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Neurobiology of infant attachment: Attachment despite adversity and parental programming of emotionality

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Abstract

We review recent findings related to the neurobiology of infant attachment, emphasizing the role of parenting quality in attachment formation and emotional development. Current findings suggest that the development of brain structures important for emotional expression and regulation (amygdala, prefrontal cortex, hippocampus) is deeply associated with the quality of care received in infancy, with sensitive caregiving providing regulation vital for programming these structures, ultimately shaping the development of emotion into adulthood. Evidence indicates that without sensitive caregiving, infants fail to develop mechanisms needed for later-life emotion and emotion regulation. Research suggests that a sensitive period exists in early life for parental shaping of emotional development, although further cross-species research is needed to discern its age limits, and thus inform interventions.

Introduction

Attachment, which is defined as the selective and enduring bond between individuals, occurs throughout the lifespan [1], encompassing both infant-caregiver attachment and adult romantic attachment. While substantial research has begun documenting the neurobiology of attachment, it has primarily focused on adult romantic attachment and adult attachment to their offspring [2,3,4]. However, more recent research is exploring the neurobiology of infant attachment to the caregiver. Within the context of infant-caregiver attachment, the term “attachment” has traditionally been used to describe a complex and highly specific bond an infant forms to their caregiver by 1 year of age [1]. However, newborns display highly specialized behaviors which can be characterized as bonding behaviors important for attachment formation [5], which we will discuss here.

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In this state of the art review, we review human and animal model research published within the past 5 years that advances our understanding of the neurobiology of infant attachment formation and the unique role of the primary caregiver in guiding attachment and infant emotional development. Recent evidence supports a working model of early-life parental shaping of lifelong emotional development, with quality of care greatly affecting emotionality and emotion regulation throughout the life course (Figure 1). Continued cross-species research will further our understanding of the mechanisms by which parenting quality in early life programs brain structures underlying lifelong emotionality.

Body

The Neurobiology of Infant Attachment Formation

Altricial species are not quickly mobile after birth, and rely on adults for care and nourishment. Thus, infants of altricial species, such as humans, rely on attaching to a caregiver for survival. Historically, scientists have questioned whether infant attachment is formed via biologically innate mechanisms or experience-dependent processes. To date, very little is known about the neurobiology of attachment in human infants, due to technical and ethical limitations that researchers face when working with babies. However, recent research in animal models supports a theoretical model in which the mammalian infant brain is innately biologically-predisposed to form attachments, but depends on necessary experiential input and infant learning to guide attachment formation, similar to “imprinting” which occurs in avian species [6].

Specifically, infant rat pups possess unique neurobiological mechanisms that promote preference learning and block aversion learning in order to support attachment. This specialized attachment circuit involves a hyper-functioning locus coeruleus releasing high levels of norepinephrine, and a hypo-functioning hypothalamus-pituitary-adrenal (HPA) stress axis (for review see [7]). When this circuit is activated by external somatosensory stimuli, such as stimuli naturally provided by the mother, pups (born blind and deaf) learn to prefer any olfactory stimulus paired with this stimulation during their first 10 days of life. Such preference learning occurs regardless if the stimulation is pleasurable or aversive [7]. Although seemingly paradoxical, the system has presumably evolved to promote infant attachment to a caregiver, and thus survival, regardless of the quality of care [8]. While this animal model has identified brain circuits critical for attachment formation in rodents, it is currently unclear if the mechanisms supporting attachment formation in human infants are the same. However, norepinephrine levels are very high at the time of birth and are critical to attachment formation across numerous species [9], and human children show decreased stress reactivity in early childhood in the presence of a caregiver [10]. Furthermore, it is noteworthy that Bowlby’s original description of attachment was based on animal models, providing a strong foundation for the use of animal models to further our understanding of the neurobiology of infant attachment [1].

