A Neuroscience Perspective on Emotional Development

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Abstract

In the current chapter, we suggest that a neuroscientific approach provides a valuable perspective to the study of emotional development. We discuss how a neuroscientific approach offers unique contributions to notable practical and theoretical challenges in the study of the development of emotion and emotion regulation. We exemplify these contributions by reviewing the current knowledge on the development of the expression and regulation of fear and anxiety and their associated neural bases. The literature reviewed highlights the fact that a neuroscientific approach situates the study of emotional development in a larger biological and evolutionary framework facilitating the translation of research across species and providing an account for species-typical development as well as individual variation. A neuroscientific approach also provides methods that permit studying emotional development across several levels of analyses, providing information on the similarity and/or differentiation between processes and mechanisms. We also cover literature that exemplifies how a neuroscientific approach can expand our understanding of how constitutional factors and experiences create the brain networks that support the expression and regulation of emotion across development. Finally, we discuss outstanding issues and future directions with the neuroscientific approach to the study of emotional development.

Introduction

Our ability to experience and express emotions is a core aspect of life. Moreover, regulating when and how we experience, and express, emotions is a crucial component of adaptive functioning. As such, understanding emotional development has important implications for individuals and society as emotional competence is crucial for mental health, physical well-being, and economic wealth (Eisenberg et al., 2001; Knudsen, Heckman, Cameron, & Shonkoff, 2006; Pine & Fox, 2015; Valiente, Lemery-Chalfant, Swanson, & Reiser, 2008). However, the scientific study of emotional development faces significant practical and theoretical challenges.

In the current chapter, we argue that a neuroscientific approach provides valuable insight into the study of emotional development, with the following broad contributions (Nelson, Thomas, & DeHaan, 2008): First, a neuroscientific approach places hypotheses and observations in a larger

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biological and evolutionary context, facilitating the translation of human emotional development from – and to – animal models. Second, by examining emotional development with a neuroscientific lens, we leverage several methods that allow us to examine the phenomena of interest at different levels of analysis (e.g., genetic, molecular, structure, function, social), helping differentiate distinct processes and mechanisms. Finally, as we gain a better understanding of the mechanisms and the interactions across several levels of analysis, we gain a picture of emotional development that moves beyond simplistic models (e.g., nature vs. nurture) and allows us to examine how experiences impact the brain circuits that support the expression and regulation of emotion.

In the following sections, we summarize three notable challenges and briefly highlight how a neuroscientific approach provides a unique perspective into these issues. We then briefly review the development of emotion and emotion regulation and its associated neural bases.

Challenge I: Emotion and Emotion Regulation as Distinct Constructs

The first challenge arises when trying to define primary aspects of emotional development such as emotion and emotion regulation. As with most psychological constructs, there is no standard definition of emotion and emotion regulation. However, a particular challenge when defining these constructs is that it is difficult to empirically and theoretically separate emotion from emotion regulation. Although there are considerable debates about the essence of emotions and formal definitions of emotion have been attempted since the beginning of modern psychology (James, 1884) and biology (Darwin, 1872/1998), most authors would agree that emotions are biologically based processes that quickly and automatically evaluate events as well as prepare the organism to act in order to aid well-being and survival (Cole, Martin, & Dennis, 2004; Dennis, Buss, & Hastings, 2012). Recent definitions of emotion reserve emotion terms such as "fear" to the experiential components of the emotions and label as "survival circuits" the behavioral responses and accompanying neurophysiological changes (LeDoux, 2012; LeDoux & Brown, 2017). This distinction between components of emotion is important when studying emotional development, as it is not possible to access the experiential components of emotion during early development. Here, we do not make use of terms such as "survival circuits" and instead use emotion-related terms (e.g., "emotion" and "fear") in line with most of the developmental literature. However, throughout this chapter, we explicitly label the component of emotion being discussed to emphasize this distinction between behavioral and physiological changes compared to conscious feeling states (i.e., fear-related behaviors vs. feelings of fear).

Importantly, this definition of emotion highlights that emotions are by nature regulating the physiology, behavior, and experiences of the organism. In other words, emotions are inherently regulatory; thus, it is difficult to separate them from the processes that regulate them (Campos, Mumme, Kermoian, & Campos, 1994; Cole et al., 2004). Processes that change the valence, intensity, and duration of emotions are often termed emotion regulation (Thompson, 1994).

Most definitions of emotion regulation highlight a wide breadth of such processes including conscious and unconscious processes that change emotional responses such as purposely distracting oneself by focusing on other aspects of the situation or unintentionally looking away from aversive events (Gross & Thompson, 2007). Recent models also distinguish between explicit and implicit strategies as well as automatic and controlled forms of emotion regulation (Braunstein, Gross, & Ochsner, 2017; Etkin, Büchel, & Gross, 2015), further complicating its distinction with emotional responses. For instance, imagine a child being approached by a novel stimulus like a stranger. When the child perceives the stranger approaching, their eyebrows raise, their eyes widen, their heart rate accelerates, and their pupils dilate – clear behavioral and physiological changes associated with fear. The child then turns around, runs away,

clings to their caregiver's leg, and averts their gaze away from the stranger. Although these withdrawal behaviors regulate behavior and lead to decreases in fear-related physiology and behavior, it is less clear if they are part of the expression of fear or if there are implicit and automatic forms of emotion regulation.

Cole et al. (2004) propose that to resolve this challenge it is necessary to measure emotion processes independently from implied emotion regulation processes and/or to assess changes in the emotion due to emotion regulation. However, currently, no single method in developmental psychology is able to empirically make this distinction. As such, developmental psychologists need to make inferences based on several methods. A neuroscientific approach provides a powerful way to measure mental processes as some methods have a high temporal precision, allowing to capture the chronometry of emotional and regulatory processes, while others can indicate which brain regions are involved in emotion and/or emotion regulation. For example, electroencephalography (EEG) measures of brain electrical activity over the scalp that can be decomposed into specific frequency bands or averaged around an event of interest (i.e., event-related potentials; ERP). EEG measures have an excellent temporal resolution. In general, the latency of a measure of interest (e.g., ERP) is thought to indicate whether a mental process is relatively automatic or effortful with shorter latencies indicating shorter information processing and more automatic responses.

Another commonly used measure is magnetic resonance imaging (MRI), which provides several measures of the structure of the brain such as gray matter, white matter, and myelination. Functional MRI (fMRI) provides a measure of brain activity with high spatial resolution. In general, processes that involve the same brain areas are considered to involve similar mental processes, whereas activation in different brain areas is thought to involve different cognitive processes. Moreover, it is possible to describe the interrelations among brain areas by examining structural and/or functional connectivity, providing information about brain networks. Structural connectivity consists of measuring the brain regions that are physically or anatomically connected to each other. On the other hand, functional connectivity refers to measuring brain activity that is temporally related across brain areas, implying that these areas are active at the same time, potentially working together and influencing each other.

As will be reviewed in this chapter, researchers have used these techniques in past decades to measure the time course and networks of brain areas involved in emotional development increasing our understanding of which processes are shared (and different) between emotions and their regulation. Although a neuroscientific approach does not solve this challenge on its own, it can provide important evidence and tools that together with other approaches can serve to better understand the distinction between emotion and emotion regulation.

Challenge II: Emotion and Cognition Integration

The second challenge to studying and understanding emotional development involves treating emotional and cognitive development as categorically different phenomena. The dichotomy between cognition and emotion can be rooted all the way back to dualistic models of the mind and brain/body best exemplified by Descartes and Aristotle, in which emotion and reason were treated as categorically separate constructs. For psychology in general, the dichotomy was exacerbated during the cognitive revolution, which focused on the role of information processing in human cognition. This led the field of cognitive psychology to focus on studying processes such as attention, memory, language, and decision-making, rarely including emotion (Phelps, 2006).

In developmental psychology in particular, this divide was further increased with the undertaking of charting cognitive development, led by work like Piaget's (Maccoby, 1984). For this, researchers studying cognitive development mostly employed experimental methods aimed at characterizing developmental milestones across

development. Examples of such methods can be found in the descriptions of Piaget's experiments and now-classic tasks (e.g., A-not-B task) used to understand differences across development by carefully controlling the environment (Piaget, 1954). Perhaps because emotional development was less amenable to experimental manipulation, emotional development was mostly studied using a more naturalistic and descriptive approach. As such, cognitive development was historically studied mostly by experimental paradigms, while emotional development was studied mostly by correlational work (Maccoby, 1984; Pérez-Edgar & Hastings, 2018). This led to a large corpus of empirical and theoretical work in each subfield emerging from different conceptual and methodological approaches, making it harder to integrate cognitive and emotional development theoretically and empirically. However, advancements in neuroscientific methods and increased understanding of the brain have demonstrated that cognitive and emotional development, not only interact but are deeply integrated in the individual throughout development (Bell & Wolfe, 2004; Lewis, 2005; Pessoa, 2008; Phelps, 2006). As will be discussed in this chapter, the brain networks associated with emotion and emotion regulation span across areas commonly studied in cognitive processes, illustrating that several cognitive functions such as attention, learning, memory, and cognitive control are deeply involved in emotional development.

Challenge III: Variations Across Development and Across Individuals

Developmental science aims to describe change over time as well as the mechanisms and consequences of change. The traditional goal of developmental science is to uncover laws concerning the development of human thought and behavior. In order to achieve this, developmental science commonly characterizes phenomena of interest by pooling information across people to summarize the data using measures of central tendency (e.g., average changes across age). Although this approach is highly valuable to characterize normative development, it often ignores the variability around the normative patterns of change. Importantly, most psychological processes have developmental trajectories that vary across individuals (Molenaar, 2004). As such, any individual developmental trajectory in the sample rarely represents the average trajectory.

Individual differences have important implications for our understanding of emotional development for practical and theoretical reasons. First, evidence suggests that many individual differences, for instance, in emotion expression, are stable over time, suggesting that they are not random noise. Second, many of the practical applications of the study of emotional development depend on the variability of emotion and emotion regulation across individuals as they are important predictors and markers of adjustment. For instance, identifying individuals at risk for later psychopathology or determining which factors predict developmental trajectories of resilience or risk relies on differences between individuals rather than norms (e.g., Kagan & Snidman, 1999; Mischel, Shoda, & Rodriguez, 1989; Moffitt et al., 2011). Finally, considering the diversity in the development of emotion is crucial to help elucidate some of the mechanisms behind the developmental outcomes. As such, the study of emotional development is faced with the challenge to not only consider both variations across normative development and variations across individuals but the need to integrate them into a framework that can account for both types of variation. We suggest that a neuroscientific approach can aid with this challenge by examining emotional development through an evolutionary biology lens that integrates species-typical and individual differences (Scarr, 1992). In this chapter, we will review empirical and theoretical literatures suggesting that individual variation due to constitutional factors such as temperament or early experiences have important implications for emotional development, including modifying the rate of developmental change. This growing literature leverages animal models to examine

how variations in evolutionarily conserved mechanisms shape emotional development across species.

We have summarized three important challenges to the study of emotional development. In addition, we have conceptually outlined some of the benefits of a neuroscientific approach to the study of emotional development. In the next section, we select a few examples from our work and others studying the development of the expression and regulation of fear and anxiety. The aim of the next section is to discuss examples that highlight the benefits of the neuroscientific approach, in particular, as it contributes to solving the challenges outlined above. Finally, we discuss outstanding issues and future directions with this approach. For each section below, we will first describe normative development, followed by a discussion on individual differences.

Neural Correlates of Emotion

Normative Development

Since the foundational work of Broca (1878/2015) and Papez (1937) on the limbic system, emotions have been thought to arise from subcortical brain structures. For example, individuals with damage to subcortical areas like the amygdala are reported to have abnormal fear reactions, including a marked reduction in the experience of fear, implying that the amygdala plays a critical role in the expression and experience of fear (Feinstein, Adolphs, Damasio, & Tranel, 2011). Most researchers would now agree that emotions are not localized to specific brain regions such as recent conceptualizations that propose that the amygdala is involved in processing of salient stimuli in general rather than exclusively fear-related stimuli (Adolphs, 2008).

However, the emphasis on subcortical circuits as fundamental for the development of emotion is reflected in the available literature. As such, we start by reviewing findings that focus on the development of relatively simplistic notions of mapping emotion to subcortical structures and functions with a focus on fear, anxiety, and the amygdala. We focus on fear, anxiety, and the amygdala because of the availability of developmental data with animal models as well as humans. In addition, the development of fear and anxiety have important implications as anxiety disorders are one of the most common forms of psychopathology, causing a significant burden to the individual and society (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012).

Fear and fear-related behaviors are thought to emerge around 7 months after birth (Sroufe, 1977). This is evidenced by the emergence of fear-related behaviors such as expressions of distress in response to strangers or heights around this developmental period (Braungart-Rieker, Hill-Soderlund, & Karrass, 2010; Scarr & Salapatek, 1970). For example, fear-related behaviors toward strangers are observed at 7 months (Sroufe, 1977), increase through infancy, and remain relatively high during toddlerhood before starting to decline in childhood (Brooker et al., 2013).

