Current Psychiatry Reports (2026) 28:64
https://doi.org/10.1007/s11920-026-01715-y

REVIEW

Maternal Neuroplasticity: Understanding Brain Changes with Perinatal Depression and Anxiety in Women

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Received: 22 April 2026 / Accepted: 13 August 2026

Abstract

Purpose of the review The transition to parenthood is a unique period of adulthood during which the brain undergoes profound and rapid changes, particularly in birthing mothers. This transition is also associated with a risk of developing mental illness, likely due to intersections among the neurobiological changes and a host of psychosocial factors including one's history of mental illness and lack of adequate support systems for parents.

Recent findings Emerging evidence indicates that perinatal depression and anxiety are associated with distinct neural alterations that overlap with the brain areas also involved in caregiving. Thus, there is a critical need to better understand the neurobiological mechanisms underlying perinatal mental illness for both maternal and child wellbeing. The goals of this review are to highlight key findings from current research on the ways in which human brain function and structure are associated with (1) perinatal depression, (2) perinatal anxiety, and (3) treatments aimed at promoting parental mental health.

Summary Advances in understanding the neuroplasticity associated with perinatal depression and anxiety will inform the development of targeted interventions and treatments aimed at supporting parental wellbeing, promoting healthy parent–infant relationships, and improving peripartum brain health.

Keywords Anxiety · Brain Health · Depression · EEG · MRI · Matrescence · Mental Health

Introduction

The transition to motherhood (i.e., matrescence) is the only period in adulthood when the brain exhibits such tremendous structural and functional changes within a relatively short time. In recent years, there has been a much better understanding of the parental brain in humans that complements the decades of research on non-human animals showing that the transition to parenthood, particularly in birthing mothers, is associated with widespread neuroplasticity [1–4]. Notably, the transition to parenthood is associated with a significant decrease in grey matter volumes of many brain areas across pregnancy as well as a heightened activity to infant cues in many brain areas during the postpartum period [2, 3]. Given this remarkable adaptive plasticity of the parental brain, which occurs alongside other biological and psychosocial changes experienced by most parents [5], it may not be surprising that the transition to parenthood is also a time of vulnerability to mental illness, particularly in mothers.

Recent research suggests a unique neurobiology of perinatal mental illness that overlaps with brain areas critical for parenting, emphasizing the urgency to understand the parental brain in illness as well as in health [6, 7]. Current scientific literature often points to an association between perinatal mental illness and changes in brain structure and function. It remains to be determined, however, if such neuroplasticity predicts mental health outcomes or vice versa. The goals of this review are to highlight key findings from current research on the ways in which human brain function and structure are associated with (1) perinatal depression, (2) perinatal anxiety, and (3) treatments aimed at promoting

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Published online: 20 August 2026

parental mental health. Our focus will be on mothers, given that all extant research on this topic has involved birthing women. However, there is a clear need for further research on the neuroplasticity of perinatal mental illness in adoptive parents and biological fathers [4, 8].

Furthermore, it should be noted that there are several other mental illnesses associated with the transition to motherhood, but there are limited data on related neurobiological change. For more information about parental brain changes associated with postpartum psychosis, childbirth-related PTSD, opioid use disorder, childhood trauma, borderline personality disorder, and pregnancy loss please see [9–12].

Perinatal Depression (PND) and the Parental Brain

Perinatal depression (PND) is associated with unique functional and structural neuroplastic changes in the human brain [2, 11, 13]. Much of this research has focused on neurobiological features associated with *postpartum* depression (PPD), and additional research is needed to understand how depression may impact the normative neuroplasticity of pregnancy and vice versa.

PND and Maternal Brain Function

Functional magnetic resonance imaging (fMRI), functional near-infrared spectroscopy (fNIRS), electroencephalography (EEG), or more specific event-related potentials (ERP) have regularly been used to investigate associations between maternal depressive symptoms and brain function. The majority of this research has been done in women during the postpartum period with depressive symptoms or a diagnosis of PPD [6, 7].

PND and fMRI/fNIRS

Findings from resting-state fMRI, task-based fMRI, and fNIRS from women with PPD collectively support altered communication in neural networks regulating self-referential processing, salience detection, executive regulation, reward processing, emotional memory, and social cue integration (Table 1).

