*REVIEW*

# *Maternal Factors during Pregnancy Affecting Offspring Neurodevelopmental Outcomes: A Narrative Review*

Mehran Karimi[1]*

[1] Mother and Newborn Health Research Center, Comprehensive Research Institute for Maternal and Child Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran

* Correspondence: Mehran Karimi, drmehrankarimi@yahoo.com

**Running Title:** Maternal Factors and Offspring Neurodevelopment

**Abstract**

**Background:** Gestation is a critical window during which maternal biological, psychological, and environmental factors influence offspring neurodevelopment. This structured narrative review synthesizes evidence on maternal pregnancy factors associated with adverse neurodevelopmental outcomes.

**Methods:** A structured search of PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar was conducted through September 2026, focusing mainly on recent studies. Approximately 400 records were screened. Studies were synthesized by thematic exposure domain with critical appraisal of design, confounding, and generalizability.

**Results:** Key domains included maternal metabolic conditions, mental health, immune activation/infection, nutrition, environmental toxicants, medications/substance use, and socioeconomic determinants. Evidence was strong for obesity, diabetes, depression, air pollution, and substance use; moderate for hypertensive disorders and antidepressants; growing for polyfluoroalkyl substances (PFAS) and sleep disturbance. Most evidence is observational and should not be interpreted causally. Cumulative exposure and first-trimester vulnerability emerged as key themes.

**Conclusion:** Maternal factors are associated with offspring neurodevelopmental outcomes through multiple interacting pathways. Many risk factors are modifiable, supporting integrated preconception and prenatal interventions to optimize offspring neurodevelopment and reduce the global burden of developmental delay.

**Keywords**: Maternal Exposure; Pregnancy; Neurodevelopment; Developmental Disabilities

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**How to cite:** Karimi M. Maternal Factors during Pregnancy Affecting Offspring Neurodevelopmental Outcomes: A Narrative Review. J Integr Matern Pediatr Care. 2026;9(1):21-38
DOI:10.18502/jimpc.v9i1.22981

Received: September 12, 2026
Revised: October 11, 2026
Accepted: October 21, 2026

Journal of Integrated Maternal and Pediatric Care

2026;9(1):21-38

ISSN: 3134-0792

DOI: 10.18502/jimpc.v9i1.22981

Mehran Karimi

## Introduction

The developmental origins of health and disease (DOHaD) framework posits that environmental exposures during critical developmental windows, particularly gestation, may alter developmental trajectories through epigenetic modifications, programming of physiological systems, and structural changes in developing organs, including the brain [1, 2]. This framework emphasizes that the period from conception through early childhood (the "first 1000 days") represents a uniquely sensitive window for establishing lifelong health trajectories. The present review focuses specifically on the prenatal component of this critical period (approximately 270 days of gestation), while acknowledging that postnatal factors during the remainder of the first 1000 days also contribute substantially to developmental outcomes.

For the purposes of this review, the term "neurodevelopmental outcomes" is used as an umbrella term encompassing developmental delay (delays in achieving expected milestones across cognitive,

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motor, language, and social-emotional domains), autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), intellectual disability, and related behavioral and cognitive difficulties observed from infancy through adolescence. In low- and low-middle income countries, prevalence estimates suggest that a substantial proportion of children experience some form of developmental delay or neurodevelopmental disorder [3], though estimates vary considerably depending on definitions, assessment methods, and population characteristics.

This narrative review was conducted to synthesize current evidence on maternal factors during pregnancy affecting offspring neurodevelopmental outcomes. The review was informed by a structured literature search (described in Section 2) and includes studies published predominantly in the last two years (January 2025 to September 2026), with landmark studies from earlier periods included where essential for context. The authors acknowledge that this is an expert narrative synthesis rather than a systematic review, reflecting the heterogeneity of exposures, outcomes, and study designs in this field, which precludes a formal systematic review and meta-analysis.

# Materials and Methods

## Review Design and Rationale

This is a structured narrative review. The term structured indicates that we used a predefined search strategy, explicit inclusion and exclusion criteria, and a documented screening procedure. It is not a systematic review, because we did not perform duplicate independent screening, formal risk-of-bias scoring, or quantitative meta-analysis. The narrative approach was selected because the heterogeneity of maternal exposures, neurodevelopmental outcomes, and study designs makes formal systematic review methodology impractical for this broad topic.

## Literature Search Strategy

A structured literature search was conducted using PubMed/MEDLINE as the primary database, supplemented by Scopus, Web of Science, and Google Scholar. The final search was conducted on September 8, 2026. Search terms included combinations of MeSH and free-text terms related to (“maternal factors” OR “prenatal exposure” OR “pregnancy complications”) AND (“neurodevelopment” OR “developmental delay” OR “cognitive development” OR “autism spectrum disorder” OR “attention deficit hyperactivity disorder” OR “intellectual disability”). Reference lists of included articles and relevant systematic reviews were manually screened. Approximately 400 records were screened, from which studies meeting the inclusion criteria were selected for synthesis. A PRISMA-style flow diagram is provided in Supplementary Table S1. All references were verified against PubMed or the original source.

## Study Selection Criteria

Studies were included if they: (1) examined maternal factors during the preconception, prenatal, or perinatal periods; (2) assessed offspring neurodevelopmental outcomes; and (3) were published in English in peer-reviewed journals. One non-English clinical guideline (Kautzky-Willer et al., 2026) was included because it provides updated management recommendations for hyperglycemia in pregnancy that were not available in English-language sources at the time of writing. Studies were excluded if they focused exclusively on postnatal maternal factors or examined only genetic conditions without considering environmental modifiers. One preprint (Lindseth et al.) is included because it provides directly relevant neuroimaging data for which no peer-reviewed equivalent was available at the time of writing; this exception is noted in the Limitations section.

## Data Synthesis and Critical Appraisal Approach

Given the heterogeneity of study designs, exposure assessments, and outcome measures, a narrative synthesis approach was employed. Studies were organized into thematic domains based on the nature of the maternal exposure. Within each domain, we prioritized larger prospective cohort studies, randomized controlled trials, and systematic reviews over smaller or retrospective studies. For critical appraisal, we considered the following factors consistently across domains: study design quality, sample size and power, control for confounding, exposure assessment validity, outcome measurement reliability, and generalizability of findings. Where methodological limitations substantially affect the strength of conclusions, these are explicitly noted in "Critical Appraisal" subsections within each thematic domain. The "Strength of Evidence" assessments in Table 1 reflect the authors' qualitative synthesis of these appraisal factors using a weighting approach that prioritizes consistency of findings, study design quality, and degree of confounding control.

