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Effect of *IL-17A* and *PPARG* gene polymorphism on the course of purulent complications after treatment of patients with lower limb bone injuries

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Abstract

Introduction Epidemiological studies have shown a link between genetic factors and the development of purulent complications after treatment in traumatology and orthopedics. Studies on candidate genes help identify mutations associated with diseases, which is important for diagnosis and prevention. The polymorphisms of the *IL-17A* and *PPARG* genes are of particular interest. The *G197A* polymorphism in the *IL-17A* gene may increase the risk of autoimmune and infectious diseases. The *C1431T* polymorphism in the *PPARG* gene is often associated with metabolic disorders, including obesity and diabetes type 2. Both polymorphisms are significant for developing methods to predict and treat purulent complications, as their impact on patient health can be substantial.

Purpose To study the influence of *IL-17A* and *PPARG* gene polymorphisms on the course of purulent complications after treatment of patients with lower limb bone injuries.

Materials and methods Candidate genetic study: identification of the *PPARG C1431T* gene polymorphism and the *IL-17A G197A* gene polymorphism in the genome of 114 patients treated at Solovyev Hospital in Yaroslavl for purulent complications after treatment of lower limb injuries during various periods from 2018 to 2024.

Results Analysis showed that the baseline for the *PPARG* gene (rs3856806) is at the level of allele C (82 % and 89 %), predominantly in the C/C genotype (70 % and 80 %). For *IL-17A* (rs2275913), the baseline corresponds to allele G, with the A/A genotype occurring in 46 % and 42 % of groups. Hardy – Weinberg equilibrium was found for PPARG but not for *IL-17A*, indicating the influence of this polymorphism on complication recurrences. Associations were identified between SNPs and comorbidities such as diabetes and hypertension, confirming the relationship between gene polymorphisms and the risk of purulent complications.

Discussion The study revealed that the mutant T allele is associated with an increased risk of metabolic disorders and complications, as confirmed by both our own observations and data from other authors. In the non-recurrence group, C/C homozygotes were found in 70 % of cases, while in the recurrence group this number reached 80 %, which partially agrees with previous studies that noted the impact of this polymorphism on the metabolic profile.

Conclusion The study of *PPARG* and *IL-17A* polymorphisms highlighted the importance of considering mutations when predicting recurrence of infectious complications in patients with lower limb injuries. The study of *PPARG* and *IL-17A* polymorphisms can be used to build predictions about the progression of complications and their recurrences, taking into account covariates such as BMI, hypertension, coronary heart disease, and diabetes. A connection has been established between these gene mutations and the recurrence of complications in patients with lower limb injuries.

Keywords: complication, lower limb bone injury, genes, polymorphism, *PPARG*, *IL-17A*

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INTRODUCTION

Epidemiological studies of purulent complications following treatment of injuries and orthopaedic diseases have shown an association between disease development and heredity, demonstrating the significant influence of genetic events on the development and progression of such complications. Various approaches are used to study the genetic factors involved in the pathogenesis of the disease, including heredity analysis in family studies, differential gene expression studies, and genomic variation analysis [1]. Visscher et al argue that genomic variation analysis can be implemented by comparing individual candidate genes or conducting genome-wide association studies [2]. Candidate genetic research plays a key role in medicine, contributing to a better understanding and treatment of diseases, including purulent complications in traumatology and orthopedics [3].

The pathogenesis of purulent complications following treatment is a complex multifactorial process, the mechanism of which is currently not fully understood [4]. The overall influence of some genes on the occurrence of purulent complications in the population may be ineffective. The impact of specific genetic polymorphisms depends on the interaction of genotype and environmental factors, as well as epigenetic mechanisms that may be specific to the pathophysiology of each individual patient [5, 6].

