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# Association of Polymorphisms in *PPARPGC1A, ACE*, and *DRD2* Genes with Gestational Diabetes Mellitus

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## Abstract

***The aim*** of our research was to study the distribution of polymorphic variants of the *DRD2/ANKK1* TaqIA (rs1800497 SNP), *PPARGC1A* rs8192678 SNP, and *ACE* I/D in gestational diabetes mellitus (GDM).

**Methods and Results:** The study included 383 pregnant women (gestational age of 37.0–41.0 weeks) with GDM and 68 pregnant women without disturbed carbohydrate metabolism. This was a prospective case-control study. All patients were divided into 3 groups. Group 1 included 211 pregnant women with GDM who received diet therapy only; Group 2 included 172 pregnant women with GDM who received insulin therapy; Group 3 included 68 pregnant women without metabolic disorders. For the *DRD2/ANKK1* TaqIA (rs1800497 SNP) (A1/A2; T/C), we found that the TT homozygous genotype and T allele prevailed in Groups with GDM compared with Group without metabolic disorders.

***Conclusion***: **A** study of the *DRD2/ANKK1* TaqIA (rs1800497 SNP), *PPARGC1A* rs8192678 SNP, and *ACE* I/D revealed statistically significant increased risks for GDM in carriers of the TT genotype and T allele of the *DRD2/ANKK1* TaqIA (rs1800497 SNP).(**International Journal of Biomedicine. 2021;11(1):42-45.**)

**Key Words:** gestational diabetes mellitus • dopamine D2 receptor • PPARPGC-1A • angiotensin-converting enzyme

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## Abbreviations

**DRD2**, dopamine D2 receptor; **GDM**, gestational diabetes mellitus; **PPARPGC1A**, peroxisome proliferator-activated receptor gamma coactivator 1-alpha; **ACE**, angiotensin-converting enzyme; **T2DM**, type 2 diabetes mellitus.

## Introduction

Despite the well-known role of pregestational obesity in the pathogenesis of gestational diabetes mellitus (GDM), the role of polymorphism of genes responsible for the regulation of carbohydrate and fat metabolism in GDM has been insufficiently studied.[1-3] The available data are quite contradictory. In particular, the significance of the polymorphisms in the *DRD2* gene, *PPARGC1A* gene, and *ACE* gene has been studied in most detail in relation to T2DM.[1,4,5] It should be noted that data on the role of genetic mechanisms in the formation of the complicated course of GDM are extremely few in number. However, current studies report a possible close relationship between genes responsible for the development of T2DM and of GDM.[6]

Several studies show that the Alu Insertion/Deletion (*ACE* I/D) polymorphism present on intron 16 of the *ACE* gene

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is associated with T2DM and diabetes-related complications.[6,7] Significant associations of *ACE* SNP's, C1237T, and G2350A with GDM were observed. Haplotype analysis revealed the remarkably significant evidence of association with SNP combination ACE A240T, C1237T, G2350A, and I/D with GDM patients ($P$=0.024).[6] However, the information about the role of this gene in the development of GDM in the world scientific literature is ambiguous. There are studies that disprove the association of the *ACE* gene polymorphisms with GDM.[8]

There is also little information about the role of polymorphisms of other genes responsible for carbohydrate metabolism disorders, such as *DRD2*, *PPARGC1A* (*PGC-1 alpha*), in the pathogenesis of GDM.

SNP rs8192678 (in exon 8, G1444A/Gly482Ser) is the most important polymorphism of the *PPARGC1A* gene.[9,10] It may be a functional point mutation associated with altered gene expression. Altered gene expression might contribute to insulin resistance by impaired metabolic pathways (e.g. PPAR-mediated adipocyte differentiation, lipid oxidation, gluconeogenesis in the liver or glucose transport in the muscles).[10-13] The negative effect of the minor 482Ser allele has been described on metabolic and cardiovascular traits, such as insulin sensitivity and secretion, measures of obesity, lipid and glucose concentrations, adiponectin level, aerobic fitness.[9, 10, 13]

A widely studied SNP, the so-called DRD2/ANKK1 Taq1A polymorphism (rs1800497, C32806T, Glu713Lys) is located ~10 kb downstream from the DRD2 gene in the ankyrin repeat and kinase domain containing 1 (*ANKK1*) gene.[14] However, individuals who carry the A1 (T) allele have reduced brain D2 receptor density,[15] which has been demonstrated to increase the risk for overeating and obesity.[16, 17]

The aim of our research was to study the distribution of polymorphic variants of the *DRD2/ANKK1* TaqIA (rs1800497 SNP), *PPARGC1A* rs8192678 SNP, and *ACE* I/D in GDM.

