Review

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Prenatal maternal stress and long-term neurodevelopmental outcomes: a narrative review

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Abstract

Introduction: Maternal stress during pregnancy has been increasingly recognized as a significant factor influencing fetal development, with potential long-term effects on neurodevelopmental outcomes in offspring. Understanding the mechanisms and implications of prenatal stress exposure is crucial for informing preventative and interventional strategies in perinatal care.

Content: This narrative review synthesizes findings from epidemiological, neurobiological, and clinical research on the long-term consequences of prenatal maternal stress. It explores a broad spectrum of stressors – including psychological distress, depression, anxiety, and acute traumatic events such as natural disasters – and their associations with cognitive, behavioral, and emotional outcomes in offspring from infancy through adolescence. Key biological mechanisms are discussed, including alterations in the maternal-placental-fetal axis, HPA axis dysregulation, placental gene expression changes, epigenetic modifications, and neuroinflammatory responses. Neuroimaging and biomarker studies are highlighted to provide evidence for stress-related changes in brain structure and function.

Summary: Current literature supports a robust association between maternal stress during pregnancy and a heightened risk of neurodevelopmental challenges in children. These include deficits in executive function, increased anxiety and depressive symptoms, emotional dysregulation, and susceptibility to psychiatric disorders. The effects appear to be moderated by timing of exposure, genetic predispositions, and the postnatal environment.

Outlook: While the evidence base is growing, methodological limitations such as variability in stress assessment and inconsistent follow-up durations persist. Future research should emphasize longitudinal, biomarker-informed designs and evaluate interventions aimed at reducing prenatal stress. Integrating maternal mental health support into routine prenatal care may offer a promising pathway to improving both maternal and child outcomes.

Keywords: prenatal stress; neurodevelopment; fetal programming; emotional regulation; cognitive outcomes; maternal anxiety

Introduction

Maternal stress during pregnancy has emerged as a significant area of research in recent years, with growing evidence suggesting that prenatal exposure to stress can have long-lasting effects on offspring neurodevelopment [1]. The fetal programming hypothesis suggests that the intrauterine environment can influence fetal development and subsequent health outcomes. This concept has been broadened to consider the potential impact of maternal psychological state on fetal brain development [2].

Emerging neurobiological and epidemiological evidence supports this view. Prenatal environmental insults, including stress, may disrupt the timing of critical neurodevelopmental events, potentially increasing the risk of disorders such as schizophrenia and autism spectrum disorders [1, 2]. Prenatal stress can also lead to altered corticogenesis and aberrant connectivity in the developing brain – effects linked to later emotional and cognitive difficulties [3].

From a mechanistic standpoint, prenatal maternal stress has been associated with increased risk for ADHD and

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anxiety disorders in children, likely mediated through dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis [4]. Epigenetic modifications, particularly DNA methylation changes in glucocorticoid receptor genes, may alter stress reactivity and emotional regulation across the lifespan [5]. Prenatal stress has also been shown to affect the expression of stress-regulating genes in the hippocampus [6], and can lead to persistent changes in gene expression related to synaptic plasticity and neurodevelopment [7]. In animal models, prenatal stress results in increased anxiety-like behavior and altered defensive strategies in offspring, reinforcing the link between early stress exposure, neuroendocrine changes, and long-term behavioral outcomes [8]. Together, these studies highlight that prenatal stress exerts its effects through both direct neurodevelopmental disruption and enduring changes in gene expression and stress regulation systems.

In parallel, structural and functional imaging studies have shown that exposure to maternal stress *in utero* can lead to altered amygdala-prefrontal connectivity, as well as reduced hippocampal volume – regions critical for emotion regulation and memory. Maternal stress-related placental gene expression has also been linked with newborn brain structure, suggesting a direct placenta-brain communication pathway [9].

Importantly, the timing of stress exposure plays a pivotal role. Stress during mid-gestation appears particularly disruptive to fetal neurobehavior and is associated with less optimal infant temperament, supporting the idea of "sensitive windows" during which environmental insults may exert the most profound influence [10]. From a developmental plasticity perspective, the concept of critical periods in brain development – specific time windows when the brain is especially sensitive to environmental inputs – helps explain how maternal stress during pregnancy may permanently alter neural circuitry, setting the stage for later vulnerability or resilience depending on the timing and intensity of exposure [11].

This narrative review aims to summarize current knowledge on the long-term neurodevelopmental outcomes of infants exposed to maternal stress during pregnancy, exploring the types of stress, potential mechanisms, and observed effects on cognitive, behavioral, and emotional development.

The importance of understanding the impact of prenatal maternal stress on offspring neurodevelopment has become increasingly apparent as research has revealed the potential for long-term consequences extending into adulthood. This knowledge may inform interventions to mitigate the effects of stress during pregnancy and promote optimal child development. Furthermore, it contributes to our understanding of the origins of neurodevelopmental disorders and mental health problems, potentially opening new avenues for prevention and early intervention.

