

A Systematic Review on the Impact of Mental Health on Reproductive Hormones and Fertility Outcomes

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Abstract

Psychological stress and mental health disorders may interact with reproductive function through neuroendocrine, metabolic, behavioural, and oxidative pathways. This systematic review aimed to synthesize the available evidence on the relationship between psychological stress, mental health, reproductive hormones, and fertility outcomes in women and men, including outcomes associated with assisted reproductive technology (ART).

PubMed, Scopus, Web of Science, the Cochrane Library, and Google Scholar were searched for English-language human literature published between 2015 and 2025. Of 3,265 records identified, 1,020 duplicates were removed, 2,245 records underwent title and abstract screening, and 595 full-text reports were assessed for eligibility. Fifteen publications were included in the final narrative synthesis. The included evidence was heterogeneous and consisted predominantly of narrative reviews, systematic reviews, meta-analyses, and one review of systematic reviews. A post hoc methodological appraisal was performed using Joanna Briggs Institute (JBI) critical appraisal tools appropriate to the type of review.

Stress-related activation of the hypothalamic-pituitary-adrenal axis was associated across the reviewed literature with alterations in hypothalamic-pituitary-gonadal signalling, with cortisol, prolactin, ghrelin, gonadotropins, and gonadal steroids implicated in proposed mechanisms. In women, the literature described associations with menstrual and ovulatory dysfunction, while male reproductive literature reported possible effects on testosterone and semen parameters. Evidence regarding a direct adverse effect of psychological distress on ART pregnancy outcomes was inconsistent, and meta-analytic evidence did not consistently demonstrate an association between pretreatment or treatment-related emotional distress and pregnancy outcomes. Psychological interventions showed more consistent benefits for reducing distress and improving coping than for improving reproductive endpoints.

Psychological stress and reproductive health appear to be interrelated; however, the available evidence does not establish a definitive causal relationship between stress and impaired fertility. Interpretation is limited by heterogeneity in study designs, populations, stress measures, and reproductive outcomes, as well as overlapping evidence bases and the predominance of secondary literature. Prospective primary studies using standardized measures and rigorously designed intervention trials are needed to clarify these relationships and determine whether improvements in psychological wellbeing translate into measurable improvements in reproductive outcomes.

Categories: Obstetrics/Gynecology, Public Health

Keywords: assisted reproductive technology, cortisol, infertility, mental health, psychological stress, reproductive endocrinology, reproductive hormones

Introduction And Background

Infertility has biological, psychological, relational, and social consequences. Psychological distress is common during fertility investigation and treatment, but the direction of association is complex: infertility itself can generate anxiety, depression, and stress, while stress-related physiology may also influence reproductive function [1-4]. This bidirectionality makes causal claims difficult and requires careful separation of psychological burden from biological effects on fertility.

The main mechanistic model involves interaction between the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes. Stress-related glucocorticoid signalling can modify gonadotropin-releasing hormone (GnRH) pulsatility and downstream luteinizing hormone (LH), follicle-stimulating hormone (FSH), and gonadal steroid secretion [5-7]. Other proposed pathways include stress-related prolactin changes, ghrelin signalling, oxidative stress, sleep disturbance, sexual behaviour, and treatment discontinuation [8-11].

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In women, stress-sensitive reproductive phenotypes include functional hypothalamic amenorrhea and menstrual or ovulatory disturbance, while mental-health comorbidity is also described across reproductive disorders [12–14]. In men, psychological and oxidative stress have been linked with testosterone and semen parameters, although the magnitude and consistency of associations vary [15–17]. ART adds further psychological burden, and distress can change substantially across stimulation, oocyte retrieval, embryo transfer, pregnancy testing, and unsuccessful cycles [18,19].