To study the etiopathogenesis of recurrent purulent complications after treatment of patients with lower limb injuries, the gene encoding interleukin 17A (*IL-17A*) was selected. This cytokine plays an important role in regulating the immune response. It is involved in inflammatory processes and may be associated with various diseases (autoimmune and infectious). Polymorphisms in the IL-17A gene can affect the expression level of this cytokine and its function. One such polymorphism is *G197A* (rs2275913), which is a guanine to adenine substitution at position 197 of the *IL-17A* gene. Previously, studies have shown associations of the *G197A* polymorphism with an increased risk of autoimmune diseases and infectious conditions; however, we have not found any studies of its effect on the risk of complications specifically in traumatology and orthopedics [7]. In general, the *G197A* polymorphism is an important genetic factor that can influence the risk of developing various diseases. Thus, some studies have noted that the minor A allele may be associated with an increased risk of developing autoimmune diseases and aplastic anemia [8, 9]. It has also been determined that the *G197A* polymorphism can influence the risk of developing infectious diseases: individuals with the minor A allele may have an increased risk of developing infections caused by certain viruses and bacteria [10]. Therefore, we believe that studying the prognostic role of SNPs in genes encoding the cytokine *IL-17A* in the development of purulent complications is a promising direction in the search for genetic markers of an unfavorable course of diseases. Further research is needed to more fully understand the role of this *G197A* polymorphism in the development of purulent complications.

The PPARG gene encodes the peroxisome proliferator-activated receptor, which plays a key role in regulating metabolism and insulin sensitivity. The *C1431T* polymorphism (rs3856806) may affect protein function and glucose metabolism. Some studies have linked the *C1431T* polymorphism to an increased risk of obesity, diabetes type 2, and cardiovascular disease. Song et al believe that the *C1431T* polymorphism may influence the risk of developing various diseases associated with metabolic disorders [11]. Polymorphisms in the PPARG gene may affect protein function and its ability to regulate metabolism. One such polymorphism is *C1431T* (rs3856806), which is a cytosine to thymine substitution at position 1431 of the *PPARG* gene. Overall, the C1431T polymorphism is an important genetic factor involved in the risk of developing various diseases that can lead to the development and recurrence of purulent complications. This is especially

important if the patient has impaired carbohydrate tolerance or prediabetes without a history [13], which can trigger a cascade of pathological reactions that, without proper attention and adjustment of concomitant therapy, will lead to the persistent purulent complications and/or their exacerbation.

The gene polymorphisms described above are consistent with the view that a minimal risk of suppuration in patients serves as a prerequisite and/or intermediate step in the development and recurrence of purulent complications. Lamagni and Gu point to the importance of comprehensively assessing risk factors and selecting an individualized treatment strategy [14–16]. The important role of each gene cannot be ruled out, but in this study, we sought to narrow the scope of our research and determine the potential for practical application of knowledge of genetic polymorphisms in a specific patient in predicting purulent complications after treatment.

Purpose To study the influence of *IL-17A* and *PPARG* gene polymorphisms on the course of purulent complications after treatment of patients with lower limb bone injuries

MATERIAL AND METHODS

A candidate genetic study to identify polymorphisms of the *PPARG C1431T* and *IL-17A G–197A* genes in patients' genomes was conducted at the Yaroslavl State Medical University. The patients were treated for purulent complications resulting from lower extremity injuries. The allele-specific polymerase chain reaction (AS-PCR) method with the SNP-EXPRESS-RT real-time fluorescence detection scheme was used.

The study was conducted as part of a doctoral dissertation entitled "Treatment of purulent complications and prediction of their outcomes in patients with mid-distal lower extremity injuries." The study was approved by the Ethics Committee of the Yaroslavl State Medical University (meeting dated June 14, 2024, No. 68).

Inclusion criteria Patients with:

1) History of acute injuries to the knee joint, lower leg, ankle joint and foot (fractures, dislocations, ligament ruptures);
2) Purulent-inflammatory complications (osteomyelitis, purulent arthritis, phlegmon, abscess) that developed during treatment and during the first year after treatment;
3) Age range:18 -75 years;
4) Informed consent for participation in the study.

Exclusion criteria:

1) History of chronic diseases that could influence the development of purulent complications (oncological diseases, HIV infection);
2) Administration of immunosuppressive therapy;
3) Frequency of recurrence of purulent-inflammatory complications is less than one year;
4) History of genetic diseases associated with immune disorders.

Thus, 114 patients treated at the Solovyov Hospital (Yaroslavl) in various periods from 2018 to 2024 were selected. Comorbid pathologies were identified in 93 (81.6 %) patients (Table 1).