# Maternal Metabolic Conditions

## Obesity and Body Mass Index

Maternal obesity represents one of the most extensively studied metabolic risk factors for offspring neurodevelopmental outcomes. Multiple large cohort studies have demonstrated consistent associations between maternal pre-pregnancy body mass index (BMI) and offspring neurodevelopmental disorders, with elevated risks observed across diverse populations [4-6]. The review by Schmitt et al. detailed how maternal obesity is associated with a pro-inflammatory intrauterine environment and may induce placental changes and offspring neuro-inflammation through microglial activation [5].

**Critical Appraisal:** The association between maternal obesity and adverse neurodevelopmental outcomes is supported by consistent findings across multiple large, prospective cohort studies from diverse populations. However, residual confounding remains a significant concern. Socioeconomic status, maternal intelligence, and shared genetic factors may partially explain observed associations. Studies using sibling comparison designs or Mendelian randomization provide stronger causal inference but have important limitations, including potential pleiotropy and weak instrument bias. The evidence is rated as "Strong" due to consistency across cohorts, though causal interpretation requires caution.

Mechanistic studies have identified several pathways linking maternal obesity to developmental outcomes, including epigenetic regulation of imprinted genes critical for neurodevelopment [7]. Sex differences in vulnerability are consistently observed across multiple exposure domains, with evidence that fetal sex interacts with maternal metabolic status to determine vulnerability [8].

Importantly, gestational weight gain (GWG) independent of pre-pregnancy BMI has emerged as a modifiable risk factor. Both excessive and inadequate GWG were associated with altered neurodevelopmental trajectories in prospective cohorts [9, 10]. The pregnancy lifestyle intervention study by Geyer et al. demonstrated that interventions targeting maternal health behaviors can improve child somatic growth and neurodevelopment [11].

## Gestational Diabetes Mellitus and Hyperglycemia

Maternal diabetes, both pre-existing and gestational, is consistently associated with elevated risk of offspring neurodevelopmental disorders. Reviews have synthesized evidence specifically linking maternal diabetes to ASD and ADHD, finding consistent associations across diverse populations, with dose-response relationships observed between glycemic control and outcome severity [12]. The Finnish Gestational Diabetes register-based study extended these findings to broader mental and behavioral disorders, demonstrating elevated risks extending into adolescence [13].

**Critical Appraisal:** The evidence linking maternal diabetes to offspring neurodevelopmental outcomes is generally strong, with consistency across study designs and populations. However, observed associations may be partially confounded by environmental pollutants that co-occur with metabolic dysfunction [14]. This is particularly relevant in low-income communities where both metabolic dysfunction and environmental toxicant exposure are more prevalent. National cohort and meta-analytic evidence support the association between maternal diabetes and offspring neurodevelopmental outcomes [15, 16]. The evidence is rated as “Strong” due to the consistency of findings, dose-response relationships, and support from multiple study designs.

Mechanistic investigations suggest that prenatal glucose intolerance may be associated with altered fetal brain development detectable through neuroimaging as early as infancy. The IMPACT BCN trial, discussed in Section 7.1, demonstrated that Mediterranean diet and stress-reduction interventions during pregnancy affected fetal brain development as detected by MRI [17]. Importantly, even subclinical glucose intolerance, below diagnostic thresholds for gestational diabetes, was associated with increased neurodevelopmental disorder risk [18].

Management guidelines emphasize the importance of optimizing glycemic control during pregnancy, with emerging evidence suggesting that stringent glucose management may mitigate neurodevelopmental risks [19]. Longitudinal cohort data demonstrated that neonatal circulating amino acids and lipid metabolites mediate the association between maternal GDM and offspring neurodevelopment, providing potential biomarkers for early identification of at-risk infants [20].

## Hypertensive Disorders of Pregnancy

Borderline hypertension defined by ACC-AHA criteria, below the traditional threshold for gestational hypertension, was associated with adverse infant neurodevelopment in a large Chinese cohort [21]. The review by Qi et al. detailed how preeclampsia alters epigenetic regulation of imprinted genes critical for neurodevelopment [7].

**Critical Appraisal:** The evidence for hypertensive disorders as developmental risk factors is rated as "Moderate." While associations are consistent, separating the effects of hypertension per se

from those of associated conditions (such as obesity, diabetes, and placental dysfunction) is methodologically challenging. The finding that even borderline hypertension is associated with adverse outcomes is intriguing but requires replication in other populations.

Nivins et al. demonstrated that prenatal exposure to maternal hypertension combined with higher BMI conferred a particularly elevated risk for neurodevelopmental and psychiatric disorders, suggesting synergistic effects between metabolic and vascular pathologies [22]. The causal pathways likely involve placental insufficiency, chronic fetal hypoxia, and elevated inflammatory signaling [23].

# Maternal Mental Health

## Depression and Depressive Symptoms

Maternal depression during pregnancy is among the most robust predictors of adverse offspring neurodevelopmental outcomes. Large prospective cohort studies have demonstrated associations between antenatal depression and a wide spectrum of outcomes, including ADHD, disruptive behavioral disorders, and neurodevelopmental delay in toddlers [24-26].

**Critical Appraisal:** The association between maternal depression and offspring neurodevelopmental outcomes is among the most consistently replicated findings in perinatal epidemiology, warranting a "Strong" evidence rating. However, the challenge of confounding by indication remains paramount: women with depression may differ from non-depressed women in numerous ways (socioeconomic status, health behaviors, health care utilization) that independently influence child development. Studies that account for these confounders typically find attenuated but still significant associations. Furthermore, the distinction between the effects of depression itself versus its treatment (antidepressant medications) remains incompletely resolved.

The biological mechanisms linking maternal depression to developmental outcomes are increasingly well-characterized, including metabolic alterations [27] and increased expression of human endogenous retroviruses (HERVs) in mothers with perinatal depression [28]. Sex differences in the risk of autistic-related traits were demonstrated by Duan et al., using both human cohort and mouse model approaches [29].

## Anxiety, Stress, and PTSD

Maternal childhood maltreatment represents a particularly potent predictor of adverse pregnancy outcomes and offspring development, with evidence for biological embedding of trauma through inflammatory pathways [30, 31]. Jones et al. found that maternal perceived stress during pregnancy was associated with offspring DNA methylation changes in HPA axis genes at birth [32].

**Critical Appraisal:** The evidence linking maternal stress and anxiety to developmental outcomes is substantial but rated as "Substantial" rather than "Strong" due to measurement heterogeneity. Different studies use varying instruments and time points, making meta-analytic synthesis challenging. Furthermore, the distinction between acute stress (such as natural disasters) and chronic stress (such as childhood maltreatment) may have different mechanistic implications that are not always clearly delineated in the literature.

