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Molecular genetic markers in predicting the clinical course of cervical cancer according to data from the Samarkand regional branch of the Republican Specialized Scientific and Practical Medical Center of Oncology and Radiology.

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Article History	Abstract
Received: 7th March 2026 Accepted: 6th April, 2026	Cervical cancer (CC) remains a global health problem, particularly in developing countries. According to GLOBOCAN 2022, 604,000 new cases and 342,000 deaths from CC were recorded worldwide in 2020. In the structure of oncological morbidity and mortality in women worldwide, cervical cancer ranks fourth, and in developing countries this type of oncological pathology ranks first, while in economically developed countries it ranks third after uterine and ovarian cancer.
Keywords:	

When studying age groups of this category of patients, cervical cancer is increasingly being recorded among women of reproductive age, and the clinical course is more aggressive. Genetic factors play a key role in the disease, determining both predisposition and its clinical course [5]. This indicates the need for a more in-depth study of the molecular genetic factors contributing to the development of this pathology [10].

One such factor may be vascular endothelial growth factor A (VEGFA), which plays a key role in angiogenesis, the process of formation of new blood vessels vital for tumor growth and metastasis [6]. The VEGFA gene exhibits pronounced polymorphism, and its various variants can affect the level of protein expression, thereby promoting tumor progression [7]. Of particular interest is the single nucleotide polymorphism C-634G (rs 2010963), located in the promoter region

of the VEGFA gene, which, according to a number of studies, is associated with various types of cancer [8].

In addition, special attention is paid to genetic polymorphisms that can modify the individual risk of developing malignant neoplasms. The TGFB1 gene, encoding transforming growth factor β1 (TGF-β1), plays an important role in the regulation of cell proliferation, apoptosis, and the immune response [10]. The Arg25Pro polymorphism (rs1800471) can affect the expression level and functional activity of TGF-β1, which is potentially associated with oncogenesis [10].

Human papillomavirus is recognized as the main etiologic cause of cervical cancer; however, not all infected women develop the disease, suggesting a role for genetic factors in carcinogenesis [7]. The EDN1 gene, encoding endothelin-1, plays an important role in the regulation of angiogenesis, proliferation, and invasion of tumor cells. The Lys197Asn polymorphism in this gene can alter the biological properties of the protein and may influence susceptibility to cervical cancer [12].

The aim of the study: to study the polymorphism C-634G of the VEGFA gene, Ala114Val of the GSTP1 gene, Lys197Asn of the EDN1 gene and to predict the clinical course of each case of cervical cancer.

Research materials and methods: The study was conducted using the SNP method and the SNP Express test systems from the Scientific and Production Company Sintol. The work was conducted at the Research Institute of Hematology and Blood Transfusion of the Ministry of Health of the Republic of Uzbekistan, in the Department of Molecular Medicine and Cellular Technologies. Genotyping of these markers consisted of several stages: peripheral blood sampling, DNA extraction from lymphocytes, and polymorphism detection by PCR.

A case-control study (comparison of two comparative samples) was conducted. The case sample consisted of 72 patients, including 30 patients from the recurrent group and 42 patients from the study group undergoing evaluation at the Samarkand Branch of the Republic Scientific and Practical Medical Center of Oncology and Radiology. The study group included patients diagnosed with squamous cell carcinoma of the cervix (T1b-3a NxM0).

The average age of these patients was 45 years. The control sample consisted of 120 apparently healthy donors of Uzbek ethnicity, without any history of

oncological pathology. The control group did not differ in age from the group of cervical cancer patients.

Study results. An association analysis of the C-634G polymorphism of the VEGFA gene with the development of cervical cancer was conducted by comparing three samples (patients with newly diagnosed cervical cancer - Group 1, patients with recurrent cervical cancer after complex treatment - Group 2, and apparently healthy individuals - Group 3) using a case-control model.

Table 1.

Comparative assessment of the allele and genotype frequencies of the C-634G polymorphism in the VEGFA gene.

