

Review Article

Preventing postpartum depression through pre-delivery screening and targeted interventions

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Received: 06 November 2025

Accepted: 21 November 2025

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ABSTRACT

Postpartum depression (PPD) is a prevalent and disabling complication of childbirth with significant consequences for both maternal and infant well-being. Despite its complex etiology involving hormonal, genetic, immune, and psychosocial factors, increasing evidence supports that PPD is highly preventable through early risk identification and targeted antenatal interventions. This review synthesizes current evidence on pre-delivery screening methods and preventive strategies. The Edinburgh postnatal depression scale (EPDS), patient health questionnaire (PHQ-9), and generalized anxiety disorder scale (GAD-7) are still the main tools for screening, but new biological and epigenetic markers, such as inflammatory cytokines and gene methylation profiles, show promise as useful additional tools. Recent advances in machine-learning algorithms have further enhanced risk prediction by integrating psychosocial, clinical, and biological variables with high discriminative accuracy. Preventive interventions, particularly cognitive behavioral therapy (CBT), interpersonal psychotherapy (IPT), mindfulness-based cognitive therapy, and peer-support programs, demonstrate consistent efficacy in reducing depressive symptom onset when delivered during pregnancy and postpartum. This review aims to explore the underlying pathogenesis and contributing risk factors for PPD. It also seeks to address pre-delivery screening approaches and preventive measures, with the goal of reducing the risk of developing PPD.

Keywords: Postpartum depression, Prevention, Targeted intervention, Risk assessment, Pre-delivery

INTRODUCTION

Depression is a major global health concern, and projections by the world health organization indicate its burden are increasing. It was the second most burdensome and disability-causing disease globally by 2020 and is predicted to become the number one disease burden by 2030. Depression is more prevalent among

women, who are twice as likely as men to experience depression.¹ This can be attributed to several factors, including biological factors like the hormonal fluctuations, particularly during the menstrual cycle and after menopause.² Furthermore, 10-20% of women were reported to experience PPD.³ It is a common psychiatric complication, affecting approximately 17% of women after childbirth.⁴ Risk factors includes previous history of

depression or psychiatric illness, depressive symptoms during pregnancy, gestational diabetes, and a lack of spousal and social support were the most powerful risk factors.⁵ Of greater concern, PPD accounts for almost 20% of preventable postpartum mortality rates.⁶

PPD is defined as “a depressive episode that begins during pregnancy or the first four weeks after birth,” according to diagnostic and statistical manual of mental disorders, fifth edition (DSM-5) diagnostic criteria. Nevertheless, women remain at risk of developing depression several months after childbirth.⁷ Unattended PPD has negative consequences for both infants and mothers. Studies that followed children of untreated mothers from birth to adolescence have reported that these children are more likely to have poor cognitive function, emotional instability, and violent behavior. Furthermore, they often struggle with psychiatric disorders during adolescence. Maternal outcomes include weight problems, alcohol and drug abuse, social relationship problems, breastfeeding problems, or the development of persistent depression.^{8,9}

Despite the detrimental outcomes of PPD, its etiology remains unclear. PPD has multifaceted risk factors including mental illness, prenatal depression, partner violence, child’s health concerns, breastfeeding difficulties, lack of social support and awareness, as well as restricting cultural customs.^{10,11} Recent studies have reported that gut microbiota and inflammatory factors potentially contribute to the pathogenesis of PPD.¹² All aspects considered, this disorder is often underdiagnosed and untreated. Women are usually hesitant in seeking professional help owing to concerns about medication safety during lactation or the cultural stigma about mental illness. Nevertheless, PPD can be easily prevented through pre-delivery screening along with targeted interventions.

Pre-delivery screening identifies women at risk of developing PPD through psychological, biological, and social indicators such as the EPDS.¹³ Growing evidence reports the effectiveness of targeted interventions such as counseling, education, prenatal cognitive-behavioral therapy, mindfulness, and peer support in reducing the risk of the onset of postpartum depression.¹⁴ Moreover, the recent advancements in artificial intelligence and deep learning have led to the development of algorithms that predict women at risk of developing PPD by integrating obstetric and psychosocial data. Thus, improving sensitivity for early detection.¹⁵ This review aims to explore the underlying pathogenesis and contributing risk factors for PPD. It also seeks to address pre-delivery screening approaches and preventive measures, with the goal of reducing the risk of developing PPD.