Attachment formation begins in the womb, where infants form preferences for maternal cues, including her odor and voice, with continued learning occurring after birth (for review see [5]). Additionally, the experience-driven neurobiological mechanisms supporting attachment formation allow the infant to bond with multiple caregivers once outside of the

womb. A recent publication has highlighted the extent that attachment formation is experience-driven [11]. Perry et al. altered the smell of rat mothers' natural odor via manipulation of their diet. Maternal odor was manipulated because it is critical for pup survival; pups require the maternal odor to orient to the mother, behave socially with the mother, and nipple attach for nursing. Two weeks of rearing with these "newly-scented" mothers produced pups that showed attachment behaviors to the new maternal odor and a loss of value of the original natural maternal odor, as indicated by pups' failure to approach or nipple attach to the mother with the natural maternal odor. These behavioral changes were paired with drastic differences in infant brain processing of the natural maternal odor, following the dissociation of this odor from caregiving. In a second experiment, the researchers reared infant rat pups with both their mother and father to test whether or not pups also displayed attachment to their father. Pups reared in these conditions showed similar approach levels to their mothers' and fathers' natural odors, indicating high odor-preference learning for both. Furthermore, the father's odor induced a neural signature similar to that of the maternal odor, suggesting that infant experiences with their fathers as a co-caregiver elicited infant attachment in a similar way to mothers. Together these experiments support a neurobiological basis for plasticity within the attachment system, as well as attachment formation to multiple caregivers.

Attachment Despite Adversity

The attachment system serves the infant the immediate benefit of promoting bonding to a caregiver, and thus survival, at this vulnerable point in development. However, since the system allows attachment to caregivers regardless of the quality of parental care received, for some infants (especially those facing adverse environments) this comes at a cost [8]. Indeed, parental care has great control over the environmental and experiential impact on the developing infant, which is due to the unique and powerful control that primary caregivers have on shaping infant development [12].

Recent research in humans suggests that the quality of parental care is critical to infant emotional development, due in part to their pronounced ability to regulate infant behavior and physiology [13,14]. For example, parental presence regulates stress hormones [15,16] and brain activity in children [17,18], but not adolescents [18,19]. While few human researchers have studied brain activity in infancy during caregiver-infant interactions, the evidence thus far further supports parental regulation of the developing infant brain [20,21]. Since it is hard to directly assess what is going on in a human infant brain, clues from animal research are helping us discover how parents regulate the infant brain. A seminal study by Sarro et al. displayed for the first time that infant rat brain activity is directly influenced by interactions with the mother in their natural nest (via, in part, to a noradrenergic neurotransmitter mechanism), with the magnitude of maternal regulation decreasing with age [22].

We propose that parental regulation of infant physiology in early life, a time of heightened and rapid brain development, is critical for the programming of circuitry underlying emotion. When the infant bonds to a caregiver that provides low quality of care, however, the lack of regulation and expected species-specific experiences in early life enduringly

disrupts brain areas underlying emotion (Figure 1). Furthermore, we propose that parental regulation of the infant is tightly linked to the patterning and quality of parental care the infant receives. Indeed, parental control of infant physiology decreases as the quality of parenting decreases (i.e. intrusiveness, unpredictability, neglect) [23,24]. We draw further evidence from studies showing that stressful conditions within the home place parents at risk for becoming less sensitive caregivers [25,26], which mediates many adverse child outcomes related to emotion regulation and behavioral problems [27–29]. These enduring outcomes are associated with altered HPA-axis activity [30,31], vagal withdrawal [32], and connectivity of brain areas important for emotion and emotion regulation [33–35]. Research with animal models is providing additional mechanistic insight into how early-life parenting quality enduringly alters brain areas supporting emotion [36–39]. One such model introduces adversity to the attachment system by exposing rodent mothers and pups to a scarce resource environment, which produces an increase in negative maltreatment caregiving behaviors. This model has identified the amygdala as being particularly vulnerable to effects of caregiving quality, as indicated by an increase in depressive-like symptoms and antisocial behaviors in adult offspring who experienced negative caregiving, as well as altered fear-related behaviors, via an amygdala-dependent mechanism [8,40,41] involving decreased amygdala-prefrontal cortex functional connectivity [42]. These infant rodent results mirror altered amygdala-prefrontal cortex connectivity found in orphanage reared human children and nonhuman primates reared with maltreating caregivers [33,43].