One of the brain networks regularly implicated in PPD is the Default Mode Network (DMN), which includes the medial prefrontal cortex (mPFC), posterior cingulate cortex, precuneus, lateral parietal cortex, and middle temporal regions [32]. The DMN supports self-referential processing, autobiographical memory integration, and narrative identity construction [32]. These mental processes are especially relevant during the postpartum period, when maternal identity formation and reinterpretation of self in relation to the infant are ongoing. One study reported that higher medial frontal local synchronization at rest was correlated with depressive symptoms shortly after giving birth [14]. Women diagnosed with PPD, however, show less synchronization in the prefrontal cortex, particularly for those with higher depressive symptoms [33]. This abnormal activity within frontal region might impair cognitive control favouring rumination [33]. Other studies show increased DMN–frontoparietal network functional connectivity in women diagnosed with PPD, suggesting excessive influence of internally focused processes over executive regulation systems [17]. Impaired network switching or connectivity between DMN and ventral attention, sensorimotor, and visual networks has also been reported in PPD [17, 20, 31]. Together, these findings indicate altered coordination among attentional, sensory, and social-cognitive systems in women with PPD. This *potentially* affects maternal detection, interpretation, and behavioral responses to infant-related cues, although it remains to be determined how these functional changes are specifically linked to caregiving.

The Salience Network (SN) - anchored in the anterior insula (AI) and anterior cingulate cortex (ACC; particularly the subgenual ACC; sgACC) - detects emotionally relevant stimuli and coordinates switching between internally focused and externally directed brain systems [34]. In the postpartum period, this network can be critical for prioritizing infant cues and emotion regulation. Mothers with PPD show hyperconnectivity between the AI and sgACC at rest compared to mothers without PPD, suggesting biased processing of negative emotions [18] and potential sensitization to negative emotional contexts during mother–infant interactions.

The Fronto-Parietal Network (FPN) - including the dorsolateral prefrontal cortex, middle frontal gyrus, inferior frontal gyrus, and posterior parietal regions - supports cognitive flexibility, inhibitory control, and top-down emotion regulation [35]. Effective maternal executive control is essential during the postpartum period for regulating emotional responses and shifting attention between internal and caregiving demands. Alterations within the FPN at rest have been reported in women diagnosed with PPD suggesting diminished flexibility within the network [17, 27, 33]. Task-based findings have shown reduced frontal activation to positive relative to negative infant cues in women reporting higher depression symptoms in the postpartum period, indicating preferential sensitivity to negative contexts [26]. This altered functioning in brain areas important for emotion regulation and cognitive flexibility in mothers with PPD or high depression symptoms postnatally potentially compromises maternal responsiveness to infant needs.

Table 1 Summary of Recent Studies on Functional and Structural Alterations Associated With Perinatal Depression and/or Anxiety

