# 24 Evolutionary Biology of Hormonal Responses to Social Challenges in the Human Child

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Human children are remarkably tuned-in to their social environments. They are informational sponges, absorbing bits of knowledge from others at a phenomenal pace, equipped with life's most sophisticated and creative communication system (human language). This sensitivity to social interactions is interwoven with the ontogeny of flexible cognitive skills – including empathy, self awareness, social-scenario building, and theory of mind (ToM) – that are the foundation of human relationships. In this chapter I examine the neuroendocrine systems that facilitate the development of these distinctively human sociocognitive adaptations.

Neuroendocrine systems may be viewed as complex sets of mechanisms designed by natural selection to communicate information among cells and tissues. Steroid and peptide hormones, associated neurotransmitters, and other chemical messengers guide behaviors of mammals in many important ways (Ellison, 2001; Lee et al., 2009; Panksepp, 2009). Analysis of patterns of hormone levels in naturalistic contexts can provide important insights into the evolutionary functions of the neuroendocrine mechanisms that guide human behaviors. Here I focus on the apparent evolutionary paradox of neuroendocrine response to psychosocial stressors.

Acute and chronic stressful experiences are associated with a variety of negative health outcomes in humans, including susceptibility to upper respiratory infections (Cohen et al., 2003), anxiety and depression (Heim and Nemeroff, 2001), and coronary heart disease (McEwen, 1998). The effects of psychosocial stress can be substantial: in the rural community of Bwa Mawego, Dominica, where I have studied child health for the past 22 years, overall morbidity among children for the 3–6 days following an acute stress event is more than double the normal rate (Flinn and England, 2003). Studies of populations within the United States indicate that chronic stress is similarly associated with a long-term three-fold increase in adverse health conditions (Cohen et al., 1993, 2007). Exposure to stressful events early in development, moreover, appears to have lifelong effects (Heim et al., 2002; Fox et al., 2007; Kolassa and Elbert, 2007; Meaney et al., 2007; Champagne, 2008; Seckl, 2008). Stress endocrinology is suspected to have an important role in the links between social environment and health. Chronic release of stress hormones such as cortisol in response to psychosocial challenges is posited to have incidental deleterious effects on immune and metabolic regulatory functions (Ader et al., 2001; Sapolsky, 2005). Release of androgens such as testosterone, dehydroepiandrosterone (DHEA), and dehydroepiandrosterone sulfate (DHEAS) are also influenced by social conditions (see Chapter 16 of this volume), and can affect immunocompetence (Muehlenbein and Bribiescas, 2005; Muehlenbein, 2008).

This importance of the social environment for a child's physical and mental health presents an evolutionary puzzle. Why, given the apparent high cost to human health of psychosocial stress, would natural selection have favored links between the psychological mechanisms that assess social challenges, and the neuroendocrine mechanisms that regulate stress and reproductive physiology and downstream immune functions? I approach this question from the integrative evolutionary paradigm of Niko Tinbergen (1963), who emphasized the importance of linking proximate physiological explanations with ontogeny (development), phylogeny (ancestry), and adaptive function (natural selection). My basic argument is that hormonal stress response to psychosocial challenges facilitates the neural remodeling and potentiation that is necessary to adapt to the dynamic informational arms race of the human sociocultural environment.

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## WHY IS THE HUMAN CHILD SO SENSITIVE TO THE SOCIAL ENVIRONMENT?

The human child is a social creature, motivated by and highly sensitive to interpersonal relationships (Gopnik et al., 1999). The life history stage of human childhood enables the development of necessary social skills (Alexander, 1987; Joffe, 1997; Bogin, 1999; Geary and Bjorklund, 2000; Flinn, 2004), including emotional regulation. Learning, practice, and experience are imperative for social success. The information processing capacity used for human social interactions is considerable, and perhaps significantly greater than that involved with foraging skills (Roth and Dicke, 2005).

The child needs to master complex dynamic tasks such as learning the personalities, social biases, relationships, and so forth of peers and adults in the local community, and developing appropriate cognitive and emotional responses to these challenges (Bugental, 2000). The learning environments that facilitate and channel these astonishing aspects of human mental phenotypic plasticity appear to take on a special importance. Much of the data required for the social behavior necessary to be successful as a human cannot be "preprogrammed" into specific, detailed, fixed responses. Social cleverness in a fast-paced, cumulative cultural environment must contend with dynamic, constantly shifting strategies of friends and enemies, and hence needs information from experiential social learning (Flinn, 1997, 2006a). The links among psychosocial stimuli, emotions, and physiological stress response may guide both the acute and long-term neurological plasticity necessary for adapting to the dynamic aspects of human sociality.

## HUMAN SOCIALITY: KEY EVOLUTIONARY PUZZLES

Humans are characterized by a distinctive set of traits, including: (1) large brains; (2) long periods of juvenile dependence; (3) extensive biparental care including large transfers of information; (4) multigenerational bilateral kin networks; (5) habitual bipedal locomotion; (6) use of the upper limbs for tool use including projectile weapons; (7) concealed or "cryptic" ovulation; (8) menopause; (9) culture including language; and (10) lethal competition among kin-based coalitions. A few other species exhibit several of these traits; only humans, however, are characterized by the entire combination (Alexander, 2005). This suite of traits presents several questions or puzzles that are key to understanding human evolutionary biology. Here I first briefly describe these puzzles, and suggest a common resolution based on the importance of social competition during human evolution. I then return to the paradox of hormonal response to social challenge, hypothesizing that glucocorticoids and androgens help facilitate neural remodeling and long-term potentiation necessary for dynamic social cognition.

### Paternal care in multimale groups

Mammals that live in groups with multiple males – such as chimpanzees (*Pan troglodytes*) – usually have little or no paternal care, because the nonexclusivity of mating relationships obscures paternity (Alexander, 1974; Clutton-Brock, 1991). Chimpanzee males appear to lack reliable cues for identifying their offspring. In contrast, it is common for human fathers to provide protection, information, food, and social status for their children. Paternal care in humans appears to be facilitated by relatively stable pair bonds, which not only involves co-operation between mates that often endures over the life span, but which requires an unusual type of co-operation among coresiding males – respect for each other's mating relationships.

The relatively exclusive mating relationships that are characteristic of humans generate natural factions within the group. Mating relationships also can create alliances in human groups, linking two families or clans together. By way of comparison, in chimpanzee communities it is difficult for even the most dominant male to monopolize an estrous female; most of the males in a community mate with most of the females (Goodall, 1986). Although dominant males sire a higher proportion, chimpanzee males in effect "share" a common interest in the community's females and their offspring. Human groups, in contrast, are composed of family units, each with distinct reproductive interests. Human males do not typically share mating access to all the group's females; consequently, there are usually reliable cues identifying which children are their genetic offspring, and which are those of other males (for variations see Flinn, 1981; Beckerman and Valentine, 2002). Because humans live in multimale groups, yet typically maintain fairly stable mating relationships, the potential for fission along family lines is high. Still, human groups overcome this inherent conflict between family units to form large, stable coalitions (Chapais, 2008).

This unusual tolerance among coresidential males and females stands in contrast to the norm of polygamous mate competition in nonhuman primates. Selection pressures favoring such tolerance are uncertain, but likely involve the importance of both male parental investment (Alexander, 1990b; Geary and Bjorklund, 2000) and male coalitions for intraspecific conflict (Alexander, 1989, 2006; Wrangham, 1999; Geary and Flinn, 2001; Bernhard et al., 2006). The hormonal mechanisms that enable these unusual aspects of human male relationships are uncertain.

Analysis of patterns of levels of candidate hormones – such as vasopressin, testosterone, DHEA/S and cortisol – in natural social conditions may provide useful clues as to the evolved functions of male coalitions and pair bonding among humans.

## An extended period of juvenile dependence and child development

The human baby is unusually altricial (helpless). Infants must be carried, fed, and protected for a long period in comparison with other primates. Human childhood and adolescence are also lengthy (Smith, 1994; Bogin, 1999; Leigh, 2004). This extension of the juvenile period that delays reproduction for much longer than the other hominoids appears costly in evolutionary terms. Parental and other kin investment continues for an unusually long time, often well into adulthood and perhaps even after the death of the parents (Alexander, 1987; Coe, 2003; Hrdy, 2009).

The selective pressures responsible for this unique suite of life history characteristics appear central to understanding human evolution (Alexander, 1990a, 1990b; Kaplan et al., 2000; Bjorklund and Pellegrini, 2002; Rosenberg, 2004). The normal delay of reproduction until at least 15 years of age involves prolonged exposure to extrinsic causes of mortality and longer generation intervals. What advantages of an extended childhood could have outweighed the heavy costs of reduced fecundity and late reproduction (Williams, 1966; Stearns, 1990) for our hominin ancestors?

## Intelligence, information, and social power

The human brain is an astonishing organ. Its cortex comprises about 30 billion neurons of 200 different types, each of which are interlinked by about a thousand synapses, resulting in a million billion connections working at rates of up to 10 billion interactions per second (Williams and Herrup, 1988; Koch, 1999; Edelman, 2006). Quantifying the transduction of these biophysical actions into specific cognitive activities – e.g., thoughts and emotions – is difficult, but it is likely that humans have more information processing capacity than any other species (Roth and Dicke, 2005).

The human brain evolved at a rapid pace: hominin cranial capacity tripled (from an average of about 450 to 1350 cc) in less than 2 million years (Lee and Wolpoff, 2003) – roughly 100 000 neurons and supportive cells per generation. Structural changes such as increased convolutions, thickly myelinated cortical neurons, lateral asymmetries, increased von Economo neurons, expansion of the neo-cortex, and enhanced integration of the cerebellum also appear significant (Allman, 1999; Amodio and Frith, 2006). In comparison with most other parts of the human genome, selection on genes involved with brain development was especially intense (Gilbert et al., 2005).

The human brain has high metabolic costs: about 50% of an infant's, and 20% of an adult's, energetic resources are used to support this neurological activity (Aiello and Wheeler, 1995). Although the increase in energetic resources allocated to the brain was accompanied by a corresponding decrease in digestive tissue, this does not explain what the selective pressures were for enhanced information processing, or why the resources were not reallocated to direct reproductive function. The obstetric difficulties associated with birthing a large-headed infant generate additional problems (Rosenberg and Trevathan, 2002). The selective advantages of increased intelligence must have been high to overcome these costs.

The human brain, in short, is a big evolutionary puzzle. It is developmentally and metabolically expensive, evolved rapidly, enables uniquely human cognitive abilities such as language, empathy, foresight, consciousness, and ToM, and generates unusual levels of novelty. Advantages of a larger brain may include enhanced information processing capacities to contend with ecological pressures that involve sexually dimorphic activities such as hunting and complex foraging (Kaplan and Robson, 2002). There is little evidence, however, of sufficient domain-specific enlargement of those parts of the brain associated with selective pressures from the physical environment (Geary and Huffman, 2002; Adolphs, 2003). Indeed, human cognition has little to distinguish itself in the way of specialized ecological talents. A large brain may have been sexually selected because it was an attractive trait for mate choice (Miller, 2000; Gavrilets and Vose, 2006). However, there is little sexual dimorphism in encephalization quotient or intelligence psychometrics (Jensen, 1998), nor is there a clear reason why brains would have been a target for sexual selection driven by mate choice uniquely among hominins.

One area in which humans are truly extraordinary is sociality. Humans are able to mentally represent the feelings and thoughts of others. Humans have unusually well-developed mechanisms for ToM (Leslie et al., 2004; Amodio and Frith, 2006), and associated specific pathologies in this domain (Baron-Cohen, 1995; Gilbert, 2001). We have exceptional linguistic abilities for transferring information from one brain to another (Pinker, 1994), enabling complex social learning. Social and linguistic competencies are roughly equivalent in both males and females, although human mothers appear to have especially

important roles in the development of their offspring's sociocognitive development (Simons et al., 2001; Deater-Deckard et al., 2004).