The review by Williams et al. specifically examined maternal PTSD and its impact on pregnancy outcomes, highlighting alarming health disparities among Black women that compound developmental risk [33]. Natural disasters provide natural experiments for studying prenatal stress effects, with the Malawian cohort study demonstrating that extreme cyclone exposure during pregnancy was associated with both maternal mental health deterioration and subsequent child developmental delays [34].

## Maternal Sleep Disturbance

Emerging research has identified maternal sleep as an under-appreciated factor in offspring development. Zhang et al. found that maternal short sleep duration during pregnancy was associated with neurodevelopmental delay in offspring, with dose-response relationships observed [35]. Lunsford-Avery et al. demonstrated that sleep complaints during pregnancy were independently associated with toddler cognition even after accounting for depression [36].

**Critical Appraisal:** Research on maternal sleep and offspring development is relatively new, with a limited number of studies. While initial findings are promising, residual confounding by stress, depression, and other factors requires careful consideration. The evidence is rated as "Growing." Replication in independent cohorts with objective sleep measures (actigraphy and polysomnography) rather than self-report would strengthen causal inference.

# Maternal Immune Activation and Infections

## Prenatal Infections

The maternal immune activation (MIA) hypothesis has gained substantial empirical support. Experimental studies have demonstrated that maternal immune activation combined with postnatal anoxia impaired olfactory discrimination and increased oxidative stress in the olfactory bulb [37]. Gardner et al. critically evaluated the evidence for MIA as a causal factor in autism, concluding that while causation is plausible, rigorous causal inference requires careful consideration of confounding [38].

**Critical Appraisal:** The MIA hypothesis is supported by robust experimental evidence from animal models and consistent epidemiological associations, warranting a "Strong" rating for experimental support. However, translating these findings to human populations is complicated by the heterogeneity of infectious exposures, the timing of infection relative to neurodevelopmental events, and the challenge of distinguishing infection-specific effects from those of shared genetic or environmental factors. The finding by Zarchev et al. that infection, but not inflammation, predicts ADHD is particularly important and requires replication [39].

The population-based cohort study by Zarchev et al. made an important distinction: maternal infection, but not inflammation alone, was associated with ADHD symptoms during childhood [39]. Hardie et al. found that prenatal maternal infections were associated with early childhood developmental outcomes across multiple domains [40].

Zika virus infection during pregnancy has emerged as a particularly devastating cause of developmental disability [41]. Cytomegalovirus (CMV) infection represents another significant infectious risk [42]. The immunoglobulin findings by Borbye-Lorenzen et al. demonstrated that high levels of IgG targeting mycoplasma or CMV in newborns increased ADHD risk [43].

## Maternal Autoimmune Conditions

The nationwide cohort study by Skott et al. examined prenatal exposure to a wide spectrum of maternal autoimmune disorders and found associations with psychiatric and neurodevelopmental disorders in offspring [44]. Maternal immune-mediated conditions were associated with ADHD risk in the large Norwegian cohort study by Walle et al. [45].

# Environmental Exposures

## Air Pollution

Prenatal exposure to air pollution is among the most consistent environmental risk factors for neurodevelopmental disorders. Zhang et al. found associations between maternal fine particulate matter (PM2.5) exposure and ASD [46]. Cloutier et al. identified specific PM2.5 constituents as particularly toxic for autism risk [47]. Oh and Song extended these findings to a broad range of neurodevelopmental disorders in a nationwide Korean cohort [48].

**Critical Appraisal:** The evidence linking air pollution to neurodevelopmental outcomes is among the strongest in environmental epidemiology, warranting a "Strong" rating. Findings are consistent across diverse populations, exposure assessment methods, and outcome definitions. However, effect sizes are generally modest, and air pollution is correlated with other environmental and social factors (noise, traffic, socioeconomic deprivation), making it difficult to isolate the specific contribution of particulate matter or gaseous pollutants. Individual-level exposure misclassification may attenuate associations.

O'Sharkey et al. found that grandmaternal exposure to air pollution was associated with grandchild ASD risk, suggesting trans-generational effects [49].

## Endocrine-Disrupting Chemicals

Per- and polyfluoroalkyl substances (PFAS) have received considerable attention due to their widespread exposure and potential developmental toxicity. Yang et al. found that maternal PFAS exposure was associated with offspring growth trajectories from birth to age 2 years [50]. The parental comparison study by Guo et al. provided particularly strong evidence by comparing maternal and paternal exposure, finding that maternal exposure was more strongly associated with child behavioral difficulties, consistent with direct prenatal effects [51].

**Critical Appraisal:** The evidence for PFAS effects on neurodevelopment is rated as "Growing." Study heterogeneity in terms of specific PFAS congeners measured, exposure levels, and outcome assessments complicates synthesis. Furthermore, PFAS are highly persistent and correlated with other environmental and lifestyle factors, making confounding a persistent concern. The parental comparison design used by Guo et al. is methodologically strong and provides evidence for direct prenatal effects rather than shared environmental or genetic confounding [51].

Bisphenol A (BPA) and related bisphenols have been extensively examined. Vian Matias de Oliveira et al. found associations between prenatal BPA exposure and neurocognitive changes in

children aged 2–5 years [52]. Phthalates and plasticizers were examined in prospective cohorts, with evidence that first-trimester exposure was associated with personal-social and language deficits, with maternal inflammation mediating these associations [53].

### Heavy Metals and Trace Elements

Zhang et al. examined maternal early-pregnancy biomarkers of mercury, manganese, selenium, and zinc exposure, finding complex associations with child neurobehavioral development [54]. Kim et al. found that prenatal heavy metal exposure was associated with autistic trait trajectories in childhood [55].

**Critical Appraisal:** The effects of heavy metals on neurodevelopment are well-established for lead and mercury, with strong evidence from both epidemiological and experimental studies. Essential trace elements such as zinc and selenium have U-shaped relationships with neurodevelopment, with both deficiency and excess being problematic. This complexity requires careful consideration of dose-response relationships rather than simple dichotomous exposure classifications. The evidence is rated as "Strong" for lead and mercury, but less consistent for other metals.

### Pesticides and Industrial Chemicals

Mwakalasya et al. found that maternal pesticide exposure during pregnancy was associated with adverse neurodevelopmental outcomes in Tanzanian children [56]. Wager and Thompson synthesized evidence on synthetic chemicals broadly, emphasizing the need for a precautionary approach to chemical exposure during pregnancy [57].

## Nutritional Factors

### Maternal Dietary Patterns

López-Yerena et al. examined how modern eating patterns endanger maternal and offspring health, highlighting that Western dietary patterns are associated with adverse developmental outcomes [58]. The IMPACT BCN trial suggested that Mediterranean diet and stress-reduction interventions during pregnancy may affect fetal brain development as detected by MRI, providing direct evidence that maternal diet influences neurodevelopment [17]. This is the primary discussion of the IMPACT BCN trial in this review.

