

# Neural Consequences of Infant Attachment

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## Abstract

Typical studies of the impact of the quality and presence of attachment relationships on child development have focused on the child's safe-base behavior. In terms of neurobiology, this has primarily led to investigations of the child's control over negative affect. In nonhuman primates, early investigations into the neurobiological consequences of attachment used models where attachment relationships were absent or severely curtailed. Institutionalization of infants, a common practice, mirrors these early primate studies since attachment relationships are limited or absent. These investigations are based on models of disruptions in attachment and used here to illustrate the impact of attachment relationships on two neural systems not typically considered: the neural substrates of reward learning and the neural substrates supporting complex cognitive function such as executive function. While attachment is central to the development of negative affect regulation, it is argued that the context in which the brain develops can also serve as an additional focus of early attachment relationships. This offers insight into the multiple functions served by attachment, and thus the role it plays in the development of other neural systems.

## Introduction

In this chapter, we review evidence of the neurodevelopmental consequences of attachment. We discuss the neural mechanisms that support the emotionally positive bond between caregivers and infants, and the neural mechanisms which support the regulation of infant distress. We suggest that these two components act together in support of the homeostatic functioning of the secure base phenomenon. According to traditional attachment theory, this allows the infant to explore while in the presence of the attachment figure, and to seek safety in close contact with the caregiver when distressed (Ainsworth

1985). The emotional bond and the regulation of negative emotion functions develop from birth. Thus, attachment relationships are a constant source of experiences which are likely to shape neurodevelopment during infancy, i.e., periods of peak developmental plasticity (Greenough et al. 1987; Fox et al. 2010; Nelson and Sheridan 2011). Attachment theory focuses primarily on one function of attachment (i.e., regulation of distress). We propose that the neural correlates of attachment also include positive emotional bonds, which undoubtedly are present earlier in development and influence neurodevelopmental outcomes. These attributes of attachment relationships give them the potential to influence neural development profoundly and thus impact developmental outcomes.

Much has been written about the importance of attachment relationships for many developmental outcomes but, to date, relatively little is known about the impact of attachment relationships on neural development. Moreover, this relative paucity of information neglects due consideration of cross-cultural perspectives of attachment, including attachment as it naturally occurs in well-functioning environments. Much of the information we have about the impact of attachment on neural development relies on studies of individuals raised in grossly impoverished settings and resultant groups of people with dysfunctional attachments. This means that much of our knowledge is about neural consequences of the lack of an adequately or well-functioning attachment system. Secondly, most of our knowledge about attachment comes from cultural settings (western, educated, industrialized, rich and democratic settings, or WEIRD; Henrich et al. 2010) where monotropic attachment is the stated norm. While Western readers may find this characterization of attachment relationships familiar, numerous sources indicate that in other settings, attachment networks comprised of multiple attachments (between the infant and a number of important caregivers) are normative (see Keller and Chaudhary as well as Morelli et al., this volume). After reviewing the extant literature, we will advance some suggestions on how current findings might apply to infants with fully functioning attachment systems, including those with an attachment network, rather than a monotropic attachment.

In addition to the issues regarding the sample, there are other issues to consider in the ascertainment of neural substrates and neural pathways of attachment. We distinguish attachment from general mother-infant bonding, and thus exclude much of the rodent work that focuses on the neural mechanisms involved in the basic mammalian mother-infant bond (e.g., Moriceau and Sullivan 2005). As many chapters in this volume attest, attachment differs from and is much more than the initial emotional bond. Most definitions focus on the function served by attachment figures, such as aids for the regulation of negative emotions or for general psychobiological regulation, although new proposals also include aids for privileged access to the social world (see Chapter 8, this volume).

