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Biological Perspectives on Social Attachment and Bonding

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ABSTRACT

The purpose of this review is to examine factors influencing both the development and expression of mammalian social bonds in the biological context of natural history, evolution, and neuroendocrinology. Animal research suggests that social bonding can be modulated by various hormones including oxytocin, vasopressin, opioids, corticotropin-releasing hormone (CRH), dopamine, and adrenal steroids, including corticosterone or cortisol. The effects of these hormones on social bond formation are especially apparent following periods of stress or anxiety. For example, social bonds are associated with birth or the introduction of a novel partner. Of possible relevance to behavioral attachment is the capacity of hormones, such as oxytocin and vasopressin, to overcome anxiety or fear. Brief exposure to oxytocin or vasopressin can facilitate social contact and in some species selective sociality. Neural systems associated with reward, including those that rely on dopamine and possibly the endogenous opioids, may help to regulate responses to the presence or absence of a preferred partner. The capacity to form social bonds emerges as a function of interactions among genetics and developmental experiences. Systems associated with sociality are designed to accommodate species and individual variations. Within a genetic background, social and hormonal experiences can reprogram the nervous system and thus potentially alter the tendency of individuals to form social bonds. An understanding of the physiological mechanisms responsible for social bonding may provide clues to the biological and psychological benefits of social support.

INTRODUCTION

Social relationships, including those structured around social bonds and emotional attachments, can be a source of life's greatest pleasures and deepest pain. The best described examples of social bonds, including parent–offspring and adult heterosexual relationships, are associated with reproduction and are most readily interpreted in the context of their evolution and adaptive functions (Bowlby 1969). The consequences of social bonds also extend beyond reproduction, with potential benefits for all aspects of life. Understanding the

neurobiology of social bonds provides insights into the protective mechanisms underlying perceived social support (Carter 1998).

Adult pair bonds and bonds between mothers and their infants are based at least in part on shared neurobiological systems. Hormones and neural systems that are responsive to these hormones coordinate the formation of social bonds with other events such as birth or emotional states. There also is evidence that these same hormones may help to program the nervous system, especially during early life, contributing to individual and sex differences, and species-typical patterns of social behavior.

SOCIAL BONDS

What Is a Social Bond?

Social bonds are a subset of affiliative or positive social behaviors, which in turn are most simply defined by approach rather than avoidance or withdrawal. Positive sociality is sometimes defined by the absence of defensive or aggressive behavior. Social behaviors, including social bonds, are active processes that involve more than simply a failure to avoid another individual (Carter 1998).

Several features are common to most definitions of social bonds. Social engagement and individual recognition are the first steps in social bonding. Differential behavioral or emotional interactions in the presence or absence of the partner are most commonly used to define a social bond. Although the term "pair bond" implies only two partners, the tendency or willingness to form social bonds may extend to more than one partner. The capacity to form either one or more social bonds varies across species and, in some cases, may lead to extended family groups.

The most accessible operational definitions of social bonds are behavioral. Social bonds are often indexed experimentally by selective approach in partner preference tests. Such tests typically include a choice between a familiar partner or a stranger or the option to remain alone. Physical contact seems to be a particularly sensitive indicator of social bonding (Carter et al. 1995), although a choice between other types of social stimuli (such as odors) may be sufficient to detect preferences. In adult animals, there have been attempts to use sexual preferences to index pair bonding; however, these are complicated by the fact that some animals are more particular or exclusive in choosing their social partners than they are in selecting a mating partner.

Defense of the mate or territory can also be a defining feature of a social bond. For example, socially monogamous species tend to be aggressive toward strangers. Although aggression can occur toward familiar conspecifics, it is less common. Intense aggression, usually toward unfamiliar animals of the same sex, can be triggered by sexual experience (Winslow et al. 1993) and/or prolonged periods of cohabitation (Bowler et al. 2002). The selective aggression seen in socially monogamous species may serve to defend either the mate or

family after a social bond has been established. Aggression is also seen in polygamous species; however, in such species, aggression may occur before mating, presumably in competition for resources or rank.

