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The promise of infant MRI in psychiatry: toward a framework for neural network measures in early emotional and behavioral risk identification and new intervention targets

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Infancy marks a critical period of developing brain-behavior relationships that might influence the emergence of emotional and behavioral problems and psychopathology later in childhood and adolescence. In this review, we describe infant MRI studies that examined the development of neural networks, their associations with emerging emotional reactivity and regulation, and the relationships of caregiver factors, such as psychopathology, parenting behaviors and socioeconomic status (SES), with these developmental trajectories. We highlight the potential of utilizing infant MRI methodologies to identify key neural network structural and functional substrates of current and future emotional reactivity and regulation. Such an approach could identify early objective neural markers of dysregulation problems that are precursors of emotional and behavioral disorders, help monitor the effectiveness of existing interventions, and ultimately guide the development of new interventions for at-risk infants.

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INTRODUCTION

The rapid development of the human brain in the first years of life [1–4] determines critical brain-behavior relationships that set the stage for future clinical and functional outcomes. In particular, a parallel, growing literature highlights the critical role of the development of large-scale neural networks supporting early emotional reactivity and emerging emotional regulation (ER) in infancy [5–7]. Given that emotional *dys*regulation is a transdiagnostic risk factor for future child emotional problems and psychopathology [8–12], elucidating neural substrates/mapping brain markers early in development could aid identification of infants at risk before onset of disorder or manifestation of symptoms. However, these brain-behavior relationships also need to be considered in the context of caregiver characteristics such as the presence of postpartum psychopathology, the availability of responsive parenting, social and economic resources (SES) [13–16], each of which is known to play a significant role in shaping the infant's developing capacity for ER. Given the enormous neuroplasticity of the infant brain during the first year of life [17–21], intervening during this developmental window might have the potential to adaptively alter ER systems and decrease the likelihood of later psychiatric disorders [22]. Along these lines, understanding the contribution of modifiable caregiving factors to the development of infant neural networks supporting ER could also inform family-based intervention targets to improve the health and well-being of at-risk infants.

The goal of this review is to describe the current literature regarding associations among the development in infancy of large-scale neural networks and infant emotional reactivity and emerging ER, and the relationships of caregiver factors with these developing trajectories. We first summarize evidence showing that emotional dysregulation is a transdiagnostic risk for future psychopathology. We then describe the roles of large-scale neural networks in emotional reactivity and ER, and how these neural networks develop in the first years of life. We next present findings from studies that examined relationships among the development of these large-scale neural networks and emerging ER in infancy, and relationships among the development of these large-scale neural networks in infancy and future psychopathology later in childhood. Following this, we highlight how caregiver factors are associated with these developing brain-behavior relationships in infancy, and the roles of social contextual factors and the sex of the child in moderating these relationships. We conclude with a summary of these findings and limitations, future research directions and clinical implications.

DISRUPTED ER IN INFANCY IS A TRANSDIAGNOSTIC RISK FOR FUTURE PSYCHOPATHOLOGY

ER involves the integration of emotion and reward evaluative, attentional, self-referential monitoring, self-regulation/inhibition, and executive function processes that modulate or maintain the intensity and valence of emotional experiences [23, 24]. The Emotion Dynamics Model [25] provides a framework for operationalizing ER along key dimensions (e.g., intensity of emotional reactivity; latency from initial arousal to peak arousal intensity; duration of response; time to recovery). Thus, when the intensity, duration and/or rapidity of the emotional experience

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(e.g., frustration, joy) is 'too much' *or* 'too little' to function appropriately within a social environment, then ER is needed [24–27]. Positive and negative forms of emotional reactivity, i.e., positive and negative emotionality, can be measured reliably in human infants within the first months of life [28–31]. Infants displaying high levels of negative emotionality (NE) cry frequently and intensely in response to novelty and limitations and are difficult to soothe [32, 33]. These constitutionally-based behaviors are consistent across settings [34], and tend to be more common in males than females [35, 36]. Although there is modest rank-order stability in NE across the first years of life [37–39], this is superimposed on mean-level *increases* in NE during this period of development [28, 40–42]. High NE in infancy is also a robust predictor of behavioral and emotional problems later in childhood [33, 43–46] and even into adolescence and adulthood [47]. When assessed in the childhood years, NE predicts the development of depression and anxiety, suicidal behavior, behavior problems and substance abuse [48–55]. Thus, high infant NE represents an important early vulnerability factor for a broad range of later emerging functional impairments [56].

Displays of positive emotionality (PE) such as smiling, laughter and high intensity pleasure, may be orthogonal to NE [57, 58], and can also be assessed reliably in the first months of life [59, 60]. PE shows significant change across the first year [61]. Although less research has focused on the predictive utility of PE, low PE in infancy may predict behavioral inhibition in early childhood [8], whereas by middle childhood, low PE is associated with later depression [9–11, 62–64]. Alternatively, some research suggests that it is the ratio of low PE relative to high NE that represents the greatest risk for later depressive symptoms [9].

