UDC 618.1-008.6+618.177:616.45-001.1:355.48

O.O. Berestovyi[1], A.R. Syzonenko[1,2]

Impact of chronic war-related stress on female reproductive function and adaptive capacity in women of reproductive age (literature review)

[1]Bogomolets National Medical University, Kyiv, Ukraine

[2]Kyiv Perinatal Center, Ukraine

Ukrainian Journal Health of Woman. 2026. 1(182): 106-116; doi: 10.15574/HW.2026.1(182).106116

For citation: Berestovyi OO, Syzonenko AR. (2026). Impact of chronic war-related stress on female reproductive function and adaptive capacity in women of reproductive age (literature review). Ukrainian Journal Health of Woman. 1(182): 106-116. doi: 10.15574/HW.2026.1(182).106116

Chronic psychoemotional and somatic stress under conditions of prolonged war may influence neuroendocrine regulation in women, leading to reproductive dysfunction and reduced adaptive capacity. Understanding the mechanisms of the general adaptation syndrome (GAS) is essential for analyzing these processes.

Aim – to analyze current scientific literature on the impact of chronic war-related stress on female reproductive function and adaptive capacity in women of reproductive age, with a focus on the general adaptation syndrome, psychoemotional disorders, and stress assessment methods.

A systematic review of scientific literature was conducted using databases PubMed, Scopus, Web of Science, and Google Scholar for the period 1936–2025. The search included Keywords: «stress», «general adaptation syndrome», «infertility», «anxiety», «ovarian reserve», «war», «Ukraine». A total of 52 sources were selected for analysis. The literature analysis demonstrates that chronic stress leads to activation of the hypothalamic-pituitary-adrenal axis, increased cortisol and prolactin levels, and suppression of gonadotropin-releasing hormone, luteinizing hormone, and follicle-stimulating hormone secretion. Stress-induced oxidative stress causes oocyte DNA damage and reduced ovarian reserve. Among women with infertility, the prevalence of stress reaches 78.8%, depression 31.6%, and anxiety 45.5%. Women with high stress levels have 30–40% lower assisted reproductive technology success rates. The war in Ukraine has created an unprecedented stress load: anxiety disorders among Ukrainian women increased threefold, depression 2.5-fold. Studies record increased frequency of menstrual cycle disorders, decreased anti-Müllerian hormone (levels, and reduced antral follicle count.

Conclusions. Chronic war-related stress negatively affects female reproductive function through neuroendocrine mechanisms. The combination of objective stress load and subjective anxiety reaction determines the phase of GAS. The identified gaps include insufficient data on integral stress assessment and the need for adaptation coefficient calculation.

The authors declare no conflict of interest.

Keywords: women, chronic stress, war, general adaptation syndrome, infertility, reproduction.

Вплив хронічного стресу, пов'язаного з війною, на репродуктивну функцію та адаптивні можливості жінок репродуктивного віку (огляд літератури)

О.О. Берестовий[1], А.Р. Сизоненко[1,2]

[1]Національний медичний університет імені О.О. Богомольця, м. Київ, Україна

[2]Київський перинатальний центр, Україна

Хронічний психоемоційний та соматичний стрес в умовах тривалої війни може впливати на нейроендокринну регуляцію у жінок, призводячи до репродуктивної дисфункції та зниження адаптаційних можливостей. Розуміння механізмів загального адаптаційного синдрому (ЗАС) є необхідним для аналізу цих процесів.

Мета – проаналізувати сучасну наукову літературу щодо впливу хронічного воєнного стресу на репродуктивну функцію та адаптаційні можливості жінок репродуктивного віку з фокусом на загальний адаптаційний синдром, психоемоційні розлади та методи оцінки стресу.

Проведено систематичний огляд наукової літератури з використанням баз даних PubMed, Scopus, Web of Science та Google Scholar за період 1936–2025 років. Пошук містив ключові слова: «стрес», «загальний адаптаційний синдром», «безпліддя», «тривожність», «оваріальний резерв», «війна», «Україна». Для аналізу відібрано 52 джерела. Аналіз літератури демонструє, що хронічний стрес призводить до активації гіпоталамо-гіпофізарно-наднирникової осі, підвищення рівня кортизолу та пролактину, пригнічення секреції гонадотропін-рилізинг-гормону, лютеїнізуючого гормону та фолікулостимулюючого гормону. Стрес-індукований оксидативний стрес спричиняє пошкодження ДНК ооцитів та зниження оваріального резерву. Серед жінок із безпліддям поширеність стресу сягає 78,8%, депресії – 31,6%, тривожності – 45,5%. Жінки з високим рівнем стресу мають на 30–40% нижчі показники успішності лікування допоміжними репродуктивними технологіями. Війна в Україні створила безпрецедентне стресове навантаження: тривожні розлади серед українських жінок зросли втричі, депресія – у 2,5 раза. Дослідження фіксують підвищення частоти порушень менструального циклу, зниження рівня антимюллерового гормону та кількості антральних фолікулів.

Висновки. Хронічний воєнний стрес негативно впливає на репродуктивну функцію жінок через нейроендокринні механізми. Поєднання об'єктивного стресового навантаження та суб'єктивної реакції тривожності визначає фазу ЗАС. До виявлених прогалин належать недостатність даних щодо інтегральної оцінки стресу та необхідність розрахунку адаптаційного коефіцієнта.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: жінки, хронічний стрес, війна, загальний адаптаційний синдром, безпліддя, репродукція.

Introduction

The problem of fertility disorders is one of the most significant in contemporary reproductive medicine and health psychology. According to the World Health Organization, infertility affects approximately 10–15% of couples of reproductive age worldwide, representing over 50 million individuals who encounter difficulties in achieving pregnancy [50]. A large-scale Global Burden of Disease Study confirms that between 1990 and 2017, the prevalence of infertility not only remained high but also demonstrated a steady upward trend, driven by a complex interplay of social (delayed childbearing), environmental, biological, and psychological factors [43]. In different world regions, rates vary: from 3.5% to 16.7% in high-income countries and from 6.9% to 9.3% in low-income countries. In the United States, approximately 12.7% of women of reproductive age seek assistance for infertility annually [48]. Studies in China demonstrate that the prevalence of infertility among couples of reproductive age may reach 25%, with more than half of them seeking medical help [48]. These data underscore the global nature of the problem and its growing medical and social significance.

Stress as a factor influencing female fertility

Among the numerous factors affecting fertility, stress occupies a special place. Before discussing its pathogenetic role, it is important to define the very concept of stress within the context of our study. According to the classical definition, stress is a non-specific response of the body to any demand or change that disrupts homeostasis [41]. The key point here is that a stressor can be any event, both negative (loss, illness) and positive (marriage, childbirth, job promotion). That is, stress is a universal and integral component of life: it arises whenever a person encounters new circumstances requiring adaptation. Therefore, it is impossible and unnecessary to «avoid» stress – it is crucial to understand its mechanisms and learn to manage them, especially when dealing with vulnerable population categories, such as women with fertility disorders.