Another source of evidence comes from the development of threat processing during infancy. For instance, infants around 7 months of age develop a normative attentional preference for threatening information (e.g., fearful or angry facial expressions) (LoBue & DeLoache, 2010; Nelson & Dolgin, 1985; Peltola, Leppänen, Palokangas, & Hietanen, 2008). Importantly, these changes in fear-related behaviors, including fear processing, develop during a developmental period in which fear becomes functionally relevant as infants become independently mobile and start to explore the environment and spend time away from the caregiver (Campos, Barrett, Lamb, Goldsmith, & Stenberg, 1983; Leppänen & Nelson, 2012).

Although evidence on the development of subcortical neural structures that may underlie the development of fear-related behaviors during infancy in humans is only emerging, nonhuman animal models can be highly informative. Other species show a similar developmental trajectory of fear-related behaviors (Sullivan & Holman, 2010). For example, rhesus monkeys do not display fear-related behaviors in the presence of a stranger until approximately 2–3 months

of age, the rough equivalent of 7–12 months of age in human infants (Kalin, Shelton, & Takahashi, 1991).

Neuroanatomical work suggests that these changes in behavior are associated with changes in subcortical regions, particularly the amygdala. For instance, in rhesus monkeys, the amygdala undergoes exponential developmental changes during the first months of life before stabilizing and displaying slower rates of change (Chareyron, Lavenex, Amaral, & Lavenex, 2012; Payne, Machado, Bliwise, & Bachevalier, 2010). Moreover, disruptions to the amygdala during this developmental period lead to abnormal threat detection and fear-related responses to strangers (Bauman, Lavenex, Mason, Capitanio, & Amaral, 2004; Raper, Wilson, Sanchez, Machado, & Bachevalier, 2013). Similarly, rat pups do not exhibit threat learning during their first 10 days of life, a developmental period approximately equivalent to the second half of the first year in humans (Haroutunian & Campbell, 1979; Sullivan, Hofer, & Brake, 1986). Elegant work has delineated the neural changes that accompany the expression of fear-related behaviors. This work suggests that around the 10th day of life, hormonal changes lead to transitions in the structure and function of the amygdala, which permit rat pups to display adult-like threat learning (Sullivan & Holman, 2010). In sum, in animal models, the developmental changes in structure and function of the amygdala largely parallel the timing of the development of fear-related behaviors.

These findings are consistent with the few human studies in which the amygdala displays rapid increases in volume during the first year of life, compared to the second year of life and other subcortical structures like hippocampus (Gilmore et al., 2012). However, continued increases in volume continue to be observed during early childhood, through adolescence and into young adulthood (Giedd et al., 1996; Goddings et al., 2014; Herting et al., 2018; Østby et al., 2009). Although evidence directly linking amygdala activity to fear-related behaviors during infancy is lacking, emerging data using fMRI suggests that the amygdala shows stronger responses to emotional stimuli during childhood and adolescence compared to adulthood (Decety, Michalska, & Kinzler, 2011; Gee, Humphreys, et al., 2013; Hare et al., 2008; Silvers et al., 2016; Silvers, Shu, Hubbard, Weber, & Ochsner, 2015; Swartz, Carrasco, Wiggins, Thomason, & Monk, 2014). This pattern of amygdala reactivity is in line with normative decreases in the expression and reports of fear and some forms of anxiety (e.g., separation anxiety) from early childhood into adolescence and adulthood (Gee, Humphreys, et al., 2013).

The limited experimental evidence concerning the development of subcortical areas in infancy comes from EEG measures. Although EEG does not directly index activity from subcortical structures (e.g., the amygdala), it is possible that the observed activity reflects neural circuits that involve the amygdala. EEG measures of threat processing in infancy reveal a pattern that parallels the development of fear expression and behavioral measures of threat processing. For example, at 7 months, infants begin to display a larger Nc (a component related to attention toward salient stimuli) to threat-related facial expressions compared to other facial expressions (Kobiella, Grossmann, Reid, & Striano, 2008; Leppänen, Moulson, Vogel-Farley, & Nelson, 2007; Peltola, Leppänen, Maki, & Hietanen, 2009). Interestingly, larger Nc to threats emerging at 7 months also occurs to only eye whites expressing fear, even in the absence of conscious perception (Jessen & Grossmann, 2014, 2016). The automatic and unconscious perception of threats is thought to occur in subcortical brain regions like the amygdala (Whalen, 2004). Together, these neuroimaging evidence suggests that infants, by the second half of their first year, process threat-related facial expressions like adults by subcortical mechanisms that operate automatically and outside of conscious awareness (Jessen & Grossmann, 2015).

These emerging data in humans, together with data from animal models, suggests that the amygdala is functional from early development and seems to have periods of change that coincide with the expression of fear-related behaviors. Importantly, this early developmental

period of rapid change in structure and function of the amygdala has been proposed as a period in which the amygdala is particularly susceptible to environmental factors leading to lasting changes in socioemotional development (Tottenham & Gabard-Durnam, 2017). In the next section, we review studies that suggest that individual differences in fear and anxiety are also associated with changes in the amygdala and have implications to the development of social behaviors and psychopathology.

Individual Differences

Variations in the expression of fear and anxious behaviors across development are often studied under the umbrella of constitutional and/or contextual factors. One line of work has focused on fearful temperament – the expression of wariness, distress, negative affect, or avoidance in response to novel stimuli (N. A. Fox, Henderson, Rubin, Calkins, & Schmidt, 2001). Fearful temperament is most often assessed in toddlerhood and has been most often studied as behavioral inhibition (N. A. Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Importantly, fearful temperament is one of the best early predictors of later anxiety (Buss, 2011; Chronis-Tuscano et al., 2009; Pérez-Edgar & Fox, 2005; Schwartz, Snidman, & Kagan, 1999). A recent meta-analysis found that children characterized as temperamentally fearful are at a 7.5-fold increase in the odds of developing anxiety problems, especially social anxiety (Clauss & Blackford, 2012).

The antecedents of fearful temperament are early emerging and can be observed in 4-month-old infants' reactions to novelty. Infants who display high levels of motor behavior (e.g., thrashing their arms and legs), negative affect, as well as arching their back are more likely to become temperamentally fearful children (N. A. Fox, Snidman, Haas, Degnan, & Kagan, 2015). Kagan and Snidman (1991) proposed that these behaviors were caused by infants' amygdala responses in the face of novelty. Kagan suggested that these highly reactive infants, who were more likely to go on to become temperamentally fearful children, had a lower threshold for amygdala responsivity (Kagan, 1994). Later studies provided further evidence for this as children who were characterized as fearful in toddlerhood or as highly reactive as infants displayed more amygdala reactivity to faces as adolescents or adults (Pérez-Edgar et al., 2007; Schwartz et al., 2011; Schwartz, Wright, Shin, Kagan, & Rauch, 2003). Although, to our knowledge, no study has examined relations between amygdala activity and fearful temperament during infancy and early childhood, analogous findings have been found in animal models of fearful temperament. For example, an extensive literature suggests that in rhesus monkeys the amygdala plays a key role in the development of fearful temperament and anxiety (e.g., Birn et al., 2014; A. S. Fox et al., 2012; Kalin, Shelton, & Davidson, 2004; Oler et al., 2010).

Early life experiences are another widely studied source of individual differences in the development of fear and anxiety. Early adverse experiences place individuals at increased risk for psychopathology, including anxiety disorders (National Scientific Council on the Developing Child, 2010; Shonkoff et al., 2012). This literature also suggests that early experiences have implications for the development of the amygdala. A large corpus of evidence with animal models suggests that early experiences are related to the structure of the amygdala. For example, early stressors are predictive of larger amygdala volumes (e.g., Howell et al., 2014; Salm et al., 2004). Neuroimaging studies in humans have confirmed that, like in animal models, a wide range of early experiences are related to the functional and structural development of the amygdala.

These experiences range from normative variations in parenting (Gard et al., 2017), early life stress (Herringa et al., 2016; Suzuki et al., 2014), childhood poverty (Gianaros et al., 2008; Kim et al., 2013; Noble, Houston, Kan, & Sowell, 2012), to more extreme forms of early experiences such as maltreatment and social deprivation (Mehta et al., 2009; Olsavsky et al., 2013; Tottenham et al., 2010). In general, but with some exceptions, these studies find that early adverse experiences are related to increased

amygdala activity and larger amygdala volume. Importantly, in some instances the associated changes of amygdala function and structure are in turn associated with the known outcomes of early experiences such as increased anxiety (Tottenham et al., 2010).

Outstanding Issues and Future Directions

The first outstanding issue concerns the developmental trajectory of amygdala function. Although studies concur that adults display lower amygdala activity compared to children and adolescents, the evidence is mixed as to whether the amygdala displays a linear or a quadratic trajectory with adolescence being a period of increased amygdala activity (Gee, Humphreys, et al., 2013; Hare et al., 2008; Silvers et al., 2016, 2015). This difference has important implications as some models propose that adolescence is a unique period for the expression and regulation of emotions (Casey, 2015; Pattwell et al., 2012). One important factor to be considered is the task involved to elicit amygdala activity as the study finding linear decreases used explicit instructions to regulate emotion (Silvers et al., 2015, 2016), whereas others showing nonlinear changes involved implicit forms of emotion regulation (Hare et al., 2008) (see next section on Neural Correlates of Emotion Regulation). It is also worth noting that the amount of longitudinal data on the function of the amygdala is scarce. Future longitudinal studies with multiple assessments will be needed to better characterize the development of the amygdala's structure, function, and its role in emotional development.

Although our discussion has focused on the development of the amygdala and its role in the development of fear and anxiety, it is clear that the amygdala does not solely relate to fear-related behaviors, nor does it function in isolation. For example, amygdala activity has been related not only to the perception and expression of fear but also has been involved in other emotions such as disgust, happiness, sadness, and anger (Lindquist, Wager, Kober, Bliss-Moreau, & Barrett, 2012). This is in line with recent conceptualizations that suggest that amygdala is involved in the processing of salient stimuli more generally, which can be threatening, rewarding, or unpredictable (Adolphs, 2008).

Further complicating this picture, both animal and human works suggest that some of the regions commonly thought to be implicated in emotion, like the amygdala, are composed of subregions that have unique functions and are associated with specific patterns of connectivity (Etkin et al., 2004; Roy et al., 2009). For example, the basolateral subregion of the amygdala is most strongly functionally connected to temporal and frontal regions, while activity in the centromedial subregion is more closely related to activity in other subcortical structures (e.g., striatum) (Qin, Young, Supekar, Uddin, & Menon, 2012; Roy et al., 2009). Importantly, although some of this functional specificity is evident from early in development, the patterns of connectivity with the amygdala subregions become more segregated and specialized during development (Gabard-Durnam et al., 2014, 2018; Qin et al., 2012). On the other hand, other animal work suggests that subregions, which are commonly considered to be distinct, are functionally similar and closely work together to organize fear-related behaviors (A. S. Fox & Shackman, 2019).

Given this complexity and the lack of one-to-one mapping between brain structure and function to emotions, some authors posit that rather than trying to focus on specific brain regions, the emphasis should be in characterizing circuits or networks (Barrett, 2017; Casey, Galván, & Somerville, 2016; Pessoa, 2017). Crucially, many of these systems are not circumscribed to traditionally conceptualized emotional processes or brain areas (i.e., subcortical structures). Rather, they involve a wide variety of processes and brain areas, including sensorimotor and cognitive (e.g., memory and language) processes. As such, recent frameworks attempt to integrate and define emotion as the interaction across circuits (Barrett, 2017; Casey et al., 2016; Pessoa, 2017). Although these recent circuit- or network-based frameworks are potentially highly fruitful, little work has tested developmental changes using these

broader frameworks. There is emerging developmental work examining the brain networks related to emotional development, rather than specific brain areas, utilizing connectivity approaches. However, most of these studies have been focused on or conceptualized as examining emotion regulation rather than emotion expression based on the measures used. In the next section, we discuss the development of emotion regulation and its neural correlates, reviewing some of this emerging network-based literature.

Neural Correlates of Emotion Regulation

Normative Development

The development of emotion regulation begins in infancy, as the infant relies on automatic physiological processes and their caregiver to regulate their homeostatic and arousal states (Kopp, 1982; Sameroff, 2010). Children begin regulating distress in early infancy, mostly with aid of their caregivers, by using several strategies like distraction, which is accomplished by the early-appearing orienting response (Harman, Rothbart, & Posner, 1997). As children develop, they begin to internalize these strategies and start to use them on their own with more frequency to reduce distress and negative affect (Mangelsdorf, Shapiro, & Marzolf, 1995; Morasch & Bell, 2012; Rothbart, Ziaie, & O'Boyle, 1992); however, their effectiveness is limited during infancy (Stifter & Braungart, 1995), especially when regulating fear (Buss & Goldsmith, 1998).

During toddlerhood, children's deliberate use of such strategies shows a marked increase during the third year of life (e.g., the use of distraction; Cole et al., 2011; Kochanska, Coy, & Murray, 2001), which has been theorized as the developmental period in which self-regulation emerges (Kopp, 1982). This age also corresponds with considerable increases in the development of other cognitive processes such as executive function (Zelazo, 2004) and executive attention (Posner, Rothbart, Sheese, & Voelker, 2014; Rothbart, Sheese, Rueda, & Posner, 2011) believed to underlie the development of effortful control of behavior (e.g., attention; Ruff & Capozzoli, 2003). Moreover, the number of emotion regulation strategies used by children increases during childhood (Hodgins & Lander, 1997). Similarly, from childhood into adulthood, the use of more complex and adaptive emotion regulation strategies continues to increase (Zimmermann & Iwanski, 2014).

