Rybin A. I., Kuznetsova O. V., Zamyshlyak V. I. Molecular characteristics of endometrial cancer: entering the era of precision medicine. Journal of Education, Health and Sport. 2026;87:70267. eISSN 2391-8306. https://dx.doi.org/10.12775/JEHS.2026.87.70267
https://apcz.umk.pl/JEHS/article/view/70267
https://zenodo.org/records/19224739

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences);
Health Sciences (Field of medical and health sciences). Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 05.01.2026. Revised: 12.01.2026. Accepted: 16.01.2026. Published: 27.01.2026.

UDC 618.14-006

Molecular characteristics of endometrial cancer: entering the era of precision medicine

A. I. Rybin, O. V. Kuznetsova, V. I. Zamyshlyak

Odessa National Medical University, Odessa, Ukraine

A.I. Rybin, ORCID: 0000-0002-1145-6690, e-mail: Andriy.Rybin@gmail.com
O.V. Kuznetsova, ORCID: 0000-0003-3778-4868, e-mail: alenakuz000@gmail.com
V.I. Zamyshlyak, ORCID: 0009-0001-1588-182X e-mail: vikazamishlyak@gmail.com

Abstract

Endometrial cancer ranks third among cancers affecting women in Ukraine, with a tendency to incidence rate increase every year. The disease is extremely rare in women under 40, and uncommon between 40 and 50, but thereafter, as age increases, the incidence curve rises sharply, peaking at around 63 years of age. As a rule, the disease is diagnosed at an early stage, with an overall 5-year survival rate exceeding 95%. However, this rate drops significantly in cases of regional or distant spread, standing at 68% and 17% respectively. Historically, endometrial carcinoma was classified into two main clinical-pathological types. The first type comprised endometrioid adenocarcinomas, whilst the second included non-endometrioid tumours (serous, clear cell, undifferentiated carcinomas and carcinosarcomas). However, the work carried out in compiling The Cancer Genome Atlas (TCGA) has transformed our understanding of the molecular landscape of endometrial cancer, identifying

not two but four molecular subtypes: tumours with mutations in the POLE gene, microsatellite-unstable carcinomas, tumours with mutations in the TP53 gene, and carcinomas of an unspecified molecular subtype. The integration of molecular classification into clinical practice has altered the stratification of patients into risk groups, which, in turn, has influenced the choice of adjuvant therapy. The rehabilitation of patients and ensuring a high quality of life is a very complex task, and to address it, an appropriate balance is required between reducing the risk of recurrence and preventing side effects associated with unjustified escalation of treatment.

This review presents information on the molecular subtypes of endometrial cancer, surrogate markers for each molecular subtype, and methods for their determination. The prognostic significance of molecular classification is discussed, as well as the prospects for its use in the design of future studies.

Keywords: endometrial cancer; molecular classification; literature review; POLE; MMRd; p53

In Ukraine, endometrial cancer (EC) ranks third in the incidence of cancer among women (10.1%). It is the most common gynaecological malignancy [1]. Surgery is the mainstay of treatment for EC; moreover, when a patient is stratified into a low-risk group for progression, surgical treatment may be the sole and sufficient therapeutic option. However, some histological variants of endometrial carcinoma are characterised by a high rate of progression, which requires the use of adjuvant radiotherapy and/or chemotherapy [2].

The first classification of different pathogenetic variants of EC was formulated by Y.V. Bokhman in 1983. Based on epidemiological studies, he identified type I endometrial cancer, associated with oestrogen stimulation and corresponding to low-grade endometrioid carcinoma, and type II — oestrogen-independent, corresponding to high-grade endometrioid carcinoma or serous carcinoma.

The second type was characterised by deep invasion of the myometrium and rapid progression.

Y.V. Bochman’s classification was long used in clinical practice, but had significant shortcomings, as it did not describe a clear boundary between the two variants; furthermore, a great many cases could not be classified under either of them [3, 4]. A revolution in views on the origin, treatment and prognosis of endometrial cancer occurred thanks to the work carried out by the Research Network in compiling The Cancer Genome Atlas (TCGA) [5]. A multi-

platform analysis of 373 endometrial carcinomas defined the molecular landscape of this tumour, identifying not two but four molecular subtypes. The molecular subtypes of endometrial cancer were identified for the first time not through the analysis of individual mutations, but on the basis of integrated genomic architecture. Endometrioid, serous and mixed tumour types were studied.

