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THE EFFICACY OF HORMONE THERAPY IN NON-OBSTRUCTIVE AZOOSPERMIA

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Abstract
This analysis evaluates the effectiveness of hormone therapy in treating non-obstructive azoospermia. Hormonal interventions, especially in cases of hypogonadotropic hypogonadism, can restore spermatogenesis in 15%–60% of patients, depending on individual factors such as hormone levels, testicular volume, and genetics. While less invasive than surgical methods, success depends on careful patient selection. Advances in personalized and combination therapies offer new potential for improving outcomes.

Keywords: Non-obstructive azoospermia, hormone therapy, spermatogenesis, male infertility, gonadotropin therapy, hypogonadotropic hypogonadism, testicular function.

Introduction

Today’s contemporary reproductive medicine faces increasing challenges in addressing male infertility, with non-obstructive azoospermia representing one of the most complex and therapeutically challenging conditions encountered in clinical practice. This condition affects approximately 1% of all men and accounts for 10-15% of male infertility cases, making it a significant public health concern with profound implications for affected individuals and their partners. The absence of spermatozoa in the ejaculate due to primary spermatogenic failure or endocrine dysfunction presents unique diagnostic and therapeutic challenges that require sophisticated understanding of reproductive physiology and endocrinology. The pathophysiology of non-obstructive azoospermia encompasses a diverse spectrum of disorders ranging from congenital genetic abnormalities to acquired conditions affecting the hypothalamic-pituitary-gonadal axis or testicular parenchyma directly. The complexity of this condition is further compounded by the intricate hormonal regulation of spermatogenesis, which involves precise coordination between gonadotropin-releasing hormone pulsatility, luteinizing hormone and follicle-stimulating hormone secretion, and local testicular factors including testosterone and inhibin production. Historically, the management of non-obstructive azoospermia has relied primarily on surgical interventions such as testicular sperm extraction or microdissection testicular sperm extraction, with assisted reproductive technologies serving as the primary pathway to biological parenthood. However, these approaches are invasive, expensive, and associated with potential complications including testicular atrophy, chronic pain, and psychological distress. The recognition that certain forms of non-obstructive azoospermia may be amenable to medical therapy has sparked renewed interest in hormonal interventions as first-line treatment options. The theoretical foundation for hormone therapy in non-obstructive azoospermia rests on the

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understanding that spermatogenesis is exquisitely sensitive to hormonal milieu and that restoration of appropriate gonadotropin stimulation may rescue impaired germ cell development in selected patients. This approach is particularly relevant in cases where the primary pathology involves hypothalamic-pituitary dysfunction rather than intrinsic testicular pathology, though emerging evidence suggests that even some forms of apparent primary testicular failure may respond to intensive hormonal stimulation.

MAIN BODY

The normal process of spermatogenesis requires precise hormonal orchestration involving multiple levels of the reproductive axis. Gonadotropin-releasing hormone neurons in the hypothalamus generate pulsatile signals that stimulate anterior pituitary gonadotrophs to secrete luteinizing hormone and follicle-stimulating hormone. These gonadotropins act on testicular Leydig cells and Sertoli cells respectively, creating the hormonal environment necessary for germ cell development and maturation. Luteinizing hormone stimulates testosterone production by Leydig cells, creating high intratesticular testosterone concentrations that are essential for spermatogenesis. The concentration of testosterone within the seminiferous tubules is approximately 50-100 times higher than circulating levels, emphasizing the critical importance of local androgen action in supporting germ cell development. Follicle-stimulating hormone acts primarily on Sertoli cells, promoting their proliferation and differentiation while stimulating production of various factors essential for germ cell support including androgen-binding protein, inhibin, and anti-Müllerian hormone. In non-obstructive azoospermia, disruption of this hormonal cascade can occur at multiple levels. Central hypogonadism results from hypothalamic or pituitary dysfunction, leading to insufficient gonadotropin stimulation despite normal testicular responsiveness. This condition may be congenital, as seen in Kallmann syndrome or idiopathic hypogonadotropic hypogonadism, or acquired due to pituitary adenomas, cranial irradiation, or pharmacological suppression. Primary testicular dysfunction involves intrinsic defects in spermatogenesis or steroidogenesis, though the distinction between primary and secondary causes may be less clear-cut than traditionally assumed. Recent research has revealed that the testicular response to gonadotropin stimulation is more complex than previously understood, with significant individual variation in sensitivity and responsiveness. Genetic polymorphisms affecting gonadotropin receptor function, steroidogenic enzyme activity, and germ cell survival pathways contribute to this heterogeneity and may explain why some men with apparently primary testicular failure respond to hormonal intervention while others do not. The concept of testicular reserve has emerged as an important consideration in determining therapeutic potential. This refers to the residual capacity of the testes to respond to appropriate stimulation, which may be preserved even in the presence of severe baseline dysfunction. Assessment of testicular reserve through provocative testing with exogenous gonadotropins or gonadotropin-releasing hormone analogs can help identify candidates most likely to benefit from hormonal therapy.