№	Group	Allele frequency				Genotype distribution frequency					
		C		G		C/C		C/G		G/G	
		n	%	n	%	n	%	n	%	n	%
	Total number of patients (n = 72)	113	78.5	31	21.5	47	65.3	19	26.4	6	8.33
1	Recurrent group (n = 30)	38	63.3	22	36.7	12	40	14	46.7	4	13.3
2	Study group (n = 42)	75	89.3	9	10.7	35	83.3	5	11.9	2	4.76
3	Control group (healthy group) (n = 75)	123	82.0	27	18.0	51	68.0	21	28.0	3	4.0

Statistical analysis of expected and observed frequencies was performed using the Hardy-Weinberg equilibrium distribution (HWED). Comparative analysis revealed a tendency toward deviation from Hardy-Weinberg equilibrium in the study group, primarily due to an excess of homozygotes for the G allele and a deficit of heterozygotes. This may indicate a possible genotype influence on the pathological condition (cervical cancer) in the population (see Table 1).

Table 2

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Differences in the frequency of allelic and genotypic variants of the C-634G polymorphism in the VEGFA gene in patient groups.

Alleles and genotypes	Number of alleles and genotypes				χ2	p	RR	95%CI	OR	95%CI
	Study group		Control group							
	n	%	n	%						
C	113	78,5	123	82,0	0,6	0,6	1,0	0,56 - 1,64	0,8	0,45 - 1,42
G	31	21,5	27	18,0	0,6	0,6	1,0	0,58 - 1,89	1,2	0,7 - 2,22
C/C	47	65,3	51	68,0	0,1	0,8	1,0	0,49 - 1,88	0,9	0,45 -1,76
C/G	19	26,4	21	28,0	0,0	0,9	0,9	0,45 - 1,98	0,9	0,45 -1,91
G/G	6	8,3	3	4,0	1,2	0,4	2,1	0,79 - 5,48	2,2	0,54 -8,81

The results of the genotype distribution frequency analysis showed that the homozygous genotype C/C prevailed in the control group compared to the study group, with a frequency of 68.0% versus 65.3%, respectively (χ2=0.1; p=0.8; RR=1.0; 95% CI: 0.49-1.88; OR=0.9; 95% CI: 0.45-1.76). The heterozygous genotype C/G also prevailed in the control group compared to the study group with a frequency of 28.0% versus 26.4%, respectively (χ2=0.0; p=0.9; RR=0.9; 95% CI: 0.45-1.98; OR=0.9; 95% CI: 0.45-1.91). The homozygous mutant genotype G/G was more common in the study group compared to the control group with a frequency of 8.3% versus 4.0%, respectively (χ2=1.2; p=0.4; RR=2.1; 95% CI: 0.79-5.48; OR=2.2; 95% CI: 0.54-8.81. (see Tables 1, 2).

Analysis of the allele distribution frequency of the C634G polymorphism in the VEGFA gene revealed the presence of statistically significant differences between the subgroup of patients with cervical cancer recurrence and the group of healthy controls, while the odds ratio OR=0.4 (95%CI: 0.2-0.73) indicates a significant protective effect of the C allele, reducing the likelihood of disease recurrence by more than 2 times. At the same time, the G allele was statistically

significantly ($\chi2$=8.3; p=0.01) predominant in patients with recurrence (36.7%), and the odds ratio (OR=2.6; 95%CI: 1.37-5.09) indicates its association with an increased risk of recurrence.

