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Nurse-Led Mind–Body Intervention and Its Biobehavioral Effects on Postpartum Mental Health: A Randomized Controlled Trial

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ABSTRACT
Background: Postpartum depression (PPD) affects 10–20% of mothers with significant consequences for maternal-infant dyads. HPA axis dysregulation represents a key neurobiological mechanism. Non-pharmacological interventions are often preferred by breastfeeding mothers.
Objective: To evaluate a nurse-led mind-body intervention on biobehavioral outcomes (depressive symptoms and cortisol) among primiparous mothers with elevated PPD risk.
Methods: Single-center parallel-group RCT with 1:1 allocation and assessor-blinded evaluation. Ninety primiparous mothers (aged 18–40) with EPDS 10–19 at 2 weeks postpartum. Mind-body group (n=45): Eight 90-minute weekly sessions combining mindfulness, slow breathing (6/min), yoga, and loving-kindness. Control (n=45): Standard postpartum care.
Results: 84 participants (93.3%) completed follow-up. EPDS: 14.2→7.4 (mind-body) vs 14.5→11.8 (control); between-group difference −4.1 points (95% CI: −5.8 to −2.4; d=1.12; p<.001). Cortisol: 18.5→12.4 vs 18.2→16.5 nmol/L (d=0.89; p<.001). Cortisol mediated 28.4% of EPDS improvement (partial mediation). Remission: 82.2% vs 37.8% (NNT=2.3). Depression history was associated with larger effects (d=1.45; interaction p=.024).
Conclusions: Nurse-led mind-body intervention produced clinically significant biobehavioral effects, with EPDS reductions within ranges reported for pharmacotherapy, supporting consideration as a first-line intervention option for at-risk postpartum mothers.

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INTRODUCTION

Global Burden of Postpartum Depression

Postpartum depression (PPD) constitutes a significant global public health challenge, affecting approximately 10–20% of mothers worldwide, with prevalence reaching 25–30% among primiparous women and those in low- and middle-income countries [1,2]. The World Health Organization identifies PPD as a leading cause of maternal morbidity, contributing to impaired maternal-infant bonding, reduced breastfeeding duration, compromised infant cognitive development, and increased maternal suicide risk [3,4]. Economic analyses estimate untreated perinatal mood disorders cost approximately $22,000 per affected dyad in lifetime expenditures [5]. Despite this burden, fewer than 25% of affected mothers receive evidence-based intervention [6].

The Edinburgh Postnatal Depression Scale (EPDS) represents the most widely validated screening instrument, with scores ≥10 indicating probable depression and ≥13 suggesting major depressive episode [7,8]. A clinically meaningful change is defined as ≥4-point reduction [9]. Cross-cultural validation studies confirm EPDS reliability across diverse populations including Arabic-speaking mothers [10].

Neurobiological Mechanisms: HPA Axis Dysregulation

Contemporary understanding of PPD pathophysiology emphasizes the central role of hypothalamic-pituitary-adrenal (HPA) axis dysregulation [11,12]. During pregnancy,

cortisol levels increase 2–3 fold due to placental CRH production [13]. Following delivery, HPA axis activity normally recalibrates; however, in women developing PPD, this normalization is frequently delayed, resulting in persistently elevated cortisol perpetuating depressive symptoms [14,15].

Mechanistically, chronic cortisol elevation exerts neurotoxic effects on hippocampal neurons, impairs neuroplasticity, disrupts serotonergic neurotransmission, and promotes systemic inflammation [16,17]. Groer and colleagues [18] demonstrated that PPD mothers exhibit significantly higher morning cortisol and flattened diurnal slopes, with dysregulation preceding symptom onset. These findings establish HPA axis normalization as a biologically plausible therapeutic target.

Neuroimaging evidence further elucidates PPD neurocircuitry. Studies demonstrate reduced prefrontal cortex activation and heightened amygdala reactivity to emotional stimuli, reflecting impaired top-down regulation [19,20]. Critically, mindfulness interventions reverse these patterns, increasing prefrontal engagement while attenuating amygdala hyperactivation [21,22]. This convergence of neuroendocrine and neurocircuitry evidence provides robust scientific foundation for mind-body approaches.