Materials and methods

Data sources and search strategy

In order to identify and select all the related studies, an extensive search was conducted in the databases PubMed, Scopus, EMBASE and Google Scholar without time restrictions, from June to December 2024. The following algorithm was used consecutively with the key words "Prenatal Stress, Neurodevelopment, Cognition, Emotional Regulation":

> ((Prenatal) OR (Pregnancy) OR (Gestational) AND (Stress) AND ((Fetal) OR (Neonatal)) AND ((Neurodevelopment) OR (Cognition) OR (Behaviour) OR (Emotional Regulation))

Screening and eligibility criteria

During the screening of the bibliography, filters were applied to refine the incoming volume of information, focusing on studies written in English and trials conducted on humans. This process was independently conducted by two blinded researchers and involved three consecutive stages. Initially, articles were assessed based on their titles, followed by an evaluation of their abstract content. Studies deemed relevant were then reviewed in their full manuscript form. The selected studies specifically examined the impact of various types of maternal stress, such as anxiety, depression, and natural disasters during pregnancy. We prioritized studies that assessed fetal brain anatomy and the socio-cognitive development of the offspring from infancy through adulthood. Research papers focusing solely on physical outcomes of prenatal stress, such as body weight and head circumference, were excluded. Additionally, manuscripts evaluating labor complications or maternal drug use were not considered.

Any disagreement between the two investigators on the selection of articles and their evaluation for the presence of bias, was resolved consensually.

Assessment of risk of bias

Both researchers were involved in evaluating the quality of the selected literature. Non-experimental studies were

assessed using the Newcastle-Ottawa scale, which focuses on the selection of the study population, the comparability between control and intervention groups, and the validation of the final results. This scale assigns a maximum of nine stars to each study based on these criteria.

Results

Study selection and characteristics

Through a comprehensive research in current literature, we identified 22 observational cohort studies that met the inclusion criteria for this review. Our sample comprised 15,664 children whose mothers experienced varying degrees of stress during pregnancy. The follow-up period spanned from 15.6±4.2 days to 37 years. Maternal stress ranged from anxiety and depression to trauma induced by a natural disaster and was assessed using self-reported symptoms, scales such as the Perceived Stress Scale (PSS), Edinburgh Perinatal Depression Scale (EPDS), and State-Trait Anxiety Interview (STAI), as well as cortisol levels during pregnancy. Evaluations of outcomes related to their offspring encompassed a broad spectrum of measures, including mental health prescriptions, the Child Behavior Checklist, and the Bayley Scales of Infant and Child Development (Table 1). Maternal demographic characteristics, including age, education, socioeconomic status, and ethnicity/race, varied across studies and are summarized in Table 2.

Quality assessment of included studies

The quality assessment results are summarized in Table 3. The majority of the clinical studies included were of moderate to high quality, with scores ranging from 6 to 9 out of a possible nine stars.

Types of maternal stress

Maternal stress during pregnancy covers a wide spectrum of experiences and conditions, each potentially influencing fetal neurodevelopment in unique ways. Psychological distress and anxiety during pregnancy, including general psychological distress and maternal anxiety, have been linked to a range of neurodevelopmental outcomes in offspring [26]. For instance, higher levels of maternal anxiety are associated with an increased risk of attention deficit hyperactivity disorder (ADHD) symptoms, emotional problems, and cognitive deficits in children. A longitudinal study by Van den Bergh and Marcoen demonstrated that even after accounting for the child's gender, parental education level, smoking, birth weight, and postnatal maternal anxiety, prenatal maternal anxiety remained significantly linked to ADHD symptoms and externalizing problems in 8 and 9 year-old children [33].

Similarly, maternal depression during pregnancy is consistently linked to adverse neurodevelopmental outcomes, such as increased risks of depression, anxiety, and behavioral problems in children [30]. A longitudinal study by Babineau et al. indicated that maternal depression during pregnancy was connected to higher levels of internalizing, externalizing, and general psychopathology in offspring, with some studies suggesting these effects could extend into adolescence and early adulthood [14].

Exposure to severe stressors like natural disasters, terrorist attacks, or personal trauma during pregnancy is also associated with various neurodevelopmental outcomes [12, 15, 16, 18, 25, 28]. For example, 'Project Ice Storm', which studied children whose mothers were pregnant during a severe ice storm in Quebec, found that higher levels of objective maternal stress during pregnancy were linked to poorer cognitive and language development in children at age 2 [28]. Further research has shown associations between prenatal exposure to severe stress and increased risks of autism spectrum disorders and schizophrenia [34, 35].

Chronic, low-level stress from daily hassles or ongoing life difficulties may also influence fetal neurodevelopment. Although less dramatic than acute traumatic events, these more common forms of stress may have cumulative effects on child outcomes. A study by Huizink et al. found that daily hassles and pregnancy-specific anxiety were associated with lower mental and motor development scores in infants at 8 months of age [31].

Biological mechanisms

Multiple biological mechanisms have been proposed to elucidate how maternal stress during pregnancy influences fetal neurodevelopment. A key pathway involves dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. In response to stress, maternal activation of the HPA axis results in elevated cortisol production, which can cross the placenta and impact the developing fetal brain [34, 36]. It is known that excess cortisol affects nearly all organs and therefore it can cross the placenta and impact fetal brain development, particularly in areas like the hippocampus, amygdala, and prefrontal cortex, which are rich in glucocorticoid receptors [34]. Animal studies have shown that prenatal exposure to elevated glucocorticoids can result in

Table 1: Characteristics of included studies on prenatal maternal stress and offspring neurodevelopment.

