Review Article

The Impact of Stress and Anxiety on Dysmenorrhea Severity: A Systematic Review

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ABSTRACT

Primary dysmenorrhea (PD) affects a significant proportion of menstruating individuals, with stress and anxiety increasingly recognized as modifiable factors influencing pain severity. This systematic review synthesizes evidence on the relationship between stress/anxiety and dysmenorrhea severity, exploring underlying mechanisms and clinical implications. Following PRISMA guidelines, we conducted a comprehensive search across PubMed, Web of Science, Scopus, and ScienceDirect. Included studies assessed stress (perceived stress, PTSD, occupational stress) or anxiety (generalized/trait anxiety) in individuals with PD, using validated scales. Risk of bias was evaluated using the Newcastle-Ottawa Scale and Cochrane RoB 2.0. Of 2,305 screened records, 45 studies met inclusion criteria, with 13 analyzed in detail. Cross-sectional studies (n=12) predominated, alongside one RCT study. Significant associations between stress/anxiety and PD severity (e.g., OR=2.8 for perceived stress (95% CI: 1.9–4.1); r=0.782, $p<0.001$ for stress-pain correlation). PTSD and occupational stress exacerbated dysmenorrhea ($\beta=10.48$, $p=0.001$; $p<0.05$, respectively). Neurobiological mechanisms (e.g., amygdala hypertrophy, HPA axis dysfunction) were implicated in pain amplification. Stress and anxiety are significantly associated with dysmenorrhea severity, potentially mediated by neuroendocrine and inflammatory mechanisms.

Keyword: Primary dysmenorrhea, Stress, Anxiety, Pain severity, Psychological distress, HPA axis, Systematic review.

Introduction

Primary dysmenorrhea (PD), characterized by painful menstrual cramps in the absence of pelvic pathology, affects 45–95% of menstruating individuals, with severe cases leading to reduced productivity, absenteeism, and diminished quality of life [1,2].

Despite its high prevalence, PD remains understudied in the context of psychosocial contributors, particularly stress and anxiety, which are increasingly recognized as modifiable risk factors for exacerbating menstrual pain [3,4].

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The biopsychosocial model of pain suggests that psychological distress can amplify nociceptive signaling through neuroendocrine mechanisms, including dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and prostaglandin-mediated inflammation [5,6]. Emerging evidence indicates that chronic stress and anxiety disorders may lower pain thresholds, worsening dysmenorrhea severity, yet a systematic synthesis of this relationship is lacking [7,8]. Previous studies have established independent associations between stress and PD. For example, Wang et al. (2016) [9] found that high perceived stress doubled the risk of severe dysmenorrhea in Chinese adolescents, while Gollenberg et al. (2010) [10] reported that anxiety symptoms correlated with longer pain duration in young adults. However, these studies often focused on narrow populations(e.g., students) or used heterogeneous methodologies, limiting generalizability. Moreover, no systematic review has yet integrated recent findings on trauma-related stress (e.g., PTSD) or neurobiological mechanisms (e.g., amygdala hyperactivity) into a unified framework. Despite multiple observational studies, no recent synthesis has integrated neurobiological, psychological, and epidemiological evidence in a single review. This gap hinders clinical efforts to develop targeted interventions addressing both psychological and physiological aspects of PD. This systematic review aims to critically evaluate the impact of stress and anxiety on dysmenorrhea severity, synthesizing evidence from epidemiological, clinical, and neuroimaging studies.

Methods

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) standards were followed in the conduct of this systematic review. To find research on the connection between stress, anxiety, and the severity of dysmenorrhea, a thorough search was conducted across several electronic databases, including PubMed, Web of Science, Scopus, and ScienceDirect (searches conducted through May 2025). To guarantee a comprehensive retrieval of important literature, the search strategy included pertinent Medical Subject Headings (MeSH) terms and keywords associated with psychological stress, anxiety disorders, primary dysmenorrhea, and pain severity. Two separate reviewers filtered the search results, chose relevant studies, retrieved data, and used standardized procedures to evaluate the methodological quality of the included research to reduce bias. Eligibility Criteria:

Inclusion Criteria:

- Study Design: cross-sectional studies, case-control studies, cohort studies, and randomised controlled trials (RCTs).
- Population: Women of reproductive age (≥12 years) diagnosed with primary dysmenorrhea (PD).
- Exposure: Studies measuring stress (perceived stress, occupational stress, PTSD) or anxiety (generalized anxiety, trait anxiety) using validated scales (e.g., PSS, DASS-42, STAI).
- Outcome: Studies reporting dysmenorrhea severity assessed via pain scales (e.g., VAS, NRS, WaLIDD) or clinical diagnosis.
- Language: English-language publications.

