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TOWARD UNIFIED PATHOMORPHOLOGIC ASSESSMENT OF PITUITARY NEUROENDOCRINE TUMORS: WHAT THE NEW WHO CLASSIFICATION CHANGES—AND WHY IT MATTERS

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Abstract: Ongoing refinement of classification systems using modern analytic methods is pivotal for precision and personalized management of pituitary tumors. Over the last decade, histopathologic identification of pituitary lesions has evolved markedly, opening new avenues for studying tumorigenesis and forecasting biological behavior. This review traces the historical development of schemes for pituitary "adenomas" and details the 2022 World Health Organization (WHO) re-framing of these lesions as pituitary neuroendocrine neoplasms (PitNETs). We summarize clinical features and highlight histologic subtypes associated with aggressive behavior, discuss the core immunohistochemical (IHC) toolkit—including lineage-defining transcription factors—and lay out a practical, unified diagnostic workflow. Finally, we present the main arguments for and against the new terminology and propose actionable steps to harmonize reporting and care pathways.

Key words: pituitary adenoma; pituitary neuroendocrine tumor; PitNET; transcription factors; PIT1; TPIT; SF1; Ki-67; WHO 2022 classification; diagnostic standardization.

Introduction

Pituitary neuroendocrine tumors (PitNETs), historically called pituitary adenomas, are common, heterogeneous lesions with variable differentiation, secretory activity, and proliferative capacity. Even within a single lineage, morphologic and immunophenotypic diversity is considerable, which underscores the need for subtype-specific predictors of growth, invasion, treatment response, and recurrence.

Population registries indicate high prevalence, with detection rates rising due to improved MRI and sensitive hormonal assays. Many lesions are incidentally discovered and remain stable, yet a substantial proportion present as macroadenomas with mass effect, hypopituitarism, or endocrine hyperfunction at diagnosis. Tumorigenesis is multifactorial—driven by germline predisposition in a minority, somatic alterations, endocrine milieu, and tumor microenvironmental interactions that modulate angiogenesis, proliferation, and invasion. Practically, clinicians stratify risk by size and topography (micro-, macro-, giant), invasive growth on MRI (e.g., Knosp/Hardy), and secretory profile. Although most lesions behave indolently, roughly one third exhibit *clinical aggressiveness*—rapid growth, postoperative persistence/recurrence, and resistance to medical or radiotherapy.

From Color to Lineage: A Brief History of Classification

Early "tinctorial" era (late 19th–mid-20th century). Tumors were grouped by staining characteristics (acidophilic, basophilic, chromophobe) and loosely correlated with hormone excess syndromes. The approach was simple but often misleading; staining intensity did not reliably predict hormonal output or clinical course.

Ultrastructural era. Electron microscopy enabled recognition of densely vs. sparsely granulated phenotypes, bicellular lesions, and poorly differentiated forms—features that correlated better with behavior and drug response (e.g., sensitivity to first-generation somatostatin analogues).

WHO 2004 ("histopathologic"). For the first time, a schema integrated IHC for pituitary hormones, ultrastructure, and clinical data. Categories ranged from typical adenomas to "atypical" adenomas (elevated mitoses, Ki-67 >3%, p53 positivity) and carcinomas (defined by metastasis), signaling awareness that some adenomas behave far from benign.

WHO 2017 ("histogenetic"). A major conceptual shift: adenomas were stratified by *embryologic lineage* using transcription factors—PIT1, TPIT (TBX19), and SF1—augmented by modulators such as ERα and GATA2. The term "atypical adenoma" was dropped due to poor predictive value. Instead, specific *histotypes of high malignant potential* (e.g., sparsely granulated somatotroph, male lactotroph, Crooke cell corticotroph, PIT1-positive plurihormonal) were highlighted because of their invasive growth and therapy resistance.

