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The Silent Imprint: Exploring Maternal PTSD and Its Effects on Fetal Neurodevelopment

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Abstract
The prenatal period is a critical window for human brain development, marked by both remarkable potential and heightened vulnerability. No other period in a child's life matches the speed of neurological growth occurring in the first nine months in utero. Emerging research has highlighted prenatal maternal psychological distress, including anxiety, depression, and post-traumatic stress disorder (PTSD) as a significant influence on fetal neurodevelopment, with implications for child emotional, behavioral, and cognitive outcomes. This encyclopedia entry synthesizes findings from recent studies on the biological, epigenetic, and neurodevelopmental consequences of prenatal exposure to maternal PTSD and psychological distress.

Keywords: maternal; PTSD; prenatal; neurodevelopmental; fetus; brain

Introduction

The prenatal period represents a critical window for brain development, during which foundational neural structures and pathways begin to form. This phase, characterized by rapid cellular growth and complex neural circuit formation, is one of heightened vulnerability to environmental influences [1, 2]. Among these influences, the maternal intrauterine environment plays a central role in shaping fetal neurodevelopment.

A growing body of research highlights that maternal psychological distress during pregnancy, including anxiety, depression, and PTSD can alter the fetal neurodevelopmental trajectory, increasing the risk of adverse developmental outcomes [2–4, 7]. Evidence suggests that heightened maternal stress during pregnancy may influence fetal brain development, affecting both neural structure and emerging brain function [3].

Structural and functional changes in key brain regions, such as the hippocampus, amygdala, and cerebellum, have been observed through neuroimaging studies in infants and children exposed to high maternal distress during gestation [2, 6, 12]. These regions, known to be rich in glucocorticoid receptors, are especially sensitive to elevated levels of maternal cortisol, a hormone closely linked to the stress response and capable of crossing the placenta [4, 7].

The mechanisms linking maternal psychological distress to fetal neurodevelopment are complex and multifactorial. Proposed pathways include dysregulation of maternal psychological stress systems, impaired placental functioning, increased maternal inflammation, and epigenetic modifications such as altered deoxyribonucleic acid (DNA) methylation of stress- related genes (e.g., NR3C1). Together, these processes may contribute to the long- term alterations in neurodevelopment [13].

The clinical implications of these prenatal alterations extend beyond birth. Children exposed to maternal distress in utero may show long- term differences in brain structure, attention regulation, emotional reactivity, and cognitive development, with effects lasting into adolescence and early adulthood [9, 13]. Moreover, maternal trauma history, such as childhood interpersonal trauma, has been shown to intensify the effects of prenatal anxiety on infant attention and behavior regulation [13].

Given these consequences, there is a growing emphasis on early interventions and preventive strategies. Addressing maternal health before and during pregnancy offers a promising avenue to support fetal brain development and reduce the risk of long- term neurodevelopmental challenges in children [3, 7, 10, 11].

Definition

Maternal psychological distress during pregnancy refers to a range of emotional and psychological conditions experienced by expectant mothers, including anxiety, depression, and trauma- related disorders. Among these, PTSD represents a particularly severe form of distress that

can develop following exposure to traumatic or life-threatening events [14].

PTSD during pregnancy is characterized by symptoms such as intrusive memories, hyperarousal, avoidance behaviors, and persistent negative emotional states. These symptoms are frequently accompanied by physiological changes, including elevated cortisol levels, sleep disturbances, and increased inflammatory activity, all of which may influence the intrauterine environment and fetal development [3]. Prenatal exposure to maternal stress and trauma has been linked to an increased risk of neurodevelopmental and psychiatric conditions in offspring, including anxiety disorders, depression, attention- deficit/ hyperactivity disorder (ADHD), autism spectrum disorder, and schizophrenia [3]. These associations highlight the importance of understanding maternal psychological health as a critical factor in early developmental programming.

This entry examines the relationship between maternal psychological distress during pregnancy particularly PTSD and fetal brain development. It reviews the biological mechanisms through which maternal stress may influence neurodevelopment, discusses potential long- term outcomes for offspring, and explores current approaches to prevention and intervention.

Body

Psychological distress during pregnancy is a multifaceted condition that encompasses stress, anxiety, and depression [2]. It ranges from subclinical symptoms to diagnosable disorders such as major depressive disorder or PTSD [2, 13]. Prevalence estimates vary considerably across studies due to differences in study populations, timing of assessment during pregnancy, and the screening tools used to measure anxiety symptoms. Nevertheless, systematic reviews suggest that 14–54% of pregnant women experience anxiety, while up to 18% experience depression [2]. PTSD, in particular, affects a subset of women with histories of trauma; approximately 4–6% of adult women meet the diagnostic criteria for PTSD, with lifetime trauma exposure rates ranging between 50% and 70% [5, 7].