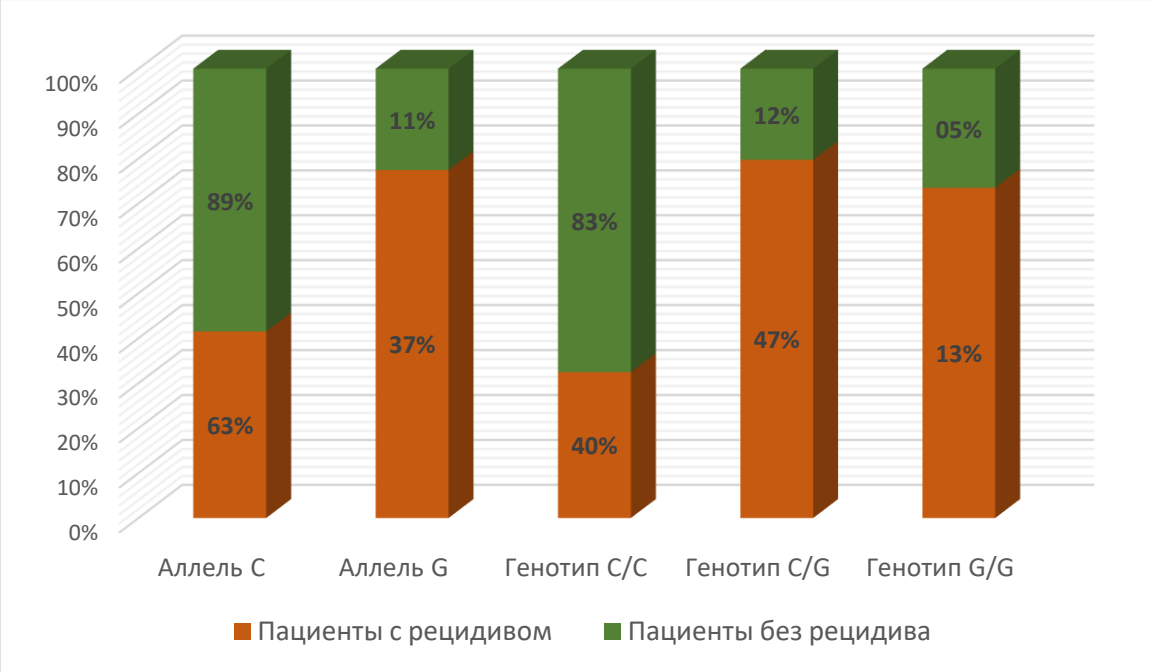

Figure 1. Frequency of allelic and genotypic variants of the C634G polymorphism in the VEGFA gene in subgroups of patients with and without recurrent.

The genotype distribution of the C634G polymorphism in the VEGFA gene demonstrated significant differences between subgroups. The homozygous C/C genotype was significantly more prevalent in patients without recurrent and the control group (83.3%). Statistical data ($\chi2$=14.0; p=0.01; OR=0.1; 95% CI: 0.05-0.38) indicate its protective role. The heterozygous C/G genotype was significantly ($\chi2$=10.9; p=0.01; OR=6.5; 95% CI: 2.13-19.64) more common in the recurrent subgroup (13.3%), indicating an increased risk of recurrence in carriers of the heterozygous C/G genotype. The frequency of the homozygous G/G genotype in the subgroups was statistically significant ($\chi2$=1.7; p=0.3; OR=3.1; 95% CI: 0.56-16.81). (Figure 1).

Thus, the C634G/C polymorphism of the VEGFA gene may serve as a prognostic marker for cervical cancer, with the G allele associated with an increased risk of disease recurrence and the C allele as a potential protective marker.

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Table 3.
Allele and genotype distribution frequencies of the Arg25Pro polymorphism in the TGFb1 gene in patient and control groups.

Num	Group	Allele frequency				Genotype distribution frequency					
		Arg		Pro		Arg/Arg		Arg/Pro		Pro/Pro	
		n	%	n	%	n	%	n	%	n	%
1	Study group (n = 72)	121	84,03	23	15,97	53	73,61	15	20,83	4	5,56
2	Recurrent (n = 30)	45	75	15	25	18	60	9	30	3	10
3	Non-recurrent (n = 42)	76	90,48	8	9,52	35	83,33	6	14,29	1	2,38
4	Control group (n = 75)	140	93,33	10	6,67	66	88	8	10,67	1	1,33

Statistical analysis of expected and observed frequencies was performed using the Hardy-Weinberg equilibrium distribution (HWED). Comparative analysis revealed that the study group exhibited a borderline statistically significant deviation from the Hardy-Weinberg equilibrium ($\chi 2=3.6$; $p=0.05$), while the control group's distribution corresponded to equilibrium. In the other subgroups, deviations from the Hardy-Weinberg equilibrium were statistically insignificant.

The results of the analysis of the allele distribution frequency of the Arg25Pro polymorphism in the TGFb1 gene for the presence of differences in their distribution in the study group of patients and in the group of healthy controls showed that the Arg allele did not prevail in the control group compared to the study group of patients, and its frequency was 93.3% versus 84.0% ($\chi 2=6.4$; $p=0.1$; RR=0.9; 95% CI: 0.54-1.5; OR=0.4; 95% CI: 0.18-0.8), and the Pro allele also did not prevail in the study group of patients compared to the control group, its frequency was 16.0% versus 6.7%, respectively ($\chi 2=6.4$; $p=0.1$). However, the calculated chance of detecting a mutant allelic variant among patients was 2.7 times compared to the control (OR=2.7; 95% CI: 1.25-5.69).