Infant efforts to self-regulate (e.g. self-soothing when distressed) are evident as early as the first few months of life [65]. Although developmental advances in the ability to regulate emotional responding may continue into adulthood [66], the most dramatic gains in ER capacities occur in the first few years of life [67]. Normative increases in ER capacities contribute to other development progressions, e.g., decreases in aggression and increases in compliance, and effective cognitive and social engagement [68, 69]. Conversely, deficits in ER in infancy are implicated in the etiology of childhood psychopathology [70–72], including aggression [12, 73, 74] and other disruptive behavior disorders [69, 72, 75, 76].

IN ADULTHOOD, LARGE-SCALE NEURAL NETWORKS PLAY IMPORTANT ROLES IN EMOTIONAL REACTIVITY AND ER

Neural networks contributing importantly to ER include: the medial prefrontal cortical (mPFC)-posterior cingulate/posterior parietal cortical (precuneus)-centered default mode network (DMN) [77, 78]; the dorsal and rostral anterior cingulate cortical (d/rACC)-anterior insula-centered salience network (SN) [79]; and the dorsolateral prefrontal cortex (dlPFC) and lateral posterior parietal cortex (lPPC)-centered central executive network (CEN)/ frontoparietal network (FPN) [79, 80].

The DMN has long been thought to support self-referential processing [81, 82]. More recently, however, the DMN has emerged as a key neural network in which different DMN nodes, including mPFC and posterior cingulate cortex (PCC), integrate self reference, social cognition, episodic memory, language and semantic memory to develop "frames of thought" within an internal narrative that are suppressed during cognitive task performance, but which can lead to mind wandering during rest or cognitively-undemanding contexts [83]. The internal narrative shaped by these cognitive processes is key to the development of a "sense of self" that in turn is a prerequisite for the capacity for ER and other adaptive regulatory processes [84].

Critically, the DMN is very closely connected with other neural networks, which facilitates the switching between the internally-driven processes subserved by the DMN nodes described above and externally-driven cognitive processes that are subserved by these other neural networks. A key neural network thought to control such switching is the SN, which detects the most contextually important information to guide behavior and social cognition [82, 85]. To facilitate this, the SN, and the anterior insula node in the SN in particular [86, 87], is thought to suppress the DMN and engage neural networks such as the CEN [88]. Here, the CEN dlPFC integrates information from multiple sources to support rule-based decision-making, the lPPC, attentional processes [89, 90], and the CEN as a whole, planning and top-down inhibitory control processes [91]. This Triple Network Model is supported by an increasing literature indicating inverse connectivity between the DMN and CEN during cognitive task performance [92–94], with the nodes within the DMN that subserve different internally-driven cognitive processes having specific patterns of connectivity with neural networks important for externally-driven cognitive task performance [95]. Furthermore, difficulty in disengaging the DMN, reflected in aberrant connectivity between the DMN and the CEN during cognitive task performance and rest, is evident in individuals with various psychiatric disorders, including mood and anxiety disorders [92, 93, 96–99]. These findings highlight the importance of the DMN and its context-dependent interactions with other neural networks in ER capacity [100–102]. It should be noted, however, that DMN nodes can show increased connectivity during task performance with cortical regions that are important for the organization of goal-directed behavior (multiple demand cortical regions centered on CEN dlPFC and dACC and SN anterior insula) [103] when it is important to combine cognitive processes subserved by the DMN (e.g., prior knowledge about the self) with the goals of the current task [104]. This aligns with the idea that the DMN is a central neural network for integrating external and internally-focused (i.e., self-related) information [105], which further underscores the importance of the DMN and its connectivity with other neural networks in ER and self-regulation processes in general.

The SN and CEN also have specific roles in ER. The SN is critical for attention to emotionally salient information, as well as switching between DMN-centered internally-focused and CEN-centered externally-focused processing, as described above. Other large-scale neural networks that are closely aligned and often equated with the SN include the cinguloopercular network (CO), centered on more dorsal regions of the ACC than the SN and the opercular region of the ventrolateral prefrontal cortex, and the ventral attention network (VAN), centered on the right ventro-lateral prefrontal cortex and temporoparietal junction [106, 107]. Together, these three closely aligned neural networks are important for attending to and integrating sensory, self-referential and emotionally-salient information with cognitive processes in order to guide complex behaviors such as social behavior that include ER [108]. Contributing to the specific role of the SN in ER is the amygdala, a key region supporting emotion processing [109] and a component of the SN [79, 82], which allows the SN to integrate emotional perceptual and motivational processes. Within the SN, the medial PFC, in particular the rostral ACC (rACC), is thought to modulate amygdala activity in response to emotionally salient stimuli [110], and has strong connectivity with CEN and SN prefrontal regions [111]. For example, more resilient individuals show greater mPFC-amygdala connectivity [112, 113], which is supported by a large rodent literature indicating greater mPFC (rACC and dACC homologs) projections to the amygdala in more resilient/ dominant animals [114–116]. The CEN has a critical role in ER via the roles of the dlPFC in integrating information from multiple sources and supporting rule-based decision-making and planning, the lPPC in attentional processes [89, 90], and the CEN as a whole, in planning and top-down inhibitory control processes [91], and reappraisal of negative emotional contexts [102, 117].