This review addressed the following PECO-framed question: among reproductive-age adults (Population), how is exposure to psychological stress and mental health disturbances (Exposure), compared with lower or absent psychological distress where comparator data were available (Comparator), associated with reproductive hormone alterations and fertility-related outcomes (Outcome), including menstrual and ovulatory function, semen parameters, conception, and ART outcomes? Proposed biological mechanisms and the effects of psychological interventions were examined as secondary outcomes.

Review

Methods

Study Design and Reporting Framework

The review was conducted using the PRISMA 2020 reporting framework [20]. The underlying MSc project received low-risk ethics approval from the Faculty of Education and Life Sciences, University of South Wales, on February 27, 2025. No primary participant data were collected. No prospective systematic review protocol was registered because the review was initially undertaken as part of an academic postgraduate project and prospective registration was not incorporated into the study design before commencement of the literature search. To mitigate this limitation, the review methods were subsequently reported in detail, including the eligibility criteria, information sources, study-selection process, and synthesis approach, and the selection process was documented using the PRISMA 2020 framework. A post hoc methodological appraisal of the included publications was also undertaken using appropriate Joanna Briggs Institute (JBI) critical appraisal tools. Nevertheless, these measures cannot substitute for prospective protocol registration, and the possibility of methodological decisions being influenced after review initiation remains a limitation.

Eligibility Criteria

Eligible publications comprised English-language human studies published between 2015 and 2025 that examined psychological stress, anxiety, depression, or related mental health factors in relation to reproductive hormones, fertility outcomes, or clinically relevant reproductive life-course factors. The eligibility framework permitted observational studies, randomized controlled trials, systematic reviews, meta-analyses, narrative reviews, and other relevant review-level evidence. Outcomes of interest included reproductive hormone alterations, menstrual and ovulatory function, semen parameters, conception, implantation, pregnancy, and assisted reproductive technology (ART) outcomes. Publications addressing mental health within a relevant reproductive-life-course context were also considered where they provided contextual evidence pertinent to the interpretation of reproductive health, although these were not treated as direct evidence of fertility effects. Non-human studies, publications unrelated to reproductive health or fertility, studies without a relevant psychological or mental-health component and without either a reproductive outcome or relevant reproductive-life-course context, and reports with insufficient information for interpretation were excluded. The 2015–2025 publication restriction applied specifically to publications evaluated for inclusion in the systematic review. This 10-year period was selected to focus the review on contemporary evidence reflecting more recent approaches to psychological assessment, reproductive endocrinology, fertility treatment, and assisted reproductive technology, while providing a sufficiently broad interval to capture relevant literature. Earlier literature was used only when relevant to provide background information, describe established biological mechanisms, support methodological approaches, or provide clinical context and was not included among the 15 publications comprising the final evidence synthesis.

Given the broad scope of the review, eligible evidence included publications representing different levels of evidence, including primary studies and review-level publications. These evidence types were not treated as equivalent or independent units of evidence. Primary studies, where available, were used to describe direct clinical associations; systematic reviews and meta-analyses to summarize broader evidence patterns; and narrative reviews primarily to provide mechanistic or contextual interpretation. Because review-level publications may contain overlapping primary studies, findings from different reviews were not quantitatively pooled or interpreted as independent observations. Instead, the evidence was synthesized narratively, with consideration of the methodological design and potential overlap of the underlying evidence base. During interpretation of the narrative synthesis, greater emphasis was placed on evidence that was more directly relevant to the review question and demonstrated stronger methodological quality on critical appraisal, whereas narrative, mechanistic, or methodologically limited sources were used primarily for contextual or explanatory interpretation and were not given equivalent evidentiary weight. Publications providing reproductive-life-course context without directly assessing fertility outcomes were interpreted as contextual evidence only and were not used to support conclusions regarding direct effects on fertility.