Importantly, relatively little is known about the neural consequences of variations in attachment (e.g., individual differences in security). In part, this is because such individual differences would need to be profound and permanent, and need to be found in well-studied and understood neural systems in order to be observable using current neuroimaging methods (but see Serra et al. 2015). Interestingly, individual differences in maternal caregiving do appear to be accompanied by differences in neural functioning. For example, maternal styles of high interactive responsivity with three- to five-month-old infants (vs. a maternal style of high intrusiveness) were related to significant differences in neural function in support of reward and stress-related action (Atzil et al. 2011). We do not know how maternal styles map onto individual differences in attachment. Using neuroimaging techniques such as MRI with infants is difficult, primarily because of their inability to stay still, lie by themselves in a scanner while awake, or follow directions. To address these difficulties, infants only participate in MRI or fMRI studies while asleep or sedated. Thus, neuroscientists tend to rely on studying the neural substrates of attachment that result from profound disruptions of attachment relationships, such as the absence of any primary caregivers, the presence of maltreatment (neglect or abuse), or disrupted caregiving.

The use of these indirect measures to assess the impact of attachment on neurodevelopment is justified because we know that these adverse early experiences often result in disorders of attachment, including reactive attachment disorder and disinhibited social engagement disorder, or indiscriminate friendliness (Zeanah and Gleason 2015). Reactive attachment disorder is a disorder of attachment characterized by a lack of developmentally appropriate attachment behaviors, including the failure to seek comfort from a caregiver. This disorder is commonly accompanied by disruptions both in emotion regulation and positive affect. Disinhibited social engagement disorder is a disorder characterized by a lack of specificity in attachment behaviors, which includes children exhibiting overly familiar or intimate behaviors with unfamiliar adults. The tight links between severe disruptions in early caregiving environments and disorders of attachment allow neuroscientists to study neurodevelopment in these populations, to gain insight into neural substrates of attachment.

It is not enough to ask whether variation in attachment impacts neural development. We must also investigate the ways in which variation in attachment shapes neural development. Neuroscientists wish to identify the pathways and developmental processes through which attachment, and variations in attachment, impact neural structure and function. Delineating these processes is useful in part to further our knowledge, but also to facilitate the creation of targets for both remedial interventions and prevention of negative outcomes. Ultimately, as a consequence of identifying these targets for intervention and prevention, all children should be able to experience the benefits of satisfying, early caregiving relationships.

In this chapter, we review the evidence that describes the processes and pathways by which severely disrupted attachment relationships impact neurodevelopment. We explore the implications of these findings for understanding the neural foundations for well-functioning attachment, hint at what might be found in studies across cultural contexts, and present our ideas for future directions in the neuroscience of attachment.

### **Institutional Care: A Model of Disruptions in Early Attachment**

Some aspects of the importance of attachment relationships to neural development may be studied by examining instances where children have no primary caregivers as a result of growing up in settings of institutional care. Institutional care, as we use the term, refers to very poor quality care as a function of rotating and overworked caregivers and a high ratio of infants to caregivers. Institutional caregivers are neither consistently present nor frequently able (or willing) to interact positively with infants in activities unrelated to health concerns. Such disrupted caregiving creates a context in which children are unable to form close relationships with any specific adult figure, or even a set of adult figures, and where caregivers are not able (or willing) to form a special bond with a specific child. Although the physical environment of most orphanages is most decidedly not stimulating, it is not consistently unsafe, lacking in nutrition, or without access to medical care. Thus, researchers have concluded that psychosocial deprivation and thus poor or absent early caregiving is the primary adversity to which these children are exposed (McCall et al. 2016). The poor quality of caregiving is measurable both in frequency and quality; in institutionalized settings, significantly fewer child-caretaker interactions occur and these are significantly lower in quality compared to interactions within families (Smyke et al. 2007). Additionally, quality of attachment is higher in children who live outside institutions (Smyke et al. 2010). Although it would be difficult to support the idea that all neural deficits found in children from institutionalized settings can be attributed to a lack of attachment figures, we can support the claims that institutionalized settings are primarily characterized by psychosocial deprivation for infants and young children, in the absence of other forms of adversity (e.g., inadequate nutrition, poor medical care, physical and sexual abuse).

