. (2015). A life history approach to the dynamics of social selection. In V. Zeigler-Hill (Ed.), *Evolutionary perspectives on social psychology* (pp. 363–372). Springer.

Figueredo, A. J., & Rushton, J. P. (2009). Evidence for shared genetic dominance between the general factor of personality, mental and physical health, and life history traits. *Twin Research and Human Genetics*, *12*(6), 555–563.

(p. 23) Figueredo, A. J., Vásquez, G., Brumbach, B. H., Schneider, S. M., Sefcek, J. A., Tal, I. R., Hill, D., Wenner, C. J., & Jacobs, W. J. (2006). Consilience and life history theory: From genes to brain to reproductive strategy. *Developmental Review*, *26*(2), 243–275.

Flinn, M. V. (2006). Evolution and ontogeny of stress response to social challenges in the human child. *Developmental Review*, *26*(2), 138–174.

Flinn, M. V., & England, B. G. (1995). Childhood stress and family environment. *Current Anthropology*, *36*(5), 854–866.

Frankenhuis, W. E., Panchanathan, K., & Nettle, D. (2016). Cognition in harsh and unpredictable environments. *Current Opinion in Psychology*, *7*, 76–80.

Geary, D. C., & Flinn, M. V. (2001). Evolution of human parental behavior and the human family. *Parenting*, *1*(1–2), 5–61.

Geronimus, A. T. (1996). What teen mothers know. *Human Nature*, *7*(4), 323–352.

Gianaros, P. J., & Hackman, D. (2013). Contributions of neuroscience to the study of socioeconomic health disparities. *Psychosomatic Medicine*, *75*(7), 610–615.

Gianaros, P. J., Marsland, A. L., Sheu, L. K., Erickson, K. I., & Verstynen, T. D. (2013). Inflammatory pathways link socioeconomic inequalities to white matter architecture. *Cerebral Cortex*, *23*(9), 2058–2071.

Gianaros, P. J., & Wager, T. D. (2015). Brain-body pathways linking psychological stress and physical health. *Current Directions in Psychological Science*, *24*(4), 313–321.

Hagen, E. (2005). Controversial issues in evolutionary psychology. In D. M. Buss (Ed.), *The handbook of evolutionary psychology* (pp. 145–176). Wiley.

Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., Shirtcliff, E. A., Pollak, S. D., & Davidson, R. J. (2015). Behavioral problems after early life stress: Contributions of the hippocampus and amygdala. *Biological Psychiatry*, *77*(4), 314–323.

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). Most people are not WEIRD. *Nature*, *466*(7302), 29–29.

Hostinar, C. E., Lachman, M. E., Mroczek, D. K., Seeman, T. E., & Miller, G. E. (2015). Additive contributions of childhood adversity and recent stressors to inflammation at midlife: Findings from the MIDUS study. *Developmental Psychology*, *51*(11), 1630.

Hoyt, L. T., & Falconi, A. M. (2015). Puberty and perimenopause: Reproductive transitions and their implications for women's health. *Social Science & Medicine*, *132*, 103–112.

Jablonka, E., & Raz, G. (2009). Transgenerational epigenetic inheritance: Prevalence, mechanisms, and implications for the study of heredity and evolution. *The Quarterly Review of Biology*, *84*(2), 131–176.

James, J., Ellis, B. J., Schlomer, G. L., & Garber, J. (2012). Sex-specific pathways to early puberty, sexual debut, and sexual risk taking: Tests of an integrated evolutionary–developmental model. *Developmental Psychology*, *48*(3), 687.

Juraska, J. M., & Willing, J. (2017). Pubertal onset as a critical transition for neural development and cognition. *Brain Research*, *1654*, 87–94.

Ketelaar, T., & Ellis, B. J. (2000). Are evolutionary explanations unfalsifiable? Evolutionary psychology and the Lakatosian philosophy of science. *Psychological Inquiry*, *11*(1), 1–21.

King, A. C., & Cabeza de Baca, T. (2011). The stagnancy of family studies in modern academia: Resistances toward the integration of evolutionary theory. *Evolution: Education and Outreach*, *4*(1), 64–74.

Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Pearson, D., Plotsky, P. M., & Meaney, M. J. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science*, *277*(5332), 1659–1662.

Lupien, S. J., Ouellet-Morin, I., Hupbach, A., Tu, M. T., Buss, C., Walker, D., Pruessner, J., & McEwen, B. S. (2006). Beyond the stress concept: Allostatic load: A developmental biological and cognitive perspective. In D. Cicchetti & D. J. Co-hen (Eds.), *Developmental psychopathology: Vol. 2. Developmental neuroscience* (2nd ed., pp. 578–628). Hoboken, NJ: Wiley.