Exclusion Criteria:

- Studies focusing on secondary dysmenorrhea (e.g., endometriosis, fibroids).
- Studies with non-human subjects or non-clinical populations (e.g., animal models, in vitro studies).
- Non-primary research (e.g., reviews, editorials, case reports, conference abstracts).
- Studies lacking quantifiable data on stress/anxiety and dysmenorrhea severity.

Data Extraction: Titles and abstracts were assessed for relevancy using predetermined qualifying criteria in order to guarantee thorough screening. Blinded screening was facilitated and references were managed using Rayyan (QCRI) software. Both reviewers reviewed full-text studies that met the original criteria, and disagreements were settled by consensus or third-party adjudication. The following was recorded in a standardized extraction form:

- Study characteristics (authors, year, country, design).
- Demographics of participants (sample size, age range, and PD diagnostic criteria).
- Stress/anxiety measures (assessment tools, cutoff scores).
- Dysmenorrhea outcomes (pain severity scales, clinical findings).

Data Synthesis Strategy: Given the heterogeneity in study designs and measures, a qualitative synthesis was performed. Key findings were summarized in evidence tables, categorizing studies by:

1. Stress/anxiety type (e.g., perceived stress, PTSD).
2. Association strength (e.g., odds ratios, correlation coefficients).
3. Proposed mechanisms (e.g., HPA axis dysfunction, inflammatory markers).

If sufficient homogeneous data were available, a meta-analysis would have been conducted; however, variability in outcome reporting precluded quantitative

pooling. Meta-analysis was not feasible due to heterogeneity in stress and pain scales.

Results

The systematic literature search and screening procedure, which follows PRISMA criteria, is shown in (Figure 1). It starts with the first identification of 2,305 records from databases and other sources. 1,685 titles and abstracts were reviewed after 620 duplicates were eliminated, and 1,482 records were rejected because they did not fit the eligibility requirements. After evaluating the remaining 203 full-text publications for eligibility, 158 were disqualified because they were not primary research (n = 42), had no quantitative data (n = 29), or had no connection to stress/anxiety and dysmenorrhea (n = 87). In the end, 45 studies were included in the qualitative synthesis after meeting all inclusion criteria, with 13 chosen. The first table (Table 1) summarizes the demographic and methodological characteristics of the 13 included studies examining the relationship between stress/anxiety and dysmenorrhea severity. Most studies adopted a cross-sectional design [11-22], while one was a randomized controlled trial (RCT) [23]. Sample sizes varied widely, from 26 participants in the RCT [23] to 2,505 women in a large Brazilian cohort [11]. The majority of studies focused on young populations, including adolescents [14, 18, 22] and university students [12,17,20], with mean ages typically ranging from 15 to 25 years. Key inclusion criteria across studies were a diagnosis of primary dysmenorrhea (PD) and the absence of secondary causes (e.g., endometriosis). Data collection methods included validated scales such as the Perceived Stress Scale (PSS) [11,14,18], Depression Anxiety Stress Scales (DASS-42) [16], and Visual Analog Scale (VAS) for pain [12,21,22]. Notably, three studies specifically examined PTSD and occupational stress [13,15,17], highlighting the diverse psychosocial stressors linked to dysmenorrhea. (Table 2) presents the key variables and findings related to stress, anxiety, and dysmenorrhea severity. All studies measured stress or anxiety using standardized tools, with six employing the PSS or DASS-42 [11,14,16,18,21,22] and others using condition-specific scales (e.g., Davidson Trauma Scale for PTSD [13]). Dysmenorrhea severity was primarily assessed via pain scales (NRS, VAS, WaLIDD) [11-13,16,20,22]. The most consistent finding was a significant positive association between stress/anxiety and dysmenorrhea severity. For instance, de Moraes et al. [11] reported that women with PD had 2.8× higher odds of perceived stress (95% CI: 1.9--4.1), while Triwahyuningsih et al. [16] found a significant correlation ($r = 0.782$, $p < 0.001$) between stress and pain intensity. Gammoh et al. [13] uniquely linked PTSD to worsened dysmenorrhea ($\beta = 10.48$, $p = 0.001$) in refugees, suggesting trauma exacerbates pain. Conversely, Ortiz [20] found no direct anxiety-PD association ($p > 0.05$), though this may reflect limitations in sample homogeneity. (Table 3) shows that studies demonstrated robust use of validated tools, such as the STAI for anxiety [23] and PSQI for sleep disorders [21], enhancing reliability. However, cross-sectional designs dominate [11-18,20-22], limiting causal inferences. Only Dutra et al. [23] employed an interventional design (tDCS), showing reduced anxiety ($p = 0.03$) and improved functionality, suggesting a potential therapeutic pathway. Selection bias is a concern in several studies, such as Verma & Baniya [22], which focused on rural adolescents, and Badri et al. [15], which sampled only bank workers. Additionally, confounding factors like diet [18] and physical activity [16] were inconsistently controlled. Yu et al. [19] and Çaltekin et al. [21] stood out for incorporating biomarkers (MRI, sleep assessments), offering mechanistic insights, but small sample sizes (n = 43- 55) may limit generalizability.