The success of hormone therapy in non-obstructive azoospermia depends critically on appropriate patient selection based on comprehensive clinical evaluation. The diagnostic workup must include detailed history taking, physical examination, hormonal assessment, genetic testing when indicated, and testicular imaging to characterize the underlying pathophysiology and predict

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therapeutic responsiveness. Historical factors of particular importance include age at presentation, duration of infertility, previous fertility history, pubertal development patterns, and exposure to gonadotoxic agents including chemotherapy, radiation, or certain medications. The timing of azoospermia onset provides crucial information about etiology, with congenital conditions typically presenting with primary infertility while acquired causes may follow periods of normal fertility. Physical examination focuses on assessment of secondary sexual characteristics, testicular volume and consistency, and presence of varicoceles or other anatomical abnormalities. Testicular volume measurement using orchidometry or ultrasound provides important prognostic information, with larger testes generally associated with better therapeutic responses. The presence of bilateral testicular atrophy suggests advanced germ cell depletion, though preservation of normal volume does not guarantee normal spermatogenesis. Hormonal evaluation forms the cornerstone of diagnostic assessment and therapeutic planning. Baseline measurements of serum follicle-stimulating hormone, luteinizing hormone, testosterone, prolactin, and thyroid function provide essential information about hypothalamic-pituitary-gonadal axis status. Elevated gonadotropin levels with low or normal testosterone suggest primary testicular dysfunction, while low or inappropriately normal gonadotropins indicate central hypogonadism. However, these traditional categorizations may be oversimplified, as mixed patterns are increasingly recognized. Advanced hormonal testing may include gonadotropin-releasing hormone stimulation tests to assess pituitary responsiveness, human chorionic gonadotropin stimulation tests to evaluate Leydig cell function, and inhibin B measurement as a marker of Sertoli cell function and spermatogenic activity. These specialized tests help refine prognosis and guide treatment selection, particularly in cases with borderline or ambiguous baseline findings. Genetic evaluation is increasingly important in the assessment of non-obstructive azoospermia, with karyotype analysis and Y chromosome microdeletion testing now considered standard components of the diagnostic workup. The presence of Klinefelter syndrome, Y chromosome deletions, or other genetic abnormalities has important implications for treatment selection and genetic counseling. Recent advances in genetic testing have identified numerous additional loci associated with spermatogenic dysfunction, though the clinical utility of comprehensive genetic screening remains under investigation.