Beginning as early as 1975, many studies have examined the impact of institutionalization on child development. Tizard and Rees (1975), for example, studied children from London orphanages. An issue that arises in studying eventual outcomes is that the reason why any infant has been placed into the institution is often not known. Some infants are placed in orphanages due to illness, failure to thrive, or other perceived deficiencies, whereas others are placed for political reasons. The Bucharest Early Intervention Project (BEIP)

is a longitudinal study of a sample of children raised from early infancy in institutions in Bucharest, Romania. BEIP was initiated at the request of the Secretary of State for Child Protection in Romania. Its major advantage over other studies of children from orphanages is that assignment to foster care, as an alternative to institutional rearing, was randomized. Thus, these outcomes are most precisely related to the interventions since interventions were randomly assigned. All study procedures were approved by the local commissions on child protection in Bucharest, the Romanian Ministry of Health, and the institutional review boards of the home institutions of the three principal investigators (Zeanah et al. 2006; Miller 2009). Studies using BEIP, therefore, provide the best available evidence for a causal relationship between lack of an attachment figure during infancy and early toddlerhood and disrupted neurodevelopment.

In BEIP, a sample of 136 children (aged 6–30 months) was recruited from each of the six institutions for young children in Bucharest. An age-matched sample of 72 community-reared children was recruited from pediatric clinics in Bucharest and comprised the never-institutionalized group. Half of the children initially raised in institutional care in Bucharest, Romania, were randomly assigned to high-quality foster care (Smyke et al. 2009) with a primary caregiver. The other half was assigned to care as usual in the institution, with several infrequently available and rotating caregivers and no primary attachment figure (Smyke et al. 2007).

Given the circumstances in which these children were raised, it is not surprising that the BEIP has provided clear evidence for disrupted attachment relationships resulting from institutional care. Children in the foster care group and care-as-usual institutionalized group exhibited increased rates of reactive attachment disorders and indiscriminate friendliness compared to the never-institutionalized group (Zeanah et al. 2005; Gleason et al. 2014). However, placement into foster care earlier than 24 months of age decreased rates of attachment disorder in the foster care group compared to the care-as-usual institutionalized group (Smyke et al. 2010), indicating that the compromised early care in institutions led to the observed disruptions in attachment. In sum, early postnatal exposure to institutionalization can be used to model the impact of a lack of early attachment on neurodevelopment.

### **Neurocognitive Impact of Disruptions in Early Caregiving**

Several neurocognitive domains have been identified as being susceptible to disruptions in early caregiving. These include the neural bases for emotion expression (both positive and negative), emotion regulation, and neural substrates across multiple cognitive domains. In this section we review findings

and explore implications of extending our knowledge about the neural correlates of disruptions attachment across these various domains.

### **Lack of Early Caregiving Relationships Disrupts Emotional Control over Negative Affect**

There is robust evidence that early exposure to institutional care causes disruptions in (a) emotional reactivity and (b) control over negative affect. The lack of an attachment relationship in infancy and early toddlerhood, caused by institutionalization, is strongly related to pathological disruptions in negative affect (Zeanah et al. 2009) and leads to elevated rates of depression and anxiety. In addition, this exposure is related to the development of neural systems which support emotion regulation and reactivity. Specifically, lack of early attachment relationships that result from institutionalization has been linked with medial prefrontal cortex function, amygdala volume and reactivity, as well as the quality and extent of connectivity between the amygdala and medial prefrontal control regions (Mehta et al. 2009a; Tottenham et al. 2010, 2011). Relatedly, early exposure to institutionalization causes a blunted stress response to interpersonal stress and rejection. In BEIP, children from the care-as-usual institutionalized group, compared to foster care and never-institutionalized children, showed blunted sympathetic and hypothalamic-pituitary-adrenal (HPA) axis responses to a laboratory Trier social stress test and a social rejection paradigm (McLaughlin et al. 2015). It has been proposed that the lack of early attachment figures may speed neurodevelopment of negative affect regulatory systems, shifting limbic/prefrontal connectivity so that the neural correlates of emotional reactivity and control over emotional responses look more “adult like” in children exposed to institutionalization at younger ages (Ganzel et al. 2013; Gee et al. 2013; Tottenham 2014). Supporting this theoretical model, evidence from BEIP indicates that disruptions in attachment relationships moderate the impact of institutionalization on pathological disruptions in regulation of negative affect. Specifically, for children in the BEIP study who were randomly assigned to foster care, improvements in their attachment relationships (self-reported and reported by their foster parent) moderated the association between institutionalization and psychopathology (McLaughlin et al. 2011; Humphreys et al. 2015).