Marceau, K., Ruttle, P. L., Shirtcliff, E. A., Essex, M. J., & Susman, E. J. (2015). Developmental and contextual considerations for adrenal and gonadal hormone functioning during adolescence: Implications for adolescent mental health. *Developmental Psychobiology*, *57*(6), 742–768.

Matheny, A. P., Wachs, T. D., Ludwig, J. L., & Phillips, K. (1995). Bringing order out of chaos: Psychometric characteristics of the confusion, hubbub, and order scale. *Journal of Applied Developmental Psychology*, *16*(3), 429–444.

(p. 24) McDade, T. W., Chyu, L., Duncan, G. J., Hoyt, L. T., Doane, L. D., & Adam, E. K. (2011). Adolescents' expectations for the future predict health behaviors in early adulthood. *Social Science & Medicine*, *73*(3), 391–398.

McDade, T. W., Georgiev, A. V., & Kuzawa, C. W. (2016). Trade-offs between acquired and innate immune defenses in humans. *Evolution, Medicine, and Public Health*, *2016*(1), 1–16.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*(1), 33–44.

McEwen, B. S., & Gianaros, P. J. (2010). Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences*, *1186*, 190.

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones and Behavior*, *43*(1), 2–15.

Meaney, M. J. (2010). Epigenetics and the biological definition of gene x environment interactions. *Child Development*, *81*(1), 41–79.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*(6), 959.

Mirowsky, J., & Ross, C. E. (2000). Socioeconomic status and subjective life expectancy. *Social Psychology Quarterly*, *63*(2), 133–151.

Mittal, C., Griskevicius, V., Simpson, J. A., Sung, S., & Young, E. S. (2015). Cognitive adaptations to stressful environments: When childhood adversity enhances adult executive function. *Journal of Personality and Social Psychology*, *109*(4), 604–621.

Panksepp, J., Moskal, J., Panksepp, J. B., & Kroes, R. (2002). Comparative approaches in evolutionary psychology: Molecular neuroscience meets the mind. *Neuroendocrinology Letters*, *23*(Suppl. 4), 105–115.

Parikh, N. I., Cnattingius, S., Dickman, P. W., Mittleman, M. A., Ludvigsson, J. F., & Ingelsson, E. (2010). Parity and risk of later-life maternal cardiovascular disease. *American Heart Journal*, *159*(2), 215–221.

Piquero, A. R. (2016). “Take my license n’ all that jive, I can’t see… 35”: Little hope for the future encourages offending over time. *Justice Quarterly*, *33*(1), 73–99.

Promislow, D. E., & Harvey, P. H. (1990). Living fast and dying young: A comparative analysis of life-history variation among mammals. *Journal of Zoology*, *220*(3), 417–437.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, *128*(2), 330–366.

Ricklefs, R. E., & Wikelski, M. (2002). The physiology/life-history nexus. *Trends in Ecology & Evolution*, *17*(10), 462–468.

Rijsdijk, F. V., & Sham, P. C. (2002). Analytic approaches to twin data using structural equation models. *Briefings in Bioinformatics*, *3*(2), 119–133.

Roff, D. (2012). *Evolutionary quantitative genetics*. Springer Science & Business Media.

Ross, L. T., & Hill, E. M. (2000). The family unpredictability scale: Reliability and validity. *Journal of Marriage and Family*, *62*(2), 549–562.

Ross, L. T., & Hill, E. M. (2002). Childhood unpredictability, schemas for unpredictability, and risk taking. *Social Behavior and Personality: An International Journal*, *30*(5), 453–473.

Ross, L. T., Hood, C. O., & Short, S. D. (2016a). Unpredictability and symptoms of depression and anxiety. *Journal of Social and Clinical Psychology*, *35*(5), 371–385.

Ross, L. T., Short, S. D., & Garofano, M. (2016b). Scale of unpredictability beliefs: Reliability and validity. *The Journal of Psychology*, *150*(8), 976–1003.

Rowe, D. C. (1994). *The limits of family influence: Genes, experience, and behavior*. Guilford Press.

Rowe, D. C. (2002). On genetic variation in menarche and age at first sexual intercourse: A critique of the Belsky–Draper hypothesis. *Evolution and Human Behavior*, *23*(5), 365–372.

Sanghavi, M., Kulinski, J., Ayers, C. R., Nelson, D., Stewart, R., Parikh, N., de Lemos, J. A., & Khera, A. (2015). Association between number of live births and markers of subclinical atherosclerosis: The Dallas Heart Study. *European Journal of Preventive Cardiology*, *23*, 391–399.

Schlomer, G. L., Del Giudice, M., & Ellis, B. J. (2011). Parent–offspring conflict theory: An evolutionary framework for understanding conflict within human families. *Psychological Review*, *118*(3), 496.

