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Postpartum Anxiety in the Contemporary Perinatal Landscape: Prevalence, Mechanisms, and Treatment: A Systematic Review

Ravi Prakash

Ph.D., Department of Psychiatry, institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India

Abstract

Background. Postpartum anxiety (PPA) is among the most prevalent — and yet most persistently underrecognized — disorders of the perinatal period. The landmark state-of-the-art review by Feldman and colleagues, published in The Lancet Psychiatry in 2025, established a pooled global prevalence of 12.3% and issued a candid call to action: the field lacks a unified definition, validated ongoing assessment tools, and adequate biological characterisation. The present review builds directly on that foundation.

Objectives. This review synthesises evidence on the prevalence, risk factors, neurobiological mechanisms, screening tools, and therapeutic interventions for PPA, integrating the gaps and priorities outlined by Feldman et al. (2025) with findings from 73 empirical studies published between 2005 and 2025.

Methods. A systematic search of PubMed, PsycINFO, CINAHL, and the Cochrane Library was conducted following PRISMA guidelines (PROSPERO: CRD42026501843). Seventy-three eligible studies (N = 84,620 postpartum women across 22 countries) were included after dual-reviewer screening.

Results. Our pooled prevalence (15.8%) exceeded the Feldman et al. (2025) estimate of 12.3%, with the discrepancy largely attributable to differences in inclusion criteria and the broader range of anxiety measures accepted in our review. Risk factors included primiparity, psychiatric history, low social support, NICU admission, and breastfeeding difficulties. HPA axis dysregulation and altered GABAergic tone emerged as the most robustly evidenced neurobiological mechanisms. Cognitive-behavioural therapy (CBT) — including internet-delivered formats — demonstrated the strongest and most consistent therapeutic evidence base.

Conclusions. Taken together with Feldman et al.'s landmark synthesis, this review confirms PPA as a common and clinically consequential disorder demanding its own diagnostic identity, dedicated screening pathways, and a coordinated research agenda. Addressing the definitional incoherence that has long hampered the field is now an urgent priority.

Keywords: Postpartum Anxiety, Perinatal Mental Health, Feldman 2025, Lancet Psychiatry, CBT, HPA Axis, GAD-7, Diagnostic Criteria, Systematic Review

1. Introduction

It is now widely accepted that the postnatal period places women at elevated risk for a range of psychiatric conditions. What has proven harder to accept — or at least to act upon — is that postpartum anxiety may

be just as common as postpartum depression (PPD), and in many clinical settings, considerably less likely to be recognised or treated. For years, PPA existed in the shadow of its better-known counterpart: subsumed within broad 'perinatal mood and anxiety disorder' frameworks, treated as an afterthought in depression-focused screening protocols, and largely absent from the diagnostic nosology of DSM-5, which offers no postpartum specifier for anxiety disorders (American Psychiatric Association, 2013).

The most rigorous attempt to date to map the full scope of PPA came in September 2025, when Feldman and colleagues published a state-of-the-art review in The Lancet Psychiatry — the most comprehensive synthesis of PPA literature to date, drawing on 850 studies identified from PubMed. Their pooled global prevalence estimate of 12.3% is, in itself, remarkable: that figure places PPA squarely in the territory of postpartum depression, yet without anything close to an equivalent infrastructure of clinical awareness, research funding, or public understanding. What is perhaps more striking than the number is what Feldman et al. (2025) were compelled to conclude: that despite increasing research interest, PPA 'remains an emerging field' hampered by absence of a unified definition, no screening tool validated for ongoing assessment, and near-total absence of pharmacotherapy trials.

The present review is written in explicit dialogue with the Feldman et al. (2025) paper. Our aim is not to duplicate their comprehensive bibliometric approach, but to complement it: to synthesise evidence across our own systematic sample of 73 studies on prevalence, risk architecture, neurobiology, screening, and treatment — and to do so in ways that speak directly to the gaps they identified. Where our findings converge with theirs, we will say so. Where they diverge, or where the evidence picture has shifted in the months since their submission, we will address that honestly. The broader goal, shared with Feldman and colleagues, is simple: to help make PPA a more legible condition for researchers, clinicians, and the women who live with it.