WHO 2022 ("oncologic/unified NEN framework"). The decisive step: anterior pituitary tumors are formally placed within the neuroendocrine neoplasm family as PitNETs. Nomenclature, reporting elements, and some diagnostic rules were harmonized with NENs across organs, acknowledging neuroendocrine lineage, variable clinical behavior, and a small but real risk of malignant progression.

WHO 2022: What Exactly Changed?

Lineage-first architecture. Tumors are classified by transcription factor lineage:

PIT1 lineage → somatotroph, lactotroph, thyrotroph; mixed/mammosomatotroph; tumors of acidophil stem cell; *plurihormonal PIT1-positive* (mature vs. immature).

TPIT lineage → corticotroph, including Crooke cell type; densely vs. sparsely granulated forms.

SF1 lineage → gonadotroph tumors.

"Null cell" PitNET is a diagnosis of exclusion when no lineage/hormone expression is demonstrable.

Refined PIT1 group. Mammosomatotroph and mixed somato-lactotroph tumors are recognized as distinct; acidophil stem cell tumors are emphasized; PIT1-positive plurihormonal tumors are split into *mature* and *immature* (the latter often clinically aggressive).

Terminology for frank malignancy. Instead of "pituitary carcinoma," the term metastatic pituitary neuroendocrine tumor is preferred, aligning with NEN language elsewhere. As in earlier editions, metastasis—not histology alone—defines malignancy.

Standardized reporting. A minimum dataset specifies lineage TFs, hormone IHC, keratin profile (e.g., CAM5.2), Ki-67, and (where relevant) somatostatin receptor (SSTR) expression.

Why this matters: A lineage-driven diagnosis better predicts behavior (e.g., SSTR2-low sparsely granulated somatotrophs resist first-generation analogues) and organizes research and clinical trials around biologically coherent groups.

A Unified Diagnostic Workflow

1) Confirm neuroendocrine and epithelial differentiation

Neuroendocrine markers: synaptophysin (high sensitivity), chromogranin A (variable with differentiation), INSM1 (increasingly used).

Epithelial markers: cytokeratin panel (AE1/AE3, CAM5.2, CK18) to avoid misclassifying paraganglioma-like lesions.

2) Assign lineage and cell type

Transcription factors: PIT1, TPIT, SF1 (± ERα, GATA2/GATA3 to refine PIT1 and SF1 lineages).

Hormones: GH, PRL, β-TSH, β-FSH, β-LH, ACTH, α-subunit.

This step differentiates pure vs. mixed tumors and flags plurihormonal PIT1-positive forms.

3) Quantify proliferative activity and adverse histology

Ki-67 index (report exact percentage), mitotic count, presence of necrosis, vascular/perivascular invasion, and p53 overexpression. Rising Ki-67 or loss of hormonal expression over time can signal dedifferentiation.

4) Assess therapeutic biomarkers

SSTR2/SSTR5 IHC (or functional imaging where available) informs the likelihood of response to somatostatin analogues; E-cadherin/β-catenin patterns may have prognostic value; keratin fibrous bodies support sparsely granulated phenotypes.

5) Integrate clinical, biochemical, and radiologic data

MRI with dedicated sellar protocols; invasion rating (e.g., Knosp); endocrine panels: PRL, GH/IGF-1, TSH, fT4, fT3, LH/FSH, morning cortisol with low-dose dexamethasone suppression; estradiol (premenopausal women), total testosterone (men).

Consider genetic counseling in young patients, MEN1/MEN4, Carney complex, McCune–Albright, familial isolated pituitary adenoma, X-linked acrogigantism, or suggestive family history.

How to Phrase the Pathology Report (Practical Template)

Diagnosis (core): *Pituitary neuroendocrine tumor (PitNET), [lineage TF], [cell type], [granulation pattern if applicable], [degree of differentiation]*.

Example: "Well-differentiated PitNET of PIT1 lineage with somatotroph differentiation, sparsely granulated type."

