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CHRONIC PSYCHOLOGICAL STRESS AND FEMALE REPRODUCTIVE DYSFUNCTION: EFFECTS ON OVARIAN RESERVE – A NARRATIVE REVIEW

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ABSTRACT

Chronic psychological stress negatively affects systems including the female reproductive axis. Ovarian reserve, reflecting oocyte quantity and quality, is a determinant of fertility and reproductive aging. In contemporary society, uncertainty, economic instability, poor work-life balance, and expanding social roles of women contribute to delayed childbearing and attention to age-related fertility decline. This review evaluates the impact and long-term consequences of chronic psychological stress on ovarian reserve and reproductive aging in women. A narrative review was performed using PubMed and Scopus. The search included publications from the past 20 years on chronic psychological stress, ovarian reserve, ovarian dysfunction, premature ovarian insufficiency, and reproductive aging. Guidelines, original studies, and reviews were analyzed. Studies suggest that women exposed to chronic stress often report increased perceived stress before reproductive dysfunction appears. Clinical and demographic data indicate that chronic stress, emotional trauma, and major life events may contribute to ovarian dysfunction and act as triggers of premature ovarian insufficiency. These factors are associated with reduced ovarian reserve markers and hormonal changes, including altered AMH, FSH, and cortisol. Overall, evidence supports a possible link between chronic psychological stress and accelerated reproductive aging, although findings remain heterogeneous due to differences in study design, stress assessment, populations, and outcomes. Chronic stress appears to be associated with impaired ovarian function, decreased ovarian reserve, and earlier reproductive aging. These findings underline the need to identify at-risk women and integrate stress management, support, and preventive counseling into reproductive health care. Further studies are needed to clarify mechanisms, define biomarkers, and guide clinical practice.

KEYWORDS

Chronic Psychological Stress; Ovarian Reserve; Ovarian Function; Reproductive Aging; Premature Ovarian Insufficiency; Diminished Ovarian Reserve; Female Infertility

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1. Introduction

Ovarian reserve is a key determinant of female fertility and reproductive lifespan. Women are born with a finite number of oocytes that progressively decline throughout life, leading to reproductive aging. Conditions such as diminished ovarian reserve and primary ovarian insufficiency-the latter defined as oligo- or amenorrhea for at least four months before the age of 40 years accompanied by elevated serum follicle-stimulating hormone levels (>25 IU/L) on two separate occasions-are associated not only with reduced fertility but also with long-term health consequences, including cardiovascular disease, skeletal morbidity, and earlier menopause (figure 1). [1-3]