The calculated odds ratio (OR = 0.4; 95% CI: 0.16-0.89) indicates a possible protective role of the wild-type genotype of this gene. The Arg/Pro and Pro/Pro genotypes were more common in the patient group (20.8% and 5.6%) than in the control group (10.7% and 1.3%), but also without statistical significance (p = 0.2 and p = 0.3). (See Tables 5.2.3).

Table 4.

Differences in the frequency of allelic and genotypic variants of the Arg25Pro polymorphism in the TGFb1 gene in the recurrent patient group and the control group.

Alleles and genotypes	Number of alleles and genotypes				*χ2*	*p*	*RR*	*95%CI*	*OR*	*95%CI*
	Recurrent group		Control group							
	n	%	n	%						
Arg	45	75,0	140	93,3	13,7	0,01	0,8	0,36 - 1,79	0,2	0,09 - 0,48
Pro	15	25,0	10	6,7	13,7	0,01	1,2	0,48 - 3,23	4,7	2,07 - 10,54
Arg/Arg	18	60,0	66	88,0	10,5	0,01	0,7	0,23 - 2,01	0,2	0,08 - 0,53
Arg/Pro	9	30,0	8	10,7	5,9	0,08	2,8	0,9 - 8,82	3,6	1,28 - 10,06
Pro/Pro	3	10,0	1	1,3	4,4	0,10	7,5	2,09 - 26,88	8,2	1,15 - 58,98

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Analysis of the allele distribution frequency of the Arg25Pro polymorphism in the TGFb1 gene between patients with recurrent cervical cancer and the control group revealed significant differences ($\chi 2=13.7$; $p=0.01$). The Arg allele was significantly more common in the control group (93.3%), with an odds ratio (OR) of 0.2 (95%CI: 0.09-0.48) indicating a significant protective effect of this allele, reducing the likelihood of disease recurrence by more than 2-fold.

In turn, the Pro allele was significantly ($\chi 2=13.7$; $p=0.01$) more prevalent in patients with recurrent (25.0%) compared to the control group (6.7%) with OR=4.7 (95% CI: 0.48-3.23), which indicates a potential association of this allele with an increased risk of disease recurrent.

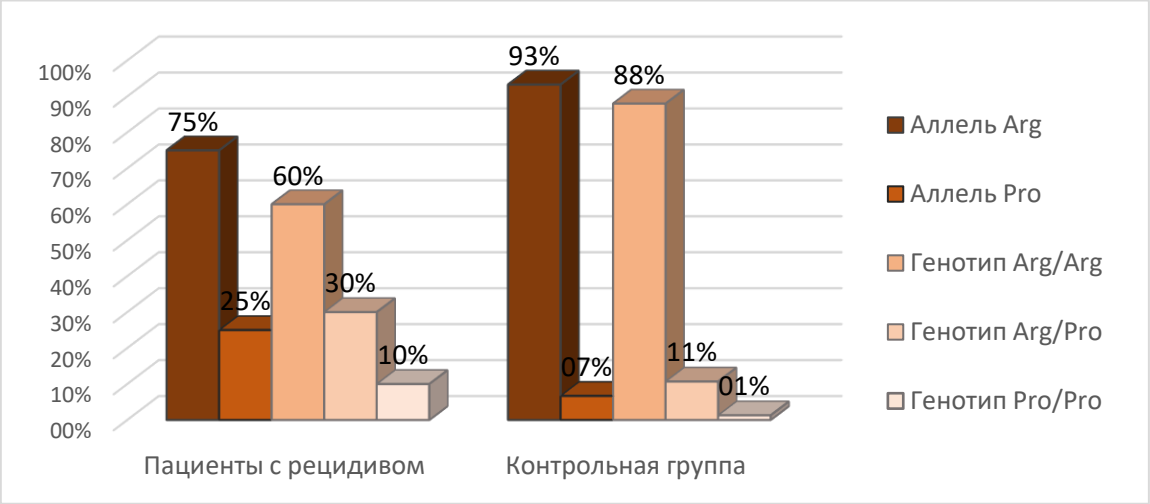

Figure 2. Frequency of allelic and genotypic variants of the Arg25Pro polymorphism in the TGFb1 gene in the group of patients with recurrence and in the control group.