Author, year	Country	Study design	Sample size	Type of stress	Stress evaluation	Follow-up duration	Endpoints	Endpoint assessment	Key findings
Nazzari et al. 2023 [12]	Italy	Observational cohort	90	Natural disaster (pandemic)	Self-reported maternal symptoms	12 months	Sociocognitive development	Auditory Stimuli/SCS	Pandemic-related stress during pregnancy associated with lower infant sociocognitive development at 12 months
McGuinn et al. 2022 [13]	Mexico	Observational cohort	496	Anxiety	Cortisol levels (saliva), STAI	8–11 years	Late childhood anxiety	RCMAS-2	Higher maternal anxiety during pregnancy predicted higher anxiety levels in children at age 8–11 years
Wu et al. 2022 [3]	USA (Washington)	Observational cohort	97	Anxiety, depression	PSS, EPDS, STAI	18 months	Fetal brain development, neurodevelopment, parenting stress	MRI, BSID III, ITSEA, PSI-SF	Maternal distress associated with altered fetal brain development and offspring cognitive and social-emotional outcomes at 18 months
Babineau et al. 2021 [14]	USA	Observational cohort	730	Anxiety, depression	PSS, EPDS	4–6 years	Executive functions, motor skills, psychiatric problems, fetal growth velocity	NIH-TB, CBCL, SCQ	Prenatal maternal stress and depressive symptoms associated with child neurobehavioral outcomes, with fetal growth velocity as a potential mediator
Berthelon et al. 2021 [15]	Chile	Observational cohort	900	Natural disaster (earthquake)	Self-reported maternal symptoms, earthquake proximity	35 months	Prenatal stress exposure on child's early cognitive and socioemotional outcomes	BDI, TADI, CBCL	Prenatal stress from earthquake exposure negatively impacted child cognitive and socioemotional development at 35 months
Clark et al. 2021 [16]	UK	Observational cohort	Case 445, control 10,108	Traumatic events (economic crisis)	Self-reported maternal symptoms	25 years	BW and HC, educational cognitive performance	High school math, physics and science tests	Economic shocks during pregnancy associated with lower birth weight and poorer educational outcomes
Thiel et al. 2021 [17]	Norway	Observational cohort	3,572	Anxiety	SCL-A, EPDS	8 weeks and 2 years	Infant temperament at 8 weeks, child development at 2 years, perinatal life stress	ICQ, ASQ-3 and ASQ-SE	Perinatal life stress associated with more difficult infant temperament and developmental problems at 2 years
Persson and Rossin-Slater, 2018 [18]	Sweden	Observational cohort	Case 127,406, control 3,988,858	Traumatic events (death in family)	Administrative population data	36 years	Birth outcomes, physical and cognitive outcomes until adulthood	Mental health drug prescriptions	Family deaths during pregnancy associated with increased risk of ADHD medication use in offspring
Aizer et al. 2016 [19]	USA (Boston)	Observational cohort	1,093 (386 siblings)	Anxiety	3rd trimester cortisol levels	7 years	Cognition and health at age 7 years, effect of stress at maternal human capital	Wechsler intelligence tests (IQ), completed schooling years	Higher prenatal cortisol levels associated with lower IQ scores and fewer years of schooling in offspring

Table 1: (continued)

Author, year	Country	Study design	Sample size	Type of stress	Stress evaluation	Follow-up duration	Endpoints	Endpoint assessment	Key findings
Räikkönen et al. 2015 [20]	Helsinki	Observational cohort	67	Depression	CES-D, placental RNA	15.6±4.2 days	Infant regulatory behaviors at 15.6±4.2 days	Neonatal perception inventory	More infant regulatory behavioral challenges. In women who reported higher depressive symptoms in the third trimester
Zhu et al. 2014 [21]	China	Observational cohort	60	Stressful life events	Prenatal life events checklist	16 months, 18 months	Cognition and temperament traits at 16 and 18 months, maternal postnatal depression	BSID-CR, TTS, Edinburgh postnatal depression scale	Lower MDI scores and less optional behavioral response in children of exposed than unexposed mothers
Buss et al. 2012 [22]	USA	Observational cohort	65	Depression	Cortisol levels (saliva)	7 years	Amygdala and hippocampus volumes, child affective problems	MRI, CBCL	Higher maternal cortisol levels during pregnancy associated with larger amygdala volumes and more affective problems in girls at age 7
Blair et al. 2011 [23]	USA (California)	Observational cohort	120	Anxiety	Pregnancy related anxiety scale, STAI	2 years	Child temperament at age 2 years	Early childhood behavior questionnaire	Higher prenatal anxiety associated with more difficult child temperament at age 2
Pluess et al. 2011 [24]	Netherlands	Observational cohort	1,513	Anxiety	Self-reported maternal symptoms	6 months	5-HTTLPR allele infants susceptibility to maternal anxiety	Infant behavior questionnaire	Infants with the short 5-HTTLPR allele more susceptible to effects of maternal anxiety on negative emotionality
Simcock et al. 2011 [25]	Australia	Observational cohort	130	Natural disaster (floods)	QFOSS (objective stress), COSMOSS (subjective stress)	30 months	ToM at 30 months	Diverse desire scenarios	Higher objective stress during pregnancy associated with poorer theory of mind development at 30 months
Bergman et al. 2010 [26]	UK	Observational cohort	125	Anxiety	3rd trimester cortisol levels (amniotic fluid+blood), STAI	14 months, 19 months	Cognition and health at 14 and 19 months, infant/parent relationship as a moderator of prenatal cortisol exposure	BSID II, Ainsworth strange situation	Higher prenatal cortisol levels associated with lower cognitive ability, moderated by infant-mother attachment
Davis and Sandman, 2010 [27]	USA (California)	Observational cohort	125	Anxiety, depression	Cortisol levels (Saliva), PSS, CES-D, STAI, PSA	3 months, 6 months, 12 months	Cognition at 3, 6 and 12 months, effects of timing of exposure to stress on infant development	BSID, MDI, PSI	Lower cortisol in 1st trimester, as well as higher in 3rd trimester, correlates with accelerated infant development.
Laplante et al. 2008 [28]	Canada	Observational cohort	89	Natural disaster (ice storm)	Storm32, impact of events scale	5.5 years	Cognition and language abilities at age 5.5 years	Wechsler preschool and primary school scale of intelligence, peabody picture vocabulary test	Higher prenatal stress from ice storm exposure associated with lower IQ and language abilities at 5.5 years