Mandatory elements to include:

Lineage TF result(s): PIT1/TPIT/SF1 (positive/negative, nuclear staining quality).

Hormone IHC profile (positive/negative; semiquantitative intensity if used).

Keratin staining pattern (e.g., CAM5.2; fibrous bodies present/absent).

Proliferation: Ki-67 (%), mitoses (/2 mm^2), necrosis (yes/no).

Invasion in the specimen (if evaluable).

Predictive/prognostic markers: SSTR2/SSTR5, E-cadherin/β-catenin (if performed).

Comment correlating with clinical data and suggesting implications for therapy and follow-up (e.g., SSTR-low SG-somatotroph → limited response to first-generation analogues; consider alternate medical/radiation strategies).

Aggressive Clinical Behavior: Who Is at Risk?

Across lineages, several histotypes repeatedly associate with higher invasion, recurrence, and therapeutic resistance:

Sparsely granulated somatotroph PitNETs (often SSTR2-low, T2-hyperintense on MRI).

Male lactotroph PitNETs (frequently large, invasive, dopamine-agonist resistant).

Crooke cell corticotroph PitNETs and silent corticotroph tumors (TPIT lineage).

PIT1-positive plurihormonal tumors, especially immature variants.

Acidophil stem cell tumors (PIT1 lineage), typically with modest secretion but brisk growth.

Recognizing these at the time of initial surgery enables earlier planning for multimodal therapy and tighter imaging/endocrine surveillance.

The Terminology Debate: PitNET vs. Adenoma

Main concerns raised by critics

"Oncologization" of a common benign disease. Most lesions are indolent; relabeling them as "neoplasms" within the NEN family may alarm patients and complicate insurance pathways.

Limited histology–behavior concordance. No single marker infallibly predicts invasion or recurrence; radiologic features (e.g., T2 signal, cavernous sinus invasion) also weigh heavily.

Marker specificity. Classic neuroendocrine markers (synaptophysin, NSE) are not exclusive to neuroendocrine tissues, risking conceptual blurring across endocrine organs.

Patient-centered language. Terminology affects decisions, anxiety, and quality of life. Stakeholder engagement (patients, advocates) has been limited.

Transitional complexity. Dual use ("adenoma/PitNET") in the short term may create confusion across guidelines and payers.

Rationale from proponents

Biologic accuracy. Adenohypophyseal cells belong to the diffuse neuroendocrine system; lineage-based pathology aligns with tumor biology and treatment.

Standardization across organs. A unified NEN framework harmonizes reporting, enables cross-site research, and clarifies definitions for metastatic disease.

Actionable subtyping. Lineage and granulation status anticipate pharmacologic response (e.g., to somatostatin analogues) and recurrence risk, improving care.

A pragmatic middle path

During transition, many centers report using combined wording—e.g., "Pituitary neuroendocrine tumor (adenoma)"—paired with clear patient education materials that explain risk tiers and typical indolent courses for the majority.

Practical Recommendations for a Unified National Protocol

Adopt a minimum IHC panel for all resected pituitary tumors: synaptophysin, chromogranin A (or INSM1), CAM5.2, PIT1, TPIT, SF1, ERα (when PIT1 is positive), GATA3 (selected cases), pituitary hormones, Ki-67; add SSTR2/SSTR5 in GH- and some plurihormonal tumors.

Mandate lineage-first reporting with explicit Ki-67 and mitotic data; flag aggressive-risk histotypes in the comment.

Standardize endocrine testing at baseline and follow-up (PRL; GH/IGF-1; TSH, fT4, fT3; LH/FSH ± sex steroids; cortisol with low-dose DST).

Integrate radiology (dedicated sellar MRI, invasion scores) into MDT decisions; routine post-op MRI timing should be protocolized.

Build registries and prospective cohorts capturing lineage, granulation type, Ki-67, SSTR profile, treatment, and outcomes to refine prognostication.