Human coalitionary dynamics appear to have become increasingly based on information and social skills. Intense intergroup competition created pressure for within-group social cohesion (Alexander, 1990a; Flinn et al., 2005a) that required not only fighting abilities, but complex social strategies.

### Kin networks and multiple caretakers

All human societies recognize kinship as a key organizational principle (Brown, 1991). All languages have kinship terminologies and concomitant expectations of nepotism (Murdock, 1949; Fortes, 1969). Human kinship systems appear unique in the consistency of both bilateral (maternal and paternal) and multigenerational structure, with a general trend for coresidence of male kin. These aspects of human kinship link families into broader co-operative systems, and provide additional opportunities for alloparental care during the long social childhood. Human grandparents stand out as unusually important in this regard (Hrdy, 2005; Flinn and Leone, 2006, 2009).

Grandparents and grand-offspring share 25% of their genes identical by descent, a significant opportunity for kin selection. Few species, however, live in groups with multiple overlapping generations of kin. Fewer still have significant social relationships among individuals two or more generations apart. Humans appear rather exceptional in this regard. Grandparenting is cross-culturally ubiquitous and pervasive (Murdock, 1967; Sear et al., 2000). Our life histories allow for significant generational overlaps, including an apparent extended postreproductive stage facilitated by the unique human physiological adaptation of menopause (Alexander, 1974, 1987; Hawkes, 2003).

The significance of emotional bonding between grandparents and grandchildren is beyond doubt. The evolved functions are uncertain, but likely involve the exceptional importance of long-term extensive and intensive investment for the human child. The emotional and cognitive processes that guide grand-relationships must have evolved because they enhanced survival and eventual reproductive success of grandchildren. In addition to the physical basics of food, protection, and hygienic care, development of the human child is strongly influenced by the dynamics of the social environment (Konner, 1991; Hetherington, 2003a, 2003b; Dunn, 2004). Grandparents may have knowledge and experience that are important and useful for helping grandchildren and other relatives succeed in social competition (Coe, 2003). Humans are unusual in the role of kin in alloparental care and group coalitions (Hrdy, 2009).

## THE SOCIAL ENVIRONMENT AS A KEY SELECTIVE PRESSURE

### Information processing is a core human adaptation

Children are especially tuned to their social worlds and the information that it provides. The social world is a rich source of useful information for cognitive development. The human brain appears designed by natural selection to take advantage of this bonanza of data (Tooby and Cosmides, 1992; Bjorklund and Pellegrini, 2002; Belsky, 2005). "Culture" may be viewed as a highly dynamic information pool that coevolved with the extensive information processing abilities associated with our flexible communicative and sociocognitive competencies (Alexander, 1979). With the increasing importance and power of information in hominin social interaction, culture and tradition may have become an arena of social co-operation and competition (Coe, 2003; Flinn, 2004, 2006a; Baumeister, 2005).

The key issue is *novelty*. One of the most difficult challenges to understanding human cognitive evolution, and its handmaiden culture, is the unique informational arms race that underlies human behavior. The reaction norms posited by evolutionary psychology to guide evoked culture within specific domains may be necessary but insufficient (Chiappe and MacDonald, 2005). The mind does not appear limited to a predetermined Pleistocene set of options – such as choosing mate A if in environment X, but choose mate B if in environment Y – analogous to examples of simple phenotypic plasticity (MacDonald and Hershberger, 2005).

Keeping up in the hominin social chess game requires imitation. Getting ahead favors creativity to produce new solutions to beat the current winning strategies. Random changes, however, are risky and ineffective. Hence the importance of cognitive abilities to hone choices among imagined innovations in ever more complex social scenarios. The theater of the mind that allows humans to "understand other persons as intentional agents" (Tomasello, 1999, p. 526) provides the basis for the evaluation and refinement of creative solutions to the never-ending novelty of the social arms race. This process of filtering the riot of novel information generated by the creative mind favored the cognitive mechanisms for recursive pattern recognition in the "open" domains of both language (Pinker, 1994, 1997; Nowak et al., 2001) and social dynamics (Geary, 2005; Flinn, 1997, 2006a). Cultural "traditions" passed down through the generations also help constrain the creative mind (Coe, 2003; Flinn and Coe, 2007). The evolutionary basis for these psychological mechanisms underlying the importance of social learning and culture appears rooted in a process of "runaway social selection" (Alexander, 2005; Flinn and Alexander, 2007).

## Runaway social selection

Darwin (1871) recognized that there could be important differences between: (1) selection occurring as a consequence of interaction with ecological factors such as predators, climate, and food; and (2) selection occurring as a consequence of interactions among conspecifics, i.e., members of the same species competing with each other over resources such as nest sites, food, and mates. The former is termed "natural selection" and the latter "social selection" of which sexual selection may be considered a special subtype (West-Eberhard, 1983). The pace and directions of evolutionary changes in behavior and morphology produced by these two types of selection – natural and social – can be significantly different (Fisher, 1930; West-Eberhard, 2003).

Selection that occurs as a consequence of interactions between species can be intense and unending – for example with parasite-host red queen evolution (Hamilton et al., 1990) and other biotic arms races. Intraspecific social competition may generate selective pressures that cause even more rapid and dramatic evolutionary changes. Relative to natural selection, social selection has the following characteristics (West-Eberhard, 1983): (1) The intensity of social selection (and consequent genetic changes) can be very high because competition among conspecifics can have especially strong effects on differential reproduction. (2) Because the salient selective pressures involve competition among members of the same species, the normal ecological constraints are often relaxed for social selection. Hence traits can evolve in seemingly extreme and bizarre directions before counter-balancing natural selection slows the process. (3) Because social competition involves *relative* superiority among conspecifics, the bar can be constantly raised in a consistent direction generation after generation in an unending arms race. (4) Because social competition can involve multiple iterations of linked strategy and counter-strategy among interacting individuals, the process of social selection can become autocatalytic, its pace and directions partly determined from within, generating what might be termed "secondary red queens." For example, reoccurrence of social competition over lifetimes and generations can favor flexible phenotypic responses such as social learning that enable constantly changing strategies. Phenotypic flexibility of learned behavior to contend with a dynamic target may benefit from enhanced information processing capacities, especially in regard to foresight and scenario-building.

Human evolution appears characterized by these circumstances generating a process of runaway social selection (Alexander, 2005; Flinn and Alexander, 2007). Humans, more so than any other species, appear to have become their own most potent selective pressure via social competition involving coalitions (Alexander, 1989; Geary and Flinn, 2002), and dominance of their ecologies involving niche construction (Laland et al., 2000). The primary functions of the most extraordinary and distinctive human mental abilities – language, imagination, self-awareness, ToM, foresight, and consciousness – involve the negotiation of social relationships (Siegal and Varley, 2002; Tulving, 2002; Flinn et al., 2005a). The multiple-party reciprocity and shifting nested subcoalitions characteristic of human sociality generate especially difficult information processing demands for these cognitive facilities that underlie social competency. Hominin social competition involved increasing amounts of novel information and creative strategies. Culture emerged as an intensive selective pressure on the evolving brain.

## Evolution of the cultural brain

As noted above, the human brain is a big evolutionary paradox. It has high metabolic costs, takes a long time to develop, evolved rapidly, enables behavior to change quickly, has unique linguistic and social aptitudes, and generates unusual levels of informational novelty. Its primary functions include dealing with other human brains (Adolphs, 2003; Alexander, 2005; Amodio and Frith, 2006). The currency is not foot-speed or antibody production, but the generation and processing of data in the social worlds of the human brain's own collective and historical information pools. Some of the standout features of the human brain that distinguish us from our primate relatives are asymmetrically localized in the prefrontal cortex, including especially the dorsolateral prefrontal cortex and frontal pole (Ghazanfar and Santos, 2004; for review see Geary, 2005). These areas appear to be involved with "social scenario building" or the ability to "see ourselves as others see us so that we may cause competitive others to see us as we wish them to" (Alexander, 1990b, p. 7), and are linked to specific social abilities such as understanding sarcasm (Shamay-Tsoory et al., 2005) and morality (Moll et al., 2005). An extended childhood seems to enable the development of these necessary social skills (Joffe, 1997).

## Evolution of the human family as a nest for the child's social mind

To summarize, the human family is the nexus for the suite of extraordinary and unique human traits. Humans are the only species to live in large multimale groups with complex coalitions and extensive paternal and alloparental care, and the altricial infant is indicative of a protective environment provided by intense parenting

and alloparental care in the context of kin groups (Chisholm, 1999). The human baby does not need to be physically precocial, instead the brain continues rapid growth, and the corresponding cognitive competencies largely direct attention toward the social environment. Plastic neural systems adapt to the nuances of the local community, such as its language (Alexander, 1990a; Geary and Bjorklund, 2000; Bjorklund and Pellegrini, 2002; Fisher, 2005). In contrast to the slow development of ecological skills of movement, fighting, and feeding, the human infant rapidly acquires skill with the complex communication system of human language (Pinker, 1994; Sakai, 2005). The extraordinary information-transfer abilities enabled by linguistic competency provide a conduit to the knowledge available in other human minds. This emergent capability for intensive and extensive communication potentiates the social dynamics characteristic of human groups (Deacon, 1997; Dunbar, 1998) and provides a new mechanism for social learning and culture.

An extended childhood appears useful for acquiring the knowledge and practice to hone social skills and to build coalitional relationships necessary for successful negotiation of the increasingly intense social competition of adolescence and adulthood. Ecologically related play and activities (e.g., exploration of the physical environment) are also important (e.g., Geary et al., 2003), but appear similar to that of other primates. The unusual scheduling of human reproductive maturity, including an "adrenarche" (patterned increases in adrenal activities preceding puberty) and a delay in direct mate competition among males appears to extend the period of practicing social roles and extends social ontogeny (Campbell, 2006; Del Giudice 2009; Flinn et al., 2009).

The advantages of intensive parenting, including paternal protection and other care, require a most unusual pattern of mating relationships: moderately exclusive pair bonding in multiple-male groups. No other primate (or mammal) that lives in large, co-operative multiple-reproductive-male groups has extensive male parental care, although some protection by males is evident in baboons (Buchan et al., 2003). Competition for females in multiple-male groups usually results in low confidence of paternity (e.g., chimpanzees). Males forming exclusive "pair bonds" in multiple-male groups would provide cues of nonpaternity to other males, and hence place their offspring in great danger of infanticide (Hrdy, 1999). Paternal care is most likely to be favored by natural selection in conditions where males can identify their offspring with sufficient probability to offset the costs of investment, although reciprocity with mates is also likely to be involved (Smuts and Smuts, 1993; Geary and Flinn, 2001; Chapais, 2008). Humans exhibit a unique "nested family" social structure, involving complex reciprocity among males and females to restrict direct competition for mates among group members.

It is difficult to imagine how this system of pair bonds and male coalitions could be maintained in the absence of another unusual human trait: concealed or "cryptic" ovulation (Alexander and Noonan, 1979). Human groups tend to be male philopatric (males tending to remain in their natal groups), resulting in extensive male kin alliances, useful for competing against other groups of male kin (Wrangham and Peterson, 1996; LeBlanc, 2003). However, unlike chimpanzees, human groups and communities are often composed of several bilateral kin factions, interwoven by pair bond relationships among them. Human females also have complex alliances, but usually are not involved directly in the overt physical aggression characteristic of intergroup relations (Campbell, 2002; Geary and Flinn, 2002). Parents and other kin may be especially important for the child's mental development of social and cultural maps because they can be relied upon as landmarks who provide relatively honest information. From this perspective, the evolutionary significance of the human family in regard to child development is viewed more as a nest from which social skills may be acquired than just as an economic unit centered on the sexual division of labor (Flinn et al., 2005b).