Table 1: (continued)

Author, year	Country	Study design	Sample size	Type of stress	Stress evaluation	Follow-up duration	Endpoints	Endpoint assessment	Key findings
Davis et al. 2007 [29]	USA (California)	Observational cohort	247	Anxiety	3rd trimester cortisol levels (saliva), 2 months postpartum	2 months	Infant temperament	Infant behavior questionnaire	Higher prenatal cortisol levels associated with more fearful and reactive infant temperament
Van den Bergh et al. 2004 [30]	Belgium	Observational cohort	72	Anxiety	STAI	8 and 9 years	Childhood disorders (ADHD, anxiety, aggression) at 8 and 9 years	CBCL, TRF, STAIC, GBO	High maternal anxiety during pregnancy associated with ADHD symptoms and externalizing problems in 8–9 year-olds
Huizink et al. 2003 [31]	Netherlands	Observational cohort	170	Daily hassles	PRAQ-R, 3rd trimester cortisol levels (saliva), everyday problem list	3 and 8 months	Physical and cognitive outcomes at 3 and 8 months	BSID	Higher prenatal anxiety and daily hassles associated with lower mental and motor development scores in infants
O'Connor et al. 2003 [32]	UK	Observational cohort	6,996	Anxiety, depression	Self-reported anxiety (crown-crisp index), EPDS	47 and 81 months	Behavioral and emotional problems at 47 and 81 months	SDQ	Prenatal maternal anxiety predicted behavioral and emotional problems in children at 47 and 81 months

ASQ-3, ages and stages questionnaire, third edition; ASQ-SE, ages and stages questionnaire: social-emotional; BDI, beck depression inventory; BSID, bayley scales of infant development; BW, birth weight; CBCL, child behavior checklist; COSMOSS, composite score for mothers' subjective stress; EPDS, edinburgh postnatal depression scale; GBO, goal-based outcome measure; HC, head circumference; ICQ, infant characteristics questionnaire; ITSEA, infant-toddler social and emotional assessment; MRI, magnetic resonance imaging; NIH-TB, NIH toolbox; PRAQ-R, pregnancy-related anxiety questionnaire-revised; PSI-SF, parenting stress index-short form; PSS, perceived stress scale; QFOSS, queensland flood objective stress scale; RCMAS-2, revised children's manifest anxiety scale, second edition; SCL-A, symptom checklist-90 anxiety subscale; SCQ, social communication questionnaire; SDQ, strengths and difficulties questionnaire; STAI, state-trait anxiety inventory; STAIC, state-trait anxiety inventory for children; TADI, test of learning and child development; ToM, theory of mind; TRF, teacher's report form.

Table 2: Maternal demographic characteristics of included studies.

Author, year	Age, years	Education	Socioeconomic status	Ethnicity/race
Nazzari et al. 2023 [12]	33.15±4.68	15.86±2.91 years	Not reported	Not reported
McGuinn et al. 2022 [13]	28.1±6	<High school: 41 % High school: 35 % >High school: 24 %	Low: 54 % Middle: 36 % High: 10 %	Not reported
Wu et al. 2022 [3]	34.79±5.64	<High school: 3 % >High school: 93.8 %	Not reported	Not reported
Babineau et al. 2021 [14]	Asian/Pacific islander: 31.16±4.54 Hispanic: 28.53±5.87 Non-Hispanic Black: 24.82±5.45 Non-Hispanic White: 30.95±4.19	<High school: 11.1 % High school: 16 % >High school: 72.9 %	<$50,000: 55 % $50,000–$100,000: 21 % >$100,000: 13.8 % Not reported: 10.2 %	Asian/Pacific islander Hispanic non-Hispanic Black White
Berthelon et al. 2021 [15]	28.3±6.89	11.7±2.71 years	Not reported	Not reported
Clark et al. 2021 [16]	27.87±4.86	Not reported	Not reported	Not reported
Thiel et al. 2021 [17]	31±5	≤12 years: 62 % >12 years: 38 %	Not reported	Not reported
Persson et al. 2018 [18]	27.88 (mean at conception)	<High school: 17.7 % High school: 31.4 % >High school: 20.2 %	Not reported	Not reported
Aizer et al. 2016 [19]	Full sample: 25.2 Cortisol sample: 24.9	Full sample: 11.4 years Cortisol sample: 11.07 years	Full sample: 26,013$ Cortisol sample: 24,403$	Not reported
Räikkönen et al. 2015 [20]	32.2±5.3	>High school: 63 %	Not reported	Not reported
Zhu et al. 2014 [21]	27.8±2.4	>High school: 100 %	78.9 % 2,000–4,000 Rmb per month	Not reported
Buss et al. 2012 [22]	31.1±6.5	≤High school: 15.3 % >High school: 83.7 %	<$60,000: 48.3 % $60,000–$100,000: 32.7 % >$100,000: 19 %	Not reported
Blair et al. 2011 [23]	30±5.2	<High school: 19.1 % >High school: 45 % Not reported: 35.8 %	<$60,000: 39.1 % $60,000–$100,000: 22.5 % >$100,000: 21.6 %	Not reported
Pluess et al. 2011 [24]	31.81±4.03	No education: 1.3 % Low (≤15 years): 32.9 % Mid-high (16–17 years): 26.4 % High (>17 years): 39.4 %	<€1,200: 4.6 % €1,200–€2,200: 17.3 % >€2,200: 78.1 %	Not reported
Simcock et al. 2011 [25]	32.06±5.10	Not reported	Not reported	Not reported
Bergman et al. 2010 [26]	36.62±4.12	<High school: 3.2 % ≥High school: 45.6 % (GSCE 11.2 %, a level 15.2 %, diploma 19.2 %) >College: 51.2 %	Not reported	White: 82.4 % Asian-Indian: 5.6% Afro-Caribbean: 8 % Middle-Eastern: 2.4 % Far-Eastern: 1.6 %
Davis et al. 2010 [27]	29.9	<High school: 5 % ≥High school: 49 % >College: 46 %	≤30,000: 17 % 30,000–60,000: 26 % $60,000–$100,000: 33 % >$100,000: 24 %	Asian: 10 % Hispanic: 30 % Non-Hispanic White: 50 %
Laplante et al. 2008 [28]	30.2±4.9	15.2 years	Lower class: 3.4 % Lower middle class: 1.1 % Middle class: 24.7 % Upper middle class Upper class: 19.1 %	Not reported