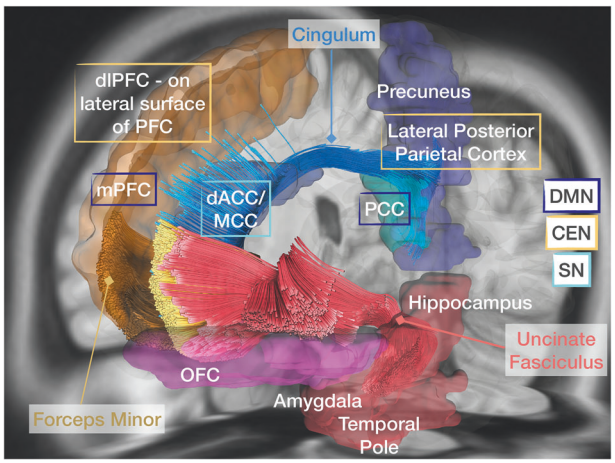

Fig. 1 Key regions and white matter (WM) tracts in large-scale neural networks important for emotion regulation (ER). [mPFC (Medial Prefrontal Cortex), dlPFC (Dorsolateral Prefrontal Cortex), dACC (Dorsal Anterior Cingulate Cortex)/MCC (Midcingulate Cortex), PCC (Posterior Cingulate Cortex), OFC (Orbitofrontal Cortex), DMN (Default Mode Network), CEN (Central Executive Network), SN (Salience Network)].

Major white matter (WM) tracts within and among these networks are the cingulum bundle (CB), connecting CEN parietal and prefrontal cortical regions [118]; the anterior corpus callosum (including the forceps minor; FM), providing lateral and medial prefrontal cortical interhemispheric connectivity [119] across the CEN and DMN; and the uncinate fasciculus (UF), which connects the temporal pole with the inferior frontal lobe and posterior orbitofrontal cortices (OFC) [120, 121], regions important for monitoring and learning the emotional (especially reward) value of stimuli [122] (Fig. 1).

The following sections focus on MRI studies that have advanced understanding of the development of these neural networks, and the relationships with developing ER capacity, in infancy.

FEASIBILITY OF MRI IN INFANTS

Recent and safe pediatric MRI developments have made it possible to conduct MRI studies in infants. For example, the majority of infant MRI studies that are included in this review (except for Ball et al. [123] who used sedation) [124] used the feed-and-bundle approach in order to ensure imaging quality by minimizing excessive movement without sedation. Additionally, pediatric imaging-optimized head coils [125] and acquisition sequences [126] have been implemented to enhance scanning quality while ensuring safety through low specific absorption rate (SAR) and fast acquisition time. Furthermore, manual inspection remains a standard quality control approach to ensure data integrity for subsequent processing and analysis. These infant MRI methodological developments have advanced understanding of the following early neurodevelopmental processes.

EARLY DEVELOPMENT OF LARGE-SCALE NEURAL NETWORKS

It is well-established that large-scale neural networks associated with emotional reactivity and ER are developing during the first years of life [6, 127–129] and continue throughout childhood and adulthood [130–134]. While sensorimotor, auditory and visual networks develop early [135–137], the DMN, SN and CEN, and amygdala connectivity with these large-scale networks, continue to develop during early childhood [138–140], including large increases in gray matter (GM) volume in cortical and subcortical regions in these networks in the first year [19, 129, 141] and into childhood [142–145]. In parallel, resting-state functional connectivity (rsFC) among these networks undergoes substantial developmental changes during infancy, initially strengthening within individual networks before increasing inter-network integration [146, 147]. Both intra- and inter-network changes predominantly reflect logarithmic patterns of growth in connectivity [148].

The DMN is initially weaker in neonates, but in infancy becomes more synchronized, particularly between mPFC and PCC regions [147, 149]. Overall adult-like DMN topology is reached around one year old, with continued maturity in the second year of life, mirroring the emergence of early self-referential and social cognitive processing [147, 149]. However, the DMN is still immature and/or sparsely connected until ages 7–9 and continues strengthening into early adolescence [150, 151]. The DMN is the fastest developing among the higher order networks, followed by the right CEN (frontoparietal), SN and the left CEN (frontoparietal) [6] with the CEN still being immature at the end of the first year [152]. The amygdala's connections with these networks also continue to develop throughout infancy and childhood, reflecting increasing functional specialization in affective and cognitive processing [104, 121].

