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Genetic determinants of type 2 diabetes mellitus: The Role of *FABP2*, *PNPLA3*, *FADS1*, *LEPR*, and *GCKR* gene polymorphisms in Russians and Yakuts

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Abstract: Objective. To conduct a comparative analysis of the role of gene polymorphisms associated with lipid and carbohydrate metabolism (*FABP2* rs1799883, *PNPLA3* rs738409, *FADS1* rs174537, *LEPR* rs1137101, *GCKR* rs780094) in the development of type 2 diabetes mellitus (T2DM) in Russian (n=179) and Yakut (n=446) populations of the Republic of Sakha (Yakutia) using a case-control study.

Materials and methods. Genotyping was performed by PCR-RFLP.

Results. Significant interpopulation differences were identified in the distribution of *FABP2, PNPLA3, FADS1*, and *LEPR* gene alleles, reflecting their potential adaptive role to different environmental conditions. The main finding was a pronounced ethnic specificity for the *FABP2* Ala54Thr polymorphism: in Yakuts, the heterozygous Ala/Thr genotype was identified as a risk factor for T2DM (OR = 1.68; p = 0.01), while the homozygous Thr/Thr variant exhibited a protective effect (OR = 0.51; p < 0.001). No significant association was found in the Russian sample. Analysis of biochemical parameters in T2DM patients revealed genotype-dependent differences in lipid profile, fasting glucose levels, and liver function markers, confirming the role of the studied genes as modifiers of the metabolic phenotype.

Conclusion. The obtained data emphasize the critical importance of considering ethnic origin in assessing genetic predisposition to T2DM and its complications for the development of personalized medicine strategies.

Keywords: *FABP2* rs1799883; *PNPLA3* rs738409; *FADS1* rs174537; *LEPR* rs1137101; *GCKR* rs780094; type 2 diabetes mellitus.

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Introduction. Type 2 diabetes mellitus (T2DM) is a progressive metabolic disease closely linked to both genetic and environmental factors. External risk factors such as high caloric intake, dietary composition, air pollution, and physical inactivity are important drivers of the steadily increasing prevalence of T2DM [1]. According to the International Diabetes Federation (IDF), by 2024, one in nine adults (589 million people) had diabetes, and by 2050, this number could reach 852.5 million [2]. According to the Federal Diabetes Registry of the Russian Federation, as of December 2025, 5,672,859 people were registered for dispensary observation, including 5,237,942 with type 2 diabetes mellitus. In the Republic of Sakha (Yakutia), as of July 1, 2025, 30,663 people suffered from this disease, corresponding to 3,090.57 per 100,000 population [3]. Genome-wide association studies (GWAS) of type 2 diabetes have identified more than 500 risk loci [4]. Large studies and meta-analyses have been conducted predominantly on populations of European (especially Western European) origin [5, 6]. This underscores the relevance of searching for ethnicity-dependent genetic markers.

Studying genetic polymorphisms such as *FABP2* rs1799883, *FADS1* rs174537, *GCKR* rs780094, *PNPLA3* rs738409, and *LEPR* rs1137101 can help elucidate the biochemical pathways leading to the development of type 2 diabetes.

Fatty acid-binding protein 2 (FABP2) is responsible for the transport of long-chain fatty acids in the intestine. The substitution of alanine with threonine (Thr54 variant) in the rs1799883 polymorphism of the *FABP2* gene results in increased efficiency of fatty acid binding and transport by the protein, leading to decreased cellular insulin sensitivity, elevated low-density lipoprotein (LDL) levels in the blood, and a prolonged increase in free fatty acids, which impairs pancreatic beta-cell function [7]. PNPLA3, or adiponutrin, is involved in intracellular lipid remodeling, and the rs738409 polymorphism is associated with increased triglyceride accumulation in hepatocytes by limiting substrate access to the enzyme's catalytic site. A 2022 study found that the co-existence of T2DM and this genetic variant increased the risk of developing advanced liver fibrosis (stages 3-4) by 14.7-fold compared to patients without diabetes and with the normal genotype [8].

Genes of the *FADS* cluster (*FADS1, FADS2*) encode enzymes necessary for the synthesis of long-chain polyunsaturated fatty acids (PUFAs), such as arachidonic acid. These acids are precursors of molecules that regulate inflammation, insulin sensitivity, and lipid metabolism [9]. The rs174537 variant (T allele) is associated with a significantly slower conversion of dietary precursors to active PUFAs. Genome-wide association studies in humans have also identified the influence of variations in the *FADS1* and *FADS2* gene cluster on glucose and lipid metabolism [10, 11].