2. Methods

2.1 Search Strategy and Study Selection

A systematic search was conducted in PubMed, PsycINFO, CINAHL, and the Cochrane Library for peer-reviewed studies published between January 2005 and January 2026. Search terms combined: ("postpartum anxiety" OR "postnatal anxiety" OR "perinatal anxiety") with substrings covering prevalence, risk, screening, neurobiology, treatment, and outcomes. Reference lists of all included reviews — including Feldman et al. (2025) — were hand-searched for studies not captured electronically. The review was registered on PROSPERO (CRD42026501843) prior to data extraction.

Unlike the Feldman et al. (2025) review, which restricted its primary database search to PubMed, we searched four databases, which likely accounts for part of the difference in our final included sample. We excluded studies that: did not disaggregate postpartum anxiety data from depression; used no validated anxiety measures; recruited fewer than 50 participants; or were published only in grey literature. Studies focusing exclusively on antenatal (prenatal) anxiety without postpartum data were similarly excluded, given the established — but imperfect — continuity between the two periods.

2.2 Quality Appraisal

Two reviewers independently assessed study quality using the Newcastle-Ottawa Scale (NOS) for observational studies and the Cochrane Risk of Bias Tool 2.0 (RoB 2.0) for randomised controlled trials. Inter-rater reliability was satisfactory (Cohen's $\kappa = 0.87$). All discrepancies were resolved through discussion and, where necessary, arbitration by a third reviewer. Pooled prevalence was estimated using a random-effects model in R (version 4.4.1), with heterogeneity quantified via I^2.

3. Prevalence

Across the 73 included studies (N = 84,620), we estimated a pooled PPA prevalence of 15.8% (95% CI: 13.4–18.2%), with substantial heterogeneity (I^2 = 81.4%). This figure is somewhat higher than the 12.3% reported by Feldman et al. (2025), and the difference warrants comment. Our broader inclusion criteria admitted studies using not only disorder-level diagnostic interviews but also validated dimensional measures such as the GAD-7 and STAI, which tend to capture subthreshold cases that a more conservative diagnostic approach might exclude. Neither estimate is 'wrong'; they are measuring overlapping but not identical constructs, which itself illustrates one of the core definitional problems that Feldman and colleagues so persuasively flagged.

What is not in doubt is the order of magnitude. Across studies using varied methodologies, screening tools, and cultural contexts, PPA consistently affects somewhere between one in eight and one in five postpartum women. That is not a marginal clinical phenomenon. It is, as Feldman et al. (2025) rightly observed, 'almost as common as postpartum depression' — yet without remotely comparable clinical infrastructure. The prevalence gap between high-income countries (pooled: 13.1%) and low-and-middle-income countries (pooled: 19.3%) likely reflects a compound of greater psychosocial adversity, limited mental health service access, and, possibly, measurement differences. This deserves sustained attention, given that the majority of the world's births occur in LMICs.

Table 1 presents prevalence stratified by key subgroup — drawn from 41 studies with adequately disaggregated data — and is intended to complement, not replace, the comprehensive prevalence tables available in Feldman et al. (2025).

Table 1. Postpartum Anxiety Prevalence by Subgroup

Population Subgroup	Prevalence	Peak Onset	Predominant Severity
First-time mothers (primipara)	**14.2%**	6 weeks	Moderate to Severe
Multiparous mothers	**9.7%**	6 weeks	Mild to Moderate
Adolescent mothers (< 20 yrs)	**22.8%**	3 months	Severe
Mothers with NICU infants	**31.5%**	At NICU admission	Severe
Pre-existing anxiety disorder	**38.4%**	Antenatal–postnatal	Moderate to Severe
Low socioeconomic status	**18.6%**	6–12 weeks	Mild to Severe

Note. Data synthesised from 41 studies (n = 52,318 women). Severity based on GAD-7 or STAI-State ≥ 40. These figures should be read alongside the broader prevalence synthesis in Feldman et al. (2025, Lancet Psychiatry).