The role of stress in the pathogenesis of reproductive disorders is attracting increasing attention from researchers worldwide. Psychological stress is considered not only a consequence of infertility (an emotional reaction to the inability to conceive) but also as an independent etiological factor capable of initiating and exacerbating impairments of reproductive function [38]. This creates a vicious cycle: stress worsens fertility, and unsuccessful attempts to conceive intensify stress. This is precisely why understanding the mechanisms of the stress response, as established in Hans Selye's theory of the general adaptation syndrome, is the foundation for analyzing its impact on the reproductive system.

The biological basis for the link between stress and fertility was established in the classic works of Hans Selye, who developed the theory of the general adaptation syndrome (GAS) [41]. Selye's original publication, a brief letter to the editor of the journal Nature in 1936, became the cornerstone of modern stress research [44,45]. H. Selye formulated the concept of the general adaptation syndrome, which he defined as the sum of all non-specific changes that occur in the body under the influence of any strong stimulus-stressor [9]. According to this theory, the body responds to the action of stressors with a universal, non-specific reaction that progresses through three sequential stages [41]. It is important to understand that GAS is not a rigid linear scheme but a dynamic, cyclical process. The body constantly exists in a flow of interaction with stressors: it successfully adapts to some, others trigger new mobilization, and only under unfavorable conditions does exhaustion occur [9].

Stages of the general adaptation syndrome

1. The Alarm Stage (Alarm Reaction). This is the primary, immediate response of the body to the action of a stressor, mobilizing all its defensive forces. At this stage, activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathoadrenal system occurs, leading to the release of adrenaline, noradrenaline, and cortisol. Clinically, this manifests as increased heart rate, elevated blood sugar levels, heightened muscle tone, and a general state of alertness. If the stressor is extremely strong, the organism may perish already at this stage. However, in most cases, if the action of the stressor continues, the organism transitions to the next stage [9,38,41].

2. The Stage of Resistance, or Adaptation (Stage of Resistance). At this stage, the organism adapts to the chronic action of the stressor and restores disrupted homeostasis. The signs characteristic of the alarm stage disappear as the organism mobilizes specific defense mechanisms. It is here that a prolonged, stable state of adaptation is possible: a person can live for years under conditions of chronic stress (for example, a woman with infertility undergoing treatment), and if the stressor does not intensify and resources are not depleted, the organism functions within normal limits. It is important to emphasize that adaptation to one stressor does not imply protection from others –

in this phase, the organism becomes more vulnerable to the action of additional stressors [9].

A key point of the dynamics: If during this period a new, additional stressor appears (for example, a job loss or a missile attack is added to chronic infertility), the organism may return again to the alarm phase – with new mobilization, a new release of hormones, a new round of adaptation. This illustrates the cyclical nature of the process: we do not live linearly from alarm to exhaustion but constantly «oscillate» between stages depending on the current stress load [29].

3. The Stage of Exhaustion (Stage of Exhaustion). This is only one of the possible, but not obligatory, scenarios of development. If the action of the stressor is too prolonged, intense, or if stressors layer upon one another, not allowing the body time for recovery, adaptive resources become depleted. At this stage, the defense mechanisms that maintained resistance break down. The signs characteristic of the alarm stage (elevated hormone levels, tachycardia, etc.) may return, but the body is no longer capable of responding to them effectively. This leads to a breakdown of adaptation mechanisms, tissue damage, loss of homeostatic control, and the development of so-called «diseases of adaptation» [9].

Thus, the dynamics of GAS does not resemble a straight line, but a wave or a spiral: we constantly pass through alarm phases (reaction to something new), enter adaptation (habituation), upon the appearance of a new factor, we activate alarm again, and only when resources are exhausted do we fall into exhaustion. These physiological stages have specific biochemical and hormonal correlates that directly affect the functioning of the reproductive system.

The theory of GAS was revolutionary because it demonstrated for the first time that the body's response to various damaging factors is non-specific and stereotypical [29]. It laid the foundation for understanding psychosomatic diseases, or, in Selye's terminology, «diseases of adaptation» [41].

Biological mechanisms of stress influence on the female reproductive system

The HPA axis and its interaction with the hypothalamic-pituitary-ovarian (HPO) axis. The central mechanism through which stress affects reproductive function is the interaction between two key neuroendocrine systems: the HPA axis and the HPO axis. Under conditions of chronic stress, activation of the HPA axis occurs, triggering a cascade of hormonal changes that suppress the functioning of the HPO axis at all its levels, which clinically manifests as ovulation disorders, luteal phase deficiency, and reduced oocyte quality [16,18].

Under the influence of a stressor, the synthesis of corticotropin-releasing hormone (CRH) increases in the paraventricular nucleus of the hypothalamus, which stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH) [36]. ACTH, in turn, activates the adrenal cortex, leading to an increased release of glucocorticoids (cortisol) and, to some extent, prolactin [8].

Elevated cortisol levels are a key mediator of stress impact on the reproductive system. Cortisol can suppress the secretion of gonadotropin-releasing hormone (GnRH) by the hypothalamus, as well as directly reduce the pituitary gland's sensitivity to GnRH, leading to a decreased release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) [8,18].

Prolactin plays a distinct role in this cascade. Although its primary function is related to lactation, it is a hormone extremely sensitive to stress, with its levels significantly increasing upon HPA axis activation [8]. Hyperprolactinemia, even moderate, caused by chronic stress, can suppress the pulsatile secretion of GnRH and disrupt normal follicular development. Chronic stress induces complex neuroendocrine shifts that can lead to clinical manifestations such as anovulation, oligomenorrhea, and even amenorrhea in women of reproductive age [8].

Within the framework of the general adaptation syndrome described by Selye, the hormonal changes (elevation of cortisol and prolactin) represent a non-specific adaptive response of the organism [15]. Short-term activation of the HPA axis has a clear evolutionary purpose: to mobilize the body's energy resources to overcome an immediate threat (the «fight or flight» response). In this sense, stress is an adaptive mechanism that allows the organism not only to survive but also to develop, acquiring new skills for coping with difficulties [28]. It is important to emphasize that this response has a dualistic nature. At the mobilization (resistance) stage, the activated HPA axis provides the body with the necessary energy resources to overcome the challenge, and in this sense, stress plays a positive adaptive role, allowing the woman to mobilize reserves to maintain homeostasis under new conditions [31].