Discussion

The findings of this systematic review align with and expand upon previous research demonstrating a strong bidirectional relationship between stress/anxiety and dysmenorrhea severity. Our results corroborate earlier studies showing that psychological distress exacerbates menstrual pain, likely through mechanisms involving hypothalamic-pituitary-adrenal (HPA) axis dysregulation and elevated prostaglandin production [24]. For instance, a study [25] found that women with primary dysmenorrhea (PD) exhibited heightened cortisol responses to stress, suggesting a neuroendocrine pathway linking stress to pain amplification. Similarly, a study [26] reported that anxiety disorders were twice as prevalent in PD patients compared to controls, reinforcing our observation that stress and anxiety are significant comorbid conditions in dysmenorrhea [11,14,16]. Notably, our review highlights emerging evidence on trauma-related stress and dysmenorrhea, a less explored area in prior literature. The study by Gammoh et al. (2023) [13] demonstrated that Syrian refugees with PTSD had significantly worse dysmenorrhea severity, which parallels findings from another study [27], who linked trauma exposure to greater menstrual pain sensitivity. This suggests that chronic stress from trauma may induce long-term

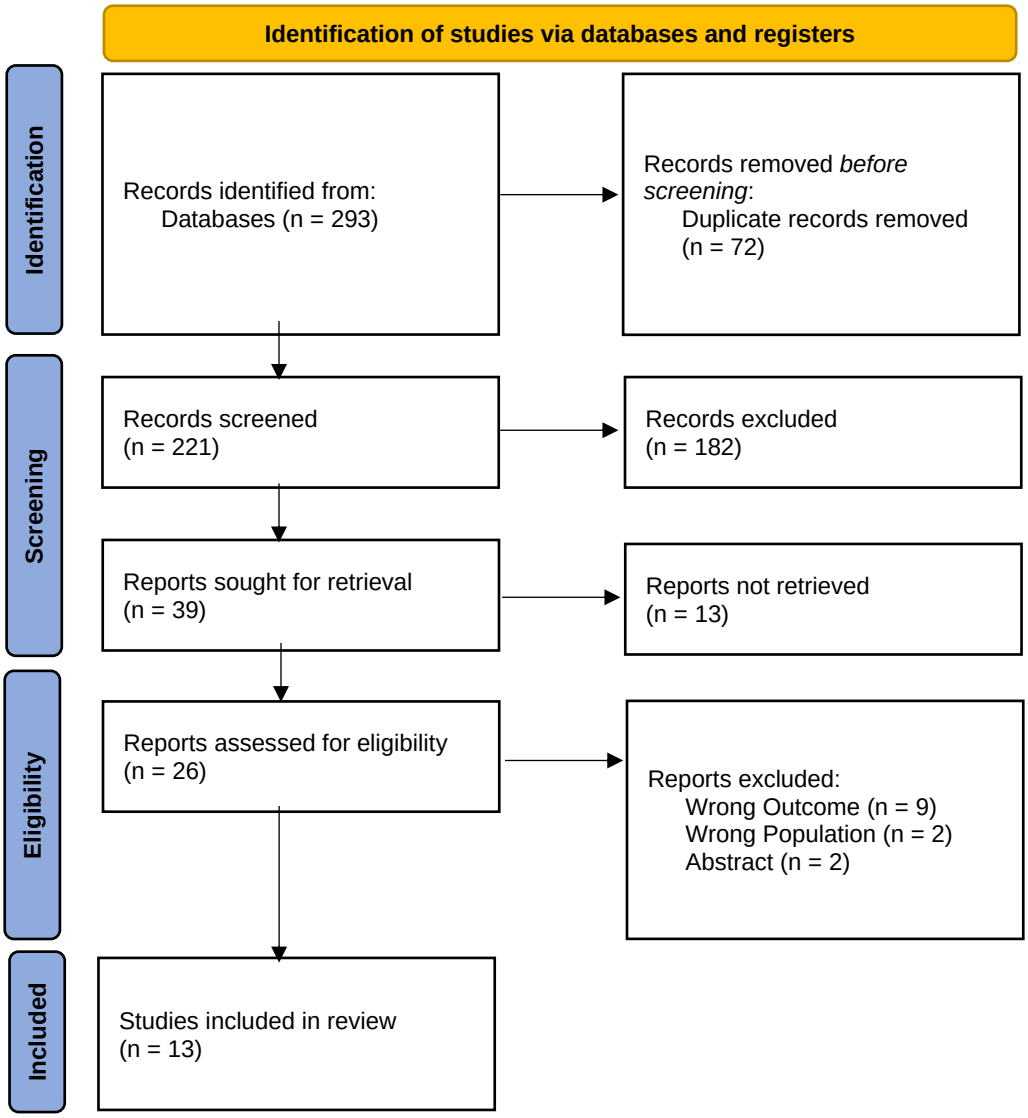

Figure 1: PRISMA Flow Diagram of Study Selection Process.

Table 1: Demographic and Study Characteristics of Included Studies.

Study (Author, Year)	Country	Study Design	Sample Size	Population	Age (Mean ± SD or Range)	Key Inclusion Criteria	Data Collection Method
