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When Do Adaptive Developmental Mechanisms Yield Maladaptive Outcomes?

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This article discusses 3 ways in which adaptive developmental mechanisms may produce maladaptive outcomes. First, natural selection may favor risky strategies that enhance fitness on average but which have detrimental consequences for a subset of individuals. Second, mismatch may result when organisms experience environmental change during ontogeny, for instance, because they move from one environment to another. Third, organisms may learn about their environment in order to develop an appropriate phenotype; when cues indicate the environmental state probabilistically, as opposed to deterministically, sampling processes may produce mismatch. For each source of maladaptation, we present a selection of the relevant empirical research and illustrate how models from evolutionary biology can be used to make predictions about maladaptation. We also discuss what data can be collected to test these models in humans. Our goal is to show that evolutionary approaches not only yield insights into adaptive outcomes but can also illuminate the conditions leading to maladaptation. This perspective provides additional nuance to the dialectic between the developmental psychopathology model and evolutionary developmental psychology.

Keywords: adaptation, maladaptation, developmental plasticity, risk-taking, probabilistic sampling

The concept of adaptation is central to both evolutionary biology and developmental psychology, but its meaning differs between these two fields. In biology, the term *adaptation* refers to traits that evolved via natural selection, because of their effects on survival and reproductive success (Williams, 1966). Traits evolve because they promote the inclusive fitness of individuals, furthering the replication of their genes into future generations. In contrast, developmental psychologists typically adopt what we refer to as the *developmental psychopathology model* (see Ellis et al., 2012). In this framework, socially undesirable or subjectively distressing behavior is viewed as maladaptive, whereas behavior enhancing an individual's well-being, cooperation, and social integration is considered adaptive (e.g., Cicchetti & Blender, 2004; Hartup & Stevens, 1997; Kochenderfer-Ladd, 2004; Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998). These different notions of adaptation are conceptually orthogonal: desirable behavior may (or may not) enhance reproductive success, and fitness-enhancing behavior may (or may not) have socially approved features. For example, a child growing up in a dangerous environment may develop high levels of vigilance, which can be detrimental to his or her subjective well-being but also promote survival (e.g., by facilitating detection of threats). Conversely, a trusting child may experience fewer negative emotions but suffer a reduction in biological fitness because he or she less effectively avoids danger.

In the current article, we adopt an evolutionary developmental approach, examining developmental processes and outcomes in light of their fitness consequences (Belsky, Steinberg, & Draper, 1991; Bjorklund & Pellegrini, 2000; Ellis & Bjorklund, 2005; Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011; Geary & Bjorklund, 2000). This approach views aggression, early sexual debut, risk taking, and other behaviors that are often considered maladaptive in a developmental psychopathology perspective, as potentially resulting from adaptive biological mechanisms (for examples, see Belsky et al., 1991; Chisholm, 1999; Daly & Wilson, 1988, Daly & Wilson, 2005; Del Giudice, 2009; Hawley, 1999; Kruger & Nesse, 2006; Mishra & Lalumière, 2008; Wilson, Daly, & Pound, 2002). The evolutionary perspective can be contrasted with the developmental psychopathology model, which routinely interprets socially undesirable or psychologically distressing outcomes as pathological, dysfunctional, or dysregulated.

An evolutionary developmental approach considers mechanisms to be functioning "normally" when they perform their evolved function, interacting with the environment to produce outcomes favored by natural selection. The term *malfunction* is reserved for cases in which a mechanism fails to execute its evolved operations. Malfunction can occur, for instance, due to genetic mutations, developmental insults (e.g., ingestion of toxins during embryogenesis), or manipulation by other organisms (e.g., pathogens). The evolutionary perspective does not deny the existence of pathology and dysregulation; however, it defines them much more narrowly,

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guarding against automatically inferring pathology from undesirable outcomes.[1]

Adaptation and Maladaptation

Our central goal in this article is to show that developmental mechanisms, even when they perform their evolved operations, may produce maladaptive outcomes. This insight is important, because observations of fitness-detrimental outcomes (e.g., injury resulting from risk taking) may otherwise be taken to invalidate adaptive explanations of behavior or to indicate malfunction of an adaptive system. The key is to distinguish between the fitness of *mechanisms*, averaged across individuals and states of the environment, and the fitness of *individual organisms* (e.g., Maynard Smith, 1989; Orr, 2009); a mechanism may be fitness-enhancing *on average*, while imposing fitness costs on *some* individuals.[2] We will illustrate this point using several examples.

The majority of research in evolutionary psychology focuses on the beneficial effects of adaptive mechanisms. This emphasis is justified, because adaptive mechanisms tend to provide a net benefit to fitness. However, as a result, a systematic framework for studying maladaptation is lacking. Some researchers have explored the psychological mismatch that might result when current environments differ from ancestral environments, generating valuable insights along with empirical results. However, even when adaptive developmental systems develop within the "species-typical" environmental range, they might still produce maladaptive outcomes. Our goal, here, is to show that evolutionary approaches do not only yield insights into adaptive development, but can also illuminate the conditions leading to maladaptive developmental outcomes.

Outline of the Article

In the next sections, we focus on three different processes that may result in maladaptive outcomes for individuals. First, risky strategies may be favored by natural selection, even when they entail the potential of detrimental outcomes. Second, mismatch may result when organisms experience environmental change, for instance, because they move from one environment to another or because the environment itself changes. Third, organisms may learn about their environment in order to develop an appropriate phenotype; when cues indicate the environmental state probabilistically, as opposed to deterministically, sampling processes may produce mismatch. For each of these sources of maladaptation, we present a selection of the exciting research that is being done on this subject. We show how evolutionary models can be used to make predictions about maladaptation, discuss what data can be collected to apply these models to real-world contexts, and explore their implications for developmental research and intervention.

Maladaptive Outcomes of Risky Strategies

Risky behavior is part and parcel of daily life: many activities that contribute to survival and reproduction also increase the probability of harm, injury, loss, or death. For instance, a bird may be more exposed to predators while foraging; a male displaying to females may be more likely attacked by rivals; and copulation itself increases the risk of contracting disease. Despite these dire potentials, animals must forage, court, and have sex to be able to perpetuate their genes into future generations—avoiding risk at all costs is not a viable strategy. From an evolutionary perspective, natural selection would be expected to favor mechanisms that produce risk taking when the fitness benefits outweigh the costs. Since these costs and benefits often depend on an individual's state and environmental context, so should risk taking.

The definition of risk is *unpredictable variation in outcomes* (Kacelnik & Bateson, 1997; Smallwood, 1996; Winterhalder, 2007). Whereas some behavioral decisions offer a narrow range of possible outcomes (low risk), others entail widely variable outcomes (high risk), with the potential for large gains as well as large losses. Consider a predator that can choose between two types of prey: larger and hard-to-catch animals versus smaller and easily caught ones. Imagine also that the expected (i.e., weighted average) energetic returns associated with hunting each type of prey are identical: one results in a high reward with a low probability, the other in a low reward with a high probability. In this scenario, hunting larger prey qualifies as more risky, because it entails more variable outcomes. In other words, the same expected return may be determined by different probabilistic combinations of outcomes, some of which are riskier (i.e., more variable) than others.

Often, natural selection results in organisms that are *risk sensitive*. A risk-sensitive organism does not only weigh expected outcomes, but chooses among options based on outcome expectations and outcome variability. Natural selection favors risk aversion when the relationship between behavioral outcomes (e.g., resource acquisition) and fitness is characterized by diminishing returns (Figure 1; see also Kacelnik & Bateson, 1997; Smallwood, 1996). For instance, a well-fed animal should forage on low-risk items (or not forage at all) when additional calories only slightly improve its condition. Conversely, when better outcomes yield increasing fitness returns, organisms may become risk prone (Figure 1). For example, an animal on the brink of starvation may choose to forage in a nutrient-rich habitat, even if it is densely populated by predators, because it has so much to gain from additional calories—in fact, it might need those calories to make it through the night.

An analogous logic applies to competition for reproduction. In some mating systems, reproductive benefits are highly skewed toward top-ranking individuals (i.e., "winner takes all" systems). In such conditions, males are intensely selected to compete for top rank, even if this implies a greater risk of fitness-detrimental outcomes. For instance, male elephant seals engage in fights that often cause harm and sometimes result in death. Despite these potentials, males benefit from participating in fights, because not participating implies being shut out from reproduction. Mathematical models reveal a similarity between a hungry bird and male elephant seals entering the reproductive arena: both are in a con-

[1] For an introduction to evolutionary approaches to pathology and disease, see Nesse (2005a) and Nesse and Jackson (2006).

[2] In this article, we adopt a *teleonomic* perspective on maladaptation, focusing on fitness costs and benefits to individual organism. This is the one usually employed in biology when studying the adaptive design of phenotypes. Other perspectives on maladaptation (e.g., those from phylogenetics or population genetics) may be appropriate for different purposes (Crespi, 2000).

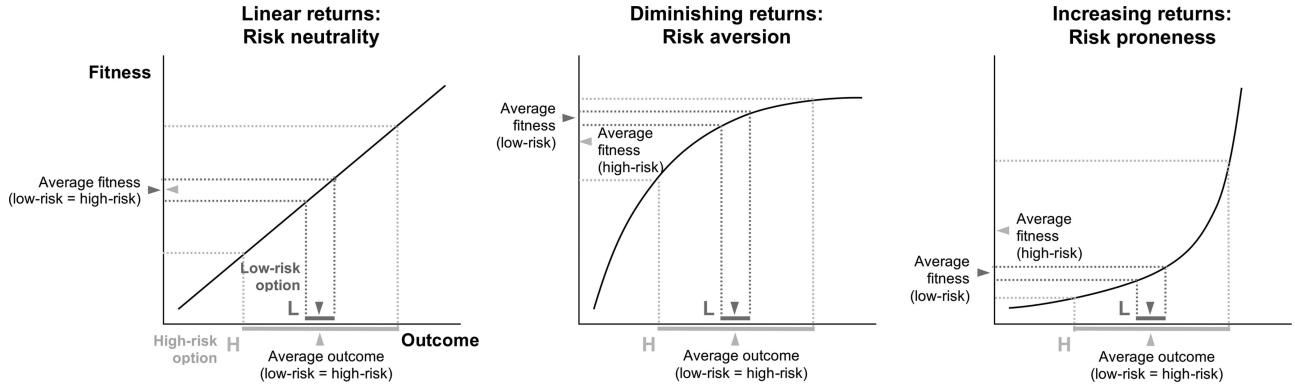

Figure 1. Risk sensitivity depends on the fitness function. With linear fitness returns (left panel), the average fitness does not depend on outcome variance, and organisms are expected to be risk neutral. When the fitness function is nonlinear, it is adaptive to be sensitive to risk. With diminishing returns (middle panel), low-risk options yield higher average fitness, favoring risk aversion. With increasing returns (right panel), high-risk options yield higher average fitness, favoring risk proneness.

dition in which the sure flow of resources (or access to mates) is below a desired level (they are in a "poor" condition). Therefore, they have little to lose and much to gain from choosing risky behaviors.

Models further show that under certain assumptions, a moderate degree of risk taking can be adaptive when an individual is in especially good condition (McNamara, Merad, & Houston, 1991; Nettle, 2009). For instance, the primary goal of an organism might be to avoid falling below some critical threshold (e.g., a dangerously low energy state). However, when the organism is well above this threshold (e.g., well nourished), it can afford substantial losses. In this "safe zone," it may become more risk prone in order to maximize potential gains (e.g., growing the largest body possible).

Risk and Human Behavior

Sex and age differences in risk taking. Men tend to be more risk taking than women—although the pattern varies across ages and contexts (Byrnes, Miller, & Schafer, 1999; Wang, Kruger, & Wilke, 2009)—and as a consequence, behavior-caused mortality is cross-culturally biased toward males (Kruger & Nesse, 2006). An evolutionary explanation for this sex-biased pattern is that men tend to have larger variance in reproductive success than women do: they are more likely to have no children and to achieve very high reproductive success (Wilson, Daly, & Pound, 2002; but see Brown, Laland, & Borgerhoff Mulder, 2009). Also, male reproductive success is limited primarily by reproductive access to women, while women's fitness is not equally constrained by reproductive access to men but rather by the energetic demands of internal gestation and lactation. Hence, women have evolved to be more selective in choosing mates than men (Schmitt, Shackelford, & Buss, 2001). Accordingly, men who fall below a desired threshold of attractiveness—determined by such factors as social status, skill, resources, and willingness to invest in offspring—may find no mate at all. This threshold effect, coupled with larger male reproductive variance, can favor risk taking in males, especially when social status or resources are at stake (Low, 1993).