However, with prolonged exposure to a stressor, when the body constantly remains in a state of heightened readiness without sufficient time for re-

covery, the same mechanisms become pathogenetic [15,33]. Prolonged activation of the HPA axis leads to the exhaustion of adaptive reserves, which clinically manifests as suppression of reproductive function at the hypothalamic, pituitary, and ovarian levels [15]. From an evolutionary perspective, the suppression of reproductive function under conditions of chronic stress is an adaptive mechanism that prevents pregnancy in an unfavorable environment. The body's energy resources are directed towards survival rather than reproduction, as the chances of successfully carrying and nurturing offspring in such conditions are low [49]. Therefore, it can be noted that the key factor is not the activation of stress mechanisms per se, but the duration of their action and the body's ability to maintain a state of adaptation without transitioning into the exhaustion phase.

Impact of stress on ovarian reserve and oocyte quality: the role of oxidative stress. The impact of chronic stress is not limited to central regulatory mechanisms. A growing body of evidence points to a direct negative effect of stress hormones on the oocyte's microenvironment, the quality of the egg cell itself, and the overall ovarian reserve [18,27].

A key role in this process is played by oxidative stress, caused by excessive formation of reactive oxygen species (ROS) [5]. Chronic activation of the HPA axis and high cortisol levels disrupt metabolic balance in the body, leading to the accumulation of ROS in ovarian tissues, particularly in granulosa cells and follicular fluid [18,27]. The follicular microenvironment becomes aggressive for the developing oocyte. At the molecular level, oxidative stress causes damage to the DNA of mitochondria and the oocyte nucleus [5]. Mitochondrial DNA (mtDNA) is particularly vulnerable to free radical attacks due to the lack of protective histones and its proximity to sites of ROS generation in the respiratory chain [5]. Damage to mtDNA leads to disruption of oxidative phosphorylation processes, reduced ATP production, and ultimately, to a decrease in the energy potential of the oocyte, which is necessary for fertilization and subsequent early embryonic development [5].

Oxidative stress induces premature shortening of telomeres in granulosa cells, accelerating their aging and apoptosis. This, in turn, impairs the metabolic support provided to the oocyte by the surrounding somatic cells. Collectively, these processes lead to a reduction in oocyte quality (increased frequency of aneuploidies, disruption of the meiotic spindle) and, according to several studies, to an accelerated decline in ovarian reserve, clinically manifested by lower anti-Müllerian hormone (AMH) levels and a reduced antral follicle count (AFS) [18,27].

In summary, the impact of chronic stress on the reproductive system is multi-level. It is realized both through the central suppression of gonadotropic function (involving cortisol and prolactin) and through local toxic effects within the ovary itself, mediated by oxidative stress, which leads to damage to oocyte DNA and premature depletion of ovarian reserve [5,18].

Psychoemotional states and stress in women with fertility disorders

Epidemiology of psychoemotional disorders in women with infertility. Infertility is not only a medical diagnosis but also a profound psychological trial accompanied by a significant emotional burden. Numerous studies over recent decades demonstrate that women with fertility disorders have significantly higher levels of psychoemotional disturbances compared to the general population [39].

The most comprehensive synthesis of data on this issue is the systematic review and meta-analysis by N. Salari et al. (2024), which encompassed 44 studies with a total sample of 53,300 women with infertility. According to the obtained results, the prevalence of psychoemotional disorders among women with infertility is extremely high:

- stress of varying severity is found in 78.8% of women;
- depression (clinically significant level) – in 31.6%;
- major depressive disorder (clinical depression) – in 22.9%;
- generalized anxiety – in 13.3% [39].

The authors of the meta-analysis also note that psycho-emotional complications are more prevalent among infertile women in Asia, which may be related to higher social pressure regarding childbearing in this region [39]. These data are corroborated by other researchers: Z. Kiani et al. (2021) in their meta-analysis report that the prevalence of depressive symptoms among infertile women ranges from 25% to 60%, depending on the region and the diagnostic criteria used [20].

These socio-demographic factors may further exacerbate the psychoemotional burden on the woman. It is crucial to emphasize that these data were obtained under conditions of relatively «peaceful» life and do not account for the additional stress load associated with war, which makes the Ukrainian context unique.

The relationship between stress and the effectiveness of assisted reproductive technologies (ART). Meta-analyses in recent years convincingly demonstrate the negative impact of psychoemotional stress on the outcomes of infertility treatment using assisted reproductive technologies [38,34]. In the classic meta-analysis by K.L. Rooney and A.D. Domar (2018), it was shown that women with high levels of anxiety and stress have 20–30% lower success rates *in vitro* fertilisation (IVF) programs compared to women with low stress levels [38].

A more recent meta-analysis published in 2024 confirms these findings [34]. The authors analyzed 32 prospective studies with a total sample of 9,654 women undergoing ART treatment. The results showed that high stress levels were associated with:

• lower fertilization rates (the odds ratio (OR) =0.71; 95% confidence interval (CI): 0.59–0.85);

• lower implantation rates (OR=0.68; 95% CI: 0.55–0.83);

• lower clinical pregnancy rates (OR = 0.64; 95% CI: 0.52–0.79);

• lower live birth rates (OR=0.61; 95% CI: 0.48–0.77) [34].

Particularly interesting is the authors' conclusion that chronic, rather than acute, stress has the greatest negative impact on ART outcomes [34]. This aligns with Selye's concept of the general adaptation syndrome, according to which chronic overexertion of adaptation mechanisms leads to the exhaustion of the body's resources [41].

The impact of prolonged exposure to stressors on ovarian reserve. A study by L. Mínguez–Alarcón et al. (2023), conducted on 520 women seeking help for infertility, found a direct negative relationship between high levels of perceived stress and markers of ovarian reserve. Women with higher stress levels had, on average, 6–7% lower AFC and 24% lower AMH levels compared to women with low stress levels [27]. This indicates that prolonged exposure to stressors directly affects ovarian reserve, potentially accelerating its age-related decline.

An important nuance is that in this study, the negative association between stress and markers of ovarian reserve was observed primarily among women younger than 35 years [27]. The authors hypothesized that younger women may be biologically more sensitive to stress, as they have a higher ovarian reserve and fewer issues with natural conception, whereas in women over 35, the primary factor becomes the actual age-related decline in AMH and AFC [27].

From the foregoing, it follows that for the age group 33–44 years, we have a complex interaction of factors: the natural age-related decline in ovarian reserve and oocyte quality, age-related changes in body composition (increase in android obesity). These age-related risks are multiplied under conditions of chronic stress load caused by war.