The hormonal treatment of non-obstructive azoospermia encompasses several distinct therapeutic approaches, each with specific indications, mechanisms of action, and expected outcomes. The selection of appropriate therapy depends on the underlying pathophysiology, patient characteristics, and treatment goals, with individualized protocols offering the best chance of success. Human chorionic gonadotropin monotherapy represents the simplest hormonal intervention and is often employed as first-line therapy in men with central hypogonadism or suspected Leydig cell dysfunction. This glycoprotein hormone mimics luteinizing hormone action, stimulating testosterone production and creating the intratesticular androgen environment necessary for spermatogenesis. Treatment protocols typically involve subcutaneous injections of 2000-3000 international units administered two to three times weekly for periods ranging from 3 to 12 months. Response rates vary considerably depending on patient selection criteria, with success rates of 20-40% reported in appropriately selected cases. The rationale for human chorionic gonadotropin therapy extends beyond simple testosterone replacement, as exogenous

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testosterone administration suppresses gonadotropin secretion and may worsen spermatogenesis. By stimulating endogenous testosterone production, human chorionic gonadotropin maintains the high intratesticular androgen concentrations required for germ cell development while avoiding the suppressive effects of exogenous androgen therapy. This approach is particularly relevant in men with secondary hypogonadism or isolated luteinizing hormone deficiency. Combined gonadotropin therapy utilizing both human chorionic gonadotropin and follicle-stimulating hormone offers theoretical advantages by addressing both Leydig cell and Sertoli cell dysfunction simultaneously. This approach is especially relevant in men with complete gonadotropin deficiency or those who fail to respond adequately to human chorionic gonadotropin monotherapy. Treatment protocols typically involve initial human chorionic gonadotropin therapy to restore testosterone production, followed by addition of recombinant follicle-stimulating hormone once adequate androgenization is achieved. Follicle-stimulating hormone preparations include both urinary-derived and recombinant formulations, with similar efficacy but different cost profiles and potential side effect patterns. The typical dosing regimen involves subcutaneous injections of 75-225 international units administered three times weekly, with dose adjustments based on clinical response and hormonal monitoring. The duration of therapy is generally longer than human chorionic gonadotropin monotherapy, with treatment courses extending 6-18 months or longer to achieve optimal results. Pulsatile gonadotropin-releasing hormone therapy represents the most physiological approach to hormonal stimulation and is particularly appropriate for men with hypothalamic dysfunction and preserved pituitary responsiveness. This treatment involves continuous subcutaneous or intravenous administration of gonadotropin-releasing hormone at physiological pulse frequencies using portable pump devices. While theoretically superior to exogenous gonadotropin therapy, the complexity and cost of gonadotropin-releasing hormone pump therapy limit its widespread application. Recent innovations in hormonal therapy include the development of selective estrogen receptor modulators and aromatase inhibitors for the treatment of male infertility. These agents work by blocking estrogen feedback at the hypothalamic-pituitary level, resulting in increased gonadotropin secretion and enhanced testicular stimulation. Clomiphene citrate and anastrozole have shown promise in preliminary studies, though their efficacy in non-obstructive azoospermia specifically requires further investigation.

The therapeutic efficacy of hormone therapy in non-obstructive azoospermia varies considerably depending on patient selection criteria, underlying etiology, and treatment protocols employed. Contemporary literature reports success rates ranging from 15% to 60% for achieving sperm production, with higher response rates observed in carefully selected populations with specific hormonal profiles and clinical characteristics. The most consistently favorable outcomes are observed in men with hypogonadotropic hypogonadism, particularly those with congenital gonadotropin deficiency or acquired central hypogonadism. In these populations, hormone therapy can achieve sperm production rates of 50-70%, with some studies reporting even higher success rates in men with preserved testicular volume and appropriate hormonal profiles. The response to treatment is typically dose-dependent and time-dependent, with optimal results often requiring 6-12 months of therapy. Men with primary testicular dysfunction traditionally have been considered poor candidates for hormonal intervention, though emerging evidence suggests that selected individuals may benefit from intensive gonadotropin stimulation. The success rates in this