Morphological and ethnographic evidence suggests that humans have been moderately polygynous across evolutionary history (see Gangestad & Simpson, 2000; Marlowe, 2003; Quinlan, 2008). Indeed, males possess a number of traits that may have evolved specifically for the purpose of male–male competition (Archer, 2009), including a high ratio of upper-to-lower-body muscle mass, fast twitch muscle for power, high levels of testosterone, risk taking, intimidation, and other aggressive behaviors. Men also seem to tailor their behavioral strategy to their own physical formidability. For instance, stronger men endorse violence more, are more prone to anger, and feel more entitled to better treatment (Sell, Tooby, & Cosmides, 2009).

Consistent with this evolutionary argument, research shows that the male-to-female mortality ratio is greater in polygynous societies (compared with monogamous ones), and also in societies in which access to resources is more positively skewed (Kruger, 2010), meaning that some individuals have privileges that most of the others do not have. Data show that higher mortality rates in males are partially driven by increased rates of homicide, which is often the result of male–male competition (Daly & Wilson, 1988). This perspective is further supported by the existence of high correlations between economic inequality and homicide rates on spatial scales ranging from nations to neighborhoods (Kruger, 2010; Wilson & Daly, 1997).

Research shows that young men are more risk prone than any other demographic (Wilson et al., 2002). Young men may benefit from competing particularly hard in early adolescence, because during this time they have yet to attain social status and wealth, assets that increase their mate value and abilities to provision for future offspring. In other words, they have little to lose and much to gain (e.g., Kruger, 2010; Kruger & Nesse, 2006; Wilson et al., 2002), which can lead to a psychology that steeply discounts the future (e.g., Rogers, 1994) and which is less deterred by the negative consequences of actions (Cross, Copping, & Campbell, 2011), focusing instead on immediate competition for mates (Frankenhuis & Karremans, in press) and status (Mishra & Lalumière, 2008).

The function of aggressive and externalizing behavior. Some behaviors observed in children and adolescents may reflect high-risk, high-stakes strategies. Consider externalizing behaviors,

characterized by attention seeking, defiance, bragging and boasting, disruption of adult-imposed discipline, teasing and threatening, and physical aggression. From an evolutionary perspective, these behaviors can be viewed as high-risk tactics of social competition (Del Giudice, Ellis, & Shirtcliff, 2011; McIntyre & Hooven, 2009). In some cases, aggressive children and adolescents become dominant, respected, and popular leaders in their peer groups, especially if they are able to skillfully mix coercive strategies with prosocial ones, such as cooperation, affiliation, and negotiation (Hawley, 1999, Hawley, 2007; Pellegrini & Bartini, 2001; Rodkin, Farmer, Ruth, & Acker, 2006). However, in other cases, aggressive children may not succeed and become unpopular or rejected, even incurring physical harm. These outcomes can be individually maladaptive, yet they may result from an adaptive strategy designed to achieve dominance and social status.

The current perspective suggests that an exclusive focus on average or modal outcomes of risky behavior (i.e., ignoring outcome variability) can be misleading. For example, it is possible that most aggressive children become unpopular and rejected, and only a few end up as popular leaders—yet, this would not imply that aggression is maladaptive. What matters is whether the expected fitness of an aggressive strategy is greater than that of alternative strategies, given an individual's current circumstances. As noted, being at the bottom of the status hierarchy may increase the appeal of a high-risk strategy because here individuals have little to lose and much to gain, formally equivalent to a hungry bird foraging in a risky patch (Wilson et al., 2002).

The development of risky strategies. The developmental environment also exerts a powerful influence on risk-taking propensities. Life history theorists have identified *resource availability, extrinsic mortality–morbidity* (i.e., all unpreventable sources of mortality or physical damage), and *unpredictability* (i.e., the extent to which individuals can predict future events) as key dimensions that modulate how organisms allocate their resources (including time and energy) across competing fitness-relevant tasks, including growth and reproduction, long-term investments, and future orientation (see Ellis, Figueredo, Brumbach, & Schlomer, 2009). Higher mortality–morbidity and unpredictability tend to shift organisms toward "fast" life history strategies, characterized by early sexual maturation and reproduction, reduced investment in long-term bonding and parenting, and increased investment in short-term mating (for a review, Ellis et al., 2009; see also Belsky et al., 1991; Chisholm, 1993; Del Giudice, 2009; Quinlan, 2007). Fast life history strategies also promote risk taking, including aggression, high-risk status competition, and various forms of antisocial behavior (e.g., Brezina, Tekin, & Topalli, 2009; Mishra & Lalumière, 2008; Wang et al., 2009).

Life history strategies are determined by a combination of genetic and environmental influences (e.g., Ellis, Boyce, et al., 2011; Figueredo, Vásquez, Brumbach, & Schneider, 2004). The quality of family relations and attachment processes in early life are likely to provide developing children with information about the safety and predictability of the local environment, such that insecure attachment and stressful family interactions may shift life history strategies toward the fast end, accelerating reproductive development (e.g., Chisholm, 1993, Chisholm, 1999; Geronimus, Bound, & Waidmann, 1999; Nettle, 2011; Quinlan, 2010) and increasing risk taking (Belsky et al., 1991; Daly & Wilson, 1988). Modulation of life history strategies based on early experience can be mediated by the stress response system (see Del Giudice et al., 2011). Such modulation of developmental processes to match fitness-relevant dimensions of the local environment is called *developmental plasticity* (West-Eberhard, 2003). In the next section, we discuss how this form of adaptive matching creates the potential for maladaptation.

Implications and Future Directions

Implications for developmental theory and research. The evolutionary perspective on risk, as presented here, can be contrasted with the standard developmental psychopathology model. The developmental psychopathology model focuses on the costs of risky behaviors to well-being and society, downplaying or ignoring potential benefits to individuals engaging in these behaviors (Ellis et al., 2012; Hawley, 1999, Hawley, 2007). In contrast, the evolutionary approach emphasizes *fitness-relevant* costs and benefits to individuals. This perspective asks whether risky strategies may have adaptive value to the actor, given his or her social (e.g., relative rank) and ecological (e.g., distributions of resources) context. An evolutionary approach does not imply that risky behavior should be left unchecked: as noted, fitness relevance and social desirability are conceptually orthogonal. Rather, giving due consideration to the fitness benefits of risk taking, including the role of outcome variability, can contribute to a deeper understanding of the myriad forces influencing risk taking. This knowledge may help researchers in devising successful intervention programs (for examples, see Brezina et al., 2009; Ellis et al., 2012).

That unpredictable outcome variability is essential to risk taking also has methodological implications. The standard way of analyzing data in psychology is to focus on average effects, treating "residual" variation as noise. When dealing with risky strategies, however, variation around the mean (or around the best fit line) is essential. First, for some individuals, a risky behavioral strategy may be adaptive, even if the expected average outcome is a net loss. Second, when the relationship between behavioral outcomes and fitness is nonlinear, the difference between two types of behavior may lie not in their average effects, but in the variance of their outcomes (i.e., their riskiness), an exclusive focus on the mean ignores that difference. The mean effect of aggression on social standing may be negligible, or even negative; yet, aggression could be an adaptive strategy for some children (e.g., those with currently low status or diminished prospects), precisely because it is associated with variable outcomes. And since the costs and benefits of risky behavior may differ between males and females, sex differences in outcome variance should be considered as well.

Analyses of risk taking also suggest that chance may play a role in determining developmental outcomes. In a nontrivial sense, the difference between those who become "winners" and "losers" may partly be random. For instance, what factors determine whether a particular individual, who behaves antisocially, becomes popular or rejected? Research shows that the popularity of antisocial adolescents depends on such qualities as social skills and intelligence (Hawley, 2007). In addition, however, part of the answer may be sheer luck (e.g., winning or losing a particular fight). Accordingly, in analyses of risky behavioral strategies, our ability to predict individual outcomes (as opposed to statistical trends) may be inherently limited.

Finally, we noted that risk taking is primarily favored when individuals are in poor condition; however, with strongly increasing returns, risk taking may also be adaptive for individuals in a very good condition (McNamara et al., 1991; Nettle, 2009). To date, little research has explored the risk-taking propensities of individuals who can lose some without suffering much, yet who have a lot to gain from winning—for them, risk taking may result from factors other than harsh, unpredictable environments.

Implications for intervention. Understanding the adaptive logic of behavior may help adjust behavior in desirable directions. For instance, if in dangerous environments (e.g., gang-infested neighborhoods), individuals develop a tendency to strongly discount the future (e.g., rather $10 now than $15 next week)—an adaptive response when extrinsic mortality rates are high (Daly & Wilson, 2005)—then threatening with longer prison sentences may not be effective at deterring crime:

> Research on risk and future uncertainty highlights the potential limitations of crime-control strategies that seek only to increase the future costs of criminal behavior (e.g., an increase in criminal penalties). Such efforts may have little impact on young people who anticipate a short life and who therefore discount the future. (Brezina et al., 2009, p. 1122)

Instead, it may be more effective to improve the actual or perceived future prospects of children, increasing their sensitivity to future costs and rewards and reducing their desire for immediate rewards (with associated potential costs). The implications of evolutionary developmental psychology for managing adolescent risk taking are discussed in more detail by Ellis et al. (2012).

Developmental Mismatch Resulting From Environmental Change or Migration

Developmental plasticity—the ability to modify developmental trajectories based on experience—is ubiquitous in plants and animals (Schlichting & Pigliucci, 1998; West-Eberhard, 2003) and in humans (e.g., Belsky et al., 1991; Chisholm, 1993; Ellis, 2004; Kuzawa, 2005; Nettle, 2011; Quinlan, 2007). Natural selection may favor developmental plasticity when the environmental state varies across time and space, because it enables organisms to tailor their developmental trajectories to local conditions. However, plasticity may also entail costs: for instance, plastic mechanisms do not always "hit" their adaptive targets, and adaptive targets might change over time, creating the potential for mismatched phenotypes (e.g., Auld, Agrawal, & Relyea, 2010; DeWitt, Sih, & Wilson, 1998; Schlichting & Pigliucci, 1998). In this section, we explain how mismatched phenotypes may arise during development, either because individuals move from one environment to another or because the environment itself changes.

The Evolution of Developmental Plasticity

Some features of the environment may remain stable over long time periods (e.g., oxygen levels, seasonal cycles, and so forth), but others are more variable across time and space (e.g., resource availability, mortality rates, and the like). When an environmental parameter is static for many generations, natural selection may favor canalized development of a specific phenotype, which is optimally adapted (within constraints) to the recurring parameter values. When environments are variable, however, different phenotypes may be optimal in different environmental states, favoring the evolution of plasticity (Dall, Giraldeau, Ollson, McNamara, & Stephens, 2005; Figueredo, Hammond, & McKiernan, 2006; Schlichting & Pigliucci, 1998). Adaptive plasticity may entail using experiences from previous life stages to "forecast" future states of the environment in order to develop adaptively matching phenotypes. The long-term adaptive modification of developmental trajectories has been referred to as *conditional adaptation* (Boyce & Ellis, 2005) and *predictive adaptive response* (Gluckman, Hanson, & Beedle, 2007; see also Kuzawa, 2005).

To illustrate how evolved mechanisms can tailor development to local conditions, consider the soapberry bug, a half-inch-long, seed-eating insect (Carroll, 1993; Carroll & Corneli, 1995). While it takes a male soapberry bug only 10 min to copulate with a female, he may spend hours more anchored to her by means of specially designed genital hooks. This mate-guarding behavior increases fitness by preventing rival males from copulating with a female before she lays her eggs. However, guarding comes at a price—the male could be copulating with additional females. The trade-off between guarding and mating is influenced by how many male competitors are around; as the number of males relative to females (i.e., the sex ratio) increases, the benefits of guarding increase as well. In Oklahoma, where sex ratios vary between populations, males exhibit plasticity, calibrating the amount of mate guarding to the sex ratio they experienced during development. By contrast, in Florida, where sex ratios remain constant, males engage in a fixed amount of mate guarding, and, when raised in lab conditions with variable sex ratios, are incapable of calibrating. Soapberry bugs offer a striking demonstration that natural selection and developmental plasticity should not be seen as opposites; natural selection designs developmental mechanisms, which in turn give rise to phenotypes adapted to their local ecologies (e.g., Panchanathan, Frankenhuis, & Barrett, 2010).

Some variation in humans may result from conditional adaptation, analogous to mate guarding in soapberry bugs. For example, women who grow up in high-mortality environments—where life expectancies are lower—exhibit earlier onset of menarche and younger age of first birth than women growing up in safe environments (Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Low, Hazel, Parker, & Welch, 2008; Nettle, 2011). Psychologically, women expecting to die young tend to experience menarche at an earlier age and conceive their first child earlier in life (Chisholm, Quinlivan, Peterson, & Coall, 2005). A plausible explanation for this accelerated reproductive strategy is that dangerous environments entrain "fast" life history strategies, favoring early reproduction. When long life is a given, organisms invest in prolonged growth and development, resulting in fewer but higher quality offspring; when life is short, organisms forgo further growth and development, focusing instead on maximizing the number of offspring (Belsky et al., 1991). The developmental system, in this case determining the timing of reproduction, can thereby produce a correlation between ecology and behavior. Variation in cultural practices, like coming-of-age rituals, may then partially reflect the interaction between evolved developmental processes and the state of the environment.

