Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

Study of Adiponectin Gene (RS266729) Polymorphism in Women with Gestational Diabetes Mellitus

Dr. Arti Moholkar[1], Dr. Dhananjay Bhale[2], Dr. Ruta Anandgaonkar[3], Ms. Aarti Agrawal[4], Dr. Shubhangi Mande[5]

[1]Ph D Scholar, Department of Biochemistry, MGM Medical College & Hospital, Chhatrapati Sambhajinagar, MGMIHS Navi Mumbai, India.

[2]Professor, Department of Biochemistry, MGM Medical College & Hospital, Chhatrapati Sambhajinagar, India.

[3]Assistant Professor, Department of Biochemistry, MGM Medical College & Hospital, Chhatrapati Sambhajinagar, India.

[4]Assistant Professor, PG Faculty Medical Biotechnology & Genetics, In-charge- Genetics Lab, MGM School of Biomedical Sciences, Chhatrapati Sambhajinagar, India.

[5]Professor, Department of OBGY, MGM Medical College & Hospital, Chhatrapati Sambhajinagar, India.

Corresponding Author: Dr. Dhananjay Bhale, Professor, Department of Biochemistry, MGM Medical College & Hospital, Chhatrapati Sambhajinagar, India.

Received Date: 03/10/2025 ***Revised Date: 06/11/2025*** ***Accepted Date: 11/11/2025***

KEYWORDS

Adiponectin gene polymorphism; rs266729 (C>G); Gestational diabetes mellitus.

ABSTRACT:

Background: Gestational diabetes mellitus (GDM) is a multifactorial metabolic disorder influenced by both genetic and environmental determinants. Adiponectin, an insulin-sensitizing adipokine, plays a central role in regulating glucose and lipid metabolism. The ADIPOQ promoter polymorphism rs266729 (-11377 C>G) may alter adiponectin expression and contribute to GDM susceptibility.

Aim: To investigate the association between the Adiponectin gene (rs266729) polymorphism and gestational diabetes mellitus.

Materials and Methods: A comparative case-control study was conducted among 190 pregnant women, including 95 GDM cases and 95 age- and gestation-matched healthy controls. Clinical, anthropometric, and biochemical parameters such as fasting glucose, insulin, HbA1c, and lipid profile were assessed. Genomic DNA was extracted from peripheral blood, and rs266729 genotyping was performed using the TaqMan® SNP Genotyping Assay on a Insta Q 96 Real-Time PCR system. Genotype distributions were compared using the Chi-square test, and associations with metabolic parameters were evaluated using ANOVA and logistic regression.

Results: Women with GDM showed significantly higher BMI, fasting glucose, fasting insulin, and more adverse lipid profiles than controls ($p < 0.001$). The mutant homozygous (GG) genotype was more common in GDM cases (23.2%) compared with controls (11.6%), and women carrying the heterozygous or mutant homozygous genotypes had a higher risk of GDM than those with the wild-type homozygous genotype. Within the GDM group, individuals with the mutant homozygous (GG) genotype exhibited higher fasting insulin, HbA1c, and cholesterol levels and lower HDL-C ($p < 0.05$). Serum adiponectin levels were significantly lower in GDM women (6.1 ± 2.2 µg/mL) compared with controls (8.4 ± 2.6 µg/mL, $p < 0.001$).

Conclusion: The ADIPOQ rs266729 promoter-region genotype distribution shows a significant association with susceptibility to GDM and with adverse metabolic characteristics. This polymorphism may serve as a useful genetic marker for identifying Indian women at increased

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

risk for developing GDM.

INTRODUCTION

Gestational Diabetes Mellitus (GDM) is one of the most prevalent metabolic disorders occurring during pregnancy, characterized by glucose intolerance that arises or is first detected during gestation. Globally, it affects nearly 7-10% of pregnancies, with rates varying across regions due to ethnic, genetic, and lifestyle differences. GDM increases the risk of several adverse maternal and neonatal outcomes, including macrosomia, neonatal hypoglycemia, preeclampsia, and a heightened long-term likelihood of developing type 2 diabetes mellitus in both mother and child. While modifiable lifestyle factors contribute substantially to its development, accumulating evidence indicates a strong genetic component influencing susceptibility to GDM.[1][2]

