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Peroxisome Proliferator-Activated Receptor Gamma (PPARG2) rs3856806 Gene Polymorphism as a Risk Factor for Type 2 Diabetes Mellitus

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ABSTRACT

Background: Diabetes Mellitus (DM) is a long-term metabolic illness brought on by either insufficient or inefficient insulin production by the pancreas. Worldwide, 537 million adults between the ages of 20 and 79 are thought to have DM. Genetics is one of the risk factors involved in the pathophysiology of type 2 DM. The gene encodes PPARG2 protein, a group of proteins that are part of the core receptor in carbohydrate and lipid metabolism. Among the two isoforms, PPARG2 is specific to adipose tissue and plays an important role in adipogenesis and mediating insulin sensitivity.

Aims: This study aims to examine the PPARG2 rs3856806 gene polymorphism as a risk factor for the occurrence of type 2 diabetes mellitus in the Cirebon population.

Methods: This case-control study involved 30 cases of type 2 DM and 30 healthy controls. Data were obtained with blood sugar level test, DNA extraction, and PCR-RFLP with Eco72I restriction enzyme, followed by visualization of results using gel electrophoresis.

Results: The C Allele frequency was higher in the case group (76.6%) while the T Allele frequency was higher in the control group (56.6%). The CT heterozygote genotype frequency was more common in the control group (86.7%) and the TT homozygous mutant genotype frequency was higher in the case group (3%) compared to the control group (0%). The results of the Chi-Square Test obtained a p-value of 0.001 with an OR value of 0.118 (CI95%=0.033-0.422).

Conclusion: The PPARG2 rs3856806 polymorphism was significantly associated with lower odds of type 2 diabetes mellitus in the population of Cirebon, Indonesia, when analyzed under a dominant genetic model comparing T-allele carriers (CT + TT) to non-carriers (CC).

Keywords: *PPARG2; rs3856806 Polymorphism; Type 2 Diabetes Mellitus.*

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1. Introduction

Diabetes mellitus (DM) is characterized by hyperglycemia, a chronic metabolic caused by the body's inability to produce insulin or use it effectively It can observed by the elevated blood glucose levels above normal, accompanied by disturbances in carbohydrate, fat, and protein metabolism with characteristic clinical symptoms such as polydipsia, polyuria, polyphagia, and weight loss (Perkumpulan Endokrinologi Indonesia, 2021).

Indonesia had 8.4 million cases of diabetes mellitus in 2000, according to estimates from the World Health Organization (WHO), and this number is projected to increase to approximately 21.3 million by 2030 (World Health Organization, 2023). In addition, according to International Diabetes Federation (IDF) data in 2021, one of the fastest-growing global health emergencies of the 21st century is diabetes. Southeast Asia represents one of the regions with a substantial and rapidly increasing diabetes burden globally. The number of affected adults is expected to increase markedly in the coming decades, and several countries in the region, including Indonesia, are consistently reported among those with the highest numbers of individuals living with diabetes (Kementrian Kesehatan RI, 2020).

Genetic, lifestyle, and environmental factors are major contributors for Type 2 Diabetes Mellitus (Ley, Schulze, Hivert, Meigs, & Hu, 2018). The majority of patients with this disease have at least one parent with T2DM. More than 50 polymorphisms have been identified to date contributing to the risk of type 2 DM. These genes encode proteins that are implicated in a number of DM causing pathways, such as insulin production, secretion, amyloid deposition in beta cells, insulin resistance, alterations in the mechanisms regulating gluconeogenesis (Sapra & Bhandari, 2023). The complex nature of T2DM, which involves the interaction of multiple genes and environmental factors, makes it challenging to establish a definitive relationship between any single genetic variant and disease risk. Furthermore, the genetic diversity of the Indonesian population adds an additional level of complexity to studying gene-disease associations.

Genetically, one of the genes suspected as a factor in diabetes mellitus type 2 disease is the PPARG2 gene. The PPARG2 gene encodes the PPARG2 protein, which is a group of proteins classified within the nuclear receptor family involved in carbohydrate, lipid, and insulin sensitivity regulation (Priya, Sankaran, Ramalingam, Sairam, & Somasundaram, 2016). PPARG2 regulates adipogenesis, energy homeostasis, and lipid metabolism; it is mostly expressed in adipose tissue (Medina-Gomez, Gray, & Vidal-Puig, 2007).