**Critical Appraisal:** Dietary studies face substantial methodological challenges, including measurement error in dietary assessment and confounding by socioeconomic status and lifestyle factors. The randomized controlled trial design used in the IMPACT BCN trial provides stronger causal evidence but may not be generalizable to populations outside the Mediterranean region. The evidence is rated as "Moderate" for dietary patterns, with the RCT evidence providing stronger support than observational studies.

### Micronutrient Deficiencies and Supplementation

The randomized controlled trial by Kvestad et al. examined vitamin B-12 supplementation during pregnancy, finding beneficial effects on early motor performance [59]. This RCT provides valuable causal evidence that addressing micronutrient deficiencies can improve developmental outcomes. Ledowsky et al. examined the effects of excess folic acid and high blood folate during preconception and pregnancy, highlighting that both deficiency and excess can be problematic [60].

**Critical Appraisal:** The evidence for micronutrient supplementation is strongest when baseline deficiency is prevalent. The finding that excess folic acid may also be problematic underscores the importance of personalized nutritional recommendations. The evidence is rated as "Strong" for specific nutrients (such as vitamin B-12, iron, omega-3 fatty acids) when deficiency is present, but the generalizability to well-nourished populations is uncertain.

Musillo et al. examined prenatal omega-3 fatty acid supplementation, finding beneficial effects on cognitive outcomes [61].

## Medications and Substance Use

### Antidepressant Medications

The systematic review and meta-analysis by Chan et al. examined maternal and paternal antidepressant use before and during pregnancy, finding elevated offspring risk of neurodevelopmental disorders [62]. The authors appropriately noted the challenge of confounding by indication.

**Critical Appraisal:** This is one of the most methodologically challenging areas in perinatal epidemiology. Untreated maternal depression carries its own developmental risks for offspring, making it unethical to randomize pregnant women to treatment versus no treatment groups. Observational studies that adjust for maternal depression severity, typically find attenuated associations between antidepressant exposure and adverse outcomes, suggesting that residual confounding by

indication remains a substantial concern. Sibling comparison studies, which control for stable maternal characteristics, have generally found smaller or null associations, though they have their own limitations. The evidence is rated as "Moderate" due to the inherent difficulty of establishing causal effects. The clinical implications are nuanced: the decision to use antidepressants during pregnancy must balance the risks of untreated depression against the potential risks of medication exposure.

### Antiepileptic Medications

Saramak et al. examined lacosamide safety during pregnancy and breastfeeding [63]. König et al. found that in utero exposure to antiepileptic drugs was associated with altered cortical networks and neurophysiological outcomes at age 6 years [64].

### Opioids and Substance Use

Kanervo et al. found challenges at age four following fetal exposure to maternal opioid maintenance treatment [65]. Young-Wolff et al. found that prenatal cannabis use was associated with ADHD and disruptive behavior disorders [66]. Park et al. found that maternal smoking based on pre-birth health examination was associated with neurodevelopmental disorders in offspring [67].

**Critical Appraisal:** The epidemiology of prenatal substance use is complicated by poly-substance use, making it difficult to attribute outcomes to any single substance. Furthermore, women who use substances during pregnancy differ from non-users in numerous ways (socioeconomic status, mental health, prenatal care utilization, and nutrition) that independently influence child development. The evidence is rated as "Strong" for alcohol and tobacco, but less consistent for cannabis and opioids due to confounding and poly-substance use.

## Paternal Factors

While this review focuses on maternal factors, emerging evidence demonstrates that paternal factors also contribute to offspring development. Paternal obesity is associated with altered sperm epigenetics, including DNA methylation and microRNA profiles, and with offspring metabolic and neurodevelopmental outcomes [68]. Paternal age, smoking, alcohol use, PFAS exposure, and psychosocial stress may also influence offspring development through seminal plasma factors, epigenetic reprogramming, and shared familial liability. Parental comparison studies have shown that maternal PFAS exposure is more strongly associated with child behavioral outcomes than paternal exposure, supporting direct prenatal effects [51]. This section is included to acknowledge the importance of both parents while maintaining the maternal focus in this review; a comprehensive review of paternal factors is beyond the scope of this manuscript.

## Mechanisms and Mediators

### Epigenetic Mechanisms

Qi et al. detailed epigenetic regulation of imprinted genes in neurodevelopment [7]. Jones et al. found that maternal stress was associated with offspring DNA methylation changes in HPA axis genes [32]. Wang et al. tracked molecular profiles from pregnancy through childhood, identifying metabolic signatures predictive of neurodevelopmental disorders at age 10 years [69].

**Critical Appraisal:** While epigenetic mechanisms are biologically plausible and supported by experimental evidence, human studies face significant challenges. DNA methylation patterns are cell-type specific, and most studies use heterogeneous tissue samples (such as cord blood) that may not reflect brain-specific epigenetic modifications. Furthermore, establishing causality in epigenetic studies requires longitudinal designs that distinguish cause from consequence, and the functional significance of observed methylation changes is often unclear.

### Microbiome and Immune Mechanisms

Matera et al. examined the maternal microbiome in fetal programming [70]. Chen et al. mapped the complex interactions between environmental exposures, the microbiome, and infant health [71].

### Oxidative Stress and Mitochondrial Mechanisms

Gouveia et al. demonstrated that maternal immune activation increased oxidative stress in the olfactory bulb [37]. Nguyen et al. found that maternal diet was associated with malondialdehyde in cord blood [72]. Peeples et al. examined mechanisms linking pregnancy exposures to autism through sterol pathway disruption [73].

### Sex Differences in Vulnerability

Sex differences in vulnerability are consistently observed across multiple exposure domains, including maternal obesity [8], maternal depression [29], and maternal immune activation. These differences likely reflect interactions between sex hormones, epigenetic programming, and differential

timing of neurodevelopmental events in male versus female fetuses. Male offspring generally show greater vulnerability to prenatal exposures, though the mechanisms remain incompletely understood. A more integrated discussion of sex-specific effects is warranted, as these differences may have important implications for risk stratification and intervention.

## Cumulative and Interactive Effects

A key theme emerging from this review is that maternal risk factors rarely occur in isolation. Metabolic dysfunction, mental health conditions, environmental exposures, and socioeconomic disadvantage often cluster within the same individuals and communities. The cumulative burden of multiple exposures likely has greater impact on neurodevelopmental outcomes than any single factor. Emerging methods for studying exposure mixtures (such as Bayesian kernel machine regression, weighted quantile sum regression) are beginning to address this complexity but have not yet been widely applied in this field. Future research should prioritize multi-exposure models that account for these complex interactions. A conceptual overview of these interacting mechanistic pathways and key modifying factors is provided in Figure 1.