During these important changes in children's ability to use self-initiated emotion regulation strategies, it is assumed that not only the number of strategies increases but also the effectiveness with which these strategies are used improves through childhood to adulthood, allowing children to better regulate their emotions. For example, children's emotion regulation strategies are more effective at regulating fear responses at age 5 compared to age 2 (Morales et al., 2017). Likewise, the ability to reinterpret the meaning of an emotional event (i.e., reappraisal), a sophisticated emotion regulation strategy, improves with age from childhood to adulthood (Silvers et al., 2015, 2016). Although these behavioral examples provide initial evidence, a neuroscience approach allows examining the engagement of emotion-related and regulation processes (e.g., control of attention) that are not evident in behavior, especially at later developmental periods in which regulatory processes may mask emotional responses.

Emotion regulation is thought to involve several anterior areas of the brain including the medial prefrontal cortex (mPFC), ventrolateral PFC (vlPFC), dorsolateral PFC (dlPFC), and anterior cingulate cortex (ACC) (Etkin et al., 2015). In general, the brain areas associated with emotion regulation show a protracted developmental trajectory with important structural and functional changes during infancy and early childhood (Gilmore et al., 2012; Li et al., 2012) as well as preadolescence that last until the end of adolescence (Giedd et al., 1999; Gogtay et al., 2004; Lenroot & Giedd, 2006) and emerging adulthood (Taber-Thomas & Pérez-Edgar, 2015).

For example, in one of the few available longitudinal studies, the PFC showed significantly more volume change than subcortical structures

(e.g., amygdala and nucleus accumbens) from late childhood into adulthood (Mills et al., 2014), providing support for its protracted development compared to other subcortical structures involved in processing and expressions of emotion. The development of these areas also corresponds with the observed changes in behaviors that these areas are thought to support (Crone & Steinbeis, 2017; Ordaz, Foran, Velanova, & Luna, 2013). For example, effortful control, the ability to inhibit a prepotent response in order to enact another response, shows a similar protracted developmental trajectory with continued improvements in late childhood and adolescence. Moreover, the size of these brain areas, such as the ACC, is a significant predictor of effortful control across development, with the largest predictive power during the periods of rapid change (<12 years; Fjell et al., 2012). Similarly, EEG measures that reflect activity from these anterior areas (e.g., ACC) and index aspects of cognitive control also suggest a prolonged developmental period from early childhood to well into adulthood (Buzzell, Richards, et al., 2017; Hoyniak, 2017; Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013).

Importantly, recent studies examining the development of emotion regulation have focused on the brain networks that support emotion regulation, rather than studying single brain areas. These network approaches are one step closer to examining the process of regulation, moving beyond brain activity in isolated areas. For example, rather than examining if the amygdala is differentially active across conditions (e.g., emotion regulation vs. no emotion regulation), these approaches measure the degree to which activity in regulation-related areas is related to emotion-related areas – under the assumption that we utilize regulatory processes (e.g., PFC activity) to dampen our emotional reaction (e.g., amygdala activity). A first step in characterizing the development of emotion regulation using this approach is to characterize the developmental trajectories in the patterns of connectivity among the brain regions of interest. Given the paucity of developmental data on the structure and function of these networks, animal models as well as a growing literature characterizing these patterns of connectivity during rest in human infants are of great importance.

Animal models with rodents and non-human primates suggest largely reciprocal anatomical connections between the amygdala and the PFC, especially mPFC (Ghashghaei, Hilgetag, & Barbas, 2007; Öngür & Price, 2000). Developmentally, tracing studies in rodents show that these projections emerge during infancy or early childhood. The projections between the amygdala and PFC continue to develop during adolescence and early adulthood (Cressman et al., 2010). Importantly, functional networks do not perfectly overlap with structural networks (e.g., Honey et al., 2009). As such, several PFC areas, which do not share direct connections to the amygdala, can be involved in its regulation. For example, in non-human primates, the amygdala showed significant functional connectivity with areas that have direct anatomical connections to the amygdala-like mPFC as well as areas that lack direct anatomical connections to the amygdala-like dlPFC (Birn et al., 2014).

In humans, similar patterns of connectivity have been found from early development. Emerging work characterizing functional connectivity networks in humans at rest suggests that from infancy to adulthood, the functional connectivity with amygdala is largely stable and displays similar topology (Gabard-Durnam et al., 2014, 2018). This is in line with recent studies examining functional connectivity of brain networks at rest more broadly (i.e., not specific to the amygdala), in which adult-like topology of networks is found from early childhood and infancy (De Asis-Cruz, Bouyssi-Kobar, Evangelou, Vezina, & Limperopoulos, 2015; Gilmore, Knickmeyer, & Gao, 2018) and even before birth (van den Heuvel et al., 2018). One exception are networks involved in regulatory processes such as frontoparietal brain regions which show considerable changes in connectivity from infancy to childhood (Gao et al., 2009, 2014; Gao, Alcauter, Smith, Gilmore, & Lin, 2015) and from childhood to adolescence (Fair et al., 2009). Similarly, for emotion-related networks specifically, the connectivity between the

amygdala and the mPFC shows significant increases in connectivity from early childhood to adulthood (Gabard-Durnam et al., 2014; Qin et al., 2012). However, a recent accelerated cohort longitudinal study found significant decreases in functional and structural mPFC-amygdala connectivity from childhood to adulthood (Jalbrzikowski et al., 2017). Overall, these changes in connectivity are in line with the developmental changes observed in emotion regulation and the development of executive functions more broadly.

Other sources of evidence examining the neural bases of the development of emotion regulation using a network approach come from task-based studies. Recent models of the neural bases of emotion regulation make the useful distinction between implicit and explicit emotion regulation strategies (Braunstein et al., 2017; Etkin et al., 2015; Gyurak, Gross, & Etkin, 2011).

Implicit emotion regulation development Implicit emotion regulation strategies do not require instructions and occur automatically and largely outside of conscious awareness such as fear extinction, emotional conflict, and affect-biased attention. These emotion regulation strategies tend to activate ACC, mPFC, and vlPFC (Braunstein et al., 2017; Etkin et al., 2015). Support for the involvement of these areas comes from several sources of evidence. The original support for the involvement of these areas (e.g., mPFC) came from lesion studies with animal models examining fear extinction – the process of learning that a previously threatening stimulus is no longer dangerous (Milad & Quirk, 2012; Morgan, Romanski, & LeDoux, 1993). Later work in adults also involving fear conditioning confirmed the role of similar brain areas in the inhibition of fear in humans (Phelps, Delgado, Nearing, & LeDoux, 2004; Phelps & LeDoux, 2005). Other evidence comes from paradigms examining emotional conflict, in which emotional cues are distracting and incompatible or irrelevant to task demands such as the emotional Stroop (Mathews & MacLeod, 1985) or the emotional variants of the Go/No-go tasks (Casey et al., 2011; Hare et al., 2008).

Importantly, regulating affective stimuli is incidental to successfully carrying out goal-directed behavior in these paradigms. In studies using such paradigms, increased brain activation in the ACC and mPFC is commonly observed for conditions that involve conflict (Egner, Etkin, Gale, & Hirsch, 2007; Etkin, Egner, Peraza, Kandel, & Hirsch, 2006). Finally, affect-biased attention is the tendency to selectively attend to environmental cues that are pertinent to the one psychological state, facilitating the processing of stimuli in the environment and influencing one's emotional processes (Morales, Fu, & Pérez-Edgar, 2016; Todd, Cunningham, Anderson, & Thompson, 2012). Studies examining the neural bases of affect-biased attention have found greater activity in vlPFC for trials that involve orienting away from threatening facial expressions (Fu, Taber-Thomas, & Pérez-Edgar, 2017; Liu, Taber-Thomas, Fu, & Pérez-Edgar, 2018; Monk et al., 2006; Telzer et al., 2008).

Most studies examining developmental patterns have used emotional variants of the Go/No-go task. These studies find that the connectivity between the amygdala and mPFC becomes more adult-like with age (Gee, Humphreys, et al., 2013; Perlman & Pelphrey, 2011). Interestingly, the amygdala-mPFC connectivity in response to fearful faces shows a valence shift from positive connectivity during early childhood to negative connectivity during late adolescence and adulthood, paralleling normative decreases in amygdala reactivity to fear faces, increases in effortful control behaviors, decreases in anxiety, and improvement in emotion regulation across development (Gee, Humphreys, et al., 2013). The reason for this shift may be due to immature structural connectivity between the amygdala and the mPFC (Moreira & Silvers, 2018). As reviewed above, the PFC continues to structurally mature well into adulthood, and structural maturity has been associated with regulation of amygdala reactivity (Swartz et al., 2014). Another complementary hypothesis suggests that inputs from subcortical areas to the PFC lead to the

development of adult-like connectivity between the PFC and subcortical regions (Casey et al., 2019; Tottenham & Gabard-Durnam, 2017). This hypothesis is based on positive connectivity during early childhood and the animal work suggesting the emergence of subcortical to cortical before cortical to subcortical connections (Bouwmeester, Smits, & Ree, 2002; Bouwmeester, Wolterink, & Ree, 2002).

Although most studies examining emotion regulation have focused on characterizing influences between subcortical and cortical areas, recent studies suggest that connectivity between subcortical and subcortical areas may also play an important role in emotion regulation. A recent study found that amygdala and ventral striatum connectivity decreased with age from childhood to adulthood. Moreover this amygdala-ventral striatum connectivity was related to worse effortful control, especially to emotional cues (Heller, Cohen, Dreyfuss, & Casey, 2016). On the other hand, increased mPFC-amygdala connectivity was related with better effortful control to emotional cues. Moreover, mPFC-amygdala connectivity mediated the relation between amygdala-ventral striatum connectivity and effortful control. These findings suggest that subcortical to subcortical connectivity plays an important role in the development of emotion and emotion regulation. Furthermore, this study provides further support for the role of cortical to subcortical connectivity in the regulation of emotion (Heller et al., 2016).

Explicit emotion regulation development Explicit emotion regulation strategies involve the conscious desire to change one's emotions by following either intrinsic or extrinsic goals (e.g., following instructions). The principal explicit emotion regulation strategies are selective attention, distraction, and reappraisal (Ochsner & Gross, 2005). Selective attention involves actively attending to either the nonemotional features of the environment to downregulate emotion or to focus one's attention on the emotional features of the stimuli to upregulate emotion (Hariri, Bookheimer, & Mazziotta, 2000; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003). Another attention-related emotional regulation strategy is distraction, which involves becoming immersed in another task to limit or diminish attention to the emotional stimuli (Pessoa, McKenna, Gutierrez, & Ungerleider, 2002; Tracey et al., 2002). Finally, reappraisal involves reinterpreting the meaning of an emotional stimulus to change ones reaction to it (Gross, 1998). Studies examining these emotion regulation strategies generally find activation of frontal areas including vlPFC and dlPFC (as well as parietal areas commonly involved in the executive control network) (Buhle et al., 2014; Kohn et al., 2014).

Although there are far fewer studies examining the development of explicit emotion regulation strategies, recent studies have started to examine explicit emotion regulation strategies in children. These studies show that the ability to use reappraisal as an emotion regulation strategy increases with age from childhood to adulthood (DeCicco, O'Toole, & Dennis, 2014; Silvers et al., 2015, 2016, 2017). Evidence for these studies comes from two main neuroimaging modalities, EEG and fMRI.

EEG studies have examined the effects of reappraisal on the late positive potential (LPP), an ERP component thought to index facilitated attention to emotional stimuli (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000). In these studies, listening to a reappraisal story before observing an aversive picture significantly reduced the LPP (Dennis & Hajcak, 2009). These studies find that children's ability to use reappraisal increases with age (DeCicco et al., 2014). Moreover, the effect of reappraisal on the LPP seems to occur at later latencies than previously shown in adults, implying that the timing of the emotion regulation process may change with age (Dennis & Hajcak, 2009). Finally, the extent of reappraisal is related concurrently and longitudinally to children's ability to use adaptive emotion regulation strategies during emotional challenges (Babkirk, Rios, & Dennis, 2015).

Similarly, studies using fMRI find that the age-related increases in emotion regulation are

evident by reductions in feelings of negative affect and amygdala reactivity as well as increases in vlPFC when instructed to reappraise aversive stimuli. Further analyses suggest that age increases in the vlPFC mediate the relation between age and amygdala reactivity, suggesting that as individuals grow older, they are better able to recruit vlPFC during reappraisal to dampen amygdala activity (Silvers et al., 2016). In addition, the effects of the vlPFC on amygdala were stronger for individuals with a "mature" pattern of mPFC-amygdala connectivity. This suggests that the ability to regulate emotions via more cognitively advanced regulation strategies may depend on previously developing simpler forms of emotion regulation such as implicit emotion regulation strategies via mPFC-amygdala connectivity.