### **How Does Lack of Early Attachment Lead to Disruptions in Control over Negative Affect?**

Early in development, young infants lack the ability to regulate their own emotions, and attachment relationships play a role in providing emotion regulation. Attachment relationships also play a role in scaffolding their developing abilities to regulate emotion, especially negative emotion (Ainsworth 1985;

Morton and Browne 1998; Zeanah and Gleason 2015; Chapters 6 and 8, this volume). Indeed, one of the core criteria of secure attachments, as assessed in the Strange Situation Procedure, is seeking proximity in the face of stress and maintaining contact with the attachment figure(s) until the distress is resolved (i.e., securely attached children use primary caregivers as a source of emotion regulation). Even in cultures in which infants have multiple attachment figures, each caregiver engages with distressed infants and contact is maintained until crying and fussing are successfully reduced (e.g., Meehan and Hawks 2013). In some cultures (e.g., when infants are often in cradles or slings), this regulatory function of attachment appears to be so well developed that infants rarely appear distressed (Gaskins 2013).

Given this central role of attachment figures in modulating negative emotions during infancy, it is perhaps not surprising that infants who have not experienced a primary attachment relationship have deficits in emotion regulation, which later results in psychopathology as children and adolescents. Infancy is a period of peak neural plasticity and is thus highly responsive to environmental inputs (Fox et al. 2010). The neural and physiological systems that regulate negative affect are likely “tuned” by early caregiving experiences. If infants do not have external sources of emotion regulation to calm them during infancy, these systems may develop in aberrant ways. For example, infants may be in a constant state of distress with an inability to downregulate their distress, or infants may develop a systematic unresponsiveness to distress which may develop as a “too mature” response, as described above. Conceptually, infants could be thought to be spending much of their time in a context of danger. In this way, the lack of an attachment figure in infancy and early toddlerhood is an environmental signal that lets the developing neural system “know” what the future is likely to hold. The resultant mismatch between the early developmental context with no attachment relationships and future experiences of relatively safe environments in middle childhood and adolescence may affect emotion regulation and stress physiology in ways that can be understood as pathological.

### **Lack of Early Caregiving Relationships Disrupts Processing of Positive Affect**

In the English and Romanian Adoptees study (Mehta et al. 2009b), exposure to institutionalization during early childhood was found to be associated with blunted striatal activation during reward anticipation. Reward anticipation is measured using neuroimaging during the monetary incentive delay task. This task links a previously neutral stimulus (e.g., a circle) with reward (e.g., winning money) by iteratively pairing responses to this stimulus with a reward over time (Knutson et al. 2001). Neural activation in response to reward anticipation is indexed by measuring responses in the brain to the stimulus which predicts reward, before any reward has been administered. This finding

suggests that disruptions in early caregiving may affect neural circuitry involved in reward processing and in learning to anticipate reward. In typically developing children and adolescents, reward anticipation is associated with increased activation of the ventral striatum, ventral medial prefrontal cortex, and dorsal anterior cingulate cortex (Knutson et al. 2003; Haber and Knutson 2010). The ventral striatum is activated during reward receipt, reward learning, and processing of secondary reward stimuli (e.g., happy faces) across numerous studies in humans and animals (for reviews, see Schultz et al. 1997; Haber and Knutson 2010). Similar to findings from the English and Romanian Adoptees study, neural activation was assessed using fMRI while adolescents and children viewed happy and fearful faces in another sample of participants exposed to institutionalization during infancy. In this study, when happy faces were viewed, the ventral striatum was less activated in adolescents exposed to institutionalization early in life compared with age-matched controls (Goff et al. 2013). The degree of ventral striatal activation in this study was also associated with symptoms of depression. Finally, in BEIP, adolescents in the care-as-usual group showed a reduced behavioral response to monetary reward, relative to adolescents in the foster care group and never-institutionalized group in the monetary incentive delay task. This suggests that it is the lack of an attachment relationship in childhood—a species-expected caregiving experience—that leads to a disruption in reward processing (Sheridan et al., under review).