The results of the analysis of the genotype distribution frequency of the Arg25Pro polymorphism in the TGFb1 gene for differences in their distribution in the

subgroup of patients with recurrence and in the group of healthy controls showed that the homozygous Arg/Arg genotype was significantly more prevalent in the control group compared to the subgroup of patients with recurrence, its frequency was 88.0% versus 60.0%, respectively ($\chi2$=10.5; p=0.01; RR=0.7; 95% CI: 0.23-2.01; OR=0.2; 95% CI: 0.08-0.53). The heterozygous Arg/Pro genotype, on the c).

Analysis of the distribution of genotypes of the Arg25Pro polymorphism in the TGFb1 gene also revealed significant differences in the homozygous genotype Arg/Arg, which was significantly more common ($\chi2$=10.5; p=0.01) in the control group (88.0%) than in the subgroup of patients with recurrence (60.0%), and the odds ratio (OR=0.2; 95% CI:0.08-0.53) indicates a protective role of this genotype. The heterozygous Arg/Pro genotype was more common in patients with recurrence (30.0%) compared to the control group, but the difference was not statistically significant (p=0.08). The odds ratio (OR=3.6) (95% CI: 1.28-10.06) suggests a potential association with an increased risk of disease recurrence.

The homozygous Pro/Pro genotype was also more common in the subgroup of patients with recurrence (10.0%) than in the control group (1.3%), but the difference was also statistically insignificant, although the values of the risk and odds ratio (RR=7.5; 95% CI: 2.09-26.88; OR=8.2; 95% CI: 1.15-58.98) indicate a possible association of this genotype with the risk of recurrence. (See Tables 4, Figure 1).

Table 5.

Differences in the frequency of allelic and genotypic variants of the Arg25Pro polymorphism in the TGFb1 gene in the subgroup of patients without recurrence and in the control group.

Alleles and genotypes	Number of alleles and genotypes				$\chi2$	p	RR	95%CI	OR	95%CI
	Study group		Control group							
	n	%	n	%						
Arg	76	90,5	140	93,3	0,6	0,6	1,0	0,33 - 2,83	0,7	0,26 - 1,78

Pro	8	9,5	10	6,7	0,6	0,6	1,0	0,45 - 2,37	1,5	0,56 - 3,87
Arg/Arg	35	83,3	66	88,0	0,5	0,6	0,9	0,28 - 3,17	0,7	0,24 - 1,98
Arg/Pro	6	14,3	8	10,7	0,3	0,7	1,3	0,37 - 4,88	1,4	0,45 - 4,32
Pro/Pro	1	2,4	1	1,3	0,2	0,7	1,8	0,11 - 28,17	1,8	0,11 - 28,51

The results of the analysis of the allele distribution frequency of the Arg25Pro polymorphism in the TGFb1 gene for the presence of differences in their distribution in the subgroup of patients without recurrence and in the group of healthy controls showed that the Arg allele was insignificantly predominant in the control group compared to the subgroup of patients without recurrence, its frequency was 93.3% versus 90.5%, respectively ($\chi2$=0.6; p=0.6; RR=1.0; 95% CI: 0.33-2.83; OR=0.7; 95% CI: 0.26-1.76), and the Pro allele was insignificantly predominant in the subgroup of patients without recurrence compared to the control group, its frequency was 9.5% versus 6.7%, respectively ($\chi2$=0.6; p=0.6; RR=1.0; 95% CI: 0.45-2.37; OR=1.5; 95%CI: 0.56-3.87).

Thus, analysis of the distribution of the Arg25Pro polymorphism alleles in the TGFb1 gene in the subgroup of patients without cervical cancer recurrence and the control group revealed no statistically significant differences ($\chi2$=0.6; p=0.6). The data do not support a reliable association between the alleles and the absence of disease recurrence.

Table 6.