As a way of summarizing this section on the development of the neural bases on emotion regulation, we briefly review recent developmental models of the neural bases of emotion regulation. Recent developmental models propose that changes in the emotion and emotion regulation circuitry across development occur hierarchically (Casey, Heller, Gee, & Cohen, 2019; Tottenham & Gabard-Durnam, 2017). Namely, that connectivity changes take place from subcortical to subcortical, followed by subcortical to cortical, followed by cortical to subcortical, followed by cortical to cortical (Casey et al., 2019). Evidence of this developmental trajectory is only emerging, but these models highlight the main points of the literature reviewed above: (1) the role of subcortical to subcortical connectivity in emotion during childhood (Heller et al., 2016); (2) the potential role of subcortical areas (e.g., amygdala) driving the connectivity with the PFC and shaping the development of these cortical areas (Tottenham & Gabard-Durnam, 2017); (3) Finally, in addition to the more commonly studied role of the PFC to subcortical areas, they highlight the role of connectivity across cortical areas, especially when using more advanced and cognitively demanding emotion regulation strategies such as reappraisal (Casey et al., 2019; Silvers et al., 2016).

Individual Differences

As with individual differences in emotion, studies have also found considerable variation across individuals in their development of emotion regulation. Temperament is an important source of variation in emotion regulation. Although there is evidence of differences in emotion regulation based on fearful temperament, this evidence is limited almost exclusively to implicit forms of emotion regulation (e.g., Morales, Pérez-Edgar, & Buss, 2015; Morales, Taber-Thomas, & Pérez-Edgar, 2016; Penela, Walker, Degnan, Fox, & Henderson, 2015; Pérez-Edgar et al., 2010), and information about their neural bases is even more sparse. The studies that do exist suggest that individuals characterized as temperamentally fearful either early in development or concurrent with the neural measures show important differences in brain structure and function in areas associated with emotion regulation (Hardee et al., 2013; Taber-Thomas, Morales, Hillary, & Pérez-Edgar, 2016). For example, structurally, adults characterized as temperamentally fearful in early childhood had a larger mPFC (Schwartz et al., 2010), but smaller ACC (Sylvester et al., 2016). Connectivity analyses suggest that fearful temperament is associated with increased negative connectivity between the amygdala and ACC and dlPFC (Hardee et al., 2013; Roy et al., 2014). Notably, these findings closely parallel findings with animal models of fearful temperament and anxiety, suggesting an evolutionarily conserved network (Birn et al., 2014).

Furthermore, other studies suggest that fearful temperament is related to more activity in the dlPFC and mPFC when instantiating effortful control in an emotional context (Fu et al., 2017; Jarcho et al., 2013, 2014). Intriguingly, these differences in brain activity are typically evident in the absence of differences in performance, suggesting that fearful individuals may need to engage these control-related areas to compensate for higher levels of emotional reactivity (Fu et al., 2017; Jarcho et al., 2014). This interpretation is in line with the previously reviewed findings of increased amygdala reactivity in individuals characterized as temperamentally fearful in early

childhood (Pérez-Edgar et al., 2007; Schwartz et al., 2003). In addition, these findings also concur with studies examining EEG measures of effortful control, which find that fearful temperament is related to increased control-related ERP components such as error monitoring (Brooker & Buss, 2014; Buzzell, Troller-Renfree, et al., 2017; Lahat et al., 2014; McDermott et al., 2009).

Most studies investigating the development of individual differences in emotion regulation focus on the effects of early life experiences in the development of emotion regulation and its associated brain networks. Together, this literature suggests that in animal models as well as in humans, a broad range of early experiences are related to the functional and structural development of emotion regulation networks (Callaghan, Sullivan, Howell, & Tottenham, 2014; McEwen et al., 2015). These experiences range from normative variations in parenting (Kopala-Sibley et al., 2018), early life stress (Burghy et al., 2012; Hanson et al., 2012), and low socioeconomic status (Gianaros et al., 2007; Kim et al., 2013), to more extreme forms of early experiences such as maltreatment and social deprivation (Gee, Gabard-Durnam, et al., 2013; Hanson et al., 2013; McLaughlin et al., 2014; McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015; Sheridan, Fox, Zeanah, McLaughlin, & Nelson, 2012). In general, this literature finds that more stressful experiences are associated with worse emotion regulation, increased amygdala reactivity, reduced structural measures in the PFC, and increased negative connectivity between mPFC and amygdala. Developmentally, studies from animal models as well as humans imply that early adversity may accelerate the development of the mPFC-amygdala connectivity (Callaghan et al., 2014). This is in line with models that suggest that early experiences shape the brain and physiological mechanisms involved in emotional responses and their regulation in an adaptive manner for that context by accelerating maturation (Del Giudice, Ellis, & Shirtcliff, 2011), albeit potentially by forgoing plasticity by closing sensitive periods earlier in development (Callaghan & Tottenham, 2016). More research is needed to reveal the functional implications of this accelerated developmental trajectory. It is possible that accelerated development comes at the expense of the development of other circuits also needed for emotional competence and may help explain the increased levels of psychopathology among children who experience high levels of early life adversity (Callaghan & Tottenham, 2016). Moreover, an accelerated developmental trajectory may lead to worse long-term health outcomes and premature aging (Belsky & Shalev, 2016).

Outstanding Issues and Future Directions

The first outstanding issue reflects the lack of evidence concerning developmental changes in the neural networks associated with emotion regulation early in development. As reviewed above, most developmental neuroscience work has focused on late childhood, early adolescence, and adulthood. This is in stark contrast to the behavioral work, in which there are decades of work on the early development of emotion regulation – work that implies that important changes in emotion regulation occur during the first years. To our knowledge, there is no neuroimaging work describing critical periods of the development of emotion regulation such as the emergence of independent forms of emotion regulation during toddlerhood and early childhood (Kopp, 1982, 1989). The lack of work during this developmental period probably stems from the difficulty of using neuroscientific measures during toddlerhood and early childhood. Neuroscientific measures are highly susceptible to motion, especially fMRI, and require a high degree of compliance. In addition, when studying developmental patterns other methodological considerations must be considered such as brain templates, skull thickness, etc. Although there is emerging work characterizing brain networks in infancy and toddlerhood during sleep (Gilmore et al., 2018; Graham et al., 2015), future research will require novel paradigms that allow the study of emotion-related processes (e.g., Graham, Fisher, & Pfeifer, 2013) during this important developmental period.

Another future avenue of research is the application of computational models to study the development of emotion regulation. Emotion regulation is a complex process composed of several cognitive processes, each of which is developing, potentially at different rates. A fruitful approach to understanding the development of emotion regulation is to focus on examining and differentiating the cognitive processes needed for successful emotion regulation. In adults, there are proposals (old and new) that highlight the utility of parsing the different cognitive processes involved in emotion regulation (Carver & Scheier, 1990; Etkin et al., 2015; Gyurak et al., 2011). However, to our knowledge, there has been little to no application of computational models to the development of emotion regulation. For example, although models differ in the specific cognitive components, most of these models agree that a key component is the ability to detect discrepancies between expected outcomes and current experiences, namely monitoring of prediction errors. There is considerable work in adults and in children examining this process, its development, and relations with the development of fear and anxiety (Buzzell, Troller-Renfree, Morales, & Fox, 2018; Meyer, 2017; Tamnes et al., 2013). Broadly, this line of research suggests that error monitoring increases from childhood to adulthood (Tamnes et al., 2013) and that increased error monitoring is related to increased levels of anxiety, especially clinical levels (Meyer, 2017). Moreover, increased error monitoring moderates the relation between early fearful temperament and later anxiety problems, such that fearful temperament is especially predictive of anxiety problems for children with increased error monitoring (Buzzell et al., 2018). Integrating this knowledge into an emotion regulation framework would be beneficial to our understanding how we control our emotions across development.

As with the study of the neural bases of emotion, there is a dearth of longitudinal studies. To our knowledge, all of the studies on the development of the brain networks associated with emotion regulation are cross-sectional. Future longitudinal studies in which the same individuals are assessed at multiple time points are needed in order to capture intraindividual variations as well as interindividual variations. For example, utilizing an accelerated cohort longitudinal design (Jalbrzikowski et al., 2017) would allow capturing within-person changes across a wide age range. Moreover, multiple time measures on the same individual will provide a better characterization of nonlinear changes as well as time periods that are highly susceptible to change. Finally, future studies should utilize multiple neural measures on the same individuals leveraging the strengths of each modality to better understand emotion regulation and its development.

Conclusion

In the present chapter, we propose that a neuroscientific approach provides a unique and valuable perspective to the study of emotional development. In particular, we suggest that a neuroscientific approach can contribute to three theoretical and methodological challenges to the study of emotional development. The first challenge involves the distinction between emotion and emotion regulation as separate processes. A neuroscientific approach can contribute to this challenge by providing tools and concepts that allow the examination of emotional development across several levels of analyses, aiding the differentiation of distinct processes and mechanisms. For example, in the literature reviewed, the amygdala was commonly involved in emotion-related processes such as fear-related behaviors. On the other hand, prefrontal areas (e.g., mPFC and vlPFC) were mostly involved in the regulation of emotion processes, including emerging connectivity data that implies that engagement in prefrontal regions regulates amygdala activity. Furthermore, different patterns of connectivity were involved with different forms of emotion regulation (i.e., explicit vs. implicit), implying different processes. Future studies leveraging a combination of neuroimaging methods (e.g., multimodal imaging) will further increase our understanding by not only characterizing the brain areas involved but also

the unique chronometry of emotion and emotion regulation processes.

The second challenge regards the common conceptualization of emotion and cognitive processes as categorically distinct processes. As reviewed in this chapter, several of the emotion regulation strategies and its associated brain networks emphasized the integration of emotion-related processes and other processes traditionally conceptualized as purely cognitive processes such as attention and cognitive control. Further supporting this integration, the developmental data reviewed in this chapter suggests that several emotion and emotion regulation processes follow a similar developmental trajectory as closely related cognitive processes. For instance, emotion regulation and cognitive control both rely heavily on prefrontal regions, and they follow a similar protracted developmental trajectory from early childhood into adulthood. Given the novelty of network or systems approaches to the study of the neural bases of emotional development, future studies should continue to characterize development of these networks. For instance, recent studies with adults utilizing multivariate pattern classification techniques have found that emotion processes involve highly distributed patterns of brain activation across cortical and subcortical areas (e.g., Kragel & LaBar, 2015). However, to our knowledge, no study has utilized similar pattern classification techniques to examine the development of emotion and emotion regulation.

The third challenge involves accounting for variation across development and across individuals. The literature reviewed highlights the fact that a neuroscientific approach situates the study of emotional development in a larger biological and evolutionary framework by using animal models and accounting for species-typical as well as individual variation. We also reviewed literature illustrating how a neuroscientific approach can further our understanding of how constitutional factors and experiences shape the brain networks that associated with the expression and regulation of emotion across development. Future longitudinal research utilizing a combination of methods is needed to increase our understanding on the development of the brain networks that underlie emotional development, including early identification of risk and the periods most susceptible to change.

We believe that a neuroscientific approach will continue to provide valuable contributions and help inform when and how to assist children's developmental trajectories to promote successful emotional development.

References

Adolphs, R. (2008). Fear, faces, and the human amygdala. *Current Opinion in Neurobiology, 18*(2), 166–172. https://doi.org/10.1016/j.conb.2008.06.006

Babkirk, S., Rios, V., & Dennis, T. A. (2015). The late positive potential predicts emotion regulation strategy use in school-aged children concurrently and two years later. *Developmental Science, 18*(5), 832–841.

Barrett, L. F. (2017). The theory of constructed emotion: An active inference account of interoception and categorization. *Social Cognitive and Affective Neuroscience, 12*(1), 1–23.

Bauman, M. D., Lavenex, P., Mason, W. A., Capitanio, J. P., & Amaral, D. G. (2004). The development of social behavior following neonatal amygdala lesions in rhesus monkeys. *Journal of Cognitive Neuroscience, 16*(8), 1388–1411. https://doi.org/10.1162/0898929042304741

Bell, M. A., & Wolfe, C. D. (2004). Emotion and cognition: An intricately bound developmental process. *Child Development, 75*(2), 366–370. https://doi.org/10.1111/j.1467-8624.2004.00679.x

Belsky, J., & Shalev, I. (2016). Contextual adversity, telomere erosion, pubertal development, and health: Two models of accelerated aging, or one? *Development and Psychopathology, 28*(4pt2), 1367–1383.

Birn, R. M., Shackman, A. J., Oler, J. A., Williams, L. E., McFarlin, D. R., Rogers, G. M., … Kalin, N. H. (2014). Evolutionarily conserved prefrontal-amygdalar dysfunction in early-life anxiety. *Molecular Psychiatry, 19*(8), 915–922. https://doi.org/10.1038/mp.2014.46

Bouwmeester, H., Smits, K., & Ree, J. M. V. (2002). Neonatal development of projections to the basolateral amygdala from prefrontal and thalamic structures in rat. *Journal of Comparative Neurology, 450*(3), 241–255. https://doi.org/10.1002/cne.10321

Bouwmeester, H., Wolterink, G., & Ree, J. M. V. (2002). Neonatal development of projections from the basolateral amygdala to prefrontal, striatal, and thalamic structures in the rat. *Journal of Comparative Neurology, 442*(3), 239–249. https://doi.org/10.1002/cne.10084

Braungart-Rieker, J. M., Hill-Soderlund, A. L., & Karrass, J. (2010). Fear and anger reactivity trajectories from 4

to 16 months: The roles of temperament, regulation, and maternal sensitivity. *Developmental Psychology, 46*(4), 791–804. https://doi.org/10.1037/a0019673

Braunstein, L. M., Gross, J. J., & Ochsner, K. N. (2017). Explicit and implicit emotion regulation: A multi-level framework. *Social Cognitive and Affective Neuroscience, 12*(10), 1545–1557.