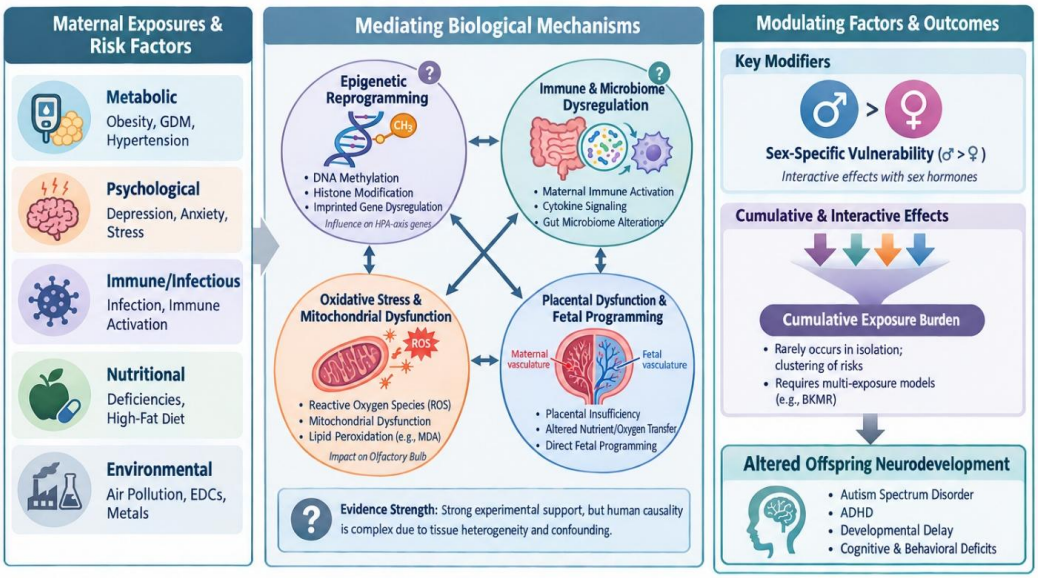



**Figure 1.** Conceptual model of the multifaceted mechanisms linking prenatal exposures to offspring neurodevelopment. Arrows indicate established or hypothesized pathways. Abbreviations: EDC, Endocrine-Disrupting Chemical; GDM, Gestational Diabetes Mellitus; HPA, Hypothalamic-Pituitary-Adrenal; MDA, Malondialdehyde; ROS, Reactive Oxygen Species. Graphic generated using OpenAI ChatGPT (GPT-5.6 Luna).

To further contextualize these interactions within a temporal framework, Figure 2 provides a detailed timeline of sensitive windows of vulnerability, aligning specific maternal exposures with the corresponding stages of fetal brain development.

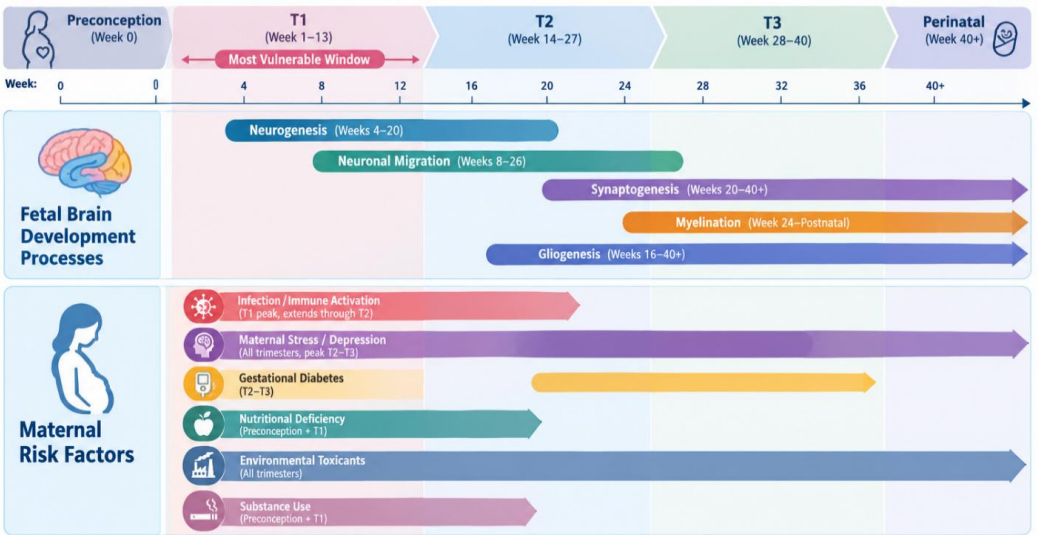



**Figure 2.** Sensitive windows of vulnerability: timing of maternal exposures and fetal brain development. This temporal framework illustrates the critical overlap between specific stages of fetal neurogenesis (including neurogenesis, neuronal migration, synaptogenesis, myelination, and gliogenesis) and the distinct periods during

which various maternal exposures (including infection, stress, metabolic dysfunction, nutritional deficiency, and environmental toxicants) exert their greatest influence. Notably, the first trimester (T1) is highlighted as the "Most Vulnerable Window." Abbreviations: T1, first trimester; T2, second trimester; T3, third trimester. Abbreviations: EDC, Endocrine-Disrupting Chemical; GDM, Gestational Diabetes Mellitus; HPA, Hypothalamic-Pituitary-Adrenal; MDA, Malondialdehyde; ROS, Reactive Oxygen Species. Graphic generated using OpenAI ChatGPT (GPT-5.6 Luna).

### Genetic Moderation, Polygenic Susceptibility, and Shared Familial Liability

Neurodevelopmental disorders are highly heritable. Twin and family studies have consistently demonstrated substantial genetic contributions to both ASD and ADHD, though recent meta-analyses have revised heritability estimates downward and highlighted the importance of shared environmental effects. For ASD, meta-analytic estimates of heritability range from approximately 64% to 74%, with shared environmental factors accounting for a non-negligible proportion of variance [74]. For ADHD, heritability estimates are consistently in the range of approximately 70–80%, with meta-analyses reporting heritability of approximately 71% for inattention and 73% for hyperactivity/impulsivity [75, 76]. Shared familial liability may therefore confound associations between maternal exposures and offspring outcomes. For example, maternal obesity, depression, and ADHD may share genetic architecture with offspring neurodevelopmental traits, and maternal genetic variants can influence the intrauterine environment through pathways such as inflammation, metabolic dysfunction, and placental function [77, 78]. This phenomenon, sometimes termed genetic nurture, complicates causal interpretation of observational associations [77].