To summarize my argument to this point, human childhood is viewed as a life history stage that appears necessary and useful for acquiring the information and practice to build and refine the mental algorithms critical for negotiating the social coalitions that are key to success in our species. Mastering the social environment presents special challenges for the human child. Social competence is difficult because the target is constantly changing and similarly equipped with ToM and other cognitive abilities. The family environment, including care from fathers and grandparents, is a primary source and mediator of the ontogeny of social competencies. Human biology has been profoundly affected by our evolutionary history as unusually social creatures, including, perhaps, a special reliance upon smart mothers, co-operative fathers, and helpful grandparents. Indeed, the mind of the human child may have design features that enable its development as a group project, guided by the multitudinous informational contributions of its ancestors and codescendants (Coe, 2003; Hrdy, 2009). Studies of the patterns of hormonal responses to these complex components of human sociality may provide important clues about the selective pressures that guided human evolution.

## NEUROENDOCRINE RESPONSE TO THE SOCIAL ENVIRONMENT

The constellation of behaviors associated with the human family and the dynamics of social competition described in previous sections are enabled by complex regulatory systems. In this section, I first briefly review the potential mechanisms for human pair bonding, maternal and paternal attachment to offspring, kin attachment, and male coalitions. Much of the research on the basic mechanisms has been done with nonhuman models and is not easily applied directly to some aspects of human psychology. I then turn to a more detailed analysis of how the neuroendocrine stress response system functions to enable acquisition of social competencies during childhood in the context of the human family environment.

The chemical messenger systems that orchestrate the ontogeny and regulation of sexual differentiation, metabolism, neurogenesis, immune function, growth, and other complex somatic processes, tend to be evolutionarily conservative among primates and more generally among mammals. Hence rodent and nonhuman primate models provide important comparative information about the functions of specific human neuroendocrine systems, for which we often have little direct empirical research. It is the particular balance of human mechanisms and abilities that is unique and reflects the history of selection for complex social interactions that shaped the human lineage.

### The chemistry of affection

Some of the most precious of all our human feelings are stimulated by close social relationships: a mother holding her newborn infant for the first time, brothers reunited after a long absence, or lovers entangled in each other's arms. Natural selection has designed our neurobiological mechanisms, in concert with our endocrine systems, to generate potent sensations in our interactions with these most evolutionarily significant individuals. We share with our primate relatives the same basic hormones and neurotransmitters that underlie these mental gifts. But our unique evolutionary history has modified us to respond to different circumstances and situations; we are rewarded and punished for somewhat different stimuli than our phylogenetic cousins. Chimpanzees and humans share the delight – the sensational reward – when biting into a ripe, juicy mango. But the endocrine, neurological, and associated emotional responses of a human father to the birth of his child (e.g., Storey et al., 2000) are likely to be quite different from those of a chimpanzee male. Happiness for a human (Buss, 2000) has many unique designs, such as romantic love (Fisher et al., 2002), that involve shared endogenous messengers from our phylogenetic heritage.

Attachments or bonding are central in the lives of the social mammals. Basic to survival and reproduction, these interdependent relationships are the fabric of the social networks that permit individuals to maintain co-operative relationships over time. Although attachments can provide security and relief from stress, close relationships also exert pressures on individuals to which they continuously respond. It should not be surprising, therefore, that the neuroendocrine mechanisms underlying attachment and stress are intimately related to one another. And although at the present time a good deal more is known about the stress response systems than the affiliative systems, some of the pieces of the puzzle are beginning to fall into place (Panksepp, 2004).

The mother–offspring relationship is at the core of mammalian life, and it appears that some of the biochemistry at play in the regulation of this intimate bond was also selected to serve in primary mechanisms regulating bonds between mates, paternal care, the family group, and even larger social networks (Hrdy, 1999; Fisher et al., 2002). Although a number of hormones and neurotransmitters are involved in attachment and other components of relationships, the two peptide hormones, oxytocin (OT) and arginine-vasopressin (AVP), appear to be primary (Carter, 2002; Young and Insel, 2002; Curtis and Wang, 2003; Lim et al., 2004; Heinrichs and Domes, 2008; Lee et al., 2009), with dopamine, cortisol, and other hormones and neurotransmitters having mediating effects.

The hypothalamus is the major brain site where OT and AVP (closely related chains of nine amino acids) are produced. From there they are released into the central nervous system (CNS) as well as transported to the pituitary where they are stored until secreted into the bloodstream. Oxytocin and AVP act on a wide range of neurological systems, and their influence varies among mammalian species and stage of development. The neurological effects of OT and AVP appear to be key mechanisms (e.g., Bartels and Zeki, 2004) involved in the evolution of human family behaviors. The effects of OT and AVP in humans are likely to be especially context dependent, because of the variable and complex nature of family relationships.

### Parental care

Along with OT and AVP, prolactin, estrogen, and progesterone are involved in parental care among mammals (Insel and Young, 2001). The roles of these hormones vary across species and between males and

females. The effects of these hormones are influenced by experience and context. Among rats, for example, estrogen and progesterone appear to prime the brain during pregnancy for parental behavior. Estrogen has been found to activate the expression of genes that increase the receptor density for OT and prolactin, thus increasing their postnatal influence (Young and Insel, 2002).

Oxytocin is most well known for its role in regulating birth and lactation, but along with AVP, it has also been found to play a central role in maternal care and attachment (Fleming et al., 1999). Just prior to birth, an increase in OT occurs, which is seen as priming maternal care. An injection of OT to virgin rats has been found to induce maternal care, while an OT antagonist administered to pregnant rats interferes with the development of maternal care (Carter, 2002).

The new rat mother requires hormonal activation to initially stimulate maternal behavior. Once she has begun to care for her pups, however, hormones are not required for maternal behavior to continue. Olfactory and somatosensory stimulation from interactions between pups and mother are, however, required for the parental care to continue (Fleming et al., 1999). The stimulation from suckling raises OT levels in rodents and breast-feeding women, which then results in not only milk letdown but also a decrease in limbic hypothalamic-anterior pituitary-adrenal cortex system (HPA) activity and a shift in the autonomic nervous system (ANS) from a sympathetic tone to a parasympathetic tone. This results in a calmness seen as conducive to remaining in contact with the infant. It also results in a shift from external-directed energy toward the internal activity of nutrient storage and growth (Uvnas-Moberg, 1998).

Experience also affects the neuroendocrine systems involved in the expression of maternal care. The HPA system of offspring during development is influenced by variation in maternal care, which then influences their maternal behavior as adults. Such changes involve the production of, and receptor density for, stress hormones and OT (Fleming et al., 1999; Champagne and Meaney, 2001).

The HPA-modulated hormones and maternal behavior are related in humans during the postpartum period (Fleming et al., 1997). During this time, cortisol appears to have an arousal effect, focusing attention on infant bonding. Mothers with higher cortisol levels were found to be more affectionate, more attracted to their infant's odor, and better at recognizing their infant's cry during the postpartum period.

Functional magnetic resonance imaging (fMRI) studies of brain activity involved in maternal attachment in humans indicate that the activated regions are part of the reward system and contain a high density of receptors for OT and AVP (Bartels and Zeki, 2004; Fisher et al., 2006). These studies also demonstrate that the neural regions involved in attachment activated in humans are similar to those activated in nonhuman animals. Among humans, however, neural regions associated with social judgment and assessment of the intentions and emotions of others exhibited some deactivation during attachment activities, suggesting possible links between psychological mechanisms for attachment and management of social relationships. Falling in love with a mate and affective bonds with offspring may involve temporary deactivation of psychological mechanisms for maintaining an individual's social "guard" in the complex reciprocity of human social networks. Dopamine levels are likely to be important for both types of relationship but may involve some distinct neural sites. It will be interesting to see what fMRI studies of attachment in human males indicate because that is where the most substantial differences from other mammals would be expected. Similarly, fMRI studies of attachment to mothers, fathers, and alloparental-care providers in human children may provide important insights into the other side of parent–offspring bonding.

## Paternal care

Paternal care is not common among mammals. For evolutionary reasons noted earlier, it is found among some rodent and primate species, including humans. The extent and types of paternal care vary among species. The hormonal influence in parental care among males appears to differ somewhat from that found among females. Vasopressin (AVP) appears to function as the male addition to OT (Young and Insel, 2002). Along with prolactin and OT, AVP prepares the male to be receptive to and care for infants (Bales et al., 2004).

Paternal care is more common in monogamous than polygamous mammals and is often related to hormonal and behavioral stimuli from the female. In the monogamous California mouse, disruption of the pair bond does not affect maternal care but does diminish paternal care (Gubernick, 1996). In other species with biparental care, however, paternal care is not as dependent on the presence of the female (Young and Insel, 2002). Experience also plays a role in influencing hormonal activation and paternal behavior. Among tamarins, experienced fathers have higher levels of prolactin than first-time fathers (Ziegler and Snowdon, 1997).

Androgens including testosterone also appear to be involved in the regulation of paternal behavior. For example, human fathers tend to have lower testosterone levels when they are involved in childcare activities (Berg and Wynne-Edwards, 2002; Fleming et al., 2002;

Gray and Campbell, 2009; also see Chapter 16 of this volume), although the relation with the key paternal role of offspring protection is uncertain. Human males stand out as very different from our closest relatives the chimpanzees in the areas of paternal attachment and investment in offspring. Investigation of the neuroendocrine mechanisms that underpin male parental behavior may provide important insights into these critical evolutionary changes.

## Pair bonding

Like male parental care, bonding between mates is also uncommon among mammals but has been selected for when it has reproductive advantages for both parents (Clutton-Brock, 1991; Carter, 2002; Young et al., 2002). Monogamy is found across many mammalian taxa, but most of the current knowledge related to the neuroendocrine basis of this phenomenon has been obtained from the comparative study of two closely related rodent species. The prairie vole (*Microtus ochrogaster*) mating pair nest together and provide prolonged biparental care, while their close relatives, the meadow vole (*Microtus pennsylvanicus*), do not exhibit these behaviors (Young et al., 2002). As with other social behaviors in rodents, OT and AVP have been found to be central in the differences these related species exhibit with respect to pair bonding.

Pair bonding occurs for the prairie vole following mating. Vagino-cervical stimulation results in a release of OT and the development of a partner preference for the female (Carter, 2002). For the male, it is an increase in AVP following mating and not just OT that results in partner preference. Exogenous OT injected in the female and exogenous AVP in the male prairie vole result in mate preference even without mating. This does not occur with meadow voles (Young et al., 2002).

The receptor density for OT and AVP in specific brain regions might provide the basis for mechanisms underlying other social behaviors. Other neurotransmitters, hormones, and social cues also are likely to be involved, but slight changes in gene expression for receptor density, such as those found between the meadow and prairie voles in the ventral palladium (located near the nucleus accumbens, an important component of the brain's reward system), might demonstrate how such mechanisms could be modified by selection (Lim et al., 2004). The dopamine D2 receptors in the nucleus accumbens appear to link the affiliative OT and AVP pair bonding mechanisms with positive rewarding mental states (Aragona et al., 2003; Curtis and Wang, 2003). The combination results in the powerful addiction that parents have for their offspring.

Given the adaptive value of extensive biparental care and prolonged attachment found in the mating pair and larger family network, it is not surprising that similar neurohormonal mechanisms active in the maternal–offspring bond would also be selected to underlie these other attachments. Though there is some variation among species and between males and females, the same general neurohormonal systems active in pair bonding in other species are found in the human (Wynne-Edwards, 2003; Panksepp, 2004; Lee et al., 2009). Androgen response to pair bonding appears complex (e.g. van der Meij et al., 2008), but similar to parent–offspring attachment in that pair bonded males tend to have lower testosterone levels in nonchallenging conditions (Alvergne et al., 2009; Gray and Campbell, 2009). Moreover, males actively involved in caretaking behavior appear to have temporarily diminished testosterone levels (Gray et al., 2007).