Developmental Mismatch

To illustrate how developmental plasticity may result in mismatched phenotypes, we consider the development of vigilance and stress responsivity. Based on archeological evidence, historical records, and contemporary variation in human childrearing, it is plausible that human children have been exposed to varying levels of threat across our evolutionary history. Across the millennia, some youngsters grew up in war-torn regions and abusive or neglectful families, while others were nurtured in safer circumstances (e.g., Hrdy, 1999; Maestripieri, 2005; Maestripieri & Carroll, 1998). If such variation was stably recurring, natural selection might have favored developmental mechanisms that use cues to the environmental state to calibrate levels of vigilance and stress responsivity (Boyce & Ellis, 2005, 2008; Del Giudice et al., 2011; Ellis, Essex, & Boyce, 2005; Glover, 2011).

This hypothesis is supported by research showing that children growing up in hostile environments tailor their emotion systems, arousal responses, and perceptual abilities to the detection and monitoring of danger. For instance, abused children show more rapid orienting to (and delayed disengagement from) angry faces compared with peers developing in a safer environment (Pollak, 2008). This difference in attention is specific to angry facial expressions—it does not occur with fearful or happy faces (Pollak & Sinha, 2002). Neurological and behavioral data further indicate that the degree of sensitivity to danger correlates with the magnitude of abuse the child endured (Shackman, Shackman, & Pollak, 2007). For instance, when abused children overhear two unfamiliar adults engage in an argument not personally relevant to them, they maintain a state of anticipatory monitoring longer than nonabused peers (Pollak, Vardi, Bechner, & Curtin, 2005). These results fit with the evolutionary developmental hypothesis that children calibrate their levels of vigilance and stress responsivity to the state of their environment.

Heightened levels of vigilance and stress responsivity can persist long after a child has been adopted by caring, supportive foster parents (e.g., Fries, Ziegler, Kurian, Jacoris, & Pollak, 2005; Gunnar, Morison, Chisholm, & Schuder, 2001; for a review, see Marshall & Kenney, 2009). In the new environment, constantly elevated stress levels can have maladaptive effects, for instance, by negatively impacting immune functioning (Shirtcliff, Coe, & Pollak, 2009). The aggressive or withdrawn behaviors that co-occur with heightened stress responsivity (see Del Giudice et al., 2011) may alienate the child from its new social environment, where such responses are no longer useful. Thus, when the environment changes from harsh to safe, the maintenance of a profile of high vigilance and stress responsivity may create a mismatch between the child and its new environment. A similar mismatch may occur when individuals stay in the same location (i.e., no migration), but the environment itself changes (e.g., due to cultural change or modernization).

Developmental mismatch and physical health outcomes. Over the last century, many societies have undergone major and rapid transitions along social–ecological dimensions relevant to biological fitness, including nutrition, hygiene, pathogens, and so on. Although these changes have improved health outcomes for many people, recent evidence suggests that for some individuals, favorable environmental change may *increase* susceptibility to disease, including elevated risk of cardiovascular disease, obesity, diabetes, allergies, reduced bone health, and compromised immune function (Barker, 1994; Gluckman, Hanson, Spencer, & Bateson, 2005; Heijmans et al., 2008; Kuzawa, 2005; Kuzawa & Quinn, 2009).

Currently, biologists, psychologists, and anthropologists are jointly studying to what extent this maladaptive pattern may be attributable to developmental mismatch. One hypothesis is that individuals calibrate to environmental parameter values early in life, and when these values differ from those experienced later in life, a mismatched phenotype may result, increasing the likelihood of physical health problems. For instance, prenatal exposure to undernutrition may result in the development of metabolic processes designed to retain and store insulin and fatty acids (Barker, 1994). However, if resources are plentiful in the postnatal environment, the individual may be at increased risk for health problems. This hypothesis is supported by data showing that detrimental effects seem absent when the postnatal environment continues to be lacking in resources (Stanner & Yudkin, 2001), suggesting that mismatch (rather than undernutrition) may be the root cause.

Developmental mismatch and psychological outcomes. The developmental psychopathology model—the currently dominant paradigm for understanding children's psychological and behavioral responses to harsh environments—views early adversity as a stressor that perturbs "normal" development, placing individuals at risk for pathologies, such as depression, anxiety disorders, and drug abuse. The likelihood of developing a disorder is especially high in children who are vulnerable due to temperamental, genetic, and neurobiological risk factors (see, e.g., Cicchetti & Lynch, 1995; for discussion, see Ellis, Boyce, et al., 2011; Oitzl, Champagne, van der Veen, & de Kloet, 2010). This perspective has generated important discoveries concerning risk factors that contribute to undesirable developmental outcomes. However, the developmental psychopathology model has a limited scope: it focuses on pathological effects of early adversity, ignoring the benefits for those individuals who develop fitness-enhancing responses to harsh environments. In contrast, an evolutionary developmental approach suggests that development generally increases the adaptive fit between organisms and their environment—unless environmental conditions fall outside the species-typical range.

Research based on the developmental psychopathology model shows that individuals developing in harsh environments are more at risk for psychological and behavioral problems than individuals growing up in supportive environments and that the effects of early adversity may persist even when individuals move to more supportive conditions (e.g., after adoption). These findings are critically important. However, little research has explored whether children growing up in harsh conditions may achieve better developmental (fitness-relevant) outcomes in harsh environments, compared with children who develop in supportive environments and confront harsh conditions later in life. An evolutionary developmental perspective suggests that mismatch *in either direction* entails fitness costs (Cameron et al., 2005; Cameron et al., 2008; Ellis, Boyce, et al., 2011; Schmidt, 2011). This prediction does not follow from the developmental psychopathology model, which regards harsh environments as generally placing individuals at risk. Thus, although both models predict that moving from a harsh to a safe environment may put individuals at a disadvantage (compared with individuals who spend their entire lifetime in the

safe environment), only the evolutionary developmental viewpoint predicts that moving to a harsh environment after developing in safe conditions also carries costs (compared with being born and raised in the harsh environment).

This key prediction has received initial support from studies of rodent cognition and behavior (for reviews, see Champagne, de Kloet, & Joëls, 2009; Oitzl et al., 2010). The early rearing environment of rat pups, specifically the extent of maternal care (licking and grooming), influences stress responsivity, brain development, and cognitive performance in later life (Champagne et al., 2008; for a recent review, see Weaver, 2009). In one study, the offspring of high-caring mothers exhibited improved learning and memory compared with offspring of low-caring mothers when they were tested in a *low to moderately stressful* learning context (Bredy, Humpartzoomian, Cain, & Meaney, 2003). This result is consistent with both the developmental psychopathology model and the evolutionary developmental perspective. But what happens when rats with different early experiences are compared in a *highly stressful* learning context? When tested in high-stress conditions, rat pups of low-caring mothers learned fearful stimuli faster than pups of high-caring mothers, also displaying better memory performance. The finding that the pattern of performance reversed when pups were tested in high-stress conditions suggests it is mismatch itself that imposes costs to cognitive performance.

Subsequent studies have replicated these results and illuminated the neurohormonal mechanisms involved: high levels of the stress hormone corticosterone facilitate long-term potentiation in the dentate gyrus, improving hippocampal synaptic plasticity (Oomen et al., 2010). These results support the evolutionary hypothesis and contradict the belief that nurturing environments always enrich emotional and cognitive development. In a similar vein, other rodent studies show that early handling by humans (a stressful experience) benefits animals only when their later environment includes stress, but creates disadvantages if animals are later placed in nonstressful environments (Laban, Dimitrijevic, van Hoersten, Markovic, & Jancovic, 1995).

The notion that stressors may positively influence developmental outcomes in some conditions is also part and parcel of research on arousal regulation and resilience (Rutter, 2006). Research on rodents, monkeys, and humans reveals that individuals exposed to mild stressors during development cope better with stressors later in life than individuals who were never exposed to such stressors—the stress inoculation hypothesis (Garmezy, 1991). For instance, squirrel monkeys who were never separated from their mothers in infancy respond to separations later in life with greater anxiety, diminished exploration of novel situations, and increased stress levels of cortisol compared with controls who did experience brief separations from their mothers as infants (Lyons, Parker, Katz, & Schatzberg, 2009). Similar results have been obtained in mice (for a review, see Macrì, Zoratto, & Laviola, 2011).

Current explanations of stress inoculation effects focus primarily on physiological mechanisms. A complementary concern is why mechanisms are structured such that later resilience is a function of earlier experience. The evolutionary developmental perspective proposes that early experiences with stressful events regulate and direct the developing organism toward an appropriate life history trajectory, tuning its stress response system to the state of the environment (Boyce & Ellis, 2005; Del Giudice et al., 2011; Ellis et al., 2005). When developmental conditions correspond to those experienced later in life, the outcome of this process is adaptive. However, when these conditions differ—for instance, when an organism develops in a supportive environment but is confronted with stressful events later in life—individuals may become maladapted and be worse off compared with what they would have been if they were developmentally prepared to deal with particular hardships.

The evolutionary developmental hypothesis of bidirectional costs to mismatch does not imply that irrespective of environmental conditions, well-matched individuals attain equal fitness (Ellis et al., 2012). For instance, individuals who develop in harsh environments may on average achieve lower fitness than individuals born in supportive environments; nonetheless, these individuals should be better adapted to environmental harshness than conspecifics who developed in safer conditions. Nor does an evolutionary developmental perspective imply that mismatch in either direction imposes equivalent costs: for instance, individuals moving from harsh to safe environments may suffer a smaller reduction in fitness than individuals moving from safe to harsh environments.

Prenatal Programming and Epigenetic Transmission

We have thus far focused on developmental mismatch resulting from the effects of postnatal environments. However, mismatch can also be induced prenatally. Fetuses may be sensitive, for instance, to maternal stress hormones (which carry information about the dangerousness and unpredictability of the external environment) or to the rate of their own growth (which carries information about the availability of food and the mother's energetic reserves). Many studies have found links between maternal stress during pregnancy and subsequent developmental patterns in offspring. Maternal stress appears associated with a range of psychological outcomes in offspring, including hyperactivity, anxiety and vigilance, aggression and externalizing behavior, and readily distracted attention (reviewed in Glover, 2011). These effects may reflect predictive adaptive responses, with the fetus preparing for a dangerous or unpredictable postnatal environment.

The transfer of an adaptive phenotype from one generation to the next is often achieved by *epigenetic transmission*, that is, the transmission of specific patterns of gene expression from parents to offspring (Bateson et al., 2004; Harper, 2005; Meaney, 2001, 2010). Recent studies suggest that common pathways may be involved in epigenetic transmission occurring prenatally and postnatally. For instance, modulation of rat pups' stress responsivity is achieved by epigenetic regulation of the expression of the glucocorticoid receptor gene, based on early maternal care (licking and grooming; see Meaney, 2010, for a comprehensive review). In humans, modifications in the epigenetic regulation of the glucocorticoid receptor are linked to both maternal stress during pregnancy (Oberlander et al., 2008) and childhood abuse (McGowan et al., 2009). Thus, the same epigenetic mechanism may function as the common pathway for prenatal and postnatal effects on developmental trajectories.

Epigenetic mismatch may arise when the environment of the previous generation provides the fetus with a wrong estimate about its own postnatal environment (e.g., via either the intrauterine environment or postnatal care). Epigenetic transmission (and subsequent mismatch) may also occur across more than two genera-

tions, reflecting a combination of the maternal and grandmaternal environments (Bateson et al., 2004; Champagne, 2010). Thus, paradoxically, rapid improvements in nutrition and other environmental dimensions may have damaging effects on the health of those people whose parents and grandparents lived in impoverished conditions (Bateson et al., 2004; Heijmans et al., 2008).

Implications and Future Directions

Implications for developmental theory and research. Developmental mismatch has emerged as an important topic in the study of health and disease (Barker, 1994; Gluckman et al., 2005; Heijmans et al., 2008; Kuzawa, 2005; Kuzawa & Quinn, 2009). Psychology also has a rich tradition of studying mismatch, documenting the effects of adoption on biopsychological outcomes (e.g., Gunnar et al., 2001; Rutter, 2006). However, extant research (based on the developmental psychopathology model) has focused primarily on developmental outcomes of moving *from* harsh *to* supportive environments (e.g., Gunnar et al., 2001). Only recently, scholars have begun investigating the costs associated with moving *from* supportive *to* harsh environments by comparing individuals who develop and remain in a harsh environment with those who move from a supportive to a harsh environment (Champagne et al., 2008; Oomen et al., 2010). This bidirectional focus fits with an evolutionary developmental perspective, which predicts that individuals will benefit from tailoring their phenotype to local environmental conditions—"good" and "bad." To our knowledge, empirical work has yet to begin exploring the physical and psychological consequences of bidirectional mismatch in humans; there is much scope for important and innovative research.