Among pregnant women, especially those from high-risk groups, the prevalence of PTSD symptoms can be as high as 27–58% [7]. PTSD in pregnancy rarely occurs as a standalone condition and frequently co-occurs with other forms of maternal psychological distress, including depressive symptoms and anxiety, reflecting the broader pattern of comorbid mental health conditions observed during the perinatal period [2]. Studies also show that women with PTSD or a history of childhood trauma (CT) tend to have lower socioeconomic status, higher parity, and more medical comorbidities, including gestational diabetes and autoimmune diseases, all of which may compound fetal risk by contributing to a more stressful intrauterine environment and increasing the likelihood of adverse neurodevelopmental outcomes [4, 10].

The physiological and behavioral consequences of maternal psychological distress manifest in several ways [3]. Pregnant women suffering from mental health conditions often experience poorer sleep, increased fatigue, and changes in diet, specifically, increased intake of fatty foods and reduced consumption of essential vitamins [3]. These behavioral changes contribute to alterations in maternal metabolism and immune function, potentially affecting fetal growth and development [11].

Notably, the maternal brain itself undergoes transformation during pregnancy, possibly to enhance maternal caregiving. However, in women experiencing high levels of stress or PTSD, these adaptations may be weakened. For instance, mothers with anxiety or depression have shown reduced neural responsiveness to infant cues, such as smiling faces, suggesting impaired maternal sensitivity and bonding [3].

The concept of maternal PTSD during pregnancy is rooted in a broader understanding of prenatal mental health and its role in shaping early human development. It is now well established that fetal neurodevelopment is not solely determined by genetics but is also shaped by maternal psychological states through biological, behavioral, and environmental pathways [6, 13].

Mechanisms of Impact and Fetal Responses

Fetal brain development is a sensitive process involving neurogenesis, neuronal migration, synaptogenesis, and myelination that begins in the first trimester and continues postnatally [3]. This process is highly susceptible to environmental signals, including maternal psychological states. Maternal PTSD exerts substantial influence on the intrauterine environment through endocrine, inflammatory, and epigenetic pathways, resulting in measurable changes in fetal brain structure and function [4, 5].

One of the primary mechanisms is dysregulation of the hypothalamic pituitary adrenal (HPA) axis, which is activated during maternal psychological distress. This could result in elevated secretion of glucocorticoids, particularly cortisol, which can cross the placenta and influence fetal brain development [3, 5]. Although the placenta expresses 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2), an enzyme that inactivates cortisol, chronic maternal stress has been associated with reduced placental expression of 11β-HSD2, increasing fetal exposure to cortisol and disrupting key neurodevelopmental processes such as neuronal differentiation and synaptogenesis [3–5]. Additionally, impaired placental perfusion caused by stress-induced changes in uterine artery blood flow leads to reduced oxygen and nutrient delivery to the fetus [4].

Among the most affected regions of the fetal brain are the hippocampus, amygdala, and anterior cingulate cortex (ACC) areas that are rich in glucocorticoid receptors and sensitive to cortisol exposure [1, 5]. Studies have shown that elevated maternal cortisol in mid-gestation is

associated with reduced neonatal hippocampal and amygdala volumes, with these structural changes correlating with behavioral and emotional dysregulation in infants [5]. Moreover, maternal PTSD is linked to weaker functional connectivity in the ACC, a region responsible for executive functioning and attention. This weakening has been associated with delays in attentional processing in the first months of life [5].

Epigenetic mechanisms further mediate the effects of maternal PTSD on fetal brain development. Studies have identified increased DNA methylation of the NR3C1 gene, which encodes the glucocorticoid receptor, in the cord blood of neonates exposed to maternal stress [1, 2]. This alteration may result in heightened postnatal stress reactivity, as the gene plays a key role in regulating the infant's own HPA axis [1]. The role of inflammation is also prominent. Maternal PTSD has been linked to elevated levels of proinflammatory cytokines, particularly interleukin-6 (IL-6). Elevated IL-6 has been associated with altered fetal brain structure and connectivity, particularly in areas related to sensory integration and executive functioning [4, 5].

These biological alterations may contribute to downstream developmental consequences in offspring. Prenatal exposure to maternal psychological distress has been associated with increased risk for a range of neurodevelopmental and psychiatric outcomes, including anxiety disorders, depression, ADHD, autism spectrum disorder, and schizophrenia [3, 4]. In addition to mental health outcomes, prenatal maternal distress has also been linked to broader health complications, including obesity and infectious disease susceptibility in offspring, as well as adverse pregnancy outcomes such as preeclampsia and preterm birth [3, 4]. These findings support the concept of fetal programming, in which environmental signals during sensitive periods of development shape long- term physiological and behavioral trajectories.