Network	Canonical Function	Key Alterations (↑/↓)	Phenotype	Perinatal Period	Reference
Default Mode Network (DMN)	Self-referential processing, autobiographical memory, maternal identity integration	↑ SMFG local connectivity	PPD - Higher depression symptoms	Postpartum	[14]
		↓ LPC–PCC connectivity	Remitted depression	Postpartum	[15]
		↓ mPFC–PCC/mPFC–PCL/mPFC–AG FC/mPFC entropy	PPD	Postpartum	[16]
		↑ FPN–DMN FNC	PPD	Postpartum	[17]
		↑ DMN→FPN effective connectivity	PPD & PPD-A	Postpartum	[17]
		↓ sgACC–MTG connectivity	PPD & PPD-A	Postpartum	[18]
		↑ hippocampus–precuneus/SFG FC	PPD	Postpartum	[19]
		↓ MTG–precuneus FC	PPD	Postpartum	[20]
Salience Network (SN)	Emotional relevance detection, interoception, network switching	↑ sgACC–vAI static FC	PPD	Postpartum	[18]
		↑ sgACC dynamic ALFF	PPD-A	Postpartum	[18]
		↓ VMHC (insula, amygdala, medial frontal); normalized post-SAINT	PPD	Postpartum	[21]
Fronto-Parietal Network (FPN)	Cognitive flexibility, inhibitory control, top-down regulation	↓ DLPFC ReHo	PPD	Postpartum	[19]
		↑ DLPFC fALFF	PPD	Postpartum	[22]
		↑ DLPFC–ACG FC	PPD	Postpartum	[23]
		↓ MFG synchronization	PPD	Postpartum	[17]
		↑ vlPFC FCS	PPD-A	Postpartum	[24]
		↓ SMN→FPN effective connectivity	PPD-A	Postpartum	[17]
		↓ DLPFC activation (fNIRS during positive FAST)	Pregnancy - Higher depression symptoms	Pregnancy	[25]
		↓ IFG activation (fNIRS during negative FAST)	Pregnancy - Higher depression symptoms	Pregnancy	[25]
		↓ response to positive infant cues	Higher depression/anxiety symptoms	Postpartum	[26]
		↓ dALFF variability	PPD	Postpartum	[27]
Reward/Motivational Circuit	Reward anticipation, motivational salience	↑ VS FCD	PPD-A	Postpartum	[24]
		↓ VS–dmPFC rsFC	PPD & PPD-A	Postpartum	[24]
Amygdala, Hippocampus	Emotional salience encoding, affective memory, hormone sensitivity	↓ Right amygdala response to positive faces	History of PPD	Parous women	[28]
		↑ Amygdala activation	Pregnancy – Higher depression symptoms	Pregnancy	[29]
		↓ Lateral amygdala GMV	PPD	Postpartum	[30]
		↑ Hippocampal ReHo & DC	PPD	Postpartum	[19, 21]
		↑ Hippocampal FC	PPD	Postpartum	[21]
		↑ Parahippocampus FCS	PPD	Postpartum	[24]
Attention Networks (DAN/VAN)	External attention allocation & salience-driven attention	↑ Precuneus (DAN) synchronization	PPD	Postpartum	[17]
		↓ FPN–VAN FNC	PPD	Postpartum	[17]
		↓ VAN–DMN FNC	PPD	Postpartum	[17]
		↓ VAN↔SMN effective connectivity	PPD & PPD-A	Postpartum	[17]
		↑ VIS–VAN FNC	PPD-A	Postpartum	[17]
		↓ VIS–DAN FNC	PPD-A	Postpartum	[17]
Sensorimotor Network (SMN)	Embodied simulation, motor readiness	↓ Precentral gyrus fALFF & ReHo	PPD	Postpartum	[22]
		↓ dALFF variability	PPD	Postpartum	[27]
		↑ PrCG–DCG FC correlated with EPDS	PPD	Postpartum	[23]
Visual Network (VIS)	Sensory-affective integration	↑ VIS internal FNC	PPD	Postpartum	[17]
		↓ DMN↔VIS effective connectivity	PPD	Postpartum	[17]

Table 1 (continued)

Network	Canonical Function	Key Alterations (↑/↓)	Phenotype	Perinatal Period	Reference
Social Cognition Network	Mentalizing, infant cue interpretation	↓ TPJ–lateral PFC connectivity	Higher depression symptoms	Postpartum	[31]
		↑ TPJ–mPFC connectivity	Higher depression symptoms	Postpartum	[31]
		↓ MTG activation (fNIRS during neutral FAST)	Pregnancy - Higher depression symptoms	Postpartum	[25]

ACG, Anterior Cingulate Gyrus; *AG*, Angular Gyrus; *DAN*, Dorsal Attention Network; *DCG*, Dorsal Cingulate Gyrus; *dALFF*, Dynamic Amplitude of Low-Frequency Fluctuations; *DLPFC*, Dorsolateral Prefrontal Cortex; *DMN*, Default Mode Network; *EPDS*, Edinburgh Postnatal Depression Scale; *FAST*, Free Association Semantic Task; *FC*, Functional Connectivity; *FCD*, Functional Connectivity Density; *FCS*, Functional Connectivity Strength; *FNC*, Functional Network Connectivity; *fALFF*, Fractional Amplitude of Low-Frequency Fluctuations; *FPN*, Fronto-Parietal Network; *IFG*, Inferior Frontal Gyrus; *LPC*, Lateral Parietal Cortex; *MFG*, Middle Frontal Gyrus; *mPFC*, Medial Prefrontal Cortex; *MTG*, Middle Temporal Gyrus; *PCC*, Posterior Cingulate Cortex; *PCL*, Paracentral Lobule; *PPD*, Postpartum Depression; *PPD-A*, Postpartum Depression with Anxiety; *PrCG*, Precentral Gyrus; *ReHo*, Regional Homogeneity; *rsFC*, Resting-State Functional Connectivity; *SFG*, Superior Frontal Gyrus; *sgACC*, Subgenual Anterior Cingulate Cortex; *SMFG*, Superior Medial Frontal Gyrus; *SMN*, Sensorimotor Network; *SN,* Salience Network; *TPJ*, Temporoparietal Junction; *vAI*, Ventral Anterior Insula; *VAN*, Ventral Attention Network; *VIS*, Visual Network; *VMHC*, Voxel-Mirrored Homotopic Connectivity; *VS*, Ventral Striatum