Other avenues for future research include the role of epigenetic mechanisms in the emergence of developmental mismatch. To date, nearly all research has focused on the effects of environmental changes on *physical* health outcomes. However, just as physical environments change (e.g., improved nutrition, health care), so do *psychological* environments (e.g., changes in family structure, childrearing, size and structure of the peer group, access to media). Recent studies reveal that epigenetic regulation of gene expression can influence psychological traits, including the development of social behavior, cognition, personality, and psychopathology (e.g., Isles, Davies, & Wilkinson, 2006; Kaminsky et al., 2008; Mill & Petronis, 2008). Combining these findings with high modern migration rates and rapidly changing environments in many parts of the world, it seems plausible that epigenetic mismatch of psychological traits will become an important avenue of future research.

Implications for intervention. Assuming that future work will uncover more instances of developmental mismatch, interventions that "repair" some of those mismatches may become desirable. Insight into the evolved design of calibration mechanisms could then provide useful indications about how to facilitate desirable outcomes. We hypothesize that developmental trajectories may be more easily reversed when the mechanisms involved are confronted with inputs that reliably co-occurred with environmental change during human evolutionary history. What inputs are relevant will depend on the developmental domain of interest.

One intriguing possibility, raised by recent advances in epigenetic research, is to act directly on the mechanisms regulating genetic expression. New studies with rodents show that the effects of epigenetic programming—a product of early care experiences (maternal licking and grooming, and arched-back nursing)—can sometimes be undone later in life (see Weaver, 2009). These reversals are accomplished by chemically modifying DNA methylation and chromatin, which affect the hypothalamic–pituitary–adrenal (HPA) axis and brain development/cognitive performance, removing the long-term effects of maternal care on gene expression in the offspring. This method has been used to alter stress responses in adult rats. Although the current state of knowledge about epigenetic programming is limited, and so caution is appropriate, it is not inconceivable that in the future humans may benefit from similar techniques. These options, if they become available, will raise important ethical issues in psychological intervention.

Finally, if future research confirms that mismatch in either direction entails costs, this raises still more ethical questions. In particular, when children live in harsh, dangerous social contexts, promoting "adaptive" development (in the developmental psychopathology sense) through external intervention may result in mismatches between the child and its environment, leaving the child unprepared to deal with environment-specific challenges. For example, if high vigilance reflects defensive processes (rather than dysfunction), lowering these defenses without changing the broader developmental context may actually increase the child's vulnerability (e.g., of attack or disrespect by peers).

Developmental Mismatch Resulting From Probabilistic Sampling

In the previous section, we described how developmental mismatch can result from changing environments. In this section, we explain how reliance on probabilistic information can result in developmental mismatch, even when the developmental environment remains stable over time (i.e., within the life span of individuals). We note upfront that some of the ideas presented in this section are speculative and remain to be tested; we include them because they might prove empirically productive and because they help illustrate how evolutionary models can contribute to the study of developmental maladaptation.

Probabilistic Sampling

As noted, organisms often assess fitness-relevant dimensions of their environment—such as mortality rates, local sex ratios, resource distributions—and adjust phenotypic development accordingly. In mathematical models of plasticity, this assessment stage is often conceptualized as a sampling process (see Houston & McNamara, 1999; Mangel & Clark, 1988; Schlichting & Pigliucci, 1998), in which individuals obtain *cues* to the state of the environment (e.g., secure attachment relations may function as "safety" cues). These cues determine the organism's estimate of the environmental state, which guides its developmental trajectory (e.g., specializing in the detection of threats; Pollak, 2008). In the real world, a cue could be any property of the environment, social or physical, that is predictive of current or future states of the environment.

In a world of perfect information, a cue correlates perfectly and exclusively with the corresponding environmental state (e.g., high levels of aggression always and only occur in dangerous environments). In such conditions, organisms need to obtain only a single cue to "know" the environmental state: the cue provides, as it

were, a transparent window onto reality. Often, however, cues may indicate the environmental state probabilistically. After having sampled a probabilistic cue, the likelihood of different environmental states changes depending on the *cue validity* (McNamara, Green, & Olssen, 2006), which specifies the extent to which a particular cue is more likely in one environmental state than another (e.g., how much more likely violence is in a dangerous world compared with a safe world). Of course, cues are useful to the extent that they discriminate between different states of the environment.

Because natural selection is an optimizing process (within constraints), it should favor information processing *consistent* with Bayesian learning, the optimal way of information updating (McNamara et al., 2006).[3] In Bayesian terms, the evidence provided by a cue will be used to update the existing estimate of the environment (prior probability)—in evolutionary models, this estimate often reflects the distribution of ancestral environmental states—yielding a revised estimate (posterior probability). The higher the cue validity, the larger its effect on belief updating. Unless cues are very reliable, observing a single cue does not provide much certainty; however, by sampling repeatedly (McNamara et al. 2006) or by aggregating different types of cues (Fawcett & Johnstone, 2003), organisms attain more precise estimates of the environmental state they are in.

When cues are probabilistic, careful "developmental decision making" may be adaptive. Rather than favoring phenotypes that irrevocably commit to a particular developmental trajectory based on a small set of cues, natural selection may favor incremental tailoring of the phenotype to the local environment, combined with continued sampling. Most evolutionary models of plasticity, however, do not incorporate such continued sampling; they represent ontogeny as a two-stage stage process (for exceptions, see Houston & McNamara, 1999; Mangel & Clark, 1988; Schlichting & Pigliucci, 1998). In the first stage, organisms may sample a cue to the environmental state (e.g., a cue suggesting the environment is dangerous). In the second stage, organisms adopt a phenotype (e.g., they become danger-adapted). The goal of these models is to understand when organisms maximize their fitness by sampling environmental cues (thus improving their estimate) versus developing a fixed phenotype without ever sampling.

Incorporating Development in Models Of Plasticity

Despite the fact that most evolutionary models of plasticity assume a two-stage life history, development is typically a constructive process, in which phenotypes incrementally adapt to local ecologies (Frankenhuis & Panchanathan, 2011a, Frankenhuis & Panchanathan, 2011b). The constructive nature of developmental processes is especially apparent in organisms that start building traits during a life stage at which they are not needed yet (Gluckman et al., 2005). Organisms might do this because it takes time to develop a phenotype. For instance, before birth—inside the mother or the egg—some organisms begin developing protective armor to reduce predation risk in the postnatal environment (Agrawal, Laforsch, & Tollrian, 1999), thick fur coats for staying warm, or a wing shape, coloration, and even behavior patterns appropriate to the postnatal environment (see Gluckman et al., 2005).

When the time allocated to constructing a phenotype correlates with the adaptive fit to the environment (i.e., the more time invested, the better the fit), organisms may benefit from specializing earlier in ontogeny (Frankenhuis & Panchanathan, 2011a, Frankenhuis & Panchanathan, 2011b; see also Auld et al., 2010; DeWitt et al., 1998). For instance, water fleas that start tailoring their phenotype prenatally toward a predator-rich environment develop more effective protective helmets than water fleas that specialize only after birth (Agrawal et al., 1999). Social insects gradually develop increased efficiency at performing a variety of everyday tasks, including food handling techniques, identification of food sources, nest repair, nestmate recognition, comb building, and nest climate control (Chittka & Muller, 2009). Many fish species incrementally fine-tune morphology and behavior for capturing locally abundant prey, increasing foraging efficiency (Dill, 1983). Human development is a particularly time-consuming process. For instance, in many hunter–gatherers societies, men begin to produce more calories than they consume only as late as their late teens (or even early twenties), because high levels of knowledge, coordination, skill, and strength are required to detect and extract the difficult-to-acquire resources that humans consume (Kaplan, Hill, Lancaster, & Hurtado, 2000; Walker, Hill, Kaplan, & McMillan, 2002).

If prolonged practice is required for the successful function of some activity in adulthood, then it may be optimal to establish a learning track early in development (Draper & Harpending, 1982). In humans, middle childhood constitutes an important phase of social learning (Campbell, 2006; Lancy & Grove, 2011). In fact, middle childhood may be a critical stage in the development of individual and sex differences in life history strategies (Del Giudice, 2009; Del Giudice, Angeleri, & Manera, 2009; Del Giudice & Belsky, 2011). Starting to enact one's behavioral strategies before reproductive maturity may allow children to engage in specialized learning (e.g., by practicing cooperative or aggressive social tactics), while receiving feedback about the success (or lack thereof) of the chosen strategy.

The Trade-Off Between Sampling and Specialization as a Source of Mismatch

If there is a premium on earlier specialization, why not develop traits as early as possible? Despite its benefits, earlier specialization may also entail costs (Boyce & Ellis, 2005; Frankenhuis & Panchanathan, 2011a, Frankenhuis & Panchanathan, 2011b). In particular, specializing earlier implies less time for sampling environmental cues, increasing the risk of miscalibration (Nepomnaschy & Flinn, 2009). Miscalibration may be costly for a number of reasons (Gluckman et al., 2005). First, even when phenotypic development can be reversed in light of new experiences, there may be costs to shifting from one phenotype to another. For example, it may require reworking of tissues that have committed to other functions. Second, development is a path-dependent process, and what happens during earlier stages of ontogeny might narrow the range of future options. For instance, developing a

[3] This assumption does not imply that animals cognitively represent information in Bayesian terms (McNamara et al., 2006). These programs could be instantiated in organisms as nothing more than a series of if–then rules, which can be quite simple and do not require sophisticated computation.

larger body now may preclude faster running speed in the future, because developing the required musculature would be too metabolically expensive. Third, in some cases developmental specialization may be irreversible, in which case individuals are "stuck" their entire lifetime with a mismatched phenotype (see West-Eberhard, 2003).

It follows that organisms may face a crucial trade-off between sampling and specialization. On the one hand, individuals benefit from specializing earlier, because earlier specialization allows for more time to achieve an adaptive fit with the estimated environmental state. On the other hand, earlier specialization implies more uncertainty about the state of the environment, and therefore a greater likelihood of developing a maladaptive phenotype.

Frankenhuis and Panchanathan (2011a, 2011b) incorporated this trade-off in a mathematical model of plasticity. This model explores how different evolutionary environments favor more or less sampling of cues in early ontogeny, before organisms start specializing their phenotype. As one might expect, the results show that organisms are more likely to develop a mismatched phenotype when cues are less informative. When cue validities are moderate to low, it takes too many samples to obtain an accurate estimate of the environment; hence, organisms may learn some, but not much, specializing early in ontogeny and sometimes developing a mismatched phenotype. However, counter to intuitions, some individuals develop an incorrect phenotype even when cues are highly informative. These individuals have sampled a small number of cues and happened to obtain a sample that is unlikely, yet consistently, present in their environment. When cues are highly reliable, organisms evolve to trust them, usually to their advantage (enabling them to specialize early) but sometimes to their detriment. As with risk taking, natural selection may favor strategies (in this case, minimal sampling) that on average maximize fitness, even when this strategy harms some individuals who follow it.

The extent of mismatch also depends on the fitness function, which describes how increments in specialization translate into fitness. Modeling results show that organisms are most likely to develop a mismatched phenotype when marginal fitness returns increase with each increment of specialization. The reason is that progressive increments in specialization garner larger and larger fitness gains; hence, organisms are pressed to specialize early in ontogeny, even though their estimate about the environmental state is not very accurate. Mismatch is least likely to result when the marginal fitness function is diminishing: when each subsequent increment of specialization yields a smaller and smaller increment in fitness. In this case, much fitness can be gained with little specialization; hence, it is worth sacrificing some degrees of specialization in order to obtain a better estimate of the environmental state, reducing the risk of mismatch (Frankenhuis & Panchanathan, 2011a, Frankenhuis & Panchanathan, 2011b).

Overall, the model shows that even when organisms have repeated opportunities to sample probabilistic cues, they might still develop mismatched (maladaptive) phenotypes. This is most likely to happen when additional degrees of specialization yield marginally increasing fitness returns and least likely when marginal fitness returns decrease. Strikingly, mismatch may result even when the cue validity is high, and when the environment remains stable across the life span of individuals. It is often assumed that mismatch does *only* occur when the environment is unstable or available cues are of low quality. Both these assumptions are incorrect when there is a trade-off between sampling and specialization.

Implications and Future Directions

Implications for developmental theory and research. Biologists studying developmental plasticity recognize the challenges inherent to probabilistic sampling and therefore use explicit models, methods, and tools for addressing information use by animals. Psychologists can draw upon these models, learning from existing findings and using similar techniques to explore novel research questions. In evolutionary developmental psychology, it is sometimes assumed that organisms have access to highly reliable cues to the environmental state. This assumption may keep out of sight inferential processes that contribute to developmental mismatch. More detailed consideration of how organisms use information to adjust their development can illuminate the design of adaptive developmental mechanisms, including the conditions that may result in them producing maladapted phenotypes. How do individuals estimate current and future environmental states? How do developing organisms handle conflicting information? Can we measure cue validities in humans? These and other questions remain to be resolved.