Broca, P. (1878). Comparative anatomy of the cerebral convolutions: The great limbic lobe and the limbic fissure in the mammalian series. *Journal of Comparative Neurology, 523*(17), 2501–2554.

Brooker, R. J., & Buss, K. A. (2014). Toddler fearfulness is linked to individual differences in error-related negativity during preschool. *Developmental Neuropsychology, 39*(1), 1–8. https://doi.org/10.1080/87565641.2013.826661

Brooker, R. J., Buss, K. A., Lemery-Chalfant, K., Aksan, N., Davidson, R. J., & Goldsmith, H. H. (2013). The development of stranger fear in infancy and toddlerhood: Normative development, individual differences, antecedents, and outcomes. *Developmental Science*. https://doi.org/10.1111/desc.12058

Buhle, J. T., Silvers, J. A., Wager, T. D., Lopez, R., Onyemekwu, C., Kober, H., … Ochsner, K. N. (2014). Cognitive reappraisal of emotion: A meta-analysis of human neuroimaging studies. *Cerebral Cortex, 24*(11), 2981–2990.

Burghy, C. A., Stodola, D. E., Ruttle, P. L., Molloy, E. K., Armstrong, J. M., Oler, J. A., … Essex, M. J. (2012). Developmental pathways to amygdala-prefrontal function and internalizing symptoms in adolescence. *Nature Neuroscience, 15*(12), 1736.

Buss, K. A. (2011). Which fearful toddlers should we worry about? Context, fear regulation, and anxiety risk. *Developmental Psychology, 47*(3), 804–819. https://doi.org/10.1037/a0023227

Buss, K. A., & Goldsmith, H. H. (1998). Fear and anger regulation in infancy: Effects on the temporal dynamics of affective expression. *Child Development, 69*(2), 359–374. https://doi.org/10.1111/j.1467-8624.1998.tb06195.x

Buzzell, G. A., Richards, J. E., White, L. K., Barker, T. V., Pine, D. S., & Fox, N. A. (2017). Development of the error-monitoring system from ages 9–35: Unique insight provided by MRI-constrained source localization of EEG. *NeuroImage, 157*, 13–26. https://doi.org/10.1016/j.neuroimage.2017.05.045

Buzzell, G. A., Troller-Renfree, S. V., Barker, T. V., Bowman, L. C., Chronis-Tuscano, A., Henderson, H. A., … Fox, N. A. (2017). A neurobehavioral mechanism linking behaviorally inhibited temperament and later adolescent social anxiety. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(12), 1097–1105. https://doi.org/10.1016/j.jaac.2017.10.007

Buzzell, G. A., Troller-Renfree, S. V., Morales, S., & Fox, N. A. (2018). Relations between behavioral inhibition, cognitive control, and anxiety: Novel insights provided by parsing subdomains of cognitive control. In K. Pérez-Edgar & N. A. Fox (Eds.), *Behavioral inhibition: Integrating theory, research, and clinical perspectives* (pp. 213–235). Cham, Switzerland: Springer. https://doi.org/10.1007/978-3-319-98077-5_10

Callaghan, B. L., Sullivan, R. M., Howell, B., & Tottenham, N. (2014). The International Society for Developmental Psychobiology Sackler Symposium: Early adversity and the maturation of emotion circuits—A cross-species analysis. *Developmental Psychobiology, 56*(8), 1635–1650.

Callaghan, B. L., & Tottenham, N. (2016). The neuro-environmental loop of plasticity: A cross-species analysis of parental effects on emotion circuitry development following typical and adverse caregiving. *Neuropsychopharmacology, 41*(1), 163.

Campos, J. J., Barrett, K. C., Lamb, M. E., Goldsmith, H. H., & Stenberg, C. (1983). Socioemotional development. In M. M. Haith & J. J. Campos (Eds.), *Handbook of child psychology* (Vol. 2, 4th ed., pp. 783–915). New York, NY: Wiley.

Campos, J. J., Mumme, D. L., Kermoian, R., & Campos, R. G. (1994). A functionalist perspective on the nature of emotion. *Monographs of the Society for Research in Child Development, 59*(2/3), 284–303. https://doi.org/10.2307/1166150

Carver, C. S., & Scheier, M. F. (1990). Origins and functions of positive and negative affect: A control-process view. *Psychological Review, 97*(1), 19.

Casey, B. J. (2015). Beyond simple models of self-control to circuit-based accounts of adolescent behavior. *Annual Review of Psychology, 66*, 295–319.

Casey, B. J., Galván, A., & Somerville, L. H. (2016). Beyond simple models of adolescence to an integrated circuit-based account: A commentary. *Developmental Cognitive Neuroscience, 17*(Suppl. 1), 128–130.

Casey, B. J., Heller, A. S., Gee, D. G., & Cohen, A. O. (2019). Development of the emotional brain. *Neuroscience Letters*, 693, 29–34. https://doi.org/10.1016/j.neulet.2017.11.055

Casey, B. J., Somerville, L. H., Gotlib, I. H., Ayduk, O., Franklin, N. T., Askren, M. K., … Teslovich, T. (2011). Behavioral and neural correlates of delay of gratification 40 years later. *Proceedings of the National Academy of Sciences, 108*(36), 14998–15003.

Chareyron, L. J., Lavenex, P. B., Amaral, D. G., & Lavenex, P. (2012). Postnatal development of the amygdala: A stereological study in macaque monkeys. *Journal of Comparative Neurology, 520*(9), 1965–1984.

Chronis-Tuscano, A., Degnan, K. A., Pine, D. S., Perez-Edgar, K., Henderson, H. A., Diaz, Y., … Fox, N. A. (2009). Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry, 48*(9), 928–935. https://doi.org/10.1097/CHI.0b013e3181ae09df

Clauss, J. A., & Blackford, J. U. (2012). Behavioral inhibition and risk for developing social anxiety disorder: A meta-analytic study. *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(10), 1066–1075.e1. https://doi.org/10.1016/j.jaac.2012.08.002

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75*(2), 317–333. https://doi.org/10.1111/j.1467-8624.2004.00673.x

Cole, P. M., Tan, P. Z., Hall, S. E., Zhang, Y., Crnic, K. A., Blair, C. B., & Li, R. (2011). Developmental changes in anger expression and attention focus: Learning to wait. *Developmental Psychology, 47*(4), 1078–1089. https://doi.org/10.1037/a0023813

Cressman, V. L., Balaban, J., Steinfeld, S., Shemyakin, A., Graham, P., Parisot, N., & Moore, H. (2010). Prefrontal cortical inputs to the basal amygdala undergo pruning during late adolescence in the rat. *Journal of Comparative Neurology, 518*(14), 2693–2709.

Crone, E. A., & Steinbeis, N. (2017). Neural perspectives on cognitive control development during childhood and adolescence. *Trends in Cognitive Sciences, 21*(3), 205–215.

Cuthbert, B. N., Schupp, H. T., Bradley, M. M., Birbaumer, N., & Lang, P. J. (2000). Brain potentials in affective picture processing: Covariation with autonomic arousal and affective report. *Biological Psychology, 52*(2), 95–111. https://doi.org/10.1016/S0301-0511(99)00044-7

Darwin, C. (1998). *The expression of the emotions in man and animals*. New York, NY: Oxford University Press.

De Asis-Cruz, J., Bouyssi-Kobar, M., Evangelou, I., Vezina, G., & Limperopoulos, C. (2015). Functional properties of resting state networks in healthy full-term newborns. *Scientific Reports, 5*, 17755.

Decety, J., Michalska, K. J., & Kinzler, K. D. (2011). The contribution of emotion and cognition to moral sensitivity: A neurodevelopmental study. *Cerebral Cortex, 22*(1), 209–220.

DeCicco, J. M., O'Toole, L. J., & Dennis, T. A. (2014). The late positive potential as a neural signature for cognitive reappraisal in children. *Developmental Neuropsychology, 39*(7), 497–515. https://doi.org/10.1080/87565641.2014.959171

Del Giudice, M., Ellis, B. J., & Shirtcliff, E. A. (2011). The adaptive calibration model of stress responsivity. *Neuroscience & Biobehavioral Reviews, 35*(7), 1562–1592. https://doi.org/10.1016/j.neubiorev.2010.11.007

Dennis, T. A., Buss, K. A., & Hastings, P. D. (2012). Physiological measures of emotion from a developmental perspective: State of the science. *Monographs of the Society for Research in Child Development, 77*(2), i–204.

Dennis, T. A., & Hajcak, G. (2009). The late positive potential: A neurophysiological marker for emotion regulation in children. *Journal of Child Psychology and Psychiatry, 50*(11), 1373–1383. https://doi.org/10.1111/j.1469-7610.2009.02168.x

Egner, T., Etkin, A., Gale, S., & Hirsch, J. (2007). Dissociable neural systems resolve conflict from emotional versus nonemotional distracters. *Cerebral Cortex, 18*(6), 1475–1484.

Eisenberg, N., Cumberland, A., Spinrad, T. L., Fabes, R. A., Shepard, S. A., Reiser, M., … Guthrie, I. K. (2001). The relations of regulation and emotionality to children's externalizing and internalizing problem behavior. *Child Development, 72*(4), 1112–1134. https://doi.org/10.1111/1467-8624.00337

Etkin, A., Büchel, C., & Gross, J. J. (2015). The neural bases of emotion regulation. *Nature Reviews Neuroscience, 16*(11), 693.

Etkin, A., Egner, T., Peraza, D. M., Kandel, E. R., & Hirsch, J. (2006). Resolving emotional conflict: A role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron, 51*(6), 871–882.

Etkin, A., Klemenhagen, K. C., Dudman, J. T., Rogan, M. T., Hen, R., Kandel, E. R., & Hirsch, J. (2004). Individual differences in trait anxiety predict the response of the basolateral amygdala to unconsciously processed fearful faces. *Neuron, 44*(6), 1043–1055. https://doi.org/10.1016/j.neuron.2004.12.006

Fair, D. A., Cohen, A. L., Power, J. D., Dosenbach, N. U. F., Church, J. A., Miezin, F. M., … Petersen, S. E. (2009). Functional brain networks develop from a "local to distributed" organization. *PLoS Computational Biology, 5*(5), e1000381. https://doi.org/10.1371/journal.pcbi.1000381

Feinstein, J. S., Adolphs, R., Damasio, A., & Tranel, D. (2011). The human amygdala and the induction and experience of fear. *Current Biology, 21*(1), 34–38. https://doi.org/10.1016/j.cub.2010.11.042

Fjell, A. M., Walhovd, K. B., Brown, T. T., Kuperman, J. M., Chung, Y., Hagler, D. J., … Gruen, J. (2012). Multimodal imaging of the self-regulating developing brain. *Proceedings of the National Academy of Sciences, 109*(48), 19620–19625. https://doi.org/10.1073/pnas.1208243109

Fox, A. S., Oler, J. A., Shelton, S. E., Nanda, S. A., Davidson, R. J., Roseboom, P. H., & Kalin, N. H. (2012). Central amygdala nucleus (Ce) gene expression linked to increased trait-like Ce metabolism and anxious temperament in young primates. *Proceedings of the National Academy of Sciences of the United States of America, 109*(44), 18108–18113. https://doi.org/10.1073/pnas.1206723109

Fox, A. S., & Shackman, A. J. (2019). The central extended amygdala in fear and anxiety: Closing the gap between mechanistic and neuroimaging research. *Neuroscience Letters*, 693, 58–67. https://doi.org/10.1016/j.neulet.2017.11.056

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*(1), 235–262. https://doi.org/10.1146/annurev.psych.55.090902.141532

Fox, N. A., Henderson, H. A., Rubin, K. H., Calkins, S. D., & Schmidt, L. A. (2001). Continuity and discontinuity of behavioral inhibition and exuberance: Psychophysiological and behavioral influences across the first four years of life. *Child Development, 72*(1), 1–21. https://doi.org/10.1111/1467-8624.00262

Fox, N. A., Snidman, N., Haas, S. A., Degnan, K. A., & Kagan, J. (2015). The relations between reactivity at 4 months and behavioral inhibition in the second year: Replication across three independent samples. *Infancy, 20*(1), 98–114. https://doi.org/10.1111/infa.12063

Fu, X., Taber-Thomas, B. C., & Pérez-Edgar, K. (2017). Frontolimbic functioning during threat-related attention: Relations to early behavioral inhibition and anxiety in children. *Biological Psychology, 122*, 98–109. https://doi.org/10.1016/j.biopsycho.2015.08.010

Gabard-Durnam, L. J., Flannery, J., Goff, B., Gee, D. G., Humphreys, K. L., Telzer, E., … Tottenham, N. (2014). The development of human amygdala functional connectivity at rest from 4 to 23 years: A cross-sectional study. *NeuroImage, 95*, 193–207.