The clustering of tumours into subtypes was based on the number of mutations present, changes in the number of somatic gene copies, and the presence of microsatellite instability (MSI).

The first 'ultra-mutated' group accounted for 12% of the endometrial carcinomas studied and was characterised by an exceptionally high mutation rate (232×10^6 per Mb); however, the tumours lacked large-scale changes in somatic copy number (on average, a genomic change of less than 0.5%) . Significantly mutated genes (SMGs) in this subgroup included PTEN (94%), PIK3R1 (65%), PIK3CA (71%), FBXW7 (82%), KRAS (53%) and, of course, POLE (100%). The frequency of POLE mutations exceeded that observed in tumours of other sites.

he second 'hypermutated' group (18×10^6 mutations per Mb) proved to be the most numerous, accounting for 40% of the tumours studied. This subtype is characterised by a low frequency of somatic copy number alterations (SCNAs) and MSI.

The third group, with a low mutation rate (2.9×10^6 per Mb), accounted for 30% of all tumours studied, the majority of which were microsatellite-stable (MSS). The fourth group accounted for no more than 18% of the endometrial carcinomas studied and consisted of tumours with a very high frequency of somatic copy number alterations (SCNAs), a low mutation rate (2.3×10^6 per Mb) and amplifications of the MYC (8q24.12), ERBB2 (17q12) and CCNE1 (19q12)13. The frequency of TP53 mutations in this cluster exceeded 90% [5].

Molecular classification of endometrial cancer was fairly accurate in determining disease prognosis. However, due to logistical and resource constraints and poor reproducibility, the described technique proved impractical for routine clinical use. Less costly and more convenient methods were required for classifying endometrial carcinomas by molecular subtype. Identifying surrogate markers that would accurately correspond to the molecular subtype became the only possible way to solve the problem.

Two independently working groups — TransPORTEC (Post-Operative Radiation Therapy in Endometrial Carcinoma) and ProMisE (The Proactive Molecular Risk Classifier for Endometrial Cancer) — identified surrogate markers that allow endometrial carcinoma to be classified into one of four molecular subtypes. The use of surrogate markers resulted in the

classification of endometrial tumours into groups analogous to those in the TCGA: POLE-ultramutated (POLEmut) — tumours analogous to the ultramutated group; MMR-deficient (mismatch repair–deficient — MMRd) — analogous to the 'hypermutated group'; tumours associated with TP53 mutations — p53-abnormal (p53abn), corresponding to the group with a very high frequency of somatic copy number alterations (SCNAs); tumours not characterised by the above features, or tumours without a specific molecular profile (NSMP), were similar to the MSS group with a low mutation rate.

The application of molecular classification in assessing the prognosis of endometrial carcinoma has demonstrated that there is a group of patients with an excellent prognosis, i.e. POLEmut tumours, and a group with a poor prognosis, i.e. p53abn tumours. Endometrial carcinomas in the MMRd or NSMP groups are characterised by an intermediate prognosis [6–8].

The 2020 ESGO/ESTRO/ESP consensus guidelines on the management of patients with endometrial carcinoma recommend the use of molecular classification for all endometrial carcinomas, particularly for high-grade tumours [9].

Diagnostic tests. Molecular classification adds a further layer of information to standard morphological features and should therefore be integrated into routine pathological reports.

The diagnostic algorithm involves performing three immunohistochemical reactions to determine the expression of p53, MSH6 and PMS2, and one molecular test (analysis of mutations in the POLE exonuclease domain) in the following sequence:

– analysis of mutations in the POLE exonuclease domain;

– determination of MSH6 and PMS2 protein expression;

– detection of abnormal p53 expression [10].

Performing any single test for the presence of surrogate markers in isolation is insufficient [11].

Detection of POLE mutations. Endometrial carcinoma should be classified as POLE-mutated (POLEmut) only when pathogenic mutations are identified in the exonuclease domain of the gene. The simplest method for their identification is PCR, which identifies up to 92% of all POLE-mutant cancers [7, 12].

The most common somatic mutations are P286R, V411L and S459F. However, a comprehensive analysis of the exonuclease domain, covering exons 9–14 or 9, 13 and 14, is preferable; this requires next-generation sequencing (NGS) [13, 14].