Implications for intervention. The sampling versus specialization model (Frankenhuis & Panchanathan, 2011a, Frankenhuis & Panchanathan, 2011b) further shows that individuals may come to vary in the fraction of their ontogeny spent sampling, depending on the consistency in their sampled cue set. Because sampling is a probabilistic process, some individuals receive a homogenous sample, resulting in a confident estimate about the environmental state. These individuals will specialize early. Others receive a heterogeneous, uninformative set of cues. They will keep sampling for longer. As a consequence, individual differences in plasticity (as in sensitivity to environmental cues used to regulate developmental trajectories) may result.

This finding suggests a novel, and perhaps counterintuitive, implication for intervention. Potentially, the duration of plasticity can be prolonged by feeding the developmental system an ambiguous cue set (as this increases uncertainty about the environmental state), facilitating continued flexibility in adapting to changing conditions. If so, children living in harsh family environments may benefit from receiving highly nurturing experiences, even if in brief bouts, if it is likely that their environment will improve later in life. Moreover, it could be more beneficial to distribute such inconsistent experiences throughout ontogeny, as opposed to all at once (since the latter would reduce uncertainty, potentially increasing specialization). This would maximize the duration of plasticity, preventing children from either (a) fully specializing to a harsh environment that will eventually change or (b) specializing too soon to a safe environment, which has not arrived yet, based on consistent cues of safety and nurturance (provided by the intervention). Note that this prediction has not been tested yet and thus should be weighted appropriately. Crucially, it only applies to those developmental domains in which a trade-off between sampling and specialization was consistently present across evolutionary time.

Conclusions

We have described three ways in which biologically adaptive developmental mechanisms can result in maladaptive individual-level outcomes: (a) natural selection may favor risky strategies that enhance fitness on average, but which have detrimental consequences for a subset of individuals; (b) mismatch may result when organisms experience environmental change during ontogeny, for instance, because they move from one environment to another; and (c) organisms may learn about their environments in order to develop an appropriate phenotype; when cues indicate the environmental state probabilistically, sampling processes may produce mismatch. We have outlined important implications for psychological theory, research, and intervention.

As developmental psychology starts to embrace evolution as its metatheory (Bjorklund & Pellegrini, 2000; see also Ploeger, van der Maas, & Raijmakers, 2008), it becomes important for researchers to develop a nuanced understanding of adaptive developmental processes and their diverse outcomes. Because adaptation and maladaptation are intertwined in the natural world (Nesse, 2005a), theoretical and empirical investigations of maladaptation should be incorporated. Without a good understanding of maladaptation, there is the risk that adaptive responses, which appear harmful to well-being, are mistaken for pathological responses (see Nesse & Jackson, 2006).

Biological theories of maladaptation tend to be grounded in mathematical modeling. In our view, evolutionary developmental scholars can learn from models developed in evolutionary biology and can use similar techniques to advance the study of humans. Crespi (2000) noted that "conditions for unambiguously identifying maladaptation are considerably more stringent than those for demonstrating adaptation" (p. 623). If so, mathematical models will prove invaluable in endeavors aimed at discerning the precise conditions leading to maladaptation, singling out key variables, and determining what counts as relevant data for demonstrating maladaptation.

The processes we discussed in this article represent a subset of the myriad sources of maladaptation in nature, making the biology of maladaptation a rich and complex topic. Other sources of maladaptation include exposure to evolutionarily novel environments (Ellis et al., 2012), trade-offs in the regulation of adaptive defenses (Nesse, 2005a, Nesse, 2005b), and social learning of fitness-detrimental behaviors (Boyd & Richerson, 1985). In addition, maladaptation can result from deleterious mutations; from pleiotropic effects, whereby a genetic variant that is beneficial for one phenotypic trait or at a particular life stage, imposes costs on another; from genetic conflicts between parent and offspring, or between maternal and paternal genes within the genome; and from sexually antagonistic selection, occurring when a genetic variant is advantageous to members of one sex but damaging to the other sex (see Crespi, 2000, Crespi, 2010; Gangestad & Yeo, 1997).

Natural selection is a hill-climbing process, incrementally favoring better psychological and behavioral designs. However, this by no means implies that the developmental mechanisms it produces always generate optimal phenotypes: natural selection is a blind process, with no foresight, constrained by chance, trade-offs, probability, and changing environments. For these and other reasons, adaptation and maladaptation are two sides of the same coin and should not be studied independently. Here, we demonstrated that evolutionary approaches not only yield insights into adaptive outcomes but can also illuminate the conditions leading to maladaptation. We hope this perspective provides additional nuance to the dialectic between the developmental psychopathology model and the evolutionary approach to human development.

References

Agrawal, A. A., Laforsch, C., & Tollrian, R. (1999, September 2). Transgenerational induction of defences in animals and plants. *Nature, 401,* 60–63. doi:10.1038/43425

Archer, J. (2009). Does sexual selection explain human sex differences in aggression? *Behavioral and Brain Sciences, 32,* 249–266. doi:10.1017/S0140525X09990951

Auld, J. R., Agrawal, A. A., & Relyea, R. A. (2010). Re-evaluating the costs and limits of adaptive phenotypic plasticity. *Proceedings of the Royal Society B: Biological Sciences, 277,* 503–511. doi:10.1098/rspb.2009.1355

Barker, D. (1994). *Mothers, babies, and disease in later life.* London, England: British Medical Journal.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, Foley, R. A., . . . Sultan, S. E. (2004, July 22). Developmental plasticity and human health. *Nature, 430,* 419–421. doi:10.1038/nature02725

Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development, 62,* 647–670. doi:10.2307/1131166

Bjorklund, D. F., & Pellegrini, A. D. (2000). Child development and evolutionary psychology. *Child Development, 71,* 1687–1708. doi:10.1111/1467-8624.00258

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology, 17,* 271–301. doi:10.1017/S0954579405050145

Boyd, R., & Richerson, P. J. (1985). *Culture and the evolutionary process.* Chicago, IL: University of Chicago Press.

Bredy, T. W., Humpartzoomian, R. A., Cain, D. P., & Meaney, M. J. (2003). Partial reversal of the effect of maternal care on cognitive function through environmental enrichment. *Neuroscience, 118,* 571–576. doi:10.1016/S0306-4522(02)00918-1

Brezina, T., Tekin, E., & Topalli, V. (2009). "Might not be a tomorrow": A multimethods approach to anticipated early death and youth crime. *Criminology, 47,* 1091–1129. doi:10.1111/j.1745-9125.2009.00170.x

Brown, G. R., Laland, K. N., & Borgerhoff Mulder, M. (2009). Bateman's principles and human sex roles. *Trends in Ecology and Evolution, 24,* 297–304. doi:10.1016/j.tree.2009.02.005

Byrnes, J. P., Miller, D. C., & Schafer, W. D. (1999). Gender differences in risk taking: A meta-analysis. *Psychological Bulletin, 125,* 367–383. doi:10.1037/0033-2909.125.3.367

Cameron, N. M., Champagne, F. A., Parent, C., Fish, E. W., Osaki-Kuroda, K., & Meaney, M. J. (2005). The programming of individual differences in defensive responses and reproductive strategies in the rat through variations in maternal care. *Neuroscience and Biobehavioral Reviews,* 29, 843–865. doi:10.1016/j.neubiorev.2005.03.022

Cameron, N. M., Del Corpo, A., Diorio, J., Mackallister, K., Sharma, S., & Meaney, M. J. (2008). Maternal programming of sexual behavior and hypothalamic-pituitary gonadal function in the female rat. *PLoS ONE,* 3, e2210. doi:10.1371/journal.pone.0002210

Campbell, B. (2006). Adrenarche and the evolution of human life history. *American Journal of Human Biology, 18,* 569–589. doi:10.1002/ajhb.20528

Carroll, S. P. (1993). Divergence in male mating tactics between two populations of the soapberry bug: I. Guarding versus nonguarding. *Behavioral Ecology, 4,* 156–164. doi:10.1093/beheco/4.2.156

Carroll, S. P., & Corneli, P. S. (1995). Divergence in male mating tactics between two populations of the soapberry bug: II. Genetic change and the evolution of a plastic reaction norm in a variable social environment. *Behavioral Ecology, 6,* 46–56. doi:10.1093/beheco/6.1.46

Champagne, D. L., Bagot, R. C., van Hasselt, F., Ramakers, G., Meaney, M. J., de Kloet, . . . Krugers, H. (2008). Maternal care and hippocampal plasticity: Evidence for experience-dependent structural plasticity, altered synaptic functioning, and differential responsiveness to glucocorticoids and stress. *The Journal of Neuroscience, 28,* 6037–6045. doi:10.1523/JNEUROSCI.0526-08.2008

Champagne, D. L., de Kloet, R., & Joëls, M. (2009). Fundamental aspects of the impact of glucocorticoids on the (immature) brain. *Seminars in Fetal & Neonatal Medicine, 14,* 136–142. doi:10.1016/j.siny.2008.11.006

Champagne, F. A. (2010). Epigenetic influence of social experiences across the lifespan. *Developmental Psychobiology, 52,* 299–311. doi:10.1002/dev.20436

Chisholm, J. S. (1993). Death, hope, and sex: Life-history theory and the development of reproductive strategies. *Current Anthropology, 34,* 1–24. doi:10.1086/204131

Chisholm, J. S. (1999). Attachment and time preference: Relations between early stress and sexual behavior in a sample of American university women. *Human Nature, 10,* 51–83. doi:10.1007/s12110-999-1001-1

Chisholm, J. S., Quinlivan, J. A., Petersen, R. W., & Coall, D. A. (2005). Early stress predicts age at menarche and first birth, adult attachment, and expected lifespan. *Human Nature, 16,* 233–265. doi:10.1007/s12110-005-1009-0

Chittka, L., & Muller, H. (2009). Learning, specialization, efficiency and task allocation in social insects. *Communicative & Integrative Biology, 2,* 151–154.

Cicchetti, D., & Blender, J. A. (2004). A multiple-levels-of-analysis approach to the study of developmental processes in maltreated children. *Proceedings of the National Academy of Sciences of the United States of America, 101,* 17325–17326. doi:10.1073/pnas.0408033101

Cicchetti, D., & Lynch, M. (1995). Failures in the expectable environment and their impact on individual development: The case of child maltreatment. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology: Vol. 2. Risk, disorder, and adaptation* (pp. 32–71). New York, NY: Wiley.

Crespi, B. J. (2000). The evolution of maladaptation. *Heredity, 84,* 623–629. doi:10.1046/j.1365-2540.2000.00746.x

Crespi, B. J. (2010). The origins and evolution of genetic disease risk in modern humans. *Annals of the New York Academy of Sciences, 1206,* 80–109. doi:10.1111/j.1749-6632.2010.05707.x

Cross, C. P., Copping, L. T., & Campbell, A. (2011). Sex differences in impulsivity: A meta-analysis. *Psychological Bulletin, 137,* 97–130. doi:10.1037/a0021591

Dall, S. R. X., Giraldeau, L.-A., Ollson, O., McNamara, J. M., & Stephens, D. W. (2005). Information and its use by animals in evolutionary ecology. *Trends in Ecology and Evolution, 20,* 187–193. doi:10.1016/j.tree.2005.01.010

Daly, M., & Wilson, M. (1988). *Homicide.* New York, NY: de Gruyter.

Daly, M., & Wilson, M. (2005). Carpe diem: Adaptation and discounting the future. *The Quarterly Review of Biology, 80,* 55–60. doi:10.1086/431025

Del Giudice, M. (2009). Sex, attachment, and the development of reproductive strategies. *Behavioral and Brain Sciences, 32,* 1–21. doi:10.1017/S0140525X09000016

Del Giudice, M., Angeleri, R., & Manera, V. (2009). The juvenile transition: A developmental switch point in human life history. *Developmental Review, 29,* 1–31. doi:10.1016/j.dr.2008.09.001

Del Giudice, M., & Belsky, J. (2011). The development of life history strategies: Toward a multi-stage theory. In D. M. Buss & P. H. Hawley (Eds.), *The evolution of personality and individual differences* (pp. 154–176). New York, NY: Oxford University Press.

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience and Biobehavioral Reviews, 35,* 1562–1592. doi:10.1016/j.neubiorev.2010.11.007

DeWitt, T. J., Sih, A., & Wilson, D. S. (1998). Costs and limits of plasticity. *Trends in Ecology and Evolution, 13,* 77–81. doi:10.1016/S0169-5347(97)01274-3

Dill, L. M. (1983). Adaptive flexibility in the foraging behavior of fishes. *Canadian Journal of Fisheries and Aquatic Sciences, 40,* 398–408. doi:10.1139/f83-058

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

Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin, 130,* 920–958. doi:10.1037/0033-2909.130.6.920

Ellis, B. J., & Bjorklund, D. F. (Eds.). (2005). *Origins of the social mind.* New York, NY: Guilford Press.