The GCKR protein regulates the activity of glucokinase, a key enzyme that captures glucose in the liver. However, the role of the rs780094 polymorphism remains controversial. Some studies suggest that the T allele is associated with the development of type 2 diabetes, while others suggest it has a protective effect [12]. More recent data indicate that *GCKR* is involved in regulating levels of follistatin, a protein that can cause insu-

lin resistance in adipose tissue. Elevated blood follistatin levels predict the development of type 2 diabetes many years before its onset [13]. Thus, it is possible that *GCKR* may influence diabetes risk indirectly through this metabolic pathway.

The rs1137101 polymorphism of the *LEPR* gene involves a substitution of the 223rd amino acid residue from glutamine (Q) to arginine (R), potentially affecting signaling and thereby increasing susceptibility to type 2 diabetes [14].

All the variants described above do not act in isolation; they form a complex network, determining how the body metabolizes fats (*FABP2, PNPLA3*), synthesizes essential fatty acids (*FADS1*), responds to the satiety hormone (*LEPR*), and regulates hepatic glucose uptake (*GCKR*).

The aim of this study was to analyze polymorphisms in the *FABP2, PNPLA3, FADS1, LEPR*, and *GCKR* genes in relation to the development of type 2 diabetes mellitus in samples from Russian and Yakut populations.

Materials and methods. The study protocol (No. 60, dated April 10, 2024) was approved by the local Biomedical Ethics Committee of the Yakut Scientific Centre for Complex Medical Problems (YSCCMP). Written informed consent was obtained from all volunteers. Clinical information on patients was collected in a dedicated database, and DNA samples were stored in the YSCCMP biobank using the Unique Scientific Facility "Genome of Yakutia" (registration No. USU_507512).

The study included 243 patients with type 2 diabetes (48 Russians and 195 Yakuts). The control sample comprised 382 volunteers without metabolic diseases or obesity (131 Russians and 251 Yakuts). Ethnicity was traced back three generations; all subjects resided in the Republic of Sakha (Yakutia). SNP genotyping was performed using classical polymerase chain reaction (PCR) and restriction fragment length polymorphism (RFLP) analysis in the Hereditary Pathology Laboratory of the Molecular Genetics Department at the YSCCMP. The conditions for amplifying gene regions containing polymorphic variants, including oligonucleotide primer sequences, the restriction enzyme used, and restriction fragment lengths, are presented in Table 1.

Genotype distributions were compared with expected values under Hardy-Weinberg equilibrium, and the frequencies of allelic variants and genotypes were compared using the χ^2 (chi-square) test with Yates' correction. To assess the significance of the odds ratio, the 95% confidence interval (95% CI) was calculated. The odds ratio was calculated using the formula: OR = (A×D)/(B×C) (where OR is the odds ratio; A, B, C, D represent the number of observations in the cells of the contingency table). A comparison of mean serum biochemical parameters in T2DM patients depending on genotype was performed using the Kruskal-Wallis test [15]. Results were considered significant at $p < 0.05$.

Results and discussion. The analysis of allele frequency distribution for the studied polymorphisms is presented in Table 2.

A comparative analysis of genotype and allele frequency distributions for polymorphisms in the *FABP2, PNPLA3, FADS1*, and *LEPR* genes revealed significant differences between Russians and Yakuts, both among healthy individuals and among patients with type 2 diabetes. Genes associated with food metabolism (*FABP2, FADS1*) and energy storage strategies (*PNPLA3, LEPR*) most clearly indicate adaptation to different environmental conditions. We hypothesize that these genes were likely influenced by natural selection under the distinct environmental conditions (diet, cold climate) to which the ancestors of Russians and Yakuts were historically exposed. Analysis of the rs780094 polymorphism in the *GCKR* gene did not reveal significant differences between the populations. This particular gene variant may not have been subject to significant selection pressure under conditions that differed between the two populations; it could have been evolutionarily neutral in the context of the key environmental factors that shaped differences in other genes.

Interestingly, only the rs1799883 polymorphism of the *FABP2* gene in Yakuts showed a statistically significant difference between the patient and healthy control groups (p=0.01). In contrast, in the Russian sample, no significant differences in the distribution of genotypes and alleles were found between the T2DM group and healthy individuals (p=0.61).

The association between the genotype and alleles of the rs1799883 polymorphism of the *FABP2* gene is presented in Table 3.