Table 2: (continued)

Author, year	Age, years	Education	Socioeconomic status	Ethnicity/race
Davis et al. 2007 [29]	30.7±5.4	<High school: 2 % ≥High school: 47 % >College: 51 %	≤30,000: 19.7 % 30,000–60,000: 29.3 % $60,000–$100,000: 28.4 % >$100,000: 22.6 %	Asian: 9 % African American: 11 % Hispanic: 20 % Non-Hispanic White: 49 %
Van den Bergh et al. 2004 [30]	18–30 years	<High school: 13.8 % ≥High school: 55.9 % >College: 32.3 %	Not reported	Caucasian
Huizink et al. 2003 [31]	31.3±4.9	≤High school: 13.6 % Secondary school: 67.5 % >College: 18.9 %	Low: 8 % Middle: 54.6 % Upper: 37.4 %	Caucasian: 96 %
O'Connor et al. 2003 [32]	14–46 years (mean 28)	≤High school: 84.4 (GCSE 22.2 %, O level 36.1 %, A level 26 %) >College: 15.6 %	Not reported	Not reported

Table 3: Quality assessment of included studies using the Newcastle-Ottawa scale.

Author, year	Selection (max 4★)	Comparability (max 2★)	Outcome (max 3★)	Total score (max 9★)
Clark et al. 2021 [16]	★★★★	★★	★★★	9/9
Babineau et al. 2021 [14]	★★★	★★	★★★	8/9
Nazzari et al. 2023 [12]	★★★	★★	★★★	8/9
Aizer et al. 2016 [19]	★★★★	★	★★★	8/9
Räikkönen et al. 2015 [20]	★★★★	★★	★★★	8/9
Zhu et al. 2014 [21]	★★★★	★★	★★★	8/9
Buss et al. 2012 [22]	★★★	★★	★★★	8/9
Pluess et al. 2011 [24]	★★★★	★	★★★	8/9
Bergman et al. 2010 [26]	★★★	★★	★★★	8/9
Davis and Sandman, 2010 [27]	★★★	★★	★★★	8/9
Huizink et al. 2003 [31]	★★★	★★	★★★	8/9
O'Connor et al. 2003 [32]	★★★★	★★	★★	8/9
McGuinn et al. 2022 [13]	★★★	★★	★★	7/9
Wu et al. 2022 [3]	★★★	★	★★★	7/9
Berthelon et al. 2021 [15]	★★★	★	★★★	7/9
Thiel et al. 2021 [17]	★★★	★★	★★	7/9
Persson et al. 2018 [18]	★★★	★	★★★	7/9
Blair et al. 2011 [23]	★★★	★★	★★	7/9
Simcock et al. 2011 [25]	★★★	★★	★★	7/9
Laplante et al. 2008 [28]	★★	★	★★★	6/9
Davis et al. 2007 [29]	★★	★★	★★	6/9
Van den Bergh et al. 2004 [30]	★★	★★	★★	6/9

The Newcastle-Ottawa Scale assesses the quality of nonrandomized studies. It evaluates three domains: selection of study groups (max 4★), comparability of groups (max 2★), and ascertainment of exposure or outcome (max 3★). A higher score indicates better quality, with a maximum possible score of 9★. Studies scoring 8–9★ are considered high quality, 6–7★ moderate quality, and ≤5★ low quality. Our assessment found that seven studies were of high quality, 10 studies were of moderate quality, and 0 studies were of low quality. This overall high quality of included studies strengthens the reliability of our findings.

structural and functional changes in these brain regions, leading to behavioral alterations such as impaired feeling of fear [36].