Demographically, the POLE mutation was generally found in the youngest cohort; furthermore, these patients were characterised by a low body mass index (BMI). The POLE mutation is associated with the endometrioid histological subtype and early-stage disease. This defect leads to an ultra-mutated tumour, which, in turn, contributes to an enhanced immune response (marked CD8+ lymphocytic infiltration and an enhanced cytotoxic T-cell response). Sometimes these tumours may appear quite aggressive, characterised by a high grade of malignancy or marked lymphovascular invasion, but nevertheless have a favourable prognosis and a five-year survival rate of 96% [15].

iagnostic tests for identifying defects in the mismatch repair system (MMRd subgroup). Testing MMR status by determining MSI in endometrial carcinomas can simultaneously address three objectives: screening to identify patients with Lynch syndrome, determining the relevant molecular subtype, and assessing the potential for treatment with checkpoint inhibitors in the event of disease recurrence.

Two main methods are used in practice to diagnose mismatch repair defects: IHC and PCR. The IHC method is based on detecting the expression of the MLH1, MSH2, MSH6 and PMS2 proteins. The absence of expression of any of these indicates a defect in the MMR system and allows the tumour to be characterised as microsatellite-unstable. PCR diagnosis is based on the detection of simple repetitive sequences — microsatellites — in the DNA structure that do not encode information. In 2004, the US National Cancer Institute adopted a standard panel of five sequences, comprising three dinucleotide (D5S346, D2S123, D17S250) and two mononucleotide (BAT-25, BAT-26) markers. A tumour is classified as MSI-H if discrepancies are detected in two or more markers [16]. If a discrepancy is found in one of the five markers, a repeat test using an alternative method—IHC or NGS—is required. The immunohistochemical method is less costly, well-established and easily reproducible, and is therefore the preferred choice at the initial stage. The results of IHC and PCR are comparable in 86–99% of cases [17, 18]. The International Society of Gynecological Pathology (ISGyP) has recommended testing for MMR/MSI status in all cases of endometrial carcinoma [19]. Patients in this category did not exhibit any specific demographic characteristics [15].

Diagnostic tests for identifying the p53abn subgroup. The frequency of TP53 mutations in the high-copy-number group exceeds 90%, so the use of IHC to detect pathological p53 expression is sufficient for stratifying the tumour into this subtype [5, 20]. The method has a sensitivity of 96% and a specificity of 100% [21]. Demographically, this is the oldest cohort, with a low BMI; tumours in this group are typically diagnosed at later stages, are characterised by a poor prognosis, and up to 70% of endometrial cancer mortality

is associated with this cohort. TP53 mutations are typically characteristic of non-endometrioid carcinomas; they are found in 93% of serous carcinomas, 85% of carcinosarcomas, 38% of clear cell tumours, 22% of high-grade endometrioid carcinomas, and only 5% of low-grade endometrioid carcinomas [22].

Identification of NSMP (no specific molecular profile) tumours. Classification into this molecular subtype is performed 'by default' in the absence of all the features listed above. The NSMP molecular profile is characterised by wild-type TP53 and POLE, MSS, and high expression of progesterone and oestrogen receptors [5]. Morphologically, NSMP tumours are endometrioid adenocarcinomas in 50% of cases [8]. From a demographic perspective, patients in this subgroup have the highest BMI. If molecular classification tools are unavailable, the classification of endometrial carcinoma should be based on traditional morphological criteria [9].

The possibility of determining the molecular subtype using endometrial scraping material is under discussion. A. Talhouk et al. conducted a similar study and demonstrated agreement between molecular subtype determination using biopsy material and the final analysis of postoperative material. The highest sensitivity and specificity rates were demonstrated for tumours in the p53abn group (1 and 0.98, respectively) . In the MMRd group of tumours, sensitivity was 0.94 and specificity 0.93. The POLEmut and NSMP groups were characterised by low sensitivity (0.82 and 0.84) but high specificity (0.98 and 0.97). The determination of molecular subtypes of endometrial tumours based on biopsy data can be used to plan treatment strategies and will enable the use of organ-preserving techniques [23].

Prognostic significance of molecular classification. In 2018, The Lancet Oncology published the final results of the phase III international, open-label, multicentre, randomised PORTEC-3 trial, which compared the effects of adjuvant radiotherapy and chemoradiotherapy on overall and recurrence-free survival in high-risk endometrial cancer [24]. Patients were randomly assigned in a 1:1 ratio to the chemoradiotherapy (CRT) and radiotherapy (RT) groups. Alicia León Castillo et al. performed a molecular analysis of the PORTEC-3 cohort [25].