Researchers are now exploring what specific aspects of sensitive caregiving promote optimal emotional development in humans, and have identified two main elements of sensitive caregiving that provide the greatest benefits, even in adverse environments: nurturance to the infant, such as sensitivity following a distressful event [44,45], and synchrony, such as caregiver responsiveness to a child's bid [15,46]. These aspects of sensitive caregiving are associated with many behavioral and physiological outcomes, with parental regulation of infant physiology hypothesized as a mediator [47]. Animal models of caregiver nurturance and caregiver-infant synchrony are needed to better understand the mechanism by which these caregiver styles in early life promote optimal emotional development throughout the lifespan. However, insight can be drawn from existing rodent and primate studies related to mother-infant social buffering [23,48], and social learning [49], which provide evidence of strong maternal regulation of emotional states, emotional learning, and the associated underlying physiology in early life.

Infant attachment to the caregiver occurs regardless of the quality of care received. However, both caregiver quality and the quality of the rearing environment impact the caregiver's ability to regulate their infant's brain and physiology, ultimately determining the quality of the parent-infant attachment, and the infant's emotional development throughout the lifespan. Importantly, sensitive caregiving, such as nurturing and synchronous interactions with the infant, can buffer the effects of adverse environments on infant outcome, making sensitive caregiving an important target for interventions for at-risk families. Lastly, a sensitive period for parental shaping of emotional development occurs in early life and is thought to coincide with strong maternal regulation of infant behavior and physiology for the programming of emotion circuitry. Caregiver regulation of offspring persists into childhood

(as a function of attachment quality), although it wanes as offspring approach adolescence, and transition to independence.

Sensitive Periods of Emotional Development

Sensitive periods are conceptualized as developmental windows during which a system displays high plasticity and vulnerability to shaping and attunement by environmental input [50]. A growing body of literature suggests that there is a sensitive period for when parenting can influence systems underlying emotional development of their offspring, although the exact time points of this sensitive period remain to be determined (for review see [14,51]).

We argue that this sensitive period may be intimately linked to the caregiver's ability to profoundly regulate infant physiology during typical caregiver-infant interactions [22], and in the presence of stressors by buffering infant reactivity [52] (Figure 1). In rodents, powerful maternal regulation of stress hormone reactivity and amygdala fear learning occurs throughout the first two weeks of life [7,49], and maternal regulation of infant brain state during caregiving interactions wanes as pups approach weaning [22], supporting the notion that this sensitive period is confined to infancy and childhood.

In humans, caregivers provide strong regulation of behavior and physiology in childhood (by decreasing amygdala and stress reactivity through prefrontal cortex engagement) but not in adolescence, similarly supporting the idea that a sensitive period exists prior to adolescence [18]. Studies following the outcome of children reared in orphanages with low-quality caregiver interactions found that foster care interventions effectively restored HPA-axis and parasympathetic nervous system reactivity only among children placed in foster care prior to two years of age, suggesting that the first two years of life are particularly open to parental shaping of stress reactivity [53]. Additionally, a recent study provided evidence that a sensitive period for maternal support influencing hippocampal development occurs in preschool [54]. In this longitudinal study, hippocampal development from school-aged children to adolescents increased faster for children with higher levels of preschool maternal support.