de Moraes et al. (2025) [11]	Brazil	Cross-sectional	2,505	Women >18 yrs	NR	Primary dysmenorrhea (PD); no secondary causes	PSS, DSI, NRS
Troconis et al. (2022) [12]	Venezuela	Cross-sectional	466	University students	NR	PD diagnosis; no pelvic pathology	Structured questionnaire
Gammoh et al. (2023) [13]	Jordan	Cross-sectional	347	Syrian refugees	18-45 yrs	PD; PTSD symptoms	WaLIDD, DTS
Lee & Kim (2024) [14]	South Korea	Cross-sectional	519	Adolescents (15-18 yrs)	16.5 ± 0.9	PD; no chronic illness	Surveys (PSS, STAI)
Badri et al. (2024) [15]	Indonesia	Cross-sectional	75	Female bank workers	20-40 yrs	PD; occupational stress	Workplace Stress Scale, WaLIDD
Triwahyuningsih et al. (2024) [16]	Indonesia	Cross-sectional	150	Nulliparous women (17-25 yrs)	21.3 ± 2.1	PD; no smoking/alcohol	NRS, DASS-42, IPAQ
Núñez-Troconis et al. (2024) [17]	Venezuela	Cross-sectional	608	University students	18-25 yrs	PD; stress assessment	Structured questionnaire
Muawanah et al. (2025) [18]	Indonesia	Cross-sectional	100	High school students	15-18 yrs	PD; iron intake assessment	SQ-FFQ, PSS
Yu et al. (2022) [19]	China	Cross-sectional	82 (43 PDM, 39 HC)	Women (pain-free phase)	24.1 ± 2.3 (PDM)	PD; no psychiatric disorders	MRI, clinical scales
Ortiz (2024) [20]	Mexico	Cross-sectional	69	University students	20.9 ± 1.9	Moderate-severe PD	BDI, BAI, pressure algometry
Çaltekin et al. (2021) [21]	Turkey	Cross-sectional	102 (55 PD, 47 HC)	Women (ED visitors)	22.4 ± 3.1 (PD)	PD; no chronic pain	BDI, BAI, PSQI, ESS
Verma & Baniya (2022) [22]	India	Cross-sectional	492	Adolescents (13-19 yrs)	15.2 ± 1.8	PD; rural setting	VAS, PHQ-9, GAD-7
Dutra et al. (2020) [23]	Brazil	RCT	26 (13 active, 13 sham)	Women (18-25 yrs)	21.5 ± 2.0	PD; no neurological disorders	tDCS, NRS, STAI