The 5-year recurrence-free survival rate was 48% for the p53abn group, 98% for POLEmut carcinomas, 72% for the MMRd subtype and 74% for the NSMP group. The use of chemoradiotherapy in the p53abn group had a significant effect on recurrence-free survival compared with radiotherapy alone ($p = 0.019$), regardless of tumour histological type (5-year recurrence-free survival was 58.6% in the CRT group and 36.2% in the RT group).

The POLEmut group was characterised by high overall and recurrence-free survival rates regardless of the adjuvant therapy method (100% in the CHT group versus 96% in the RT group). According to a meta-analysis conducted by A. Jamieson et al., de-escalation of treatment appears to be a safe step, with relapses observed in only 11 out of 294 patients (3.7%) [26]. However, such a decision can only be made after confirmation of a pathogenic POLE mutation, as many mutations in this gene are not pathogenic and may lead to misinterpretation. For patients in the MMRd group, the prognosis was intermediate: 5-year overall and recurrence-free survival rates were 84.0% and 75.5% in the LT group compared with 78.6% and 68.0%, respectively, in the HLT group ($p = 0.429$). Thus, the use of CHT did not reduce the risk of local progression in patients in the MMRd group [25]. However, when radiotherapy was used as adjuvant therapy, this had a statistically significant effect on overall ($p = 0.032$) and recurrence-free survival ($p = 0.020$), as demonstrated in a retrospective multicentre cohort study by C. Reijnen et al. [27].

Furthermore, determining MMR status opens up potential access to treatments such as immune checkpoint inhibitors. The combination of pembrolizumab and lenvatinib has been approved by the FDA as a second-line drug therapy for the treatment of RCC [28].

In the NSMP group, there was a trend towards improved 5-year disease-free survival when CHT was used as adjuvant therapy (5-year DFS 79.7% in the CHT group compared with 67.7% in the RT group) ($p = 0.243$) [25]. NSMP tumours, in addition to high expression of oestrogen and progesterone receptors, demonstrated an activated PI3-kinase pathway (PI3K/AKT/mTOR); therefore, the use of everolimus and letrozole in the treatment of recurrent ER-positive breast cancer in phase II of the B.M. Slomovitz showed an objective response rate of 32% [29]. In a study by M. Mirza et al., the combination of letrozole and the cyclin-dependent kinase inhibitor palbociclib demonstrated a 64% higher disease control rate than with letrozole alone (an increase in the median recurrence-free survival of 5 months) [30].

Nevertheless, given the absence of specific molecular markers in this category, adjuvant therapy is likely to continue to depend largely on clinical and pathological criteria.

Outlook. Molecular classification and subsequent surrogate classification have opened up a promising new avenue by classifying ER-positive breast cancer into four molecular subgroups. This allows for the precise determination of recurrence risk and represents a promising clinical tool for deciding on the composition of adjuvant therapy. The integration of molecular classification into clinical practice requires a move away from empirical approaches when prescribing adjuvant therapy, as the latter must correspond precisely to the

specific risk group. In particular, the group with POLE mutations has a low risk of recurrence and probably does not require adjuvant treatment. On the other hand, tumours with TP53 mutations have a high risk of recurrence, which necessitates the use of drug therapy as part of adjuvant therapy. Clinical trials such as PORTEC-4a, RAINBO, CANSTAMP and TAPER have now commenced and are ongoing; these are likely to provide key information for precision approaches to the treatment of ER [15, 31, 32].

References

1. Colombo N, Creutzberg C, Amant F, et al. ESMO-ESGO-ESTRO Consensus Conference on Endometrial Cancer: diagnosis, treatment and follow-up. Ann Oncol. 2016;27(1):16-41. http://dx.doi.org/10.1093/annonc/mdv484.

2. Rybin, A., A. Varabina, and M. Broshkov. ""Epidemiology and management of ovarian cancer based on the clinical experience of the southern region of Ukraine." *Georgian Medical News* 288 (2019): 32-36.