LITERATURE SEARCH

This review is based on a comprehensive literature search performed on November 2nd, 2025, in the PubMed and

Clinical Key databases, as well as Google Scholar. Utilizing MeSH (Medical Subject Headings) and relevant keywords such as “postpartum depression”, “prevention”, “targeted intervention”, “risk assessment”, “Pre-delivery”, and “Prediction”. This review aims to explore the underlying pathogenesis and contributing risk factors for PPD. It also seeks to address pre-delivery screening approaches and preventive measures, with the goal of reducing the risk of developing PPD. The search was not restricted by date, language, or type of publication to ensure a broad exploration of the available literature.

DISCUSSION

Sociocultural and demographic risk factors

An association was reported between women’s age and the onset of PPD, where women younger than 25 years are at a higher risk of developing PPD. Furthermore, women from a lower socioeconomic class are also more vulnerable. In detail, low income, level of education, or unemployment often result in a lack of understanding of effective methods for childbearing and postpartum care. Additionally, limited finances may lead to compromising the mother’s health and well-being to lower expenses.^{16,17} Cultural beliefs, such as the preference for a male child, are also among the factors affecting a mother’s psychological well-being.¹⁸

Psychological risk factors

A history of depressive episodes, as well as suffering from depression or anxiety during pregnancy, are among the strongest predictors of PPD. Strong evidence to date from studies conducted on a large-scale correlate’s family history of psychiatric illness with a higher risk of PPD onset. It has repeatedly been observed that past depression is among the most influential risk factors for PPD, where it was reported that women who have history of mood disorders are 3 times more likely to develop PPD than women with no history of psychiatric illness.¹⁹ Additionally, low self-esteem and struggling with stressful life circumstances, including marital issues, were also reported to contribute to higher risk of PPD.²⁰ While these sociocultural and psychiatric characteristics are confirmed as risk factors for PPD underlying mechanisms behind these associations are yet to be explored.

Biological risk factors and pathophysiology

Being a multifaceted complex condition, the pathophysiology of PPD remains unclear. Nevertheless, growing evidence suggests that hormonal fluctuations, genetic variations, immune function, and inflammation may play a role in the pathogenesis of PPD.

Hormones

Rapid fluctuations in reproductive hormones during pregnancy and after giving birth have been associated

with the development of PPD. Considering that estrogen and progesterone are particularly implicated in mechanisms that regulate thyroid function, the hypothalamic-pituitary-adrenal axis, and immune responses, they influence emotional processing, cognition, and motivation. Brain imaging studies indicate that these hormones affect the neuron circuits underlying mood regulation. However, the variability in the timing of PPD's onset and presenting symptoms, attributed to individual histories and external factors, complicates the identification of specific hormonal causes.²¹

Most studies showed no significant difference in reproductive hormone levels associated with depression between depressed and nondepressed postpartum women. Some studies in which women with PPD were treated with estradiol showed promise. Nevertheless, estradiol treatment has not been clinically proven to be successful. Some authors noted that fluctuations in reproductive hormone concentrations can provoke affective mood dysregulation in women with a genetic susceptibility across their lifespan, particularly during premenstrual, perinatal, and perimenopausal stages. For instance, a double-blinded pharmacofMRI study investigating the triggering of anhedonia and reward circuit activity in women with a history of PPD. Anhedonia increased during addback and withdrawal in women with a history of PPD but not in euthymic women. In the reward feedback, both hormone-sensitive women (n=10) and non-hormone-sensitive women (n=18) showed reduced activation in clusters located in the right putamen ($p < 0.031$, FWE-corrected) and left postcentral and supramarginal gyri ($p < 0.014$, FWE-corrected) during the withdrawal scans, compared to pre-treatment scans. While the results did not report variations in hormone sensitivity, they highlight the notable impact of reproductive hormones on reward-associated brain activity in women.²² Recent research has shown that the sudden decrease in the levels of allopregnanolone, a major progesterone metabolite, after childbirth, may play an essential role in triggering PPD. This can be attributed to the fact that allopregnanolone affects both anxiety and depression through the modulation of GABA receptors.²³