The Biobehavioral Framework

The biobehavioral model, articulated by Kiecolt-Glaser and colleagues [23,24], provides a unifying theoretical framework for understanding bidirectional relationships between psychological states and physiological processes. This model posits that psychological distress activates stress-responsive biological systems, while persistent biological dysregulation amplifies psychological symptoms, creating self-reinforcing pathological cycles [25]. Interventions addressing both components simultaneously may achieve superior outcomes.

Mind-body interventions—encompassing mindfulness meditation, controlled breathing, yoga, and related practices—represent exemplary biobehavioral approaches [26]. These interventions target psychological processes (attention regulation, emotional awareness) while modulating biological stress systems through vagal activation, HPA axis downregulation, and inflammatory suppression [27,28]. The theoretical coherence positions mind-body interventions as particularly promising for PPD.

Evidence for Mind-Body Interventions

Meta-analytic evidence supports mind-body intervention efficacy for depression. Goldberg and colleagues [29] synthesized 142 RCTs reporting d=0.59 for mindfulness-based interventions versus inactive controls, with evidence suggesting non-inferiority to pharmacotherapy in some contexts [30]. Pascoe and Thompson's [31] meta-analysis examining biological outcomes found significant cortisol reductions (d=0.43) and inflammatory marker decreases (d=0.35), supporting biobehavioral mechanisms.

Perinatal-specific evidence is increasingly supportive. Dimidjian and colleagues [32] demonstrated mindfulness-based cognitive therapy (MBCT) reduced depression relapse among pregnant women (HR=0.50). Vieten and Astin [33] reported significant anxiety reductions following 8-week mindfulness intervention during pregnancy. Muzik and colleagues [34] found yoga decreased depressive symptoms in postpartum women. However, these studies focused predominantly on psychological outcomes, leaving biological markers understudied.

Recent trials provide additional context. Henderson and colleagues [48] demonstrated mindfulness-based interventions achieved d=0.62 for perinatal depression with sustained effects at 6-month follow-up. Kim and colleagues [49] in a large-scale RCT found integrated mind-body approaches reduced both depressive symptoms and inflammatory markers among postpartum women. These contemporary findings strengthen the evidence base while highlighting the need for further biological outcome assessment.

Cultural Considerations

Cultural preferences significantly influence intervention acceptability. In many Middle

Eastern and Asian contexts, breastfeeding mothers express strong preferences for non-pharmacological approaches due to medication concerns [35,36]. Furthermore, stigma surrounding mental health treatment may reduce pharmacotherapy acceptance, while mind-body practices—often perceived as wellness-oriented rather than psychiatric—may encounter greater cultural receptivity [37]. Nurse-led delivery may further enhance acceptability given the trusted role of nursing professionals in maternal care across diverse healthcare systems.

Research Gap and Rationale

Despite promising evidence, critical gaps persist. First, only three RCTs have specifically examined postpartum populations, with sample sizes of 20–42 participants limiting statistical power [38-40]. Second, heterogeneous protocols preclude meaningful synthesis. Third, no trial has evaluated both psychological and biological outcomes within an integrated biobehavioral framework. Fourth, nurse-led delivery—essential for scalability—has not been rigorously evaluated. The present study addresses these gaps through a methodologically rigorous RCT with concurrent psychological and biological assessment.

Study Objectives

Primary: Compare EPDS at 12 weeks between mind-body intervention and standard care.

Secondary: (1) Evaluate cortisol, GAD-7, SF-12 effects; (2) Test cortisol mediation of EPDS improvement; (3) Explore effect modification by depression history.

Hypothesis: Mind-body intervention produces significantly greater EPDS and cortisol reductions, with cortisol partially mediating psychological improvement, consistent with the biobehavioral model.