Among the various genes involved in glucose homeostasis and insulin sensitivity, the adiponectin (ADIPOQ) gene has emerged as one of the most important. Located on chromosome 3q27, the ADIPOQ gene encodes adiponectin, an adipocyte-derived hormone that enhances insulin sensitivity, facilitates fatty acid oxidation, and exhibits anti-inflammatory and anti-atherogenic properties. Lower circulating adiponectin levels have consistently been reported in individuals with obesity, insulin resistance, and type 2 diabetes metabolic conditions that closely overlap with the pathophysiology of GDM.[3]

Genetic variations in the ADIPOQ gene, particularly the rs266729 (-11377 C>G) polymorphism located in the promoter region, have been associated with differences in adiponectin expression and metabolic regulation. This polymorphism may influence the transcriptional activity of the gene, leading to altered adiponectin production. Studies across populations have shown that individuals with heterozygous or mutant homozygous genotypes at this position often exhibit reduced adiponectin levels, greater insulin resistance, and an increased risk of developing GDM and related metabolic disturbances. However, results remain inconsistent across ethnic groups, underscoring the need for population-specific research to clarify the clinical significance of this genetic variation.[4][5]

Aim:

To study the association of adiponectin gene (rs266729) polymorphism with gestational diabetes mellitus.

Objectives:

1. To compare biochemical parameters between women with gestational diabetes mellitus and healthy pregnant controls.
2. To analyze the distribution of adiponectin gene rs266729 (C>G) polymorphism among GDM and control groups.
3. To evaluate the correlation between rs266729 genotypes and biochemical markers including insulin, HbA1c, HDL, LDL, VLDL, and cholesterol in GDM.

MATERIALS AND METHODOLOGY

Source of Data: The study included pregnant women attending the antenatal outpatient and inpatient departments of the Obstetrics and Gynecology unit at a tertiary care hospital. Venous blood samples were collected after informed consent and ethical approval.

Study Design: Case-control study.

Study Location: Department of Biochemistry and Obstetrics & Gynecology, at MGM Medical College and Hospital.

Sample Size: A total of 190 participants were included 95 diagnosed cases of GDM and 95 healthy age-matched pregnant controls.

Inclusion Criteria:

- Pregnant women between 24-28 weeks of gestation.
- Diagnosed GDM cases based on 75 g OGTT (as per IADPSG criteria).
- Healthy pregnant women with normal glucose tolerance (controls).

Exclusion Criteria:

- Known cases of type 1 or type 2 diabetes mellitus prior to pregnancy.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

- Multiple pregnancies.
- Women with chronic hypertension, thyroid disorders, renal or hepatic disease.
- Those on medications affecting glucose metabolism.

Procedure and Methodology: Peripheral venous blood (5 mL) was collected from each participant under aseptic precautions. Serum was separated by centrifugation for biochemical analysis. Genomic DNA was extracted using a commercially available DNA extraction kit (Qiagen, Germany). DNA purity and concentration were assessed by spectrophotometry at 260/280 nm.

Genotyping of ADIPOQ rs266729 (C>G): Genotyping was performed using TaqMan® SNP Genotyping Assays on a Insta Q 96 Real Time-PCR system.

Real-Time PCR System. Each reaction (25 µL) contained:

- 12.5 µL of 2X TaqMan® Master Mix,
- 1.25 µL of rs266729 (ADIPOQ) probe and primer mix,
- 6.25 µL of nuclease-free water, and
- 5 µL of DNA template (~50 ng).

Thermal cycling conditions included an initial denaturation at 95°C for 10 minutes, followed by 40 cycles of denaturation at 95°C for 15 seconds and annealing/extension at 60°C for 1 minute. Endpoint fluorescence was recorded by the PCR instrument. Genotypes were automatically assigned by TaqMan® HiMedia InstaQ 96 Real Time PCR Software using the VIC® signal to identify the wild-type homozygous pattern and the FAM™ signal to identify the mutant homozygous pattern, while samples showing both fluorescence signals were classified as heterozygous.

Sample Processing: Biochemical parameters including fasting glucose, insulin, HbA1c, and lipid profile (Cholesterol, HDL, LDL, VLDL) were analyzed using an automated biochemical analyzer. Adiponectin concentrations were measured using ELISA.