In some cases behavioral, autonomic, or other physiological responses to the presence or absence of a partner have been used to index social bonds. For example, heart rate or endocrine responses, such as increases in heart rate or adrenal hormone levels following separation, may reflect emotional changes associated with the absence of a preferred social partner. Physiological changes can be difficult to measure, especially during naturalistic behavioral interactions. Even more challenging are attempts to interpret physiological measures in behavioral or emotional terms (Hennessy 1997; Mason and Mendoza 1998; Porges 2001 and this volume). For example, changes in heart rate or stress hormones may occur as the result of mobilization, fear, changes in thermoregulation, or other processes that are not specifically related to emotions experienced during separation from a partner. Thus, without careful consideration of the context in which they occur, physiological measures may not be meaningful measures of social bonds.

Evolutionary Context

At the center of evolutionary theories is the concept of genetic survival or "fitness." Individual survival to at least reproductive age is a precondition for reproduction, and reproduction is essential for the transmission and survival of genes. Mammals produce young that are nutritionally dependent for at least some part of their lives on the mother. In most — but not all — mammals, milk and maternal behavior are both delivered primarily to the mother's genetic offspring. For the mammalian infant, the importance of the mother is clear. In addition, many benefits of the maternal or, where appropriate, paternal role may be provided by "alloparents," who are sometimes, but not always, genetically related to the young (Carter and Roberts 1997; Hrdy, this volume).

Parents and even alloparents can also benefit from interactions with young animals. In addition to genetic fitness, the mother may gain physically and emotionally from interactions with her own offspring. This is seen in the capacity of lactating women to be more resilient in the face of stressors (Carter and Altemus 1997; Carter et al. 2001). It is often difficult for a male to determine whether a female's young are his own offspring. Perhaps as a result it is rare for males to provide care for infants. However, in a limited number of mammalian species, both males and females exhibit selective social behaviors, sometimes termed "pair bonds," which may encourage the male and female to remain together after mating and even during nonreproductive periods. There also is a strong association between the tendency to form adult pair bonds and the expression of male parental behavior. The coincidence of these behaviors has been used to categorize species as "monogamous" (Kleiman 1977).

The term monogamy has also been used to describe sexual exclusivity or fidelity. The multiple uses of this word have created some confusion. It is now well-established that sexual exclusivity is not necessarily a reliable trait of "monogamous" species; i.e., those species that tend to live in pairs and show male parental behavior (Carter et al. 1995; Gowaty 1997). However, a male that remains with a female sexual partner and invests in her offspring may increase the chances that his own genetic offspring will survive. The advantages of one mating strategy over another in a particular situation are difficult to determine. However, as a lifestyle, polygamy, which does not demand either pair bonds or paternal behavior, can be successful under conditions when a single female can rear her offspring unaided by the father.

It is often argued that monogamy — or what is now sometimes called "social monogamy" — may be especially beneficial when two caretakers are necessary to rear a family. However, in apparent contradiction to Kleiman's original definition of monogamy (1977), Komers and Brotherton (1997) claim that social monogamy is observed more often in the absence of paternal care than in its presence. Thus, the latter propose that monogamy may have evolved first (for reasons other than the "need" for a second parent) and that the availability of a second parent as a caretaker is a secondary, but not essential, benefit of monogamy. Regardless of their evolutionary origins, socially monogamous species have provided a novel opportunity to understand the biological processes responsible for social bonds.

What Are the Proximate Causes of Social Bonding?

Social bonds are interwoven with the capacity to adapt to an ever-changing and challenging environment. Stable companionship may function to provide a sense of safety and reduce anxiety. Stressful experiences (such as pregnancy and parturition), anxiety, neophobia, and isolation often precede the formation of long-lasting social attachments (Bowlby 1969; Panksepp 1998; Carter. 1998).