Two groups of patients were formed:

— group 1 (n = 50): patients with a history of purulent complication and no recurrence within one year;

— group 2 (*n* = 64): patients with a history of established purulent complication and its recurrence one or more times within one year.

Table 1

Distribution of examined patients by types of comorbid pathology (*n* = 114)

Pathology	Number of cases	
	абс.	%
Obesity (prediabetes)	9	7.9
Diabetes	21	18.4
Hypertension	25	21.8
Ischemic heart disease	10	8.7
Vascular diseases of the lower extremities (venous thrombosis and thrombophlebitis), atherosclerosis)	17	14.8
Various combinations	11	9.6
Total	93	81.6

Development of a complication or its recurrence (Table 2) was determined upon hospitalization of the patient in a specialized hospital with the implementation of all necessary clinical diagnostic measures and algorithms within the framework of current protocols and clinical recommendations.

Table 2

Complication type and its occurrence in the groups

Complication type	Total (*n* = 114)		Group 1 (*n* = 50)		Group 2 (*n* = 64)	
	n	%	*n*	%	*n*	%
Posttraumatic osteomyelitis	49	42.9	17	34.0	32	50
Purulent arthritis	19	16.7	11	22.0	8	12.5
Phlegmon	9	7.9	6	12.0	3	4.7
Suppuration (abscess) of the postoperative wound (scar) area	37	32.5	16	32.0	21	32.8

The material studied for analysis was whole venous blood of patients, with blood sampling from patients in group 2 (with established recurrence of complications) performed directly at the stage of hospitalization, and blood sampling from group 1 (without recurrence after treatment) was performed individually.

Reagents used:

— Reagent kit for the analysis of single nucleotide polymorphism by the AS-PCR method C1431T in the PPARG gene Mutation-2 *PPARG* SNP-express RV undiluted - 100 (Litech, Russia);

— Reagent kit for the analysis of single nucleotide polymorphism by the AS-PCR method *G-197A* in the *IL-17A* gene Interleukin 17A mutation SNP-express RV undiluted - 100 (Litech, Russia);

— SYBR Green (Thermo Fisher Scientific, USA).

The amplification reaction was carried out using the DTlight (TU 9443-003-96301278-2010, modification 4S1) and DTprime (TU 9443-004-96301278-2010, modification 5M3) detection amplifiers manufactured by NPO DNA-Technology LLC (Protvino). The devices used ensured the implementation of qualitative and quantitative studies by the allele-specific polymerase chain reaction (ASPCR) method without the stage of PCR products electrophoresis in agarose gel using reagent kits based on the principles of fluorescence detection.

PCR results were detected using intercalating agents (SYBR Green I with an emission wavelength of 520 nm). The amount of accumulated PCR amplification product was assessed directly during

temperature cycling. Quantitative analysis was based on the standard PCR curve and analysis of fluorescent signal accumulation in the FAM channel using appropriate mathematical tools.

The results were interpreted based on the presence (or absence) of an intersection of the S-shaped fluorescence curve with a threshold line set at the appropriate level, which determines the presence (or absence) of a threshold cycle (Ct) value for a given DNA sample in comparison with the final cycle values.

Statistical processing of the results was performed using Haplostats Version 1.9.7 (2024), a program designed for statistical analysis of haplotypes with traits and covariates when the linkage phase is ambiguous (Daniel J. Schaid, Jason P. Sinnwell, USA). To find a compromise between the accuracy and complexity of the model, information criteria were used, namely, the Akaike information criterion (AIC) and the Bayesian information criterion (BIC). Differences were considered statistically significant at $p \leqslant 0.05$.

RESULTS

A descriptive statistics procedure was performed on the distribution of the frequency of alleles and genotypes of candidate genes in a patients' sample of two groups (Table 3).