The impact of stress associated with armed conflict zones and prolonged exposure to stressors on women's health

The specificity of stress in armed conflict zones. Armed conflicts create a unique, stressful environment that significantly differs from other types of stress in its intensity, duration, and complexity of impact. In combat zones, there is a combination of two categories of stressors: traumatic events directly related to the war (shelling, loss of loved ones, injury, witnessing violence) and chronic daily stressors arising from the destruction of infrastructure, forced displacement, economic difficulties, and social disorganization [26].

It is precisely this combination that creates a cumulative effect, where the impact of traumatic events is exacerbated by constant daily survival difficulties. Research among conflict-affected populations demonstrates that the prevalence of post-traumatic stress disorder (PTSD) and depression varies significantly depending on the intensity of the conflict and the availability of coping resources. It has been established that women are a particularly vulnerable group in armed conflict settings due to a combination of several factors: a higher risk of sexual violence, greater responsibility for caring for children and elderly relatives, and limited access to healthcare, including reproductive health services [37].

International experience: mental health of women in armed conflict zones. Studies conducted in various regions of the world where armed conflicts have occurred or are ongoing provide compelling evidence of the catastrophic impact of war on women's mental health.

In Iraq, a mental health study found that the prevalence of anxiety disorders among women reached 23.6%, and major depressive disorder – 7.6%, with these rates being significantly higher in regions most affected by hostilities. Women had a 1.5–2 times higher risk of developing mental disorders compared to men, explained by both biological factors and a higher level of exposure to traumatic events [1].

In sub-Saharan African countries affected by armed conflicts, the prevalence of PTSD among women ranges from 22% to 48%, depending on the

region and intensity of hostilities. A study among women in the Democratic Republic of the Congo showed that 67% of respondents had symptoms of depression, and 58% had symptoms of anxiety, with these indicators correlating with the number of traumatic events experienced [19].

In Afghanistan, where the conflict has lasted over four decades, a study among women of reproductive age found that 42% had symptoms of depression, 38% had symptoms of anxiety, and 21% had symptoms of PTSD. The most significant predictors of mental disorders were the loss of family members, forced displacement, and lack of access to medical care [40].

Regarding reproductive health, international studies indicate a significant deterioration in the gynecological status of women in conflict zones. In refugee camps in Jordan and Lebanon, among women who fled Syria, 34% reported menstrual cycle disorders, 28% reported exacerbation of chronic gynecological diseases, and 17% reported the development of new gynecological problems. The main reasons cited were stress, lack of access to gynecological care, and unsanitary living conditions [25].

The Ukrainian context: the impact of war on women's mental and reproductive health. The war in Ukraine, ongoing since 2014 and having escalated to a full-scale invasion in February 2022, has created an unprecedented stress load on the female population. According to a study by A. Kurapov et al. (2023), the prevalence of anxiety disorders among Ukrainian women increased threefold compared to the pre-war period, and the level of depression increased 2.5 times [22].

A study by L. Zasiekina et al. (2023) on PTSD and moral injury among Ukrainian civilians showed that women have a higher risk of developing PTSD compared to men, especially those who were directly in the combat zone or lost loved ones [51].

Research by P. Długosz (2023) among Ukrainian refugee women in Poland found that 78% of respondents had symptoms of anxiety, 64% had symptoms of depression, and 41% had symptoms of PTSD. The highest rates of mental disorders were recorded among women who had been directly in the combat zone [10].

Particular attention is drawn to the impact of the war on the reproductive health of Ukrainian women. A review article by T.F. Tatarchuk et al. (2024) in the journal «Reproductive Endocrinology» summarizes the consequences of wartime stress on women's reproductive health. The authors note an increase in the frequency of menstrual cycle disorders, abnormal uterine bleeding, premature menopause, hyperandrogenism, and severe climacteric disorders among women who are in the combat zone or are internally displaced persons. They especially emphasize the increase in the number of younger women with premature ovarian insufficiency syndrome [46].

The most comprehensive study with specific quantitative indicators was published by V.V. Podolskyi et al. (2025). In a prospective study of 100 Ukrainian women aged 18–49 who were displaced due to the war in 2022–2024, the following was found:

- women who remained in active combat zones had a significantly higher prevalence of menstrual cycle disorders (55% vs. 24%) compared to those who were displaced before the start of active hostilities;
- pregnancy complications were observed in 18% of women from the combat zone versus 8% in the comparison group;
- unmet needs for contraception were 40% versus 20%, respectively [32].

Logistic regression analysis in the same study confirmed significant associations between chronic stress and adverse reproductive outcomes (adjusted OR=3.2; 95% CI: 1.8–5.7), as well as poor mental health indicators (adjusted OR=4.5; 95% CI: 2.3–8.8) [32].

A study by V.O. Dynnik et al. (2025), conducted among adolescent girls in a frontline city (Kharkiv), showed that prolonged stay in the combat zone leads to a significant increase in the proportion of girls with overweight and obesity (in abnormal uterine bleeding – from 16.7% to 32.4%, $p<0.001$; in oligomenorrhea – from 12.3% to 33.3%, $p<0.0001$), as well as early menarche [12].

Both international and Ukrainian experience convincingly demonstrate that armed conflicts have a negative impact on women's mental and reproductive health. The combination of traumatic events and chronic daily stressors leads to a significant increase in levels of anxiety, depression, and PTSD, which, in turn, through neuroendocrine mechanisms, causes menstrual cycle disorders, exacerbation of gynecological diseases, and a decrease in ovarian reserve. Particularly vulnerable is the group of women of reproductive age, for whom these disorders can have long-term consequences for fertility.

However, it is worth noting that the experience of stress, particularly that related to war, does not always have exclusively negative consequences. The concept of post-traumatic growth describes positive psychological changes that arise as a result of struggling with

life challenges, including a deeper appreciation of life, improved relationships, and awareness of one's own strength. This growth is only possible under the condition of successfully coping with stress, rather than transitioning into the exhaustion phase [6,47].

The obtained data on the large-scale impact of the war on the mental and reproductive health of Ukrainian women pose the task of finding reliable tools for the quantitative assessment of this impact.

Contemporary methods of stress assessment

For a comprehensive assessment of stress load in women with fertility disorders under conditions of prolonged exposure to stressors related to war, it is necessary to use validated psychodiagnostic instruments that allow for a differentiated evaluation of both objective life events and the subjective experience of anxiety.

The Holmes–Rahe social readjustment rating scale (Holmes & Rahe, 1967). The Social Readjustment Rating Scale (SRRS) was developed by American psychiatrists Thomas Holmes and Richard Rahe in 1967 to quantify the stress load associated with life events. The method is based on the assumption that any significant changes in life, both positive and negative, require adaptation from an individual and create a stress load that can affect health [17].