Information Sources and Search Strategy

PubMed, Scopus, Web of Science, the Cochrane Library, and Google Scholar were searched for relevant literature. Search concepts combined terms relating to psychological stress and mental health with reproductive hormones and fertility outcomes, including infertility, ovulation, pregnancy, and assisted reproductive technology (ART). Boolean operators were used, and eligibility was restricted to English-language human literature published between 2015 and 2025. The original academic project did not preserve the complete database-specific search strings or exact final search date for each database. To improve transparency, the search strategy was retrospectively reconstructed from the original search concepts and eligibility criteria. As the exact historical search dates and verbatim database-specific syntax could not be independently verified, these were not retrospectively estimated, and this is acknowledged as a limitation of the review.

Study Selection and Data Extraction

Titles and abstracts were screened for relevance, followed by full-text eligibility assessment against the predefined inclusion and exclusion criteria. During the original full-text assessment, publications with substantial methodological or reporting limitations that prevented reliable extraction or interpretation of relevant exposure-outcome data were excluded. This eligibility decision preceded and was distinct from the post hoc JBI critical appraisal, which was subsequently applied only to the 15 included publications and was not used as an additional exclusion criterion. Screening and data extraction were conducted by a single reviewer as part of the original postgraduate academic project; therefore, independent duplicate screening and extraction by two reviewers were not performed. Data were extracted using a structured spreadsheet covering author, year, country, study design, population, stress or mental health exposure, assessment method, reproductive hormone measures, fertility outcomes, and reported interventions. To promote consistency and reduce selection bias, the predefined eligibility criteria were applied throughout the screening process, and extracted information was cross-checked against the source publications during preparation of the revised manuscript. As independent duplicate review was not undertaken, the possibility of reviewer-dependent selection or extraction error cannot be completely excluded and is acknowledged as a methodological limitation. Given the heterogeneity and predominantly secondary nature of the final evidence set, findings were synthesized narratively rather than statistically pooled.

Critical Appraisal and Risk of Bias

In response to editorial review, a post hoc critical appraisal was undertaken using standardized Joanna Briggs Institute (JBI) tools selected according to publication type [21,22]. Systematic reviews, meta-analyses, and the review of systematic reviews were assessed using the 11-item JBI Critical Appraisal Checklist for Systematic Reviews and Research Syntheses. Narrative and mechanistic reviews were assessed using the JBI textual-evidence framework, focusing on identifiable expertise, population relevance, logical argumentation, use of extant literature, and handling of discordant evidence. Responses were recorded as Yes, No, Unclear, or Not Applicable. Because this appraisal was performed during manuscript revision and not prospectively prespecified, results are presented as a transparency and quality assessment rather than as an exclusion rule. The presentation of the study-selection flow diagram was additionally aligned with the format used in a recently published Cureus systematic review [23].

Synthesis

Meta-analysis was not performed because of marked heterogeneity in publication type, populations, stress measures, hormonal assays, and fertility outcomes. The synthesis therefore emphasizes consistency, uncertainty, and conflicts between sources rather than calculating a pooled effect. Evidence was compared according to publication type, methodological quality, directness to the review question, and consistency of findings, with review-level findings interpreted cautiously where overlap in underlying primary studies was possible.

Results

Study Selection

The search identified 3,265 records. After removal of 1,020 duplicates, 2,245 records were screened by title and abstract, of which 1,650 were excluded. Of the 595 full-text reports assessed, 580 were excluded: incomplete outcome data (n=180), methodological or reporting limitations that prevented reliable extraction or interpretation of the outcomes relevant to this review (n=145), absence of reproductive hormone or fertility outcomes (n=110), absence of a primary stress or mental-health exposure (n=85), and duplicate or overlapping populations (n=60). These 145 exclusions were made during the original full-text eligibility assessment and were not based on the subsequently performed JBI critical appraisal. Fifteen publications were included in the narrative synthesis. The study-selection process is summarized in Figure 1.