Ellis, B. J., & Boyce, W. T. (2008). Biological sensitivity to context. *Current Directions in Psychological Science, 17,* 183–187. doi:10.1111/j.1467-8721.2008.00571.x

Ellis, B. J., Boyce, W. T., Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2011). Differential susceptibility to the environment: An evolutionary neuro-developmental theory. *Development and Psychopathology, 23,* 7–28. doi:10.1017/S0954579410000611

Ellis, B. J., Del Giudice, M., Dishion, T. J., Figueredo, A. J., Gray, P., Griskevicius, V., . . . Wilson, D. S. (2012). The evolutionary basis of risky adolescent behavior: Implications for science, policy, and practice. *Developmental Psychology, 48,* 598–623. doi:10.1037/a0026220

Ellis, B. J., Essex, M. J., & Boyce, W. T. (2005). Biological sensitivity to context: II. Empirical explorations of an evolutionary-developmental theory. *Development and Psychopathology, 17,* 303–328. doi:10.1017/S0954579405050157

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,* 204–268. doi:10.1007/s12110-009-9063-7

Ellis, B. J., McFadyen-Ketchum, S., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1999). Quality of early family relationships and individual differences in the timing of pubertal maturation in girls: A longitudinal test of an evolutionary model. *Journal of Personality and Social Psychology, 77,* 387–401. doi:10.1037/0022-3514.77.2.387

Fawcett, T. W., & Johnstone, R. A. (2003). Optimal assessment of multiple cues. *Proceedings of the Royal Society B: Biological Sciences, 270,* 1637–1643. doi:10.1098/rspb.2003.2328

Figueredo, A. J., Hammond, K. R., & McKiernan, E. C. (2006). A Brunswikian evolutionary developmental theory of preparedness and plasticity. *Intelligence, 34,* 211–227. doi:10.1016/j.intell.2005.03.006

Figueredo, A. J., Vásquez, G., Brumbach, B. H., & Schneider, S. M. R. (2004). The heritability of life history strategy: The K-factor, covitality, and personality. *Social Biology, 51,* 121–143.

Frankenhuis, W. E., & Karremans, J. C. (in press). Uncommitted men match their risk taking to female preferences, while committed men do the opposite. *Journal of Experimental Social Psychology*. doi:10.1016/j.jesp.2011.09.001

Frankenhuis, W. E., & Panchanathan, K. (2011a). Balancing sampling and specialization: An adaptationist model of incremental development. *Proceedings of the Royal Society B: Biological Sciences.* Advance online publication, doi:10.1098/rspb.2011.0055

Frankenhuis, W. E., & Panchanathan, K. (2011b). Individual differences in developmental plasticity may result from stochastic sampling. *Perspec-*

tives on Psychological Science, 6, 336–347. doi:10.1177/1745691611412602

Fries, A. B. W., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuropeptides critical for regulating social behavior. *Proceedings of the National Academy of Sciences, 102,* 17237–17240. doi:10.1073/pnas.0504767102

Gangestad, S. W., & Simpson, J. A. (2000). On the evolutionary psychology of human mating: Trade-offs and strategic pluralism. *Behavioral and Brain Sciences, 23,* 573–587. doi:10.1017/S0140525X0000337X

Gangestad, S. W., & Yeo, R. A. (1997). Behavioral genetic variation, adaptation, and maladaptation: An evolutionary perspective. *Trends in Cognitive Sciences, 1,* 103–108. doi:10.1016/S1364-6613(97)89056-0

Garmezy, N. (1991). Resilience in children's adaptation to negative life events and stressed environments. *Pediatric Annals, 20,* 463–466.

Geary, D. C., & Bjorklund, D. F. (2000). Evolutionary developmental psychology. *Child Development, 71,* 57–65. doi:10.1111/1467-8624.00118

Geronimus, A. T., Bound, J., & Waidmann, T. A. (1999). Health inequality and population variation in fertility timing. *Social Science & Medicine, 49,* 1623–1636. doi:10.1016/S0277-9536(99)00246-4

Glover, V. (2011). Prenatal stress and the origins of psychopathology: An evolutionary perspective. *Journal of Child Psychology and Psychiatry, 52,* 356–367. doi:10.1111/j.1469-7610.2011.02371.x

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007). Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology, 19,* 1–19. doi:10.1002/ajhb.20590

Gluckman, P. D., Hanson, M. A., Spencer, H. G., & Bateson, P. (2005). Environmental influences during development and their later consequences for health and disease: Implications for the interpretation of empirical studies. *Proceedings of the Royal Society B, 272,* 671–677. doi:10.1098/rspb.2004.3001

Gunnar, M. R., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology, 13,* 611–628. doi:10.1017/S095457940100311X

Harper, L. V. (2005). Epigenetic inheritance and the intergenerational transfer of experience. *Psychological Bulletin, 131,* 340–360. doi:10.1037/0033-2909.131.3.340

Hartup, W. W., & Stevens, N. (1997). Friendship and adaptation in the life course. *Psychological Bulletin, 121,* 355–370. doi:10.1037/0033-2909.121.3.355

Hawley, P. H. (1999). The ontogenesis of social dominance: A strategy-based evolutionary perspective. *Developmental Review, 19,* 97–132. doi:10.1006/drev.1998.0470

Hawley, P. H. (2007). Social dominance in childhood and adolescence: Why social competence and aggression may go hand in hand. In P. H. Hawley, T. D. Little, & P. Rodkin (Eds.), *Aggression and adaptation: The bright side to bad behavior* (pp. 1–29). Hillsdale, NJ: Erlbaum.

Heijmans, B. T., Tobi, E. T., Stein, A. D., Putter, H., Blauw, G. J., Susser, E. S., . . . Lumey, L. H. (2008). Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences of the United States of America, 105,* 17046–17049. doi:10.1073/pnas.0806560105

Houston, A. I., & McNamara, J. M. (1999). *Models of adaptive behavior: An approach based on state.* Cambridge, England: Cambridge University Press.

Hrdy, S. B. (1999). *Mother nature.* New York, NY: Pantheon.

Isles, A. R., Davies, W., & Wilkinson, L. S. (2006). Genomic imprinting and the social brain. *Philosophical Transactions of the Royal Society B: Biological Sciences, 361,* 2229–2237. doi:10.1098/rstb.2006.1942

Kacelnik, A., & Bateson, M. (1997). Risk-sensitivity: Crossroads for theories of decision-making. *Trends in Cognitive Science, 1,* 304–309. doi:10.1016/S1364-6613(97)01093-0

Kaminsky, Z., Petronis, A., Wang, S., Levine, B., Ghaffar, O., Floden, D., & Feinstein, A. (2008). Epigenetics of personality traits: An illustrative study of identical twins discordant for risk-taking behavior. *Twin Research and Human Genetics, 11,* 1–11. doi:10.1375/twin.11.1.1

Kaplan, H., Hill, K., Lancaster, J., & Hurtado, A. M. (2000). A theory of human life history evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology, 9,* 156–185. doi:10.1002/1520-6505(2000)9:4<156::AID-EVAN5>3.0.CO;2-7

Kochenderfer-Ladd, B. (2004). Peer victimization: The role of emotions in adaptive and maladaptive coping. *Social Development, 13,* 329–349. doi:10.1111/j.1467-9507.2004.00271.x

Kruger, D. J. (2010). Socio-demographic factors intensifying male mating competition exacerbate male mortality rates. *Evolutionary Psychology, 8,* 194–204.

Kruger, D. J., & Nesse, R. M. (2006). An evolutionary life-history framework for understanding sex differences in human mortality rates. *Human Nature, 17,* 74–97. doi:10.1007/s12110-006-1021-z

Kuzawa, C. W. (2005). Fetal origins of developmental plasticity: Are fetal cues reliable predictors of future nutritional environments? *American Journal of Human Biology, 17,* 5–21. doi:10.1002/ajhb.20091

Kuzawa, C. W., & Quinn, E. A. (2009). Developmental origins of adult function and health: Evolutionary hypotheses. *Annual Review of Anthropology, 38,* 131–147. doi:10.1146/annurev-anthro-091908-164350

Laban, O., Dimitrijevic, M., van Hoersten, S., Markovic. B. M., & Jancovic, B. D. (1995). Experimental allergic encephalomyelitis in the rat. *Brain, Behavior, and Immunity, 9,* 9–19. doi:10.1006/brbi.1995.1002

Lancy, D. F., & Grove, M. A. (2011). Getting noticed: Middle childhood in cross-cultural perspective. *Human Nature.* Advance online publication. doi:10.1007/s12110-011-9117-5

Leve, L. D., Winebarger, A. A., Fagot, B. I., Reid, J. B., & Goldsmith, H. H. (1998). Environmental and genetic variance in children's observed and reported maladaptive behavior. *Child Development, 69,* 1286–1298. doi:10.2307/1132266

Low, B. S. (1993). Ecological demography: A synthetic focus in evolutionary anthropology. *Evolutionary Anthropology, 1,* 177–187. doi:10.1002/evan.1360010507

Low, B. S., Hazel, A., Parker, N., & Welch, K. B. (2008). Influences on women's reproductive lives: Unexpected ecological underpinnings. *Cross-Cultural Research, 42,* 201–219. doi:10.1177/1069397108317669

Lyons, D. M., Parker, K. J., Katz, M., & Schatzberg, A. F. (2009). Developmental cascades linking stress inoculation, arousal regulation, and resilience. *Frontiers in Behavioral Neuroscience, 3,* 32. doi:10.3389/neuro.08.032.2009

Macrì, S., Zoratto, F., & Laviola, G. (2011). Early stress regulates resilience, vulnerability, and experimental validity in laboratory rodents through mother–offspring hormonal transfer. *Neuroscience and Biobehavioral Reviews, 35,* 1534–1543. doi:10.1016/j.neubiorev.2010.12.014

Maestripieri, D. (2005). Early experience affects the intergenerational transmission of infant abuse in rhesus monkeys. *Proceedings of the National Academy of Sciences of the United States of America, 102,* 9726–9729. doi:10.1073/pnas.0504122102

Maestripieri, D., & Carroll, K. A. (1998). Child abuse and neglect: Usefulness of the animal data. *Psychological Bulletin, 123,* 211–223. doi:10.1037/0033-2909.123.3.211

Mangel, M., & Clark, C. W. (1988). *Dynamic modeling in behavioral ecology.* Princeton, NJ: Princeton University Press.

Marlowe, F. (2003). The mating system of foragers in the Standard Cross-Cultural Sample. *Cross-Cultural Research, 37,* 282–306. doi:10.1177/1069397103254008

Marshall, P. J., & Kenney, J. W. (2009). Biological perspectives on the effects of early psychosocial experience. *Developmental Review, 29,* 96–119. doi:10.1016/j.dr.2009.05.001

Maynard Smith, J. (1989). *Evolutionary genetics.* Oxford, England: Oxford University Press.

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., . . . Meaney, M. J. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience, 12,* 342–348. doi:10.1038/nn.2270

McIntyre, M. H., & Hooven, C. K. (2009). Human sex differences in social relationships: Organizational and activational effects of androgens. In P. B. Gray & P. T. Ellison (Eds.), *Endocrinology of social relationships* (pp. 225–245). Cambridge, MA: Harvard University Press.

McNamara, J. M., Green, R. F., & Olssen, O. (2006). Bayes' theorem and its applications in animal behavior. *Oikos, 112,* 243–251. doi:10.1111/j.0030-1299.2006.14228.x

McNamara, J. M., Merad, S., & Houston, A. I. (1991). A model of risk-sensitive foraging for a reproducing animal. *Animal Behaviour,* 41, 787–792. doi:10.1016/S0003-3472(05)80345-8

Meaney, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24,* 1161–1192. doi:10.1146/annurev.neuro.24.1.1161

Meaney, M. J. (2010). Epigenetics and the biological definition of Gene × Environment interactions. *Child Development, 81,* 41–79. doi:10.1111/j.1467-8624.2009.01381.x

Mill, J., & Petronis, A. (2008). Pre- and peri-natal environmental risks for attention-deficit hyperactivity disorder (ADHD): The potential role of epigenetic processes in mediating susceptibility. *Journal of Child Psychology and Psychiatry, 49,* 1020–1030. doi:10.1111/j.1469-7610.2008.01909.x

Mishra, S., & Lalumière, M. L. (2008). Risk-taking, antisocial behavior, and life histories. In J. Duntley & T. K. Shackelford (eds.), *Evolutionary forensic psychology* (pp. 139–159). New York, NY: Oxford University Press. doi:10.1093/acprof:oso/9780195325188.003.0008

Nepomnaschy, P., & Flinn, M. (2009). Early life influences on the ontogeny of the neuroendocrine stress response in the human child. In P. Gray & P. Ellison, *The endocrinology of social relationships* (pp. 364–382). Cambridge, MA: Harvard University Press.