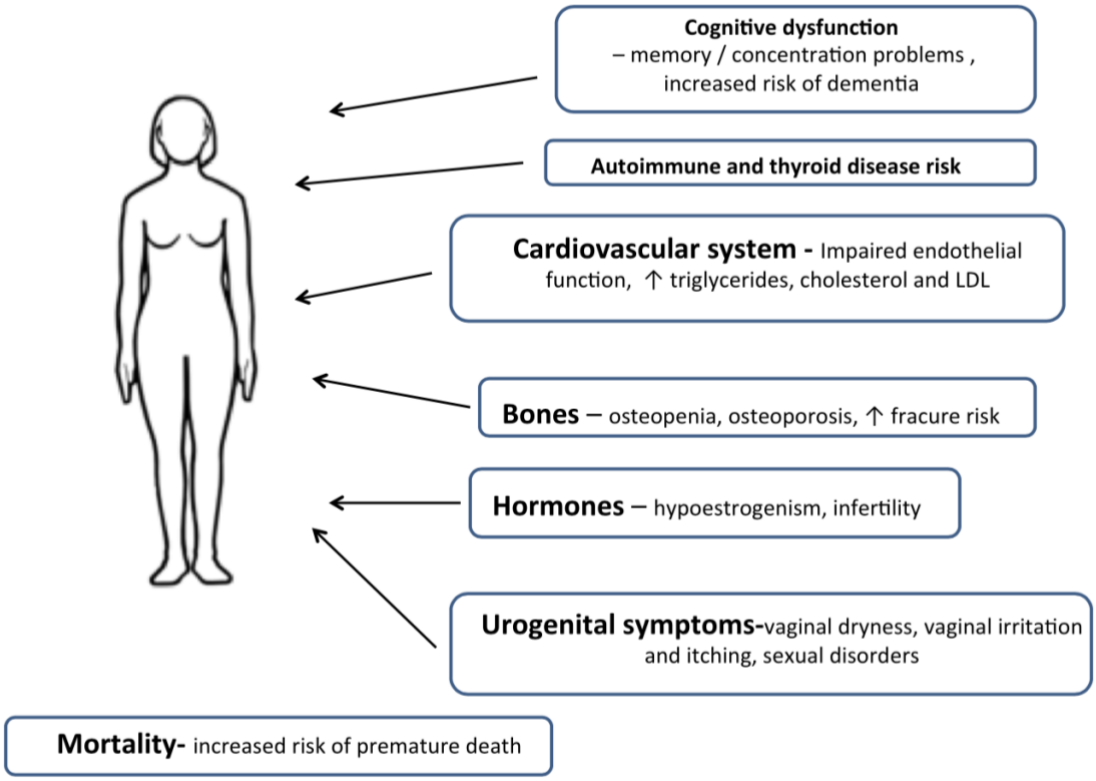

Fig. 1. *Long-term consequences of premature ovarian insufficiency (reproduced from Podfigurna-Stopa et al., 2016). [3]*

In modern society, the expanding social roles of women have contributed to delayed childbearing, which has increased clinical attention to ovarian reserve and fertility. [4] Infertility affects approximately 10-15% of couples worldwide. [5] The development of assisted reproductive technologies and improved access to fertility services has led to a growing number of patients seeking treatment, which has also increased awareness of the psychological burden associated with infertility. [6]

Chronic psychological stress is increasingly prevalent and has been shown to affect multiple physiological systems. [7,8] In women, stress may dysregulate the hypothalamic-pituitary-ovarian axis and increase cortisol production, which can inhibit estradiol synthesis by follicular cells. This may negatively affect reproductive function through endocrine disruption as well as indirect mechanisms, including lifestyle changes such as increased alcohol consumption and smoking, which promote oxidative stress and may further impair fertility. [9,10] Women may be particularly vulnerable to these effects due to both biological and psychosocial factors, potentially resulting in a self-reinforcing cycle between stress and reproductive dysfunction (figure 2).

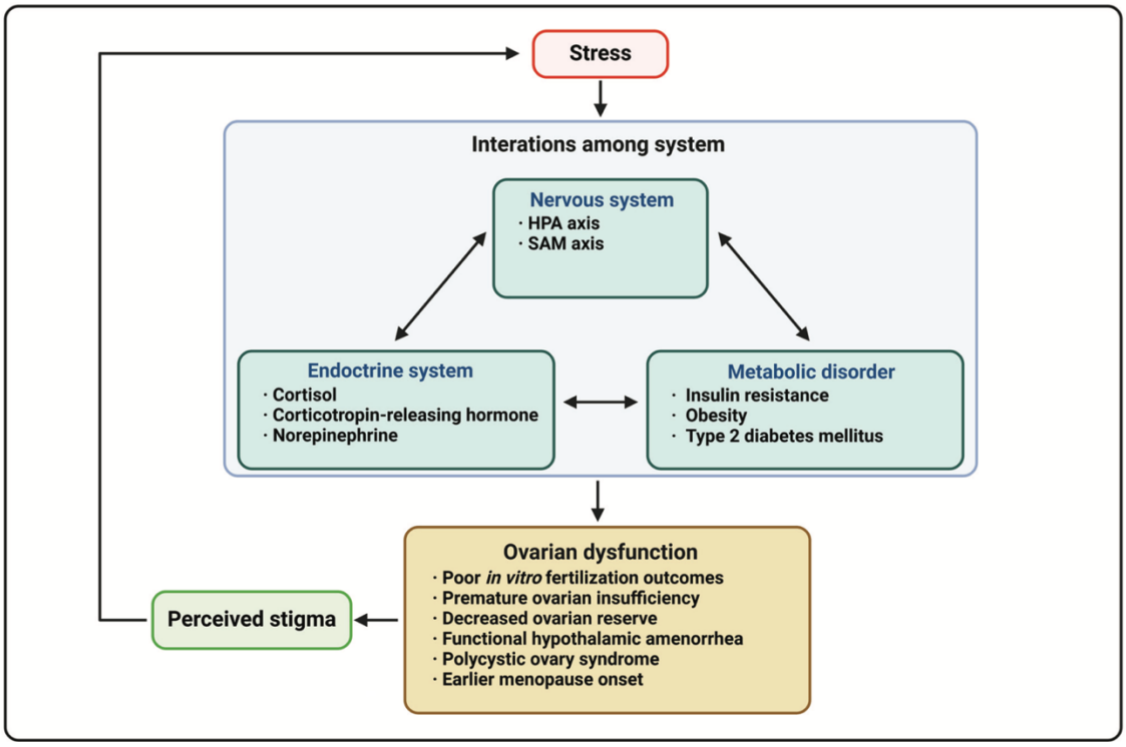

Fig. 2. *Association between elevated psychological stress and reproductive function. [9]*

Despite growing evidence, findings remain inconsistent and the underlying mechanisms are not fully understood. Therefore, this narrative review aims to synthesize current evidence on the impact of chronic psychological stress on ovarian function and reproductive aging in women.