Neurochemical changes that are capable of overcoming neophobia may also lead to a decrease in social inhibitions and increased sociality, a first step in social bond formation. However, under a variety of experimental conditions, animals may be indiscriminantly social. The tendency toward selectivity in social responses probably relies on mechanisms that promote both general sociality as well as specific neurophysiological processes necessary to reinforce or reward selective sociality (Insel 2003). In addition, inherent to the concept of a social bond is recognition memory. The neural substrates for nonselective social engagement and selective social bonding are related but not identical (Cho et al. 1999; Insel and Young 2001).

Transformations from behavioral avoidance to approach and the subsequent preferential or selective social behaviors are critical elements in the formation of social bonds. As described below, several hormones and receptor systems have been implicated in this transformation.

Table 5.1 Features of the neurobiology of oxytocin (OT) and arginine vasopressin (AVP). For details and exceptions, see Carter (1998) and Russell et al. (2003).

- Ancient origins prior to the separation between vertebrates and invertebrate
- Specific 9-amino acid structures of OT and AVP are novel to mammals
- OT is most abundant neuropeptide in the hypothalamus as indexed by mRNA
- Synthesized in largest cells in the CNS (magnocellular neurons) as well as other smaller cells
- Transported by neurosecretion to posterior pituitary, but also released in CNS
- Sibling hormones, OT and AVP, have consequences for each other's functions
- OT has only one known receptor but may bind to one or more of the three AVP receptors

Neuroendocrine Substrates for Social Bonding

Among the compounds that have been implicated in social engagement, social recognition, and bond formation are two uniquely mammalian peptide hormones: oxytocin and arginine vasopressin (Table 5.1). Vasopressin and oxytocin are closely related compounds that probably originated from a common gene. Both consist of nine amino acids. Oxytocin and vasopressin are present in many of the same regions of the brain, although usually not in the same cells (Russell et al. 2003). Oxytocin and vasopressin are made in and act on the brain, especially in the hypothalamus and areas of the nervous system that regulate the autonomic nervous system. Oxytocin has only one known receptor; however, vasopressin has three receptor subtypes, of which the V1a type has been implicated in social bonding (Young 1999). These two peptides have dynamic interactions that help to integrate emotional and autonomic states with social behaviors.

Oxytocin is associated with birth, lactation, and sexual behavior as well as modulation or down regulation of the hypothalamic–pituitary–adrenal (HPA) axis. It may help mammals to manage the stress of birth and the postpartum period (Carter et al. 2001), and remarkably, this same hormone has the capacity to produce behavioral and physiological calming (Uvnas-Moberg 1998). Oxytocin also has been implicated in various forms of positive social interactions including social bonding.

Model Systems

Early studies of the mechanisms associated with social bonds in animals focused primarily on primates, dogs, guinea pigs, and ungulates (reviewed by Hennessy 1997; Mason and Mendoza 1998; Panksepp 1998). Many of these studies were aimed at understanding the bonds between adults and infants and often emphasized the response to separation. For example, one of the first attempts to understand the regulation of separation distress in infant guinea pigs focused on opioids, suggesting that these endogenous peptides could modulate the crying

normally associated with separation. Keverne and his colleagues, working with sheep, were the first to document a clear role for another neuropeptide hormone, oxytocin, in the formation of mother–infant bonds (Keverne et al. 1997).

Oxytocin

A more recent literature examining the neurobiology of adult–adult social bonding has come from socially monogamous rodents of the genus Microtus (voles). Several species from this genus (including prairie and pine voles) are capable of pair bonding as well as male parental behaviors, whereas others (including meadow and montane voles) that do not show pair bonds or male parental behavior are usually classified as nonmonogamous or polygamous. Among these species, prairie voles (*Microtus ochrogaster*) have been most extensively studied in the field as well as in the laboratory (Carter et al. 1995).