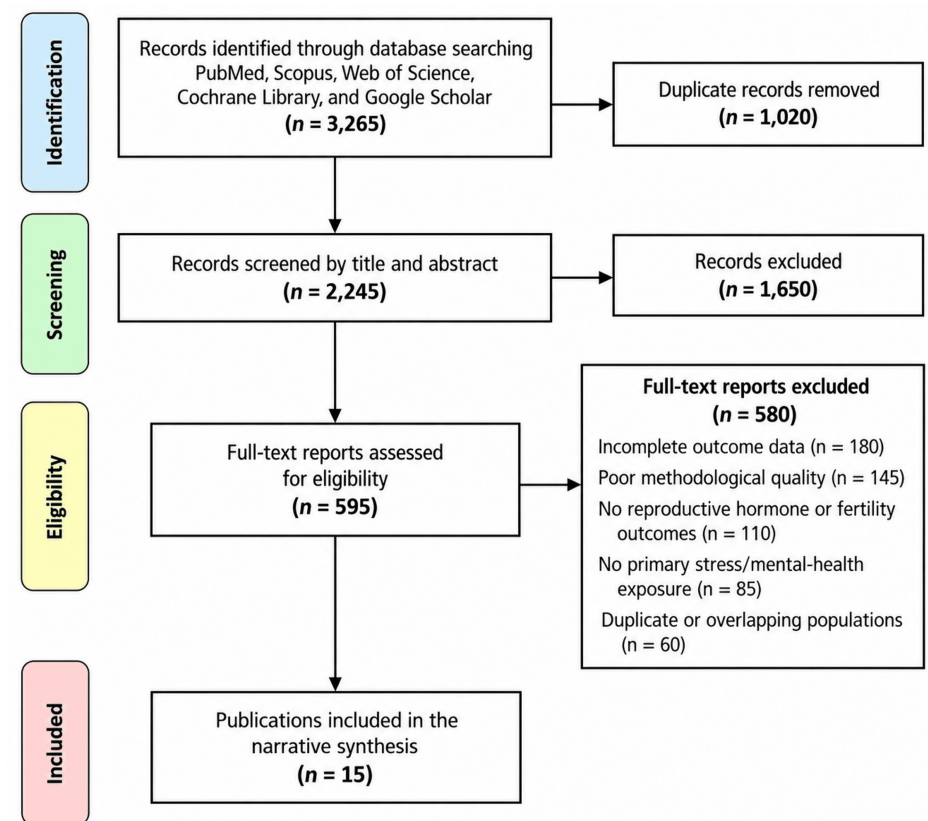


FIGURE 1: PRISMA 2020 flow diagram of study selection

The 15 included publications comprised narrative and mechanistic reviews, systematic reviews, meta-analyses, and a review of systematic reviews addressing stress, mental health, reproductive endocrinology, fertility, and assisted reproductive outcomes. The characteristics, primary focus, and key findings of the included publications are summarized in Table 1.