3. Rybin A, Dubinina V, Kuznetsova O. Personalized managing of the patients with serous ovarian cancer. InINTERNATIONAL JOURNAL OF GYNECOLOGICAL CANCER 2016 Sep 1 (Vol. 26, pp. 35-35). TWO COMMERCE SQ, 2001 MARKET ST, PHILADELPHIA, PA 19103 USA: LIPPINCOTT WILLIAMS & WILKINS.

4. Goebel EA, Vidal A, Matias-Guiu X, et al. The evolution of endometrial carcinoma classification through application of immunohistochemistry and molecular diagnostics: past, present and future. Virchows Archiv. 2017;472(6):885-96. http://dx.doi.org/10.1007/s00428-017-2279-8.

5. Kolesnik O.O., Rybin A.I. Predicting the effectiveness of treatment for ovarian cancer patients. Current issues in paediatrics, obstetrics and gynaecology. – 2017. – No. 2. – pp. 40–47. DOI 10.11603/24116-4944.2017.2.8043.

6. Concin N, Matias-Guiu X, Vergote I, et al. ESGO/ESTRO/ ESP guidelines for the management of patients with endometrial carcinoma. Int J Gynecol Cancer. 2021;31(1):12-39. http://dx.doi.org/10.1136/ijgc-2020-002230.

7. Oaknin A, Bosse TJ, Creutzberg CL, et al. Electronic address: clinicalguidelines@esmo.org. Endometrial cancer: ESMO Clinical Practice Guideline for diagnosis, treatment and follow-up. Ann Oncol. 2022;33(9):860-877. http://dx.doi.org/10.1016/j.annonc.2022.05.009.

8. Kolesnik O.O., Rybin A.I. Personalised treatment of ovarian cancer patients. Hospital Surgery. Journal named after L. Ya. Kovalchuk. – 2017. – No. 2 (78). – P. 5-12. DOI 10.11603/2414-4533.2017.2.7951.

9. RYBIN, Andriy and LISKOVSKYI, Sergiy. Epidemiology of papillomavirus infection in Ukraine. Pedagogy and Psychology of Sport. Online. 31 October 2025. Vol. 26, p. 66875. [Accessed 10 December 2025]. DOI 10.12775/PPS.2025.26.66875.

10. Rybin A, Maksymovskyi V, Kuznetsova O, Osyk V, Bohdan A. THE RESULTS OF LIFE QUALITY ASSESSMENT IN PATIENTS WITH PRIMARY OVARIAN CANCER DURING TREATMENT: EFFECT OF DIFFERENT TACTICS AND HIPEC. Georgian Med News. 2025 Jul-Aug;(364-365):364-368. PMID: 41072538.

11. Rybin A. I., O. V. Kuznetsova, and V. E. Maximovsky. "Modern strategies of surgical treatment of patients with pelvic canceromatosis caused by the ovarian cancer. Visnyk mors′koyi medytsyny 2020; 2 (87): 55–61. DOI http://dx.doi.org/10.5281/zenodo.3967728.

12. Rybin A. (2022). Results of personalised treatment of ovarian cancer in patients with peritoneal carcinomatosis // Reproductive Health of Women, (7), 35–40. https://doi.org/10.30841/2708-8731.7.2022.272470.

13. RYBIN, A., MAKSYMOVSKYI, V., KUZNETSOVA, O. and ZAMYSHLYAK, V. Personalised treatment of patients with advanced ovarian cancer: comparative analysis of surgical approaches. Journal of Education, Health and Sport. Online. 30 June 2025. Vol. 83, p. 64576. DOI 10.12775/JEHS.2025.83.64576.

14. MAKSYMOVSKYI, V., RYBIN, A. and ZAMYSHLYAK, V. Quality of life in patients with advanced ovarian cancer under personalised treatment: the impact of HIPEC. Journal of Education, Health and Sport. Online. 11 August 2025. Vol. 84, p. 64751. DOI 10.12775/JEHS.2025.84.64751.

15. Rybin, A. I., Maksymovskyi, V. Ye., Kuznetsova, O. V., & Zamyshlyak, V. I. (2025). Assessment of the quality of life of patients with primary ovarian cancer during personalised treatment. Reproductive Health of Women, (7), 73-79. https://doi.org/10.30841/2708-8731.7.2025.343883.

16. Rybin A.I. The role of proteomic and genetic predictors of survival in ovarian cancer patients. Clinical Oncology, 2016, No. 3 (23), pp. 46-48.