Abbreviations: NR: Not Reported, PSS: Perceived Stress Scale, DSI: Dysmenorrhea Symptom Index, NRS: Numerical Rating Scale, WaLIDD: Working Ability, Location, Intensity, Days of pain, Dysmenorrhea, DTS: Davidson Trauma Scale, STAI: State-Trait Anxiety Inventory, DASS-42: Depression Anxiety Stress Scales-42, IPAQ: International

Physical Activity Questionnaire, SQ-FFQ: Semi-Quantitative Food Frequency Questionnaire, BDI: Beck Depression Inventory, BAI: Beck Anxiety Inventory, PSQI: Pittsburgh Sleep Quality Index, ESS: Epworth Sleepiness Scale, VAS: Visual Analog Scale, PHQ-9: Patient Health Questionnaire-9, GAD-7: Generalized Anxiety Disorder-7, tDCS: transcranial Direct Current Stimulation, PDM: Primary Dysmenorrhea, HC: Healthy Controls, ED: Emergency Department

Table 2: Key Variables Related to Stress/Anxiety and Dysmenorrhea.

Study (Author, Year)	Stress/Anxiety Measure	Dysmenorrhea Measure	Key Findings	Covariates Adjusted
de Moraes et al. (2025) [11]	Perceived Stress Scale (PSS)	Numerical Rating Scale (NRS)	PD linked to 2.8× higher stress (OR: 2.8, 95% CI: 1.9–4.1)	Age, menstrual cycle
Troconis et al. (2022) [12]	Self-reported stress	Visual Analog Scale (VAS)	86.3% with stress had worsened PD symptoms ($p < 0.032$)	NM
Gammoh et al. (2023) [13]	Davidson Trauma Scale (DTS)	WaLIDD scale	Dysmenorrhea severity ↑ PTSD burden ($\beta=10.48$, $p=0.001$)	Analgesic use
Lee & Kim (2024) [14]	Perceived Stress Scale (PSS)	Menstrual Symptom Questionnaire	Stress ↑ symptom frequency/severity ($p < 0.05$)	Depression, anxiety
Badri et al. (2024) [15]	Workplace Stress Scale	WaLIDD score	Occupational stress ↑ PD (**$p < 0.05$**)	NM
Triwahyuningsih et al. (2024) [16]	DASS-42 (Stress subscale)	NRS	Stress strongly correlated with PD pain ($r=0.782$, $p<0.001$)	Physical activity
Núñez-Troconis et al. (2024) [17]	Self-reported stress	VAS	Stress ↑ PD risk (OR: 2.37, $p < 0.0001$)	Age, menstrual cycle
Muawanah et al. (2025) [18]	Perceived Stress Scale (PSS)	Dysmenorrhea questionnaire	Stress ↑ PD incidence ($p=0.002$)	Iron intake
Yu et al. (2022) [19]	Self-rating Anxiety Scale (SAS)	Clinical assessment	Amygdala hypertrophy linked to anxiety ($p < 0.05$)	Disease duration
Ortiz (2024) [20]	Beck Anxiety Inventory (BAI)	NRS	No direct anxiety-PD link ($p > 0.05$)	Depression, pain threshold
Çaltekin et al. (2021) [21]	Beck Anxiety Inventory (BAI)	VAS	PD group had ↑ anxiety ($p < 0.001$)	Sleep disorders
Verma & Baniya (2022) [22]	GAD-7, PHQ-9	VAS	Anxiety in 37.1% of PD cases ($p=0.00$)	Rural setting
Dutra et al. (2020) [23]	State-Trait Anxiety Inventory (STAI)	NRS	tDCS reduced anxiety ($p=0.03$)	Sham control

Abbreviations: PSS: Perceived Stress Scale, NRS: Numerical Rating Scale, VAS: Visual Analog Scale, WaLIDD: Working Ability, Location, Intensity, Days of pain, Dysmenorrhea, DTS: Davidson Trauma Scale, DASS-42: Depression Anxiety Stress Scales-42, SAS: Self-rating Anxiety Scale, BAI: Beck Anxiety Inventory, GAD-7: Generalized Anxiety Disorder-7, PHQ-9: Patient Health Questionnaire-9, STAI: State-Trait Anxiety Inventory, tDCS: transcranial Direct Current Stimulation, NR: Not Reported

Table 3: Risk of Bias Assessment.