The brain's reward network - including the anterior cingulate cortex, ventral striatum, and midbrain dopaminergic sites - mediates reward anticipation and motivational salience [36] and is thus vital to maternal caregiving and parent-infant bonding. Functional imaging has revealed higher ventral striatal connectivity at rest in mothers with PPD and anxiety, but reduced ventral striatal to dorsal mPFC connectivity in mothers with PPD with and without anxiety, suggesting impaired integration between motivational and higher-order reflective and regulatory systems compared to healthy mothers [24]. Such imbalance may impact women's caregiving motivation and induce reactive emotional responses to infant distress. Task-based studies involving positive words, an infant cry, an infant smiling face, and even a monetary reward have also reported decreased activity in the brain reward network in mothers with PPD or high levels depressive symptoms postpartum compared to healthy mothers [37].

Functional alterations in the amygdala and hippocampus of women with PPD implicate emotional memory and stress regulation systems in maternal mental health and caregiving concerns. For example, mothers with a history of PPD or PPD symptomology show less amygdala activation in response to a positive emotional stimulus (adult happy face vs. neutral) [38] and to a threat stimulus (threatening words) compared with women without PPD [39], which suggest information processing bias, with reduced attention to positive stimuli and blunted responses to threats in PPD. However, when exposed to an infant's emotional cues such as infant smiling face or infant cry, mothers with depressive symptoms showed higher amygdala activity compared to what was seen in mothers without depressive symptoms postpartum [40]. This research underscores the importance of cue type when characterizing the association between PPD and maternal brain function, and shows that some brain responses in mothers with PPD are atypically heightened in response to infant cues. Findings from the hippocampus extend these alterations to brain networks involved in memory and stress axis regulation, with studies reporting greater hippocampal centrality and enhanced hippocampus–DMN connectivity at rest [21, 33] in mothers with PPD compared with mothers without PPD. Collectively, these results suggest that PPD is associated with functional reorganization involving altered amygdala activation patterns and increased hippocampal connectivity, but that this activity varies depending on whether the response is to an infant cue or a non-infant cue as well as if the measurement is done at rest.

Taken together, PPD appears to involve multi-network imbalance that may undermine flexible maternal identity formation, emotion regulation, and adaptive responsiveness during matrescence (Fig. 1). Whether these functional brain changes are evident with antenatal depression remain to be determined.

PND and EEG

Changes in brain activity with perinatal mental illness have also been detected by EEG. EEG determines brain activity by detecting electrical signals across a larger brain region, as opposed to more targeted blood oxygenation or blood flow changes revealed by fMRI or fNIRS. Several studies examining maternal EEG activity show links between perinatal depressive symptoms and maternal right frontal alpha activity (see [41] for a review), with more recent efforts attempting to leverage larger-scale EEG networks in the context of maternal depression (e.g. [42]),.