Study	Year	Design	Focus	Key finding
Palomba et al. [24]	2018	Narrative review	Female fertility, stress and quality of life	Stress and infertility are closely associated; directionality is difficult to establish, and lifestyle/psychological factors may interact with reproductive function.
Rooney and Domar [1]	2018	Narrative review	Infertility-related stress and treatment	Infertility clearly causes distress; whether stress causes infertility is less certain. Psychological interventions reduce distress, with variable evidence for pregnancy benefit.
Ramya et al. [25]	2023	Narrative/mechanistic review	Multiple stressors and reproductive hormones	Describes HPA-HPG interactions, cortisol, prolactin, gonadal steroids, and oxidative pathways that may impair reproduction.
Karunyam et al. [10]	2023	Systematic review	Cortisol and infertility	Cortisol findings differed by population, sampling, and study design; the review supports a stress-fertility association but emphasizes heterogeneity.
Wieczorek et al. [14]	2023	Narrative review	Reproductive hormones and female mental wellbeing	Reviews bidirectional relationships between reproductive hormone fluctuations and mental wellbeing across reproductive stages.
Deierlein et al. [26]	2024 online/2025 volume	Systematic review	Mental health across reproductive life course in women with disabilities	Found substantial mental-health burden, but no included studies specifically examined fertility, menstruation or menopause; direct fertility inference is therefore limited.
Sominsky et al. [9]	2017	Mechanistic review	Stress, ghrelin and reproduction	Proposes ghrelin as a mediator connecting stress, energy balance and suppression of reproductive function.
Odetayo et al. [27]	2024	Narrative/mechanistic review	Stress, GnIH and male fertility	Describes GnIH-related suppression of the male reproductive axis as a plausible stress pathway.
Lamprou et al. [28]	2021	Review of systematic reviews	Stress/anxiety during fertility treatment	Fourteen reviews/meta-analyses showed conflicting reproductive-outcome results; psychological support consistently aided adjustment and well-being.
Galst [29]	2018	Research review	Stress and infertility	Concludes the stress-infertility relationship is clinically important but causally difficult to resolve.
Zaks et al. [30]	2023	Systematic review/meta-analysis	Mental health and female reproductive disorders	Reported approximately two- to three-fold higher odds of psychiatric disorders among women with reproductive system disorders; temporal and causal direction remained uncertain.
Dehkordi et al. [11]	2025	Systematic review	Occupational stress and fertility	Reported associations between job stress and fertility-related outcomes or intentions, with heterogeneous measures and designs.
Lewinski and Brzozowska [31]	2022/2023 publication record	Short narrative review	Stress-related hormonal changes and female infertility	Summarizes glucocorticoid, autonomic, and endocrine mechanisms potentially linking chronic stress with female reproductive dysfunction.
Nicoloro-SantaBarbara et al. [32]	2018	Meta-analysis	Emotional distress and ART outcomes	Across 20 prospective studies (N=4,308), pretreatment anxiety, depressive symptoms, and perceived stress were not associated with less favorable ART pregnancy outcomes.
Szkodziak et al. [4]	2020	Systematic review	Psychological aspects of infertility	Synthesized evidence linking mental disorders and reproductive physiology; methodological heterogeneity limits causal interpretation.

TABLE 1: Summary of the 15 included publications and key findings

Quality and Risk-of-Bias Assessment

The critical appraisal was undertaken post hoc during manuscript revision and was not prespecified in a

prospectively registered review protocol. Accordingly, the appraisal was used to characterize the methodological strengths and limitations of the included evidence rather than to determine study eligibility or generate definitive quality grades; the numerical JBI summaries should therefore be interpreted descriptively. Systematic reviews and meta-analyses generally reported clear research questions and inclusion criteria, while the principal methodological limitations included variable reporting of duplicate critical appraisal, methods for minimizing errors during data extraction, assessment of publication bias, and prospective protocol or review-method reporting. Narrative and mechanistic reviews were generally well-referenced and biologically coherent but were limited by the absence of systematic and reproducible search, selection, and appraisal methods. The results of the JBI critical appraisal for the included publications are summarized in Table 2. Importantly, this post hoc JBI appraisal was applied only to the 15 publications already included in the final synthesis and was not used to exclude publications during the original study-selection process. Accordingly, mechanistic conclusions from narrative sources were interpreted as explanatory evidence rather than as evidence of causality.