Gabard-Durnam, L. J., O'Muircheartaigh, J., Dirks, H., Dean, D. C., III, Tottenham, N., & Deoni, S. (2018). Human amygdala functional network development: A cross-sectional study from 3 months to 5 years of age. *Developmental Cognitive Neuroscience, 34*, 63–74.

Gao, W., Alcauter, S., Elton, A., Hernandez-Castillo, C. R., Smith, J. K., Ramirez, J., & Lin, W. (2014). Functional network development during the first year: Relative sequence and socioeconomic correlations. *Cerebral Cortex, 25*(9), 2919–2928.

Gao, W., Alcauter, S., Smith, J. K., Gilmore, J. H., & Lin, W. (2015). Development of human brain cortical network architecture during infancy. *Brain Structure and Function, 220*(2), 1173–1186.

Gao, W., Zhu, H., Giovanello, K. S., Smith, J. K., Shen, D., Gilmore, J. H., & Lin, W. (2009). Evidence on the emergence of the brain's default network from 2-week-old to 2-year-old healthy pediatric subjects. *Proceedings of the National Academy of Sciences, 106*(16), 6790–6795. https://doi.org/10.1073/pnas.0811221106

Gard, A. M., Waller, R., Shaw, D. S., Forbes, E. E., Hariri, A. R., & Hyde, L. W. (2017). The long reach of early adversity: Parenting, stress, and neural pathways to antisocial behavior in adulthood. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 2*(7), 582–590.

Gee, D. G., Gabard-Durnam, L. J., Flannery, J., Goff, B., Humphreys, K. L., Telzer, E. H., … Tottenham, N. (2013). Early developmental emergence of human amygdala–prefrontal connectivity after maternal deprivation. *Proceedings of the National Academy of Sciences, 110*, 15638–15643, 201307893.

Gee, D. G., Humphreys, K. L., Flannery, J., Goff, B., Telzer, E. H., Shapiro, M., … Tottenham, N. (2013). A developmental shift from positive to negative connectivity in human amygdala–prefrontal circuitry. *The Journal of Neuroscience, 33*(10), 4584–4593. https://doi.org/10.1523/JNEUROSCI.3446-12.2013

Ghashghaei, H. T., Hilgetag, C. C., & Barbas, H. (2007). Sequence of information processing for emotions based on the anatomic dialogue between prefrontal cortex and amygdala. *NeuroImage, 34*(3), 905–923. https://doi.org/10.1016/j.neuroimage.2006.09.046

Gianaros, P. J., Horenstein, J. A., Cohen, S., Matthews, K. A., Brown, S. M., Flory, J. D., … Hariri, A. R. (2007). Perigenual anterior cingulate morphology covaries with perceived social standing. *Social Cognitive and Affective Neuroscience, 2*(3), 161–173. https://doi.org/10.1093/scan/nsm013

Gianaros, P. J., Horenstein, J. A., Hariri, A. R., Sheu, L. K., Manuck, S. B., Matthews, K. A., & Cohen, S. (2008). Potential neural embedding of parental social standing. *Social Cognitive and Affective Neuroscience, 3*(2), 91–96. https://doi.org/10.1093/scan/nsn003

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., … Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2*(10), 861–863. https://doi.org/10.1038/13158

Giedd, J. N., Snell, J. W., Lange, N., Rajapakse, J. C., Casey, B. J., Kozuch, P. L., … Kaysen, D. (1996). Quantitative magnetic resonance imaging of human brain development: Ages 4–18. *Cerebral Cortex, 6*(4), 551–559.

Gilmore, J. H., Knickmeyer, R. C., & Gao, W. (2018). Imaging structural and functional brain development in early childhood. *Nature Reviews Neuroscience, 19*(3), 123.

Gilmore, J. H., Shi, F., Woolson, S. L., Knickmeyer, R. C., Short, S. J., Lin, W., … Shen, D. (2012). Longitudinal development of cortical and subcortical gray matter from birth to 2 years. *Cerebral Cortex, 22*(11), 2478–2485. https://doi.org/10.1093/cercor/bhr327

Goddings, A.-L., Mills, K. L., Clasen, L. S., Giedd, J. N., Viner, R. M., & Blakemore, S.-J. (2014). The influence of puberty on subcortical brain development. *NeuroImage, 88*, 242–251. https://doi.org/10.1016/j.neuroimage.2013.09.073

Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., … Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences of the United States of America, 101*(21), 8174–8179. https://doi.org/10.1073/pnas.0402680101

Graham, A. M., Fisher, P. A., & Pfeifer, J. H. (2013). What sleeping babies hear: A functional MRI study of interparental conflict and infants' emotion processing. *Psychological Science, 24*(5), 782–789.

Graham, A. M., Pfeifer, J. H., Fisher, P. A., Lin, W., Gao, W., & Fair, D. A. (2015). The potential of infant fMRI research and the study of early life stress as a promising exemplar. *Developmental Cognitive Neuroscience, 12*, 12–39. https://doi.org/10.1016/j.dcn.2014.09.005

Gross, J. J. (1998). Antecedent-and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224.

Gross, J. J., & Thompson, R. A. (2007). Conceptual foundations for the field. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 3–24). New York, NY: Guilford.

Gyurak, A., Gross, J. J., & Etkin, A. (2011). Explicit and implicit emotion regulation: A dual-process framework. *Cognition and Emotion, 25*(3), 400–412.

Hanson, J. L., Adluru, N., Chung, M. K., Alexander, A. L., Davidson, R. J., & Pollak, S. D. (2013). Early neglect is associated with alterations in white matter integrity and cognitive functioning. *Child Development*, n/a–n/a. https://doi.org/10.1111/cdev.12069

Hanson, J. L., Chung, M. K., Avants, B. B., Rudolph, K. D., Shirtcliff, E. A., Gee, J. C., … Pollak, S. D. (2012). Structural variations in prefrontal cortex mediate the relationship between early childhood stress and spatial working memory. *Journal of Neuroscience, 32*(23), 7917–7925. https://doi.org/10.1523/JNEUROSCI.0307-12.2012

Hardee, J. E., Benson, B. E., Bar-Haim, Y., Mogg, K., Bradley, B. P., Chen, G., … Pérez-Edgar, K. (2013). Patterns of neural connectivity during an attention bias task moderate associations between early childhood temperament and internalizing symptoms in young adulthood. *Biological Psychiatry, 74*(4), 273–279. https://doi.org/10.1016/j.biopsych.2013.01.036

Hare, T. A., Tottenham, N., Galvan, A., Voss, H. U., Glover, G. H., & Casey, B. J. (2008). Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. *Biological Psychiatry, 63*(10), 927–934. https://doi.org/10.1016/j.biopsych.2008.03.015

Hariri, A. R., Bookheimer, S. Y., & Mazziotta, J. C. (2000). Modulating emotional responses: Effects of a neocortical network on the limbic system. *Neuroreport, 11*(1), 43–48.

Hariri, A. R., Mattay, V. S., Tessitore, A., Fera, F., & Weinberger, D. R. (2003). Neocortical modulation of the amygdala response to fearful stimuli. *Biological Psychiatry, 53*(6), 494–501.

Harman, C., Rothbart, M. K., & Posner, M. I. (1997). Distress and attention interactions in early infancy. *Motivation and Emotion, 21*(1), 27–44.

Haroutunian, V., & Campbell, B. A. (1979). Emergence of interoceptive and exteroceptive control of behavior in rats. *Science, 205*(4409), 927–929. https://doi.org/10.1126/science.472715

Heller, A. S., Cohen, A. O., Dreyfuss, M. F., & Casey, B. J. (2016). Changes in cortico-subcortical and subcortico-subcortical connectivity impact cognitive control to emotional cues across development. *Social Cognitive and Affective Neuroscience, 11*(12), 1910–1918.

Herringa, R. J., Burghy, C. A., Stodola, D. E., Fox, M. E., Davidson, R. J., & Essex, M. J. (2016). Enhanced prefrontal-amygdala connectivity following childhood adversity as a protective mechanism against internalizing in adolescence. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 1*(4), 326–334.

Herting, M. M., Johnson, C., Mills, K. L., Vijayakumar, N., Dennison, M., Liu, C., … Whittle, S. (2018). Development of subcortical volumes across adolescence in males and females: A multisample study of longitudinal changes. *NeuroImage, 172,* 194–205.

Hodgins, M. J., & Lander, J. (1997). Children's coping with venipuncture. *Journal of Pain and Symptom Management, 13*(5), 274–285. https://doi.org/10.1016/S0885-3924(96)00328-4

Honey, C. J., Sporns, O., Cammoun, L., Gigandet, X., Thiran, J. P., Meuli, R., & Hagmann, P. (2009). Predicting human resting-state functional connectivity from structural connectivity. *Proceedings of the National Academy of Sciences, 106*(6), 2035–2040. https://doi.org/10.1073/pnas.0811168106

Howell, B. R., Grand, A. P., McCormack, K. M., Shi, Y., Laprarie, J. L., Maestripieri, D., … Sanchez, M. M. (2014). Early adverse experience increases emotional reactivity in juvenile rhesus macaques: Relation to amygdala volume. *Developmental Psychobiology, 56*(8), 1735–1746.

Hoyniak, C. (2017). Changes in the NoGo N2 event-related potential component across childhood: A systematic review and meta-analysis. *Developmental Neuropsychology, 42*(1), 1–24.

Jalbrzikowski, M., Larsen, B., Hallquist, M. N., Foran, W., Calabro, F., & Luna, B. (2017). Development of white matter microstructure and intrinsic functional connectivity between the amygdala and ventromedial prefrontal cortex: Associations with anxiety and depression. *Biological Psychiatry, 82*(7), 511–521.

James, W. (1884). What is an emotion? *Mind, 9*(34), 188–205.

Jarcho, J. M., Fox, N. A., Pine, D. S., Etkin, A., Leibenluft, E., Shechner, T., & Ernst, M. (2013). The neural correlates of emotion-based cognitive control in adults with early childhood behavioral inhibition. *Biological Psychology, 92*(2), 306–314. https://doi.org/10.1016/j.biopsycho.2012.09.008

Jarcho, J. M., Fox, N. A., Pine, D. S., Leibenluft, E., Shechner, T., Degnan, K. A., … Ernst, M. (2014). Enduring influence of early temperament on neural mechanisms mediating attention–emotion conflict in adults. *Depression and Anxiety, 31*(1), 53–62.

Jessen, S., & Grossmann, T. (2014). Unconscious discrimination of social cues from eye whites in infants. *Proceedings of the National Academy of Sciences, 111*(45), 16208–16213. https://doi.org/10.1073/pnas.1411333111

Jessen, S., & Grossmann, T. (2015). Neural signatures of conscious and unconscious emotional face processing in human infants. *Cortex, 64*, 260–270.

Jessen, S., & Grossmann, T. (2016). The developmental emergence of unconscious fear processing from eyes during infancy. *Journal of Experimental Child Psychology, 142*, 334–343. https://doi.org/10.1016/j.jecp.2015.09.009

Kagan, J. (1994). *Galen's prophecy: Temperament in human nature*. New York, NY: Basic Books.

Kagan, J., & Snidman, N. (1991). Infant predictors of inhibited and uninhibited profiles. *Psychological Science, 2*(1), 40–44. https://doi.org/10.1111/j.1467-9280.1991.tb00094.x

Kagan, J., & Snidman, N. (1999). Early childhood predictors of adult anxiety disorders. *Biological Psychiatry,*

46(11), 1536–1541. https://doi.org/10.1016/S0006-3223(99)00137-7

Kalin, N. H., Shelton, S. E., & Davidson, R. J. (2004). The role of the central nucleus of the amygdala in mediating fear and anxiety in the primate. *The Journal of Neuroscience, 24*(24), 5506–5515. https://doi.org/10.1523/JNEUROSCI.0292-04.2004

Kalin, N. H., Shelton, S. E., & Takahashi, L. K. (1991). Defensive behaviors in infant rhesus monkeys: Ontogeny and context-dependent selective expression. *Child Development, 62*(5), 1175–1183. https://doi.org/10.1111/j.1467-8624.1991.tb01598.x

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research, 21*(3), 169–184. https://doi.org/10.1002/mpr.1359

Kim, P., Evans, G. W., Angstadt, M., Ho, S. S., Sripada, C. S., Swain, J. E., … Phan, K. L. (2013). Effects of childhood poverty and chronic stress on emotion regulatory brain function in adulthood. *Proceedings of the National Academy of Sciences, 110*, 18442–18447, 201308240.

Knudsen, E. I., Heckman, J. J., Cameron, J. L., & Shonkoff, J. P. (2006). Economic, neurobiological, and behavioral perspectives on building America's future workforce. *Proceedings of the National Academy of Sciences of the United States of America, 103*(27), 10155–10162. https://doi.org/10.1073/pnas.0600888103

Kobiella, A., Grossmann, T., Reid, V. M., & Striano, T. (2008). The discrimination of angry and fearful facial expressions in 7-month-old infants: An event-related potential study. *Cognition and Emotion, 22*(1), 134–146.