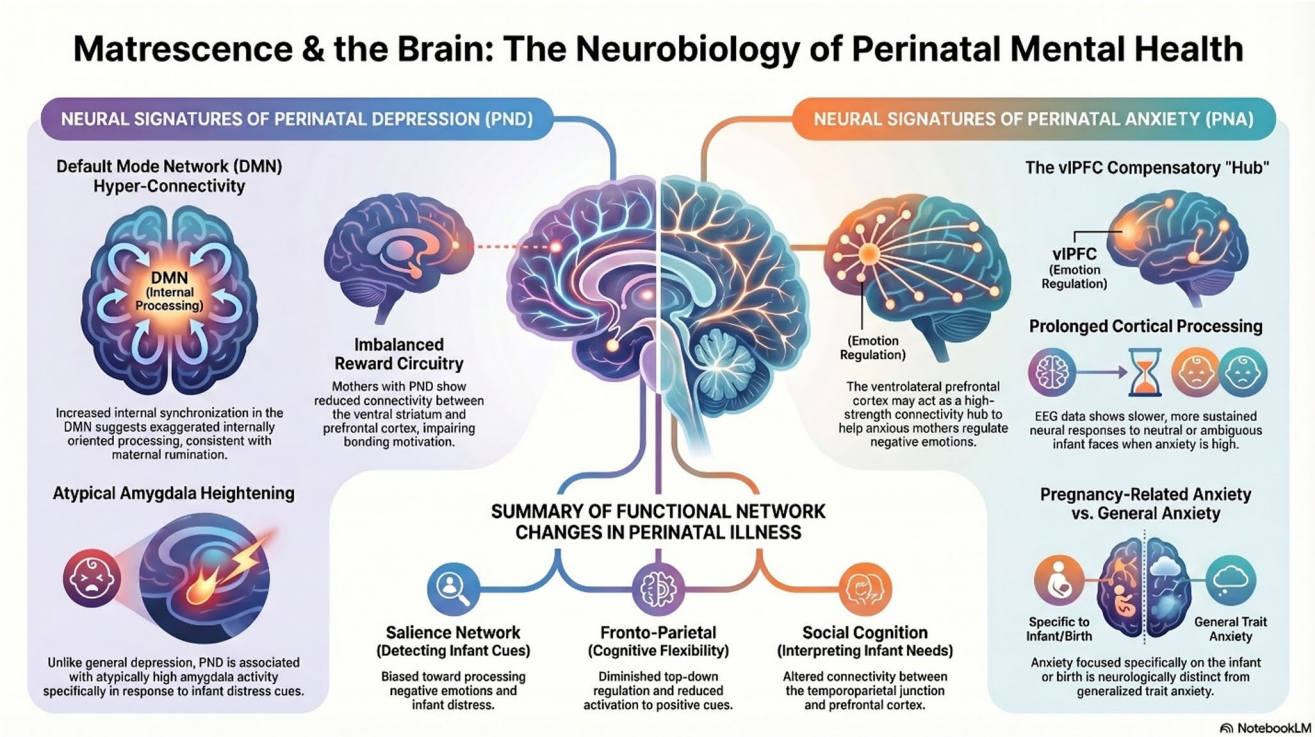

Fig. 1 A summary of key factors that can contribute to maternal brain health. A growing body of research is elucidating how perinatal mental illness, psychotherapeutic interventions, and medications are associated with plasticity in the parental brain. To promote parental brain health, we need to continue to understand how these factors affect the neurobiological determinants of parenting. (created with NotebookLM)

Event-related potentials (ERPs), measures of brain electrical activity occurring in response to a specific event or stimulus, are derived from EEG and have been used to examine the temporal dynamics of neural processes in the context of maternal mental health [43]. Common ERPs, based on specific peaks or waves of EEG activity, are the P300 related to attention allocation and the N170 linked to face recognition. Higher levels of antenatal depressive symptoms are associated with blunted P300 responses to infant distress faces, but not happy or neutral infant faces or to infant cries [44]. This suggests that antenatal depressive symptoms affect neural processing of infant distress cues at the level of visual attention allocation. In turn, these effects may be more selective to a mothers' initial attention to infant faces as recent work found that antenatal depressive symptoms in pregnancy women are not associated with sustained attentional processing of infant faces (captured by the LPP ERP; [45]).

How these EEG changes in pregnancy relate to postpartum infant care remains to be determined, but assessment of ERPs during pregnancy has been leveraged to predict postpartum depression symptoms [46] as well as mother-infant bonding [47]. For example, in healthy mothers, there is an association between changes in particular EEG measures in response to infant faces (e.g., P1 and P2 ERPs) from pregnancy to postpartum and bonding with the infant [47]. Additionally, higher antenatal depressive symptoms are associated with stronger negative association between P300 in pregnancy and postpartum depressive symptoms [46]. Taken together, these studies point to the potential of ERPs in pregnancy being utilized as predictive tools for later risk of PPD and mother-infant bonding.

Other research focused on the *postpartum* period showed that maternal postpartum depression symptoms were related to larger N170 responses to infant faces in mothers, although the same was also found in non-mothers [48]. Higher postpartum levels of depression (as well as anxiety and substance use) also contribute to membership in latent profiles of psychological risk, are associated with slower neural responses to infant faces (N170), and are related to challenges in maternal caregiving such as lower certainty about their infant's mental states [49].

PND and Maternal Brain Structure

There is an emerging collection of brain structural features associated with *postpartum* depression. Mothers

with PPD show greater cortical thickness in the frontal gyrus and fusiform gyrus at two months postpartum [50] and greater volumes of the dorsolateral PFC and insula three months postpartum compared to mothers without PPD [51]. These brain areas are involved in decision making, emotion regulation and empathy, which are essential for parenting. In line with this, mothers diagnosed with PPD within one year of giving birth had decreased cortical thickness in brain areas involved in sensory processing, such as the parietal lobe, and greater surface area of brain regions involved in empathy, such as the insula [19] compared to mothers without PPD. In addition, higher levels of depressive symptoms were associated with increased surface area of the insula in mothers diagnosed with PPD [19, 51]. This research suggests that the typical decrease in brain structure during the transition to motherhood may not occur to the same degree in mothers diagnosed with PPD.