## Materials and Methods

The study included 383 pregnant women with GDM and 68 pregnant women without disturbed carbohydrate metabolism, who gave birth between the second quarter of 2019 and the third quarter of 2020 (gestational age of 37.0–41.0 weeks). This was a prospective case-control study. Diagnosis of GDM was based on the criteria of the American College of Obstetricians and Gynecologists.[18] All patients were divided into 3 groups. Group 1 included 211 pregnant women with GDM who received diet therapy only; Group 2 included 172 pregnant women with GDM who received insulin therapy; Group 3 included 68 pregnant women without metabolic disorders.

The surveyed patients were questioned about: 1) family history of disorders of carbohydrate metabolism and obesity; 2) chronic somatic and gynecological diseases; 3) reproductive history; and 4) complications of current pregnancy, the timing of GDM detection.

After DNA extraction, the samples were subjected to a PCR-RFLP reaction to analyze the *DRD2/ANKK1* TaqIA (rs1800497 SNP), the *PPARGC1A* rs8192678 SNP, and the *ACE* I/D polymorphism.

Genomic DNA was isolated the peripheral blood leukocytes using standard extraction technique using kits from QIAmpDNABloodMiniKit» («Qiagen», Germany). Genotyping of the *ACE* I/D polymorphism was performed using a PCR method as previously described.[19] The *PPARGC1A* rs8192678 SNP was genotyped by PCR-RFLP analysis.[20] Genotyping of the *DRD2/ANKK1* TaqIA (rs1800497) polymorphism was performed by PCR-RFLP analysis.[21]

Statistical analysis was performed using the Statistica v.10 software package (*StatSoft Inc*, USA). The frequency distribution of genotypes for the studied polymorphic loci was checked for compliance with the Hardy–Weinberg equilibrium (HWE).[22] To check the statistical significance of the differences between the groups, we used Pearson $\chi 2$ test. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. A value of $P$<0.05 was considered statistically significant.

## Results and Discussion

The genotype frequencies of all studied polymorphisms were in compliance with HWE ($P$>0.05) (Tables 1-3).

An analysis of the frequency distribution of genotypes and alleles of the *PPARGC1A* rs8192678 SNP showed no differences between groups (Table 1).

***Table 1.***

***Distribution of alleles and genotypes of the PPARGC1A rs8192678 SNP in the studied groups***

| Genotype/ allele | Group 1 (n=133) | Group 2 (n=110) | Group 3 (n=50) | Statistics |
|---|---|---|---|---|
| Ser/Ser | 15 (11.38%) | 18 (16.4%) | 4 (8%) | $\chi^2$=3.261 df=4; $P$=0.5151 |
| Gly/Ser | 72 (54.1%) | 61(55.4%) | 30 (60%) | |
| Gly/Gly | 46 (34.6%) | 31 (28.2%) | 16 (32%) | |
| Ser | 102 (38.3%) | 97 (44.1%) | 38 (38%) | $\chi^2$=1.959 df=2; $P$=0.3774 |
| Gly | 164 (61.7%) | 123 (55.9%) | 62 (62%) | |
| Compliance with HWE | $\chi^2$ =2.79 | $\chi^2$ =1.71 | $\chi^2$ =3.74 | |

For the *DRD2/ANKK1* TaqIA (rs1800497 SNP) (A1/A2; T/C), we found that the TT homozygous genotype and T allele prevailed in Groups 1 and 2 compared with Group 3 (Table 2). The carriage of the T allele of the *DRD2/ANKK1* TaqIA (rs1800497 SNP) was associated with increased *risk* of GDM (Group 1: OR=2.286; 95% CI: 1.320-3.957; $P$<0.0.1 Group 2: OR=2.436; 95% CI: 1.384-4.289; $P$<0.01)

An analysis of the frequency distribution of genotypes and alleles of the *ACE* I/D polymorphism showed no differences between groups (Table 3).