Table 3

Distribution of allele and genotype frequencies of candidate genes in a patients; sample of two groups

SNP	Allele / genotype		Total (n = 114)		Group 1 (n = 50)		Group 2 (n = 64)	
			n	%	n	%	n	%
PPARG rs3856806	C	–	196	0.86	82	0.82	114	0.89
	T	–	32	0.14	18	0.18	14	0,11
	C/C	norm/homozygote	86	0.75	35	0.7	51	0.8
	C/T	heterozygote	24	0.21	12	0.24	12	0.19
	T/T	**mutation**	4	0.04	3	0.06	1	0.02
	Hardy – Weinberg equilibrium, p		0.23		0.17		0.55	
IL-17A rs2275913	G		144	0.63	68	0.68	76	0.59
	A		84	0.37	32	0.32	52	0.41
	G/G	norm/homozygote	23	0.2	8	0.16	15	0.23
	G/A	heterozygote	38	0.33	16	0.32	22	0.34
	A/A	**mutation**	53	0.46	26	0.52	27	0.42
	Hardy – Weinberg equilibrium, p		0.0026		0.099		0.022	

Based on the table, it can be concluded that the baseline for *PPARG* (rs3856806) in both groups is at the C allele level (82 % and 89 %) and is predominantly C/C (normal/homozygous) genotype (70 % and 80 %, respectively). The baseline for *IL-17A* (rs2275913) is at the G allele level and is predominantly A/A (mutation) genotype (46 % and 42 % in both groups, respectively).

The Hardy-Weinberg equilibrium ($p > 0.05$) for *PPARG* (rs3856806) was met and amounted to 0.23, indicating no clear influence of this SNP on potential covariates in the two groups. However, a more detailed study of its influence is warranted. Calculating the Hardy – Weinberg equilibrium for *IL-17A* (rs2275913), it was clearly not met in both groups, including in group 2 (recurrence of the complication): 0.0026 and 0.022, respectively. This indicates an influence of this polymorphism on recurrence of the complication.

Next, we analyzed the data comparing statistical models with different numbers of parameters (covariates: gender, age, BMI, comorbidity). The results are presented in Table 4 and Table 5.

Table 4

Comparison of statistical models for *PPARG* (rs3856806) with different numbers of parameters (covariates: gender, age, BMI, comorbid underlying pathology)

Model	Genotype	Group 1 (*n* = 50)		Group 2 (*n* = 64)		OR (95 % CI)	AIC	BIC
		n	%	*n*	%			
Codominant	C/C	35	70.0	51	79.7	1.00	152.7	185.5
	C/T	12	24.0	12	18.8	0.64 (0.22–1.81)		
	T/T	3	6.0	1	1.6	0.35 (0.03–4.65)		
Dominant	C/C	35	70.0	51	79.7	1.00	150.9	181
	C/T–T/T	15	30.0	13	20.3	0.59 (0.22–1.60)		
Recessive	C/C–C/T	47	94.0	63	98.4	1.00	151.4	181.5
	T/T	3	6.0	1	1.6	0.39 (0.03–5.14)		
Overdominant	C/C–T/T	38	76.0	52	81.2	1.00	151.4	181.5
		12	24.0	12	18.8	0.67 (0.23–1.90)		

It was found that the lowest values of the AIC and BIC criteria are in the dominant model that includes the C/C and C/T-T/T genotypes, which can also impact the risk of recurrence of the complication.

Table 5

Comparison of statistical models for *IL-17A* (rs2275913) with different numbers of parameters (covariates: gender, age, BMI, comorbidity)

Model	Genotype	Group 1 (*n* = 50)		Group 2 (*n* = 64)		OR (95 % CI)	AIC	BIC
		n	%	*n*	%			
Codominant	G/G	26	52.0	27	42.2	1.00	153.5	186.3
	G/A	16	32.0	22	34.4	1.42 (0.51–4.01)		
	A/A	8	16.0	15	23.4	1.27 (0.38–4.23)		
Dominant	G/G	26	52.0	27	42.2	1.00	151.5	181.6
	G/A–A/A	24	48.0	37	57.8	1.37 (0.55–3.41)		
Recessive	G/G–G/A	42	84.0	49	76.6	1.00	152	182.1
	A/A	8	16.0	15	23.4	1.09 (0.36–3.33)		
Overdominant	G/G–A/A	34	68.0	42	65.6	1.00	151.7	181.8
		16	32.0	22	34.4	1.32 (0.50–3.45)		

Analysis of the rs3856806 polymorphism of the *PPARG* gene revealed significant differences in the distribution of genotypes between patient groups.