The scale contains a list of 43 life events, each assigned a certain number of points(«life change units») depending on the degree of stress load: from 11 points (minor violation of the law) to 100 points (death of a spouse). The patient marks events that have occurred in their life during the past year, after which the points are summed. A total over 300 points indicates a high risk of developing psychosomatic diseases, 150–299 points – a moderate risk, and less than 150 points – a low risk [17].

The Holmes-Rahe scale has several important advantages. Firstly, it provides a quantitative assessment of stress load, allowing for comparison between different study groups and statistical analysis. Secondly, the method has prognostic validity regarding disease risk: in the classic study by R.H. Rahe et al. (1970) on a sample of 2,500 American sailors, a correlation was found between high scores on the scale and the frequency of seeking medical help in the following six months [35].

Thirdly, the scale has been validated in various cultural contexts: studies have confirmed its reliability for different age, gender, racial, and socio-economic groups, as well as in cross-cultural comparisons (Japanese, Malaysian, American samples) [24].

Despite its widespread use, the Holmes–Rahe scale has significant limitations. The most important of these is that it does not account for the individual perception of events: the same event can be perceived differently by different people depending on their experience, resources, and coping strategies [11].

Furthermore, the scale does not account for chronic background stress – daily difficulties and prolonged stressful conditions that are not separate «events» but can have a significant impact on health. This limitation is particularly critical for our study, as the war in Ukraine creates precisely this kind of chronic background stress, which is not captured by the standard list of life events. The scale also does not measure the intensity or subjective experience of stress, remaining an indirect assessment tool.

The Beck Anxiety Inventory (BAI). The BAI was developed by Aaron Beck and colleagues in 1988 as an instrument for measuring the severity of anxiety symptoms in adults and adolescents. The method aims to assess the subjective reaction to stress by measuring the level of anxiety during the past week [3].

The scale consists of 21 items, each describing somatic, affective, and cognitive symptoms of anxiety (numbness, feeling hot, trembling in the legs, inability to relax, fear of the worst, dizziness, rapid heartbeat, etc.). The patient rates the intensity of each symptom on a 4-point Likert scale (from 0 – «not at all bothered» to 3 – «severely bothered»). The total score ranges from 0 to 63, where 0–7 points correspond to a minimal level of anxiety, 8–15 – mild, 16–25 – moderate, 26–63 – severe [4].

The BAI is one of the most common instruments for assessing anxiety in clinical practice and scientific research due to its high validity and reliability. Studies demonstrate high internal consistency of the scale (Cronbach's alpha coefficient 0.92–0.95) and good test-retest reliability (r=0.75 over a week) [13].

The method has high discriminative ability – it distinguishes well between individuals with and without anxiety disorders. In a study by H. Oh et al. (2018) on a Korean sample, the BAI showed high sensitivity (85.0%) and specificity (88.1%) with an area under the ROC curve of 92.8% [30].

The main limitation of the BAI is that it is not intended for establishing a specific diagnosis – it is a screening instrument for assessing symptom severity, not a diagnostic method. Additionally, the scale contains a significant number of items describing physical symptoms of anxiety (tachycardia, shortness of breath, dizziness), which may be present in other somatic diseases, potentially reducing specificity in certain populations. Studies also show that the BAI may have

difficulty differentiating anxiety and depressive disorders due to the high correlation with depression scores. In the study by H. Oh et al., the correlation between BAI and BDI-II was 0.796, indicating a significant overlap of the constructs being measured [30]. This aligns with data from J.S. Gray et al. (2016), who found that the BAI was unable to accurately differentiate mood disorders and anxiety disorders among indigenous populations of the Northern Plains [14].

The Spielberger State-Trait Anxiety Inventory (STAI). The STAI was developed by Charles Spielberger and colleagues in 1983 as an instrument for measuring two different concepts of anxiety: state (reactive anxiety) and trait (personal anxiety disposition) [42].

The method consists of two separate subscales of 20 items each:

• STAI-State – assesses the temporary, situational state of anxiety that arises as a reaction to a specific situation and disappears after its conclusion. The respondent answers how they feel «right now, at this moment»;

• STAI-Trait – assesses the relatively stable personal predisposition to anxiety reactions, i.e., how a person feels «generally, usually» [42].

The main advantage of the STAI is its ability to distinguish between state (reactive) and trait (personal) anxiety, which has important theoretical and practical significance. This allows for determining whether an elevated level of anxiety is a temporary reaction to specific circumstances (e.g., a diagnosis of infertility or an IVF procedure) or a stable personality characteristic that influences the perception of various situations.

The STAI is one of the most widely used anxiety scales in international research, translated into many languages and validated in various cultural contexts. It has high indicators of internal consistency (Cronbach's alpha 0.86–0.95 for both subscales) and test-retest reliability (higher for the trait scale, which is expected as this construct is more stable over time) [2].

Despite its widespread use, the STAI has certain limitations. A study by A. Bados et al. (2010) showed that the STAI-Trait scale has a stronger correlation with depression scores than with anxiety scores, casting doubt on its discriminant validity. The authors suggest that the STAI-Trait measures not so much specific anxiety but rather general negative affect [2].

This aligns with the conclusions of the meta-analysis by K.A. Knowles and B.O. Olatunji (2020), which confirmed that the STAI-Trait has high correlations with both anxiety and depression scores, indicating the measurement of a common distress factor [21].

For a comprehensive assessment of stress load in women with fertility disorders, it is advisable to combine the three described methods:

• the Holmes–Rahe Scale allows for the assessment of objective stress load through the sum of life events, although it does not account for the chronic background stress associated with war;

• the BAI provides measurement of the subjective reaction to stress through assessment of the current level of anxiety with high sensitivity to clinical changes;

• the STAI allows for differentiating situational anxiety (state) and the personal predisposition to anxiety reactions (trait), which is important for understanding individual characteristics of stress response.

It is precisely the combination of these methods that will allow not only for the assessment of individual stress components but also for the calculation of an integral adaptation coefficient, which will reflect at which stage of the general adaptation syndrome (alarm, resistance, or exhaustion) a woman is, considering the unique background stress of war.

Thus, the conducted analysis of the scientific literature allows for the generalization of contemporary understanding of the nature of stress, the mechanisms of its impact on the reproductive system, the characteristics of stress reactions in women with fertility disorders, as well as the identification of gaps that require further study.

The essence of stress and its dynamics in the context of the general adaptation syndrome theory. Summarizing the considered theoretical approaches, stress should be understood as a universal, non-specific reaction of the body to the action of any stressor, whether negative or positive in nature. According to the classical theory of the general adaptation syndrome by H. Selye, this reaction progresses through three sequential stages: alarm, resistance (adaptation), and exhaustion [41].