Differences in the frequency of allelic and genotypic variants of the Arg25Pro polymorphism in the TGFb1 gene in subgroups of patients with and without recurrence

Alleles and genotypes	Number of alleles and genotypes				$\chi2$	p	RR	95%CI	OR	95%CI
	With relapse		Without relapse							
	n	%	n	%						

Arg	45	75,0	76	90,5	6,2	0,1	0,8	0,4 - 1,74	0,3	0,13 - 0,78
Pro	15	25,0	8	9,5	6,2	0,1	1,2	0,39 - 3,73	3,2	1,28 - 7,82
Arg/Arg	18	60,0	35	83,3	4,9	0,1	0,7	0,27 - 1,95	0,3	0,1 - 0,87
Arg/Pro	9	30,0	6	14,3	2,6	0,3	2,1	0,74 - 5,99	2,6	0,82 - 8,07
Pro/Pro	3	10,0	1	2,4	1,9	0,3	4,2	1,21 - 14,64	4,6	0,54 - 38,56

The results of the analysis of the allele distribution frequency of the Arg25Pro polymorphism in the TGFb1 gene for the presence of differences in their distribution in the subgroups of patients with and without recurrence showed that in the subgroup of patients without recurrence, compared with the subgroup of patients with recurrence, the Arg25Pro allele was 90.5% versus 75.0% ($\chi2$=6.2; p=0.1; RR=0.8; 95% CI: 0.4-1.74; OR=0.3; 95% CI: 0.13-0.78), and the Pro allele prevailed in the subgroup of patients with recurrence compared with the subgroup without recurrence, its frequency was 25.0% versus 9.5%, respectively ($\chi2$=6.2; p=0.1; RR=1.2; 95% CI: 0.39-3.73; OR=3.2; 95%CI: 1.28-7.82).

Analysis of the genotype distribution of the Arg25Pro polymorphism in the TGFb1 gene demonstrated a statistically significant difference between the subgroups (p=0.1). The homozygous wild genotype Arg/Arg prevailed in patients without recurrence (83.3%), the calculated odds ratio (OR=0.3; 95% CI: 0.1-0.87) may indicate its possible role in relation to relapse. The heterozygous genotype Arg/Pro was more common in the subgroup with relapse (30.0%), but the differences were insignificant. The homozygous genotype Pro/Pro was also more common in the subgroup of patients with recurrence (10.0%), but the differences were not statistically significant (p=0.3), and the odds ratio RR=4.2 (95% CI: 1.21-14.64) suggest a possible pathogenic role of this genotype.

Table 7

Allele and genotype distribution frequencies of the Lys197Asn polymorphism in the EDN1 gene in patient and control groups.

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Num	Group	Allele frequency				Genotype distribution frequency					
		Lys		Asn		Lys/Lys		Lys/Asn		Asn/Asn	
		n	%	n	%	n	%	n	%	n	%
1	Study group (n = 72)	103	71,53	41	28,47	37	51,39	29	40,28	6	8,33
2	Recurrent (n = 30)	34	56,67	26	43,33	8	26,67	18	60	4	13,33
3	Non-recurrent (n = 42)	69	82,14	15	17,86	29	69,05	11	26,19	2	4,76
4	Control group (n = 75)	127	84,67	23	15,33	54	72	19	25,33	2	2,67

The results of the analysis of the distribution frequency of the alleles of the Lys197Asn polymorphism in the EDN1 gene for the presence of differences in their distribution in the study group of patients and in the group of healthy controls showed that the Lys allele was significantly predominant in the control group compared to the study group of patients, and its frequency was 84.7% versus 71.5%, respectively ($\chi2=7.4$; $p=0.01$; RR=0.8; 95% CI: 0.54-1.33; OR=0.5; 95% CI: 0.26-0.8), and the Asn allele was statistically significantly dominant in the study group of patients compared to the control group, its frequency was 28.5% versus 15.3%, respectively ($\chi2=7.4$; $p=0.01$; RR=1.2; 95% CI: 0.6-2.34; OR=2.2; 95%CI: 1.25-3.87).

Analysis of differences in the allele frequencies of the Lys197Asn polymorphism in the EDN1 gene showed that the Lys allele was significantly more common in the control group than in the study group of patients, indicating its protective role against the development of cervical cancer ($\chi2=7.4$; $p=0.01$; OR=0.5; 95% CI: 0.26-0.8). The Asn allele was significantly more common in patients with cervical cancer (28.5% versus 15.3%), indicating a potential association of this allele with an increased risk of the disease ($\chi2=7.4$; $p=0.01$; OR=2.2; 95% CI: 1.25-3.87), that is, the presence of the Asn allele increases the risk of the disease by 2.2 times.