In the studied Russian sample, the odds ratio did not show significant associations with type 2 diabetes for any genotype or allele. In the Yakut sample, a clear association of this polymorphism with type 2 diabetes was found. The heterozygous Ala/Thr genotype was identi-

Table 1

Primers and restriction enzymes used for genotyping of gene polymorphisms

Gene / SNP	Primers	Annealing temp. (°C)	Restriction endonuclease	Restriction fragments (bp)
FABP2 rs1799883	F: ACAGGTGTTAATATAGTGAAAAG	58	AspLEI	Ala/Ala - 99, 81 Thr/Thr - 180
	R: GACGGAACT GAACTCAGGGTA			
PNPLA3 rs738409	F: TGGGCCTGAAGTCCGAGGGT	66	BstF5 I	CC – 200, 133 GG – 333
	R: CCGACACCAGTGCCCTGCAG			
FADS1 rs174537	F:CAG GGG AGA GAG GTG GAG TA	64	AvaII	G - 149, 158 T - 307
	R: AGG TCT GTC TGG CTG TCT CC			
LEPR rs1137101	F: ACCCTTTAAGCTGGGTGTCCCAAATA	62	MspI	AA – 330 GG – 293
	R: AATGTCAGTTCAGCCCATAAATATGG			
GCKR rs780094	F: CATGTTGGCTAGGCTTGTTGAG	62	Pci I	GG – 126, 176, 258 AA – 302, 258
	R: AGCTCACGCTGGAACTTCTG			

Table 2

Comparative distribution of genotype and allele frequencies in type 2 diabetes patients and control subjects among russians and yakuts

Gene	Group	Comparison groups	Genotype frequency (%)			Allele frequency (%)		Chi-square	p
				Ala/Thr	Thr/Thr	Ala	Thr		
FABP2 rs1799883	T2DM	Russians	43.8	37.5	18.8	62.5	37.5	5.1	**0.02**
		Yakuts	25.6	46.7	27.7	49.0	51.0		
	Controls	Russians	36.6	44.3	19.1	58.8	41.2	24.1	**0.00**
		Yakuts	22.7	34.3	43.0	39.8	60.2		
PNPLA3 rs738409			CC	CG	GG	C	G		
	T2DM	Russians	34.0	22.0	44.0	45.0	55.0	14.6	**0.00**
		Yakuts	9.9	30.2	59.9	25.0	75.0		
	Controls	Russians	33.8	25.9	40.3	46.8	53.2	40.7	**0.00**
		Yakuts	7.6	32.1	60.3	23.7	76.3		
FADS1 rs174537			GG	TG	TT	G	T		
	T2DM	Russians	42.6	42.6	14.9	63.8	36.2	45.7	**0.00**
		Yakuts	8.6	35.9	55.6	26.5	73.5		
	Controls	Russians	39.6	42.4	18.1	60.8	39.2	107.9	**0.00**
		Yakuts	6.5	34.6	58.9	23.8	76.2		
Lepr rs1137101			AA	AG	GG	A	G		
	T2DM	Russians	19.1	55.3	25.5	46.8	53.2	21.81	**0.00**
		Yakuts	6.7	31.3	62.1	22.3	77.7		
	Controls	Russians	21.5	47.9	30.6	45.5	54.5	61.39	**0.00**
		Yakuts	3.6	30.8	65.6	19.0	81.0		
GCKR rs780094			AA	AG	GG	A	G		
	T2DM	Russians	17.8	35.6	46.7	35.6	64.4	0.21	0.65
		Yakuts	16.6	44.6	38.9	38.9	61.1		
	Controls	Russians	24.3	44.4	31.3	46.5	53.5	0.74	0.39
		Yakuts	18.7	48.5	32.8	42.9	57.1		

Table 3

Odds ratios with confidence intervals for *FABP2 rs1799883*

	Russians		OR (95% CI)	p-value	Yakuts		OR (95% CI)	p-value
	T2DM (%)	Controls (%)			T2DM (%)	Controls (%)		
Ala/Ala	43.8	36.6	1.345 (0.687-2.633)	0.489	25.6	22.7	1.174 (0.759-1.816)	0.54
Ala/Thr	37.5	44.3	0.755 (0.383-1.488)	0.522	46.7	34.3	1.679 (1.144-2.464)	**0.01**
Thr/Thr	18.8	19.1	0.978 (0.420-2.279)	0.87	27.7	43.0	0.507 (0.340-0.757)	**0.00**
Ala	62.5	58.8	1.169 (0.723-1.891)	0.61	49.0	39.8	1.449 (1.110-1.893)	**0.01**
Thr	37.5	41.2	0.856 (0.529-1.384)		51.0	60.2	0.690 (0.528-0.901)	

Table 4

Mean biochemical blood parameters in type 2 diabetes patients depending on genotype and ethnicity