The functional integration and roles of these networks evolve across development. Specifically, over the first two years of life, decreasing network-level connectivity occurs between DMN-SN and CEN-SN, reflecting the emergence of distinct specializations, while increasing connectivity occurs between right CEN-DMN and left and right CEN [6]. In childhood through emerging adulthood (ages 7–20), inter-network connectivity between higher-order large-scale networks undergoes much development [153], with children displaying weaker DMN-SN and CEN-SN connectivity compared with adults [82]. In particular, the flexibility of switching between distinct inter-network dynamics is weaker in childhood, with adults having rapid-switching of more transient connections between DMN, CEN and SN [154].

WM tracts in these networks develop throughout infancy [155–157]. Several tracts, including those described above, show significant increases in fractional anisotropy (FA, the ratio of longitudinal versus transverse water diffusivity, reflecting the degree of longitudinal fiber alignment/collinearity of fibers), and decreases in radial diffusivity (RD, the extent of transverse water diffusivity in WM tracts, thought to reflect non-collinearity of fibers and/or damage to myelin) during the first two years of life [158, 159]. Increases in FA in the majority of WM tracts, especially those connecting visual and subcortical regions implicated in emotional reactivity and ER, continue across the early school years [160–163]. These microstructural changes reflect the progressive assembly of pathways critical for integrating ER and executive function [18, 164].

The UF demonstrates rapid early development in infancy [18, 165]; maturation of this tract is particularly relevant for emotion-processing circuits, with aberrant UF microstructure linked to less ER capacity [166]. Similarly, the CB develops rapidly over the first year of life [18], and has a long developmental trajectory into adulthood [161] facilitating cognitive control and attentional flexibility [167]. The FM also undergoes significant maturation during infancy [18, 168], contributing to large-scale network integration. This tract plays an important role in the development of higher-order cognitive functions, including ER with patients with emotional dysregulation disorders displaying lower FM structural integrity [169]. These changes in WM microstructure correspond with increasing rsFC within and among the DMN, SN, and CEN [5, 170], with evidence suggesting that infants exhibiting stronger intra- and inter-network connectivity patterns demonstrate more stable regulatory behaviors later in infancy [170, 171].

RELATIONSHIPS AMONG THE DEVELOPMENT OF THESE LARGE-SCALE NEURAL NETWORKS AND ER IN INFANCY

A small but increasing number of studies have examined relationships among the development of these large-scale neural networks and ER in infancy [123, 170, 172–174]. We reported associations among greater amygdala-DMN and -SN, and lower amygdala-CEN, FC and lower infant positive emotionality (PE), which we replicated in an independent infant sample [94]. These findings parallel those from other infant studies that reported significant relationships between greater amygdala-SN and -DMN FC and greater NE [171, 175, 176]. One study, however, reported an association between greater DMN internetwork FC in early infancy and lower NE later in infancy [177]; and another study reported inverse associations among DMN, SN and CEN and novelty-evoked distress in 4-month infants [178]. Our group also showed associations between greater CB volume, potentially reflecting reduced synaptic pruning [179], and dampened concurrent PE [93]; and reported in 3-month infants that lower UF fiber collinearity predicted greater NE at 9-months [180]. Similarly, another study reported an association between lower early infant fiber collinearity in the inferior stria terminalis, a white matter (WM) tract connecting the amygdala and anterior hypothalamus, and greater infant fear later in infancy [181]. Using multimodal mediation, we further showed that lower rsFC between CEN and DMN structures suppressed the otherwise negative relationship between greater CB volume and dampened concurrent PE [93]. These findings highlight the complex interplay between infant emotionality, brain structure, and functional connectivity in large-scale networks during early development. Further, infant emotionality and rsFC share a bidirectional relationship, where early emotional behaviors can shape neural connectivity patterns in ER networks, and this dynamic interaction underscores the importance of neural plasticity in early emotional development [182].

In a recent study, we used Neurite Orientation Dispersion and Density Imaging (NODDI) to estimate the WM microstructural integrity and myelination of prefrontal cortical regions, using the neurite density index (NDI), and dispersion, using the orientation dispersion index (ODI). These indices of WM microstructure have potential to more accurately evaluate microstructural alterations in the developing brain [183, 184]. We examined relationships in infants among these measures and NE and PE, and reported positive associations among 3-month rACC ODI and cACC NDI and concurrent NE, and 3-month lateral orbitofrontal cortex (lOFC), a region important for decision-making about potential reward value [185, 186], ODI and prospective NE [187]. We also reported a negative association between 3-month dlPFC ODI and concurrent PE. These findings parallel those from our earlier studies of WM microstructure-NE and PE relationships in infants, as they suggest that greater NDI and ODI, reflecting greater microstructural complexity and, likely, more diffuse patterns of connectivity, among prefrontal cortical regions supporting salience perception (rACC), decision-making (lOFC), action selection (cACC), and attentional processes (dlPFC) might result in greater integration of these prefrontal cortical regions with other neural networks, greater attention to salient negative external cues, and thus higher NE and/or lower PE. Furthermore, we have recently shown that in infancy larger increases in ODI in major WM tracts interconnecting neural networks supporting ER, in particular the UF, FM and CB, are associated with disrupted developmental changes, i.e., smaller increases or larger decreases, in PE and ER [188].