Other forms of disrupted early caregiving (including emotional neglect, cumulative adversity, and maltreatment) have also been linked with blunted neural and behavioral responses to reward as measured by the monetary incentive delay task and similar computerized tasks (Guyer et al. 2006; Dillon et al. 2009; Pechtel and Pizzagalli 2013; Hanson et al. 2015a, b). Taken together, existing evidence indicates that disruptions in early caregiving may confer risk for dysfunction in basic learning mechanisms around reward which ultimately support healthy mood function.

### **How Does Lack of Early Attachment Lead to Disruptions in Control over Positive Affect?**

Currently there is no consensus as to the mechanism underlying the association between early caregiving and reward processing (for reviews, see Pechtel and Pizzagalli 2011; Goff and Tottenham 2015). Considering aspects of attachment relationships may, however, shed some light on potential mechanisms. As we mentioned in the introduction, attachment relationships appear to have at least two core properties: secure base and positive emotional bonds. While the “safe base” behavior is the defining feature of attachment relationships, as measured in the Strange Situation Procedure, the positive emotional bond is a defining feature in more naturalistic contexts, and may be the core emotional aspect of the developing system of trust (Keller 2013a; Gaskins 2014). Attachment

relationships play several roles in early child development. In addition to providing a source of regulation over negative emotions, the attachment figure also provides initial instances of reward learning. In WEIRD settings, a primary task for a child is to elicit caretaking behaviors, such as provision of food and comfort, through the use of vocalizations and behaviors. An attuned caregiver will use infant's hunger signals to guide their behavior to provide food. Through this process, the child of an attuned caregiver will learn that some behaviors will elicit reward (e.g., food) and will learn to perform these more readily, particularly when hungry. This is just one example, in particular cultural settings, of the manner in which infant behaviors with a caregiver may be linked with reward. In other cultures where constant physical contact is the norm, there is a more immediate pairing of the provision of comfort and food with attachment figures. In the case of severely disrupted or absent attachment in early childhood, it is likely that the infant has requested and received fewer rewards and less clear learning opportunities, because fewer rewards and interaction opportunities were available from which to learn. If reward (e.g., food, comforting, play) occurs randomly with respect to their behavior, infants will not form a strong association between their attachment figures and positive reinforcement. For the institutional infant, it is possible that the neural circuits which underlie reward learning (connectivity between dopamine-rich sites in the prefrontal cortex and ventral striatum) will have less "practice" during this period of peak developmental plasticity, thus resulting in the observed long-term disruptions in both reward learning and activation of the ventral striatum in the context of reward.

### **Lack of Early Caregiving Relationships Results in Global Cognitive Deficits**

Children exposed to institutional care in early childhood exhibit clear neurocognitive deficits. Exposure to institutional care shows reductions in IQ, which can partially be remediated following randomization to foster care (Nelson et al. 2007). This suggests that the presence of an attached early caregiver is important for intellectual development. Exposure to institutional care is also associated with general cognitive deficits, including deficits in executive function, which is defined as the ability to hold in mind rules and ideas no longer present in the environment and to inhibit immediate responses (Bos et al. 2009; Beckett et al. 2010). Relatedly, disrupted early caregiving following institutionalization is associated with disruptions in attention and impulsivity (Zeanah et al. 2009), which is the behavioral manifestation of poor executive function (Tibu et al. 2016). Finally, early institutional care is associated with global reductions in cortical volume, neural function, and cortical thickness across studies (Chugani et al. 2001; Vanderwert et al. 2010; Sheridan et al. 2012a). Importantly, these general reductions in cortical thickness and neural function statistically mediate the association between institutionalization

and behavioral manifestations of attention and impulsivity, indicating that it is because of the reduced thickness that attention and impulsivity are elevated (McLaughlin et al. 2010, 2013).