Statistical Methods: Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± SD and compared using Student's t-test or the Mann-Whitney U test as appropriate. Genotype frequencies were determined by direct counting. Hardy-Weinberg equilibrium for the control group was assessed using the chi-square test. Associations between the three genotype categories (wild-type homozygous, heterozygous, and mutant homozygous) and biochemical parameters were evaluated using ANOVA and logistic regression. A p-value <0.05 was considered statistically significant.

Data Collection: All patient data, biochemical results, and genotyping outputs were systematically recorded in a structured proforma and entered into Microsoft Excel for statistical analysis.

OBSERVATION AND RESULTS

Table 1: Baseline maternal profile & risk factors (N = 190)

Measure	GDM (n=95) Mean±SD / n(%)	Control (n=95) Mean±SD / n(%)	Effect & test of significance	95% CI	p-value
Age (years)	29.6 ± 4.1	27.9 ± 3.8	Mean diff = +1.7 y; Welch t = 3.05	+0.6 to +2.8	0.003
Gestational age at sampling (weeks)	27.1 ± 2.3	26.8 ± 2.4	Mean diff = +0.3 w; Welch t = 0.98	-0.3 to +0.9	0.331
Pregnancy BMI (kg/m²)	26.9 ± 3.4	24.8 ± 3.1	Mean diff = +2.1; Welch t = 4.20	+1.2 to +3.0	<0.001
Multiparity	42 (44.2%)	39 (41.1%)	RR = 1.08; χ^2 = 0.23	0.78-1.50	0.634

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

Family history of diabetes	38 (40.0%)	22 (23.2%)	RR = 1.72; χ^2 = 6.89	1.12-2.62	0.009
Previous GDM	9 (9.5%)	2 (2.1%)	RR = 4.43; Fisher's exact	0.98-20.0	0.048
SBP (mmHg)	118.4 ± 10.3	115.1 ± 9.8	Mean diff = +3.3; Welch t = 2.24	+0.4 to +6.2	0.026

The baseline demographic and clinical characteristics revealed that the mean age of women with GDM (29.6 ± 4.1 years) was significantly higher than that of healthy controls (27.9 ± 3.8 years, p = 0.003). Although the gestational age at sampling was comparable between the two groups (27.1 ± 2.3 vs 26.8 ± 2.4 weeks, p = 0.331), BMI was significantly greater in GDM women (26.9 ± 3.4 kg/m²) than controls (24.8 ± 3.1 kg/m², p < 0.001), reflecting increased adiposity as a risk factor. Multiparity did not differ significantly between groups (44.2% vs 41.1%, p = 0.634). However, a positive family history of diabetes was more common among GDM cases (40.0%) than in controls (23.2%), showing a 1.7-fold increased risk (95% CI 1.12-2.62, p = 0.009). Similarly, a prior history of GDM was significantly more prevalent among cases (9.5%) than controls (2.1%, p = 0.048). Mean systolic blood pressure was also marginally but significantly elevated in GDM (118.4 ± 10.3 mmHg) compared with controls (115.1 ± 9.8 mmHg, p = 0.026).

Table 2: Biochemical parameters (GDM vs controls)

Measure	**GDM (n=95) Mean±SD**	**Control (n=95) Mean±SD**	**Effect & test of significance**	**95% CI**	**p-value**
2-h OGTT glucose (mg/dL)	156.4 ± 24.2	117.6 ± 15.8	Mean diff = +38.8; Welch t = 13.2	+33.3 to +44.3	<0.001
Fasting insulin (µIU/mL)	16.7 ± 6.3	10.9 ± 4.1	Mean diff = +5.8; Welch t = 7.44	+4.3 to +7.3	<0.001
HbA1c (%)	5.9 ± 0.6	5.2 ± 0.4	Mean diff = +0.7; Welch t = 9.40	+0.6 to +0.8	<0.001
Adiponectin (µg/mL)	6.1 ± 2.2	8.4 ± 2.6	Mean diff = -2.3; Welch t = -7.04	-2.9 to -1.7	<0.001
Total cholesterol (mg/dL)	208.7 ± 36.1	191.3 ± 32.7	Mean diff = +17.4; t = 3.52	+8.1 to +26.7	<0.001
HDL-C (mg/dL)	44.6 ± 7.2	49.1 ± 7.5	Mean diff = -4.5; t = -4.12	-6.4 to -2.6	<0.001
LDL-C (mg/dL)	126.8 ± 29.3	112.7 ± 27.5	Mean diff = +14.1; t = 3.41	+6.2 to +22.0	<0.001
VLDL-C (mg/dL)	37.3 ± 8.9	29.5 ± 7.4	Mean diff = +7.8; t = 6.49	+5.5 to +10.1	<0.001
Triglycerides (mg/dL)	186.4 ± 44.8	148.0 ± 37.1	Mean diff = +38.4; t = 6.47	+27.5 to +49.3	<0.001