The challenge before human evolutionary biologists and psychologists is to understand how these general neuroendocrine systems have been modified and linked with other special human cognitive systems (e.g., Allman et al., 2001; Blakemore et al., 2004) to produce the unique suite of human family behaviors. Analysis of hormonal responses to social stimuli may provide important insights into the selective pressures that guided the evolution of these key aspects of the human mind.

## The chemistry of stress, family, and the social mind

The evolutionary scenario proposed in previous sections posits that the family is of paramount importance in a child's world. Throughout human evolutionary history, parents and close relatives provided calories, protection, and information necessary for survival, growth, health, social success, and eventual reproduction. The human mind, therefore, is likely to have evolved special sensitivity to interactions with family care providers, particularly during infancy and early childhood (Bowlby, 1969; Baumeister and Leary, 1995; Daly and Wilson, 1995; Belsky, 1997, 1999; Geary and Flinn, 2001; Flinn et al., 2009).

The family and other kin provide important cognitive "landmarks" for the development of a child's understanding of the social environment. The reproductive interests of a child overlap with those of its parents more than with any other individuals. Information (including advice, training, and incidental observation) provided by parents is important for situating oneself in the social milieu and developing a mental model of its operations. A child's family environment may be an especially important source and mediator of stress, with consequent effects on health.

Psychosocial stressors are associated with increased risk of infectious disease (Cohen et al., 2003) and a variety of other illnesses (Ader et al., 2001). Physiological stress responses regulate the

allocation of energetic and other somatic resources to different bodily functions via a complex assortment of neuroendocrine mechanisms. Changing, unpredictable environments require adjustment of priorities. Digestion, growth, immunity, and sex are irrelevant while being chased by a predator (Sapolsky, 1994). Stress hormones help shunt blood, glucose, and so on to tissues necessary for the task at hand. Chronic and traumatic stress can diminish health, evidently because resources are diverted away from important health functions. These costs can be referred to as "allostatic load" (McEwen, 1995). Such diversions of resources may have special significance during childhood because of the additional demands of physical and mental growth and development and possible long-term ontogenetic consequences.

## Stress response mechanisms and theory

Physiological response to environmental stimuli perceived as stressful is modulated by the limbic system (amygdala and hippocampus) and basal ganglia. These components of the CNS interact with the sympathetic and parasympathetic nervous systems and two neuroendocrine axes, the sympathetic-adrenal medullary system (SAM) and the HPA. The SAM and HPA systems affect a wide range of physiological functions in concert with other neuroendocrine mechanisms and involve complex feedback regulation. The SAM system controls the catecholamines norepinephrine and epinephrine (adrenalin). The HPA system regulates glucocorticoids, primarily cortisol (for reviews, see Weiner, 1992; McEwen, 1995; Sapolsky et al., 2000).

Cortisol is a key hormone produced in response to physical and psychosocial stressors (Mason, 1968; Selye, 1976). It is produced and stored in the adrenal cortex. Release into the plasma is primarily under the control of pituitary adrenocorticotropic hormone (ACTH). The free or unbound portion of the circulating cortisol may pass through the cell membrane and bind to a specific cytosolic glucocorticoid receptor. This complex may induce genes coding for at least 26 different enzymes involved with carbohydrate, fat, and amino acid metabolism in brain, liver, muscle, and adipose tissue (Yuwiler, 1982).

Cortisol modulates a wide range of somatic functions, including: (1) energy release (e.g., stimulation of hepatic gluconeogenesis in concert with glucagon and inhibition of the effects of insulin); (2) immune activity (e.g., regulation of inflammatory response and the cytokine cascade); (3) mental activity (e.g., alertness, memory, and learning); (4) growth (e.g., inhibition of growth hormone and somatomedins); and (5) reproductive function (e.g., inhibition of gonadal steroids, including testosterone). These complex multiple effects of cortisol muddle understanding of its adaptive functions. The demands of energy regulation must orchestrate with those of immune function, attachment bonding, and so forth. Mechanisms for localized targeting (e.g., glucose uptake by active versus inactive muscle tissues and neuropeptide-directed immune response) provide fine-tuning of the preceding general physiological effects. Cortisol regulation allows the body to respond to changing environmental conditions by preparing for *specific* short-term demands (Mason, 1971; Munck et al., 1984; Weiner, 1992).

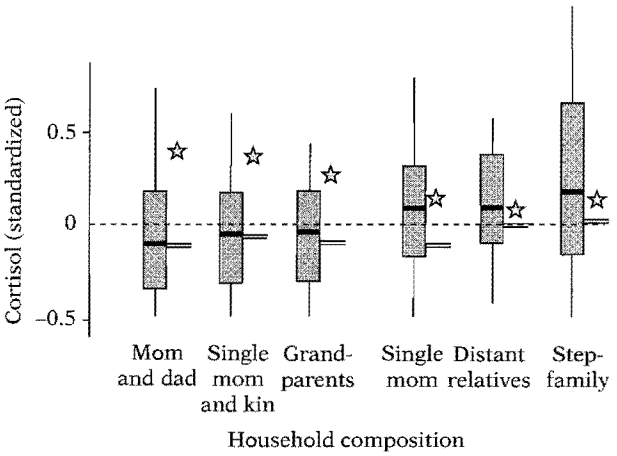
These temporary beneficial effects of glucocorticoid stress response, however, are not without costs. Persistent activation of the HPA system is associated with immune deficiency, cognitive impairment, inhibited growth, delayed sexual maturity, damage to the hippocampus, and psychological maladjustment (Glaser and Kiecolt-Glaser, 1994; Dunn, 1995; McEwen, 1995; Ader et al., 2001). Chronic stress may diminish metabolic energy (Ivanovici and Wiebe, 1981; Sapolsky, 1991) and produce complications from autoimmune protection (Munck and Guyre, 1991). Stressful life events – such as divorce, death of a family member, change of residence, or loss of a job – are associated with infectious disease and other health problems (Herbert and Cohen, 1993; Maier et al., 1994).

Current psychosocial stress research suggests that cortisol response is stimulated by uncertainty that is perceived as significant and for which behavioral responses will have unknown effects (Weiner, 1992; Kirschbaum and Hellhammer, 1994). That is, important events are going to happen; the child does not know how to react but is highly motivated to figure out what should be done. Cortisol release is associated with unpredictable, uncontrollable events that require full alert readiness and mental anticipation. In appropriate circumstances, temporary moderate increases in stress hormones (and associated neuropeptides) may enhance mental activity for short periods in localized areas, potentially improving cognitive processes for responding to social challenges (Beylin and Shors, 2003; Lupien, 2009). Other mental processes may be inhibited, perhaps to reduce external and internal "noise" (Servan-Schreiber et al., 1990; cf. Newcomer et al., 1994).

Relations between cortisol production and emotional distress, however, are difficult to assess because of temporal and interindividual variation in HPA response (Kagan, 1992; Nachmias et al., 1996). Habituation may occur to repeated events for which a child acquires an effective mental model. Attenuation and below-normal levels of cortisol may follow a day or more after emotionally charged events. Chronically stressed children may develop abnormal cortisol response, possibly via changes in binding globulin levels and/or reduced affinity or density of

glucocorticoid or corticotrophin-releasing hormone (CRH)/vasopressin receptors in the brain (Fuchs and Flugge, 1995). Early experience – such as perinatal stimulation of rats (Meaney et al., 1991), prenatal stress of rhesus macaques (Schneider et al., 1992; Clarke, 1993), and sexual abuse among humans (de Bellis et al., 1994) – may permanently alter HPA response. Personality may also affect HPA response (and vice versa) because children with inhibited temperaments tend to have higher cortisol levels than extroverted children (Kagan et al., 1988; cf. Gunnar et al., 1995; Hertsgaard et al., 1995; Nachmias et al., 1996).

Further complications arise from interaction between the HPA stress response and a wide variety of other neuroendocrine activities, including modulation of catecholamines, melatonin, testosterone, serotonin, β-endorphins, cytokines, and enkephalins (de Kloet, 1991; Sapolsky, 1992; Saphier et al., 1994). Changes in cortisol for energy allocation and modulation of immune function may be confused with effects of psychosocial stress. As reviewed in the previous section, OT and vasopressin intracerebral binding sites are associated with familial attachment in mammals and may influence distress involving caretaker–child relationships. Other components of the HPA axis such as CRH and melanocyte-stimulating hormone have effects that are distinct from cortisol.




**24.1.** Cortisol levels and household composition of children living in Bwa Mawego, Dominica. Box and whisker plots are for children's mean values of cortisol standardized for time since awakening (for descriptions of methods see Flinn, 2006b). Double lines represent average cortisol levels when an absence of stressful events were observed or reported. Stars indicate average cortisol levels during holidays (day before Christmas and August holiday weekends). Data include 21 673 salivary cortisol samples from 268 children collected from 1989 to 2001.

## Stress response and family environment

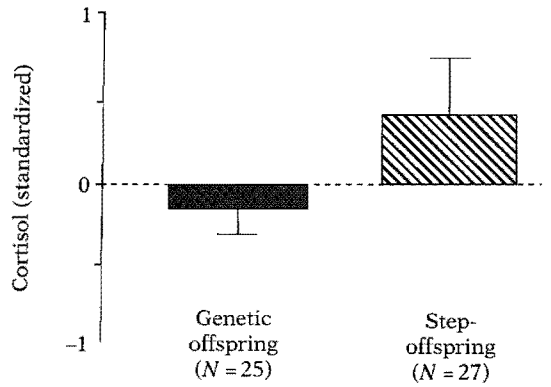
Composition of the family or caretaking household may have important effects on child development (Kagan, 1984; Whiting and Edwards, 1988). For example, in Western cultures, children with divorced parents may experience more emotional tension or "stress" than children living in a stable two-parent family (Wallerstein, 1983; Pearlin and Turner, 1987; Gottman and Katz, 1989).

Investigation of physiological stress responses in the human family environment has been hampered by the lack of noninvasive techniques for measurement of stress hormones. Frequent collection of plasma samples to assess temporal changes in endocrine function is not feasible in nonclinical settings. The development of saliva immunoassay techniques, however, presents new opportunities for stress research. Saliva is relatively easy to collect and store, especially under adverse field conditions faced by anthropologists (Ellison, 1988). In this section I review results from a longitudinal, 20-year study of child stress and health in a rural community on the island of Dominica (for reviews see Flinn and England, 1995, 1997, 2003; Flinn, 1999, 2006b). The research design uses concomitant monitoring of a child's daily activities, stress hormones, and psychological conditions to investigate the effects of naturally occurring psychosocial events in the family environment.

Associations between average cortisol levels of children and household composition indicate that children living with nonrelatives, stepfathers, and half-siblings (stepfather has children by the stepchild's mother), or single parents without kin support had higher average levels of cortisol than children living with both parents, single mothers with kin support, or grandparents (Figure 24.1). Note, however, that these differences in cortisol levels are diminished when comparisons are made during nonstressed conditions (double line bars in Figure 24.1). Moreover, the pattern is reversed during the excitement of holidays and other apparently hedonic emotional circumstances (stars in Figure 24.1). Hence cortisol appears to be elevated during "positive" as well as "negative" social challenges.

A further test of the hypothesis that difficult family environments are stressful is provided by comparison of step- and genetic children residing in the same households. Stepchildren had higher average cortisol levels than their half-siblings residing in the same household who were genetic offspring of both parents (Figure 24.2).