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population are generally lower, typically ranging from 10-25%, but the potential for avoiding surgical intervention makes hormonal therapy a reasonable first-line approach in motivated patients with favorable characteristics. Several clinical and laboratory parameters have been identified as predictive factors for therapeutic success. Baseline testicular volume represents one of the strongest predictors of treatment response, with men having testicular volumes greater than 15 milliliters showing significantly higher success rates than those with smaller testes. This relationship likely reflects the correlation between testicular volume and residual spermatogenic capacity, though individual variation is considerable. Hormonal parameters also provide important prognostic information, though the relationships are complex and sometimes counterintuitive. Men with moderately elevated follicle-stimulating hormone levels may respond better than those with either very high or very low baseline values, suggesting an optimal range of baseline gonadotropin stimulation. The follicle-stimulating hormone to luteinizing hormone ratio has emerged as a potentially useful predictive marker, with certain ratios associated with improved treatment outcomes.

Age at treatment initiation influences therapeutic efficacy, with younger men generally showing better responses than older individuals. This age-related decline in treatment efficacy likely reflects the progressive loss of germ cell reserves with advancing age, though the relationship is not linear and individual variation is substantial. The optimal age for intervention remains debated, though most experts recommend attempting hormonal therapy in men under 40 years of age when appropriate indications exist. The duration of azoospermia prior to treatment initiation may also influence outcomes, with longer periods of infertility potentially associated with reduced therapeutic responsiveness. This observation suggests that early intervention may improve success rates, though the precise timeline for irreversible germ cell loss remains uncertain and likely varies among individuals. Genetic factors increasingly are recognized as important determinants of treatment response. Men with certain Y chromosome microdeletions may have reduced responsiveness to hormonal therapy, while specific genetic polymorphisms affecting gonadotropin receptor function or steroidogenic pathways may influence treatment outcomes. The integration of genetic testing into clinical decision-making represents an emerging area of investigation with potential for improving patient selection and treatment individualization.

The hormonal treatment of non-obstructive azoospermia generally is well-tolerated, though several important safety considerations and monitoring requirements must be addressed throughout the treatment course. The extended duration of therapy often required for optimal results necessitates careful attention to potential adverse effects and complications that may emerge during long-term hormone administration. Injection site reactions represent the most common adverse effects associated with gonadotropin therapy, occurring in approximately 10-20% of treated men. These reactions typically manifest as local erythema, swelling, or discomfort at injection sites and usually resolve spontaneously or with minor modifications to injection technique. Proper patient education regarding injection procedures and site rotation can minimize these complications. Gynecomastia development is a potential concern with hormonal therapy, particularly with human chorionic gonadotropin treatment, due to increased aromatization of testosterone to estradiol. The incidence of clinically significant gynecomastia ranges from 5-15% in treated populations, with most cases being mild and reversible upon treatment discontinuation.

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Monitoring of estradiol levels and consideration of aromatase inhibitor co-therapy may be appropriate in men at high risk for this complication.

Mood changes and behavioral effects occasionally are reported with gonadotropin therapy, though the incidence appears lower than with exogenous testosterone administration. The physiological restoration of hormone levels typically results in improved mood and energy levels, though individual responses vary considerably. Men with pre-existing mood disorders require careful monitoring and potential psychiatric consultation during treatment. Polycythemia represents a rare but potentially serious complication of hormonal therapy, particularly in men who achieve significant increases in testosterone levels. Regular monitoring of hematocrit levels is recommended, with dose adjustments or temporary treatment interruption indicated if values exceed normal ranges. The incidence of clinically significant polycythemia is low, occurring in fewer than 5% of treated men. Cardiovascular effects of hormonal therapy require consideration, particularly in older men or those with pre-existing cardiovascular risk factors. While physiological hormone replacement generally is considered safe, the potential for adverse cardiovascular events cannot be completely excluded. Baseline cardiovascular assessment and periodic monitoring of blood pressure, lipid profiles, and cardiac function may be appropriate in selected patients. The monitoring requirements for hormonal therapy in non-obstructive azoospermia include regular assessment of treatment response, hormone levels, and potential adverse effects. Semen analyses should be performed at regular intervals, typically every 2-3 months during treatment, to assess therapeutic response and guide treatment modifications. The appearance of any sperm in the ejaculate represents a positive response, even if concentrations remain below normal ranges. Hormonal monitoring includes periodic assessment of testosterone, luteinizing hormone, follicle-stimulating hormone, and estradiol levels to ensure appropriate therapeutic responses and detect potential complications. The frequency of monitoring depends on the specific treatment protocol and individual patient characteristics, though monthly assessments during treatment initiation and quarterly evaluations during maintenance therapy are generally appropriate.