We also reported that greater 3-month DMN medial superior frontal cortical volume was associated with higher infant 3-month NE, and that greater 3-month SN and/or ventral attention network (VAN) ventrolateral prefrontal cortical volume predicted lower infant 9-month PE, even after controlling for 3-month PE, highlighting the importance of these latter neural measures in explaining PE *change*. These findings were replicated in an independent sample [189]. These results add to our previous findings by indicating that greater GM volume in prefrontal cortical regions important for salience perception and attention also predispose to higher levels of infant NE and lower levels of infant PE with increasing age. Other findings indicate positive and inverse associations among subcortical GM volumes and indices of NE in infants [190].

Overall, findings from the above studies suggest that higher levels of NE and lower levels of PE in infancy are associated with: 1. greater levels of endogenous functional connectivity, measured by rsFC, among the amygdala and regions within the DMN and SN, and among regions within the CEN and DMN; 2. lower collinearity (i.e., more diffuse structural connectivity) of fibers in WM tracts among the CEN and DMN (e.g., the CB) and WM tracts connecting the amygdala with the hypothalamus, with worsening ER associated with increasing magnitude of these alterations in WM microstructure during infancy; and 3. greater GM volume in prefrontal cortical regions important for salience perception and attention. One interpretation of these findings is that these patterns of connectivity, WM and GM might result in greater interference by interoceptive and salience processing on executive processing, resulting in greater attention to potentially threatening/worrying emotional stimuli and expression of higher levels of NE and lower levels of PE. Interestingly, greater UF volume was significantly associated with lower concurrent infant NE; but lower orbitofrontal cortex (OFC)-amygdala rsFC, suppressed this otherwise negative relationship, while greater OFC-CEN rsFC mediated this relationship [93]. This finding likely reflects the more specific role of the UF in ER, as it connects prefrontal cortical regions implicated in the evaluation of emotional value of stimuli with the amygdala [191, 192].

RELATIONSHIPS AMONG DEVELOPMENT OF THESE LARGE-SCALE NEURAL NETWORKS AND PSYCHOPATHOLOGY RISK

A small number of studies report prospective relationships among infant development of large-scale neural networks, and amygdala rsFC with these networks, and psychopathology risk in early childhood [193–195]. Other findings indicate inverse relationships among rsFC among regions in the DMN and SN in infancy and behavioral inhibition at 2 years [170]. Furthermore, there are positive associations among infant amygdala-SN FC and internalizing behaviors in the first 2 years, while inverse associations were found with infant striatal-DMN FC [176, 196, 197]. Additionally, greater levels of atypical development of infant amygdala-DMN FC were associated with greater anxiety at 4 years [198]. Other work has reported prospective associations with other aspects of neurodevelopment. For example, greater amygdala volume in early infancy predicted poorer working memory, a key executive function process, in girls at three years of age [199]. Regarding WM, smaller corpus callosum length measured by cranial ultrasound in 7-week-old infants was associated with greater executive functioning deficits at 4 years of age [200]. Yet, few studies examined associations among large-scale neural networks in infancy and indices of future psychopathology later in childhood. Determining these relationships is a critically important, yet understudied, step toward identifying neural markers of pathophysiological processes in infancy that predispose to future mental health problems in childhood.

ASSOCIATIONS BETWEEN CAREGIVER DEPRESSION AND ANXIETY AND INFANT NEURAL NETWORK-ER RELATIONSHIPS

To a large extent, adaptive development of ER systems in infancy is shaped by the quality of interactions with primary caregivers [201, 202]. Developmental studies are consistent in documenting