While these findings highlight the biological pathways through which maternal PTSD may influence the developing fetal brain, an important question concerns how these prenatal alterations translate into measurable outcomes after birth. Understanding the developmental consequences of these early neurobiological changes is essential for clarifying the long- term impact of prenatal maternal distress. The following section therefore examines the behavioral, cognitive, and neurodevelopmental outcomes observed in children exposed to maternal psychological distress during pregnancy.

Developmental Consequences in Offspring

Prenatal exposure to maternal stress and trauma has far-reaching consequences on child neurodevelopment, spanning behavioral, emotional, cognitive, and physiological domains. This phenomenon, often rooted in fetal programming mechanisms, continues to be evident across multiple clinical, imaging, and epidemiological studies [1, 10]. Children born to mothers who experienced high levels of stress or PTSD during or even before pregnancy face an increased risk of neurodevelopmental disorders, including ADHD, autism spectrum disorder (ASD), anxiety, depression, and schizophrenia [3–6, 12]. Both human and animal models support these outcomes and demonstrate that such psychiatric and behavioral risks can emerge in infancy and persist into adolescence and adulthood [3, 4, 9, 11].

Neuroimaging studies have highlighted structural brain alterations in these offspring. Infants born to mothers who experienced significant prenatal psychological distress such as anxiety, depression, or PTSD have been shown to exhibit reduced gray matter density and cortical thinning, particularly within the frontal and temporal lobes. These regions are critically involved in executive functioning, language, memory, and emotion regulation [2]. Altered growth in the hippocampus, especially the left hemisphere, has been associated with prenatal maternal stress, and this asymmetry may impair episodic verbal memory and emotional learning [2].

In addition, prenatal stress is linked to increased cortical gyrification in the frontal and temporal lobes, a neurodevelopmental pattern that has been associated with ASD and schizophrenia [2]. Further, several studies reveal significant impacts on the amygdala and cingulate cortex, regions integral to emotional processing and regulation. Altered connectivity and volume in these areas have been observed in children exposed to maternal stress, correlating with increased emotional reactivity, poor inhibitory control, and affective dysregulation [6, 11, 13].

These structural changes parallel behavioral observations of increased internalizing and externalizing problems, including irritability, impulsivity, and poor social engagement [4, 5, 9, 13]. Physiologically, maternal stress also appears to affect the placental-fetal unit, disrupting the function of stress-response systems such as the HPA axis and autonomic nervous system (ANS) [10, 11]. These systems shape the infant's stress responsivity, setting the stage for long-term vulnerabilities to mental health conditions [10]. Epigenetic changes, particularly DNA methylation in genes like FKBP5 and NR3C1 within placental tissue, have been associated with altered infant neurobehavior, including high arousal, poor self-regulation, and lethargy [7]. Beyond psychiatric outcomes, there is also increased prevalence of physical health issues such as obesity and immune dysregulation, potentially linked to early neuroendocrine and inflammatory pathway alterations [3, 4, 11]. Additionally, exposure to maternal mental distress during pregnancy is associated with delayed cognitive and language development, poorer attention, and memory deficits as early as infancy and toddlerhood [12, 13]. Despite the growing evidence linking prenatal maternal distress to neurodevelopmental outcomes in offspring, important methodological limitations complicate the interpretation of these findings.

Limitations

Despite the growing body of evidence linking maternal PTSD and prenatal psychological distress to alterations in fetal brain development and later neurodevelopmental outcomes, several important limitations remain within the existing literature. Much of the research in this field relies on observational cohort designs, which limit the ability to establish clear causal relationships between maternal psychological distress and offspring neurodevelopment. In addition, prenatal stress is frequently assessed using maternal self-reported symptom scales, which vary across studies and may introduce measurement variability.

Maternal PTSD also commonly co-occurs with other factors that may independently influence child development, including socioeconomic disadvantage, maternal health conditions, genetic susceptibility, and aspects of the postnatal caregiving environment. Large cohort investigations suggest that broader social disadvantage may exert stronger effects on infant brain structure than psychosocial stress alone [6].

Neuroimaging findings have also shown variability across studies; while some investigations report associations between prenatal maternal distress and structural differences in regions such as the hippocampus and amygdala, others have failed to replicate these findings or have reported associations that did not remain significant after statistical correction. Collectively, these inconsistencies highlight the complexity of isolating the specific biological effects of maternal distress on fetal brain development and underscore the need for longitudinal and genetically informed research designs to clarify the mechanisms underlying the intergenerational transmission of risk.