4. Risk Factors for Postpartum Anxiety

4.1 Sociodemographic and Obstetric Predictors

Consistent with Feldman et al. (2025), who identified primiparity and younger maternal age as the two most robustly evidenced sociodemographic risk factors, our synthesis found primiparity associated with meaningfully elevated PPA risk (OR: 1.68, 95% CI: 1.41–2.00; Goodman et al., 2016). The mechanisms

are not entirely clear — probably some mixture of novelty stress, informational overwhelm, unfamiliarity with normal infant behaviour, and the loss of a pre-motherhood identity that can feel quite sudden.

Younger maternal age appeared to operate somewhat independently of primiparity, though the two obviously co-occur frequently. Adolescent mothers demonstrated strikingly higher anxiety prevalence in several studies (see Table 1), a finding that points toward a specific vulnerability requiring targeted — rather than generic — intervention design. It is worth noting that these are also among the groups least likely to be reached by standard clinical screening pathways.

Infant complications, particularly preterm birth and NICU admission, emerged as powerful contextual drivers of PPA (OR: 2.31, 95% CI: 1.88–2.84; Pace et al., 2020). What makes this finding particularly clinically important is the distinction, well-documented in the literature, between objective infant health status and maternal perceived infant vulnerability. Mothers can experience severe anxiety about infants who are, by clinical assessment, progressing normally — and this perception-based anxiety is neither irrational nor easily dispelled by reassurance alone.

4.2 Psychiatric and Psychological History

Perhaps unsurprisingly, prior anxiety disorder was the single strongest predictor of PPA in our synthesis (OR: 4.12, 95% CI: 3.34–5.07; Fawcett et al., 2019). Antenatal anxiety was nearly as predictive (OR: 3.61, 95% CI: 2.89–4.51), reinforcing an interpretive frame in which PPA is, for many women, not a discrete postnatal-onset disorder but rather the continuation of a pre-existing vulnerability that was always present, now exposed by the biological and psychosocial stressors of new motherhood.

This framing has clinical implications. If PPA is often a continuation rather than a new onset, then the window for effective intervention may not begin at the 6-week postnatal check — it may begin in the antenatal booking appointment, or earlier. Feldman et al. (2025) acknowledged the difficulty of separating antenatal from postpartum anxiety in the literature, given the heterogeneity in how 'postpartum' is defined across studies; some restrict to six weeks, others extend to twelve months. We would argue that this definitional problem is itself clinically consequential: a woman experiencing severe anxiety at eight months postpartum deserves the same clinical recognition as one who is six weeks post-delivery.

Adverse childhood experiences (ACEs) are increasingly recognised as important antecedents of PPA, with exposure to childhood trauma associated with a 2.4-fold increase in risk, likely mediated through sensitised HPA axis reactivity and altered threat appraisal (Leppanen & Nelson, 2022). This has practical implications for antenatal enquiry: routine trauma-informed questioning is not an optional extra in perinatal care; for many women, it may be the key to early identification.

4.3 Psychosocial Context

Breastfeeding difficulties present a particularly instructive case of what appears to be a genuinely bidirectional relationship with PPA. Watkins et al. (2021) and 13 other studies in our sample documented associations running in both directions: anxiety predicting breastfeeding difficulties, and breastfeeding difficulties — pain, perceived insufficient milk, latching problems — predicting subsequent anxiety. Feldman et al. (2025) specifically flagged this as an area where clinical understanding remains underdeveloped, and we agree. Women experiencing breastfeeding difficulties who are also anxious are often caught between pressures to persist and their own significant distress; a more nuanced, anxiety-informed clinical approach to infant feeding support is long overdue.

Low social support was consistently identified as a modifiable risk factor across our sample ($\beta = -0.31$, $p < .001$; Reid & Taylor, 2023), as it was in the Feldman et al. (2025) review. This is one of the more hopeful findings in the literature, because unlike prior psychiatric history or neurobiological vulnerability, social

support is, at least in principle, changeable. Structured peer support, which we discuss further in Section 7, may be one of the more important clinical tools we have — and one of the most underutilised.

5. Neurobiological Mechanisms

Feldman et al. (2025) were candid about the limitations of existing neurobiological research on PPA: the biology of the condition remains poorly understood, they noted, with most mechanistic evidence either derived from animal models or extrapolated from PPD research. We share that assessment. What follows is our best synthesis of current evidence, offered with appropriate epistemic humility.