Studies have also aimed to understand whether brain structural changes during pregnancy or the early postpartum are predictive of, or only associated with, depression. Interestingly, increased amygdala volume from late pregnancy to the early postpartum period is associated with higher levels of depressive symptoms across the same time period in healthy mothers not diagnosed with PPD [52]. However, brain structure in the first week postpartum in 16 brain areas of interest, including the amygdala, hippocampus, and nucleus accumbens, was not associated with the development of postpartum depression or anxiety assessed a few months later [15]. While these results could indicate that a clear link between brain structural changes in pregnancy and PPD does not yet exist, it may instead be the case that a single timepoint in the very early postpartum period is already too late to detect predictive relationships between maternal brain structure and depressive symptoms measured soon thereafter.

In addition to understanding the structural changes during pregnancy and the early postpartum period that may be associated with PND, one study has aimed to determine whether brain structure associated with PPD can predict treatment outcomes. This study took place in an inpatient parent-child care unit and they found that mothers diagnosed with PPD who responded to the treatment (combination of medication, CBT, group therapy, and physiotherapy) with a decrease in depressive symptoms had a smaller hippocampus before treatment when compared to the non-responders [53]. In contrast, smaller hippocampal volume predicted poorer response to treatment in people with MDD outside the postpartum period [53], again pointing to a uniqueness of the neurobiology of maternal mental illness.

Perinatal Anxiety (PNA) and the Parental Brain

Anxiety disorders occur in at least 15% of women during the perinatal period [54]. In addition, up to one-half of women with PND also have clinically significant anxiety symptoms or an anxiety disorder and this comorbidity can reach 70% in certain subtypes of PPD [55, 56]. However, most studies of the neurobiology of perinatal mental illness do not assess anxiety symptoms specifically. For those that do, anxiety is typically involved as a covariate to help pinpoint the influences of depressive symptoms (e.g. [57]), or is included in a latent variable of distress that cannot distinguish among the mental health variables (e.g. [58]). While there remains a paucity of research on this maternal mental health issue, it is encouraging that the number of studies on the neurobiological correlates of high perinatal anxiety (PNA) has recently been increasing (e.g. [6, 59, 60]),. Importantly, none of the recent studies have compared a group of mothers with clinically diagnosed generalized anxiety (but no other psychopathology) with a healthy control group, although most of the studies involved some participants above the typical questionnaire cutoffs for high generalized anxiety symptoms. It should also be noted that due to the diagnosis criteria requiring 6 months of clinically relevant symptoms, many women during pregnancy and the postpartum period are living with a burden of anxiety symptoms that are often overlooked but contribute to more sever clinical profile [61].

PNA and Maternal Brain Function

PNA and fMRI

In a study examining functional connectivity strength in groups of 3–4 month postpartum women with PPD only, PPD plus generalized anxiety, or no mental health diagnosis, mothers with both PPD and generalized anxiety had the highest connectivity strength in their ventrolateral prefrontal cortex (vlPFC) [24]. Although this experimental design prohibits understanding the unique effects of postpartum anxiety alone on brain connectivity, the findings suggest that the vlPFC may be a compensatory "hub" engaged by women with high mental health burden while they process their negative emotions. The same group of researchers [24] reported elsewhere that PPD with generalized anxiety was also associated with stronger long-range connections involving the ventral striatum, with particularly strong functional connectivity between the ventral striatum and the ventrolateral prefrontal cortex. They again proposed that this strengthening may be compensatory for postpartum women suffering with both depression and generalized anxiety as they attempt to regulate their negative affect.

Cortical-amygdalar activity in response to infant cues is also influenced by postpartum anxiety. In mothers viewing negative and positive affective faces of their own and unknown 3-month-old infants, lower differential frontal lobe activation between the infants' negative and positive emotional states predicted high general anxiety symptoms more than one year later [26]. Furthermore, while listening to one's own or an unknown infant's cries, amygdala functional connectivity with the superior and middle frontal cortices as well as with the middle temporal cortex is stronger in anxious postpartum mothers [62], possibly reflecting differences in attention, social cognition, and impulse control. On the other hand, no association was found between state anxiety and fMRI activity or connectivity in any brain site studied in postpartum mothers listening to their own and other infants' cries [63]. This latter study did find, however, a significant moderating influence of state anxiety on the relation between amygdala functional connectivity with the posterior superior temporal sulcus (involved in social cognition) during cry sessions and maternal caregiving observed during a mother-infant free play. Because anxiety positively moderated the amygdala-temporal sulcus to positive caregiving relationship in high-anxiety mothers, but negatively moderated it in low-anxiety mothers, the authors proposed that anxious postpartum mothers may over-engage that pathway to help promote positive parenting [63].