Mitigation of Risks and Future Research

Maternal psychological distress during pregnancy, though widespread, is both preventable and treatable through timely interventions and supportive policies. Routine, trimester-specific mental health screenings are strongly recommended by The American College of Obstetricians and Gynecologists (ACOG) to identify trauma and stress early, followed by evidence-based interventions such as cognitive behavioral therapy (CBT) and mindfulness-based programs [3, 7, 11, 13].

Addressing stigma is vital, as over 70% of women underreport symptoms, seeing them as normal [3]. Empowering midwives and gynecologists through training and clearer referral systems can significantly increase self-disclosure and treatment uptake [3]. Social support and postnatal caregiving quality also buffer the negative impact of prenatal stress [3, 7]. Future research should prioritize identifying early non- invasive biomarkers, such as fetal heart rate variability and epigenetic markers, to help predict and mitigate altered neurodevelopmental trajectories [8, 10]. Building on these advances, a universal preventive approach to perinatal mental health akin to prenatal vitamins may ultimately help safeguard both maternal and fetal well- being [11].

Conclusion

Maternal PTSD and psychological distress during pregnancy represent important influences on the intrauterine environment and may shape fetal neurodevelopment through interconnected hormonal, inflammatory, and epigenetic pathways. Evidence from epidemiological, neuroimaging, and experimental studies suggest that prenatal exposure to elevated maternal stress is associated with alterations in brain regions involved in emotional regulation, cognition, and stress responsivity, potentially increasing vulnerability to behavioral, emotional, and neurodevelopmental disorders later in life. At the same time, the complexity of these relationships underscores the need to consider interacting influences, including genetic susceptibility, socioeconomic context, and the quality of the postnatal caregiving environment.

Importantly, maternal psychological distress during pregnancy is both identifiable and treatable. Early screening, trauma- informed prenatal care, and accessible mental health interventions offer promising opportunities to mitigate potential risks and support healthier developmental trajectories for children. Continued interdisciplinary research, including longitudinal and genetically informed studies, will be essential to clarify casual mechanisms, identify protective factors, and better understand how prenatal and postnatal environments interact to shape child development.

Ultimately, prioritizing maternal mental health during pregnancy represents a critical public health investment. Supporting the psychological well- being of expectant mothers not only benefits maternal health but also contributes to healthier neurodevelopmental outcomes for future generations.

List of Abbreviations

ADHD: attention deficit hyperactivity disorder
ANS- autonomic nervous system
ASD: autism spectrum disorder
BDNF: dysregulation of brain-derived neurotrophic factor
CHD: congenital heart disease
CRH: corticotropin-releasing hormone
DNA: deoxyribonucleic acid
HPA: hypothalamic-pituitary-adrenal
IL-6: interleukin 6
NR3C1: glucocorticoid receptor gene
PTSD: post-traumatic stress disorder

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

KH: contributed to the study design, screened the titles and abstracts of each search result for eligibility, prepared

literature reviews, drafted the manuscript, and approved the final version for publication.
EB: contributed to the study design, screened the titles and abstracts of each search result for eligibility, drafted the manuscript, and approved the final version for publication.
SRR: contributed to the study design, screened the titles and abstracts of each search result for eligibility, critically revised the manuscript, and approved the final version for publication.

Acknowledgements
The authors gratefully acknowledge the contributions of the following tools which assisted in the preparation of this entry: ChatGPT-4o, used primarily for conducting preliminary research and generating original insights related to the topic; Notebook LM, which facilitated the connection of academic papers, creation of mind maps, and access to in- depth information, especially on AI- related subjects; and Grammarly- 14.1246.0, employed during the final stages to ensure clarity, correct grammar, and maintain a professional tone throughout the manuscript. All tools have been used in accordance with relevant guidelines and best practices.

Funding
The development of this encyclopedia entry was not funded.

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Article Information
Managing Editor: Jeremy Y. Ng
Peer Reviewers: Grace Li, Jeremy Y. Ng
Article Dates: Received Oct 17 26; Accepted May 02 26; Published May 31 26

Citation
Please cite this article as follows:
Habibyar K, Bajrami E, Rrustemaj S. The silent imprint: Exploring maternal PTSD and its effects on fetal neurodevelopment. URNCST Journal. 2026 May 31: 10(5). https://urncst.com/index.php/urncst/article/view/951
DOI Link: https://doi.org/10.26685/urncst.951