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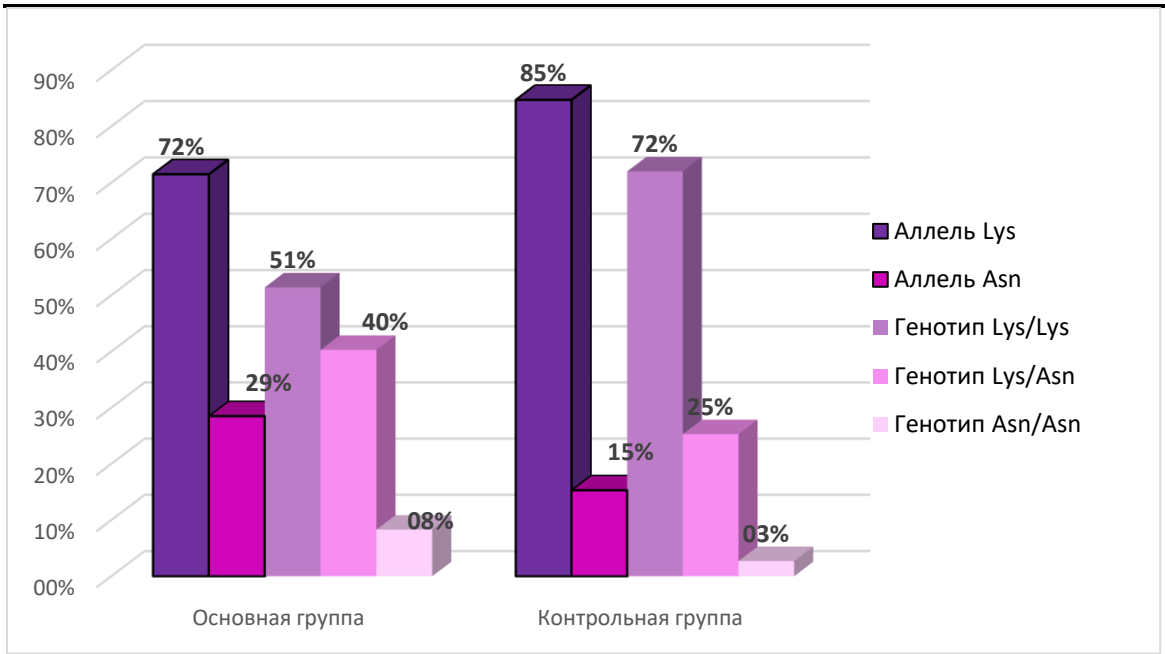

Figure 3. Frequency of allelic and genotypic variants of the Lys197Asn polymorphism in the EDN1 gene in patient groups and in the control group

The results of the analysis of the distribution frequency of the Lys197Asn polymorphism alleles in the EDN1 gene for the presence of differences in their distribution in the subgroup of patients with recurrence and in the group of healthy controls showed that the Lys allele was significantly predominant in the control group compared to the subgroup of patients with recurrence, its frequency was 84.7% versus 56.7%, respectively ($\chi 2$=18.8; p=0.01; RR=0.7; 95% CI: 0.31-1.46; OR=0.2; 95% CI: 0.12-0.45). The Asn allele was significantly more prevalent in the subgroup of patients with recurrence compared to the control group, its frequency was 43.3% versus 15.3%, respectively ($\chi 2$=18.8; p=0.01; RR=1.5; 95%CI:0.82-2.73; OR=4.2; 95%CI:2.2-8.1).

Analysis of the distribution frequency of alleles of the Lys197Asn polymorphism in the EDN1 gene revealed statistically significant differences between the subgroup of patients with recurrence and the control group, indicating a possible association of this allele with an increased risk of disease recurrence, while the risk of recurrence may increase by 4.2 times in the presence of this allele.