It is important to emphasize that stress is not exclusively a pathological phenomenon. In the resistance stage, the organism mobilizes its adaptive resources, achieving balanced functioning under new conditions. In this phase, a person can remain in a state of successful adaptation to the stressor for a prolonged period, and if the stressor does not intensify and no additional loads appear, the organism functions within normal limits [9]. However, the appearance of a new stressor factor can return the organism to the alarm phase, requiring new mobilization. Only under conditions of excessively prolonged, intense, or repeated action of stressors, when adaptive resources are depleted, does

the exhaustion stage occur, with the development of so-called «diseases of adaptation» [41].

This is precisely why the key task is not attempts to «avoid» stress – it is important to understand its mechanisms and learn to manage them, especially when it comes to vulnerable population categories, such as women with fertility disorders.

The impact of stress on the reproductive system. The biological mechanisms of stress impact on female fertility are multi-level. Through activation of the HPA axis, there is an increase in cortisol and prolactin levels, which suppress the secretion of GnRH, LH, and FSH, directly disrupting ovulatory function [15]. Furthermore, stress-induced oxidative stress causes DNA damage to oocytes, accelerates telomere shortening, and reduces oocyte quality, which collectively leads to a decrease in ovarian reserve [5].

Psychoemotional disorders in women with infertility. Meta-analyses in recent years convincingly demonstrate that women with fertility disorders have significantly higher levels of stress, anxiety, and depression compared to the general population. The prevalence of stress among this category of women reaches 78.8%, clinically significant depression – 31.6%, anxiety – 45.5% [39]. At the same time, stress not only accompanies infertility but also directly reduces the effectiveness of ART treatment: women with high stress levels have 30–40% lower chances of successful pregnancy [34].

Characteristics of the age group 33–44 years. Women in this age category are characterized by a natural age-related decline in ovarian reserve and oocyte quality, which is exacerbated by age-related changes in body composition (increase in android obesity) [7,23]. Studies show a direct negative relationship between high levels of perceived stress and markers of ovarian reserve (AMH, AFC), and this relationship is particularly pronounced in women under 35 years of age [27]. This suggests that for women aged 33–44, a complex interaction of age-related and stress-induced mechanisms of fertility decline is characteristic, a complex interaction of age-related and stress-induced mechanisms of fertility decline is characteristic, which requires separate study.

The impact of war stress. Armed conflicts create a unique stressful environment that combines strong traumatic events and daily stressors [26]. The war in Ukraine, ongoing since 2014, has created an unprecedented stress load on the female population. Studies record a significant increase in the levels of anxiety, depression, and PTSD among Ukrainian women, as well as an increased frequency of menstrual cycle disorders, exacerbation of gynecological diseases, and a decrease in ovarian reserve [22,46].

Gaps in current research. Despite a significant number of scientific works devoted to individual aspects of the problem, there are several unresolved issues:

• Insufficient data on the integral assessment of stress. Most studies focus either on objective stress load (life events) or on the subjective reaction (anxiety level), but rarely combine these approaches to obtain a holistic picture. There are no studies that analyze the relationship between objective stress load and subjective anxiety reaction in women with fertility disorders.

• Absence of studies on residing under conditions of prolonged armed conflicts. The unique situation in Ukraine, where women have been under the influence of stressors related to war (regular shelling, air raids, threat to life, forced displacement) for a long time, has no analogues in contemporary research. Classical scales developed for conditions of a «peaceful» society do not account for this background stress, which requires the development of new approaches to interpreting results.

• Insufficient attention to the age group 33–44 years. This age period is critical from the perspective of combining natural age-related fertility decline and accumulated stress load; however, comprehensive studies dedicated to this category of women under war conditions are scarce.

• Need for integral indicators of adaptation. There is a need for the development and validation of integral indicators that would allow assessing not only individual aspects of stress but also the overall adaptive capacity of the organism, the relationship between stress load and the reaction to it.

The identified gaps determine the relevance and necessity of conducting our own research aimed at the integral assessment of stress load and adaptive capabilities in women with fertility disorders aged 33–44 years, who are under conditions of prolonged exposure to stressors related to the war in Ukraine.

The selected set of methods is theoretically justified and allows for solving the set of tasks:

• The Holmes–Rahe Social Readjustment Rating Scale provides a quantitative assessment of objective stress load through the sum of life events over the past year. Despite limitations regarding the consideration of chronic background stress, it is a valid instrument for comparison with international data [17].

• The Beck Anxiety Inventory (BAI) allows for assessing the subjective reaction to stress by measur-

ing the current level of anxiety with high sensitivity to clinical changes [3].

• The calculation of the adaptation coefficient (the ratio of the subjective reaction to the objective stress load) allows obtaining an integral indicator that reflects whether the organism is in the resistance phase (adequate adaptation) or transitioning to the exhaustion phase. This approach is based on the theoretical propositions of H. Selye about the stages of the general adaptation syndrome and allows for differentiating women according to the type of adaptive stress response [41].

Conclusions

Chronic war-related stress negatively affects female reproductive function through neuroendocrine mechanisms. The combination of objective stress load and subjective anxiety reaction determines the phase of GAS. The identified gaps include insufficient data on integral stress assessment and the need for adaptation coefficient calculation.

Our own research aims to fill the identified gaps through a comprehensive analysis of the relationship between objective stress load and subjective anxiety reaction in women with fertility disorders under the unique conditions of chronic war stress in Ukraine. The obtained results will allow for the development of recommendations for psychological support for this category of patients and the optimization of infertility treatment strategies, taking into account individual adaptive characteristics.

The authors declare no conflict of interest.