Polygenic risk scores (PRS) provide one approach to indexing genetic susceptibility. Emerging evidence suggests that prenatal exposures may interact with polygenic susceptibility, though findings are disorder-specific, exposure-specific, and sometimes inconsistent in direction. Some studies report that exposure effects are stronger among genetically vulnerable individuals, while others find no significant interaction or even negative interactions depending on the disorder and exposure type [79]. Gene–environment interaction studies remain underpowered and inconsistent, particularly for ASD and ADHD [79].

Family-based designs are particularly informative. Sibling comparison studies control for stable maternal and familial characteristics and can help distinguish direct intrauterine effects from shared genetic or environmental confounding [80]. Negative-control designs, such as comparing maternal versus paternal exposure, provide another approach to distinguish direct prenatal effects from shared familial confounding. For example, Guo et al. found that maternal PFAS exposure was more strongly associated with child behavioral difficulties than paternal exposure, consistent with direct intrauterine effects [51]. Methodological work on sibling comparison designs has also highlighted important limitations, including bias from non-shared confounders and measurement error [81]. Mendelian randomization can also strengthen causal inference, but it requires valid instruments and is vulnerable to pleiotropy, weak instrument bias, and canalization [82]. Future studies should integrate PRS, family-based designs, and multi-omics to identify gene–environment interactions before strong causal claims are made.

## Clinical Implications and Interventions

### Preconception and Antenatal Care

Adsul et al. demonstrated that antenatal care shapes cognitive development in India [83]. The NICE guidelines for maternal and child nutrition provide evidence-based recommendations for nutrition and weight management in pregnancy [84]. These findings underscore the importance of early engagement with prenatal care as a foundation for optimizing neurodevelopmental outcomes.

### Risk Communication and Shared Decision-Making

Miranda Montoya et al. examined risk communication preferences among pregnant women in the context of emerging pathogens [85]. Falutz et al. examined perinatal risk factors for NEET outcomes, emphasizing the long-term societal costs of developmental vulnerability [86].

### Targeted Interventions

As discussed in Section 7.1, the IMPACT BCN trial demonstrated that the Mediterranean diet and stress-reduction interventions during pregnancy affected fetal brain development [17]. Kvestad et al. demonstrated that vitamin B-12 supplementation improves early motor performance [59]. Geyer et al. demonstrated that lifestyle interventions targeting maternal health behaviors improve child outcomes [11].

Community-based approaches have also shown promise. Mondal et al. demonstrated that community-based interventions can catalyze health and nutrition behaviors [87]. Montesanti et al. provided evidence that culturally-appropriate interventions can support Indigenous mothers [88].

**Critical Appraisal of Intervention Evidence.** While these interventions show promise, the evidence base for specific interventions remains heterogeneous. The strongest evidence exists for nutritional supplementation in deficient populations and for lifestyle interventions targeting multiple risk factors. However, few interventions have been evaluated for their long-term neurodevelopmental outcomes, and most studies have focused on short-term or intermediate outcomes. Implementation challenges, particularly in low-resource settings, require further study.

# Health Disparities and Social Determinants

The review by Williams et al. highlighted that maternal PTSD, adverse pregnancy outcomes, and health disparities are particularly pronounced among Black women [33]. This finding must be understood within the broader context of structural racism, which operates as a fundamental cause of health disparities through multiple pathways, including differential access to quality healthcare, environmental exposures, chronic stress, and economic disadvantage. The weathering hypothesis provides a useful framework for understanding how chronic exposure to racism and discrimination accelerates biological aging and increases vulnerability to adverse pregnancy outcomes. Allostatic load, the cumulative physiological burden of chronic stress, has been identified as a key mediator linking social disadvantage to health outcomes [89].

**Critical Appraisal.** Research on racial and ethnic disparities must move beyond descriptive analyses to interrogate the structural mechanisms driving observed differences. The concept of intersectionality, the recognition that multiple marginalized identities (race, class, gender, disability status) interact to produce distinct experiences, should inform both research design and intervention development. Studies that simply adjust for race without examining the underlying structural determinants risk reifying biological interpretations of what are fundamentally social phenomena.

Ingeborgrud et al. found that low perceived social support during pregnancy was associated with anxiety and ADHD symptoms in offspring [90]. Terada et al. found that allostatic load mediated the association between socioeconomic disadvantage and autism risk [89]. These findings suggest that social support and stress reduction may represent modifiable intervention targets with potential developmental benefits.

# Future Directions

## Precision Medicine Approaches

Heo et al. identified sex-based differences in prenatal and perinatal predictors of autism spectrum disorder using machine learning [91]. Tu et al. assessed the quality of machine learning techniques in identifying maternal risk factors [92].

Research Recommendations:

1. **Multi-omics integration:** Future studies should combine genomics, epigenomics, transcriptomics, proteomics, and metabolomics to identify molecular signatures predictive of developmental risk.
2. **Exposure mixture methodologies:** Bayesian kernel machine regression (BKMR), weighted quantile sum regression, and other mixture methods should be more widely applied to account for co-occurring exposures. Figure 3 provides a visual representation of this cumulative exposure burden and the rationale for utilizing these multi-exposure models.
3. **Sibling comparison designs:** To control for stable familial confounding, sibling comparison studies provide stronger causal inference.**Longitudinal trajectory modeling:** Trajectory models, which can identify distinct developmental pathways and sensitive periods of vulnerability, should be more widely applied to understand how exposures at specific developmental stages influence later outcomes.

## Neuroimaging and Biomarkers

Menu et al. highlighted recent advances in understanding prenatal determinants of neurodevelopment through large-scale neuroimaging [2]. Lindseth et al. linked pregnancy and birth-related risk factors to multivariate fusion of child cortical structure [93]. (This preprint is included because it provides the most directly relevant neuroimaging data on this topic; a peer-reviewed version was not yet available at the time of writing. Findings should be interpreted with caution pending peer review.)

Research Recommendations:

1. **Early biomarker development:** Identification of neonatal or early-infancy biomarkers predictive of later developmental delay would enable targeted intervention during critical windows of plasticity.

2. **Functional neuroimaging:** Studies that link prenatal exposures to functional brain connectivity may provide more direct evidence of altered neural processing. Functional connectivity measures may be particularly sensitive to early alterations in neural network organization, potentially providing earlier indicators of developmental risk.
3. **Replication and validation:** Biomarker research requires rigorous replication in independent cohorts before clinical translation.