Another proposed mechanism involves alterations in placental function and gene expression. Maternal stress may impair placental efficiency and modulate the expression of critical genes involved in nutrient transport, growth factor signaling, and neurotransmitter regulation, particularly dopamine and serotonin, which are essential for fetal brain development. These disturbances may disrupt the maturation

of neural circuits underlying reward sensitivity, motivation, and attentional control. Dysregulation of these systems has been linked to difficulties in experiencing pleasure and sustaining motivation during cognitive tasks. Furthermore, stress-induced downregulation of the placental enzyme 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2), which normally converts active cortisol into its inactive form cortisone, may increase fetal exposure to maternal glucocorticoids, thereby influencing brain development [36, 37].

Epigenetic modifications also play a role. Prenatal stress exposure can lead to epigenetic changes in the fetus, altering gene expression patterns that affect neurodevelopment and response to stress later in life. Studies have indicated that maternal stress during pregnancy is associated with epigenetic changes in genes involved in stress response systems, such as the glucocorticoid receptor gene, potentially setting the conditions for long-term alterations in stress reactivity [34].

Lastly, maternal stress can induce inflammatory responses that might cross the placenta and influence fetal brain development, possibly contributing to neurodevelopmental disorders. Elevated levels of pro-inflammatory cytokines have been linked to changes in fetal brain development and an increased risk of neuropsychiatric disorders in offspring [31]. Figure 1 illustrates the key biological mechanisms through which prenatal maternal stress impacts fetal brain development and long-term neurodevelopmental outcomes.

Long-term neurodevelopmental outcomes

Several studies have linked prenatal maternal stress to various developmental outcomes in offspring. A meta-analysis by Tarabulsy et al. reported small but significant

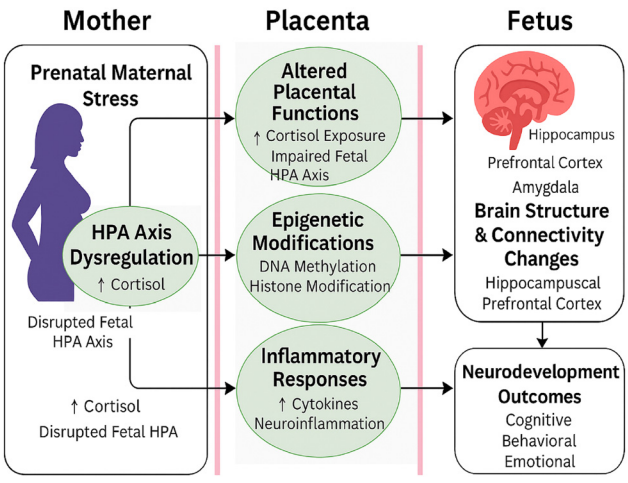

Figure 1: Pathways from maternal stress to fetal neurodevelopment.

negative effects on child cognitive development, particularly in general cognitive functioning and language development, with an overall effect size of r = −0.10 (95 % CI: −0.14 to −0.06), indicating a modest but consistent negative impact [38]. Longitudinal research, such as a study by Laplante et al., has shown that children whose mothers experienced high levels of stress during the 1998 Quebec ice storm had lower full-scale and verbal IQ scores and language abilities at age 5.5 years compared to those who experienced lower or moderate levels, especially when the stress occurred early in pregnancy [28]. Moreover, a study by Aizer et al., which included sibling pairs in its sample, provided compelling evidence that *in-utero* exposure to elevated cortisol levels adversely impacts offspring's human potential. This impact is reflected in reduced years of schooling, diminished cognitive abilities, and poorer health outcomes [19].

Prenatal maternal stress has also been consistently associated with increased behavioral problems in offspring. For example, a large cohort study by Berthelon et al., linked maternal stress during pregnancy with increased externalizing and internalizing behaviors in children at 35 months of age [15]. This study noted a dose-response relationship between the number of stressful life events during pregnancy and child behavioral problems. Further research, including findings by Van den Bergh and Marcoen, suggests that these behavioral effects can persist into adolescence, impacting areas such as ADHD symptoms and externalizing problems in teenagers [33]. In addition Räikkönen et al., which studied the behavior of infants up to 14 days post birth noticed a higher incidence of infant regulatory behavioral challenges in women who reported higher depressive symptoms in the third trimester [20]. In a study by Zhu et al., children of exposed mothers to stressful life events scored lower in the MDI scale and presented with less optimal behavioral response than those in the control group [21]. Finally O' Connor et al., has demonstrated that children whose mothers experienced high levels of anxiety during late pregnancy showed an increased likelihood of developing behavioral and emotional problems by 81 months of age. This association persisted even after accounting for obstetric risks, psychosocial disadvantages, and postnatal anxiety and depression. The odds ratio was 1.91 (95 % CI: 1.26–2.89) for girls and 2.16 (95 % CI: 1.41–3.30) for boys [32].

Additionally, exposure to maternal stress *in utero* has been connected to altered emotional regulation and a higher risk of mental health issues. A longitudinal study by McGuinn et al. demonstrated that maternal anxiety during pregnancy predicted higher anxiety levels in children at age 8–11 years, a correlation that persisted even after adjusting for postnatal maternal anxiety and other factors [13]. Other research has linked prenatal stress exposure to increased

risks of depression, anxiety disorders, and stress reactivity in later life, underscoring the potential long-term impact on mental health [17, 23, 29].