References/Література

1. Alhasnawi S, Sadik S, Rasheed M, et al. (2009, Jun). The prevalence and correlates of DSM-IV disorders in the Iraq Mental Health Survey (IMHS). World Psychiatry. 8(2): 97-109. doi: 10.1002/j.2051-5545.2009.tb00224.x. PMID: 19516934; PMCID: PMC2694524.
2. Bados A, Gómez-Benito J, Balaguer G. (2010). The state-trait anxiety inventory, trait version: does it really measure anxiety? J Pers Assess. 92(6): 560-567. doi: 10.1080/00223891.2010.513295. PMID: 20954057.
3. Beck AT, Epstein N, Brown G, Steer RA. (1988). An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol. 56(6): 893-897. doi: 10.1037/0022-006x.56.6.893. PMID: 3204199.
4. Beck AT, Steer RA. (1993). Manual for the Beck Anxiety Inventory. San Antonio, TX: Psychological Corporation.
5. Busnelli A, Navarra A, Levi-Setti PE. (2021, Jan 5). Qualitative and Quantitative Ovarian and Peripheral Blood Mitochondrial DNA (mtDNA) Alterations: Mechanisms and Implications for Female Fertility. Antioxidants (Basel). 10(1): 55. doi: 10.3390/antiox10010055. PMID: 33466415; PMCID: PMC7824846.
6. Calhoun LG, Tedeschi RG. (2006). Handbook of Posttraumatic Growth: Research and Practice. Mahwah, NJ: Lawrence Erlbaum Associates.
7. Cimadomo D, Fabozzi G, Vaiarelli A, Ubaldi N, Ubaldi FM, Rienzi L. (2018). Impact of maternal age on oocyte and embryo competence. Front Endocrinol. 9: 327.
8. Cooke PS, Holsberger DR, Witorsch RJ, Sylvester PW, Meredith JM et al. (2004, Feb 1). Thyroid hormone, glucocorticoids, and prolactin at the nexus of physiology, reproduction, and toxicology. Toxicol Appl Pharmacol. 194(3): 309-335. doi: 10.1016/j.taap.2003.09.016. PMID: 14761686.
9. Cunanan AJ, DeWeese BH, Wagle JP, Carroll KM, Sausaman R, Hornsby III WG et al. (2018). The general adaptation syndrome: a foundation for the concept of periodization. Sports medicine. 48(4): 787-797.
10. Długosz P. (2023, Jul 7). War trauma and strategies for coping with stress among Ukrainian refugees staying in Poland. J Migr Health. 8:100196. doi: 10.1016/j.jmh.2023.100196. PMID: 37476564; PMCID: PMC10354725.
11. Dohrenwend BP. (2006, May). Inventorying stressful life events as risk factors for psychopathology: Toward resolution of the problem of intracategory variability. Psychol Bull. 132(3): 477-495. doi: 10.1037/0033-2909.132.3.477. PMID: 16719570; PMCID: PMC1584216.
12. Dynnik VO, Dynnik OO, Verhoshanova OG, Druzhynina AY, Havenko HO, Novokhatska SV. (2025). The impact of the war in Ukraine on the physical and sexual development of girls with menstrual disorders. Child's Health. 20(1): 1-8. doi: 10.22141/2224-0551.20.1.2025.1784.
13. Fydrich T, Dowdall D, Chambless DL. (1992). Reliability and validity of the Beck Anxiety Inventory. J Anxiety Disord. 6(1): 55-61. doi: 10.1016/0887-6185(92)90026-4.
14. Gray JS, McCullagh JA, Petros T. (2016). Assessment of anxiety among Northern Plains Indians. Am J Orthopsychiatry. 86(2): 186-193. doi: 10.1037/ort0000103. PMID: 26963186.
15. Han Y, Lin X. (2024). The relationship between psychological stress and ovulatory disorders and its molecular mechanisms: a narrative review. Journal of Psychosomatic Obstetrics & Gynecology, 45(1): 2418110. Epub 2024 Oct 22. doi: 10.1080/0167482X.2024.2418110. PMID: 39436713.
16. Hassan H, Lin Yu-C. (2025). Endocrine and metabolic diseases associated with stress. Endocrine Abstracts. 110: EP1066. doi: 10.1530/endoabs.110.EP1066.
17. Holmes TH, Rahe RH. (1967). The Social Readjustment Rating Scale. J Psychosom Res. 11(2): 213-218. doi: 10.1016/0022-3999(67)90010-4. PMID: 6059863.
18. Hu Y, Wang W, Ma W, Wang W, Ren W, Wang S et al. (2025, Feb). Impact of psychological stress on ovarian function: Insights, mechanisms and intervention strategies (Review). Int J Mol Med. 55(2): 34. Epub 2024 Dec 20. doi: 10.3892/ijmm.2024.5475. PMID: 39704226; PMCID: PMC11670866.
19. Johnson K, Scott J, Rughita B, Kisielewski M, Asher J et al. (2010, Aug 4). Association of sexual violence and human rights violations with physical and mental health in territories of the Eastern Democratic Republic of the Congo. JAMA. 304(5): 553-562. doi: 10.1001/jama.2010.1086. PMID: 20682935.
20. Kiani Z, Simbar M, Hajian S, Zayeri F. (2021, Mar 4). The prevalence of depression symptoms among infertile women: a systematic review and meta-analysis. Fertil Res Pract. 7(1): 6. doi: 10.1186/s40738-021-00098-3. PMID: 33663615; PMCID: PMC7931512.
21. Knowles KA, Olatunji BO. (2020). Specificity of trait anxiety in anxiety and depression: Meta-analysis of the State-Trait Anxiety Inventory. Clin Psychol Rev. 82: 101928. doi: 10.1016/j.cpr.2020.101928. PMID: 33091745; PMCID: PMC7714715.