### **How Does Lack of Early Attachment Lead to Disruptions in Global Cognitive Function?**

Elsewhere, it has been argued that this impact of institutionalization on cognitive development results from deprivation in rich cognitive stimulation during early childhood (McLaughlin et al. 2014; Sheridan and McLaughlin 2014). Studies of rodents show that a lack of cognitive stimulation will increase synaptic pruning processes, and thus rodents exposed to very low levels of cognitive stimulation show overall reductions in cortical volume (Diamond et al. 1972). Here we posit that because attachment relationships are the primary source of stimulation in infancy and early toddlerhood, the lack of these relationships are likely to result in an unstimulating environment, which also increases synaptic pruning globally throughout the brain. Pruning is the mechanism by which many environmental childhood experiences (e.g., phonemic retention in the context of multiple language exposure, visual cortex organization) impact neural development (Wiesel and Hubel 1965; Hensch 2005; Morishita and Hensch 2008). The general reductions in cortical thickness and volume observed following institutionalization are likely to yield deficits in higher-order cognitive functions because these functions require coordinated activation of multiple areas of association cortex (e.g., prefrontal and parietal cortex) and rely on late-developing areas of the brain such as the prefrontal cortex.

The context of attachment relationships is the first in which a child can expect to receive cognitive stimulation. In the Western caregiving systems, this is through contingent vocalization and face-to-face play. In other cultures, social, visual, and vestibular stimulation can occur via exposure to multiple sensory and social environments experienced through carrying. In Mayan cultures, for example, the infant might be held by an older child, and together they may sit next to the mother, surrounded by numerous others, while the mother engages in social exchange and prepares food (Morelli et al., this volume). When attachment relationships are disrupted or absent, this cognitive input is grossly reduced. As a result, the typical developmental process of synaptic pruning, which creates the most efficient neural system possible given particular early inputs, may prune connections relating attachment figures with rewarding events in the world. Given that the institutional setting has a reduced sensory environment and a lack of an early attachment figure, the pruning process may tune brain function with this impoverished setting (Greenough et al. 1987; Fox et al. 2010). Unfortunately, large differences are still observable as late as 8 years of age in children raised in institutions versus those raised in families, indicating that these processes may be difficult to reverse (Sheridan et al. 2012a).

## **Multiple Roles of the Early Attachment Relationship in Shaping Development**

Currently the most common manner of assessing attachment style is through the Strange Situation Procedure. In this experimental context, infants use their primary caregiver as a source of regulation over emotions generated by the novelty of the unfamiliar setting and stranger, as well as by separation. Usually this is interpreted as fear, or wariness elicited by novelty, and distress elicited by separation from the attachment figure. The Strange Situation was meant to mimic everyday situations as well as to represent a potentially dangerous situation—being left alone in an unfamiliar environment (Ainsworth 1985). Although this approach has been valuable in linking quality of attachment to child development outcomes in Western or urban cultures, it has focused attention primarily on regulation of negative emotion as outcomes. Here we review literature which documents that a lack of early attachment results in disruptions in neural development, in part in neural systems that support emotion regulation (e.g., the amygdala and ventral medial prefrontal cortex). These types of disruptions in neural development of emotion regulation systems are consistent with our understanding that early caregiving is important in shaping infant learning about the regulation of negative affect.