Publication	Appraisal tool	Result	Interpretation
Karunyam et al. [10]	JBI systematic review	9/11 Yes	Higher methodological quality; broad search and structured synthesis; some appraisal/reporting domains not fully satisfied.
Deierlein et al. [26]	JBI systematic review	9/11 Yes	Clear methods and explicit study-quality assessment; fertility-specific applicability is limited.
Zaks et al. [30]	JBI systematic review	10/11 Yes	Strong systematic methods and quantitative synthesis; observational evidence limits causal inference.
Dehkordi et al. [11]	JBI systematic review	9/11 Yes	Structured systematic methods; heterogeneity of exposures/outcomes remains important.
Nicoloro-SantaBarbara et al. [32]	JBI systematic review	10/11 Yes	Prospective-study meta-analysis with appropriate quantitative synthesis; outcome restricted to ART pregnancy success.
Szkodziak et al. [4]	JBI systematic review	7/11 Yes	PRISMA-based search and two reviewers reported, but appraisal and synthesis methods were less detailed.
Lamprou et al. [28]	JBI systematic review/research synthesis	7/11 Yes	Umbrella-style review with multiple databases; limited detail on independent appraisal and publication bias.
Palomba et al. [24]	JBI textual evidence	5/6 Yes	Focused, well-referenced narrative review; not a formal systematic review.
Rooney and Domar [1]	JBI textual evidence	5/6 Yes	Clinically focused and well referenced; narrative selection methods limit reproducibility.
Ramya et al. [25]	JBI textual evidence	5/6 Yes	Broad mechanistic synthesis; narrative methods and mixed evidence types limit bias control.
Wieczorek et al. [14]	JBI textual evidence	5/6 Yes	Literature search described and argument supported; narrative design limits reproducibility.
Sominsky et al. [9]	JBI textual evidence	6/6 Yes	Detailed mechanistic review with extensive literature support and discussion of competing pathways.
Odetayo et al. [27]	JBI textual evidence	5/6 Yes	Mechanistically coherent and referenced; no formal systematic selection or risk-of-bias process.
Galst [29]	JBI textual evidence	5/6 Yes	Balanced clinical research review acknowledging conflicting evidence; non-systematic selection.
Lewinski and Brzozowska [31]	JBI textual evidence	5/6 Yes	Focused endocrine narrative with literature support; short-review format limits methodological detail.

TABLE 2: Post-hoc JBI critical appraisal of included publications

The appraisal was conducted by a single assessor during manuscript revision; therefore, independent duplicate appraisal and formal disagreement resolution were not undertaken. The JBI scores presented in Table 2 represent descriptive summaries of the appraisal rather than validated quality grades. Methodological quality informed the narrative synthesis by determining the weight placed on individual

sources: findings from more methodologically robust systematic reviews and meta-analyses were given greater interpretive emphasis, whereas findings from narrative or methodologically limited sources were used primarily for contextual or mechanistic interpretation and were not treated as equivalent evidence of causality.

Stress, the HPA-HPG/HPO Interface, and Reproductive Endocrinology

Stress activates the HPA axis, increasing corticotropin-releasing hormone, adrenocorticotrophic hormone, and glucocorticoid signalling. Sustained glucocorticoid exposure can interact with hypothalamic and pituitary reproductive signalling, providing a biologically plausible route through which severe or chronic stress could alter GnRH pulsatility and gonadotropin secretion [5-7]. The reproductive effect is unlikely to be uniform because stress intensity, duration, individual resilience, energy availability, sleep, medication, and underlying reproductive disease modify the response.

Prolactin and metabolic signalling provide additional links. Stress-related hyperprolactinemia can suppress reproductive function in susceptible individuals [8]. Sominsky et al. described ghrelin as a potential interface between stress, energy balance, and reproduction, particularly when stress coexists with altered nutrition or metabolic demand [9]. These mechanisms support biological plausibility but should not be interpreted as proof that ordinary psychological distress directly causes infertility.

Cortisol is the most frequently discussed stress biomarker, yet its interpretation is difficult. Karunyam et al. found substantial variation in cortisol sampling matrices, timing, populations, and infertility diagnoses [10]. A single serum or salivary measurement may not adequately represent chronic HPA activity. Circadian rhythm, sleep, acute procedural stress, medications, and ART stage can all influence measurements.