Comparative analysis of allele distribution between the subgroups of patients with and without recurrence revealed statistically significant differences ($\chi 2$=11.2; p=0.01). The Lys allele was significantly more common in patients without recurrence (82.1%) compared to patients with recurrence (56.7%), the

odds ratio (OR=0.3; 95% CI: 0.14-0.59) indicating a possible protective role. In turn, the Asn allele was significantly more common in patients with recurrence (43.3%) compared to patients without recurrence (17.9%), while OR=3.5 (95% CI: 1.68-7.36) indicating its potential association with an increased risk of recurrence.

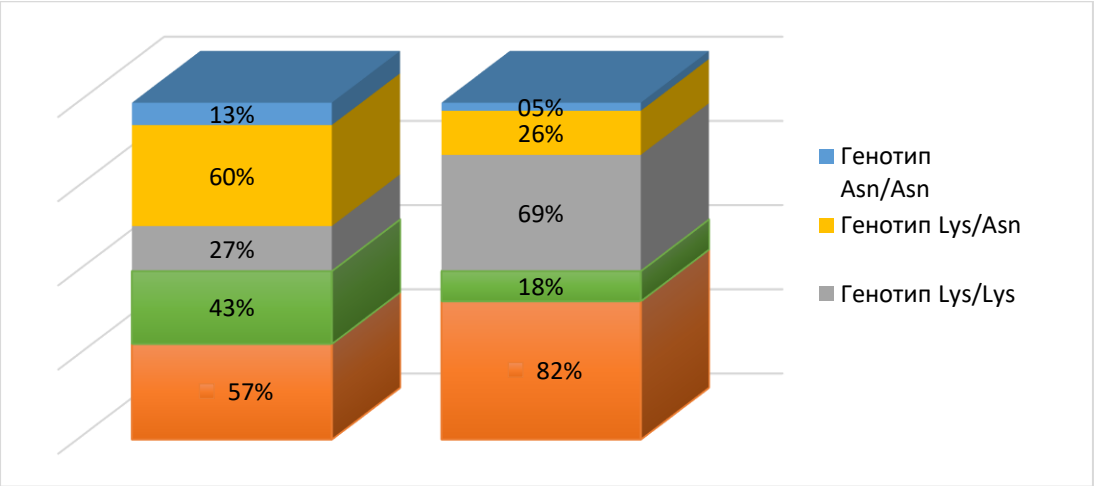

Figure 4. Frequency of allelic and genotypic variants of the Lys197Asn polymorphism in the EDN1 gene in subgroups of patients with and without recurrence.

In this study, we found that the Lys allele and the Lys/Lys genotype may be associated with a protective effect, with the protective effect of the Lys allele being associated with both primary disease development and relapse. The Asn allele and the Lys/Asn genotype demonstrated a possible association with an increased risk, with the protective effect of the Asn allele being associated with both primary disease development and relapse. The homozygous Asn/Asn genotype did not show significant differences in any of the recurrent groups, likely due to its low frequency.

The EDN1 gene may be associated with the risk of cervical cancer recurrence, increasing the feasibility of its use as a clinical marker.

Conclusion. The results of this molecular genetic study suggest a significant role for genetic polymorphisms in determining the clinical prognosis of patients with cervical cancer (CC). Despite the lack of direct associations between the studied loci and overall disease risk in the case-control model, clear genetic predictors of an unfavorable course of the disease were identified.

Carriage of the G allele of the C-634G polymorphism of the VEGFA gene and the Pro allele of the Arg25Pro polymorphism of the TGFB1 gene were found to be key determinants of a high risk of disease recurrence. Statistically significant odds ratios (ORs from 2.6 to 4.8) were found for these markers, indicating their important prognostic role. Furthermore, the heterozygous Lys/Asn genotype of the EDN1 gene significantly contributes to the risk of recurrence, increasing the likelihood of an adverse outcome by more than fourfold.

At the same time, factors such as the C allele of the VEGFA gene and the Arg allele of the TGFB1 gene demonstrate a pronounced protective effect, being associated with a more favorable clinical course.

These results may facilitate the identification of molecular genetic markers of predisposition to cervical cancer and lay the foundation for a personalized approach to treatment and prevention of relapse.

A combination of genetic profile analysis of the angiogenesis, antioxidant defense, and cytokine regulation systems allows for the stratification of patients into risk groups, which opens up opportunities for personalizing dynamic monitoring algorithms, preventing recurrence, and generally optimizing the management of patients with cervical cancer.

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