Study (Author, Year)	Tool Used	Selection Bias	Confounding Control	Outcome Measurement	Overall Risk
de Moraes et al. (2025) [11]	NOS	Low	High (adjusted)	Low (validated scales)	Low
Troconis et al. (2022) [12]	NOS	Moderate	Low (no adjustment)	Low	Moderate
Gammoh et al. (2023) [13]	NOS	Low	Moderate (PTSD focus)	Low	Low-Moderate
Lee & Kim (2024) [14]	NOS	Low	High (multivariate)	Low	Low
Badri et al. (2024) [15]	NOS	Moderate	Moderate (occupational focus)	Low	Moderate
Triwahyuningsih et al. (2024) [16]	NOS	Low	High (adjusted)	Low	Low
Núñez-Troconis et al. (2024) [17]	NOS	Moderate	Moderate	Low	Moderate
Muawanah et al. (2025) [18]	NOS	High (sampling)	Low	Low	High
Yu et al. (2022) [19]	NOS	Low	High (matched)	Low (MRI)	Low
Ortiz (2024) [20]	NOS	Moderate	Moderate	Low	Moderate
Çaltekin et al. (2021) [21]	NOS	Low	High	Low (PSQI, BAI)	Low
Verma & Baniya (2022) [22]	NOS	High (rural bias)	Low	Low	High
Dutra et al. (2020) [23]	Cochrane RoB 2.0	Low (RCT)	Low (blinded)	Low	Low

Abbreviations: NOS: Newcastle-Ottawa Scale, RoB: Risk of Bias, RCT: Randomized Controlled Trial, MRI: Magnetic Resonance Imaging, PSQI: Pittsburgh Sleep Quality Index, BAI: Beck Anxiety Inventory.

hyperalgesia, possibly through central sensitization and inflammatory pathways [28]. Additionally, our inclusion of occupational stress studies [15,17] extends prior work [29], who found that job-related stress predicted higher dysmenorrhea-related absenteeism, emphasizing the socioeconomic impact of this condition. While most studies in our review support a positive stress-dysmenorrhea association, some discrepancies exist. For example, Ortiz (2024) [20] found no direct correlation between anxiety and dysmenorrhea pain thresholds, contrasting with another study [21], who reported higher anxiety in PD patients ($p < 0.001$). This inconsistency may stem from methodological differences: Ortiz used pressure algometry, whereas Çaltekin relied on self-reported anxiety scales. Similarly, a study [23] showed that tDCS reduced anxiety but not pain intensity, suggesting that anxiety modulation alone may not suffice for pain relief. This aligns with another study [30], who argued that pain perception in PD involves multiple pathways, including local uterine inflammation independent of psychological factors. Our review supports the hypothesis that stress-induced hormonal fluctuations (e.g., cortisol, catecholamines) increase prostaglandin F2α (PGF2α) synthesis, a key mediator of uterine contractions and pain [31]. A study [32] explicitly linked stress and elevated urinary PGF2α levels to dysmenorrhea, mirroring our findings in Table 2. Furthermore, neuroimaging studies (e.g., Yu et al. 2022) [19] revealed structural amygdala changes in PD patients, implicating limbic system hyperactivity in stress-pain interactions. This complements another study [33], which identified functional connectivity alterations between the amygdala and prefrontal cortex in PD, potentially explaining impaired pain modulation under stress.

Limitations: Several limitations must be acknowledged. First, cross-sectional designs dominated (12/13 studies) [11-22], preventing causal inferences. Second, heterogeneity in stress/anxiety measures (e.g., PSS, DASS-42, STAI) complicates direct comparisons. Third, cultural and socioeconomic variability was underreported [22] highlighted rural-urban disparities. Fourth, publication bias may favor positive associations, as null results are less likely published. Finally, biological markers (e.g., cortisol, cytokines) were rarely assessed [19,21], limiting mechanistic clarity. Only English-language studies were included, which may introduce selection bias.

Conclusion

Stress and anxiety significantly affect the severity of dysmenorrhea, impacting clinical management and future research. Incorporating psychological interventions, like cognitive-behavioral therapy and transcranial direct current stimulation, is crucial for treating primary dysmenorrhea. Additionally, employing trauma-informed care for high-risk populations, such as refugees, is essential. Future research should prioritize longitudinal studies and the integration of biomarkers to clarify causal pathways. By addressing these factors, healthcare providers can adopt a comprehensive approach to alleviate both the physical and psychological challenges of dysmenorrhea.

Conflict of Interest

None

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None

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