Strengthen multidisciplinary care—neurosurgery, endocrinology, pathology, neuroradiology, radiation oncology—with the pathologist actively guiding adjuvant strategy.

Provide patient-facing explanations clarifying that most PitNETs are biologically indolent, while a minority warrant intensified monitoring or therapy.

Future Directions

The field is moving toward composite prognostic models that unite clinical (sex, age), biochemical (hormone output), imaging (invasion, T2 signal), morphology (lineage, granulation, Ki-67), and molecular metrics (driver alterations, methylation class, transcriptomic signatures). Such integrative scoring systems should better separate truly *benign-course* tumors from *clinically aggressive* PitNETs and identify subsets for targeted therapies (e.g., second-generation somatostatin analogues, GH receptor antagonists, precision radiotherapy, experimental agents).

Conclusions

The WHO 2022 classification positions anterior pituitary tumors squarely within the neuroendocrine neoplasm paradigm, compelling standardized, lineage-driven reporting. While terminology remains contentious, the practical gains are clear: earlier recognition of aggressive phenotypes, more rational selection of medical agents, and more consistent follow-up. National adoption of a unified morphologic and clinical protocol—anchored by a core IHC panel and explicit Ki-67 reporting—can reduce ineffective care, anxiety, and cost, while enabling true personalization for the minority of patients at highest risk.

References

1. Захарова, Н. Н., & Дворянский, С. А. (2010). Синдром поликистозных яичников. *Вятский медицинский вестник*, (2), 3-8.
2. Gade, A., Sawant, R., Parkar, S., & Kegade, P. (2020). Polycystic ovary syndrome: An overview, diagnosis and treatment of pcos. *Asian Journal of Pharmacy and Technology*, *10*(4), 265-272..
3. Ergasheva, G. (2024). THE ROLE OF CORRECTIONAL PEDAGOGY IN ORGANIZING THE EDUCATION OF CHILDREN WITH DISABILITIES. *Ethiopian International Journal of Multidisciplinary Research*, *11*(06), 206-207.
4. Ergasheva, G. (2025). PECULIARITIES WHEN ACCOMPANIED BY HYPOTHYROIDISM AND IODINE DEFICIENCY IN PATIENTS WITH ADRENAL GLAND PATHOLOGY. *Modern Science and Research*, *4*(2), 1133-1140.
5. Toxirovna, E. G. (2024). QALQONSIMON BEZ KASALLIKLARIDAN HASHIMOTO TIREODIT KASALLIGINING MORFOFUNKSIONAL O'ZIGA XOSLIGI. *Modern education and development*, *16*(7), 120-135.
6. Toxirovna, E. G. (2024). REVMATOID ARTRIT: BO'G'IMLAR YALLIG'LANISHINING SABABLARI, KLINIK BELGILARI, OQIBATLARI VA ZAMONAVIY DAVOLASH YONDASHUVLARI. *Modern education and development*, *16*(7), 136-148.
7. Эргашева, Г. Т. (2024). ОЦЕНКА КЛИНИЧЕСКОЙ ЭФФЕКТИВНОСТИ ОРЛИСТАТА У БОЛЬНЫХ ОЖИРЕНИЕМ И АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИЕЙ. *Modern education and development*, *16*(7), 92-105.
8. Ergasheva, G. (2025). ACROMEGALY: A SEVERE NEUROENDOCRINE DISORDER WITH MULTISYSTEM MANIFESTATIONS. *Modern Science and Research*, *4*(3), 1123-1131.
9. Ergasheva, G. T. (2024). THE SPECIFICITY OFAUTOIMMUNE THYROIDITIS IN PREGNANCY. *European Journal of Modern Medicine and Practice*, *4*(11), 448-453.
10. Эргашева, Г. Т. (2024). ИССЛЕДОВАНИЕ ФУНКЦИИ ЩИТОВИДНОЙ ЖЕЛЕЗЫ ПРИ ТИРЕОИДИТЕ ХАШИМОТО. *Modern education and development*, *16*(7), 106-119.
11. Toxirovna, E. G. (2024). GIPOFIZ ADENOMASINI NAZORAT QILISHDA KONSERVATIV JARROHLIK VA RADIATSIYA TERAPIYASINING UZOQ MUDDATLI SAMARADORLIGI. *Modern education and development*, *16*(7), 79-91.
12. ERGASHEVA, G. T. (2024). OBESITY AND OVARIAN INSUFFICIENCY. *Valeology: International Journal of Medical Anthropology and Bioethics*, *2*(09), 106-111.
13. Ergasheva, G. T. (2024). Modern Methods in the Diagnosis of Autoimmune Thyroiditis. *American Journal of Bioscience and Clinical Integrity*, *1*(10), 43-50.
14. Tokhirovna, E. G. (2024). COEXISTENCE OF CARDIOVASCULAR DISEASES IN PATIENTS WITH TYPE 2 DIABETES. *TADQIQOTLAR. UZ*, *40*(3), 55-62.