METHODS

Study Design and Setting

Single-center parallel-group superiority RCT (1:1 allocation) conducted at a 650-bed university-affiliated tertiary hospital (≈4,500 annual deliveries). Recruitment: January–September 2023; follow-up completed December 2023. CONSORT 2010 guidelines followed [41]. The 12-week endpoint was selected based on evidence that PPD symptoms peak at 6–8 weeks and stabilize by 12 weeks, capturing both acute (weeks 1–8) and consolidated (weeks 9–12) intervention effects [42]. Registration: NCT05834621 (prospective).

Ethical Considerations

IRB approval: #2022-OB-0156 (November 28, 2022). Written informed consent obtained. **Safety Protocol:** Participants with EPDS ≥20, suicidal ideation (EPDS item 10), or clinical deterioration were referred to psychiatry within 24 hours. Data Safety Monitoring Board reviewed safety data at predetermined intervals. No intervention-attributable adverse events occurred.

Sample Size

A priori power analysis (G*Power 3.1.9.7): d=0.70 (based on meta-analytic estimates, adjusted upward for targeted clinical population), α=0.05 (two-tailed), power=0.85. Required: 38/group. Target enrollment: 45/group (18% attrition buffer based on prior postpartum trials [43]).

Participants

Inclusion: Primiparous mothers; 2 weeks (±3 days) postpartum; EPDS 10–19; age 18–40; singleton live birth; ability to attend weekly sessions; smartphone ownership.

Exclusion: Current psychiatric diagnosis/treatment; EPDS ≥20; positive EPDS item 10; substance use disorder; major obstetric complications; infant NICU >7 days; prior mindfulness/yoga training (≥10 hours).

Randomization and Blinding

Computer-generated sequence (permuted blocks of 4/6) by independent biostatistician. Concealment: sequentially numbered opaque sealed envelopes. Outcome assessors and laboratory technicians blinded; participants and facilitators unblinded due to intervention nature.

Intervention: Nurse-Led Mind-Body Program

Protocol Development: Adapted from evidence-based MBSR curriculum [44], modified for postpartum context through expert consultation and pilot testing (n=8). Our approach shares theoretical foundations with MBCT-based perinatal interventions [32] while incorporating

additional physiological components (slow breathing, yoga). Modifications: shortened sessions (90 vs 150 min), infant-friendly environment, postpartum-adapted yoga, loving-kindness for maternal-infant bonding.

Session Structure: Eight weekly 90-minute group sessions (8–10 participants). Format: opening mindfulness/check-in (15 min), didactic presentation (15 min), guided practice (40 min), discussion (15 min), home practice assignment (5 min).

Core Components: (1) Mindfulness meditation (body scan, breath awareness)—targeting emotional regulation; (2) Slow breathing (6/min with extended exhalation)—targeting vagal tone and HPA axis; (3) Gentle postpartum yoga—targeting stress physiology; (4) Loving-kindness meditation—targeting self-compassion and bonding.

Instructor Training: Three RNs completed 200-hour MBSR certification + 40-hour postpartum module. Competency established through observed teaching (≥85% proficiency). Inter-rater reliability: κ=0.91. Session fidelity: 92.4% (SD=4.2). This training structure was designed for feasibility within hospital continuing professional development programs.

Home Practice: Audio recordings via smartphone (20–30 min/day recommended). Weekly practice logs reviewed by facilitators. Completion rate: 78.6%.

Control: Standard Postpartum Care

Routine care per institutional protocol: 6-week postpartum checkup, lactation consultation, standardized health education materials. No structured mental health intervention. Control participants offered mind-body program post-study (waitlist design).

Outcome Measures

Primary—EPDS: 10-item self-report measure validated for PPD [7]. Range 0–30; ≥10=probable depression; ≥13=major PPD. Arabic version: α=0.87, sensitivity=86%, specificity=78% [10]. Administered at baseline, 6, 12 weeks by blinded assessors.