Taken together, these neuroendocrine and metabolic mechanisms provide biological plausibility for an association between stress and altered reproductive physiology; however, biological plausibility is distinct from demonstrated clinical effect. Much of the evidence supporting HPA-HPG/HPO interactions is mechanistic, narrative, or observational and cannot establish that stress-related endocrine alterations directly cause infertility. Accordingly, these mechanistic findings are interpreted as explanatory evidence, while conclusions regarding fertility and ART outcomes are considered separately based on studies directly evaluating those clinical outcomes.

Female Reproductive Function and Mental Health

Reviews by Palomba et al. and Ramya et al. describe plausible associations between stress and reproductive dysfunction [24,25]. In primary observational research, Schliep et al. reported associations between preconception perceived stress, reproductive hormone levels, and longer time to pregnancy [33], while Nillni et al. examined associations of depressive symptoms, perceived stress, and psychotropic medication use with menstrual-cycle characteristics [34].

However, mental-health symptoms and reproductive disorders also share bidirectional and common-cause pathways. Zaks et al. reported approximately two- to three-fold increased odds of psychiatric disorders in women with reproductive system disorders, but emphasized limitations in temporal characterization and diagnostic measurement [30]. Thus, depression or anxiety may be a consequence, correlate, contributor, or shared manifestation rather than a single causal exposure.

Wieczorek et al. further highlighted that reproductive hormones themselves can influence mood and mental wellbeing across the menstrual cycle and reproductive lifespan [14]. This reinforces the need to avoid a one-directional model in which psychological symptoms are assumed to be the initiating cause of reproductive dysfunction.

Male Reproductive Function

Male reproductive effects have been attributed to HPA-HPG interactions, gonadotropin-inhibitory hormone signalling, oxidative stress, sleep, and behavioural changes. Odetayo et al. describe GnIH as a plausible mediator through which stress may influence GnRH, LH/FSH signalling, testosterone production, and spermatogenesis [27]. Reviews of male fertility describe potential associations between psychological or oxidative stress and semen concentration, motility, morphology, and DNA integrity [15-17]. However, Nouri et al. found that subjective male stress was not significantly associated with sperm density or progressive motility during IVF, highlighting the inconsistency of the clinical evidence [35].

Assisted Reproductive Technology Outcomes

ART is psychologically demanding, and distress commonly increases around invasive procedures, waiting periods, pregnancy testing, and unsuccessful cycles [1,18,19]. Prospective psychosocial evidence has also examined whether emotional distress predicts ART failure [36]. Nevertheless, evidence that emotional

distress itself reduces the probability of ART pregnancy is inconsistent. This distinction is clinically important because patients should not be made to feel responsible for unsuccessful treatment because they were anxious or distressed.

Nicoloro-SantaBarbara et al. pooled 20 prospective studies including 4,308 women and found that anxiety, depressive symptoms, and perceived stress before treatment, and anxiety or depressive symptoms during treatment, were not associated with less favorable pregnancy outcomes [32]. Lamprou et al.'s review of systematic reviews similarly concluded that the evidence regarding psychological factors and treatment outcomes was conflicting, while psychological support improved adjustment and wellbeing [28]. These findings temper stronger causal claims made in some narrative sources.

Psychological Interventions and Integrated Fertility Care

Psychological interventions have more consistent evidence for improving psychological wellbeing than for improving reproductive outcomes. Cognitive behavioural approaches, mind-body programs, mindfulness-based interventions, counselling, and structured psychosocial support have been associated with improvements in outcomes such as anxiety, depressive symptoms, coping, adjustment, and treatment experience [1,37-41]. Although some meta-analytic evidence has suggested possible reproductive benefits [37], these findings should be interpreted cautiously because intervention types, comparator groups, fertility diagnoses, study populations, and reproductive endpoints-including conception, clinical pregnancy, and live birth-vary substantially across studies. Consequently, the available evidence does not support a consistent or definitive effect of psychological interventions on fertility outcomes.