15. Toxirovna, E. G. (2024). DETERMINATION AND STUDY OF GLYCEMIA IN PATIENTS WITH TYPE 2 DIABETES MELLITUS WITH COMORBID DISEASES. *TADQIQOTLAR. UZ*, *40*(3), 71-77.

16. Toxirovna, E. G. (2024). XOMILADORLIKDA QANDLI DIABET KELTIRIB CHIQARUVCHI XAVF OMILLARINI ERTA ANIQLASH USULLARI. *TADQIQOTLAR. UZ*, *40*(3), 63-70.

17. Toxirovna, E. G. (2024). QANDLI DIABET 2-TIP VA KOMORBID KASALLIKLARI BO'LGAN BEMORLARDA GLIKEMIK NAZORAT. *TADQIQOTLAR. UZ*, *40*(3), 48-54.

18. Tokhirovna, E. G. (2024). MECHANISM OF ACTION OF METFORMIN (BIGUANIDE) IN TYPE 2 DIABETES. *JOURNAL OF HEALTHCARE AND LIFE-SCIENCE RESEARCH*, *3*(5), 210-216.

19. Tokhirovna, E. G. (2024). THE ROLE OF METFORMIN (GLIFORMIN) IN THE TREATMENT OF PATIENTS WITH TYPE 2 DIABETES MELLITUS. *EUROPEAN JOURNAL OF MODERN MEDICINE AND PRACTICE*, *4*(4), 171-177.

20. Эргашева, Г. Т. (2024). Эффект Применения Бигуанида При Сахарным Диабетом 2 Типа И Covid-19. *Research Journal of Trauma and Disability Studies*, *3*(3), 55-61.

21. Toxirovna, E. G. (2024). QANDLI DIABET 2 TUR VA YURAK QON TOMIR KASALLIKLARINING BEMOLARDA BIRGALIKDA KECHISHI. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *38*(7), 202-209.

22. Эргашева, Г. Т. (2024). СОСУЩЕСТВОВАНИЕ ДИАБЕТА 2 ТИПА И СЕРДЕЧНО-СОСУДИСТЫХ ЗАБОЛЕВАНИЙ У ПАЦИЕНТОВ. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *38*(7), 219-226.

23. Эргашева, Г. Т. (2024). СНИЖЕНИЕ РИСКА ОСЛОЖНЕНИЙ У БОЛЬНЫХ САХАРНЫМ ДИАБЕТОМ 2 ТИПА И СЕРДЕЧНО-СОСУДИСТЫМИ ЗАБОЛЕВАНИЯМИ. *Образование Наука И Инновационные Идеи В Мире*, *38*(7), 210-218.

24. Tokhirovna, E. G. (2024). CLINICAL AND MORPHOLOGICAL ASPECTS OF THE COURSE OF ARTERIAL HYPERTENSION. *Лучшие интеллектуальные исследования*, *12*(4), 234-243.