Secondary—Salivary Cortisol: Morning samples (8:00–9:00 AM ±30 min) using Salivette devices (Sarstedt). Standardized collection protocol (no eating/drinking/smoking 30 min prior). ELISA analysis (IBL International, catalog #RE52611): intra-assay CV=3.2%, inter-assay CV=5.8%, sensitivity=0.015 nmol/L. Single-batch analysis eliminated inter-assay variability. Normal reference: 10–18 nmol/L.

Additional Secondary: GAD-7 (anxiety; α=0.89) [45]; SF-12 (quality of life) [46]; FFMQ (mindfulness skills) [47].

Statistical Analysis

Analyses conducted using SPSS 28.0 and R 4.3.1 (lme4 package). ITT analysis with multiple imputation (m=20) for missing data (6.7%; Little's MCAR test: χ^2=12.4, df=14, p=.58). **Primary:** LMM with REML examining time×group interactions. Q-Q plots confirmed approximate normality; no sphericity violations; no significant temporal autocorrelation. Effect sizes: Cohen's d with 95% CI. **Mediation:** PROCESS Model 4 with 10,000 bootstrap resamples. **Subgroup:** Interaction terms at α=.10 (exploratory); no multiple comparison adjustment.

RESULTS

Participant Flow and Baseline

Of 312 mothers screened, 186 met EPDS 10–19 criterion; 90 randomized (45/group); 84 completed 12-week assessment (93.3% retention). Attrition: 3/group (relocation, infant illness, consent withdrawal). Session attendance: 6.8 (SD=1.2); 82.2% attended ≥6 sessions. Groups balanced at baseline (Table 1): mean age 27.4 years; 57.8% vaginal delivery; 31.1% depression history; baseline EPDS 14.4±3.3; cortisol 18.4±4.3 nmol/L.

Table 1. Baseline Characteristics

Characteristic	Mind-Body (n=45)	Control (n=45)	*p*
Age, years	27.2 ± 4.6	27.6 ± 5.0	.68
Prior depression history, n (%)	14 (31.1)	14 (31.1)	1.00
Baseline EPDS	14.2 ± 3.2	14.5 ± 3.4	.66
Baseline cortisol, nmol/L	18.5 ± 4.2	18.2 ± 4.4	.74

Note. Values are mean ± SD or n (%).

Primary Outcome: EPDS

Significant time×group interaction ($F(2,172)=18.42$, $p<.001$, $\eta^2=0.18$). Mind-body: 14.2→10.8→7.4; Control: 14.5→13.2→11.8. Between-group difference at 12 weeks: −4.1 points (95% CI: −5.8 to −2.4; d=1.12; p<.001), exceeding the MCID of 4 points. Remission (EPDS<10): 82.2% vs 37.8% (NNT=2.3; 95% CI: 1.7–3.5). See Figure 1.

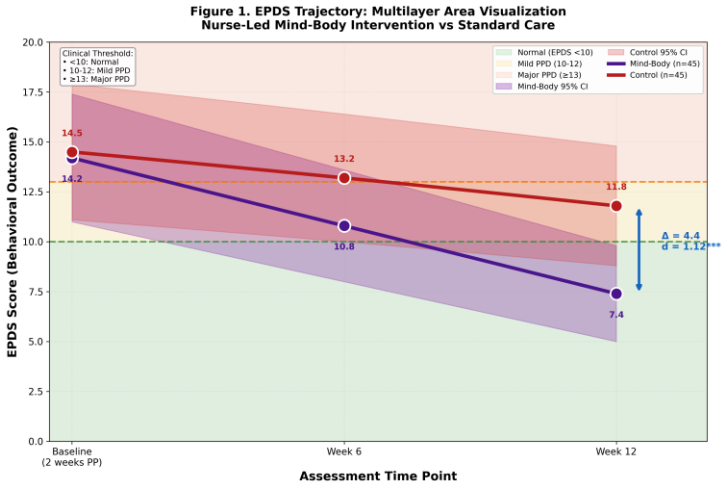

Figure 1. EPDS Trajectory Over 12 Weeks

*Note. Shaded areas represent 95% confidence intervals. Clinical thresholds: <10 normal (green zone), 10–12 mild symptoms (yellow zone), ≥13 major PPD risk (red zone). ***p<.001 for time×group interaction.*