Treatment with oxytocin quickly facilitates positive social behaviors, including selective partner preferences and maternal behavior (Pedersen and Boccia 2002; Carter and Keverne 2002). Chronic exposure to oxytocin is capable of down regulating or buffering the response to stressors and the reactivity of the autonomic nervous system, including heart rate and blood pressure (Uvnas-Moberg 1998). Oxytocin is released during positive social interactions and may permit social interactions without fear (Porges, this volume). In rats, touch and massage (presumably in a context of perceived safety) can release oxytocin, which in turn may feed back on the nervous system to further enhance relaxation (Uvnas-Moberg 1998). Thus, oxytocin is an excellent candidate for the integration of emotional experiences with the physiological processes through which social bonds bestow benefits.

Vasopressin

Some of the effects of vasopressin on social behavior appear similar to those attributed to oxytocin. In particular, both peptides may have the capacity to reduce social anxiety and both can facilitate social bonding. In prairie voles, vasopressin that is administered into the central nervous system can increase social contact as well as facilitate the onset of a preference for a familiar partner (Cho et al. 1999). The effects of vasopressin on social behavior are blocked by pretreatment with a V1a vasopressin receptor antagonist (Winslow et al. 1993). In addition, when genes for the prairie vole vasopressin (V1a) receptor were transferred by a viral vector into the limbic system of male mice, these mice showed significant increases in affiliative contact, although this social behavior was not selective (Young 1999; Insel and Young 2001).

Complicating attempts to understand the separate effects of vasopressin and oxytocin is the fact that these structurally similar peptides are capable of binding to each other's receptors (Russell et al. 2003). However, the autonomic and possibly some of the emotional properties of the effects of vasopressin are different

from those associated with oxytocin. Vasopressin is capable of increasing blood pressure and cardiovascular functions, and may facilitate the release of hormones of the HPA axis. Treatment with vasopressin is associated with alertness, behavioral reactivity, arousal, and in many cases the defense of both the individual and the family (Carter et al. 1995; Carter 1998). In certain areas of the brain, vasopressin is a sexually dimorphic hormone with males producing higher levels than females. This sex difference is due to the fact that vasopressin synthesis, especially within the limbic system, is facilitated by androgens (De Vries and Villalba 1997). Vasopressin may be especially important to the postcopulatory mate-guarding and male parental behavior that is typical of monogamous species (Winslow et al. 1993). The behavioral characteristics of vasopressin may permit males from socially monogamous species, such as prairie voles, to show necessary sexually dimorphic behaviors, such as mounting or even aggression toward strangers, while continuing to form social bonds and exhibiting high levels of social behavior within the family.

Other "Stress" Hormones

Another neuropeptide that has been implicated in stress and anxiety as well as pair bonding is corticotropin-releasing hormone (CRH). CRH is synthesized and released in several areas of the hypothalamus that are implicated in emotion and the regulation of autonomic responses. In male prairie voles there is also evidence that treatment with moderate, but not high, doses of CRH can facilitate pair bonding; pretreatment with a specific CRH antagonist blocked these effects (DeVries et al. 2002). Evidence from other species suggests that the actions of CRH may synergize with those of vasopressin to regulate the release of adrenal steroids. It is also possible that CRH and vasopressin work in synchrony to allow stressful experiences to facilitate pair bonding, especially in males. Whether CRH plays a role in pair bonding in females remains to be studied.

The relationship between anxiety or stress and social bonding also offers important clues to the identity of other hormones that may be associated with social attachment. At least some species of socially monogamous mammals have exceptionally high levels of adrenal steroids, and it is possible that these steroids have behavioral consequences related to the development and expression of the traits of monogamy (DeVries et al. 1996; Carter 1998; Carter and Keverne 2002). Also of possible relevance to the capacity of stressful experiences to facilitate social bonding is the fact that in rats the binding of oxytocin to its receptor is increased by the presence of adrenal steroids, especially in the amygdala (Liberzon and Young 1997).