Several caveats need emphasis. Firstly, not all children in difficult family environments have elevated cortisol levels. Secondly, household composition is not a uniform indicator of family environment. Some single-mother households, for example, appear more stable, affectionate, and supportive than some two-parent households. Thirdly, children appear




**24.2.** Comparison of cortisol levels (standardized for time since awakening) of children (maternal half-siblings) living in the same household that were either genetic offspring or step-offspring of the resident adult male.

differentially sensitive to different aspects of their caretaking environments, reflecting temperamental and other individual differences.

These caveats, however, do not invalidate the general association between household composition and childhood stress. There are several possible reasons underlying this result. Children in difficult caretaking environments may experience chronic stress resulting in moderate-high levels of cortisol (i.e., a child has cortisol levels that are above average day after day). They may experience more acute stressors that substantially raise cortisol for short periods of time. They may experience more frequent stressful events (e.g., parental chastisement or marital quarreling – see Wilson et al., 1980; Flinn, 1988; Finkelhor and Dzuiba-Leatherman, 1994) that temporarily raise cortisol. There may be a lack of reconciliation between parent and child. And they may have inadequate coping abilities, perhaps resulting from difficult experiences in early development.

The events in children's lives that are associated with elevated cortisol are not always traumatic or even "negative." Activities such as eating meals, hard physical work, routine competitive play (e.g., cricket, basketball, and "king of the mountain" on ocean rocks), return of a family member who was temporarily absent (e.g., father returning from a job in town for the weekend), and holiday excitement (stars in Figure 24.1) were associated with temporary moderate increases (from about 10% to 100%) in cortisol among healthy children. These moderate stressors usually had rapid attenuation (<1 hour) of cortisol levels (some stressors had characteristic temporal "signatures" of cortisol level and duration).

High-stress events (cortisol increases from 100% to 2000%), however, most commonly involved trauma from family conflict or change (Flinn et al., 1996; Flinn and England, 2003). Punishment, quarreling, and residence change substantially increased cortisol levels, whereas calm, affectionate contact was associated with diminished (–10% to –50%) cortisol levels. Of all cortisol values that were more than two standard deviations (2 SD) above mean levels (i.e., indicative of substantial stress), 19.2% were temporally associated with traumatic family events (residence change of child or parent/caretaker, punishment, "shame," serious quarreling, and/or fighting) within a 24-hour period. In addition, 42.1% of traumatic family events were temporally associated with substantially elevated cortisol (i.e., at least one of the saliva samples collected within 24 hours was >2 SD above mean levels). Chronic elevations of cortisol levels sometimes occurred among children in difficult family environments, but this was difficult to assess quantitatively (Flinn, 2009).

There was considerable variability among children in cortisol response to family disturbances. Not all individuals had detectable changes in cortisol levels associated with family trauma. Some children had significantly elevated cortisol levels during some episodes of family trauma but not during others. Cortisol response is not a simple or uniform phenomenon. Numerous factors, including preceding events, habituation, specific individual histories, context, and temperament, might affect how children respond to particular situations.

Nonetheless, traumatic family events were associated with elevated cortisol levels for all ages of children more than any other factor that we examined. These results suggest that family interactions were a critical psychosocial stressor in most children's lives, although the sample collection during periods of intense family interaction (early morning and late afternoon) may have exaggerated this association.

Although elevated cortisol levels are associated with traumatic events such as family conflict, long-term stress may result in diminished cortisol response. In some cases, chronically stressed children had blunted response to physical activities that normally evoked cortisol elevation. Comparison of cortisol levels during "nonstressful" periods (no reported or observed crying, punishment, anxiety, residence change, family conflict, or health problem during the 24-hour period before saliva collection) indicates a striking reduction and, in some cases, reversal of the family environment–stress association (see double bars in Figure 24.1). Chronically stressed children sometimes had subnormal cortisol levels when they were not in stressful situations. For example, cortisol levels immediately after school (walking home from school) and during noncompetitive play were lower among some chronically stressed children (cf. Long et al., 1993). Some chronically stressed children appeared socially "tough" or withdrawn and exhibited little

or no arousal to the novelty of the first few days of the saliva collection procedure.

Relations between family environment and cortisol stress response appear to result from a combination of factors including frequency of traumatic events, frequency of positive "affectionate" interactions, frequency of negative interactions such as irrational punishment, frequency of residence change, security of "attachment," development of coping abilities, and availability or intensity of caretaking attention. Probably the most important correlate of household composition that affects childhood stress is maternal care. Mothers in socially "secure" households (i.e., permanent amiable coresidence with mate and/or other kin) appeared more able and more motivated to provide physical, social, and psychological care for their children. Mothers without mate or kin support were likely to exert effort attracting potential mates and may have viewed dependent children as impediments to this. Hence coresidence of father may provide not only direct benefits from paternal care but also may affect maternal care (Lamb et al., 1987; Belsky et al., 1991; Flinn, 1992; Hurtado and Hill, 1992). Young mothers without mate support usually relied extensively on their parents or other kin for help with childcare.

Children born and raised in household environments in which mothers have little or no mate or kin support were at greatest risk for abnormal cortisol profiles and associated health problems. Because socioeconomic conditions influence family environment, they have consequences for child health that extend beyond direct material effects. Also, because health in turn may affect an individual's social and economic opportunities, a cycle of poor health and poverty may be perpetuated generation after generation.

## CONCLUDING REMARKS

People in difficult or inequitable social environments tend to be less healthy in comparison with their more fortunate peers (e.g., Flinn, 1999; Hertzman, 1999; Dressler and Bindon, 2000; Wilkinson, 2001; Cohen et al., 2003). Social support can have reproductive consequences in group-living species (e.g., Silk et al., 2003; Cheney and Seyfarth, 2007). If the brain evolved as a social tool, then the expenditure of somatic resources (e.g., glucose) to resolve psychosocial problems makes sense. Relationships, especially family relationships, are of paramount importance. They are likely to have been a key factor affecting human reproductive success at least for over half a million years, and selection may have shaped our hormonal, neural, and psychological mechanisms to respond to this critical selective pressure. In Bwa Mawego, and perhaps in most human societies, children elevate their stress hormone (cortisol) levels more frequently and extensively in response to psychosocial stimuli than to challenges associated with the physical environment. The adaptive effects of the major stress hormones (Huether, 1996, 1998; Koolhaas et al., 2006; Fox et al., 2007) and affiliative neurotransmitters on neural reorganization appear consistent with observations of sensitivity to the social world (Flinn, 2006b).

Social competence is extraordinarily difficult because the competition is constantly changing and similarly equipped with ToM and other cognitive abilities. The sensitivity of the stress-response and affiliative systems to the social environment may enable adaptive neural reorganization to this most salient and dynamic puzzle. Childhood appears necessary and useful for acquiring the information and practice to build and refine the mental algorithms critical for negotiating the social coalitions that are the key to success in our species. The human family provides critical support for the child to develop sociocognitive skills. Traumatic early environments may result in diminished abilities to acquire social competencies as a consequence of glucocorticoid hypersensitivity disrupting neurogenesis, particularly in the hippocampus and other components of the limbic system (Mirescu et al., 2004; Weaver et al., 2004). An improved understanding of the hormonal and neurological mechanisms that facilitate the intensive and extensive relationships involved with human families and broader kin coalitions, including comparisons between humans and our close primate relatives, may provide important insights into the selective pressures that shaped key features of human biology.

## DISCUSSION POINTS

1. What happened to our hominin ancestors? Why are we the only species left? If humans suddenly went extinct, would another life form eventually evolve high intelligence? How?
2. Do you think chimpanzee fathers love their offspring? Why or why not? And chimpanzee grandparents?
3. Do school exams make you sick? Collect data from your classmates to test your hypotheses.
4. What events cause you to become "stressed?" Why do you think natural selection produced psychological mechanisms that result in this sensitivity?
5. How are the events that cause stress for you similar and/or different from the events that were stressful for your parents, your grandparents, and your distant hominin ancestors?

## REFERENCES


Ader, R., Felten, D. L. and Cohen, N. (2001). *Psychoneuroimmunology*, 3rd edn. San Diego, CA: Academic Press.

Adolphs, R. (2003). Cognitive neuroscience of human social behavior. *Nature Reviews, Neuroscience*, **4**(3), 165–178.

Aiello, L. C. and Wheeler, P. (1995). The expensive-tissue hypothesis: the brain and the digestive system in human and primate evolution. *Current Anthropology*, **36**, 199–221.

Alexander, R. D. (1974). The evolution of social behavior. *Annual Review of Ecology and Systematics*, **5**, 352–383.

Alexander, R. D. (1979). *Darwinism and Human Affairs*. Seattle: University of Washington Press.

Alexander, R. D. (1987). *The Biology of Moral Systems*. Hawthorne, NY: Aldine de Gruyter.

Alexander, R. D. (1989). Evolution of the human psyche. In *The Human Revolution*, P. Mellars and C. Stringer (eds). Chicago: University of Chicago Press, pp. 455–513.

Alexander, R. D. (1990a). Epigenetic rules and Darwinian algorithms: the adaptive study of learning and development. *Ethology and Sociobiology*, **11**, 1–63.

Alexander, R. D. (1990b). *How Humans Evolved: Reflections on the Uniquely Unique Species. Museum of Zoology (Special Publication No. 1)*. Ann Arbor, MI: The University of Michigan.

Alexander, R. D. (2005). Evolutionary selection and the nature of humanity. In *Darwinism and Philosophy*, V. Hosle and C. Illies (eds). South Bend, IN: University of Notre Dame Press, pp. 301–348.

Alexander, R. D. (2006). The challenge of human social behavior. *Evolutionary Psychology*, **4**, 1–32.

Alexander, R. D. and Noonan, K. M. (1979). Concealment of ovulation, parental care, and human social evolution. In *Evolutionary Biology and Human Social Behavior: an Anthropological Perspective*, N. A. Chagnon and W. Irons (eds). North Scituate, MA: Duxbury Press, pp. 436–453.

Allman, J. (1999). *Evolving Brains*. New York: Scientific American Library.

Allman, J., Hakeem, A., Erwin, J. M., et al. (2001). The anterior cingulate cortex: the evolution of an interface between emotion and cognition. *Annals of the New York Academy of Sciences*, **935**, 107–117.

Alvergne, A., Faurie, C. and Raymond, M. (2009). Variation in testosterone levels and male reproductive effort: insight from a polygynous human population. *Hormones and Behavior*, **56**(5), 491–497.

Amodio, D. M. and Frith, C. D. (2006). Meeting of minds: the medial frontal cortex and social cognition. *Nature Reviews Neuroscience*, **7**(4), 268–277.

Aragona, B. J., Liu, Y., Curtis, J. T., et al. (2003). A critical role for nucleus accumbens dopamine in partner-preference formation in male prairie voles. *Journal of Neuroscience*, **23**(8), 3483–3490.

Bales, K. L., Kim, A. J., Lewis-Reese, A. D., et al. (2004). Both oxytocin and vasopressin may influence alloparental behavior in male prairie voles. *Hormones and Behavior*. **45**(5), 354–361.

Baron-Cohen, S. (1995). *Mindblindness: an Essay on Autism and Theory of Mind*. Boston, MA: MIT/Bradford.

Bartels, A. and Zeki, S. (2004). The neural correlates of maternal and romantic love. *NeuroImage*, **21**, 1155–1166.

Baumeister, R. F. (2005). *The Cultural Animal: Human Nature, Meaning, and Social Life*. New York: Oxford University Press.

Baumeister, R. F. and Leary, M. R. (1995). The need to belong: desire for interpersonal attachment as a fundamental human motive. *Psychological Bulletin*, **117**, 497–529.

Beckerman, S. and Valentine, P. (eds) (2002). *Cultures of Multiple Fathers: the Theory and Practice of Partible Paternity in South America*. Gainesville, FL: University of Florida Press.

Belsky, J. (1997). Attachment, mating, and parenting: an evolutionary interpretation. *Human Nature*, **8**, 361–381.