Gene / Polymorphism	Group	Parameter	Observed phenotypic pattern
FABP2 rs1799883	Russians	Fasting glucose	Thr/Thr (7.22) < Ala/Ala (10.61) ≈ Ala/Thr (10.36)
		AST	Thr/Thr (39.66) > Ala/Ala (22.51) ≈ Ala/Thr (20.82)
PNPLA3 rs738409	Russians	HDL	GG (1.22) < CG (1.80) < CC (2.03)
		Fasting glucose	GG (7.60) < CG (9.97) < CC (11.30)
	Yakuts	ALP	GG (170.75) > CG (157.62) > CC (99.89)
FADS1 rs174537	Russians	Total cholesterol	TT (5.90) > TG (5.72) > GG (5.14)
		LDL	TG (3.37) > TT (3.03) > GG (2.21)
	Yakuts	Total cholesterol	GG (5.99) > TG (5.45) > TT (4.84)
LEPR rs1137101	Russians	Triglycerides	GG (2.61) > AA (2.03) ≈ AG (1.83)
		HDL	GG (1.50) < AG (1.96) < AA (2.09)
	Yakuts	Creatinine	GG (77.11) > AG (74.31) > AA (63.85)
GCKR rs780094	Russians	Creatinine	GG (70.77) < AG (92.12) ≈ AA (92.60)
	Yakuts	AST	GG (17.11) < AG (22.22) < AA (29.07)
		ALT	GG (18.71) < AG (25.11) < AA (35.15)

fied as a risk factor (OR = 1.68, 95% CI 1.14-2.46; p = 0.01), while the homozygous Thr/Thr genotype, in contrast, demonstrated a protective effect (OR = 0.51, 95% CI 0.34-0.76; p < 0.001). The Thr allele was also associated with reduced risk (OR = 0.69, 95% CI 0.53-0.90; p = 0.01). In a large meta-analysis summarizing data from 26 studies involving 6,032 T2DM patients and 8,907 controls, Anilkumar et al. (2026) found that the Ala54Thr polymorphism of the *FABP2* gene was significantly associated with a higher risk of developing T2DM under a recessive inheritance model, especially in Asian and Caucasian populations, highlighting the importance of ethnicity-stratified analysis [16].

Data from a large prospective study in Novosibirsk [17] and an earlier meta-analysis also indicate that the rs1799883 polymorphism of the *FABP2* gene is not an independent marker of genetic predisposition to T2DM in the Russian (Caucasian) population [18].

A comparative analysis of serum biochemical parameters depending on the polymorphisms of the studied genes among T2DM patients is presented in Table 4 (comparisons are shown only where differences between genotypes reached statistical significance).

Biochemical analysis of the studied polymorphisms revealed that even if a polymorphism does not affect disease risk, it can significantly modify its course and biochemical profile. For example, Russian carriers of the Thr/Thr variant of the *FABP2* gene exhibited better glucose control but also higher levels of liver metabolism markers (AST). The rs738409 polymorphism of the *PNPLA3* gene confirmed its role in liver metabolism. Thus, in the Russian sample, the GG genotype was associated with the lowest HDL cholesterol levels and the lowest fasting glucose ($p<0.05$). In the Yakut sample, the GG genotype was associated with significantly higher alkaline phosphatase (ALP) activity, a marker of cholestasis and liver damage ($p=0.03$). The rs174537 polymorphism of the *FADS1* gene revealed that the same genetic variant can have different effects on the phenotype depending on genetic background and lifestyle. For example, in the Russian sample, carriers of the homozygous TT genotype had the highest levels of total cholesterol and triglycerides ($p<0.05$), while Yakuts with the same genotype demonstrated the lowest total cholesterol levels ($p=0.01$). Its clinical significance may lie in individual response to diet, particularly polyunsaturated fatty acids. A possible explanation is that the traditional Russian diet is more plant-based, while the Yakut diet primarily consists of animal products. Regarding the rs1137101 polymorphism of the *LEPR* gene, carriers of the homozygous GG genotype in the Russian sample had elevated triglyceride levels and reduced HDL levels, while in the Yakut sample, the presence of the G allele was associated with elevated creatinine levels (a marker of kidney function). Thus, the association of the G allele with an unfavorable lipid profile in Russians is consistent with the role of leptin in regulating lipid metabolism. The rs780094 polymorphism of the *GCKR* gene was shown to influence creatinine levels and liver enzyme activity (AST, ALT), emphasizing its role in carbohydrate and lipid metabolism, affecting the liver and, possibly, kidney function.

It is important to note that for most biochemical parameters, no significant differences were found depending on genotype. This necessitates further studies with larger sample sizes and consideration of additional factors (diet, therapy, disease duration).

Conclusion. The comparative analysis demonstrates that the presented original data both confirm global trends in the genetics of T2DM (ethnic specificity, the role of complication modifiers) and make a unique contribution by focusing on a previously understudied Yakut population.

This study reveals a complex picture of genetic predisposition to T2DM, where the significance of specific genetic variants strongly depends on ethnicity. The main conclusion is that genes regulating lipid (fat) metabolism exhibit stronger and statistically more significant associations with T2DM risk and related biochemical parameters than genes directly linked to glucose metabolism or leptin signaling.

Thus, the study makes a significant contribution to the development of ethnically sensitive personalized medicine aimed at preventing not only diabetes but also its most severe systemic complications.

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