Among the other hormones associated with stress and anxiety that have been implicated in pair bonding are the catecholamines, including dopamine and norepinephrine (Keverne et al. 1997). Norepinephrine, associated with arousal and activation, also helps to regulate oxytocin release (Russell et al. 2003).

Reward

The chemistry of reward, which involves dopamine, remains integral to the processes that allow attachments to form (Insel 2003). Among the possible roles for dopamine in social bond formation is enhancement of the perceived hedonic properties of the partner or stimuli associated with the partner. Both dopamine and opioid receptors are found in the nucleus accumbens, which (as described below) may be of relevance to the emotional states that accompany social bonding. In addition, in a yet to be determined manner, oxytocin and vasopressin may help to connect social stimuli to the reward system.

In prairie voles, dopamine–oxytocin interactions have been implicated in the formation of social bonds in both sexes (Aragona et al. 2003). Not all species of mammals have an anatomical association between dopamine and oxytocin; the co-localization — or alternatively lack of co-localization — of these chemicals may account in part for species differences in the capacity to form social bonds. Consistent with species differences in the tendency to show selective social behaviors is the fact that in rats and nonmonogamous voles, receptors for oxytocin and dopamine are not co-localized in classic reward pathways (Insel 2003). The rewarding effects of dopamine could serve to cement relationships and enhance emotional feelings associated with the formation of social bonds.

Addiction and Social Bonds

Early attempts to understand social bonds focused attention on possible parallels between the addictive properties of endogenous opioids and those of social bonds (Panksepp 1998). However, the effects of endogenous opioids on social behavior are complex and may vary as a function of species age, gender, and social rank. Evidence for opioid effects on social bonding came first from studies showing that blocking opioid receptors with naloxone increased separation cries in young guinea pigs, whereas morphine had the opposite effect. However, adult social behaviors are not readily blocked by naloxone treatments, leading Panksepp to conclude that although the endogenous opioids may play a role in separation distress, they are not essential for social reward. Endogenous opioids can, however, regulate oxytocin and vasopressin, including the capacity to release these peptides (Russell et al. 2003). Opioids also have been implicated in maternal behavior. For example, in sheep, blocking opioid receptors prevented both the release of oxytocin and the facilitatory effects of vaginal–cervical stimulation on social bonding (Keverne et al. 1997). It is possible that opioids have both direct and indirect modulatory effects on social behavior, although their role in social bonding is at present poorly defined (Panksepp 1998; Keverne, this volume).

Disruptions in or the absence of social bonds are associated with anxiety and vulnerability to substance abuse, and the most effective treatments for addiction often incorporate social support. As the identities of ancient and powerful

neuroendocrine systems, including those that incorporate oxytocin, have become more apparent, knowledge of their properties helps us to understand causes and consequences of social bonds, including the mechanisms through which giving and receiving social support benefit health and well-being (Carter 1998; Uvnas-Moberg 1998). Oxytocin may be especially necessary for the selective and vulnerable immobility that is characteristic of social bonds and is necessary for birth, nursing, and orgasm (Porges, this volume).

Neuroanatomical Substrates of Social Bonding

The neural mechanisms for the actions of hormones that influence social bonding remain only superficially understood (reviewed Carter and Keverne 2002). It is known that the receptors for the hormones that have been implicated in social bonding, including oxytocin and vasopressin, are differentially distributed in regions of the nervous system, such as the olfactory system, the extended amygdala (including the nucleus accumbens and ventral pallidum), the lateral septum, and lower brainstem (Young 1999; Insel and Young 2001). In turn, these areas have been implicated in social behavior and social memory, anxiety, the regulation of autonomic and visceral responses, and reward (Carter and Keverne 2002; Insel 2003; Porges 2001 and this volume).