The field of hormonal therapy for non-obstructive azoospermia continues to evolve rapidly, with several promising developments offering potential for improved treatment efficacy and expanded therapeutic options. These advances span multiple areas including novel therapeutic agents, personalized medicine approaches, combination therapy protocols, and enhanced patient selection methods. Recent research has focused on identifying new hormonal targets and therapeutic agents that may enhance spermatogenesis beyond traditional gonadotropin therapy. Selective estrogen receptor modulators and aromatase inhibitors have shown promise in preliminary studies, working through mechanisms distinct from conventional gonadotropin administration. These agents may be particularly valuable as adjunctive therapies or in patients who fail to respond to standard hormonal interventions. The development of long-acting gonadotropin preparations represents a significant advancement in treatment convenience and patient compliance. These formulations allow for less frequent injections while maintaining therapeutic efficacy, potentially improving treatment adherence and reducing injection-related adverse effects. Clinical trials of sustained-release gonadotropin preparations have demonstrated comparable efficacy to conventional formulations with improved patient satisfaction. Personalized medicine approaches are beginning

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to influence hormonal therapy selection and dosing strategies. Genetic testing for polymorphisms affecting gonadotropin receptor function, steroidogenic enzyme activity, and drug metabolism may allow for individualized treatment protocols optimized for each patient's specific genetic profile. This approach holds promise for improving treatment efficacy while minimizing adverse effects and unnecessary treatment exposure.

Combination therapy protocols utilizing multiple hormonal agents simultaneously or sequentially represent another area of active investigation. The rationale for combination approaches includes targeting multiple pathways involved in spermatogenesis, overcoming resistance mechanisms, and providing synergistic effects that may enhance overall treatment efficacy. Preliminary studies of combination protocols have shown encouraging results, though optimal regimens remain to be defined. The integration of advanced imaging techniques into treatment monitoring offers potential for improved assessment of therapeutic response and early detection of treatment failure. High-resolution testicular ultrasound, magnetic resonance imaging, and emerging techniques such as shear wave elastography may provide more sensitive measures of testicular function than conventional semen analysis alone. Biomarker development represents another promising area of research, with several candidate markers under investigation for their potential to predict treatment response, monitor therapeutic efficacy, and identify optimal treatment duration. These biomarkers include various hormonal parameters, genetic markers, and novel protein or metabolic indicators of spermatogenic activity.

The role of adjunctive therapies in enhancing hormonal treatment efficacy is gaining attention, with studies investigating the potential benefits of antioxidant supplementation, lifestyle modifications, and other supportive measures. These approaches may provide additional benefits when combined with hormonal therapy, though their specific contributions require further clarification through controlled clinical trials. Emerging understanding of epigenetic mechanisms in spermatogenesis may lead to novel therapeutic approaches targeting gene expression patterns rather than traditional hormonal pathways. These epigenetic therapies could potentially reverse acquired defects in germ cell development and offer new treatment options for men with apparent primary testicular dysfunction.