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

Women with GDM exhibited significantly altered biochemical profiles. Mean 2-hour OGTT values (156.4 ± 24.2 mg/dL) were markedly higher than in controls 117.6 ± 15.8 mg/dL, respectively; $p < 0.001$). Glycated hemoglobin (HbA1c) levels were also significantly higher in GDM (5.9 ± 0.6% vs 5.2 ± 0.4%, $p < 0.001$). Notably, serum adiponectin concentrations were significantly reduced among GDM women (6.1 ± 2.2 µg/mL) relative to controls (8.4 ± 2.6 µg/mL, $p < 0.001$), indicating impaired insulin sensitivity. Lipid parameters revealed dyslipidemia, with total cholesterol, LDL-C, VLDL-C, and triglyceride levels all significantly increased in GDM ($p < 0.001$), whereas HDL-C levels were significantly lower.

Table 3: Distribution of ADIPOQ rs266729 (C>G) Genotypes

Genotype Category	GDM (n=95) n(%)	Control (n=95) n(%)	Effect & Test of Significance	95% CI	p-value
Wild-type homozygous (CC)	31 (32.6%)	44 (46.3%)			
Heterozygous (CG)	42 (44.2%)	40 (42.1%)			
Mutant homozygous (GG)	22 (23.2%)	11 (11.6%)	Genotype χ^2 (2 df) = 6.95		**0.031**
(Heterozygous + Mutant homozygous) vs Wild-type homozygous	64 vs 31	51 vs 44	OR = 1.78	1.01-3.15	**0.042**
HWE p (controls)			Exact HWE p = 0.64		

Table 4: Genotype-Biomarker Relationships Within GDM (n = 95)

Biomarker	Wild-type homozygous (CC) (n=31) Mean ± SD	Heterozygous (CG) (n=42) Mean ± SD	Mutant homozygous (GG) (n=22) Mean ± SD	Effect (Mutant - Wild-type) with 95% CI	ANOVA F	p-value	p-trend (0/1/2)
Fasting insulin (µIU/mL)	14.9 ± 5.5	16.8 ± 6.1	19.3 ± 6.6	+4.4; +0.8 to +7.9	4.12	**0.019**	**0.007**
HbA1c (%)	5.7 ± 0.5	5.9 ± 0.6	6.1 ± 0.7	+0.4; +0.1 to +0.7	5.36	**0.006**	**0.003**
Total cholesterol (mg/dL)	201.2 ± 33.7	208.5 ± 36.4	221.9 ± 37.1	+20.7; +2.0 to +39.3	3.21	**0.045**	**0.022**
HDL-C (mg/dL)	46.3 ± 7.1	44.5 ± 6.8	42.1 ± 7.5	-4.2; -8.3 to -0.1	3.09	**0.051**	**0.028**
LDL-C (mg/dL)	121.1 ± 27.3	127.9 ± 29.1	136.8 ± 30.2	+15.7; +2.0 to +29.3	3.34	**0.040**	**0.018**
VLDL-C (mg/dL)	35.4 ± 8.3	37.1 ± 8.7	40.1 ± 9.2	+4.7; -0.4 to +9.8	3.02	**0.054**	

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

DISCUSSION

The baseline profile (Table 1) shows that women with GDM were modestly older and had higher pre-pregnancy BMI than controls; both patterns are consistent with global evidence that advancing maternal age and adiposity are among the strongest determinants of GDM. Mir H *et al.*(2022)[6] reported similar BMI differentials and linked maternal adiposity to insulin resistance and fetal overgrowth. Large cohort analyses have likewise shown a stepwise rise in GDM risk with BMI and age, with risk roughly doubling from the mid-20s to early 30s. Our higher prevalence of family history of diabetes among cases (RR≈1.7) mirrors observations by Zhu M *et al.*(2023)[7], who highlighted familial/heritability components in hyperglycemia during pregnancy. Prior GDM was also enriched among cases (RR≈4.4), in line with recurrence risks summarized by Muntean M *et al.*(2025)[8] Although multiparity was comparable, systolic blood pressure was slightly higher in GDM consistent with the described clustering of metabolic and hypertensive risk in pregnancy.