Nesse, R. M. (2005a). Maladaptation and natural selection. *Quarterly Review of Biology, 80,* 62–70. doi:10.1086/431026

Nesse, R. M. (2005b). Natural selection and the regulation of defenses: A signal detection analysis of the smoke detector principle. *Evolution and Human Behavior, 26,* 88–105. doi:10.1016/j.evolhumbehav.2004.08.002

Nesse, R. M., & Jackson, E. D. (2006). Evolution: Psychiatric nosology's missing biological foundation. *Clinical Neuropsychiatry, 3,* 121–131.

Nettle, D. (2009). An evolutionary model of low mood states. *Journal of Theoretical Biology, 257,* 100–103. doi:10.1016/j.jtbi.2008.10.033

Nettle, D. (2011). Flexibility in reproductive timing in human females: Integrating ultimate and proximate explanations. *Philosophical Transactions of the Royal Society B: Biological Sciences, 366,* 357–365. doi:10.1098/rstb.2010.0073

Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S., & Devlin, A. M. (2008). Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics, 3,* 97–106. doi:10.4161/epi.3.2.6034

Oitzl, M. S., Champagne, D. L., van der Veen, R., & de Kloet, E. R. (2010). Brain development under stress: Hypotheses of glucocorticoid actions revisited. *Neuroscience and Biobehavioral Reviews, 34,* 853–866. doi:10.1016/j.neubiorev.2009.07.006

Oomen, C. A., Soeters, H., Audureau, N., Vermunt, L., van Hasselt, F., Manders, E., . . . Krugers, H. J. (2010). Severe early life stress improves hippocampal synaptic plasticity and emotional learning under high-stress conditions in adulthood. *Journal of Neuroscience, 30,* 6635–6645. doi:10.1523/JNEUROSCI.0247-10.2010

Orr, H. A. (2009). Fitness and its role in evolutionary genetics. *Nature Review Genetics, 10,* 531–539. doi:10.1038/nrg2603

Panchanathan, K., Frankenhuis, W. E., & Barrett, H. C. (2010). Development: Evolutionary ecology's midwife. *Behavioral and Brain Sciences, 33,* 105–106. doi:10.1017/S0140525X1000021X

Pellegrini, A. D., & Bartini, M. (2001). Dominance in early adolescent boys: Affiliative and aggressive dimensions and possible functions. *Merrill–Palmer Quarterly, 47,* 142–163. doi:10.1353/mpq.2001.0004

Ploeger, A., van der Maas, H. L. J., & Raijmakers, M. E. J. (2008). Is evolutionary psychology a metatheory for psychology? A discussion of four major issues in psychology from an evolutionary developmental perspective. *Psychological Inquiry, 19,* 1–18. doi:10.1080/10478400701774006

Pollak, S. D. (2008). Mechanisms linking early experience and the emergence of emotions. *Current Directions in Psychological Science, 17,* 370–375. doi:10.1111/j.1467-8721.2008.00608.x

Pollak, S. D., & Sinha, P. (2002). Effects of early experience on children's recognition of facial displays of emotion. *Developmental Psychology, 38,* 784–791. doi:10.1037/0012-1649.38.5.784

Pollak, S. D., Vardi, S., Bechner, A. M. P., & Curtin, J. J. (2005). Physically abused children's regulation of attention in response to hostility. *Child Development, 76,* 968–977. doi:10.1111/j.1467-8624.2005.00890.x

Quinlan, R. J. (2007). Human parental effort and environmental risk. *Proceedings of the Royal Society of London B: Biological Sciences, 274,* 121–125. doi:10.1098/rspb.2006.3690

Quinlan, R. J. (2008). Human pair-bonds: Evolutionary functions, ecological variation, and adaptive development. *Evolutionary Anthropology, 17,* 227–238. doi:10.1002/evan.20191

Quinlan, R. J. (2010). Extrinsic mortality effects on reproductive strategies in a Caribbean community. *Human Nature, 21,* 124–139. doi:10.1007/s12110-010-9085-1

Rodkin, P. C., Farmer, T. W., Ruth, P., & Acker, R. V. (2006). They're cool: Social status and peer group support for aggressive boys and girls. *Social Development, 15,* 175–204.

Rogers, A. R. (1994). Evolution of time preference by natural selection. *American Economic Review, 84,* 460–481.

Rutter, M. (2006). Implications of resilience concepts for scientific understanding. *Annals of the New York Academy of Sciences, 1094,* 1–12. doi:10.1196/annals.1376.002

Schlichting, C. D., & Pigliucci, M. (1998). *Phenotypic evolution: A reaction norm perspective.* Sunderland, MA: Sinauer.

Schmidt, M. V. (2011). Animal models for depression and the mismatch hypothesis of disease. *Psychoneuroendocrinology, 36,* 330–338. doi:10.1016/j.psyneuen.2010.07.001

Schmitt, D. P., Shackelford, T. K., & Buss, D. M. (2001). Are men really more "oriented" toward short-term mating than women? A critical review of theory and research. *Psychology, Evolution, & Gender, 3,* 211–239. doi:10.1080/14616660110119331

Sell, A., Tooby, J., & Cosmides, L. (2009). Formidability and the logic of human anger. *Proceedings of the National Academy of Sciences of the United States of America, 106,* 15073–15078. doi:10.1073/pnas.0904312106

Shackman, J. E., Shackman, A. J., & Pollak, S. D. (2007). Physical abuse amplifies attention to threat and increases anxiety in children. *Emotion, 7,* 838–852.

Shirtcliff, E. A., Coe, C. L., & Pollak, S. D. (2009). Early childhood stress is associated with elevated antibody levels to herpes simplex virus type 1. *Proceedings of the National Academy of Sciences of the United States of America, 106,* 2963–2967. doi:10.1073/pnas.0806660106

Smallwood, P. D. (1996). An introduction to risk sensitivity: The use of Jensen's inequality to clarify evolutionary arguments of adaptation and constraint. *American Zoologist, 36,* 392–401.

Stanner, S. A., & Yudkin, J. S. (2001). Fetal programming and the

Leningrad Siege Study. *Twin research, 4,* 287–292. doi:10.1375/1369052012498

Walker, R., Hill, K., Kaplan, H., & McMillan, G. (2002). Age-dependency in hunting ability among the Ache of eastern Paraguay. *Journal of Human Evolution, 42,* 639–657. doi:10.1006/jhev.2001.0541

Wang, X. T., Kruger, D. J., & Wilke, A. (2009). Life history variables and risk-taking propensity. *Evolution and Human Behavior, 30,* 77–84. doi:10.1016/j.evolhumbehav.2008.09.006

Weaver, I. C. G. (2009). Shaping adult phenotypes through early life environments. *Birth Defects Research Part C: Embryo Today, 87,* 314–326.

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

Williams, G. C. (1966). *Adaptation and natural selection.* Princeton NJ: Princeton University Press.

Wilson, M., & Daly, M. (1997). Life expectancy, economic inequality, homicide, and reproductive timing in Chicago neighborhoods. *British Medical Journal, 314,* 1271–1274.

Wilson, M., Daly, M., & Pound, N. (2002). An evolutionary psychological perspective on the modulation of competitive confrontation and risk-taking. In D. Pfaff et al. (Eds.), *Hormones, brain, and behavior* (Vol. 5, pp. 381–408). San Diego, CA: Academic Press. doi:10.1016/B978-012532104-4/50096-2

Winterhalder, B. (2007). Risk and decision-making. In R. I. M. Dunbar and L. Barrett (eds.), *Oxford handbook of evolutionary psychology* (pp. 433–445). New York, NY: Oxford University Press.

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COMMENT

Match Fitness: Development, Evolution, and Behavior: Comment on Frankenhuis and Del Giudice (2012)

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The application of evolutionary thinking to human physical and psychological medicine suggests several pathways through which evolutionary processes affect risk of disease. Among these is the concept of mismatch between an individual and its environment, either because the environment has changed for the whole species (*evolutionary novelty*) or because the environment has changed for an individual during its lifetime (*developmental mismatch*). Here we set a discussion of maladaptation and mismatch as a cause of psychopathology (Frankenhuis & Del Giudice, 2012) in the broader framework of developmental plasticity and life history trade-offs.

Keywords: evolutionary medicine, evolutionary novelty, developmental plasticity, mismatch, trade-off

The concept of mismatch is now well established in evolutionary medicine as one of the major pathways that can affect health and disease risk (Gluckman, Low, Buklijas, Hanson, & Beedle, 2011). The concept has been applied extensively to the risk of diseases such as obesity, diabetes, and myopia and to life-course events such as puberty. For example, with regard to obesity, the model simply posits that humans evolved to be adapted to a particular range of nutritional density and workload, whereas the modern environment now frequently lies outside this range, leading to metabolic capacity being overloaded and consequent obesity and metabolic disease.

This concept is central to a review by Frankenhuis and Del Giudice (2012), who have summarized psychological adaptation and maladaptation within an evolutionary and developmental framework. Their review is useful in no small part because it brings attention to the problems of language and metaphor. Adaptation and maladaptation clearly have very different meanings to the evolutionary biologist and to the practicing psychologist. Beyond this, their review serves to underscore an important tenet of evolutionary medicine and psychology—physiological and psychological mechanisms have both evolved to optimize fitness in the Darwinian sense, and not necessarily health, wellbeing, or longevity. Importantly, Frankenhuis and Del Giudice—particularly in their discussion of risky adaptive strategies—emphasize the need to put the context of evolutionary perspectives on individual variation rather than on group variation. Indeed, it could be argued that one of the great insights of Darwin was to focus on individual variation rather than "type," as was the norm in 19th-century biology, and it remains too easy to fall back into typology rather than assessing the context—biological, developmental, environmental, and social—of the individual.

But the concept of mismatch extends beyond the simple *evolutionary novelty* form of mismatch to that occurring within a life course—*developmental mismatch* (Gluckman et al., 2011). As recognized by Frankenhuis and Del Giudice (2012), the most extensive use of the concept of *developmental mismatch* has been in studies that fall under the rubric of *developmental origins of health and disease*, whereby factors in early life predispose adults to have a greater risk of obesity, heart disease, and diabetes. It has been suggested that the normative processes of developmental plasticity can become maladaptive if there is a mismatch between the circumstances that induce changes in developmental trajectory and the actual environment into which the organism matures (Bateson et al., 2004; Gluckman, Hanson, Spencer, & Bateson, 2005).

There is not a one-to-one link between genotype and phenotype. In virtually all multicellular taxa, a range of phenotypes can develop from a single genotype in response to environmental cues acting in early development. These mechanisms of developmental plasticity are strongly conserved, and there is a large body of literature arguing for their adaptive (in the evolutionary sense of increasing reproductive fitness) role (West-Eberhard, 2003). Developmental plasticity and robustness are often portrayed as polar opposites, but, as has been recently pointed out, there are multiple mechanisms leading to both consistency of phenotype (robustness) and plasticity, and these interact during development (Bateson & Gluckman, 2011). Multiple levels of organization from the molecular to the whole organism are involved. Although much plasticity is limited by critical windows during development, learning can be considered as a set of plastic mechanisms with a much broader temporal capacity.

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A fundamental feature of developmental processes is that of trade-offs, which are often formulated within life-history theory, although essentially any potentially adaptive response to an environmental cue or trait involves some form of trade-off. A favored classification of developmental plasticity uses the temporal dimension of any trade-off as its basis, considering plastic responses as either immediately adaptive or predictively adaptive (Gluckman & Hanson, 2004; Gluckman, Hanson, Spencer, & Bateson, 2005). For example, fetal growth retardation is a case of immediate adaptation in which the organism has reduced its growth rate because of maternal or placental signals of undernutrition so as to survive the fetal phase, but with a reduced chance of postnatal survival being the trade-off of being born smaller. In contrast, other responses do not induce an early phenotypic change or a phenotypic change with concurrent survival advantage. A predictive adaptive response may be a phenotypic change induced early in life for advantage later in the life course, or it may be a shift in developmental trajectory that leads to a phenotype of advantage in the anticipated later environment. To be adaptive, that advantage must be reflected in increased fitness—the most obvious way is in increased survival until and through peak reproduction. Some inductive cues may be reliable, such as in the case of day length inducing differences in coat thickness in the offspring of multivoltine rodents, or they may be probabilistic, as in the case of predicting future nutritional or stressor environments from those experienced in early life. However, shifts in phenotypic development may become disadvantageous later in life if the correlation between the inductive cue and the environmental conditions actually encountered is poor. This general model is well categorized (Gluckman, Hanson, & Beedle, 2007; Gluckman, Hanson, & Spencer, 2005) and has been extensively used to explain the developmental origins of health and disease (Gluckman, Hanson, Cooper, & Thornburg, 2008).