The results obtained regarding the *ACE* I/D polymorphism were not similar to a previous study conducted by Parul Aggarwal et al.[6] and Mani Mirfeizi et al.,[8] in which the researchers found the prevalence of the DD genotype in women with GDM compared to healthy women.

***Table 2.***

***Distribution of alleles and genotypes of the DRD2/ANKK1 TaqIA (rs1800497 SNP) in the studied groups***

| Genotype/ allele | Group 1 (n=72) | Group 2 (n=61) | Croup 3 (n=51) | Statistics |
|---|---|---|---|---|
| C/T | 37 (51.4%) | 27 (44.3%) | 23 (60.5%) | $\chi^2$=12.47 df=4; *P*=0.0142 |
| C/C | 21 (29.2%) | 19( 31.1%) | 26 (34.2%) | |
| T/T | 14 (19.4%) | 15 (24.6%) | 2 (5.3%) | |
| C | 79 (54.9%) | 65 (53.3%) | 75 (73.5%) | $\chi^2$=11.578 df=2; *P*=0.0031 |
| T* | 65 (45.1%) | 57 (46.7%) | 27 (26.5%) | |
| Compliance with HWE | $\chi^2$ =0.1 | $\chi^2$ =0.75 | $\chi^2$ =1.28 | |

**Group 1: OR=2.286; 95% CI: 1.320-3.957; P<0.01*
**Group 2: OR=2.436; 95% CI: 1.384-4.289; P<0.01*

***Table 3.***

***Distribution of alleles and genotypes of the ACE I/D polymorphism in the studied groups***

| Genotype/ allele | Group 1 (n=131) | Group 2 (n=123) | Group 3 (n=58) | Statistics |
|---|---|---|---|---|
| D/D | 10 (7.6%) | 15 (12.2%) | 5 (8.6%) | $\chi^2$=2.073 df=4; *P*=0.7223 |
| I/D | 47 (35.9%) | 46 (37.4%) | 23 (39.7%) | |
| I/I | 74 (56.5%) | 62 (50.4%) | 30 (51.7%) | |
| D | 67 (25.6%) | 76 (30.9%) | 33 (28.4%) | $\chi^2$=1.779 df=2; *P*=0.4109 |
| I | 195 (74.4%) | 170 (69.1%) | 83 (71.6%) | |
| Compliance with HWE | $\chi^2$ =0.43 | $\chi^2$ =1.9 | $\chi^2$ =0.04 | |

Epidemiological studies revealed inconsistent results regarding the association of the *PPARGC1A* rs8192678 SNP with the metabolic syndrome pathology. Regarding T2DM, while some studies confirmed an increased risk,[23-27] others failed to demonstrate a significant effect, or on the contrary, observed a decreased risk in the presence of the minor Ser482 allele.[28-31]

The role of the *DRD2* SNPs in the development of diabetes is not well understood. McGuire V et al.[32] demonstrated that the frequency of the *Taq1a* 'risk' allele (A1) varies according to race/ethnicity. Multiple studies have shown that the presence of at least one A1 risk (T) allele is associated with body mass index (BMI) in adults. [33] The *DRD2/ANKK1* TaqIA polymorphism could influence individual preferences for high-fat/high-sugar foods.[34] Ramos-Lopez et al.[34] suggest that the interactions between the *DRD2/ANKK1* TaqIA polymorphism and dietary factors (sugar and fats) influence triglyceride levels in diabetic patients. Barnard et al.[34] found that the A1 (T) allele appears to be highly prevalent among individuals with type 2 diabetes. Papysheva et al.[35] found that the *DRD2/ANKK1* TaqIA (rs1800497) is involved in carbohydrate and lipid metabolism disorders in pregnant women with GDM. Potential influence of the *DRD2/ANKK1* TaqIA polymorphism on GDM merits further exploration.

**In conclusion**, a study of the *DRD2/ANKK1* TaqIA (rs1800497 SNP), *PPARGC1A* rs8192678 SNP, and *ACE* I/D revealed statistically significant increased risks for GDM in carriers of the TT genotype and T allele of the *DRD2/ANKK1* TaqIA (rs1800497 SNP).

## Competing Interests

The authors declare that they have no competing interests.

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