Gut microbiota, inflammation, and immune function

Recent studies indicate a link between gut microbiota dysbiosis and PPD, with inflammatory factors (IFs) also implicated in PPD's development. A study by Li et al to determine whether genetically predicted GM and IFs contribute to PPD and whether IFs mediate GM's effect on PPD by employing Mendelian randomization (MR) analyses. Findings revealed that class Alphaproteobacteria and certain families and genera within it demonstrated negative associations with PPD risk, with the IFs CCL5 and CCL3 correlating negatively with PPD risk but not mediating GM's impact. The study found no evidence of heterogeneity. However, this MR analysis provides genetic evidence for the roles of GM

and IFs in the pathogenesis of PPD, advancing the development of new preventive and therapeutic strategies.¹²

Given that the immune axis is regulated by estradiol, which fluctuates during the perinatal period, immune responses are implicated in PPD. Anti-inflammatories are elevated in pregnancy to suppress immunity and protect the fetus. However, the immune system rapidly becomes proinflammatory and remains so for several weeks following delivery. Studies reported a variation in genetic expression related to immune function. A recent genome-wide association study explored genetic markers associated with PPD through RNA sequencing of whole blood samples and reported that immune biomarkers (including genes that encode TNF-family receptors and A disintegrin and metalloproteinase with thrombospondin motifs (ADAMTs)) were significantly upregulated in women exhibiting depressive symptoms two months after delivery.²⁴ Moreover, elevated levels of matrix metalloproteinase-8 (MMP8) in late pregnancy were identified as a predictor of the onset of PPD. Moreover, disparities in MMPs and the ADAMTs protein subfamily were also reported. These findings may provide a valuable direction for future investigations into immune-driven modifications of the interstitial matrix and blood-brain barrier integrity.²⁵ Further research is required to validate the role of immune cytokines in PPD. In addition to immunity-related variations, other genetic factors were also reported to contribute to the development of PPD. In addition to immunity-related variations, other genetic factors were also reported to contribute to the development of PPD.

Genetics

Growing evidence suggests that a family history of mental illnesses contributes to the development of PPD.⁷ Val66Met polymorphism of brain-derived neurotrophic factor has been identified as a candidate in non-perinatal depression.²⁶ Genome-wide association studies of more than 1,200 women found genetic variations on chromosomes 1q21.3 q32.1 and 9p24.3 p22.3 and in Hemicentin-1 (HMCN1), which contains several estrogen-binding sites. All of which increase susceptibility to PPD.²⁷ Estrogen-induced epigenetic DNA methylation modifications have also been associated with PPD.²⁸ However, these findings require validation through large-scale studies. Given complexity of PPD and the stigma around mental illness, it presents a persistent challenge as it often goes undiagnosed. However, recent research shows several approaches to assessing risk of PPD onset before delivery.

Pre-delivery screening approaches

Psychological and psychometric screening

Brief self-report assessments remain the foundation of antenatal risk detection for PPD. The EPDS is the most

commonly applied assessment tool in perinatal settings for the detection of PPD, where studies have shown an association between elevated antenatal scores and later postpartum depressive symptoms. However, reported sensitivities and optimal cut points vary depending on clinical history (e.g., past depressive episodes), sociodemographic characteristics, and obstetric complications. Large-scale cohort studies reported improvements in the predictive outcomes of PDD when biological and demographic factors are taken into account alongside EPDS results.^{10,17,27} Other validated assessment tools include the PHQ-9 and GAD-7, which correlate with antenatal distress and can complement EPDS screening, particularly when anxiety is suspected.^{5,13}

Predictive biomarkers

Growing evidence identified candidate biomarkers that may enhance psychometric screening. Several studies have repeatedly reported alterations in cytokine expression profiles to be associated with perinatal depressive pathways.^{24,29} Transcriptomic and proteomic studies have reported metalloproteinase-related genes, including MMP8 and ADAMTS, to be upregulated in women who develop PPD.^{24,27} Furthermore, epigenetic modifications, such as differential methylation at HP1BP3 loci, have provided promising predictive potential in small cohorts but require wider-scale studies for validation.¹⁹ Recent studies have demonstrated that modulation of allopregnanolone and GABAergic neurotransmission, via the interplay of neurosteroid pathways and polymorphisms affecting neuroendocrine function, is also a potential biological contributor to PPD.^{22,23}