The application of evolutionary arguments to behavior has been fraught with controversy, in no small part because of the deterministic approach taken by the school of thought led by Cosmides and Tooby (Duchaine, Cosmides, & Tooby, 2001). Similarly, the widely held dichotomy of explaining behaviors as either learned (nurture) or innate (itself a word fraught with difficulty; Mameli & Bateson, 2011; but generally assumed to reflect genetic determinants) has been equally limiting. The growing understanding of development both at a systems level and at a molecular level, where there is increasing evidence for the role of epigenetic mechanisms, provides a paradigmatic shift in our understandings of how phenotypes emerge and persist.

Equally, we have a problem of translating concepts relating to behavior in a current generation to those of our evolutionary past. Nowhere is this more obvious than in the concept of fitness itself. *Fitness* has a precise meaning in evolutionary biology and relates to the probability that a trait of the organism will be transmitted to future generations. In practice this is generally interpreted as the number of grand-offspring or fertile offspring carrying that trait. Inclusive fitness extends this concept to allow for kin selection. But formal estimates of fitness in humans can take no account of evolutionarily novel cultural practices, such as contraceptive use, that disconnect behavior from fertility. This limits the way in which hypothesis testing relating to biological, as opposed to cultural, evolution is conducted in modern evolutionary medicine and psychology. A recent review by Nesse (2011) is commended for discussing how to create and test hypotheses in this domain. Equally, it is important to recognize that mismatch is but one set of pathways by which evolutionary mechanisms generate risk for an individual (Gluckman et al., 2011). For example, a number of emotional symptoms and states (anxiety, depression, and phobias) can be interpreted as exaggerated defense mechanisms (Nesse & Ellsworth, 2009).

In their discussion of psychological maladaptation, Frankenhuis and Del Giudice (2012) outline the maladaptive consequences of developmental mismatch, distinguishing two potential mechanisms: (a) a change in environment after initiation of a developmental program based on correct perception of an early life cue and (b) failure to correctly perceive an early life cue even if the lifetime environment remains constant. Here we are beginning to see tantalizing opportunities for intervention by manipulation of early life cues—an approach already demonstrated for the physiological consequences of developmental mismatch (Vickers et al., 2005)—but much research remains to be done, particularly around the identification of critical periods during which intervention is possible before the developmental program "locks in."

Within this evolutionary framework, it is interesting to consider another aspect of risk-taking behavior reflecting both developmental and evolutionary mismatch, namely that associated with adolescence. Indeed, adolescence itself might be considered a consequence of changing social and physical environments leading to a loss of temporal mismatch between different maturing systems—in this case the reproductive system and frontothalamic pathways controlling executive function (Steinberg, 2005). Gluckman and Hanson have argued that adolescence has in recent decades become a much more prominent component of the life course (Gluckman & Hanson, 2006a, 2006b). The age of puberty and therefore the age of onset of adolescence have fallen dramatically since the early 19th century. This appears to be related to improvements in maternal and child nutrition and health. Although speculative, Gluckman and Hanson suggest that this might reflect the removal of constraints on maturation associated with urbanization and agriculture that historically arose from malnutrition and infection. They further speculate that the earlier age of modern puberty might reflect a return to a putative age of maturation in a Paleolithic past. Against this background, other influences such as poor prenatal nutrition or perinatal stress may further modulate life history patterns to accelerate timing to maturation in an environment of risk. Such an explanation is given further support by the observation of accelerated maturation in modern hunter–gatherer groups with high extrinsic juvenile mortality (Walker et al., 2006). But on the other hand, Gluckman and Hanson point out that in modern Western societies the end of adolescence, in other words the age of acceptance as an adult within the community, has been further delayed. The consequence is that the duration of "adolescence" has stretched from perhaps 2 to 4 years at the beginning of the 19th century to more than a decade at present—within 200 years. This is not without consequences—both boys and girls who undergo earlier puberty exhibit higher rates of conduct disorder, depression, and other symptomatology than do their peers who undergo puberty at a normal age (e.g., Michaud, Suris, & Deppen, 2006). In that study, boys in particular showed adverse outcomes, with a nearly fivefold greater risk of attempted suicide.

Neurophysiological studies are casting considerable light on this temporal mismatch and its psychological consequences. Func-

tional studies have shown that aspects of judgment do not mature until into the third decade of life (Cauffman & Steinberg, 2000). Other studies have suggested that risk-taking behaviors and their underlying neural pathways may be particularly exaggerated during adolescence (Somerville & Casey, 2010). But the most intriguing observations are the multiple imaging studies that suggest that frontothalamic pathways are not fully mature until well into the third decade of life (Gogtay et al., 2004; Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). This raises three evolutionary hypotheses that are testable and very relevant to the issues of concern in respect of adolescent behavior.

First, is it simply that executive function, emotional control, and self-control have always been underpinned by a very slowly maturing system, which is reflected in the well-established respect all cultures give to older people for their wisdom and judgment? But what has happened is that those skills were not needed in the less complex societies of the pre-electronic age where people essentially operated in small networks, even if they lived in urban conurbations (one is reminded of Dunbar's (2003) calculation that humans evolved to live in groups of no more than 100 to 150 people). But now with the combination of greater physical and certainly greater communicative ability, these later components of neural maturation are needed to cope optimally in society. In this model, adolescent behaviors can be seen to reflect the evolutionary novelty of the more complex world, and they are given further emphasis by the shifting pattern of biological maturation induced by environmental change.

Second, it could be argued that the changed social milieu means that there are simply more skills to learn, and it takes a longer time to get to the level of social skill necessary in the typical modern environment. There is little or no evidence to support this hypothesis.

But a third hypothesis is one associated with developmental plasticity and cue perception. Could it be that the big changes in the way modern children are reared have changed the patterns of frontothalamic maturation? It is well established in neurobiology, for example in the visual system, that early experience can change the pattern of development of neural pathways. Further, there is compelling evidence that noncognitive capacities expressed in later life are dependent on the pattern of early child rearing (Knudsen, Heckman, Cameron, & Shonkoff, 2006). In the last three decades there has been a dramatic shift in patterns of child rearing, with adolescence becoming less rigid, particularly because of more affluence and more fiscal freedom alongside the freedom created by the Internet and cell phones. On the other hand, the rearing of infants and prepubertal children has become more controlled from very young ages for a variety of reasons. These include the impact of women entering the workforce, requiring greater supervisory control of children for longer each day, media-fueled concerns over child safety, and greater attention being given to formal education from a younger age at preschool. Are we seeing here an example of developmental mismatch in which the maladaptive consequences might benefit from more thoughtful attention to early childhood experience?

In the example of adolescent behavior, we see the conflation of the neurophysiological with the behavioral. Increasingly we must see these as two sides of the same coin. For too long the language of one has been ignored by the other. Indeed, Frankenhuis and Del Giudice (2012) are to be congratulated for emphasizing the need for an integrated approach. Importantly, they realize that child and adolescent development is not tightly canalized; rather, it is a process in which the inherited genotype and perhaps inherited epigenotype give rise to an organism that is subsequently molded by experiential processes, inducing epigenetic and neurobiological changes (learning) that have an evolutionary and adaptive underpinning. Such plastic responses arise from evolved mechanisms that allow an organism to adapt to circumstances that change in a time frame intermediate between that which is coped with by homeostasis on one hand and selection on the other, but they do not necessarily create behaviors that modern society judges to be optimal or healthy. Our world has changed too much for that to be the case.

References

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., . . . Sultan, S. E. (2004). Developmental plasticity and human health. *Nature, 430,* 419–421. doi:10.1038/nature02725

Bateson, P., & Gluckman, P. (2011). *Plasticity, robustness, development, and evolution.* Cambridge, England: Cambridge University Press. doi:10.1017/CBO9780511842382

Cauffman, E., & Steinberg, L. (2000). (Im)maturity of judgment in adolescence: Why adolescents may be less culpable than adults. *Behavioral Sciences & the Law, 18,* 741–760. doi:10.1002/bsl.416

Duchaine, B., Cosmides, L., & Tooby, J. (2001). Evolutionary psychology and the brain. *Current Opinion in Neurobiology, 11,* 225–230. doi:10.1016/S0959-4388(00)00201-4

Dunbar, R. I. M. (2003). The social brain: Mind, language, and society in evolutionary perspective. *Annual Review of Anthropology, 32,* 163–181. doi:10.1146/annurev.anthro.32.061002.093158

Frankenhuis, W. E., & Del Giudice, M. (2012). When do adaptive developmental mechanisms yield maladaptive outcomes? *Developmental Psychology, 48,* 628–642. doi:10.1037/a0025629

Gluckman, P. D., & Hanson, M. A. (2004, September 17). Living with the past: Evolution, development, and patterns of disease. *Science, 305,* 1733–1736. doi:10.1126/science.1095292

Gluckman, P. D., & Hanson, M. A. (2006a). Changing times: The evolution of puberty. *Molecular and Cellular Endocrinology, 254–*255, 26–31. doi:10.1016/j.mce.2006.04.005

Gluckman, P. D., & Hanson, M. A. (2006b). Evolution, development, and timing of puberty. *Trends in Endocrinology and Metabolism, 17,* 7–12. doi:10.1016/j.tem.2005.11.006

Gluckman, P. D., Hanson, M. A., & Beedle, A. S. (2007). Early life events and their consequences for later disease: A life history and evolutionary perspective. *American Journal of Human Biology, 19,* 1–19. doi:10.1002/ajhb.20590

Gluckman, P. D., Hanson, M. A., Cooper, C., & Thornburg, K. L. (2008). Effect of in utero and early life conditions on adult health and disease. *The New England Journal of Medicine, 359,* 61–73. doi:10.1056/NEJMra0708473

Gluckman, P. D., Hanson, M. A., & Spencer, H. G. (2005). Predictive adaptive responses and human evolution. *Trends in Ecology & Evolution, 20,* 527–533. doi:10.1016/j.tree.2005.08.001

Gluckman, P. D., Hanson, M. A., Spencer, H. G., & Bateson, P. (2005). Environmental influences during development and their later consequences for health and disease: Implications for the interpretation of empirical studies. *Proceedings of the Royal Society B: Biological Sciences, 272,* 671–677. doi:10.1098/rspb.2004.3001

Gluckman, P. D., Low, F. M., Buklijas, T., Hanson, M. A., & Beedle, A. S. (2011). How evolutionary principles improve the understanding of human health and disease. *Evolutionary Applications, 4,* 249–263. doi:10.1111/j.1752-4571.2010.00164.x

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., . . . Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *PNAS: Proceedings of the National Academy of Sciences of the United States of America, 101,* 8174–8179. doi:10.1073/pnas.0402680101

Knudsen, E. I., Heckman, J. J., Cameron, J. L., & Shonkoff, J. P. (2006). Economic, neurobiological, and behavioral perspectives on building America's future workforce. *Proceedings of the National Academy of Sciences of the United States of America, 103,* 10155–10162. doi:10.1073/pnas.0600888103

Lebel, C., Walker, L., Leemans, A., Phillips, L., & Beaulieu, C. (2008). Microstructural maturation of the human brain from childhood to adulthood. *NeuroImage, 40,* 1044–1055. doi:10.1016/j.neuroimage.2007.12.053

Mameli, M., & Bateson, P. (2011). An evaluation of the concept of innateness. *Philosophical Transactions of the Royal Society, Series B: Biological Sciences, 366,* 436–443. doi:10.1098/rstb.2010.0174

Michaud, P. A., Suris, J. C., & Deppen, A. (2006). Gender-related psychological and behavioural correlates of pubertal timing in a national sample of Swiss adolescents. *Molecular & Cellular Endocrinology, 254–*255, 172–178. doi:10.1016/j.mce.2006.04.037

Nesse, R. M. (2011). Ten questions for evolutionary studies of disease vulnerability. *Evolutionary Applications, 4,* 264–277. doi:10.1111/j.1752-4571.2010.00181.x

Nesse, R. M., & Ellsworth, P. C. (2009). Evolution, emotions, and emotional disorders. *American Psychologist, 64,* 129–139. doi:10.1037/a0013503

Somerville, L. H., & Casey, B. J. (2010). Developmental neurobiology of cognitive control and motivational systems. *Current Opinion in Neurobiology, 20,* 236–241. doi:10.1016/j.conb.2010.01.006

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences, 9,* 69–74. doi:10.1016/j.tics.2004.12.005

Vickers, M. H., Gluckman, P. D., Coveny, A. H., Hofman, P. L., Cutfield, W. S., Gertler, A., . . . Harris, M. (2005). Neonatal leptin treatment reverses developmental programming. *Endocrinology, 146,* 4211–4216. doi:10.1210/en.2005-0581

Walker, R., Gurven, M., Hill, K., Migliano, A., Chagnon, N., De Souza, R., . . . Yamauchi, T. (2006). Growth rates and life histories in twenty-two small-scale societies. *American Journal of Human Biology, 18,* 295–311. doi:10.1002/ajhb.20510

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

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