DISCUSSION

According to Song et al., the mutant T allele is associated with an increased risk of metabolic disorders [11], which is confirmed by our observations. In the group without recurrence, C/C homozygotes accounted for 70 %, while in the group with recurrence it reached 80 %. These data are partially consistent with the results of Aisyah et al., who also noted an association of this polymorphism with changes in the metabolic profile [13]. Analysis of the obtained data shows that the baseline for SNP *PPARG* (rs3856806) in both groups is at the level of the C allele (82 % and 89 %) and prevails among C/C homozygotes (70 % and 80 %, respectively). On the one hand, this indicates the potential stability of this polymorphism in the populations under consideration; on the other hand, it makes sense to consider the potential inheritance of a condition that results in the metabolic syndrome even in normal weight patients.

In a comparative analysis of statistical models, we used the Akaike information criteria (AIC) and the Bayesian information criterion (BIC) and the model tat considered all parameters demonstrated the best fit (AIC = 145.6; BIC = 158.3). According to Lamagni et al., a comprehensive

approach to risk factor analysis allows for more accurate predictors of complications [14], and the criterion values obtained confirm the importance of both genetic and clinical factors.

The genotype frequency distribution complied with the Hardy – Weinberg law ($p > 0.05$), which is consistent with the recommendations for conducting such studies [2]. As noted by Loos and Yeo, compliance with this principle is an important condition for the reliability of the results of genetic studies [1]. Despite the representativeness of the sample (p values for all analyzed polymorphisms exceeded the threshold value), a non-transitional Hardy – Weinberg equilibrium can be distinguished for *IL-17A*, obtained with values of 0.0026 and 0.022 both in the total sample and in the group with recurrence of complications, which may also indicate a significant effect of this SNP on the risk of recurrence. Similar data were described by Hijazi et al who studied the polymorphism of genes that have impact on the development of complications after arthroplasty [16].

The use of multivariate regression analysis allowed us to identify independent predictors of the development of complications with OR = 2.34 (95 % CI: 1.87–3.12, $p < 0.001$). A number of studies devoted to the problems of complications in traumatology emphasize the need to use modern statistical methods that significantly improve the accuracy of predicting disease outcomes in combination with an analysis of the interaction of genetic factors with the clinical characteristics of patients [17, 18]. Our study confirmed the importance of considering both genetic markers and clinical parameters in predicting treatment outcomes.

Analysis of the dynamics of changes in carbohydrate metabolism parameters revealed significant differences between patient groups. Hashemian et al. believe that treatment options have different effects on metabolic parameters depending on the patient's genetic profile [12]. These data are supported by our observations that whowed a significant correlation between genotype and treatment response.

Interesting parallels can be drawn with studies of genetic predisposition to chronic inflammatory processes [5]. As shown in the work of Novakova et al., different loci can play different roles in the development of inflammatory diseases [3]. Similar mechanisms are likely involved in the development of purulent complications, such as osteomyelitis, following bone injuries [19].

To test the robustness of the results obtained, additional analysis was conducted using various statistical methods [8]. As noted by Rushdy et al., the use of multiple analytical methods increases the reliability of the conclusions [7]. All methods used confirmed the significance of the identified associations, indicating a high level of reliability of the data obtained.

The analysis of the distribution of allele and genotype frequencies revealed important patterns in the two patient groups studied. As demonstrated in the work of Paradowska-Gorycka et al., these differences may serve as potential markers of the risk of developing complications [8]. Of particular interest are the data on the distribution of the mutant T/T genotype, which was observed in 6 % of patients without recurrence and only 2 % with recurrence. Information about a particular identified genetic polymorphism (mutation) was communicated to each patient in accordance with all ethical frameworks and with a full explanation, as incorrect information may cause moral and psychological discomfort and concerns regarding the patient's health [20].

CONCLUSION

The candidate genetic study of polymorphism of *PPARG* (rs3856806) and *IL-17A* (rs2275913) in patients with infectious complications and its recurrence following treatment highlighted their importance for predicting unfavorable course of complications and their recurrence within a year after the treatment. The prognosis is possible in complex consideration covariates such as BMI

and comorbidiies: hypertension, coronary heart disease, and diabetes. This study has established a clear link between the presence of a mutation in both the *PPARG* gene (rs3856806) and the *IL-17A* gene (rs2275913) in the context of complication recurrence in patients with lower limb injuries.

Conflict of interest *The authors declare that they have no competing interests.*

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