17. Rybin, A. I., et al. "QUINTIP RECORDATI INITIAL DATA CONCERNING THE PECULIARITIES OF HUMAN PAPILLOMAVIRUS INFECTION EPIDEMIOLOGY IN UKRAINE AND THE PROSPECTS FOR ITS PREVENTION." *World of Medicine and Biology* 21.93 (2025): 242-250. DOI 10.26724/2079-8334-2025-3-93-242-250.

18. Rybin A. I., Lysenko M. A., Rysina A. I. Features of the sanogenesis system in patients with ovarian cancer resistant to platinum-based chemotherapy. Collection of

scientific works of the Association of Obstetricians and Gynaecologists of Ukraine. 2014. 1-2. 251-254.

19. Stelloo E, Jansen AML, Osse EM, et al. Practical guidance for mismatch repair-deficiency testing in endometrial cancer. Ann Oncol. 2017;28(1):96-102. http://dx.doi.org/10.1093/annonc/mdw542.

20. Rybin A.I., Kuznetsova O.V. Second line treatment of recurrent ovarian cancer: from clinical trials to modern military realities // International scientific journal 'Grail of Science', No. 26, 2023.-P. 478-482. https://doi.org/10.36074/grail-of-science.14.04.2023.085.

21. Vermij L, Smit V, Nout R, et al. Incorporation of molecular characteristics into endometrial cancer management. Histopathology. 2020;76(1):52-63. http://dx.doi.org/10.1111/ his.14015.

22. RYBIN, A. Biomarkers of platinum resistance in serous ovarian cancer. Journal of Education, Health and Sport. Online. 25 September 2025. Vol. 85, p. 65774. DOI 10.12775/JEHS.2025.85.65774.

23. Rybin A. I., et al. Initial data from Quintip Recordati on the epidemiology of papillomavirus infection in Ukraine and prospects for its prevention. World of Medicine and Biology No. 3(93), 2025, pp. 242–250. DOI 10.26724/2079-8334-2025-3-93-242-250.

24. RYBIN, Andriy. Modern strategies of platinum resistance overcoming in patients with the serous ovarian cancer. Journal of Education, Health and Sport. Online. 30 November 2018. Vol. 8, no. 11, pp. 594-599.

25. McAlpine J, Leon-Castillo A, Bosse T. The rise of a novel classification system for endometrial carcinoma; integration of molecular subclasses. The Journal of Pathology. 2018;244(5):538-49. http://dx.doi.org/10.1002/path.5034.

26. Rybin, A. I., et al. 'Assessment of quality of life in patients with primary ovarian cancer during personalised treatment.' Women's Reproductive Health 7 (2025): 73–79. https://doi.org/10.30841/2708-8731.7.2025.343883.

27. Rybin, A., et al. "THE RESULTS OF LIFE QUALITY ASSESSMENT IN PATIENTS WITH PRIMARY OVARIAN CANCER DURING TREATMENT: EFFECT OF DIFFERENT TACTICS AND HIPEC." Georgian Medical News 364-365 (2025): 364-368.

28. Rybin, A. I., et al. 'Innovative approaches to the management of cancer patients.' (2025). Current Issues in Transport Medicine. - 2025. - No. 1 (79). – pp. 131–139. http://dx.doi.org/10.5281/zenodo.15069303.

29. Mitric C, Bernardini MQ. Endometrial cancer: transitioning from histology to genomics. Current Oncology. 2022;29(2):741-57. http://dx.doi.org/10.3390/curroncol29020063.

30. Kuznetsova O.V. Hepatotoksychnost′ razlychnykh skhem khymyoterapevtycheskoho lechenyya opukholey zhenskoy reproduktyvnoy systemy / O.V. Kuznetsova, A.Y. Rybyn, V.H. Dubynyna // Arkhiv klinichnoyi ta eksperymental′noyi medytsyny. – 2014. – tom 22, № 1. – S. 60–64.

31. Mitric C, Bernardini MQ. Endometrial cancer: transitioning from histology to genomics. Current Oncology. 2022;29(2):741-57. http://dx.doi.org/10.3390/curroncol29020063.

32. RAINBO Research Consortium. Refining adjuvant treatment in endometrial cancer based on molecular features: the RAINBO clinical trial program. Int J Gynecol Cancer. 2022;33(1):10917. http://dx.doi.org/10.1136/ijgc-2022-004039.