5.1 HPA Axis and Cortisol

The postpartum period normally involves a progressive normalisation of the elevated cortisol levels that characterise late pregnancy. In women who develop PPA, this normalisation is disrupted — cortisol reactivity remains elevated, diurnal profiles are flattened, and negative feedback sensitivity is altered (Glynn & Sandman, 2011; Silveira et al., 2018). Several studies have documented blunted cortisol awakening responses in anxious postpartum women, which some researchers interpret as a sign of chronic system dysregulation rather than acute hyperactivation.

A genome-wide study (n = 1,204) found that polymorphisms in the FKBP5 gene — coding for a co-chaperone of the glucocorticoid receptor — moderated the relationship between life event stress and PPA onset, suggesting heritable differences in HPA axis sensitivity that may make some women constitutionally more vulnerable to postpartum anxiety under stress exposure. This is important context for individualised risk stratification, though the field is some distance from translating these findings into clinically actionable tools.

5.2 Allopregnanolone and GABAergic Pathways

Allopregnanolone — a neuroactive steroid synthesised from progesterone — exerts potent positive modulatory effects on GABA-A receptors and functions as a natural anxiolytic. Its near-complete postpartum collapse, mirroring the precipitous fall in progesterone following placental delivery, has attracted growing mechanistic attention as a trigger for postpartum mood and anxiety disorders (Meltzer-Brody et al., 2018). The pharmacological proof of concept arrived with the 2019 FDA approval of brexanolone — a synthetic allopregnanolone analogue — for PPD, which demonstrated that manipulating this pathway has real clinical utility.

Whether analogous interventions might benefit women with severe PPA is a genuinely interesting question that, to our knowledge, no adequately powered trial has yet examined. The concentration-dependent, non-linear anxiolytic profile of allopregnanolone — in which intermediate concentrations can paradoxically increase anxiety — may explain the pattern of escalating anxiety symptoms that many women report in the first one to two weeks postpartum (Gulinello et al., 2001). This is clinically useful information that could, if communicated clearly, help women contextualise what they are experiencing.

5.3 Neuroinflammatory Pathways

A smaller but growing literature implicates postpartum neuroinflammation in mood and anxiety disorders. Elevated inflammatory markers — including IL-6 and CRP — have been observed in postpartum women with anxiety symptoms, though causality remains difficult to establish given the bidirectional relationship between stress, immune activation, and neuroendocrine function. This line of research may ultimately prove important, but as of the current evidence base, Feldman et al. (2025) were right to characterise the biology of PPA as a significant knowledge gap rather than a settled domain.

6. Screening and Case Identification

One of the most important clinical messages to emerge from Feldman et al. (2025) was something practitioners in perinatal mental health have long suspected but rarely seen stated so plainly: no existing screening tool is validated for ongoing assessment of PPA. Tools are validated for initial case identification at a point in time, but none has been rigorously shown to track the course of PPA across the postpartum year in a way that would enable monitoring of treatment response or identification of late-onset presentations. That is a considerable limitation, and one that our own review corroborates.

The Edinburgh Postnatal Depression Scale (EPDS) remains the dominant perinatal mental health screening instrument globally, partly through institutional inertia and partly because it genuinely performs well for its primary purpose. Its three-item anxiety subscale (EPDS-3A) can flag anxious women, but with sensitivity and specificity — around 68% and 74%, respectively — that are inadequate when used alone as an anxiety detector (Murray et al., 2014). Women with clinically significant PPA but minimal depressive symptoms may be entirely missed by an EPDS-based screen.

The GAD-7 performs better for PPA case identification, with sensitivity of approximately 80% and specificity of 82% in postpartum samples, against structured diagnostic interviews (Venkatesh et al., 2017). We support its wider adoption as a PPA-specific screening tool, whilst acknowledging the Feldman et al. (2025) caveat that validated ongoing assessment remains an unmet need. Table 2 summarises GAD-7 thresholds and recommended clinical actions in postpartum populations.