Summary

In pair-bonding species, biological changes associated with anxiety or fear set the stage for attachment. Oxytocin or vasopressin (or related hormones), released especially in the presence of or following exposure to stress hormones, could help to overcome anxiety or fear in the face of strangers. For example, we have recently observed in female prairie voles that brief exposure to an infant results in a rapid decline in corticosterone and can also facilitate the formation of subsequent adult social bonds, possibly through the release of endogenous oxytocin (Kim, Bales, and Carter, unpublished data). Oxytocin, especially in females but perhaps in males as well (Cho et al. 1999), may allow or encourage the social interactions and immobility which in turn permit social bonds to form. Other hormonal systems, including those classically associated with reward (e.g., dopamine and the endogenous opioids) may interact with oxytocin and vasopressin to specify a particular partner or regulate the response to the absence of a partner or other disruptions in social bonds.

HOW ARE INDIVIDUAL, GENDER, AND SPECIES VARIATIONS IN THE CAPACITY FOR SOCIAL BONDING PRODUCED?

Understanding differences in the tendency to form pair bonds requires an appreciation for interactions among genetic and epigenetic/developmental processes.

For example, a stressful or fearful experience, mediated in part by adrenal steroids, may have immediate consequences for social interactions and, in some cases, stress facilitates pair bonding (DeVries et al. 1996; Carter 1998). In addition, social experiences across the life span may also reprogram the later expression of peptide hormones and their receptors, further contributing to individual or species variation in the capacity or willingness to form pair bonds (Henry and Wang 1998; Pedersen and Boccia 2002). For example, in prairie voles, early handling, probably mediated by increased maternal stimulation of the pups, produces in those pups long-lasting increases in oxytocin within the brain. In separate studies we have found that exposure to oxytocin in early life increases the later production of oxytocin, especially in females, and also produces animals that form pair bonds more quickly than their untreated counterparts (reviewed Carter 2003).

The relationship between pair bonding and reproduction suggests a possible role for sex steroids. However, animals that are gonadectomized in adulthood are capable of forming pair bonds. Thus, in adult animals, gonadal steroids may not be essential for the formation of partner preferences (Carter et al. 1995). Sex steroids, however, may have a critical behavioral role during early development by altering the production of and adult responses to neuropeptides, including vasopressin and oxytocin. Over the life span of an individual, sex steroids regulate processes that lead to sexual differentiation and thus may help to explain sex differences in the tendency to form pair bonds (De Vries and Villalba 1997; Carter 2003). In addition, reproductive hormones such as estrogens and androgens can regulate the synthesis and actions of peptides, such as oxytocin and vasopressin, providing indirect effects on the ability of an individual to form social bonds.

Although gonadal hormones may not be essential for pair bonding, sex differences do exist in the capacity and mechanisms for pair bond formation (Carter 1998, 2003). For example, in prairie voles, stressful experiences and stress hormones from the adrenal gland tend to facilitate heterosexual pair bond formation in males; in contrast, in females comparable experiences or hormones inhibit female–male pair bonding (DeVries et al. 1996). However, following a stressful experience, females may form female–female bonds, suggesting that both physiological and social context must be taken into account in attempts to understand the mechanisms underlying pair bonding.

Mechanisms for sex differences in pair bond formation may involve sex differences in the behavioral effects of oxytocin and vasopressin (Carter 1998). Chronic exposure to oxytocin is associated with energy conservation and immobility, in part through interactions with the parasympathetic nervous system (Uvnas-Moberg 1998; Porges 1998, 2001, this volume). In contrast, vasopressin promotes sympathetic arousal, increases in blood pressure and cardiovascular activity, behavioral alertness, and physical mobilization. The best known functions of oxytocin (birth and lactation) are unique to females.

Estrogen tends to facilitate the synthesis and actions of oxytocin, although both males and females are capable of producing oxytocin. It is likely that the effects of vasopressin on pair bonding reflect the effects of this peptide within the limbic system, and vasopressin synthesis in the limbic system is especially androgen dependent (De Vries and Villalba 1997).

Relative differences between the production and effects of oxytocin versus vasopressin are compatible with sex differences in reproductive behaviors. However, the actions of these peptides may also differ as a function of the endocrine history of the individual. For example, castration in early life renders male prairie voles (in adulthood) less sensitive to the pair bond-facilitating effects of vasopressin (Cushing et al. 2003).