Neuroimaging studies are beginning to shed light on how prenatal maternal stress might affect offspring's brain structure and function. For instance, research by Buss et al. found that higher maternal cortisol levels during pregnancy were associated with larger amygdala volumes and affective problems in girls at age 7, emphasizing the amygdala's role in emotion processing [22]. Additionally, studies like the one by Wu et al. have observed changes in hippocampal volume, white matter microstructure, and functional connectivity in neonates related to maternal depression during pregnancy, which could underpin some of the observed cognitive, behavioral, and emotional outcomes in children exposed to prenatal stress [6].

Moderating factors

Several factors may moderate the association between prenatal maternal stress and offspring neurodevelopment. The timing of stress exposure during pregnancy is crucial; some studies indicate that early pregnancy might be a particularly sensitive period for certain outcomes, whereas other research finds stronger effects for stress exposure later in pregnancy. For instance, the 'Project Ice Storm' study highlighted that early pregnancy exposure to objective stress was more closely associated with cognitive outcomes, while late pregnancy exposure was more strongly linked to physical outcomes, such as birth weight, gestational age, and head circumference [28].

The severity and chronicity of stress also play significant roles. Chronic or severe stress exposures may have stronger effects than acute or mild stressors. Research by Huizink et al. demonstrated that pregnancy-specific anxiety had a greater association with infant development than general anxiety or daily hassles, suggesting that the nature and intensity of the stressor are crucial [31].

Genetic susceptibility also moderates the impact of prenatal stress on neurodevelopment. Variations in genes involved in stress response systems, for example, can influence susceptibility to the effects of prenatal stress. A study by Pluess et al. showed that the effect of prenatal maternal anxiety on infant temperament was moderated by a polymorphism in the serotonin transporter gene (5-HTTLPR) [24].

Finally, the postnatal environment, including factors such as maternal care, socioeconomic status, and exposure to additional stressors, can modify the long-term effects of prenatal stress exposure. A supportive postnatal environment may buffer some of the adverse effects of prenatal stress, while a stressful postnatal environment may exacerbate them. For example, Sharp et al. found that sensitive maternal care in infancy moderated the association between prenatal maternal anxiety and child behavioral problems at age 4 years [39].

Discussion

The growing body of evidence linking prenatal maternal stress to long-term neurodevelopmental outcomes in offspring underscores the critical importance of the intrauterine environment in shaping long-term cognitive, behavioral, and emotional trajectories. Studies across various populations and types of stressors consistently demonstrate associations between maternal stress during pregnancy and cognitive, behavioral, and emotional outcomes in children, with effects potentially persisting into adolescence and early adulthood [13–15, 19–21, 26, 28, 30, 32, 33, 38]. For instance, the longitudinal study by Laplante et al. showed that children whose mothers experienced high levels of stress during the 1998 Quebec ice storm had lower full-scale and verbal IQ scores at age 5.5 years, particularly when the stress occurred early in pregnancy [28]. Similarly, research by Van den Bergh and Marcoen [33] and O'Connor et al. [32] has linked prenatal maternal anxiety to increased risks of ADHD symptoms, externalizing problems, and emotional difficulties in children, even after taking into account various confounding factors. These findings are further supported by neuroimaging studies, such as those by Buss et al. [22] and Wu et al. [3], which have begun to elucidate the neurobiological underpinnings of these associations, revealing alterations in brain structure and function related to prenatal stress exposure. Collectively, these studies paint a compelling picture of the far-reaching impact of maternal stress during pregnancy on offspring neurodevelopment.

The biological mechanisms underlying the effects of prenatal maternal stress on fetal neurodevelopment are complex and multifaceted, involving alterations in the hypothalamic-pituitary-adrenal (HPA) axis, changes in placental function and gene expression, epigenetic modifications, and inflammatory responses [31, 34, 36, 37]. The interplay of these mechanisms highlights the intricate ways in which maternal stress can "program" fetal development, potentially setting the stage for long-term alterations in stress reactivity and neurobehavioral functioning. For example, elevated maternal cortisol levels during pregnancy have been associated with structural and functional changes in brain regions rich in glucocorticoid receptors, such as the hippocampus, amygdala, and prefrontal cortex [27, 34]. These alterations may underlie some of the observed

cognitive and emotional outcomes in children exposed to prenatal stress. Moreover, the emerging field of epigenetics has shed light on how prenatal stress exposure can lead to lasting changes in gene expression patterns that affect neurodevelopment and stress responsivity later in life [34]. The complexity of these biological pathways underscores the need for interdisciplinary research approaches that integrate neuroimaging, epigenetics, and stress biomarkers to provide a more comprehensive understanding of how maternal stress impacts fetal neurodevelopment.

While the association between prenatal maternal stress and offspring neurodevelopment is increasingly well-established, it is crucial to recognize the role of moderating factors in shaping these outcomes. Factors such as the timing and severity of stress exposure, genetic susceptibility, and the postnatal environment all play significant roles in determining the long-term impact of prenatal stress [3, 11, 31].

The timing of stress exposure during pregnancy appears to be a critical moderator. Stress during early gestation has been more strongly associated with cognitive delays, whereas stress in the third trimester is often linked to emotional and behavioral disturbances in children [28]. Moreover research from Davis and Sandman (2010) suggests that chronic or severe stressors may have stronger effects than acute or mild ones [27]. This reflects the notion of "critical periods" of fetal brain development, during which specific regions (e.g., prefrontal cortex, amygdala) are particularly sensitive to environmental perturbations [11].