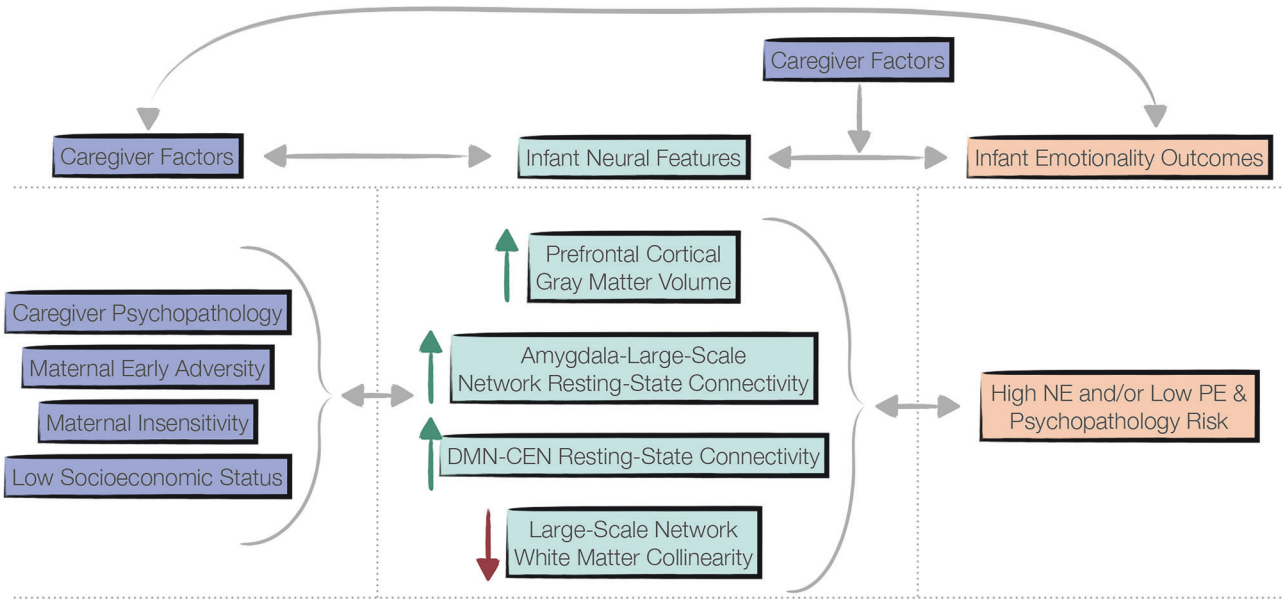

Fig. 2 Schematic depiction of demonstrated relationships between various caregiver factors and major infant neural features contributing to emotional reactivity outcomes and future psychopathology risk. While this review focuses on the influence of caregiver factors on infant neural network emotional behavior relationships, it is also acknowledged that bidirectional relationships exist among these factors. [DMN - default mode network, CEN - central executive network, NE - negative emotional reactivity, PE - positive emotional reactivity].

the adverse effects of caregiver (typically maternal) depression and anxiety in the first postpartum year on infant ER [203, 204], and associations with offspring social, emotional and behavioral problems later in childhood [205, 206].

In the field of infant MRI, several studies have reported associations between caregiver depression and anxiety, and other indices of caregiver distress, and the development of GM and WM in neural networks supporting ER in infancy [16, 190, 207–212]. In parallel, prenatal maternal depression is associated with greater infant amygdala connectivity with various SN and DMN nodes above and beyond any influence of postnatal depression [213]. These amygdala connectivity patterns are recapitulated later in life among those with major depressive disorder [214, 215]. Furthermore, sensitive parenting has been shown to affect infant ER-related neurodevelopment. For example, maternal sensitivity (timely and appropriate responsiveness to infant signals) was associated with 5-month-old infant DMN rsFC, indicating that infants of more sensitive mothers displayed greater intra-DMN connectivity [14]. Interestingly, greater maternal sensitivity was also associated with greater 6-month hippocampus (a less-well characterized zone of the DMN [216]) connectivity with CEN regions, as well as with lower amygdala connectivity with DMN-related regions [13].

Critically, an emerging literature is exploring how these caregiver factors may help shape developing neural network-ER relationships in infancy. Here, studies reported significant relationships among exposure to postpartum depression, infant WM microstructure and infant NE [217]. Our own work, supported by others [218], shows that more severe postpartum depression and anxiety are associated with greater amygdala-DMN and amygdala-SN, and lower amygdala-CEN, rsFC in 3-month-olds, which in turn are associated with lower levels of infant PE (even after accounting for parenting behaviors) [94], suggesting that caregiver depression and anxiety associate with large-scale neural networks supporting emotional reactivity in early infancy.

Indirect evidence for the effects of caregiver stress, distress or challenges to parenting on infant neural network-ER relationships are suggested by studies that have reported associations between early adversity and infant neural network-ER relationships. Here, maternal childhood and adolescence adverse experiences are associated with smaller neonatal amygdala volume and greater negative emotionality in 6-month old infants. Furthermore, childhood emotional neglect in the mother relates to stronger neonatal rsFC among the amygdala and prefrontal cortical regions in large-scale neural networks including the CEN and SN [219]. Financial strain in the family and/or low SES is also known to affect infant neurodevelopment in regions with broad implications for attention, emotional reactivity, ER and psychopathology, including temporal pole, inferior frontal and anterior cingulate regions [15, 220]. Additionally, prenatal exposure to neighborhood crime was associated with weaker neonatal thalamic-DMN and amygdala-hippocampus rsFC [221].