Recent breakthroughs in molecular genetics offer another perspective on the mechanisms for individual and species differences in the capacity to form social bonds. Species differences in the receptors for peptide hormones and their distributions in the nervous system, such as those seen in voles (Witt et al. 1991; Insel and Young 2001), are now widely accepted. However, both the synthesis of both peptide hormones and their receptors can also be altered by early social and hormonal experiences. Such processes create variations in the nervous system and may be responsible for subsequent behavioral differences among species, between males and females within a species, and among individuals (Carter 2003). For example, interspecific variations in the regulatory regions of the gene for the vasopressin V1a receptor have been implicated in social behavior (Young 1999; Insel and Young 2001).

Working within genetic constraints, early exposure to hormones, including peptides and steroids, has the capacity to reprogram the developing nervous system. In addition, early experiences can alter the capacity of the nervous system to produce hormones, including oxytocin (Carter et al. 2003). Such changes can produce adaptive and, in some cases, long-lasting changes in physiology and behavior. Patterns of social behaviors seem to be especially sensitive to the effects of early experience (Pedersen and Boccia 2002; Carter 2003). It is likely that differences in the capacity to form social bonds reflect the social and endocrine history of the individual, the consequences of sexual differentiation, as well as species variations in neuroendocrinology and neuroanatomy.

HUMAN SOCIAL BONDS

What Is Known regarding the Biological Basis of Human Social Bonding?

Studies directly investigating the biological basis of human social bonds are uncommon. However, the physiological circumstances associated with social bond formation are probably similar in humans and other mammals. The basic hormones and neural mechanisms responsible for social behaviors are also conserved among mammals, and thus shared by humans and other mammals.

As in other mammals, human physiology is usually capable of accommodating the demands of birth and lactation. However, there is considerable variation in the birth process among individuals, and the large size of human infants and their skulls relative to the human cervix/pelvis presents special challenges to the birth process. In this context, humans, along with a few domestic animals, are unique in the fact that they may experience hormonally assisted and/or surgical delivery of their young. The effects of a recently introduced oxytocin-antagonist (Atosiban) as a method for preventing premature labor remain to be studied in humans; however, in other species, blocking oxytocin in early life can disrupt social behavior (Carter 2003). Furthermore, human females may or may not nurse their own offspring. In those women who do breastfeed, patterns and durations of nursing/lactation are extremely variable. Finally, human infants are raised under extremely variable conditions (Hrdy, this volume), which based on our experience with animals, might be expected to have effects on the subsequent sociality of the infant. The consequences for the parent–infant bond of such variations and modifications in labor, lactation, and parental behavior have received little attention.

It is known that lactating women may be protected from psychological and physiological stressors, and breastfeeding women are in general less reactive to stressful experiences than women who give birth but do not lactate (Carter and Altemus 1997; Carter et al. 2001). Human pregnancy and birth are associated with transient elevations in CRH, cortisol, estrogen, catecholamines, prolactin, vasopressin, and oxytocin. Furthermore, gonadal steroids decline following parturition. Dramatic hormonal shifts which occur during pregnancy, birth, and the postpartum period may affect the emotional state of the mother and indirectly influence the child (Carter 2003). Impairments in the capacity to manage these shifts could predispose some women to emotional instability, including a vulnerability to depression or in rare cases psychosis (Carter et al. 2001). The emotional status of the mother could of course also affect the baby. Lactation may help to buffer the new mother from these hormonal and autonomic shifts, including dramatic changes in the HPA axis and other hormonal systems. The HPA axis has been implicated in depression. However, the relationship between lactation and postpartum depression is poorly understood, and this literature is both incomplete and difficult to interpret, complicating any attempts to draw strong conclusions. Even in the face of this complexity, there is evidence that the emotional reactions of both mothers and fathers to their infants may correlate with hormonal levels in late pregnancy (Storey et al. 2000; Fleming, this volume).