2. Methodology

2.1. Data collection

This study was conducted as a narrative review of the literature. A systematic search was performed in the PubMed and Scopus databases to identify publications on the impact of chronic psychological stress on ovarian function and reproductive aging in women. The search included keywords related to stress, ovarian reserve, reproductive aging, and female infertility.

Eligible studies included original research articles, review articles, case reports, and clinical guidelines published in English over the last 20 years. Studies were selected based on relevance and quality of evidence, with emphasis on hormonal, clinical, and reproductive outcomes associated with chronic psychological stress and mental health.

3.Results

3.1. Findings from experimental animal models

Experimental studies using animal models suggest that chronic psychological stress may impair ovarian function and contribute to diminished ovarian reserve. In a chronic unpredictable stress (CUS) model in mice, commonly used to simulate depression- and anxiety-like states, stress was induced through repeated exposure to diverse stressors, including restraint, sleep disruption, food and water deprivation, and environmental disturbances. [11]

Prolonged exposure to chronic stress was associated with anxiety-like behavior and disturbances in the estrous cycle, including reduced duration of the fertile phase and prolonged non-reproductive phases. These alterations were accompanied by decreased anti-Mullerian hormone (AMH) levels, increased follicle-stimulating hormone (FSH) levels, reduced expression of follicle-stimulating hormone receptor (FSHR), and impaired follicular development, including a decreased number of antral follicles and increased follicular atresia. Furthermore, stress exposure was linked to reduced reproductive outcomes, as evidenced by decreased offspring survival rates. [11]

3.2. Observational studies in women

The included studies with sample sizes ranging from 45 to 250 participants, conducted across different countries, including India, the USA, Ukraine, China, and Iran. The study populations primarily consisted of women of reproductive age presenting with fertility-related concerns, including those with newly diagnosed idiopathic primary ovarian insufficiency (POI). [10,12-15]

Participants were recruited from both urban and rural settings. Psychological stress was assessed using structured questionnaires or semi-structured interviews, while hormonal parameters, including serum cortisol, FSH, and anti-Mullerian hormone (AMH), were measured during the early follicular phase. [10,12-14]

Overall, higher perceived stress levels were associated with elevated cortisol concentrations and unfavorable reproductive hormone profiles, characterized by increased FSH and decreased AMH levels, suggesting diminished ovarian reserve. These associations were more pronounced among women reporting anxiety and chronic stress symptoms. [10,13,14]

Qualitative analyses identified a broad spectrum of adverse life events preceding reproductive dysfunction (figure 3). The most frequently reported stressors included workplace-related stress, interpersonal relationship difficulties, family conflicts, caregiving burden, serious illness or death of family members, and financial hardship. [12]

Workplace stress emerged as the most prevalent factor, particularly among employed and highly educated women, reflecting increasing socioeconomic pressures, including rising costs of living, education, and housing. The growing participation of women in the workforce and their expanding professional roles may further contribute to cumulative stress exposure. [12]

Family-related stressors were also commonly reported, highlighting the psychosocial burden associated with interpersonal relationships and caregiving responsibilities. In many cultural contexts, women continue to bear primary responsibility for household duties and caregiving, even when engaged in full-time employment, resulting in a “double burden” that may increase vulnerability to stress-related health disturbances.

In addition, sleep disturbances, including insomnia, irregular sleep patterns, and chronic sleep deprivation, were frequently observed and were often linked to prolonged stress exposure. Previous evidence suggests that disruptions in sleep and circadian rhythms may contribute to menstrual irregularities; however, their role in the progression of POI remains unclear. [10,12-14]