There are, however, two additional prominent neurocognitive deficits that result from institutional care: problems associated with reward learning and global deficits in cognitive function. These difficulties, which result from a lack of attachment in early childhood, involve neural structures and functions different from those that account for regulation of emotion. Specifically, reward response and anticipation are supported by the functioning of the striatum and dorsal medial prefrontal cortex, whereas complex cognitive function is supported by coordinated activity across association cortex, including lateral prefrontal and superior parietal cortex. In addition, aspects of attachment relationships with a primary caregiver which likely support development of these neural and cognitive functions are proposed here. Evidence from studies of institutionalization indicate that the lack of an early attachment relationship results in severe deficits in cognitive and emotional function, as well as in the neural structures that underlie these functions. The various impacts of disrupted caregiving likely transpire through multiple pathways, representing the multiple important functions of the early caregiving relationship (for further discussion of the functions of attachment, viewed from cross-cultural and cross-species perspectives, see Chapter 8, this volume).

## **Future Directions**

In this review, we have focused on extreme exposures characterized by an almost complete absence of attachment relationships. In at least one study,

however, randomly assigned interventions allowed causal inference (Zeanah et al. 2003; Nelson et al. 2007). Unfortunately, access to natural or actual experiments in rearing is limited in human studies. Most investigations of early exposures and subsequent outcomes in humans are correlational in nature and rely on naturally occurring variation in rearing environments (e.g., neglect, abuse). Because natural variation of this kind is unfortunately common in humans, these types of studies are easier to perform and should be viewed as important complements to studies with actual exogenous variation in attachment environments. For example, correlational studies could more carefully delineate and describe observations made in studies with random assignment. Equally important, the number of studies in humans where random assignment is used should increase. While it is unethical to assign children randomly to negative early environments, treatment studies where children are randomly assigned to treatments in which these environments are ameliorated (e.g., parenting interventions) are possible and increasingly common (e.g., Bernard et al. 2012; Caron et al. 2016). Unfortunately, evaluations of these interventions are unlikely to focus on the neural functioning of the child. Future work that evaluates the neurobiological consequences of early parenting interventions could contribute meaningfully to our understanding of the impact of early experience on neural structure and function.

In addition, there is a large body of work in nonhuman primates which can address gaps in current understanding of the effects of early rearing deficits on neurobiology due to a lack of experimental evidence. Indeed, since the very early days of attachment research, primate models have been informative (Suomi et al. 2008). This research often mirrors and further develops the work reviewed above, as primate models of attachment disruptions generally involve random assignment to peer or nursery rearing: a total lack of maternal care. There are many similarities in the downstream consequences of peer rearing and institutionalization. For example, both forms of early maternal deprivation disrupt stress physiology and regulation (Dettmer et al. 2012; McLaughlin et al. 2015). However, the impact of these two experiences on neural structure may differ; indeed, they may go in opposite directions (Spinelli et al. 2009; Sheridan et al. 2012a). The diversity of findings between humans and primates exposed to superficially similar experiences points to the importance of carefully considering species-specific effects and exact nature of the exposures. Importantly, studies in nonhuman primates, with greater access to random assignment, can be used to test specific theories from the human literature about the effect of variation in early caregiving on long-term neurobiological outcomes. Increased collaboration between researchers investigating early rearing exposures in animal and human models has the potential to accelerate our understanding of the impact of attachment disruption on the developing brain.

Finally, we wish to stress the importance of expanding consideration of attachment beyond the mother-infant dyad to include attachment networks (Keller and Chaudhary, this volume). In addition, the proposed functions of

attachment need to expand beyond the regulation of negative emotion to include introduction to the world (Chapter 8, this volume). Our consideration of the neural consequences of attachment, or lack thereof, provides strong support for the involvement of two neural mechanisms: one that underpins regulation of negative emotion and another that underpins reward anticipation and reward response. Although most of the evidence is conceptualized from a framework of monotropic attachment, we have purposively indicated that the evidence equally supports the interpretation that an infant's neural substrate "expects" experiences which involve both multiple functions of attachment and an attachment network, in the sense of experience-expectant and experience-dependent processes (Greenough et al. 1987) and probabilistic epigenesis (Gottlieb 2007). Further research is needed (e.g., new treatments for institutionalized children) to test the neural foundations and consequences of attachment, as reconceptualized through this volume.