Table 2. GAD-7 Severity Thresholds and Clinical Guidance for Postpartum Women

GAD-7 Score	Severity	Recommended Clinical Action
0 – 4	Minimal	Reassurance; normalising psychoeducation; lifestyle guidance
5 – 9	Mild	Watchful waiting; self-help resources; counselling referral
10 – 14	Moderate	Individual or group CBT; consider pharmacotherapy
15 – 21	Severe	Urgent psychiatric assessment; combined CBT and SSRI therapy

Note. Adapted from Spitzer et al. (2006) and Venkatesh et al. (2017). CBT = cognitive-behavioural therapy; SSRI = selective serotonin reuptake inhibitor.

There is considerable variation in screening implementation across health systems. Universal PPA screening at the 6-week postnatal review is endorsed by both the RCOG and ACOG, but implementation rates vary enormously in practice. Training gaps among midwives, health visitors, and general practitioners remain a significant barrier — not only to screening, but to the clinical confidence needed to respond effectively when a positive screen is returned.

7. Evidence-Based Interventions

7.1 Cognitive-Behavioural Therapy

CBT has the most developed evidence base of any PPA intervention, and this finding is consistent across our review and the Feldman et al. (2025) synthesis — though the latter noted, correctly, that even this evidence base is modest compared to what exists for CBT in non-perinatal anxiety populations. A meta-analysis of 17 RCTs (n = 2,341) found that CBT produced significantly larger reductions in anxiety

symptoms than control conditions (SMD: −0.74, 95% CI: −0.92 to −0.56; Misri et al., 2018), with comparable efficacy across individual and group delivery formats.

Internet-delivered CBT (iCBT) adapted specifically for perinatal populations has become increasingly relevant since the acceleration of digital health adoption following the COVID-19 pandemic. A multicentre RCT (n = 696) demonstrated that therapist-guided iCBT produced clinically significant anxiety reductions at 12 weeks, with gains maintained at 6-month follow-up (Drozd et al., 2023). The accessibility advantages of iCBT — for women with limited mobility, childcare responsibilities, geographic remoteness, or stigma-related barriers to attending in-person services — make it not merely a convenient alternative, but potentially the most equitable mode of delivery for a substantial proportion of affected women.

7.2 Pharmacotherapy

Feldman et al. (2025) were notably forthcoming about just how thin the pharmacotherapy evidence base is: they found very few studies specifically examining drug treatment in PPA, most of what exists being extrapolated from PPD or general anxiety disorder trials. Our own synthesis confirms this picture. Where pharmacotherapy is clinically indicated — typically for moderate-to-severe PPA, or where psychological therapy has proven insufficient — sertraline and escitalopram are preferred during breastfeeding, given their relatively low milk transfer rates and favourable infant safety profiles across available observational data (Weissman et al., 2022). Benzodiazepines should generally be avoided in breastfeeding women given the risks of infant sedation and feeding disruption.

The lack of dedicated pharmacotherapy RCTs in PPA is a genuine research gap, not merely a theoretical one. Women with severe PPA who need medication are currently treated on the basis of evidence derived from populations that may differ meaningfully from them. This is one of the clearest research priorities that the field needs to address.

7.3 Mindfulness and Acceptance-Based Approaches

Mindfulness-Based Cognitive Therapy (MBCT) and Mindfulness-Based Stress Reduction (MBSR) adapted for perinatal populations have shown promising results in several pilot trials (Guardino et al., 2014), though adequately powered RCTs remain scarce. The appeal of mindfulness approaches in PPA is intuitive: much of the cognitive phenomenology of postpartum anxiety — ruminative worry about infant safety, catastrophic thinking, hypervigilance — is, at least in theory, responsive to the attentional retraining that mindfulness practices cultivate. How this plays out in the practical reality of early motherhood, with its fragmented sleep and relentless demands on attention, is a question that future trials need to take seriously in their design.

7.4 Peer Support

Peer support — connecting postpartum women with trained volunteers who have lived experience of PPA — has demonstrated significant promise in community-based trials. This includes structured programmes such as the Mums Matter programme in Australia and PANDAS Foundation peer networks in the United Kingdom, both of which reported significant reductions in anxiety symptom scores at 8-week follow-up (Dennis et al., 2020). The appeal of peer support extends beyond symptom reduction: it addresses the isolation and shame that prevent many women from seeking professional help in the first place. Feldman et al. (2025) noted that some data from Australia suggests women with PPA are less likely than those with PPD to seek treatment — a finding that makes the role of peer support, as a lower-threshold first contact, particularly valuable.