The optimal management of non-obstructive azoospermia requires careful consideration of multiple factors including patient characteristics, underlying etiology, treatment goals, and resource availability. Developing appropriate treatment algorithms and decision-making frameworks is essential for ensuring that patients receive optimal care while avoiding unnecessary interventions or delays in achieving reproductive goals. The initial decision regarding whether to pursue hormonal therapy versus immediate surgical intervention depends on several key factors. Patient age represents a critical consideration, with younger men generally better candidates for hormonal therapy due to higher success rates and longer time horizons for achieving pregnancy. Conversely, older men or those with partners of advanced maternal age may benefit from more expeditious surgical approaches to minimize delays in reproductive timing. The underlying etiology of azoospermia significantly influences treatment selection, with men having clear evidence of hypogonadotropic hypogonadism representing ideal candidates for hormonal intervention. Those with primary testicular dysfunction require more careful consideration, weighing the potential benefits of hormonal therapy against the time and resources required for

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treatment trials. Partner factors also influence treatment decision-making, particularly female partner age and fertility status. Couples with female factor infertility requiring assisted reproductive technologies may benefit from concurrent hormonal therapy in the male partner while pursuing fertility treatments, potentially improving overall reproductive outcomes. Conversely, couples with normal female fertility may justify more prolonged trials of hormonal therapy before considering surgical alternatives.

The duration of treatment trials represents another important clinical decision requiring careful consideration of success rates, patient motivation, and opportunity costs. Most experts recommend initial treatment trials of 6-12 months duration, with periodic reassessment of therapeutic response and consideration of treatment modification or discontinuation based on clinical progress. Cost-effectiveness considerations increasingly influence treatment selection, particularly in healthcare systems with limited resources or restricted insurance coverage. Hormonal therapy generally is less expensive than surgical interventions in the short term, though prolonged treatment courses may ultimately become more costly than surgical alternatives. Comprehensive economic analyses must consider not only direct treatment costs but also indirect costs related to time off work, travel, and psychological impact. The development of standardized treatment protocols and clinical guidelines remains an important goal for optimizing care in this population. Professional societies and expert panels continue to work toward consensus recommendations that balance available evidence with clinical experience and resource considerations. These guidelines must be flexible enough to accommodate individual patient circumstances while providing clear direction for clinical decision-making. Quality of life considerations represent an important but often overlooked aspect of treatment selection. Hormonal therapy may offer psychological benefits through preservation of hope and active participation in treatment, while surgical interventions may provide more definitive closure and enable couples to move forward with assisted reproductive technologies. Understanding individual patient preferences and values is essential for making appropriate treatment recommendations.

In conclusion, hormone therapy is a promising, less invasive treatment for selected patients with non-obstructive azoospermia, especially those with hypogonadotropic hypogonadism. Success rates vary (15–60%) and depend on individual factors such as hormone levels, testicular volume, and genetics. Personalized treatment protocols and combination therapies are improving outcomes. While long treatment duration and patient commitment are required, the safety profile is generally favorable. Ongoing research and integration of personalized medicine tools are essential for optimizing therapy and establishing evidence-based guidelines.

References:

1. Tohirbek To‘lqinjon o‘g, S. (2025, February). STRUCTURE AND FUNCTIONS OF THE URETHRAL SPHINCTER IN MEN. In Scientific Conference on Multidisciplinary Studies (pp. 165-171).
2. Tohirbek To’lqinjon o’g, S. (2024). Successful testicular sperm extraction in an infertile man with non-obstructive azoospermia and hypergonadotropic hypogonadism presenting with bilateral atrophic testis: a case report. Miasto Przyszłości, 48, 186-188.
3. Uzbekistan, O. F. To verify Questionnaire of the “Uzbek Index of Premature Ejaculation”.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