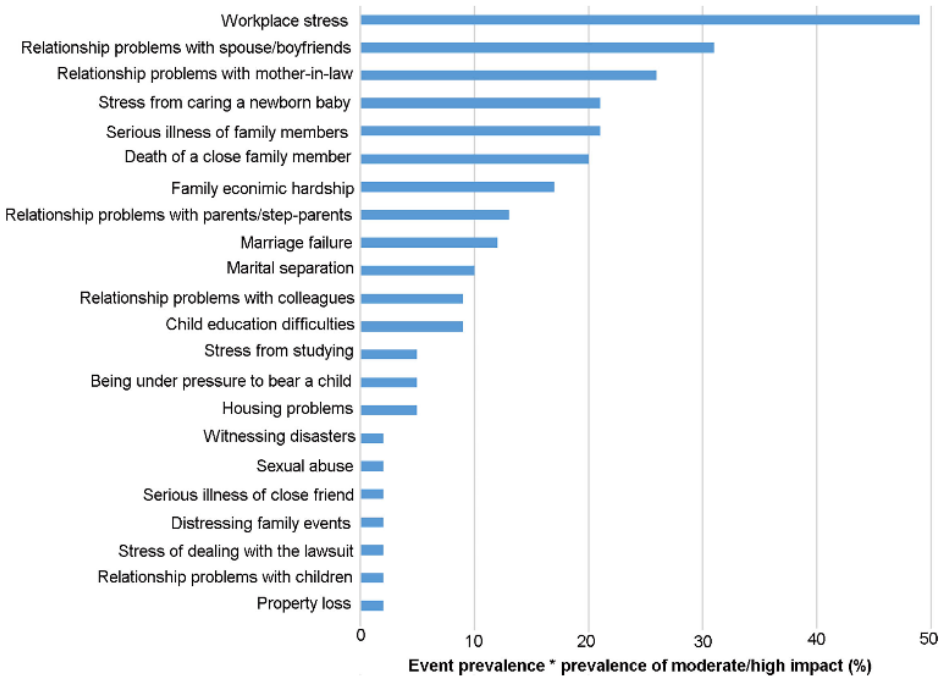

Fig. 3. *Stressful life events with the prevalence-impact scores. The prevalence of stressful life events and the frequency of moderate/high impact is treated as percentages, and their product is multiplied by 100 to obtain the frequency-impact score. [12]*

An interventional component reported in one study indicated that lifestyle modifications, including a healthy diet, physical activity, yoga, and meditation, were associated with reduced stress levels and partial improvement in hormonal parameters (figure 4). In some cases, participants were also advised to initiate hormone therapy under medical supervision. [10,17]

Table 1: The hormone levels of the selected population before intervention

No. of patients	Cortisol levels (mcg/dL)	FSH (m IU/ m L)	LH (mIU/m L)	AMH (ng/mL)
08	21-22	9-12	7-10	1-1.5
10	2.5-4.5	12-14.9	10-12	0.15-0.25
12	22-26	14.9-16	2.5-4.8	1-1.2
15	1-5.2	1-1.8	12-13.5	0.9-1
17	23-25	1.98-2.1	5-8.5	1.1-2
18	3-3.5	2.1-2.4	14-16.5	0.5-0.85
20	25-25.5	17-21.3	17-20.1	0.8-1

Table 2: The hormone levels of the selected population after intervention:

No. of patients	Cortisol levels (mcg/d L)	FSH (m IU/m L)	LH (mIU/m L)	AMH (ng/m L)
08	10-15	4-5	3.9-4.8	1.5-1.8
10	8-11	2.8-3.5	2.6-3.7	2-2.5
12	16-19	1.9-4	2- 4.1	2.5-3.3
15	20-24	5.4-7.1	5.7-7.4	3.8-4
17	18-22	3.5-7.8	3.8-7	3-3.55
18	15-17	7-8.9	7.2-9	1.9-2
20	9-13	7.5-9	9-14.5	3.5-3.95

Fig. 4. *Comparison of hormonal parameters before and after lifestyle intervention in participants from one of the included studies. [10]*

Population-based studies conducted during periods of extreme stress, such as war, further support the detrimental impact of chronic psychosocial burden on women's health. Data from Ukraine indicate a high prevalence of stress, anxiety, and sleep disturbances among women, particularly in younger age groups. Prolonged exposure to such stressors has been associated with worsening mental health and may contribute to reproductive dysfunction. [15]

Excessive stress-related and socio-economic factors have been linked to a range of reproductive disorders, including premature menopause, severe climacteric symptoms, secondary amenorrhoea, abnormal uterine bleeding, and infertility. An increased incidence of gynecological pathologies has been observed among women exposed to chronic stress conditions, including those affected by military conflict. Notably, recent observations suggest a growing number of younger women diagnosed with POI, as well as more severe menopausal symptoms among perimenopausal women. [15]

Overall, these findings indicate that chronic psychological stress represents a significant factor associated with impaired ovarian function and reproductive aging, highlighting the importance of early identification and targeted preventive strategies in women at risk.