Belsky, J. (1999). Modern evolutionary theory and patterns of attachment. In *Handbook of Attachment: Theory, Research, and Clinical Applications*. J. Cassidy and P. R. Shaver (eds). New York: Guilford Press, pp. 141–161.

Belsky, J. (2005). Differential susceptibility to rearing influence: an evolutionary hypothesis and some evidence. In *Origins of the Social Mind: Evolutionary Psychology and Child Development*, B. J. Ellis and D. F. Bjorklund (eds). New York: Guilford Press, pp. 139–163.

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

Berg, S. J. and Wynne-Edwards, K. E. (2002). Salivary hormone concentrations in mothers and fathers becoming parents are not correlated. *Hormones and Behavior*, **42**, 424–436.

Bernhard, H., Fischbacher, U. and Fehr, E. (2006). Parochial altruism in humans. *Nature*, **442**(7105), 912–915.

Beylin, A. V. and Shors, T. J. (2003). Glucocorticoids are necessary for enhancing the acquisition of associative memories after acute stressful experience. *Hormones and Behavior*, **43**, 124–131.

Bjorklund, D. F. and Pellegrini, A. D. (2002). *The Origins of Human Nature: Evolutionary Developmental Psychology*. Washington, DC: APA Press.

Blakemore, S.-J., Winston, J. and Frith, U. (2004). Social cognitive neuroscience: where are we heading? *Trends in Cognitive Neurosciences*, **8**(5), 216–222.

Bogin, B. (1999). *Patterns of Human Growth*, 2nd edn. Cambridge: Cambridge University Press.

Bowlby, J. (1969). *Attachment and Loss:* vol. 1. *Attachment*. London: Hogarth.

Brown, D. E. (1991). *Human Universals*. Philadelphia: Temple University Press.

Buchan, J. C., Alberts, S. C., Silk, J. B., et al. (2003). True paternal care in a multi-male primate society. *Nature*, **425**(6954), 179–181.

Bugental, D. B. (2000). Acquisition of the algorithms of social life: a domain-based approach. *Psychological Bulletin*, **26**, 187–209.

Buss, D. M. (2000). The evolution of happiness. *American Psychologist*, **55**, 15–23.

Campbell, A. (2002). *A Mind of Her Own: the Evolutionary Psychology of Women*. Oxford: Oxford University Press.

Campbell, B.C. (2006). Adrenarche and the evolution of human life history. *American Journal of Human Biology* **18**, 569–589.

Carter, C.S. (2002). Neuroendocrine perspectives on social attachment and love. In *Foundations in Social Neuroscience*, J.T. Cacioppo, G.G. Berntson, R. Adolphs, et al. (eds). Cambridge, MA: MIT Press, pp. 853–890.

Champagne, F.A. (2008). Epigenetic mechanisms and the transgenerational effects of maternal care. *Frontiers in Neuroendocrinology*, **29**, 386–397.

Champagne, F.A. and Meaney, M.J. (2001). Like mother, like daughter: evidence for non-genomic transmission of parental behavior and stress responsivity. *Progress in Brain Research*, **133**, 287–302.

Chapais, B. (2008). *Primeval Kinship: How Pair-bonding Gave Birth to Human Society*. Cambridge, MA: Harvard University Press.

Cheney, D.L. and Seyfarth, R.M. (2007). *Baboon Metaphysics: the Evolution of a Social Mind*. Chicago: University of Chicago Press.

Chiappe, D. and MacDonald, K. (2005). The evolution of domain-general mechanisms in intelligence and learning. *Journal of General Psychology*, **132**(1), 5–40.

Chisholm, J.S. (1999). *Death, Hope, and Sex*. Cambridge: Cambridge University Press.

Clarke, A.S. (1993). Social rearing effects on HPA axis activity over early development and in response to stress in rhesus monkeys. *Developmental Psychobiology*, **26**(8), 433–446.

Clutton-Brock, T.H. (1991). *The Evolution of Parental Care*. Princeton, NJ: Princeton University Press.

Coe, K. (2003). *The Ancestress Hypothesis: Visual Art as Adaptation*. New Brunswick, NJ: Rutgers University Press.

Cohen, S., Doyle, W.J., Turner, R.B., et al. (2003). Emotional style and susceptibility to the common cold. *Psychosomatic Medicine*, **65**(4), 652–657.

Cohen, S., Kessler, R.C. and Underwood, G.L. (1993). Negative life events, perceived stress, negative effect, and susceptibility to the common cold. *Journal of Personality and Social Psychology*, **64**, 131–140.

Cohen, S., Janicki-Deverts, D., Miller, G.E. (2007). Psychological stress and disease. *Journal of the American Medical Association*, **298**(14), 1685–1687.

Curtis, T.J. and Wang, Z. (2003). The neurochemistry of pair bonding. *Current Directions in Psychological Science*, **12**(2), 49–53.

Daly, M. and Wilson, M. (1995). Discriminative parental solicitude and the relevance of evolutionary models to the analysis of motivational systems. In *The Cognitive Neurosciences*, M.S. Gazzaniga (ed.). Cambridge, MA: MIT Press, pp. 1269–1286.

Darwin, C.R. (1871). *The Descent of Man and Selection in Relation to Sex*. London: John Murray.

de Bellis, M., Chrousos, G.P., Dorn, L.D., et al. (1994). Hypothalamic-pituitary-adrenal axis dysregulation in sexually abused girls. *Journal of Clinical Endocrinology and Metabolism*, **78**, 249–255.

de Kloet, E.R. (1991). Brain corticosteroid receptor balance and homeostatic control. *Frontiers in Neuroendocrinology*, **12**(2), 95–164.

Deacon, T.W. (1997). *The Symbolic Species: the Co-Evolution of Language and the Brain*. New York: Norton.

Deater-Deckard, K., Atzaba-Poria, N. and Pike, A. (2004). Mother- and father–child mutuality in Anglo and Indian British families: a link with lower externalizing behaviors. *Journal of Abnormal Child Psychology*, **32**(6), 609–620.

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

Dressler, W. and Bindon, J.R. (2000). The health consequences of cultural consonance: cultural dimensions of lifestyle, social support, and arterial blood pressure in an African American community. *American Anthropologist*, **102**(2), 244–260.

Dunbar, R.I.M. (1998). The social brain hypothesis. *Evolutionary Anthropology*, **6**, 178–190.

Dunn, A.J. (1995). Interactions between the nervous system and the immune system: implications for psychopharmacology. In *Psychopharmacology: the Fourth Generation of Progress*, F.R. Bloom and D.J. Kupfer (eds). New York: Raven Press.

Dunn, J. (2004). Understanding children's family worlds: family transitions and children's outcome. *Merrill-Palmer Quarterly*, **50**(3), 224–235.

Edelman, G.M. (2006). *Second Nature: Brain Science and Human Knowledge*. New Haven: Yale University Press.

Ellison, P. (1988). Human salivary steroids: methodological considerations and applications in physical anthropology. *Yearbook of Physical Anthropology*, **31**, 115–142.

Ellison, P.T. (2001). *On Fertile Ground, a Natural History of Human Reproduction*. Cambridge, MA: Harvard University Press.

Finkelhor, D. and Dzuiba-Leatherman, J. (1994). Victimization of children. *American Psychologist*, **49**(3), 173–183.

Fisher, H.E., Aron, A., Mashek, D., et al. (2002). Defining the brain systems of lust, romantic attraction and attachment. *Archives of Sexual Behavior*, **31**(5), 413–419.

Fisher, H.E., Aron, A. and Brown, L.L. (2006). Romantic love: a mammalian brain system for mate choice. *Philosophical Transactions of the Royal Society of London. Series B*, **361**, 2173–2186.

Fisher, R.A. (1930). *The Genetical Theory of Natural Selection*. Oxford: Clarendon Press.

Fisher, S.E. (2005). On genes, speech, and language. *New England Journal of Medicine*, **353**, 1655–1657.

Fleming, A.S., Steiner, M. and Corter, C. (1997). Cortisol, hedonics, and maternal responsiveness in human mothers. *Hormones and Behavior*, **32**(2), 85–98.

Fleming, A.S., O'Day, D.H. and Kraemer, G.W. (1999). Neurobiology of mother–infant interactions: experience and central nervous system plasticity across development and generations. *Neuroscience and Biobehavioral Reviews*, **23**, 673–685.

Fleming, A.S., Corter, C., Stallings, J., et al. (2002). Testosterone and prolactin are associated with emotional

responses to infant cries in new fathers. *Hormones and Behavior*, **42**, 399–413.

Flinn, M. V. (1981). Uterine and agnatic kinship variability. In *Natural Selection and Social Behavior: Recent Research and New Theory*, R. D. Alexander and D. W. Tinkle (eds). New York: Blackwell Press, pp. 439–475.

Flinn, M. V. (1988). Step and genetic parent/offspring relationships in a Caribbean village. *Ethology and Sociobiology*, **9**(3), 1–34.

Flinn, M. V. (1992). Paternal care in a Caribbean village. In *Father–Child Relations: Cultural and Biosocial Contexts*, B. Hewlett (ed.). Hawthorne, NY: Aldine de Gruyter, pp. 57–84.

Flinn, M. V. (1997). Culture and the evolution of social learning. *Evolution and Human Behavior*, **18**(1), 23–67.

Flinn, M. V. (1999). Family environment, stress, and health during childhood. In *Hormones, Health, and Behavior*, C. Panter-Brick and C. Worthman (eds). Cambridge: Cambridge University Press, pp. 105–138.

Flinn, M. V. (2004). Culture and developmental plasticity: evolution of the social brain. In *Evolutionary Perspectives on Child Development*, K. MacDonald and R. L. Burgess (eds). Thousand Oaks, CA: Sage, pp. 73–98.

Flinn, M. V. (2006a). Cross-cultural universals and variations: the evolutionary paradox of informational novelty. *Psychological Inquiry*, **17**, 118–123.

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

Flinn, M. V. (2009). Are cortisol profiles a stable trait during child development? *American Journal of Human Biology*, **21**(6), 769–771.

Flinn, M. V. and Alexander, R. D. (2007). Runaway social selection. In *The Evolution of Mind*, S. W. Gangestad and J. A. Simpson (eds). New York: Guilford Press, pp. 249–255.

Flinn, M. V. and Coe, K. C. (2007). The linked red queens of human cognition, coalitions, and culture. In *The Evolution of Mind*, S. W. Gangestad and J. A. Simpson (eds). New York: Guilford Press, pp. 339–347.

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

Flinn, M. V. and England, B. G. (1997). Social economics of childhood glucocorticoid stress response and health. *American Journal of Physical Anthropology*, **102**, 33–53.

Flinn, M. V. and England, B. G. (2003). Childhood stress: endocrine and immune responses to psychosocial events. In *Social and Cultural Lives of Immune Systems*, J. M. Wilce (ed.). London: Routledge Press, pp. 107–147.

Flinn, M. V. and Leone, D. V. (2006). Early trauma and the ontogeny of glucocorticoid stress response: grandmother as a secure base. *Journal of Developmental Processes*, **1**(1), 31–68.

Flinn, M. V. and Leone, D. V. (2009). Alloparental care and the ontogeny of glucocorticoid stress response among stepchildren. In *Substitute Parents*, G. Bentley and R. Mace (eds). Oxford: Berghahn Books, pp. 266–286.

Flinn, M. V., Quinlan, R., Turner, M. T., et al. (1996). Male–female differences in effects of parental absence on glucocorticoid stress response. *Human Nature*, **7**(2), 125–162.

Flinn, M. V., Geary, D. C. and Ward, C. V. (2005a). Ecological dominance, social competition, and coalitionary arms races: why humans evolved extraordinary intelligence. *Evolution and Human Behavior*, **26**(1), 10–46.