It is important to recognize that perinatal anxiety is not unitary and that anxieties specific to motherhood [64] may be associated with particular brain functioning. For instance, infant-focused anxiety postpartum is positively associated with connectivity between the ACC and insula or putamen at rest in mothers with and without opioid use disorder [65]. It is unknown if these infant-focused anxiety results extend to generalized anxiety, but the results may reflect differences in affective and motivational processing between mothers who are more and are less confident in their caregiving role. This same study also found that magnitude of change across the first few months postpartum in resting state functional connectivity between the amygdala and the midbrain periaqueductal gray, a brain area regulating defensive behaviour, was especially large in women with the highest infant-focused anxiety and could indicate that they neurobiologically process the infant as a threat. Lastly, data-driven modeling to determine functional connectivity across whole brain networks associated with caregiving found that higher infant-focused anxiety postpartum was related to higher connectivity between the cerebellar and motor–sensory–auditory networks, and between the frontoparietal and motor–sensory–auditory networks, even when controlling for generalized anxiety and depression symptoms. However, these strengths particularly decreased from 2 to 8 months postpartum in women with less anxiety toward the infant at 8 months [66].

PNA and EEG

Maternal EEG responses to infant faces involve a greater LPP (but not N170 or P300) to infant neutral but not distress faces in women with high anxiety during *pregnancy* [67] and the postpartum period [68] (also see[69] for replication in mothers of school-aged children), and this effect remains significant when controlling for depression symptoms [67]. This result may reflect more prolonged cortical processing of ambiguous infant faces when anxiety, but not depression, is high. This relationship between anxiety and the LPP was not found, however, in a later analysis this group conducted involving a smaller sample of pregnant women [67], highlighting the importance of robust sample sizes when examining links between the maternal brain and mental health. Anxiety symptoms were also found to influence the LPP while women attended to infant distress faces interspersed with infant cry stimuli. This suggests that studies using more naturalistic, multi-sensory experiences may best reveal how perinatal anxiety influences the maternal brain [45].

EEG resting state beta activity (involved in cortical attentional control and hyperarousal) measured in late pregnancy was also positively associated with maternal antenatal generalized anxiety [70], but the effects were no longer significant when controlling for depression symptoms or other covariates. Interestingly, beta activity was instead negatively associated with pregnancy-related antenatal anxiety that involves women's specific concerns about fetal and future infant health, the process of giving birth, changes in physical appearance, and competence in being a mother (as specifically mentioned above), even when depression and other covariates were included. Although pregnancy-related antenatal anxiety is significantly correlated with women's general anxiety symptoms [64], pregnancy-related anxiety is a unique predictor of some negative outcomes for mothers and their infants [71].

Recent EEG studies involving *postpartum* anxiety symptoms are more nuanced in their findings. In a longitudinal study involving mid-pregnancy EEG responses to infants or inanimate objects to predict concurrent or postpartum anxiety, greater LPP to images of infants with happy expressions significantly predicted higher postpartum anxiety symptoms, although the investigators noted that differences in LPP accounted for only a small amount of the variance in later postpartum anxiety levels, so was not a major predictor [46]. Similar to studies of depression, anxiety has also been linked to patterns of frontal alpha asymmetry in mothers of 9-month-old infants [72],

as well as mu EEG rhythm that captures a neural marker of processing non-verbal signals [73]. Two other perinatal longitudinal studies assessing EEG responses to infant emotional faces [47] or at rest [74] in late pregnancy and again a few months postpartum found no relationship between the EEG and anxiety measures, consistent with one postpartum cross-sectional study [75]. Across studies, it is worth noting that the sometimes restricted range of maternal anxiety symptoms may add to the nuance and variability among findings, necessitating greater heterogeneity in mental health symptoms in addition to enrolling women with a clinically diagnosed anxiety disorder.