Genetic factors, such as variations in genes involved in stress response systems, can also influence susceptibility to the effects of prenatal stress, as demonstrated by Pluess et al. [24] in their study of the serotonin transporter gene polymorphism. Furthermore, the postnatal environment, including maternal care and socioeconomic factors, can either exacerbate or buffer the effects of prenatal stress exposure [3]. These findings highlight the complex interplay between genetic predisposition, prenatal environment, and postnatal experiences in shaping neurodevelopmental trajectories. Understanding these moderating factors is crucial for identifying at-risk individuals and developing targeted interventions to mitigate the potential negative effects of prenatal stress on offspring development.

In addition to these moderating factors, it is essential to acknowledge the presence of alternative and interacting risk factors. For example, maternal stress during pregnancy is often correlated with nutritional deficiencies, sleep disturbances, and substance use, each of which can independently affect fetal development [37]. Socioeconomic status (SES) is another major contributor, influencing both maternal stress levels and access to healthcare, education, and psychosocial support. Studies consistently show that lower SES is associated with elevated prenatal stress and poorer child developmental outcomes [14, 19].

Additionally, maternal psychiatric history, especially untreated depression and anxiety, may contribute to both genetic vulnerability and environmental exposure to stress. These maternal conditions are often chronic and may affect parenting practices postnatally, compounding risks for emotional and cognitive difficulties in children [30]. Furthermore, epigenetic mechanisms, including DNA methylation changes in glucocorticoid receptor genes, represent a biological pathway through which maternal stress may shape long-term neurodevelopment [5, 22].

Limitations and areas for future research

While the body of research on prenatal maternal stress and offspring neurodevelopment is expanding, several important limitations must be acknowledged. Most critically, all studies included in this review employed observational designs, which limits our ability to establish causal relationships between maternal stress and neurodevelopmental outcomes. The observed associations, while consistent across studies, may be influenced by unmeasured confounding variables, shared genetic factors, or other environmental influences that co-occur with maternal stress. Without randomized controlled trials – which would be ethically unfeasible for stress exposure – the field remains dependent on observational evidence that can demonstrate association but not causation.

Additional methodological limitations include reliance on non-standardized measures of stress exposure, such as retrospective maternal reports, assessments at single timepoints, and study designs that may not adequately capture the complexity and temporal dynamics of stress exposure during pregnancy. Furthermore, overreliance on maternal cortisol response as a biomarker may be problematic, as cortisol reactivity attenuates as gestation progresses and with repeated stress exposure [30].

To address these limitations, future research should prioritize prospective, multi-time-point assessments of maternal stress while integrating objective biomarkers such as cortisol levels, pro-inflammatory cytokines, and placental gene expression profiles. These approaches may provide more biologically grounded insights into the associations between maternal stress and fetal development. Given that stress responsiveness varies significantly across individuals, incorporating both maternal and fetal biomarker-based assessments could enhance the precision and validity of findings [37, 22].

Additionally, although several biological mechanisms have been proposed to explain the observed associations, further research is required to clarify the specific pathways that may link maternal stress with neurodevelopmental outcomes. There is a pressing need for mechanistic studies that integrate neuroimaging, epigenetics, and stress biomarkers to provide a more detailed understanding of the biological correlates of the observed prenatal stress associations.

Future research should also employ stronger study designs to approach causal inference, such as natural experiments, instrumental variable analyses, or sibling comparison studies that can better control for unmeasured confounding factors. Mendelian randomization studies using genetic variants associated with stress response could also provide insights into potential causal relationships.

Finally, few studies have examined interventions aimed at mitigating the potential associations between maternal stress and adverse neurodevelopmental outcomes. Future research should focus on developing and testing interventions that could reduce maternal stress during pregnancy, while carefully evaluating whether such interventions lead to improved offspring outcomes. This could include stress reduction techniques, mindfulness-based interventions, or targeted psychosocial support for pregnant women experiencing high levels of stress. However, given the observational nature of current evidence, intervention studies will be critical for establishing whether the observed associations reflect causal relationships that can be modified through targeted approaches.

The field would also benefit from longer-term follow-up studies that can distinguish between transient developmental associations and those that persist into adulthood, as well as studies that examine potential protective factors and resilience mechanisms that may moderate the observed associations between prenatal stress and neurodevelopmental outcomes.

Conclusions

This narrative review highlights consistent associations between maternal stress during pregnancy and long-term neurodevelopmental outcomes in offspring across multiple observational studies. The observed relationships between prenatal stress exposure and cognitive, behavioral, and emotional outcomes in children are well-documented, with proposed biological mechanisms including HPA axis alterations, epigenetic modifications, and brain structural changes providing plausible pathways. However, causal relationships remain to be established given the observational nature of all included studies.

Future research should prioritize longitudinal studies, mechanistic investigations, and intervention trials to determine whether these associations reflect causal relationships. Understanding these associations may ultimately inform strategies to promote optimal child development, though establishing causality will be essential before definitive preventive recommendations can be made. While the consistency of findings suggests that integrating maternal mental health support into prenatal care warrants investigation, the effectiveness of such approaches requires rigorous testing through intervention studies designed to establish causality rather than association alone.

The field would benefit significantly from research designs that can more definitively establish causal relationships, including natural experiments, sibling comparison studies, and randomized controlled trials of stress reduction interventions. Only through such approaches can we move beyond demonstrating associations to establishing whether maternal stress during pregnancy represents a modifiable risk factor for adverse neurodevelopmental outcomes in children.

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