Of particular relevance to the mother–infant interaction may be the pulsatile release of oxytocin that normally induces uterine contractions and is necessary for milk ejection (Russell et al. 2003). Women who deliver by caesarian section, especially if the surgery occurs before the onset of labor, may not experience the birth-related pulses of oxytocin and may have difficulty in establishing

lactation, in part because they do not readily produce a pulsatile pattern of oxytocin release in the immediate postpartum period (Uvnas-Moberg 1998).

It is plausible that the physiological conditions that lead to parental bonding in humans are related to those that are associated with social bond formation in other species. Based on the literature in nonhuman animals, we can postulate that a woman (and her partner or other companions) who experiences high levels of emotional arousal and HPA axis activity, in the prenatal period, or even during labor, might be especially primed to form a new social bond with a child. Birth, lactation, and stimuli from a newborn may be powerful releasers of hormones such as oxytocin and dopamine, which could in turn reinforce social bonds during this period (Insel 2003). The relationship between oxytocin and dopamine might be especially relevant to social bonding if the postpartum period were accompanied by arousal followed by a sense of emotional safety. As in other mammals, human lactation and perhaps even the hormonal milieu associated with birth has, at least in theory, the potential to be emotionally protective and perhaps to predispose the mother to form a bond with her infant.

What Mechanisms Exist to Permit Fathers or Other Caretakers to Form Social Bonds with Children?

The capacity to form strong social bonds is not limited to mothers and infants. As described by Hrdy (this volume) and Keverne (this volume), the historical tendency of humans and other primates to show patterns of communal rearing of offspring would demand mechanisms to support social bonds between nonmaternal caretakers and infants. Because the hormones associated with social bonding are not unique to birth or lactation, we can postulate that the same or related hormones that are associated with maternal attachment might have the potential to encourage the formation of social bonds between children and their nonmaternal caretakers, including fathers, adoptive parents, and grandparents. For example, although less well-studied, the hormonal changes experienced by fathers may be in partial synchrony with their domestic partners (Storey et al. 2000; Fleming, this volume). Furthermore, as suggested by studies of pair bonding in animals, it is possible that a uniquely masculine hormonal cocktail, including centrally active vasopressin, predisposes the prospective father or other male caretaker to form social bonds with offspring (De Vries and Villalba 1997). Included in the features of a paternal bond may be a particular need for a father to attempt to protect his own children or acquire the resources necessary to support his family. The role of testosterone in male social attachment has not been well studied even in animal models. Testosterone does produce relatively long-lasting increases in vasopressin in areas of the nervous system, such as the amygdala, which may be of importance to the capacity of parents to respond appropriately to stimuli from an infant. Furthermore, transient declines in androgens may accompany the birth of a child or perhaps exposure to stimuli from an

infant (Storey et al. 2000). Androgens can interfere in some cases with the release or actions of oxytocin, and it is possible that temporarily lower levels of sex steroids around the time of birth could be adaptive, permitting males as well as females to experience the behavioral consequences of oxytocin. Whether oxytocin plays a role in paternal behavior remains to be determined, but the capacity of oxytocin and vasopressin to influence each other's receptors provides yet another possible explanation for the tendency of fathers or other males who are exposed to children to develop strong emotional attachments.

SUMMARY

The nervous system of modern humans is constructed from ancient neural and endocrine components that are shared among mammals. The patterns of emotion and behavior that humans call "social bonds" emerge from neurobiological roots. Even rather unsophisticated creatures, such as prairie voles, can develop lifelong social relationships with properties apparently similar to human social attachments. We now recognize that behavior is sculpted by genetic and epigenetic processes producing differences that are labeled as "species," "gender," and "individuality." Knowledge of the origins and properties of these systems does not totally explain social behavior. However, this knowledge does give us an exciting new perspective with far-reaching implications for understanding this essential aspect of human nature.

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