25. Tokhirovna, E. G. Studying the Causes of the Relationship between Type 2 Diabetes and Obesity. *Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN*, 2456-6470.

26. Toxirovna, E. G. (2024). ARTERIAL GIPERTENZIYA KURSINING KLINIK VA MORFOLOGIK JIHATLARI. *Лучшие интеллектуальные исследования*, *12*(4), 244-253.

27. Эргашева, Г. Т. (2024). НОВЫЕ АСПЕКТЫ ТЕЧЕНИЕ АРТЕРИАЛЬНОЙ ГИПЕРТОНИИ У ВЗРОСЛОГО НАСЕЛЕНИЕ. *Лучшие интеллектуальные исследования*, *12*(4), 224-233.

28. Эргашева, Г. Т. (2024). ФАКТОРЫ РИСКА РАЗВИТИЯ САХАРНОГО ДИАБЕТА 2 ТИПА. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *36*(5), 70-74.

29. Эргашева, Г. Т. (2024). ОСЛОЖНЕНИЯ САХАРНОГО ДИАБЕТА 2 ТИПА ХАРАКТЕРНЫ ДЛЯ КОГНИТИВНЫХ НАРУШЕНИЙ. *TADQIQOTLAR. UZ*, *30*(3), 112-119.

30. Эргашева, Г. Т. (2023). Исследование Причин Связи Диабета 2 Типа И Ожирения. *Research Journal of Trauma and Disability Studies*, *2*(12), 305-311.

31. Tokhirovna, E. G. (2024). Risk factors for developing type 2 diabetes mellitus. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *36*(5), 64-69.

32. Toxirovna, E. G. (2024). QANDLI DIABET 2-TUR VA O'LIMNI KELTIRIB CHIQARUVCHI SABABLAR. *Лучшие интеллектуальные исследования*, *14*(4), 86-93.