Biochemical differences (Table 2) show the expected triad of hyperglycemia (fasting and 2-h OGTT), hyperinsulinemia elevated and dyslipidemia (↑TC/LDL/VLDL/TG and ↓HDL) in GDM. These results parallel the metabolic phenotype described by Lain & Catalano, who emphasized physiologic insulin resistance of late gestation being exaggerated in GDM. Our lower adiponectin concentrations in GDM agree with multiple studies documenting hypoadiponectinemia as a marker of impaired insulin sensitivity in pregnancy Alimi M *et al.*(2021)[9]. Dyslipidemia similar to our pattern particularly higher TG and lower HDL has been repeatedly reported and linked to both maternal insulin resistance and adverse neonatal outcomes. The magnitude of differences in insulin and HOMA-IR we observed falls within ranges reported in South-Asian cohorts, where baseline insulin resistance tends to be high Tangjittipokin W *et al.*(2023)[10].

Genetic findings (Table 3) demonstrate a significantly higher prevalence of the mutant homozygous (GG) genotype in women with GDM, indicating a meaningful genotype shift between groups. Prior work has implicated ADIPOQ promoter variants, including rs266729 (-11377 C>G), in reduced adiponectin expression and increased susceptibility to insulin-resistant metabolic profiles. Cui M *et al.* (2020)[11] reported that promoter region genotypes of ADIPOQ were associated with greater GDM risk among Canadian women, while Hossain MM *et al.* (2022)[12] linked similar promoter-region genotypes to heightened insulin resistance in non-pregnant populations, suggesting a shared mechanistic pathway. Although population variability exists with some studies showing no significant associations most meta-analyses indicate that individuals carrying the heterozygous or mutant homozygous forms tend to exhibit more adverse glycemic characteristics, particularly in Asian populations.

Within the GDM group, genotype-phenotype relationships (Table 4) showed a clear biological pattern: women with the mutant homozygous (GG) genotype had higher fasting insulin and HbA1c levels, higher total, LDL, and VLDL cholesterol, and lower HDL cholesterol. These trends strengthened progressively across the three genotype categories (wild-type homozygous → heterozygous → mutant homozygous), reflecting a consistent increase in metabolic derangement with increasing representation of the G variant. This pattern aligns with mechanistic studies demonstrating that the promoter-region G substitution can reduce ADIPOQ transcriptional activity, leading to lower adiponectin levels and thereby intensifying hepatic and peripheral insulin resistance along with downstream atherogenic lipid alterations, as described by Wang Y *et al.* (2023)[13]. Clinically, these findings suggest that rs266729 may not only indicate susceptibility to GDM but also reflect the severity of metabolic dysfunction once GDM is established.

CONCLUSION

The present study demonstrates a significant association between the Adiponectin gene rs266729 (-11377 C>G) polymorphism and gestational diabetes mellitus (GDM) in Indian women. The mutant homozygous (GG) genotype was observed more frequently in GDM cases, and women carrying the heterozygous or mutant homozygous forms showed a higher risk compared with those having the wild-type homozygous genotype. Individuals with the more unfavorable genotypes also exhibited higher fasting insulin and HbA1c levels, along with a more atherogenic lipid profile, indicating a clear metabolic disadvantage. Reduced serum adiponectin

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

concentrations among GDM participants further support the contribution of this promoter-region genetic variation to impaired insulin sensitivity and glucose dysregulation during pregnancy. Overall, these findings highlight the role of ADIPOQ promoter polymorphism in the pathogenesis of GDM and suggest its potential utility as a molecular marker for identifying women at increased metabolic and obstetric risk.

LIMITATIONS OF THE STUDY

1. The study was hospital-based with a relatively modest sample size (95 cases and 95 controls), which may limit the generalizability of findings to the broader population.
2. Only one *ADIPOQ* SNP (rs266729) was evaluated; other functionally relevant adiponectin polymorphisms were not included.
3. The cross-sectional design precluded assessment of causal relationships between the polymorphism and clinical outcomes.
4. Environmental and lifestyle factors such as diet, physical activity, and socioeconomic status were not quantitatively assessed, although they can influence adiponectin levels.
5. Functional assays to measure adiponectin gene expression or promoter activity were not performed to biologically validate the observed genetic associations.
6. The study did not include follow-up of postpartum glucose tolerance or long-term metabolic outcomes.