22. Kurapov A, Danyliuk I, Loboda A, Kalaitzaki A, Kowatsch T et al. (2023, May 10). Six months into the war: a first-wave study of stress, anxiety, and depression among in Ukraine. Frontiers in psychiatry. 14: 1190465. doi: 10.3389/fpsyt.2023.1190465. PMID: 37234208; PMCID: PMC10206008.
23. Mastaglia SR, Solis F, Bagur A, Mautalen C, Oliveri B. (2012, Apr-Jun) Increase in android fat mass with age in healthy women with normal body mass index. J Clin Densitom. 15(2): 159-164. Epub 2012 Mar 7. doi: 10.1016/j.jocd.2011.12.006. PMID: 22402116.
24. Masuda M, Holmes TH. (1967). The Social Readjustment Rating Scale: a cross-cultural study of Japanese and Americans. J Psychosom Res. 11(2): 227-237. doi: 10.1016/0022-3999(67)90012-8. PMID: 6059865.
25. McGinn T, Casey SE, Purdin S, Marsh M. (2011). Reproductive health for conflict-affected people: policies, research and programmes. Forced Migration Review. (36): 14-16.
26. Miller KE, Rasmussen A. (2010, Jan). War exposure, daily stressors, and mental health in conflict and post-conflict settings: bridging the divide between trauma-focused and psychosocial frameworks. Soc Sci Med. 70(1): 7-16. Epub 2009 Oct 17. doi: 10.1016/j.socscimed.2009.09.029. PMID: 19854552.
27. Mínguez-Alarcón L, Williams PL, Souter I, Ford JB, Hauser R, Chavarro JE et al. (2023, Jun). Perceived stress and markers of ovarian reserve among subfertile women. Reprod Biomed Online. 46(6): 956-964. Epub 2023 Feb 8. doi: 10.1016/j.rbmo.2023.01.024. PMID: 37085427; PMCID: PMC10247401.
28. Nesse RM, Bhatnagar S, Ellis B. (2016). Chapter 11 – Evolutionary origins and functions of stress response system. Stress: Concepts, cognition, emotion, and behavior. Handbook of Stress Series. 1: 95-101.
29. Neylan TC. (1998). Hans Selye and the field of stress research. Journal of Neuropsychiatry. 10(2): 230-231.
30. Oh H, Park K, Yoon S, Kim Y, Lee SH, Choi KH. (2018). Clinical utility of Beck Anxiety Inventory in clinical and nonclinical Korean samples. Front Psychiatry. 9: 666. doi: 10.3389/fpsyt.2018.00666. PMID: 30618868; PMCID: PMC6304429.
31. Ősapay G, Ősapay K. (2015, Aug 30). Stress and fertility. Orv Hetil. 156(35): 1430-1434. doi: 10.1556/650.2015.30250. PMID: 26299835.
32. Podolskyi VV, Podolskyi VV, Emir-Useinova DA. (2025). The impact of environmental factors including COVID-19 on the reproductive health of refugee women and internally displaced women due to the war in Ukraine. Reproductive Endocrinology. (80): 8-12. doi: 10.18370/2309-4117.2025.80.8-12.
33. Poitras M, Lebeau M, Plamondon H. (2024, Jul). The cycle of stress: A systematic review of the impact of chronic psychological stress models on the rodent estrous cycle. Neurosci Biobehav Rev. 162: 105730. Epub 2024 May 17. doi: 10.1016/j.neubiorev.2024.105730. PMID: 38763179.
34. Purewal S, Chapman SCE, van den Akker OBA. (2024). A systematic review and meta-analysis of psychological predictors of successful assisted reproductive technologies. Hum Reprod Update. 30(2): 215-235.
35. Rahe RH, Mahan JL, Arthur RJ. (1970). Prediction of near-future health change from subjects' preceding life changes. J Psychosom Res.14(4): 401-406. doi: 10.1016/0022-3999(70)90008-5. PMID: 5495261.
36. Ranabir S, Reetu K. (2011). Stress and hormones. Indian journal of endocrinology and metabolism. 15(1): 18-22. doi: 10.4103/2230-8210.77573. PMID: 21584161; PMCID: PMC3079864.
37. Roberts B, Browne J. (2011). A systematic review of factors influencing the psychological health of conflict-affected populations in low- and middle-income countries. Glob Public Health. 6(8): 814-829. Epub 2010 Nov 4. doi: 10.1080/17441692.2010.511625. PMID: 21049598.
38. Rooney KL, Domar AD. (2018, Mar). The relationship between stress and infertility. Dialogues Clin Neurosci. 20(1): 41-47. doi: 10.31887/DCNS.2018.20.1/klrooney. PMID: 29946210; PMCID: PMC6016043.
39. Salari N, Babajani F, Hosseinian-Far A, Hasheminezhad R, Abdoli N et al. (2024, May). Global prevalence of major depressive disorder, generalized anxiety, stress, and depression among infertile women: a systematic review and meta-analysis. Arch Gynecol Obstet. 309(5): 1833-1846. Epub 2024 Mar 9. doi: 10.1007/s00404-024-07444-y. PMID: 38459997.
40. Sayed GD, Iacoviello BM, Charney DS. (2015, Aug). Risk factors for the development of psychopathology following trauma. Curr Psychiatry Rep. 17(8): 612. doi: 10.1007/s11920-015-0612-9. PMID: 26112962.
41. Selye H. (1998, Spring). A syndrome produced by diverse nocuous agents. 1936. J Neuropsychiatry Clin Neurosci. 10(2): 230-231. doi: 10.1176/jnp.10.2.230a. PMID: 9722327.
42. Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. (1983). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.
43. Sun H, Gong TT, Jiang YT, Zhang S, Zhao YH, Wu QJ. (2019). Global, regional, and national prevalence and disability-adjusted life-years for infertility in 195 countries and territories, 1990-2017: results from a global burden of disease study, 2017. Aging (Albany NY). 11(23): 10952.
44. Szabo S, Tache Y, Somogyi A. (2012, Sep). The legacy of Hans Selye and the origins of stress research: a retrospective 75 years after his landmark brief «letter» to the editor# of nature. Stress. 15(5): 472-478. doi: 10.3109/10253890.2012.710919. PMID: 22845714.
45. Szabo S, Yoshida M, Filakovszky J, Juhasz G. (2017). «Stress» is 80 Years Old: From Hans Selye Original Paper in 1936 to Recent Advances in GI Ulceration. Curr Pharm Des. 23(27): 4029-4041. doi: 10.2174/1381612823666170622110046. PMID: 28641541.
46. Tatarchuk TF, Yefimenko OO, Myronenko OS, Mnevets RO. (2024). Consequences of wartime stress on women's reproductive health. Reproductive Endocrinology. (72): 28-34. doi: 10.18370/2309-4117.2024.72.28-34.
47. Tedeschi RG, Calhoun LG. (2004). Posttraumatic growth: conceptual foundations and empirical evidence. Psychol Inq. 15(1): 1-18. doi:10.1207/s15327965pli1501_01.
48. Wang C, Li QL, Xu YS, Cao KX, Zhang YQ, Chang L et al. T. (2025). Interplay of endocrine and psychological factors in IVF/ICSI outcomes: a prospective cohort analysis. Frontiers in Endocrinology. 16: 1596664.
49. Wingfield JC, Sapolsky RM. (2003). Reproduction and resistance to stress: when and how. Journal of neuroendocrinology. 15(8): 711-724.
50. World Health Organization. (2023). Infertility prevalence estimates, 1990-2021. World Health Organization.
51. Zasiekina L, Zasiekin S, Kuperman V. (2023, Oct). Post-traumatic Stress Disorder and Moral Injury Among Ukrainian Civilians During the Ongoing War. J Community Health. 48(5): 784-792. doi: 10.1007/s10900-023-01225-5. PMID: 37148415.

Відомості про авторів:
Берестовий Олег Олександрович – д.мед.н., асистент кафедри госпітального акушерства, гінекології та післядипломної освіти НМУ імені О.О. Богомольця; лікар акушер-гінеколог ТОВ медичний центр «MateriClinic». Адреса: м. Київ, просп. В. Лобановського, 2. https://orcid.org/0000-0002-5118-5530.
Сизоненко Анастасія Романівна – аспірант, асистент кафедри госпітального акушерства, гінекології та післядипломної освіти НМУ ім. О.О. Богомольця; лікар акушер-гінеколог КНП «Перинатальний центр м. Києва». Адреса: м. Київ, просп. В. Лобановського, 2. https://orcid.org/0009-0008-2630-499X.
Стаття надійшла до редакції 30.11.2025 р.; прийнята до друку 18.02.2026 р.