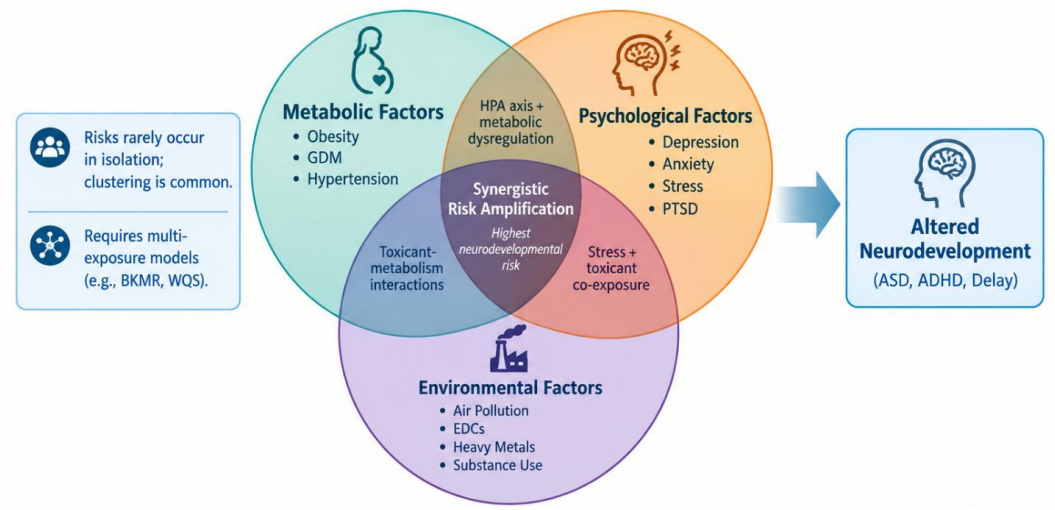



**Figure 3.** Cumulative exposure burden: overlapping maternal risk domains. This Venn diagram illustrates that maternal risk factors rarely occur in isolation; rather, metabolic, psychological, and environmental factors often cluster within the same individuals, leading to synergistic risk amplification (highest neurodevelopmental risk) at their intersections. Assessing these overlapping domains requires multi-exposure models (e.g., BKMR, WQS) to capture their combined effects on altered neurodevelopment (ASD, ADHD, Delay). Abbreviations: EDC, Endocrine-Disrupting Chemical; GDM, Gestational Diabetes Mellitus; HPA, Hypothalamic-Pituitary-Adrenal; PTSD, Post-Traumatic Stress Disorder; BKMR, Bayesian Kernel Machine Regression; WQS, Weighted Quantile Sum. Graphic generated using OpenAI ChatGPT (GPT-5.6 Luna).

### Intervention Development and Implementation Science

Research Recommendations:

1. **Implementation science:** Research is needed on how to effectively implement evidence-based interventions in diverse healthcare settings, including low-resource contexts. Specific challenges include integrating maternal mental health screening into routine prenatal care, delivering nutritional interventions in food-insecure communities, and addressing barriers to healthcare access.
2. **Adaptive intervention designs:** Sequential multiple assignment randomized trials (SMARTs) can optimize intervention strategies by allowing for treatment adaptation based on individual response.
3. **Community-based participatory research:** Engaging communities in the design and implementation of interventions ensures cultural appropriateness and enhances sustainability.
4. **Trans-diagnostic approaches:** Rather than targeting single exposures or outcomes, interventions that address common underlying mechanisms (such as maternal stress, inflammation, nutrition) may have broader benefits.
5. **Health equity focus:** Intervention research must explicitly address how to reduce, rather than inadvertently widen, health disparities. This requires attention to structural barriers and the social determinants of health.

## Strengths and Limitations

### Strengths

This review has several strengths. First, it provides a comprehensive synthesis of the maternal risk factors affecting offspring neurodevelopmental outcomes, spanning biological, psychological, environmental, and social domains. Second, the inclusion of critical appraisal sections within each thematic domain moves beyond simple description to engage with the quality and limitations of the evidence. Third, the review incorporates highly current literature (through September 2026), ensuring that the synthesis reflects the most recent developments in the field. Fourth, the attention to health disparities and social determinants is an important contribution that is often underrepresented in similar reviews. Finally, the summary table (Table 1) provides a valuable at-a-glance reference for clinicians and researchers.

## Limitations

This review has several limitations that should be acknowledged. First, as a structured narrative review, the literature selection process may be subject to selection bias, and we cannot exclude the possibility that relevant studies were overlooked. Second, given the heterogeneity of study designs, exposure assessments, and outcome measures, we were unable to quantitatively synthesize effect sizes through meta-analysis. Third, the review was limited to publications in English, with one non-English clinical guideline included for context [19]. This exception was made because the guideline provided unique management recommendations relevant to the review's clinical implications section. Fourth, the quality of included studies varied considerably, and we relied on qualitative assessment rather than formal quality scoring tools. Fifth, many of the associations described are observational in nature, and residual confounding by shared genetic, environmental, and socioeconomic factors cannot be excluded. Sixth, the literature in this field is highly susceptible to publication bias, where studies with null or negative findings are less likely to be published, potentially overestimating the strength of relationships between maternal factors and neurodevelopmental outcomes. Seventh, the outcomes grouped under neurodevelopmental outcomes are heterogeneous. ASD, ADHD, intellectual disability, developmental delay, and behavioral disorders differ substantially in etiology, age of onset, diagnostic criteria, and assessment methods. Developmental delay is often identified through screening instruments and may be transient, whereas ASD and ADHD require standardized diagnostic assessment. Combining these outcomes may obscure exposure–outcome specificity and may contribute to inconsistent findings across studies. Future research should prespecify outcomes, use harmonized diagnostic and dimensional measures, and examine exposure–outcome associations separately rather than treating neurodevelopmental outcomes as a single construct. Eighth, the review spans a rapidly evolving field, and new evidence may emerge that modifies the conclusions presented here. Finally, the review focuses exclusively on the prenatal period and does not address the substantial contribution of postnatal factors to neurodevelopmental outcomes. Ninth, one included study (Lindseth et al.) was a preprint rather than a peer-reviewed publication. While its findings are directly relevant to the review's scope, they should be interpreted with caution pending peer review.

# Conclusions

This comprehensive narrative review synthesizes current evidence demonstrating that maternal factors during pregnancy are associated with offspring neurodevelopmental outcomes through multiple interacting pathways. The evidence establishes that maternal metabolic conditions (obesity, diabetes, hypertension), mental health (depression, anxiety, stress, PTSD), immune activation and infections, nutritional factors, environmental exposures, medications, and socioeconomic determinants all contribute to offspring neurodevelopmental risk. However, it is important to emphasize that most of this evidence is observational, and causal inference requires careful consideration of confounding and alternative explanations.