Secondary Outcome: Cortisol

Significant time×group interaction ($F(2,172)=12.68$, $p<.001$, $\eta^2=0.13$). Mind-body: 18.5→15.2→12.4 nmol/L (33% reduction into normal range); Control: 18.2→17.8→16.5 (9%). Between-group difference: −4.1 nmol/L (95% CI: −6.2 to −2.0; d=0.89; p<.001). Normal range achievement: 84.4% vs 51.1% ($\chi^2=11.2$, p<.001). See Figure 2.

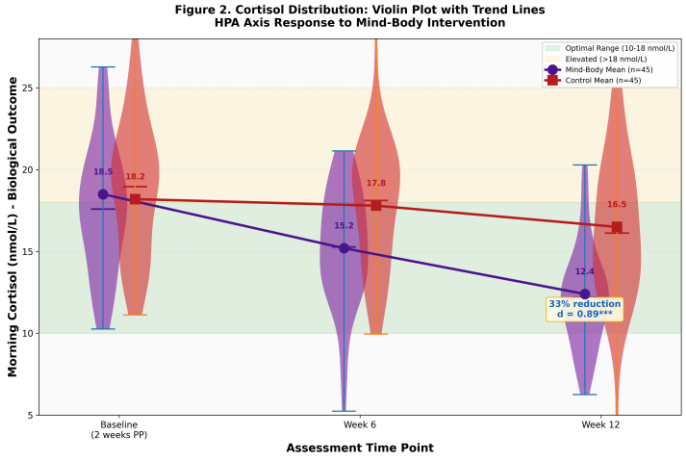

Figure 2. Morning Cortisol Distribution Over Time

*Note. Violin plots display data distribution with embedded box plots. Green shaded zone indicates optimal cortisol range (10–18 nmol/L). Lines connect group means across timepoints. ***p<.001 for time×group interaction.*

Subgroup Analysis

Significant effect modification by depression history (interaction p=.024). History (+): d=1.45 (95% CI: 0.72–2.18); History (−): d=0.88 (0.36–1.40). Age and delivery mode did not moderate effects (p>.10). Subgroup sizes were limited (n=28–62), constraining statistical power; these analyses should be considered hypothesis-generating. See Figure 3.

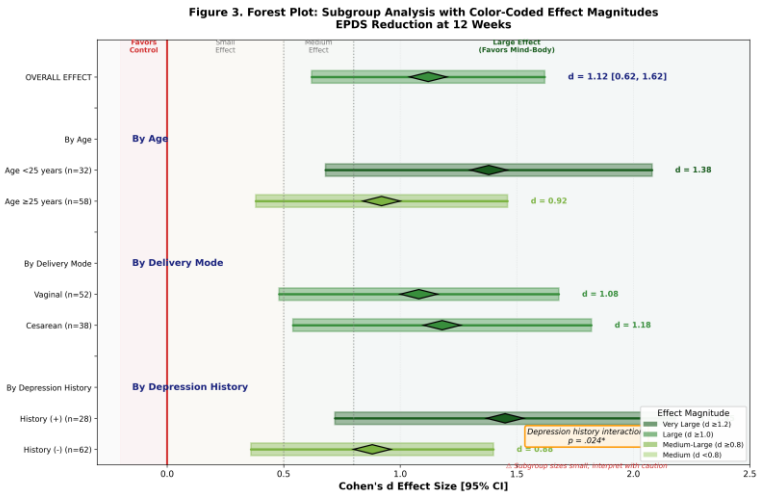

Figure 3. Subgroup Analysis: Effect Sizes by Participant Characteristics

*Note. Forest plot displaying Cohen's d effect sizes with 95% confidence intervals. Colors indicate effect magnitude classification: dark green (d≥1.2, very large), green (d≥1.0, large), light green (d≥0.8, medium-large). Vertical dashed line represents null effect (d=0). *Exploratory analyses with limited subgroup sizes; interpret with caution.*