In addition to the potential modifying effects of environmental factors such as caregiver psychopathology, parenting quality and the broader social context (e.g., financial strain, neighborhood stressors), genetic factors are also likely to play a role. For example, there is a strong genetic component to brain structure and function as evidenced by twin and family studies, with heritability estimates for brain volumes and cortical measures typically ranging from 60–80% [222, 223]. Structural and functional connectivity also show substantial heritability varying by brain white matter tract and network; heritable patterns of functional connectivity were found within the DMN and CEN, with network subregions also showing heritable intra-network connectivity [223, 224]. About half of the inter- and intra-network connectivity of canonical resting-state networks show up to 53% heritability, with stable genetic influences during adolescence [225]. A multigenerational longitudinal study found that individuals at high familial risk for depression show increased DMN connectivity and decreased DMN-CEN negative connectivity, indicating potential biomarkers for depression risk [226]. Despite increasing interest in elucidating environmental and genetic risk factors that influence the development of large-scale neural networks subserving ER in infancy through childhood, many gaps remain. In particular, work is urgently needed to understand the ways in which caregiver resilience, positive well-being and social support could serve as buffers in contexts of stress to support adaptive infant ER development.

While these findings highlight the importance of caregiver emotional reactivity and the caregiving environment on

Table 1. MRI Studies Examining Infant Brain-NE, PE, and ER Relationships.

Authors	Participant Age	MRI Modality	Emotional Outcomes	Major Findings	Caregiver Factors
Graham et al. [171]	4 weeks	rsFC	↑ 6-month fear	↑ amygdala-SN rsFC	None
Thomas et al. [176]	4 weeks	rsFC	↑ 6-month NE	↑ amygdala-SN rsFC	None
Phillips et al. [94]	3 months	rsFC	↓ PE	↑ amygdala-DMN and -SN rsFC ↓ amygdala-CEN rsFC	↑ EPDS, trait anxiety and ↑ amygdala-SN rsFC ↑ EPDS, trait anxiety and ↓ amygdala-CEN rsFC
Filippi et al. [175]	4 months	rsFC	↑ NE	↑ amygdala-DMN and -SN rsFC	None
Ravi et al. [177]	1 month	rsFC	↓ 6-month NE	↑ DMN internetwork rsFC	None
Filippi et al. [178]	4 months	rsFC	↑ novelty-evoked distress	↓ DAN-DMN, -SN, and -CEN rsFC	None
Banihashemi et al. [180]	3 months	dMRI	↑ 9-month NE	↓ UF fiber collinearity	↓ 3-month EPDS and ↑ NE
Nolvi et al. [217]	3 weeks	dMRI	↑ 6-month NE	↑ (whole brain FA, corpus callosum FA, CB FA) × 6-month EPDS	↑ 3- and 6-month EPDS and ↑ 6-month NE
Banihashemi et al. [93]	3 months	dMRI	↓ PE	↑ CB volume	↑ PAI-BOR affective instability and ↑ NE
Planalp et al. [181]	1 month	dMRI	↑ 6-month fear	↓ inferior stria terminalis fiber collinearity	None
Zhang et al. [187]	3 months	dMRI	↑ NE	↑ rACC ODI and cACC NDI	↑ caregiver age, trait anxiety, government assist sum and ↑ NE
			↑ 9-month NE	↑ lOFC ODI	↑ 3-month trait and ↑ 9-month NE
			↓ PE	↑ dlPFC ODI	↓ caregiver age, government assistance sum and ↓ PE
Zhang et al. [188]	3 and 9 months	dMRI	↑ 3-to-9-month decrease or ↓ increase in NE	↑ 3-to-9-month increases in right UF, FM, and left CB ODI	None
			↓ 3-to-9-month increase in PE	↑ 3-to-9-month increases in right UF NDI	None
Bezanson et al. [190]	3 months	sMRI	↑ 9-month NE	↑ left pallidum volume ↓ left accumbens, bilateral hippocampi volumes	↑ state anxiety and ↑ right pallidum volumes
Zhang et al. [189]	3 months	sMRI	↑ NE	↑ DMN SFC volume	↑ trait anxiety and ↑ NE
			↓ 9-month PE	↑ SN and/or VAN vlPFC volume	None

EPDS Edinburgh Postnatal Depression Scale, *PAI-BOR* Personality Assessment Inventory–Borderline Features Scale, *DAN* Dorsal Attention Network, *sMRI* structural MRI, *dMRI* diffusion MRI, *NDI* Neurite Density Index, *ODI* Orientation Dispersion Index.

developing infant neural network-ER relationships, it is noteworthy that relationships exist between infant emotional reactivity and ER and caregiver neural activity, where infant emotional behavior can activate caregiver neural networks that support parenting behaviors [227–229]. Furthermore, there are well-documented effects of infant emotional reactivity on caregiving affective responses and parenting behaviors [230, 231]. Together, these findings therefore highlight the presence of bidirectional relationships among caregiver affective responses and associated caregiver neural activity, and infant neural network activity and ER. These bidirectional relationships should be the focus of future studies.