3.3. Premature ovarian insufficiency: long-term consequences

Premature ovarian insufficiency (POI) is associated with significant long-term reproductive and systemic health consequences, affecting fertility, metabolic status, cardiovascular risk, bone health, and psychological wellbeing. [3,16,17,25]

Reproductive outcomes

POI demonstrates an intermittent course in a subset of patients, with spontaneous conception occurring in 4-10% of cases. However, cumulative pregnancy rates remain low, and outcomes are heterogeneous, including miscarriages and obstetric complications. [16,17]

Urogenital and sexual health

Estrogen deficiency leads to urogenital atrophy and sexual dysfunction, predominantly manifested as vaginal dryness, dyspareunia, and reduced lubrication. Although hormone replacement therapy improves vaginal trophic changes, sexual symptoms often persist in a substantial proportion of patients. [13,16,17]

Cardiometabolic risk

POI is associated with increased cardiovascular risk driven by endothelial dysfunction, metabolic disturbances, and adverse lipid profiles. Despite some variability across studies, overall evidence indicates elevated long-term risk of ischemic heart disease and metabolic syndrome. Hormone therapy may partially improve endothelial function but does not fully eliminate cardiovascular risk. [18-20,25]

Bone health

Early estrogen deprivation leads to reduced bone mineral density, with a high prevalence of osteopenia and osteoporosis, particularly affecting the lumbar spine and femoral neck. The duration of hypoestrogenism is a key determinant of bone loss and fracture risk. [21,22,25]

Autoimmune and endocrine disorders

A substantial proportion of POI cases have an autoimmune etiology. Increased prevalence of autoimmune thyroid disease, adrenal insufficiency, and type 1 diabetes has been consistently reported, with thyroid autoimmunity being the most frequent association. [23]

Neurocognitive and psychological effects

POI is linked to increased risk of cognitive decline and neurodegenerative disorders, particularly in cases of early or surgically induced menopause. Psychologically, POI is associated with high rates of depression, anxiety, and reduced quality of life, reflecting both hormonal and psychosocial mechanisms. [13,24-26]

Mortality

Women with POI demonstrate a modest but consistent increase in all-cause mortality, mainly driven by cardiovascular and metabolic causes, supporting the systemic impact of early ovarian failure. [19,25]

4. Discussion

This narrative review synthesizes current evidence on the association between chronic psychological stress and ovarian reserve, suggesting a biologically plausible but not yet fully defined link with accelerated reproductive aging. Across experimental and observational studies, chronic stress exposure was consistently associated with dysregulation of the hypothalamic-pituitary-ovarian axis, reflected by elevated cortisol levels and adverse reproductive hormone profiles, including increased FSH and decreased AMH. [8-11,14] Collectively, these changes are indicative of diminished ovarian reserve and may reflect accelerated ovarian aging.

Converging evidence from clinical and population-based studies further suggests that women exposed to sustained psychosocial stressors more frequently present with reproductive dysfunction, including primary ovarian insufficiency (POI) and menstrual irregularities. [10,12-15]

However, the current evidence base is predominantly observational and methodologically heterogeneous, with variability in stress assessment tools, limited sample sizes, and inconsistent endocrine endpoints. These limitations preclude causal inference and do not exclude bidirectional relationships, where reproductive dysfunction may also contribute to increased psychological stress.

Despite these constraints, the consistency of findings across biological, clinical, and epidemiological domains supports chronic psychological stress as a potential modifiable contributor to impaired ovarian function. Nevertheless, the underlying mechanisms remain insufficiently understood.

Future well-designed prospective cohort studies with standardized psychometric instruments, objective stress biomarkers, and longitudinal reproductive outcomes are essential to clarify causality, elucidate mechanistic pathways, and inform evidence-based preventive strategies in reproductive medicine.

5. Conclusions

Despite study limitations, the available evidence suggests that cumulative exposure to stressful life events may contribute to the development and progression of primary ovarian insufficiency (POI). This highlights the importance of early identification of women at risk, particularly young women exposed to high academic, occupational, and socioeconomic stress. [10,12-15]

Targeted stress-reduction strategies, including management of work-family burden and improvement of sleep quality, may represent a potentially cost-effective approach to preserving reproductive health. While broader socio-economic stressors and global instability are not always within individual control-although efforts to address them at the societal level remain essential-modifiable factors within daily life should be actively targeted. In patients with POI, addressing psychosocial stress alongside standard medical care, including appropriate hormone replacement therapy and patient education, may improve overall outcomes and quality of life. Further studies are needed to clarify mechanisms, define biomarkers, and guide clinical practice.[9,10,12,15,17]

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