One of the many potential genes discovered by multiple genome-wide association studies (GWAS) is PPARG2, a member of the nuclear hormone receptor superfamily (Sarhangi et al., 2020). The PPARG2 rs3856806 variation, a silent exon-6 mutation known as C161T (rs3856806), is one of its polymorphisms that has been linked to reduced PPARG2 transcription (Shawki et al, 2022).

In Indonesia, where genetic research on type 2 diabetes mellitus (T2DM) is still relatively limited, examining the role of the PPARG2 rs3856806 gene polymorphism as a potential risk factor for T2DM is particularly important. The country's diverse population encompasses numerous ethnic groups, each with unique genetic backgrounds. Furthermore, gene-environment interactions, including diet, physical activity, physical activity levels, and metabolic profiles may further influence the effect of the PPARG2 rs3856806 polymorphism on T2DM susceptibility. These factors underscore the complex interplay between genetic and environmental factors in the development of type 2 diabetes mellitus. Given Indonesia's substantial ethnic diversity, variations in minor allele frequency (MAF) and gene–environment interactions may contribute to population-specific variations in genetic effects (Putri, ASP, Pratamawati, TM, & Nauphar, D. 2025).

Therefore, a promising candidate for investigating genetic susceptibility to type 2 diabetes mellitus in Indonesia is the PPARG2 rs3856806 gene polymorphism. Although studies on the role of this polymorphism in T2DM have been conducted in various populations, little is known about its association with the condition in the Indonesian population. Understanding the role of genetic factors, particularly the PPARG2 rs3856806 gene, in T2DM is a key aim of this study. This research aims to investigate the association between the PPARG2 rs3856806 polymorphism and T2DM risk within the Indonesian population.

2. Methods

Study design

Investigating whether the polymorphism of the PPARG2 rs3856806 gene is linked to an elevated risk of Type 2 Diabetes Mellitus in Waled RSUD Cirebon is the aim of this case-control study. The Ethics Commission approved this study, with ethical approval number 31/EC/FKUGJ/V/2024. It also complied with the administrative requirements of the Research Permit Letter of the Genetics and Biomedical Laboratory, Faculty of Medicine, Universitas Swadaya Gunung Jati, Faculty of Medicine's Genetics and Biomedical Laboratory.

Population and sample

This study was carried out at Waled RSUD Cirebon from April to July 2024. There were 60 participants in the study, 30 of whom were cases and 30 of whom were controls. Purposive sampling was used. A total of 60 participants were recruited for the study, consisting of 30 case subjects diagnosed with T2DM and 30 control subjects without T2DM.

The clinical diagnosis of type 2 diabetes, which was validated by medical records and a review of test data showing elevated fasting blood glucose levels and HbA1c values, served as the basis for selecting the case subjects. The control group did not have a history of diabetes or other serious metabolic diseases, Controls were selected to be comparable in age and sex distribution, however, complete matching was not achieved. Patients who declined to participate in the study after completing and signing the informed consent form, however, were excluded from the study. Additionally, potential confounders such as age, sex, body mass index, and family history of diabetes were controlled for in the analyses to help ensure more accurate and reliable results.

Measurements

In this research, the independent variable was the PPARG2 rs3856806 gene polymorphism measured using the PCR-RFLP method with measurement results of the presence of polymorphism (T-allele presence) and the absence of polymorphism (absence of the T allele). While the dependent variable in this study was Type 2 Diabetes Mellitus, assessed using blood glucose measurements.

After screening and obtaining consent for sample collection, venous blood samples (3 mL) were collected into EDTA tubes. Genomic DNA was extracted by following the manufacturer’s protocol. The purity and concentration of DNA were measured using a Nano spectrophotometer, and samples were stored at –20°C until further analyses.

DNA amplification was performed using a BioRad T100 thermal cycler with
Forward primer: 5’-CAAGACAACCTGCTACAAGC-3’
Reverse primer: 5’-TTCTTGTAGATCTCCTGCAG-3’

The amplification protocol consisted of an initial denaturation at 94°C for 3 minutes, followed by 32 cycles of denaturation at 94°C for 45 seconds, annealing at 57.7°C for 45 seconds, and extension at 72°C for 60 seconds, with a final extension at 72°C for 5 minutes, and ending with 25° for infinity hold. The restriction was carried out at 37 °C overnight using 8 U of Eco72I restriction enzyme, followed by agarose gel electrophoresis. The CC homozygote had 2 bands at 120 and 80 bp. The CT heterozygote had 3 fragments at 200; 120 and 80 bp. The TT homozygote produced a single band at 200 bp.