Kochanska, G., Coy, K. C., & Murray, K. T. (2001). The development of self-regulation in the first four years of life. *Child Development, 72*(4), 1091–1111. https://doi.org/10.1111/1467-8624.00336

Kohn, N., Eickhoff, S. B., Scheller, M., Laird, A. R., Fox, P. T., & Habel, U. (2014). Neural network of cognitive emotion regulation—An ALE meta-analysis and MACM analysis. *NeuroImage, 87*, 345–355.

Kopala-Sibley, D. C., Cyr, M., Finsaas, M. C., Orawe, J., Huang, A., Tottenham, N., & Klein, D. N. (2018). Early childhood parenting predicts late childhood brain functional connectivity during emotion perception and reward processing. *Child Development*. https://doi.org/10.1111/cdev.13126

Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology, 18*(2), 199–214. https://doi.org/10.1037/0012-1649.18.2.199

Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology, 25*(3), 343–354. https://doi.org/10.1037/0012-1649.25.3.343

Kragel, P. A., & LaBar, K. S. (2015). Multivariate neural biomarkers of emotional states are categorically distinct. *Social Cognitive and Affective Neuroscience, 10*(11), 1437–1448.

Lahat, A., Lamm, C., Chronis-Tuscano, A., Pine, D. S., Henderson, H. A., & Fox, N. A. (2014). Early behavioral inhibition and increased error monitoring predict later social phobia symptoms in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(4), 447–455. https://doi.org/10.1016/j.jaac.2013.12.019

LeDoux, J. E. (2012). Rethinking the emotional brain. *Neuron, 73*(4), 653–676. https://doi.org/10.1016/j.neuron.2012.02.004

LeDoux, J. E., & Brown, R. (2017). A higher-order theory of emotional consciousness. *Proceedings of the National Academy of Sciences, 114*, E2016–E2025, 201619316.

Lenroot, R. K., & Giedd, J. N. (2006). Brain development in children and adolescents: Insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioral Reviews, 30*(6), 718–729. https://doi.org/10.1016/j.neubiorev.2006.06.001

Leppänen, J. M., Moulson, M. C., Vogel-Farley, V. K., & Nelson, C. A. (2007). An ERP study of emotional face processing in the adult and infant brain. *Child Development, 78*(1), 232–245. https://doi.org/10.1111/j.1467-8624.2007.00994.x

Leppänen, J. M., & Nelson, C. A. (2012). Early development of fear processing. *Current Directions in Psychological Science, 21*(3), 200–204. https://doi.org/10.1177/0963721411435841

Lewis, M. D. (2005). Bridging emotion theory and neurobiology through dynamic systems modeling. *Behavioral and Brain Sciences, 28*(02), 169–194. https://doi.org/10.1017/S0140525X0500004X

Li, G., Nie, J., Wang, L., Shi, F., Lin, W., Gilmore, J. H., & Shen, D. (2012). Mapping region-specific longitudinal cortical surface expansion from birth to 2 years of age. *Cerebral Cortex, 23*(11), 2724–2733.

Lindquist, K. A., Wager, T. D., Kober, H., Bliss-Moreau, E., & Barrett, L. F. (2012). The brain basis of emotion: A meta-analytic review. *Behavioral and Brain Sciences, 35*(3), 121–143. https://doi.org/10.1017/S0140525X11000446

Liu, P., Taber-Thomas, B. C., Fu, X., & Pérez-Edgar, K. E. (2018). Biobehavioral markers of attention bias modification in temperamental risk for anxiety: A randomized control trial. *Journal of the American Academy of Child & Adolescent Psychiatry, 57*(2), 103–110.

LoBue, V., & DeLoache, J. S. (2010). Superior detection of threat-relevant stimuli in infancy: Threat detection in infancy. *Developmental Science, 13*(1), 221–228. https://doi.org/10.1111/j.1467-7687.2009.00872.x

Maccoby, E. E. (1984). Socialization and developmental change. *Child Development, 55*(2), 317–328. https://doi.org/10.2307/1129945

Mangelsdorf, S. C., Shapiro, J. R., & Marzolf, D. (1995). Developmental and temperamental differences in emotion regulation in infancy. *Child Development, 66*(6), 1817–1828. https://doi.org/10.1111/j.1467-8624.1995.tb00967.x

Mathews, A., & MacLeod, C. (1985). Selective processing of threat cues in anxiety states. *Behaviour Research and Therapy, 23*(5), 563–569.

McDermott, J. M., Perez-Edgar, K., Henderson, H. A., Chronis-Tuscano, A., Pine, D. S., & Fox, N. A. (2009). A history of childhood Behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. *Biological Psychiatry, 65*(5), 445–448. https://doi.org/10.1016/j.biopsych.2008.10.043

McEwen, B. S., Bowles, N. P., Gray, J. D., Hill, M. N., Hunter, R. G., Karatsoreos, I. N., & Nasca, C. (2015). Mechanisms of stress in the brain. *Nature Neuroscience, 18*(10), 1353.

McLaughlin, K. A., Peverill, M., Gold, A. L., Alves, S., & Sheridan, M. A. (2015). Child maltreatment and neural systems underlying emotion regulation. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*(9), 753–762.

McLaughlin, K. A., Sheridan, M. A., Winter, W., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2014). Widespread reductions in cortical thickness following severe early-life deprivation: A neurodevelopmental pathway to attention-deficit/hyperactivity disorder. *Biological Psychiatry, 76*(8), 629–638.

Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C., … Sonuga-Barke, E. J. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian Adoptees study pilot. *Journal of Child Psychology and Psychiatry, 50*(8), 943–951.

Meyer, A. (2017). A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. *Developmental Cognitive Neuroscience, 27*, 58–68. https://doi.org/10.1016/j.dcn.2017.08.001

Milad, M. R., & Quirk, G. J. (2012). Fear extinction as a model for translational neuroscience: Ten years of progress. *Annual Review of Psychology, 63*, 129–151.

Mischel, W., Shoda, Y., & Rodriguez, M. I. (1989). Delay of gratification in children. *Science, 244*(4907), 933–938. https://doi.org/10.1126/science.2658056

Mills, K. L., Goddings, A.-L., Clasen, L. S., Giedd, J. N., & Blakemore, S.-J. (2014). The developmental mismatch in structural brain maturation during adolescence. Developmental Neuroscience, 36(3–4), 147–160.

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., … Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences of the United States of America, 108*(7), 2693–2698. https://doi.org/10.1073/pnas.1010076108

Molenaar, P. C. (2004). A manifesto on psychology as idiographic science: Bringing the person back into scientific psychology, this time forever. *Measurement, 2*(4), 201–218.

Monk, C. S., Nelson, E. E., McClure, E. B., Mogg, K., Bradley, B. P., Leibenluft, E., … Pine, D. S. (2006). Ventrolateral prefrontal cortex activation and attentional bias in response to angry faces in adolescents with generalized anxiety disorder. *The American Journal of Psychiatry, 163*(6), 1091–1097. https://doi.org/10.1176/appi.ajp.163.6.1091

Morales, S., Fu, X., & Pérez-Edgar, K. E. (2016). A developmental neuroscience perspective on affect-biased attention. *Developmental Cognitive Neuroscience, 21*, 26–41. https://doi.org/10.1016/j.dcn.2016.08.001

Morales, S., Pérez-Edgar, K. E., & Buss, K. A. (2015). Attention biases towards and away from threat mark the relation between early dysregulated fear and the later emergence of social withdrawal. *Journal of Abnormal Child Psychology, 43*(6), 1067–1078. https://doi.org/10.1007/s10802-014-9963-9

Morales, S., Ram, N., Buss, K. A., Cole, P. M., Helm, J. L., & Chow, S.-M. (2017). Age-related changes in the dynamics of fear-related regulation in early childhood. *Developmental Science, 21*, e12633. https://doi.org/10.1111/desc.12633

Morales, S., Taber-Thomas, B. C., & Pérez-Edgar, K. E. (2016). Patterns of attention to threat across tasks in behaviorally inhibited children at risk for anxiety. *Developmental Science.* https://doi.org/10.1111/desc.12391

Morasch, K. C., & Bell, M. A. (2012). Self-regulation of negative affect at 5 and 10 months. *Developmental Psychobiology, 54*(2), 215–221. https://doi.org/10.1002/dev.20584

Moreira, J. F. G., & Silvers, J. A. (2018). In due time: Neurodevelopmental considerations in the study of emotion regulation. In P. M. Cole & T. Hollenstein (Eds.), *Emotion regulation* (pp. 111–134). New York, NY: Routledge.

Morgan, M. A., Romanski, L. M., & LeDoux, J. E. (1993). Extinction of emotional learning: Contribution of medial prefrontal cortex. *Neuroscience Letters, 163*(1), 109–113.

National Scientific Council on the Developing Child. (2010). Persistent fear and anxiety can affect young children. Retrieved November 15, 2018, from https://developingchild.harvard.edu/resources/persistent-fear-and-anxiety-can-affect-young-childrens-learning-and-development/

Nelson, C. A., & Dolgin, K. G. (1985). The generalized discrimination of facial expressions by seven-month-old infants. *Child Development, 56*(1), 58. https://doi.org/10.2307/1130173

Nelson, C. A., Thomas, K., & DeHaan, M. (2008). Neural bases of cognitive development. In W. Damon & R. M. Lerner (Eds.), *Child and adolescent development: An advanced course* (pp. 19–51). Hoboken, NJ: Wiley.

Noble, K. G., Houston, S. M., Kan, E., & Sowell, E. R. (2012). Neural correlates of socioeconomic status in the developing human brain. *Developmental Science, 15*(4), 516–527. https://doi.org/10.1111/j.1467-7687.2012.01147.x

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9*(5), 242–249.

Oler, J. A., Fox, A. S., Shelton, S. E., Rogers, J., Dyer, T. D., Davidson, R. J., … Kalin, N. H. (2010). Amygdalar and hippocampal substrates of anxious temperament differ in their heritability. *Nature, 466*(7308), 864–868. https://doi.org/10.1038/nature09282

Olsavsky, A. K., Telzer, E. H., Shapiro, M., Humphreys, K. L., Flannery, J., Goff, B., & Tottenham, N. (2013). Indiscriminate amygdala response to mothers and strangers after early maternal deprivation. *Biological Psychiatry, 74*(11), 853–860. https://doi.org/10.1016/j.biopsych.2013.05.025

Ordaz, S. J., Foran, W., Velanova, K., & Luna, B. (2013). Longitudinal growth curves of brain function underlying inhibitory control through adolescence. *Journal of Neuroscience, 33*(46), 18109–18124. https://doi.org/10.1523/JNEUROSCI.1741-13.2013

Öngür, D., & Price, J. L. (2000). The organization of networks within the orbital and medial prefrontal cortex of rats, monkeys and humans. *Cerebral Cortex, 10*(3), 206–219.

Østby, Y., Tamnes, C. K., Fjell, A. M., Westlye, L. T., Due-Tønnessen, P., & Walhovd, K. B. (2009). Heterogeneity in subcortical brain development: A structural magnetic resonance imaging study of brain maturation from 8 to 30 years. *Journal of Neuroscience, 29*(38), 11772–11782.

Papez, J. W. (1937). A proposed mechanism of emotion. *Archives of Neurology and Psychiatry, 38*(4), 725–743.

Pattwell, S. S., Duhoux, S., Hartley, C. A., Johnson, D. C., Jing, D., Elliott, M. D., … Lee, F. S. (2012). Altered fear learning across development in both mouse and human. *Proceedings of the National Academy of Sciences, 109*(40), 16318–16323. https://doi.org/10.1073/pnas.1206834109

Payne, C., Machado, C. J., Bliwise, N. G., & Bachevalier, J. (2010). Maturation of the hippocampal formation and amygdala in *Macaca mulatta*: A volumetric magnetic resonance imaging study. *Hippocampus, 20*(8), 922–935. https://doi.org/10.1002/hipo.20688

Peltola, M. J., Leppänen, J. M., Maki, S., & Hietanen, J. K. (2009). Emergence of enhanced attention to fearful faces between 5 and 7 months of age. *Social Cognitive and Affective Neuroscience, 4*(2), 134–142. https://doi.org/10.1093/scan/nsn046

Peltola, M. J., Leppänen, J. M., Palokangas, T., & Hietanen, J. K. (2008). Fearful faces modulate looking duration and attention disengagement in 7-month-old infants. *Developmental Science, 11*(1), 60–68. https://doi.org/10.1111/j.1467-7687.2007.00659.x

Penela, E. C., Walker, O. L., Degnan, K. A., Fox, N. A., & Henderson, H. A. (2015). Early behavioral inhibition and emotion regulation: Pathways toward social competence in middle childhood. *Child Development, 86*(4), 1227–1240.