Schlomer, G. L., & Ellis, B. J. (2016). Sibling studies. In T. K. Shackelford & V. Weekes-Shackelford (Eds.), *Encyclopedia of evolutionary psychological science*. Springer. **https://doi.org/10.1007/978-3-319-16999-6_1925-1**

(p. 25) Scott-Sheldon, L. A., Carey, M. P., Vanable, P. A., & Senn, T. E. (2010). Subjective life expectancy and health behaviors among STD clinic patients. *American Journal of Health Behavior*, *34*(3), 349–361.

Segal, N. L. (1993). Twin, sibling, and adoption methods: Tests of evolutionary hypotheses. *American Psychologist*, *48*(9), 943.

Shalev, I., & Belsky, J. (2016). Early-life stress and reproductive cost: A two-hit developmental model of accelerated aging? *Medical Hypotheses*, *90*, 41–47.

Shelleby, E. C., Votruba-Drzal, E., Shaw, D. S., Dishion, T. J., Wilson, M. N., & Gardner, F. (2014). Income and children’s behavioral functioning: A sequential mediation analysis. *Journal of Family Psychology*, *28*(6), 936.

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association*, *301*(21), 2252–2259.

Shonkoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., Pascoe, J., Wood, D.L., & Committee on Early Childhood, Adoption, and Dependent Care. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*(1), e232–e246.

Simpson, J. A., Griskevicius, V., Kuo, S. I., Sung, S., & Collins, W. A. (2012). Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. *Developmental Psychology*, *48*(3), 674–686.

Slavich, G. M., O’Donovan, A., Epel, E. S., & Kemeny, M. E. (2010). Black sheep get the blues: A psychobiological model of social rejection and depression. *Neuroscience & Biobehavioral Reviews*, *35*(1), 39–45.

Sung, S., Simpson, J. A., Griskevicius, V., Sally, I., Kuo, C., Schlomer, G. L., & Belsky, J. (2016). Secure infant-mother attachment buffers the effect of early-life stress on age of menarche. *Psychological Science*, *27*(5), 667–674.

Szepsenwol, O., Simpson, J. A., Griskevicius, V., & Raby, K. L. (2015). The effect of unpredictable early childhood environments on parenting in adulthood. *Journal of Personality and Social Psychology*, *109*(6), 1045–1067.

Tither, J. M., & Ellis, B. J. (2008). Impact of fathers on daughters' age at menarche: A genetically and environmentally controlled sibling study. *Developmental Psychology*, *44*(5), 1409–1420.

Tooby, J., & Cosmides, L. (1990). The past explains the present: Emotional adaptations and the structure of ancestral environments. *Ethology and Sociobiology*, *11*(4–5), 375–424.

Turecki, G., & Meaney, M. J. (2016). Effects of the social environment and stress on glucocorticoid receptor gene methylation: A systematic review. *Biological Psychiatry*, *79*(2), 87–96.

Umberson, D., Olson, J. S., Crosnoe, R., Liu, H., Pudrovska, T., & Donnelly, R. (2017). Death of family members as an overlooked source of racial disadvantage in the United States. *Proceedings of the National Academy of Sciences*, *114*(5), 915–920.

Warner, T. D., & Swisher, R. R. (2014). The effect of direct and indirect exposure to violence on youth survival expectations. *Journal of Adolescent Health*, *55*(6), 817–822.

Waters, S. F., West, T. V., & Mendes, W. B. (2014). Stress contagion: Physiological covariation between mothers and infants. *Psychological Science*, *25*, 934–942. **http://dx.doi.org/10.1177/0956797613518352**.

Weaver, I. C., Cervoni, N., Champagne, F. A., D'Alessio, A. C., Sharma, S., Seckl, J. R., Dymov, S., Szyf, M., & Meaney, M. J. (2004). Epigenetic programming by maternal behavior. *Nature Neuroscience*, *7*(8), 847–854. **http://dx.doi.org/10.1038/nn1276**

West-Eberhard, M. J. (2003). *Developmental plasticity and evolution*. Oxford University Press.

White, S. F., Brislin, S., Sinclair, S., Fowler, K. A., Pope, K., & Blair, R. J. R. (2013). The relationship between large cavum septum pellucidum and antisocial behavior, callous-unemotional traits and psychopathy in adolescents. *Journal of Child Psychology and Psychiatry*, *54*(5), 575–581.

Williams, D. R., & Collins, C. (1995). US socioeconomic and racial differences in health: Patterns and explanations. *Annual Review of Sociology*, *21*(1), 349–386.

Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution*, *11*, 398–411.

Tomás Cabeza de Baca

Tomás Cabeza de Baca, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health Phoenix, AZ

Bruce J. Ellis

Bruce J. Ellis, University of Utah Salt Lake City, UT