Integrated and algorithmic prediction

Multivariable prediction models that integrate psychosocial, obstetric, and biological factors have demonstrated enhanced screening power compared to single-domain approaches. Recent artificial intelligence and deep learning multivariable models trained on large clinical cohorts demonstrate good discrimination, with improved performance when provided with more comprehensive sets of risk factors (history of mental illness, social support metrics, obstetric complications, and, where available, biomarker data). Of great importance, published models emphasize the need for local recalibration before clinical employment, given that predictive population case-mix and measurement differences impact accuracy.^{15,30,31}

Given the heterogeneity in measurements and effect sizes across settings, best practice is to use validated psychometric screening (EPDS±PHQ-9/GAD-7) at multiple time points during pregnancy, combine screening scores with key clinical risk indicators (prior psychiatric history, severe obstetric events, low social support), and, where available, interpret biomarker results as supplementary, research-grade information rather than

standalone diagnostics.^{17,24,27} Integration into electronic health records and the adoption of reproducible decision rules facilitate timely referral pathways and enable targeted preventive care for those at a higher risk.^{15,30}

Targeted interventions

Several targeted antenatal measures are feasible, scalable, and effective in reducing subsequent depressive symptoms and disorder onset. These interventions fall into psychological therapies, stress-reduction programs, and social/peer support strategies.

Psychological interventions

CBT

Strong evidence from several large-scale randomized-trial studies reported CBT as the first-line intervention for preventing common perinatal mental disorders when delivered antenatally. Recent high-quality trials and meta-analyses report clinically significant reductions in depressive symptoms and lower incidence of postpartum depressive episodes among women. Trials deploying CBT delivered by trained non-specialist CBT therapists versus usual care; effect sizes vary with intervention length and whether it is delivered individually, in a group, or in a non-specialist format in low-resource settings have produced substantial preventive benefits, underscoring scalability.^{32,33} IPT is an evidence-based alternative, particularly appropriate for women whose primary risk derives from relationship stressors, role transition difficulties, or bereavement; IPT targets interpersonal functioning and has demonstrated symptom reduction in perinatal populations.³⁴ Guidelines and systematic reviews recommend CBT and IPT as first-line psychosocial preventive options for at-risk pregnant women.³⁵

Mindfulness

Mindfulness-based cognitive therapy, structured relaxation training, and prenatal yoga have demonstrated reductions in perceived stress, anxiety, and depressive symptom scores in randomized and nonrandomized studies.³⁶ Proposed mechanisms include modulation of hypothalamic-pituitary-adrenal axis reactivity, improvements in sleep and emotion regulation, and enhanced parasympathetic response.^{37,38} Meta-analytic studies indicate modest to moderate effects; heterogeneity across trials can be attributed to variations in program fidelity, participant adherence, and baseline risk factors.¹⁴

Social support

Interpersonal connectedness and peer support (childcare education, home visiting, and telephone peer counseling) are robust protective factors. Randomized trials report that structured peer-support and home-visiting programs reduce depressive symptoms and, in some high-risk

cohorts, lower the incidence of PPD.³² Outcomes are most consistent when support programs are sustained across late pregnancy and the early postpartum period.¹⁸

CONCLUSION

PPD is a preventable complication with pre-delivery screening, risk detection, and subsequent intervention. The employment of psychological, biological, and digital screening tools allows clinicians to identify vulnerable women early and implement timely preventive care. The best antenatal prevention plan employs a stepwise-care model, starting with universal screening (EPDS/PHQ-9), then psychological preventive measures including mindfulness, brief CBT, and peer support for moderate risk, and finally structured psychotherapy (CBT/IPT) or psychiatric consultation for high risk or individuals with previous depressive episodes. Integrating psychosocial strategies with initiatives targeting social determinants such as housing, domestic violence, and financial strain enhances preventive effectiveness.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Felimban O, Abdalla M, Alharbi F, Altuwairqi S, Alawami S, Alramel Z, et al. Preventing postpartum depression through pre-delivery screening and targeted interventions. *Int J Community Med Public Health* 2025;12:5827-32.