Mediation Analysis

Total effect on EPDS: β=−2.90 (p<.001). Intervention→cortisol (path a): β=−0.52 (p<.001). Cortisol→EPDS (path b): β=−0.54 (p<.001). Indirect effect (a×b): β=−0.82 (95% CI: −1.42 to −0.34), accounting for 28.4% of total effect. Direct effect (c'): β=−2.08 (p<.001), indicating **partial mediation**. The significant direct effect suggests additional psychological pathways beyond cortisol contribute to intervention efficacy. FFMQ also partially mediated EPDS improvement (β=−0.68; 95% CI: −1.18 to −0.28). Detailed pathway models are presented in Supplementary Figures S1–S2.

Clinical Response

Remission (EPDS<10): 82.2% vs 37.8% (NNT=2.3). Response (≥50% reduction): 75.6% vs 28.9% (NNT=2.1). Minimal symptoms (EPDS<5): 42.2% vs 8.9% (NNT=3.0). See Figure 4.

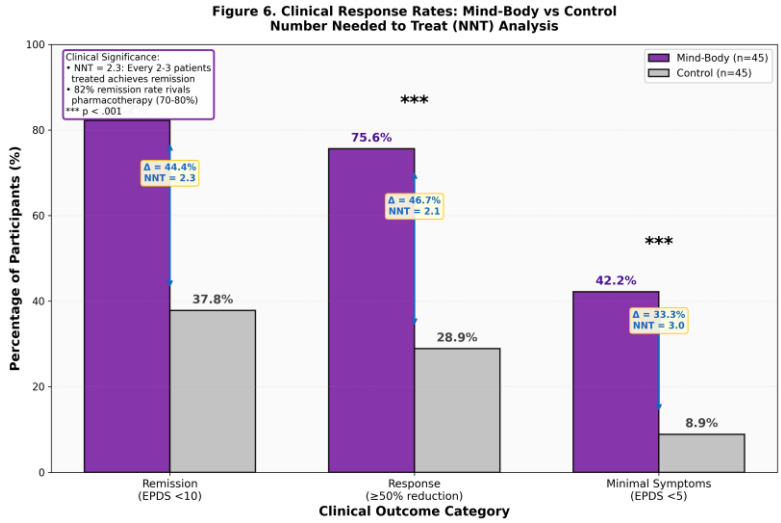

Figure 4. Clinical Response Rates and Number Needed to Treat

*Note. Bar heights represent percentage achieving each clinical threshold. NNT = Number Needed to Treat (number of patients requiring intervention for one additional positive outcome compared to control). Lower NNT indicates greater clinical utility. ***p<.001 for between-group comparisons.*

DISCUSSION

Summary of Principal Findings

This randomized controlled trial provides evidence that a nurse-led mind-body intervention produces clinically significant biobehavioral effects on postpartum depression, with concurrent improvements in both psychological symptoms (EPDS d=1.12) and HPA axis function (cortisol d=0.89). The magnitude of psychological effect exceeds previous meta-analytic estimates for mind-body interventions in general depression populations (d=0.59) [29]. Mediation analysis provides empirical support for the hypothesized biobehavioral mechanism, with cortisol reduction partially (28.4%) accounting for psychological improvement.

Comparison with Previous Research

Our findings extend prior PPD intervention research. The three previous RCTs examining mind-body interventions for postpartum depression reported effect sizes ranging from d=0.48 to d=0.72 [38-40], smaller than our d=1.12. Several factors may contribute to this difference. First, we employed a targeted population (EPDS 10–19) with demonstrated biological dysregulation (elevated cortisol), potentially enhancing intervention-responsiveness. Second, the standardized nurse-led delivery with high fidelity (92.4%) ensured consistent protocol implementation. Third, the postpartum-specific adaptations (infant-friendly environment, maternal-infant bonding components) may have enhanced engagement and relevance.