Flinn, M. V., Ward, C. V. and Noone, R. (2005b). Hormones and the human family. In *Handbook of Evolutionary Psychology*, D. Buss (ed.). New York: Wiley, pp. 552–580.

Flinn, M. V., Muehlenbein, M. P. and Ponzi, D. (2009). Evolution of neuroendocrine mechanisms linking attachment and life history: the social neuroendocrinology of middle childhood. *Behavioral and Brain Sciences*, **32**(1), 27–28.

Fortes, M. (1969). *Kinship and the Social Order*. Chicago, IL: Aldine de Gruyter.

Fox, N. A., Hane, A. A. and Pine, D. S. (2007). Plasticity for affective neurocircuitry: how the environment affects gene expression. *Current Directions in Psychological Science*, **16**, 1–5.

Fuchs, E. and Flugge, G. (1995). Modulation of binding sites for corticotropin-releasing hormone by chronic psychosocial stress. *Psychoneuroendocrinology*, **30**(1), 33–51.

Gavrilets, S. and Vose, A. (2006). The dynamics of Machiavellian intelligence. *Proceedings of the National Academy of Sciences of the United States of America*, **103**(45), 16823–16828.

Geary, D. C. (2005). *The Origin of Mind: Evolution of Brain, Cognition, and General Intelligence*. Washington, DC: American Psychological Association.

Geary, D. C. and Bjorklund, D. F. (2000). Evolutionary developmental psychology. *Child Development*, **71**(1), 57–65.

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

Geary, D. C. and Flinn, M. V. (2002). Sex differences in behavioral and hormonal response to social threat. *Psychological Review*, **109**(4), 745–750.

Geary, D. C. and Huffman, K. J. (2002). Brain and cognitive evolution: forms of modularity and functions of mind. *Psychological Bulletin*, **128**(5), 667–698.

Geary, D. C., Byrd-Craven, J., Hoard, M. K., et al. (2003). Evolution and development of boys' social behavior. *Developmental Review*, **23**, 444–470.

Ghazanfar, A. A. and Santos, L. R. (2004). Primate brains in the wild: the sensory bases for social interactions. *Nature Reviews Neuroscience*, **5**(8), 603–616.

Gilbert, P. (2001). Evolutionary approaches to psychopathology: the role of natural defences. *Australian and New Zealand Journal of Psychiatry*, **35**(1), 17–27.

Gilbert, S. L., Dobyns, W. B. and Lahn, B. T. (2005). Genetic links between brain development and brain evolution. *Nature Reviews Genetics*, **6**(7), 581–590.

Glaser, R. and Kiecolt-Glaser, J. K. (eds) (1994). *Handbook of Human Stress and Immunity*. New York: Academic Press.

Goodall, J. (1986). *The Chimpanzees of Gombe: Patterns of Behavior*. Cambridge, MA: Belknap Press of Harvard University Press.

Gopnik, A., Meltzoff, A.N. and Kuhl, P.K. (1999). *The Scientist in the Crib: Minds, Brains, and How Children Learn*. New York: William Morrow and Co.

Gottman, J.M. and Katz, L.F. (1989). Effects of marital discord on young children's peer interaction and health. *Developmental Psychology*, **25**(3), 373–381.

Gray, P.B. and Campbell, B.C. (2009). Human male testosterone, pair bonding and fatherhood. In *Endocrinology of Social Relationships*, P.B. Gray and P.T. Ellison (eds). Cambridge: Harvard University Press.

Gray, P.B., Parkin, J.C. and Samms-Vaughan, M.E. (2007). Hormonal correlates of human paternal interactions: a hospital-based investigation in urban Jamaica. *Hormones and Behavior*, **52**, 499–507.

Gubernick, D. (1996). A natural family system. *Family Systems*, **3**, 109–124.

Gunnar, M., Porter, F.L., Wolf, C.M., et al. (1995). Neonatal stress reactivity: predictions to later emotional temperament. *Child Development*, **66**, 1–13.

Hamilton, W.D., Axelrod, R. and Tanese, R. (1990). Sexual reproduction as an adaptation to resist parasites (a review). *Proceedings of the National Academy of Sciences of the United States of America*, **87**, 3566–3573.

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

Heim, C., Newport, D., Wagner, D., et al. (2002). The role of early adverse experience and adulthood stress in the prediction of neuroendocrine stress reactivity in women: a multiple regression analysis. *Depression and Anxiety*, **15**, 117–125.

Heim, C. and Nemeroff, C. (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies. *Society of Biological Psychiatry*, **49**, 1023–1039.

Heinrichs, M. and Domes, G. (2008). Neuropeptides and social behaviour: effects of oxytocin and vasopressin in humans. *Progress in Brain Research*, **170**, 337–350.

Henry, J.P. and Wang, S. (1998). Effect of early stress on adult affiliative behavior. *Psychoneuroendocrinology*, **23** (8), 863–875.

Herbert, T.B. and Cohen, S. (1993). Stress and immunity in humans: a meta-analytic review. *Psychosomatic Medicine*, **55**, 364–379.

Hertsgaard, L., Gunnar, M., Erickson, M.F., et al. (1995). Adrenocortical responses to the strange situation in infants with disorganized/disoriented attachment relationships. *Child Development*, **66**, 1100–1106.

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. *Annals of the New York Academy of Sciences*, **896**, 85–95.

Hetherington, E.M. (2003a). Intimate pathways: changing patterns in close personal relationships across time. *Family Relations: Interdisciplinary Journal of Applied Family Studies*, **52**(4), 318–331.

Hetherington, E.M. (2003b). Social support and the adjustment of children in divorced and remarried families. *Childhood: a Global Journal of Child Research*, **10**(2), 217–236.

Hrdy, S.B. (1999). *Mother Nature: a History of Mothers, Infants, and Natural Selection*. New York: Pantheon.

Hrdy, S.B. (2005). Evolutionary context of human development: the cooperative breeding model. In *Attachment and Bonding: a New Synthesis*, C.S. Carter and L. Ahnert (eds). Cambridge, MA: MIT Press, pp. 9–32.

Hrdy, S.B. (2009). *Mothers and Others: The Evolutionary Origins of Mutual Understanding*. Cambridge, MA: Harvard University Press.

Huether, G. (1996). The central adaptation syndrome: psychosocial stress as a trigger for adaptive modifications of brain structure and brain function. *Progress in Neurobiology*, **48**, 568–612.

Huether, G. (1998). Stress and the adaptive self organization of neuronal connectivity during early childhood. *International Journal of Developmental Neuroscience*, **16**(3/4), 297–306.

Hurtado, A.M. and Hill, K.R. (1992). Paternal effect on offspring survivorship among Ache and Hiwi hunter-gatherers: implications for modeling pair-bond stability. In *Father–Child Relations: Cultural and Biosocial Contexts*, B. Hewlett (ed.). Hawthorne, NY: Aldine de Gruyter, pp. 31–55.

Insel, T.R. and Young, L.R. (2001). The neurobiology of attachment. *Nature Reviews Neuroscience*, **2**, 129–136.

Ivanovici, A.M. and Wiebe, W.J. (1981). Towards a working "definition" of "stress": a review and critique. In *Stress Effects on Natural Ecosystems*, G.W. Barrett and R. Rosenberg (eds). New York: Wiley, pp. 13–17.

Jensen, A.R. (1998). *The G Factor: the Science of Mental Ability*. New York: Praeger.

Joffe, T.H. (1997). Social pressures have selected for an extended juvenile period in primates. *Journal of Human Evolution*, **32**, 593–605.

Kagan, J. (1984). *The Nature of the Child*. New York: Basic Books.

Kagan, J. (1992). Behavior, biology, and the meanings of temperamental constructs. *Pediatrics*, **90**, 510–513.

Kagan, J., Resnick, J.S. and Snidman, N. (1988). The biological basis of childhood shyness. *Science*, **240**, 167–171.

Kaplan, H.S. and Robson, A.J. (2002). The emergence of humans: the coevolution of intelligence and longevity with intergenerational transfers. *Proceedings of the National Academy of Sciences of the United States of America*, **99**(15), 10221–10226.

Kaplan, H., Hill, K., Lancaster, J., et al. (2000). A theory of human life history evolution: diet, intelligence and longevity. *Evolutionary Anthropology*, **9**, 156–183.

Kirschbaum, C. and Hellhammer, D.H. (1994). Salivary cortisol in psychneuroendocrine research: recent developments and applications. *Psychoneuroendocrinology*, **19**, 313–333.

Koch, C. (1999). *Biophysics of Computation. Information Processing in Single Neurons*. New York: Oxford University Press.

Kolassa, I.-T. and Elbert, T. (2007). Structural and functional neuroplasticity in relation to traumatic stress. *Current Directions in Psychological Science*, **16**, 321–325.

Konner, M. (1991). *Childhood*. Boston, MA: Little, Brown and Co.

Koolhaas, J. M., de Boer, S. F. and Buwalda, B. (2006). Stress and adaptation: toward ecologically relevant animal models. *Current Directions in Psychological Science*, **15**, 109–112.

Korte, S. M., Koolhaas, J. M., Wingfield, J. C., et al. (2005). The Darwinian concept of stress: benefits of allostasis and costs of allostatic load and the trade-offs in health and disease. *Neuroscience and Biobehavioral Reviews*, **29**(1), 3–38.

Laland, K. N., Odling-Smee, J. and Feldman, M. W. (2000). Niche construction, biological evolution, and cultural change. *Behavioral and Brain Sciences*, **23**, 131–175.

Lamb, M., Pleck, J., Charnov, E., et al. (1987). A biosocial perspective on paternal behavior and involvement. In *Parenting Across the Lifespan: Biosocial Dimensions*, J. B. Lancaster, J. Altmann, A. Rossi, et al. (eds). Hawthorne, NY: Aldine de Gruyter, pp. 111–142.

LeBlanc, S. A. (2003). *Constant Battles: the Myth of the Peaceful, Noble Savage*. New York: St. Martin's Press.

Lee, H.-J., Macbeth, A. H., Pagani, J. H., et al. (2009). Oxytocin: the great facilitator of life. *Progress in Neurobiology*, **88**(2), 127–151.

Lee, S. H. and Wolpoff, M. H. (2003). The pattern of evolution in Pleistocene human brain size. *Paleobiology*, **29**, 186–196.

Leigh, S. R. (2004). Brain growth, cognition, and life history in primate and human evolution. *American Journal of Primatology*, **62**, 139–164.

Leslie, A. M., Friedmann, O. and German, T. P. (2004). Core mechanisms in "theory of mind." *Trends in Cognitive Sciences*, **8**(12), 529–533.

Lim, M. M., Wang, Z., Olazabal, D. E., et al. (2004). Enhanced partner preference in a promiscuous species by manipulating the expression of a single gene. *Nature*, **429**, 754–757.

Long, B., Ungpakorn, G. and Harrison, G. A. (1993). Home-school differences in stress hormone levels in a group of Oxford primary school children. *Journal of Biosocial Sciences*, **25**, 73–78.

Lupien, S. J. (2009). Brains under stress. *Canadian Journal of Psychiatry – Revue Canadienne de Psychiatrie*, **54**(1), 4–5.

MacDonald, K. and Hershberger, S. L. (2005). Theoretical issues in the study of evolution and development. In *Evolutionary Perspectives on Human Development*, R. L. Burgess and K. MacDonald (eds). Thousand Oaks, CA: Sage, pp. 21–72.

Maier, S. F., Watkins, L. R. and Fleschner, M. (1994). Psychoneuroimmunology: the interface between behavior, brain, and immunity. *American Psychologist*, **49**, 1004–1007.

Mason, J. W. (1968). A review of psychoendocrine research on the pituitary-adrenal cortical system. *Psychosomatic Medicine*, **30**, 576–607.