Finally, a common theme in recent literature is emerging in which it is proposed that caregiver adversity promotes accelerated infant development via premature closure of sensitive periods [13,33]. While this may be true in some instances, we argue that this conceptualization is too simplistic and has the potential to dissuade researchers from exploring alternative hypotheses, thus limiting our understanding of neurobiological pathways to pathology. For example, there is evidence that early-life adversity precociously activates amygdala activity in humans and rodents [33,55]. However, using rodent models, deeper levels of analyses of gene expression, cell-type specific development, and the development of receptors used for cell-to-cell communication indicate that adversity can accelerate or delay different aspects of brain development [56,57]. Furthermore, rodent research exploring ways to repair the impact of early-life caregiver adversity on later-life emotionality discovered that in adulthood the presence of maternal odor can paradoxically normalize depressive-like and fear behaviors that arise as a result of early-life adversity [41]. Since maternal odor typically loses its regulatory power by weaning [11], this indicates that

maternal cues become regulatory at a delayed, developmentally-inappropriate time point following infant experience with negative caregiving. These findings provide exciting advances for research efforts attempting to re-open sensitive periods of maternal regulation of emotional development, in order to enduringly repair early-life effects of negative caregiving in adulthood.

Conclusion

Research on the neurobiology of infant attachment is revealing that the infant brain is uniquely primed for learning about the world in a way that promotes attachment to a caregiver. This attachment bias has immediate benefits, but enduring consequences, due to the caregiver's powerful ability to program the rapidly developing infant brain. Sensitive caregivers, particularly those who are in synchrony with their infant and provide nurturance during distress, provide the most optimal early-life programming of brain structures important for lifelong emotionality, seemingly via regulation of the infant brain and physiology. Adversity within the attachment system via negative caregiving has an enduring impact on brain areas underlying emotion and emotional regulation. Recent research suggests that there is a sensitive period for parental shaping of emotional development in early life, although further cross-species research is necessary for understanding the age limits of this period and how to re-open this sensitive period for later-life intervention efforts.

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Highlights

- Infants form attachment to their caregiver despite the quality of parenting.
- Parents program emotionality via regulation of infant physiology.
- Parental nurturance and caregiver-infant synchrony are key to infant regulation.
- Neglectful, intrusive, and/or unpredictable caregiving disrupts regulation.
- Sensitive caregiving protects infant development in adverse environments.

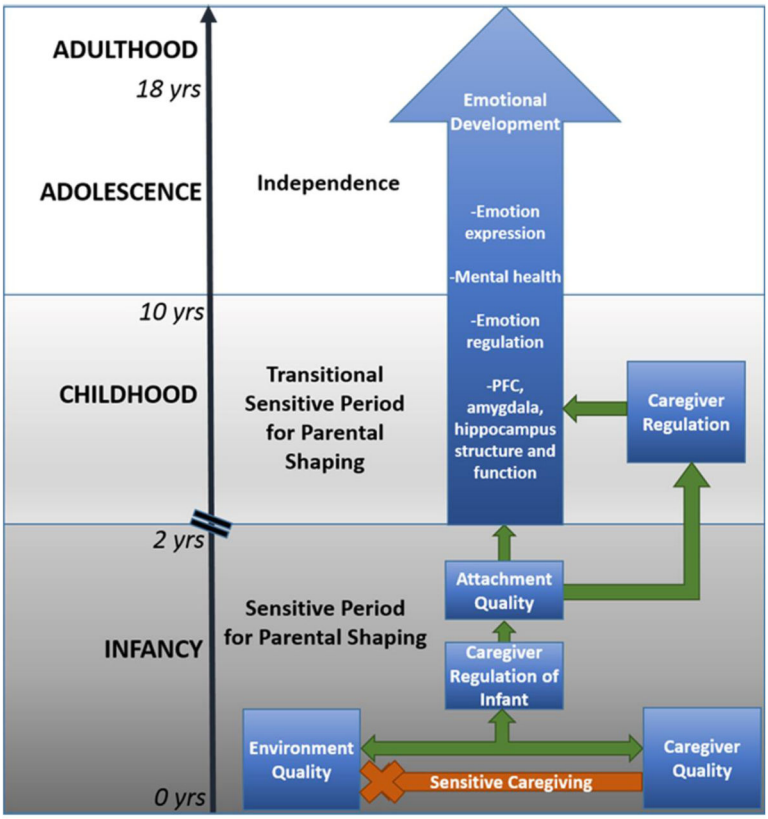

Figure 1.

Caregiver regulation of infant behavior and physiology in early life programs later life emotionality.