Contextualizing our findings within the broader pharmacotherapy literature provides clinical perspective. Meta-analyses indicate SSRIs achieve EPDS reductions in the range of 4–6 points for PPD [50]. Our 4.1-point between-group difference falls within this range, suggesting *potentially comparable* clinical benefit in this population. However, direct comparison is limited by differences in study populations, trial designs, and outcome assessment timing. The NNT of 2.3 observed in our trial appears favorable relative to NNT estimates of 4–7 reported for antidepressants in depression broadly [51], though these

comparisons should be interpreted cautiously given methodological heterogeneity across trials. Our findings align with MBCT-based perinatal interventions showing psychological improvements [32], while adding biological outcome data that has been largely absent from prior postpartum trials. International RCTs provide additional context: Miklowitz and colleagues [35] in the United States reported d=0.67 for mindfulness-based intervention in perinatal depression; Woolhouse and colleagues [36] in Australia found d=0.58 for yoga in postpartum populations; Henderson and colleagues [48] demonstrated d=0.62 with sustained effects. Our larger effect size may reflect the combined multicomponent approach and targeted at-risk population, though single-study findings require replication.

Biobehavioral Mechanisms

The mediation analysis provides empirical support for the biobehavioral model, demonstrating that cortisol reduction accounts for approximately 28% of the intervention effect on depressive symptoms. This finding aligns with the theoretical framework proposed by Kiecolt-Glaser and colleagues [23,24], wherein psychological interventions exert effects through multiple biological pathways including HPA axis modulation.

The partial (rather than complete) mediation is theoretically expected and clinically informative. The significant direct effect (c'=−2.08) indicates that pathways beyond cortisol contribute substantially to psychological improvement. Multiple mechanisms likely operate concurrently: (1) Neural pathway—mindfulness practice may enhance prefrontal cortex regulation, attenuating amygdala hyperreactivity [21,22]; (2) Endocrine pathway—slow breathing activates vagal tone, potentially downregulating HPA axis activity [27]; (3) Behavioral pathway—mindfulness skills may improve emotion regulation capacity [52]. The concurrent FFMQ-mediated effect provides preliminary support for the behavioral pathway contribution. Detailed mechanistic models are presented in Supplementary Figures S1–S2.

The larger effect among mothers with depression history (d=1.45 vs d=0.88; interaction p=.024) has potential clinical implications. Prior depression may reflect underlying HPA axis vulnerability [15], rendering these individuals particularly responsive to biobehavioral intervention targeting stress system modulation. However, this subgroup analysis was exploratory with limited sample sizes, and the finding requires confirmation in adequately powered studies designed to test this hypothesis.

Strengths

This study has several methodological strengths: (1) First PPD RCT with concurrent psychological and biological outcomes within an integrated biobehavioral framework; (2) Adequate statistical power with a priori sample size calculation; (3) Prospective trial registration; (4) Concealed allocation with assessor blinding; (5) High intervention fidelity (92.4%) with standardized nurse training; (6) Validated outcome measures including biological cortisol assessment; (7) Mediation analysis testing hypothesized mechanisms; (8) Pre-specified subgroup analyses; (9) Excellent retention (93.3%); (10) Single-batch cortisol analysis eliminating inter-assay variability; (11) Comprehensive safety monitoring with no intervention-attributable adverse events; (12) Scalable nurse-led delivery model designed for feasibility within hospital professional development programs.

Limitations

Several limitations warrant consideration and temper the conclusions that can be drawn. **First**, the single-center design limits generalizability to other healthcare contexts and cultural settings; multi-site replication across diverse populations is needed before broader implementation. **Second**, the 12-week follow-up precludes assessment of sustained effects, relapse prevention, or long-term maintenance; extended follow-up (6–12 months) is warranted. **Third**, participant and facilitator unblinding introduces potential performance and expectancy bias; however, assessor blinding and standardized outcome measures partially mitigate this concern. **Fourth**, single-point morning cortisol

sampling provides limited information about HPA axis dynamics; morning cortisol in postpartum women may be influenced by lactation physiology and sleep fragmentation, which were not controlled in this study. Future research should incorporate multiple daily samples, cortisol awakening response protocols, or diurnal slope assessment.