Key themes emerging from this synthesis include:

1. **Multi-domain risk accumulation:** Developmental outcomes are best predicted by cumulative exposure across multiple domains rather than any single factor, suggesting the need for comprehensive approaches to risk assessment and intervention.
2. **Critical windows of vulnerability:** The first trimester and immediate perinatal period represent particularly sensitive windows for many exposures, though effects are observed across gestation.
3. **Epigenetic and biological embedding:** Maternal factors may program offspring development through epigenetic modifications, microbiome alterations, and immune system programming, though the functional significance of these changes requires further investigation.
4. **Modifiability:** Many of the identified risk factors are modifiable, providing opportunities for prevention through preconception counseling, optimized antenatal care, and targeted interventions.
5. **Health equity:** Disparities in exposure to risk factors and access to protective resources underscore the need for equity-focused approaches to maternal-child health.

Future research should prioritize multi-exposure models that account for complex interactions between risk factors, mechanistic studies using multi-omics approaches, and implementation science to translate findings into effective interventions. The ultimate goal is to identify modifiable risk factors and implement evidence-based interventions during the critical prenatal window to optimize offspring neurodevelopmental outcomes and reduce the global burden of developmental delay.

**Table 1.** Summary of Major Maternal Risk Factor Domains and Associated Neurodevelopmental Outcomes.

| Risk factor domain | Associated outcomes | Proposed mechanisms | Representative effect estimates | Strength of evidence |
|---|---|---|---|---|
| **Maternal obesity/high BMI** | ASD, ADHD, intellectual disability | Inflammation, epigenetics, placental dysfunction | ASD: RR ≈ 1.3–1.6; ADHD: OR ≈ 1.2–1.5 | Strong |
| **Gestational diabetes** | ASD, ADHD, epilepsy | Metabolic programming, oxidative stress | ASD: RR ≈ 1.3–1.5; ADHD: RR ≈ 1.3–1.5 | Strong |
| **Hypertensive disorders** | Cognitive deficits, ASD | Placental insufficiency, hypoxia | ASD/cognitive: OR ≈ 1.2–1.4 | Moderate |
| **Maternal depression** | ADHD, behavioral disorders, delay | HPA axis, metabolic alterations, epigenetics | ADHD: HR ≈ 1.4–1.8; behavioral: OR ≈ 1.3–1.6 | Strong |
| **Maternal stress/PTSD** | ASD, behavioral problems | DNA methylation, inflammation | ASD: RR ≈ 1.2–1.5 | Substantial |
| **Maternal infection/immune activation** | ASD, ADHD, cognitive deficits | Inflammation, oxidative stress | ASD: RR ≈ 1.3–1.8; ADHD: HR ≈ 1.2–1.4 | Strong (experimental) |
| **Air pollution** | ASD, neurodevelopmental disorders | Neuroinflammation, oxidative stress | ASD: OR ≈ 1.1–1.5 per IQR | Strong |
| **PFAS/EDCs** | Behavioral difficulties, neurocognition | Thyroid disruption, neurotoxicity | Mixed: OR ≈ 1.1–1.3 | Growing |
| **Heavy metals (lead, mercury)** | Cognitive deficits, autistic traits | Neurotoxicity, oxidative stress | Lead: cognitive decrements; mercury: mixed | Strong |
| **Maternal nutrition (deficiency)** | Motor/cognitive delays | Nutrient deprivation, epigenetic effects | B12 supplementation improves motor scores; deficiency OR ≈ 1.5–2.0 | Strong (specific nutrients) |
| **Antidepressant exposure** | Neurodevelopmental disorders | Serotonergic disruption (unclear) | OR ≈ 1.2–1.5, attenuated in sibling designs | Moderate |
| **Substance use (alcohol, tobacco)** | ADHD, behavioral disorders | Neurotoxicity, epigenetic effects | Alcohol: RR ≈ 1.5–2.0; tobacco: OR ≈ 1.3–1.6 | Strong |

Note. Strength of Evidence Definitions: Strong: Consistent findings across multiple large, prospective cohort studies from diverse populations, with support from meta-analyses or Mendelian randomization where available. Confounding addressed, though residual confounding possible. Moderate: Consistent associations observed, but fewer studies, smaller samples, or more limited control for confounding. Findings require replication. Substantial: Evidence from multiple studies, but with notable heterogeneity in measurement or inconsistent findings across populations. Growing: Emerging evidence from a limited number of studies; preliminary but not yet firmly established. Representative effect estimates are illustrative ranges; exact pooled estimates should be substituted from the cited systematic reviews and meta-analyses where available.

## Abbreviation

| Abbreviation | Full Form |
|---|---|
| ACC-AHA | American College of Cardiology/American Heart Association |
| ADHD | Attention-deficit/hyperactivity disorder |
| ASD | Autism spectrum disorder |
| BKMR | Bayesian kernel machine regression |
| BMI | Body mass index |
| BPA | Bisphenol A |
| CMV | Cytomegalovirus |
| DNA | Deoxyribonucleic acid |
| DOHaD | Developmental Origins of Health and Disease |
| EDC | Endocrine-disrupting chemical |
| FinnGeDi | Finnish Gestational Diabetes |
| GDM | Gestational diabetes mellitus |
| GWG | Gestational weight gain |
| HERVs | Human endogenous retroviruses |
| HPA | Hypothalamic-pituitary-adrenal |
| IgG | Immunoglobulin G |
| MDA | Malondialdehyde |

| | |
|---|---|
| MeSH | Medical Subject Headings |
| MIA | Maternal immune activation |
| MRI | Magnetic resonance imaging |
| NEET | Not in employment, education or training |
| NICE | National Institute for Health and Care Excellence |
| PFAS | Per- and polyfluoroalkyl substances |
| PM2.5 | Fine particulate matter with aerodynamic diameter ≤2.5 μm |
| PTSD | Post-traumatic stress disorder |
| RCT | Randomized controlled trial |
| ROS | Reactive oxygen species |
| SMARTs | Sequential multiple assignment randomized trials |
| T1 | First trimester |
| T2 | Second trimester |
| T3 | Third trimester |
| WQS | Weighted quantile sum |

## Declaration

**Conflict of Interest:**
The authors declare no competing interests.

**Acknowledgements:**
Not applicable.

**Funding:**
This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The work was conducted as part of the authors' academic duties.

**Availability of Data and Materials:**
Not applicable.

**Consent for Publication:**
Not applicable.

**Ethics Approval and Consent to Participate:**
Not applicable.

**AI Disclosure:**
AI-assisted technologies (ChatGPT, OpenAI) were used for language editing and manuscript formatting. The conceptual models in the figures were generated using ChatGPT (OpenAI). The authors take full responsibility for the content and figures.

**Author Contributions:**
Conceptualization and design: M.K. Literature search and synthesis: M.K. Drafting: M.K. Critical revision: M.K. Final approval: all authors.

**Supplementary Information**
The online version contains supplementary material available at https://doi.org/10.18502/jimpc.v9il.22981

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