Mason, J. W. (1971). A re-evaluation of the concept of "non-specificity" in stress theory. *Journal of Psychosomatic Research*, **8**, 323–334.

McEwen, B. S. (1995). Stressful experience, brain, and emotions: developmental, genetic, and hormonal influences. In *The Cognitive Neurosciences*, M. S. Gazzaniga (ed.). Cambridge, MA: MIT Press, pp. 1117–1135.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, **338**, 171–179.

Meaney, M. J., Mitchell, J., Aitken D., et al. (1991). The effects of neonatal handling on the development of the adrenocortical response to stress: implications for neuropathology and cognitive deficits later in life. *Psychoneuroendocrinology*, **16**, 85–103.

Meaney, M. J., Szyf, M. and Seckl, J. R. (2007). Epigenetic mechanisms of perinatal programming of hypothalamic-pituitary-adrenal function and health. *Trends in Molecular Medicine*, **13**(7), 269–277.

Miller, G. E. (2000). *The Mating Mind: How Sexual Choice Shaped the Evolution of Human Nature*. New York: Doubleday.

Mirescu, C., Peters, J. D. and Gould, E. (2004). Early life experience alters response of adult neurogenesis to stress. *Nature Reviews: Neuroscience*, **7**(8), 841–846.

Moll, J., Zahn, R., de Oliveira-Souza, R., et al. (2005). The neural basis of human moral cognition. *Nature Reviews: Neuroscience*, **6**(10), 799–809.

Muehlenbein, M. P. (2008). Adaptive variation in testosterone levels in response to immune activation: empirical and theoretical perspectives. *Social Biology*, **53**, 13–23.

Muehlenbein, M. P. and Bribiescas, R. G. (2005). Testosterone-mediated immune functions and male life histories. *American Journal of Human Biology*, **17**, 527–558.

Munck, A. and Guyre, P. M. (1991). Glucocorticoids and their immune function. In *Psychoneuroimmunology*, R. Ader, D. L. Felten, and N. Cohen (eds). New York: Academic Press, pp. 447–474.

Munck, A., Guyre, P. M. and Holbrook, N. J. (1984). Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. *Endocrine Reviews*, **5**, 25–44.

Murdock, G. P. (1949). *Social Structure*. New York: Macmillan.

Murdock, G. P. (1967). *Ethnographic Atlas*. Pittsburgh, PA: University of Pittsburgh Press.

Nachmias, M., Gunnar, M., Mangelsdorf, S., et al. (1996). Behavioral inhibition and stress reactivity: the moderating role of attachment security. *Child Development*, **67**, 508–522.

Newcomer, J. W., Craft, S., Hershey, T., et al. (1994). Glucocorticoid-induced impairment in declarative memory performance in adult humans. *Journal of Neuroscience*, **14**(4), 2047–2053.

Nowak, M. A., Komarova, N. L. and Niyogi, P. (2001). Evolution of universal grammar. *Science*, **291**, 114–118.

Pande, H., Unwin, C. and Haheim, L. L. (1997). Factors associated with the duration of breastfeeding. *Acta Paediatrica*, **86**, 173–177.

Panksepp, J. (2004). *Affective Neuroscience: the Foundations of Human and Animal Emotions*. New York: Oxford University Press.

Panksepp, J. (2009). Carving "natural" emotions: "kindly" from bottom-up but not top-down. *Journal of Theoretical and Philosophical Psychology*, **28**(2), 395–422.

Pearlin, L. I. and Turner, H. A. (1987). The family as a context of the stress process. In *Stress and Health: Issues in Research Methodology*, S. V. Kasl and C. L. Cooper (eds). New York: Wiley, pp. 143–165.

Pinker, S. (1994). *The Language Instinct*. New York: William Morrow.

Pinker, S. (1997). *How the Mind Works*. New York: Norton.

Rosenberg, K. (2004). Living longer: information revolution, population expansion, and modern human origins. *Proceedings of the National Academy of Sciences of the United States of America*, **101**(30), 10847–10848.

Rosenberg, K. and Trevathan, W. (2002). Birth, obstetrics and human evolution. *BJOG: An International Journal of Obstetrics and Gynecology*, **109**(11), 1199–1206.

Roth, G. and Dicke, U. (2005). Evolution of the brain and intelligence. *Trends in Cognitive Sciences*, **9**(5), 250–257.

Sakai, K. L. (2005). Language acquisition and brain development. *Science*, **310**, 815–819.

Saphier, D., Welch, J. E., Farrar, G. E., et al. (1994). Interactions between serotonin, thyrotropin-releasing hormone and substance P in the CNS regulation of adrenocortical secretion. *Psychoneuroendocrinology*, **19**, 779–797.

Sapolsky, R. M. (1991). Effects of stress and glucocorticoids on hippocampal neuronal survival. In *Stress: Neurobiology and Neuroendocrinology*, M. R. Brown, G. F. Koob and C. Rivier (eds). New York: Dekker, pp. 293–322.

Sapolsky, R. M. (1992). *Stress, the Aging Brain, and the Mechanisms of Neuron Death*. Cambridge, MA: MIT Press.

Sapolsky, R. M. (1994). *Why Zebras Don't Get Ulcers*. New York: W. H. Freeman and Co.

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science*, **308**(5722), 648–652.

Sapolsky, R. M., Romero, L. M. and Munck, A. U. (2000). How do glucocorticoids influence stress responses? *Endocrine Reviews*, **21**(1), 55–89.

Schneider, M. L., Coe, C. L. and Lubach, G. R. (1992). Endocrine activation mimics the adverse effects of prenatal stress on the neuromotor development of the infant primate. *Developmental Psychobiology*, **25**, 427–439.

Sear, R., Mace, R. and McGregor, I. A. (2000). Maternal grandmothers improve the nutritional status and survival of children in rural Gambia. *Proceedings of the Royal Society of London. Series B*, **267**, 1641–1647.

Seckl, J. R. (2008). Glucocorticoids, developmental "programming" and the risk of affective dysfunction. *Progress in Brain Research*, **167**, 17–34.

Selye, H. (1976). *The Stress of Life*. New York: McGraw-Hill.

Servan-Schreiber, D., Printz, H. and Cohen, S. D. (1990). A network model of catecholamine effects: gain, signal-to-noise ratio, and behavior. *Science*, **249**, 892–895.

Shamay-Tsoory, S. G., Tomer, R. and Aharon-Peretz, J. (2005). The neuroanatomical basis of understanding sarcasm and its relationship to social cognition. *Neuropsychology*, **19**(3), 288–300.

Siegal, M. and Varley, R. (2002). Neural systems involved with "Theory of Mind." *Nature Reviews Neuroscience*, **3**, 463–471.

Silk, J. S., Alberts, S. C. and Altmann, J. (2003). Social bonds of female baboons enhance infant survival. *Science*, **302**, 1231–1234.

Simons, K., Paternite, C. E. and Shore, C. (2001). Quality of parent/adolescent attachment and aggression in young adolescents. *Journal of Early Adolescence*, **21**, 182–203.

Smith, B. H. (1994). Patterns of dental development in homo, Australopithecus, pan, and gorilla. *American Journal of Physical Anthropology*, **94**(3), 307–325.

Smuts, B. B. and Smuts, R. W. (1993). Male aggression and sexual coercion of females in nonhuman primates and other mammals: evidence and theoretical implications. *Advances in the Study of Behavior*, **22**, 1–63.

Stearns, S. C. (1992). *The Evolution of Life Histories*. Oxford: Oxford University Press.

Storey, A. E., Walsh, C. J., Quinton, R. L., et al. (2000). Hormonal correlates of paternal responsiveness in new and expectant fathers. *Evolution and Human Behavior*, **21**(2), 79–95.

Tinbergen, N. (1963). On the aims and methods of ethology. *Zeitschrift für Tierpsychologie*, **20**, 410–463.

Tomasello, M. (1999). *The Cultural Origins of Human Cognition*. Cambridge, MA: Harvard University Press.

Tooby, J. and Cosmides, L. (1992). The psychological foundations of culture. In *The Adapted Mind*, J. H. Barkow, L. Cosmides and J. Tooby (eds). Oxford: Oxford University Press, pp. 19–36.

Tulving, E. (2002). Episodic memory: from mind to brain. *Annual Review of Psychology*, **53**, 1–25.

Uvnas-Moberg, K. (1998). Oxytocin may mediate the benefits of positive social interaction and emotions. *Psychoneuroendocrinology*, **23**, 819–835.

van Anders, S. M. and Gray, P. B. (2007). Hormones and human partnering. *Annual Review of Sex Research*, **18**, 60–93.

van der Meij, L., Buunk, A. P., van de Sande, J. P., et al. (2008). The presence of a woman increases testosterone in aggressive dominant men. *Hormones and Behavior*, **54**, 640–644.

Wallerstein, J. S. (1983). Children of divorce: stress and developmental tasks. In *Stress, Coping, and Development in Children*, N. Garmezy and M. Rutter (eds). New York: McGraw-Hill, pp. 265–302.

Weaver, I. C. G., Cervoni, N., Champagne, F. S., et al. (2004). Epigenetic programming by maternal behavior. *Nature Reviews: Neuroscience*, **7**(8), 847–854.

Weiner, H. (1992). *Perturbing the Organism*. Chicago: University of Chicago Press.

West-Eberhard, M. J. (1983). Sexual selection, social competition, and speciation. *Quarterly Review of Biology*, **58**, 155–183.

West-Eberhard, M. J. (2003). *Developmental Plasticity and Evolution*. Oxford: Oxford University Press.

Whiting, B. B. and Edwards, C. (1988). *Children of Different Worlds*. Cambridge, MA: Harvard University Press.

Wilkinson, R. G. (2001). *Mind the Gap: Hierarchies, Health, and Human Evolution*. New Haven, CT: Yale University Press.

Williams, G. C. (1966). *Adaptation and Natural Selection*. Princeton: Princeton University Press.

Williams, R. W. and Herrup, K. (1988). The control of neuron number. *Annual Review of Neuroscience*, **11**, 423–453.

Wilson, M. I., Daly, M. and Weghorst, S. J. (1980). Household composition and the risk of child abuse and neglect. *Journal of Biosocial Sciences*, **12**, 333–340.

Wrangham, R. W. (1999). Evolution of coalitionary killing. *Yearbook of Physical Anthropology*, **42**, 1–30.

Wrangham, R. W. and Peterson, D. (1996). *Demonic Males*. New York: Houghton Mifflin Company.

Wynne-Edwards, K. E. (2003). From dwarf hamster to daddy: The intersection of ecology, evolution, and physiology that produces paternal behavior. In *Advances in the Study of Behavior*, P. J. B. Slater, J. S. Rosenblatt, C. T. Snowden et al. (eds). San Diego, CA: Academic Press, **32**, pp. 207–261.

Young, L. J. and Insel, T. R. (2002). Hormones and parental behavior. In *Behavioral endocrinology*, J. B. Becker, S. M. Breedlove, D. Crews, et al. (eds). Cambridge, MA: MIT Press, pp. 331–369.

Young, L., Wang, Z. and Insel, T. R. (2002). Neuroendocrine bases of monogamy. In *Foundations in Social Neuroscience*, J. T. Cacioppo, G. G. Berntson, R. Adolphs, et al. (eds). Cambridge, MA: MIT Press, pp. 809–816.

Yuwiler, A. (1982). Biobehavioral consequences of experimental early life stress: effects of neonatal hormones on monoaminergic systems. In *Critical Issues in Behavioral Medicine*, L. J. West and M. Stein (eds). Philadelphia: J. P. Lippincott, pp. 59–78.

Ziegler, T. E. and Snowdon, C. T. (1997). Role of prolactin in paternal care in a monogamous New World primate, *Saguinus oedipus*. The integrative neurobiology of affiliation. *Annals of the New York Academy of Sciences*, **807**, 599–601.