4. Tohirbek To‘lqinjon o‘g, S. (2025, February). STRUCTURE AND FUNCTIONS OF THE URETHRAL SPHINCTER IN MEN. In Scientific Conference on Multidisciplinary Studies (pp. 165-171).
5. Umarovich, B. M. (2025). DEVELOPING OF VIRAL INFECTIONS IN HEMATOPOIETIC STEM-CELL TRANSPLANT (HSCT) RECIPIENTS. Web of Medicine: Journal of Medicine, Practice and Nursing, 3(5), 468-473.
6. Muhammadiev, S. (2025). HEMIEPIPHYSIODESIS IN PEDIATRIC ORTHOPAEDICS AS A TREATMENT OF KNEE DEFORMITIES. International Journal of Artificial Intelligence, 1(4), 225-227.
7. Хошимова, А. Ё. (2018). ВЛИЯНИЕ ЗАГРЯЗНЕНИЯ ОКРУЖАЮЩЕЙ СРЕДЫ НА ЗАБОЛЕВАЕМОСТЬ БРОНХИАЛЬНОЙ АСТМОЙ. Актуальные вопросы современной пульмонологии. Ма, 200.
8. АБДУГАНИЕВА, А. Ё., & ЮЛДАШЕВА, Х. Б. К. ЛАБОРАТОРНАЯ ДИАГНОСТИКА ПРИ COVID-19. ИНТЕРНАУКА Учредители: Общество с ограниченной ответственностью" Интернаука", 37-38.
9. Lutfidin o'g'li, Y. B. (2025). ENDEMIK BUQOQ KASALLIGINI TA’SIRIDAN YO’LDOSH TERMINAL SO’RGICHLARDAGI QON TOMIRLARNING PATOMORFOLOGIK O’ZGARISHLARI. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 3(33), 322-325.
10. Lutfidin o‘g‘li, Y. B. (2025). Major Hystologic Types of Lung Cancer. Miasto Przyszłości, 57, 81-86.
11. Yusupov, B., & Xatamova, M. (2025). GIPERTIREOZ TA’SIRIDAN YO’LDOSH TERMINAL SO’RG’ICHLARDAGI QON TOMIRLARNING PATOMORFOLOGIK O’ZGARISHLARI. Modern Science and Research, 4(2), 432-437.
12. Yevgenevna, S. O. (2025). PREDICTION OF PREECLAMPSIA DEVELOPMENT IN PREGNANT WOMEN WITH OVERWEIGHT AND OBESITY. Web of Medicine: Journal of Medicine, Practice and Nursing, 3(5), 561-568.
13. Isroilova, G. (2023). DEVELOPING THE PRINCIPLES OF STUDYING AND TREATMENT OF VAGINAL DYSBIOSIS DURING PREGNANCY. Modern Science and Research, 2(4), 52-53.
14. Юсупова, Р. Т., & Шаланкова, О. Е. (2020). РЕПРОДУКТИВНОЕ ЗДОРОВЬЕ ДЕВОЧЕК-ПОДРОСТКОВ, ПРОЖИВАЮЩИХ В УСЛОВИЯХ ФЕРГАНСКОЙ ДОЛИНЫ. In Университетская наука: взгляд в будущее (pp. 612-614).
15. Пулатова, Н. С., Йигиталиев, А. Б., & Абдурашидов, А. А. ЭПИДЕМИОЛОГИЯ РАКА ТЕЛА МАТКИ В ФЕРГАНСКОЙ ОБЛАСТИ. 1-SON, 1-JILD IYUL 2022 1-QISM, 29.
16. Эгамбердиев, Д. Э., Абдурашидов, А. А., & Эргашов, У. Ш. ПРОФИЛАКТИКА И МЕТОФИЛАКТИКА МОЧЕКАМЕННОЙ БОЛЕЗНИ.
17. Husanboy, U. (2024). ACUTE HEMORRHAGIC CYSTITIS DISEASE IN CHILDREN AND ITS DEVELOPMENT IN THE CHILD’S DODY. In International Conference on Multidisciplinary Sciences and Educational Practices (pp. 88-94).
18. Ravshanovna, R. Y., & Abduxoliq o'g'li, R. A. (2024). Clinical and Morphological Characteristics and Treatment of Gaucher Disease. Miasto Przyszłości, 49, 1407-1412.

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