SEX OF THE INFANT EFFECTS ON THE DEVELOPMENT OF NEURAL NETWORK-ER RELATIONSHIPS

There are effects of offspring sex on neurodevelopment that vary across studies [16, 199, 212, 232–235]. For example, we reported stronger positive relationships between postpartum depression and anxiety and infant amygdala-SN, and amygdala-CEN rsFC, and between the latter infant rsFC measures and lower infant PE, in male relative to female infants [94]. Other research showed stronger relationships between lower amygdala-CEN WM FA and externalizing behaviors in boys than girls [233], while other studies showed stronger effects of early financial strain and maternal depression on neurodevelopment in girls than in boys [212, 235]. Further examination of contextual factors and offspring sex on neural indices of ER in infancy through early childhood is clearly warranted.

A FRAMEWORK FOR UNDERSTANDING NEURODEVELOPMENTAL TRAJECTORIES UNDERLYING EMERGING NE, PE AND ER IN INFANCY, AND FOR IDENTIFYING NEURAL INDICES OF FUTURE PSYCHOPATHOLOGY RISK

Emotional dysregulation, evidenced by high NE and low PE in infancy, is a robust predictor of behavioral and emotional problems later in childhood [8, 33, 45, 46] and even into adolescence and adulthood [47], and thus represents an early transdiagnostic marker of psychopathology [70–72]. Findings from MRI studies of infants indicate that large-scale networks, including the DMN, SN and CEN, are critical substrates of emotional expressivity and ER [100–102], along with the major WM tracts forming the connections within and between each network, including the CB, FM and UF. These large-scale neural networks develop and increase in synchrony by 1 year, with increasing network-level integration [6, 149] and continued maturation beyond the first postnatal year. Findings suggest that higher levels of NE and lower levels of PE in infancy are associated with greater functional connectivity among the amygdala and regions within the DMN and SN, and among regions within the CEN and DMN. This is accompanied by lower collinearity of fibers in WM tracts among the CEN and DMN and greater GM volume in prefrontal cortical regions important for salience perception and attention. Such integration of structure and function facilitates greater maladaptive CEN-DMN connectivity and integration of prefrontal cortical regions with other neural networks, yielding greater attention to salient negative external cues, and thus higher NE and/or lower PE (Fig. 2; Table 1).

Critically, these indices of neural network structure and function have been shown to be linked with future affective outcomes and psychopathology risk. For example, aberrant infant DMN-SN and amygdala-DMN rsFC were associated with greater childhood behavioral inhibition [170] and anxiety [198], respectively. Furthermore, caregiver factors (e.g., symptoms of psychopathology, parenting behaviors) and limited resources (e.g., low SES) also influence ER-related infant neurodevelopment in ways that recapitulate the infant neural patterns described above, including greater amygdala-large-scale network rsFC [13, 94, 219], and in some studies, indicating that these patterns contribute to high NE and/or low PE [94].

LIMITATIONS AND FUTURE DIRECTIONS

Limitations of the research to date include, in some cases, small sample sizes and/or samples with unbalanced male/female offspring ratios, as well as inherent sampling bias in infant and caregiver participant recruitment and the exclusion of infants who were unable to remain still during scans. Furthermore, some studies examining relationships among social contextual factors and indices of large-scale network structure and function in infancy do not then extend neural findings to examining relationships with measures of infant emotional reactivity or ER. Moreover, future work in larger samples, and studies utilizing advances of integrated multimodal imaging to examine global structure-function relationships will have a beneficial effect on the field, increasing our understanding of neural targets for intervention or prevention of deficient ER in infancy. Additionally, as dysregulated emotional and physiological response to stress in infancy is linked to later negative affective and behavioral outcomes [236–239], more research is necessary to understand contributions of proximal stress-control neural structures [240, 241] in infancy to other stress response systems and the interactions between infant stress reactivity and ER-related processes.

CONCLUSION AND FUTURE CLINICAL IMPLICATIONS

Together, findings to date highlight the promise of infant MRI as a valuable approach that can identify key neural network structural and functional correlates of current, and predictors of future, NE, PE and ER (Fig. 2; Table 1). This approach has great potential to provide neural markers to guide early emotional and behavioral disorder risk identification, help monitor the effectiveness of interventions to ameliorate aberrant NE, PE and ER in infancy, and ultimately help to develop new, targeted interventions that are based on an understanding of underlying neural network abnormalities in infancy.

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AUTHOR CONTRIBUTIONS

MLP (Conceptualization, writing—original draft, review and editing); AEH (Conceptualization, writing—original draft, review and editing); LB (Figures, writing—additional sections, review and editing); YZ (Table, writing—additional sections, review and editing). All authors approved the final version of the manuscript.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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