Pérez-Edgar, K. E., Bar-Haim, Y., McDermott, J. M., Chronis-Tuscano, A., Pine, D. S., & Fox, N. A. (2010). Attention biases to threat and behavioral inhibition in early childhood shape adolescent social withdrawal. *Emotion, 10*(3), 349–357. https://doi.org/10.1037/a0018486

Pérez-Edgar, K. E., & Fox, N. A. (2005). Temperament and anxiety disorders. *Child and Adolescent Psychiatric Clinics of North America, 14*(4), 681–706. https://doi.org/10.1016/j.chc.2005.05.008

Pérez-Edgar, K., & Hastings, P. (2018). Emotion development from an experimental and individual differences lens. In J. T. Wixted (Ed.), *Stevens' handbook of experimental psychology and cognitive neuroscience* (4th ed., Vol. 4, pp. 289–322).

Pérez-Edgar, K. E., Roberson-Nay, R., Hardin, M. G., Poeth, K., Guyer, A. E., Nelson, E. E., … Ernst, M. (2007). Attention alters neural responses to evocative faces in behaviorally inhibited adolescents. *NeuroImage, 35*(4), 1538–1546. https://doi.org/10.1016/j.neuroimage.2007.02.006

Perlman, S. B., & Pelphrey, K. A. (2011). Developing connections for affective regulation: Age-related changes in emotional brain connectivity. *Journal of Experimental Child Psychology, 108*(3), 607–620.

Pessoa, L. (2008). On the relationship between emotion and cognition. *Nature Reviews Neuroscience, 9*(2), 148–158. https://doi.org/10.1038/nrn2317

Pessoa, L. (2017). A network model of the emotional brain. *Trends in Cognitive Sciences, 21*(5), 357–371. https://doi.org/10.1016/j.tics.2017.03.002

Pessoa, L., McKenna, M., Gutierrez, E., & Ungerleider, L. G. (2002). Neural processing of emotional faces requires attention. *Proceedings of the National Academy of Sciences, 99*(17), 11458–11463.

Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology, 57*(1), 27–53. https://doi.org/10.1146/annurev.psych.56.091103.070234

Phelps, E. A., Delgado, M. R., Nearing, K. I., & LeDoux, J. E. (2004). Extinction learning in humans. *Neuron, 43*(6), 897–905. https://doi.org/10.1016/j.neuron.2004.08.042

Phelps, E. A., & LeDoux, J. E. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. *Neuron, 48*(2), 175–187. https://doi.org/10.1016/j.neuron.2005.09.025

Piaget, J. (1954). *The Construction of Reality in the Child* (Cook, M, trans.). New York, NY, US: Basic Books.

Pine, D. S., & Fox, N. A. (2015). Childhood antecedents and risk for adult mental disorders. *Annual Review of Psychology, 66*(1), 459–485. https://doi.org/10.1146/annurev-psych-010814-015038

Posner, M. I., Rothbart, M. K., Sheese, B. E., & Voelker, P. (2014). Developing attention: Behavioral and brain mechanisms. *Advances in Neuroscience, 2014*, e405094. https://doi.org/10.1155/2014/405094

Qin, S., Young, C. B., Supekar, K., Uddin, L. Q., & Menon, V. (2012). Immature integration and segregation of emotion-related brain circuitry in young children. *Proceedings of the National Academy of Sciences, 109*(20), 7941–7946.

Raper, J., Wilson, M., Sanchez, M., Machado, C. J., & Bachevalier, J. (2013). Pervasive alterations of emotional and neuroendocrine responses to an acute stressor after neonatal amygdala lesions in rhesus monkeys. *Psychoneuroendocrinology, 38*(7), 1021–1035. https://doi.org/10.1016/j.psyneuen.2012.10.008

Rothbart, M. K., Sheese, B. E., Rueda, M. R., & Posner, M. I. (2011). Developing mechanisms of self-regulation in early life. *Emotion Review, 3*(2), 207–213. https://doi.org/10.1177/1754073910387943

Rothbart, M. K., Ziaie, H., & O'Boyle, C. G. (1992). Self-regulation and emotion in infancy. *New Directions for Child and Adolescent Development, 1992*(55), 7–23.

Roy, A. K., Benson, B. E., Degnan, K. A., Perez-Edgar, K., Pine, D. S., Fox, N. A., & Ernst, M. (2014). Alterations in amygdala functional connectivity reflect early temperament. *Biological Psychology, 103*, 248–254.

Roy, A. K., Shehzad, Z., Margulies, D. S., Kelly, A. M. C., Uddin, L. Q., Gotimer, K., … Milham, M. P. (2009). Functional connectivity of the human amygdala using resting state fMRI. *NeuroImage, 45*(2), 614–626. https://doi.org/10.1016/j.neuroimage.2008.11.030

Ruff, H. A., & Capozzoli, M. C. (2003). Development of attention and distractibility in the first 4 years of life. *Developmental Psychology, 39*(5), 877–890. https://doi.org/10.1037/0012-1649.39.5.877

Salm, A. K., Pavelko, M., Krouse, E. M., Webster, W., Kraszpulski, M., & Birkle, D. L. (2004). Lateral amygdaloid nucleus expansion in adult rats is associated with exposure to prenatal stress. *Developmental Brain Research, 148*(2), 159–167. https://doi.org/10.1016/j.devbrainres.2003.11.005

Sameroff, A. (2010). A unified theory of development: A dialectic integration of nature and nurture. *Child Development, 81*(1), 6–22. https://doi.org/10.1111/j.1467-8624.2009.01378.x

Scarr, S. (1992). Developmental theories for the 1990s: Development and individual differences. *Child Development, 63*(1), 1–19. https://doi.org/10.1111/j.1467-8624.1992.tb03591.x

Scarr, S., & Salapatek, P. (1970). Patterns of fear development during infancy. *Merrill-Palmer Quarterly of Behavior and Development, 16*(1), 53–90.

Schwartz, C. E., Kunwar, P. S., Greve, D. N., Kagan, J., Snidman, N. C., & Bloch, R. B. (2011). A phenotype of early infancy predicts reactivity of the amygdala in male adults. *Molecular Psychiatry, 17*(10), 1042–1050. https://doi.org/10.1038/mp.2011.96

Schwartz, C. E., Kunwar, P. S., Greve, D. N., Moran, L. R., Viner, J. C., Covino, J. M., … Wallace, S. R. (2010). Structural differences in adult orbital and ventromedial prefrontal cortex are predicted by 4-month infant temperament. *Archives of General Psychiatry, 67*(1), 78–84. https://doi.org/10.1001/archgenpsychiatry.2009.171

Schwartz, C. E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*(8), 1008–1015. https://doi.org/10.1097/00004583-199908000-00017

Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science, 300*(5627), 1952–1953. https://doi.org/10.1126/science.1083703

Sheridan, M. A., Fox, N. A., Zeanah, C. H., McLaughlin, K. A., & Nelson, C. A. (2012). Variation in neural development as a result of exposure to institutionalization early in childhood. *Proceedings of the National Academy of Sciences, 109*(32), 12927–12932.

Shonkoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., … Care, D. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics, 129*(1), e232–e246.

Silvers, J. A., Insel, C., Powers, A., Franz, P., Helion, C., Martin, R., … Ochsner, K. N. (2017). The transition from childhood to adolescence is marked by a general decrease in amygdala reactivity and an affect-specific ventral-to-dorsal shift in medial prefrontal recruitment. *Developmental Cognitive Neuroscience, 25*, 128–137.

Silvers, J. A., Insel, C., Powers, A., Franz, P., Helion, C., Martin, R. E., … Ochsner, K. N. (2016). vlPFC–vmPFC–amygdala interactions underlie age-related differences in cognitive regulation of emotion. *Cerebral Cortex, 27*(7), 3502–3514.

Silvers, J. A., Shu, J., Hubbard, A. D., Weber, J., & Ochsner, K. N. (2015). Concurrent and lasting effects of emotion regulation on amygdala response in adolescence and young adulthood. *Developmental Science, 18*(5), 771–784.

Sroufe, L. A. (1977). Wariness of strangers and the study of infant development. *Child Development, 48*(3), 731–746. https://doi.org/10.1111/1467-8624.ep10402182

Stifter, C. A., & Braungart, J. M. (1995). The regulation of negative reactivity in infancy: Function and development. *Developmental Psychology, 31*(3), 448–455. https://doi.org/10.1037/0012-1649.31.3.448

Sullivan, R. M., Hofer, M. A., & Brake, S. C. (1986). Olfactory-guided orientation in neonatal rats is enhanced by a conditioned change in behavioral state. *Developmental Psychobiology, 19*(6), 615–623. https://doi.org/10.1002/dev.420190612

Sullivan, R. M., & Holman, P. J. (2010). Transitions in sensitive period attachment learning in infancy: The role of corticosterone. *Neuroscience & Biobehavioral Reviews, 34*(6), 835–844. https://doi.org/10.1016/j.neubiorev.2009.11.010

Suzuki, H., Luby, J. L., Botteron, K. N., Dietrich, R., McAvoy, M. P., & Barch, D. M. (2014). Early life stress and trauma and enhanced limbic activation to emotionally valenced faces in depressed and healthy children. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(7), 800–813.e10. https://doi.org/10.1016/j.jaac.2014.04.013

Swartz, J. R., Carrasco, M., Wiggins, J. L., Thomason, M. E., & Monk, C. S. (2014). Age-related changes in the structure and function of prefrontal cortex–amyg-

dala circuitry in children and adolescents: A multimodal imaging approach. *NeuroImage, 86*, 212–220. https://doi.org/10.1016/j.neuroimage.2013.08.018

Sylvester, C. M., Barch, D. M., Harms, M. P., Belden, A. C., Oakberg, T. J., Gold, A. L., … Pine, D. S. (2016). Early childhood behavioral inhibition predicts cortical thickness in adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry, 55*(2), 122–129. e1. https://doi.org/10.1016/j.jaac.2015.11.007

Taber-Thomas, B. C., Morales, S., Hillary, F. G., & Pérez-Edgar, K. E. (2016). Altered topography of intrinsic functional connectivity in childhood risk for social anxiety. *Depression and Anxiety, 33*(11), 995–1004.

Taber-Thomas, B. C., & Pérez-Edgar, K. E. (2015). Emerging adulthood brain development. In J. Arnett Jensen (Ed.), *The Oxford handbook of emerging adulthood* (pp. 126–141). New York, NY: Oxford University Press.

Tamnes, C. K., Walhovd, K. B., Torstveit, M., Sells, V. T., & Fjell, A. M. (2013). Performance monitoring in children and adolescents: A review of developmental changes in the error-related negativity and brain maturation. *Developmental Cognitive Neuroscience, 6*, 1–13. https://doi.org/10.1016/j.dcn.2013.05.001

Telzer, E. H., Mogg, K., Bradley, B. P., Mai, X., Ernst, M., Pine, D. S., & Monk, C. S. (2008). Relationship between trait anxiety, prefrontal cortex, and attention bias to angry faces in children and adolescents. *Biological Psychology, 79*(2), 216–222. https://doi.org/10.1016/j.biopsycho.2008.05.004

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*(2/3), 25–52. https://doi.org/10.2307/1166137

Todd, R. M., Cunningham, W. A., Anderson, A. K., & Thompson, E. (2012). Affect-biased attention as emotion regulation. *Trends in Cognitive Sciences, 16*(7), 365–372. https://doi.org/10.1016/j.tics.2012.06.003

Tottenham, N., & Gabard-Durnam, L. J. (2017). The developing amygdala: A student of the world and a teacher of the cortex. *Current Opinion in Psychology, 17*, 55–60.

Tottenham, N., Hare, T. A., Quinn, B. T., McCarry, T. W., Nurse, M., Gilhooly, T., … Casey, B. J. (2010). Prolonged institutional rearing is associated with atypically large amygdala volume and difficulties in emotion regulation. *Developmental Science, 13*(1), 46–61. https://doi.org/10.1111/j.1467-7687.2009.00852.x

Tracey, I., Ploghaus, A., Gati, J. S., Clare, S., Smith, S., Menon, R. S., & Matthews, P. M. (2002). Imaging attentional modulation of pain in the periaqueductal gray in humans. *Journal of Neuroscience, 22*(7), 2748–2752.

van den Heuvel, M. I., Turk, E., Manning, J. H., Hect, J., Hernandez-Andrade, E., Hassan, S. S., … Thomason, M. E. (2018). Hubs in the human fetal brain network. *Developmental Cognitive Neuroscience, 30*, 108–115. https://doi.org/10.1016/j.dcn.2018.02.001

Valiente, C., Lemery-Chalfant, K., Swanson, J., & Reiser, M. (2008). Prediction of children's academic competence from their effortful control, relationships, and classroom participation. *Journal of Educational Psychology, 100*(1), 67–77. https://doi.org/10.1037/0022-0663.100.1.67

Whalen, P. J. (2004). Human amygdala responsivity to masked fearful eye whites. *Science, 306*(5704), 2061–2061. https://doi.org/10.1126/science.1103617

Zelazo, P. D. (2004). The development of conscious control in childhood. *Trends in Cognitive Sciences, 8*(1), 12–17. https://doi.org/10.1016/j.tics.2003.11.001

Zimmermann, P., & Iwanski, A. (2014). Emotion regulation from early adolescence to emerging adulthood and middle adulthood: Age differences, gender differences, and emotion-specific developmental variations. *International Journal of Behavioral Development, 38*(2), 182–194.