Accordingly, mental-health support should be integrated into fertility care primarily because improvement in psychological wellbeing is an important clinical outcome in its own right. Screening can identify severe anxiety, depression, trauma symptoms, relationship strain, or treatment-related distress that warrants appropriate support or intervention. Any potential effect of psychological interventions on conception, pregnancy, or live-birth outcomes should be regarded as uncertain and secondary to the more consistently demonstrated psychological benefits.

Clinical Interpretation

The available evidence supports biological plausibility for interactions between psychological stress and reproductive function but does not establish a direct causal relationship between stress and infertility. Clinical associations remain heterogeneous and may reflect interactions among endocrine, metabolic, behavioural, and treatment-related factors, while infertility itself can contribute substantially to psychological distress. Patient-centered fertility care should therefore address both reproductive and mental health without implying causality or patient blame.

Limitations

This review has several important methodological limitations. The search was restricted to English-language literature published between 2015 and 2025. The final evidence set included primary studies and heterogeneous review-level evidence, including narrative reviews, systematic reviews, meta-analyses, and an umbrella-style review. Several included publications were broader than the core fertility question; notably, the Deierlein review did not identify studies specifically examining fertility outcomes. The original project did not preserve complete reproducible database-specific search strings or exact search dates, did not prospectively register a protocol, and did not prespecify a validated critical-appraisal instrument. Screening and data extraction were undertaken by a single reviewer, and the JBI critical appraisal was added post hoc during manuscript revision.

The inclusion of different levels of evidence also creates potential overlap between underlying primary studies and a risk of double-counting if review-level findings are interpreted as independent evidence. No quantitative pooling across reviews was undertaken, and a formal citation-matrix assessment of overlap was not performed; therefore, residual overlap cannot be excluded. Although the review incorporated systematic elements, including predefined eligibility criteria, searches across multiple information sources, structured study selection and data extraction, PRISMA-based reporting, and critical appraisal of the included evidence, the methodological limitations described above mean that it does not fulfil all features expected of a prospectively planned and fully reproducible systematic review. Accordingly, the findings should be interpreted as a structured systematic synthesis of heterogeneous, predominantly secondary evidence rather than as a definitive estimate of causal effects, and apparent consistency across reviews should be interpreted cautiously.

Future Research

Future work should prioritize prospective primary studies with repeated validated psychological measures, standardized hormone sampling, repeated semen assessment, and prespecified fertility endpoints. Studies should adjust for age, infertility diagnosis, BMI, sleep, medication, substance use, occupational exposures,

energy availability, and treatment stage. Randomized trials of psychological interventions should distinguish patient-reported mental-health benefits from biological reproductive outcomes and report ongoing pregnancy and live birth. Future systematic reviews should prospectively register protocols, preserve complete database-specific search strategies, avoid overlapping review-level evidence where possible, and conduct independent risk-of-bias assessment before synthesis.

Conclusions

Stress, mental health, and reproductive health are interconnected through neuroendocrine, metabolic, behavioral, and psychosocial pathways. The literature supports biological plausibility for stress-related reproductive endocrine effects, but clinical associations are heterogeneous, and directionality is often uncertain. Evidence that emotional distress directly reduces ART pregnancy success is inconsistent, whereas the psychological burden of infertility and the benefit of psychosocial support for wellbeing are much clearer. Fertility services should incorporate appropriate mental-health screening and support while avoiding deterministic messages that stress alone causes infertility or treatment failure. Better prospective primary research is needed to define which patients are biologically vulnerable to stress-related reproductive effects and whether targeted interventions improve reproductive endpoints.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Jijisha Ali, Neha Gami

Acquisition, analysis, or interpretation of data: Jijisha Ali, Neha Gami

Drafting of the manuscript: Jijisha Ali, Neha Gami

Critical review of the manuscript for important intellectual content: Jijisha Ali, Neha Gami

Supervision: Jijisha Ali, Neha Gami

Disclosures

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