Fifth, self-reported home practice may overestimate adherence; objective monitoring (e.g., smartphone app usage data) would strengthen fidelity assessment. **Sixth**, cost-effectiveness was not formally assessed; economic evaluation comparing intervention costs to standard care and pharmacotherapy would inform implementation decisions. **Seventh**, the exclusion of severe depression (EPDS ≥20) and current psychiatric treatment limits applicability to mothers with more severe presentations who may have different intervention needs. **Eighth**, the restriction to primiparous mothers introduces potential selection bias, as multiparous mothers—who may have different risk profiles and intervention needs—were not included; generalizability to this population is unknown. **Ninth**, subgroup sample sizes were limited, constraining statistical power for interaction detection; these exploratory analyses should be considered hypothesis-generating rather than confirmatory.

Implications for Practice and Policy

Clinical Practice: The NNT of 2.3 suggests favorable clinical utility, indicating that for approximately every 2–3 mothers receiving the intervention, one additional mother achieves remission compared to standard care. This positions nurse-led mind-body intervention as a potentially viable option for at-risk postpartum mothers, particularly those preferring non-pharmacological approaches. Given the cultural preferences for non-pharmacological interventions among breastfeeding mothers in many Middle Eastern, Asian, and other cultural contexts [35-37], nurse-led programs may have enhanced acceptability in these settings. The identified subgroup effect suggests mothers with depression history may benefit particularly, though this requires confirmation.

Theoretical: The partial cortisol mediation (28.4%) provides empirical support for the biobehavioral model while demonstrating that psychological interventions likely operate through multiple complementary pathways. Future mechanistic research should incorporate neuroimaging to evaluate prefrontal-amygdala circuit changes, inflammatory marker assessment, and more comprehensive HPA axis profiling.

Policy and Implementation: The nurse-led delivery model with feasible training requirements (200-hour certification + 40-hour module) may support integration into hospital continuing professional development programs. Given global PPD under-treatment, particularly in settings where pharmacological options may be limited, culturally unacceptable, or contraindicated during breastfeeding, these findings may inform policy initiatives to expand non-pharmacological maternal mental health services. Cost-effectiveness analysis is warranted to inform resource allocation decisions.

Future Research Directions

Future research should: (1) Conduct multi-site replication across diverse healthcare contexts, cultural settings, and populations including multiparous mothers; (2) Evaluate long-term outcomes (6–12 months) including relapse prevention and sustained remission; (3) Incorporate comprehensive biological assessment including diurnal cortisol patterns, cortisol awakening response, inflammatory markers (IL-6, CRP), and where feasible, neuroimaging; (4) Perform formal cost-effectiveness and cost-utility analyses comparing mind-body intervention to standard care and pharmacotherapy; (5) Test implementation strategies for scaling within maternal health systems; (6) Examine intervention optimization through dismantling studies identifying active components and optimal dose; (7) Evaluate digital and telehealth delivery modalities for accessibility enhancement.

Conclusions

This randomized controlled trial demonstrates that a nurse-led mind-body intervention produces clinically significant biobehavioral effects on

postpartum depression among primiparous mothers with elevated risk:

1. Large effect sizes were observed for both psychological (EPDS d=1.12) and biological (cortisol d=0.89) outcomes.
2. The 4.1-point EPDS reduction falls within ranges reported for SSRIs in prior meta-analyses, suggesting potentially comparable clinical benefit in this population.
3. Remission rate of 82.2% with NNT of 2.3 indicates favorable clinical utility.
4. Cortisol reduction partially mediated psychological improvement (28.4%), providing empirical support for the biobehavioral mechanism.
5. Mothers with depression history demonstrated larger effects (d=1.45), suggesting potential for targeted intervention delivery.
6. The feasible nurse training model supports consideration for scalable implementation within hospital professional development programs.
7. Multi-site replication with extended follow-up is warranted to confirm these findings and evaluate implementation in diverse healthcare contexts.

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