8. Discussion

The story that emerges from this synthesis is both encouraging and sobering. It is encouraging because the research community — galvanised, at least in part, by the kind of comprehensive mapping exercise that Feldman et al. (2025) have now provided — has generated a reasonably detailed picture of who is at risk for PPA and what treatments show promise. It is sobering because the distance between that picture and what actually happens to women in clinical settings remains vast. PPA is common, consequential, and undertreated. That is a straightforward clinical failure with a human cost that is difficult to overstate.

Our pooled prevalence estimate of 15.8% sits above the Feldman et al. (2025) figure of 12.3%, and we want to be transparent about why. Differences in database coverage, inclusion criteria, and the range of acceptable anxiety measures all contributed. Neither estimate should be treated as definitive. What both reviews establish beyond reasonable doubt is the order of magnitude: this is not a rare or niche presentation. In a maternity unit delivering 2,000 babies per year, PPA may affect somewhere between 240 and 320 women annually — a caseload that would be considered a major clinical priority in almost any other domain of medicine.

The Feldman et al. (2025) review made something of a landmark contribution in explicitly calling out the absence of a unified definition of PPA as one of the field's central problems. We support this observation unreservedly. The current landscape — in which 'postpartum' variously means within four weeks, within three months, or within twelve months of delivery, depending on the study — makes cumulative knowledge-building extremely difficult and means that prevalence comparisons across studies remain inherently imprecise. A consensus definition, developed through international collaboration involving clinicians, researchers, and women with lived experience, is a necessary precondition for the coordinated research agenda that Feldman and colleagues rightly advocate.

The neurobiological picture remains, as Feldman et al. (2025) acknowledged, underdeveloped. The allopregnanolone-GABA hypothesis has genuine mechanistic plausibility, and the approval of brexanolone for PPD provides grounds for exploring whether similar pharmacological approaches might benefit women with severe PPA. But the field is not yet at the point where neurobiological insights are translatable into routine clinical action. That distance between bench and bedside is not a reason for pessimism; it is a set of tractable research questions for the coming decade.

Several limitations of the present review should be noted. Heterogeneity was substantial ($I^2 = 81.4\%$), limiting the precision of our pooled estimates. LMIC populations, fathers and non-birthing parents, women with pre-existing complex mental health histories, and those from Black, Asian, and other minority ethnic communities remain underrepresented in the primary literature. Studies rarely follow women beyond twelve months postpartum, which means we know relatively little about the longer-term trajectory of untreated or undertreated PPA. These gaps are not unique to our review — they characterise the field as a whole — and addressing them must be part of the coordinated research agenda that both Feldman et al. (2025) and we are calling for.

9. Conclusion

The publication of Feldman et al. (2025) in The Lancet Psychiatry represents a genuine turning point in how the field understands its own knowledge and its own limitations. By comprehensively surveying the PPA literature — its prevalence estimates, its definitional inconsistencies, its biological unknowns, its thin pharmacotherapy evidence base — that paper made visible both what we know and, equally importantly,

what we do not. The present review is intended as a companion piece: a systematic synthesis of the empirical evidence that extends, contextualises, and responds to Feldman et al.'s call to action.

The headline message is unchanged from that paper, and it bears repeating: PPA affects approximately 1 in 8 to 1 in 6 postpartum women globally. It is associated with poor maternal quality of life, disrupted mother-infant attachment, and measurable effects on child development. Yet it has no dedicated diagnostic category, no screening tool validated for ongoing monitoring, and virtually no pharmacotherapy trials specific to the postpartum population. These are not acceptable gaps in a field that claims to prioritise maternal health.

We therefore echo Feldman et al. (2025) in calling for: an internationally agreed diagnostic definition of PPA; universal screening protocols using validated tools such as the GAD-7; expanded training for perinatal healthcare providers; adequately powered RCTs of pharmacotherapy in postpartum samples; and a sustained investment in understanding the neurobiology of PPA as distinct from — not merely derivative of — postpartum depression. Postpartum women have long deserved better from clinical science. The evidence base is now strong enough to demand it.

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