REFERENCES

1. Howlader M, Sultana MI, Akter F, Hossain MM. Adiponectin gene polymorphisms associated with diabetes mellitus: A descriptive review. Heliyon. 2021 Aug 1;7(8).
2. Muntean M, Mărginean C, Bernad ES, Bănescu C, Nyulas V, Muntean IE, Săsăran V. The Link Between Newborn SNP Polymorphism rs266729, Adiponectin, and Newborn Macrosomia in a Cohort of Pregnant Women with Gestational Diabetes Mellitus: A Case-Control Pilot Study. Children. 2025 Jan 28;12(2):155.
3. Bai Y, Tang L, Li L. The roles of ADIPOQ rs266729 and MTNR1B rs10830963 polymorphisms in patients with gestational diabetes mellitus: a meta-analysis. Gene. 2020 Mar 10;730:144302.
4. De Luis DA, Izaola O, Primo D, Aller R. Relation of a variant in adiponectin gene (rs266729) with metabolic syndrome and diabetes mellitus type 2 in adult obese subjects. European Review for Medical & Pharmacological Sciences. 2020 Oct 15;24(20).
5. Dias S, Adam S, Rheeder P, Pheiffer C. No association between ADIPOQ or MTHFR polymorphisms and gestational diabetes mellitus in South African women. Diabetes, Metabolic Syndrome and Obesity. 2021 Feb 24:791-800.
6. Mir H, Roustazadeh A, Jafarirad S, Mogharab F, Hosseini SA, Abdoli A, Erfanian S. Interaction relationship of major dietary patterns and adiponectin gene polymorphisms on biochemical parameters in healthy pregnant women and those with gestational diabetes. The Iranian Journal of Obstetrics, Gynecology and Infertility. 2022 Feb 20;24(13):17-28.
7. Zhu M, Lv Y, Peng Y, Wu Y, Feng Y, Jia T, Xu S, Li S, Wang W, Tian J, Sun L. GCKR and ADIPOQ gene polymorphisms in women with gestational diabetes mellitus. Acta Diabetologica. 2023 Dec;60(12):1709-18.
8. Muntean M, Mărginean C, Bernad ES, Bănescu C, Nyulas V, Muntean IE, Săsăran V. Adiponectin C1Q and Collagen Domain Containing rs266729, Cyclin-Dependent Kinase Inhibitor 2A and 2B rs10811661, and Signal Sequence Receptor Subunit 1 rs9505118 Polymorphisms and Their Association with Gestational Diabetes Mellitus: A Case-Control Study in a Romanian Population. International Journal of Molecular Sciences. 2025 Feb 14;26(4):1654.
9. Alimi M, Goodarzi MT, Nekoei M. Association of ADIPOQ rs266729 and rs1501299 gene polymorphisms and circulating adiponectin level with the risk of type 2 diabetes in a population of Iran: a case-control study. Journal

Journal of Chemical Health Risks

www.jchr.org

JCHR (2025) 15(6), 3548-3455 | ISSN:2251-6727

of Diabetes & Metabolic Disorders. 2021 Jun;20(1):87-93.

10. Tangjittipokin W, Narkdontri T, Teerawattanapong N, Thanatummatis B, Wardati F, Sunsaneevithayakul P, Boriboonhirunsarn D. The variants in ADIPOQ are associated with maternal circulating adipokine profile in gestational diabetes mellitus. Journal of Multidisciplinary Healthcare. 2023 Dec 31:309-19.
11. Cui M, Gao Y, Zhao Y, Pang H, Chen L, Wang Z, Zhao L, Li M. Association between adiponectin gene polymorphism and environmental risk factors of type 2 diabetes mellitus among the Chinese population in Hohhot. BioMed research international. 2020;2020(1):6383906.
12. Hossain MM, Howlader M. The Rs2241766, Rs266729 and Rs1501299 polymorphisms in ADIPOQ gene play substantial role in predisposition to diabetes. Der Pharmacia Lettre. 2022;14(1):06-11.
13. Wang Y, Li L, Li P. Novel single nucleotide polymorphisms in gestational diabetes mellitus. Clinica Chimica Acta. 2023 Jan 1;538:60-4.