PNA and Maternal Brain Structure

Very little research has investigated how anxiety may be related to structural brain changes across pregnancy and the postpartum period in mothers. However, recent research on the structure of the cerebral cortex during *pregnancy* found higher antenatal trait anxiety, but not general negative affect, was strongly associated with greater gray matter volume in the supramarginal gyrus – a region involved in social cognition and empathy [76]. This finding aligns with what has been found in non-pregnant anxious or depressed people [77]. Additionally, an 8-week mindfulness program aiming at reducing anxiety during pregnancy reported that pregnant women with higher levels of antenatal anxiety had greater surface area in the right superior frontal cortex during the third trimester, a brain area that plays a role in the regulation of attention and impulse control [78]. We have yet to understand how *postpartum* anxiety may alter the structure of the maternal brain; however, recent research shows that low state anxiety in mothers during the first six weeks postpartum is associated with an enlargement of the superficial amygdala, a brain area that processes olfactory inputs and is activated while experiencing a variety of emotional states. This association was not found for the centromedial or basolateral amygdala regions, and could indicate that mothers with low anxiety undergo optimal superficial amygdala plasticity during the early postpartum period not found in mothers with higher anxiety [79] (Fig. 1).

Are Perinatal Depression and Anxiety Neurobiologically Unique from Depression and Anxiety at Other Times of the Lifespan?

Most of the studies reviewed above focused on mothers with high and low depression or anxiety symptoms; however, an important question is whether brain features differ between women with PND or PNA and non-mothers with high depression or anxiety outside of the postpartum period. This would aid in our ability to develop appropriate and specific treatments for perinatal mental illnesses. Indeed, there is some work showing that although similar brain areas are involved in depression during the postpartum period and other times in life, the way these brain areas process information can be quite different [38, 80]. For example, mothers with PPD have greater right amygdala activation in response to an infant cue compared to non-mothers with major depression, showing that the brain response to an infant cue differs if depression occurs during the postpartum period or at other times in life [80]. Reasons for these differences in brain activation with motherhood remain to be determined, but despite PPD and major depression sharing similar symptomology, they are differentiated by timing of onset, physiological profiles, the presence of an infant, and the excessive worry and/or guilt surrounding parenting abilities that occurs in many women with PPD [80]. Furthermore, as noted above, pregnancy-specific anxiety and generalized anxiety measured concurrently are not completely correlated with each other and are associated with some differences in neurobiological features.

Promoting Mental Health in Parents: Limitations and Future Directions

Our understanding of the neuroplasticity of perinatal mental illness is in its infancy and much more research on the neural correlates of perinatal mental illness in all parents is warranted. Specifically, future research in this area would benefit from an increase in both longitudinal and cross-sectionals designs, larger sample sizes and an attempt to assess the multiple factors contributing to perinatal mental health such as early life adversity, social isolation and pre-conception mental health.

It is arguably more effective to support and treat parents struggling with perinatal mental illness during pregnancy and the early postpartum period than to combat the multiplicative problems that untreated perinatal mental illness can contribute in the parent's and child's life beyond the peripartum period. Interventions and treatments can be, and should be, multifaceted, focusing on several biological and social factors associated with perinatal mental illness; these include neurosteroid and neurotransmitter dysregulation, social isolation, relationship distress, poverty, early life adversity, racism, and sexism [5, 81]. Indeed, simply improving symptoms of mental illness alone does not improve maternal self-efficacy whereas interventions that involve couples or entire families have the potential for greater improvements [82]. What is interesting to note is that a range of interventions can modify

the maternal brain and improve neurobiological functioning. These include psychotherapeutic interventions [83], mindfulness interventions [84], pharmacotherapies targeting particular neurochemical systems in the parental brain [85–87], and interventions directly targeting the brain such as transcranial direct current stimulation (tDCS) [88–90]. The mechanisms underlying how these treatments affect the parental brain are mostly unknown.

Conclusions

The adult human brain of birthing (and also non-birthing [91]) parents undergoes significant modifications in function and structure with the healthy transition to parenthood. Many of these brain areas are critical for parenting but are also involved in perinatal mental illness. Given the high rates of perinatal depression and anxiety, there is an urgency to improve the well-being of parents and positively affect the next generation as well as generations to come. To do this, understanding the neurobiological underpinnings of perinatal mental health in all parents should be the focus of further research, to develop more targeted brain-based interventions to promote parental brain health. We need to continue implementing evidence-based interventions and treatments at multiple levels (e.g., individual, institutional, and community) during pregnancy and the postpartum period that support parenting. These include easier access to healthcare and childcare, to name a few. Understanding the parental brain is fundamental to society and supporting its development should be our priority.

Author contributions JLP was invited to write the article and proposed the initial topic and outline. All authors performed a literature search, wrote sections of the manuscript and critically revised the manuscript. SA created the table. JSL and JLP significantly contributed to editing the final draft of the manuscript.

Data Availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The author declares that no competing financial interests exist. JLP is the scientific advisor at Strategies for Moms. HJVR receives support from Tonix Pharmaceuticals.

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