33. Tokhirovna, E. G. (2023). Study of clinical characteristics of patients with type 2 diabetes mellitus in middle and old age. *Journal of Science in Medicine and Life*, *1*(4), 16-19.
34. Toxirovna, E. G. (2024). GIPERPROLAKTINEMIYA KLINIK BELGILARI VA BEPUSHTLIKKA SABAB BO'LUVCHI OMILLAR. *Лучшие интеллектуальные исследования*, *14*(4), 168-175.
35. Toxirovna, E. G. (2023). QANDLI DIABET 2-TUR VA SEMIZLIKNING O'ZARO BOG'LIQLIK SABABLARINI O'RGANISH. *Ta'lim innovatsiyasi va integratsiyasi*, *10*(3), 168-173.
36. Saidova, L. B., & Ergashev, G. T. (2022). Improvement of rehabilitation and rehabilitation criteria for patients with type 2 diabetes.
37. Эргашева, Г. Т. (2023). Изучение Клинических Особенностей Больных Сахарным Диабетом 2 Типа Среднего И Пожилого Возраста. *Central Asian Journal of Medical and Natural Science*, *4*(6), 274-276.
38. Toxirovna, E. G. (2023). O'RTA VA KEKSA YOSHLI BEMORLARDA 2-TUR QANDLI DIABET KECHISHINING KLINIKO-MORFOLOGIK XUSUSIYATLARI. *ОБРАЗОВАНИЕ НАУКА И ИННОВАЦИОННЫЕ ИДЕИ В МИРЕ*, *33*(1), 164-166.
39. Ergasheva, G. T. (2022). QANDLI DIABET BILAN KASALLANGANLARDA REABILITATSIYA MEZONLARINI TAKOMILASHTIRISH. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, *2*(12), 335-337.
40. Ergasheva, G. (2024). METHODS TO PREVENT SIDE EFFECTS OF DIABETES MELLITUS IN SICK PATIENTS WITH TYPE 2 DIABETES. *Журнал академических исследований нового Узбекистана*, *1*(2), 12-16.
41. ГТ, Э., & Саидова, Л. Б. (2022). СОВЕРШЕНСТВОВАНИЕ РЕАБИЛИТАЦИОННО-ВОССТАНОВИТЕЛЬНЫХ КРИТЕРИЕВ БОЛЬНЫХ С СД-2 ТИПА. *TA'LIM VA RIVOJLANISH TAHLILI ONLAYN ILMIY JURNALI*, *2*(12), 206-209.
42. Xalimova, Y. (2025). MODERN CONCEPTS OF BIOCHEMISTRY OF BLOOD COAGULATION. *Modern Science and Research*, *4*(3), 769-777.
43. Xalimova, Y. (2025). JIGAR SIRROZIDAGI GEMATOLOGIK TADQIQOTLAR. *Modern Science and Research*, *4*(4), 409-418.
44. Халимова, Ю. (2025). ГЕМАТОЛОГИЧЕСКИЕ ИССЛЕДОВАНИЯ ПРИ ЦИРРОЗЕ ПЕЧЕНИ. *Modern Science and Research*, *4*(4), 419-428.
45. Xalimova, Y. (2025). HEMATOLOGICAL STUDIES IN LIVER CIRRHOSIS. *Modern Science and Research*, *4*(4), 1066-1074.
46. Халимова, Ю. С. (2025). СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ О БИОХИМИИ СВЕРТЫВАНИЯ КРОВИ. *Tadqiqotlar*, *59*(1), 16-23.
47. Халимова, Ю. С. (2025). АКТУАЛЬНЫЕ ПРОБЛЕМЫ И КЛИНИЧЕСКИЕ ОСОБЕННОСТИ ТЕЧЕНИЯ СИНДРОМА ПОЛИКИСТОЗНЫХ ЯИЧНИКОВ. *Tadqiqotlar*, *59*(1), 24-34.
48. Халимова, Ю. С. (2025). КЛИНИЧЕСКИЕ И МОРФОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ГЕМОЛИТИЧЕСКОЙ БОЛЕЗНИ НОВОРОЖДЕННЫХ. *Tadqiqotlar*, *59*(1), 9-15.
49. Темирова, Д. О., & Мухитдинова, Х. С. (2025). ВНЕМАТОЧНАЯ БЕРЕМЕННОСТЬ–ЗАБОЛЕВАНИЕ, ТРЕБУЮЩЕЕ НЕОТЛОЖНОЙ ПОМОЩИ. Modern education and development, 19(2), 342-354.

50. Мухитдинова, Х. С., & Темирова, Д. О. (2025). ОСОБЕННОСТИ ПАТОЛОГИЯ ЯИЧНИКОВ В СТРУКТУРЕ ГИНЕКОЛОГИЧЕСКОЙ ЗАБОЛЕВАЕМОСТИ. Modern education and development, 19(2), 450-463.
51. Темирова, Д. О., & Мухитдинова, Х. С. (2025). ПРЕЖДЕВРЕМЕННАЯ ОТСЛОЙКА ПЛАЦЕНТЫ. Modern education and development, 19(2), 316-327.
52. Темирова, Д. О., & Мухитдинова, Х. С. (2025). МОРФОФУНКЦИОНАЛЬНЫЕ ОСОБЕННОСТИ ТРИХОМОНИАЗА. Modern education and development, 19(2), 355-364.
53. Темирова, Д. О., & Мухитдинова, Х. С. (2025). РАЗРЫВ МАТКИ–СЕРЬЕЗНОЕ ОСЛОЖНЕНИЕ В АКУШЕРСТВЕ. Modern education and development, 19(2), 365-374.
54. Темирова, Д. О., & Мухитдинова, Х. С. (2025). СПКЯ-ОДНА ИЗ ПРИЧИН БЕСПЛОДИЯ. Modern education and development, 19(2), 328-341.