Data collection

Data on the PPARG2 rs3856806 gene polymorphism and type 2 diabetes mellitus were collected and analyzed using IBM SPSS Statistics, employing chi-square tests for both univariate and bivariate analyses. Medical records were used as secondary data to confirm participants' type 2 diabetes diagnoses. Prior to sample collection, screening procedures were conducted to minimize potential selection bias. Following approval from the Research Ethics Committee of the Faculty of Medicine, Universitas Swadaya Gunung Jati, data collection and acquisition were subsequently completed.

Data analysis

Genotype and allele frequencies were calculated and displayed as a percentage. The distribution of genotypes and alleles in the PPARG2 SNP rs3856806 gene was examined using univariate analysis to ascertain the relationship between independent and dependent factors. Using contingency analysis and a 2×2 table, the polymorphism at rs3856806 with type 2 DM was assessed to determine the odds ratio (OR) and p-value.

3. Results

Respondent characteristics

Using a purposive sampling approach that met the inclusion and exclusion criteria, 60 respondents were selected. Based on factors such as age, sex, type 2 DM history, and length of type 2 DM, the characteristics of study participants are presented. Both primary and secondary data were utilized in this study. Table 1 shows that the distribution of sex among participants was 21 (35%) male and 39 (65%) female. The majority of subjects with type 2 DM ranged in age between 45-59 years, totaling 16 (53.3%), and the majority of subjects without a history of type 2 DM ranged in age between 45-59 years, totaling 17 respondents (56.7%). The characteristics of the duration of type 2 DM disease in the study subjects from the case group who were diagnosed with type 2 DM. The duration of the disease is divided into 2 parts; 0-10 years and 11-20 years.

Table 1. Description of the subject's characteristics

	Case		control	
	Frequency	**Percentage**	**Frequency**	**Percentage**
Gender				
Male	4	13.3%	17	56.7%
Female	26	86.7%	13	43.3%
Total	30	100%	30	100%
Type 2 Diabetes Mellitus				
Yes	30	100%	-	-
No	-	-	30	100%
Total	30	100%	30	100%
Age				
30-44	5	16.7%	9	30%
45-59	16	53.3%	17	56.7%
60-74	9	30%	4	13.3%
Total	30	100%	30	100%
Duration of type 2 diabetes mellitus				
1-10 years	22	73.3%	-	-
11-20 years	8	26.7%	-	-
Total	30	100%	29	100%

The gender distribution of the research subjects in the case group was 4 (13.3%) males and 26 (86.7%) females. This indicates that the sample of individuals with T2DM was predominantly female. Meanwhile, the frequency in the control group was 17 (56.7%) males and 13 (43.3%) females. Given the notable sex distribution difference of the case and control groups, it may be important to consider variations in T2DM prevalence that is connected to sex and how these variations may affect the study's findings.

The frequency distribution of age characteristics in the case group was categorized as follows: 30–44 years (5 respondents, 16.7%), ages 45–59 years with 16 respondents (53.3%), and ages 60–74 years with 9 respondents (30%). This distribution shows a higher concentration of T2DM cases among middle-aged to older adults, which is consistent with the known epidemiology of T2DM, as the risk increases with advancing age. In contrast, the frequency distribution of age characteristics in the control group consisted of 9 respondents (30%) aged 30–44 years, 16 respondents (53.3%) aged 45–59 years, and 4 respondents (13.3%) aged 60–74 years. The higher proportion of younger individuals in the control group may reflect age-related differences in disease onset and prevalence.

In the case group, 22 individuals (73.3%) had a disease duration of 0–10 years, while 8 individuals (26.7%) had duration of 11–20 years. In contrast, no disease duration was identified in the control group. This finding indicates a predominance of relatively shorter disease duration among the case subjects, which may have implications for understanding disease progression and its relationship with genetic factors.

Frequency Distribution of Genotypes

The frequency distribution of genotypes in the PPARG2 rs3856806 gene was obtained through PCR-RFLP examination with the results of CC wild type homozygote (120, 80 base pair), TT homozygote mutant (200 bp), CT heterozygote mutant (200, 120, 80 base pair). Table 2 displays the genotype frequency distribution of the PPARG2 rs3856806 gene.

Table 2. *Frequency distribution of genotypes*

Genotype	Case		Control	
	F	%	F	%
CC	17	56.7%	4	13.3%
TT	1	3%	0	0%
CT	12	40%	26	86.7%
Total	30	100%	30	100%

The results showed that respondents in the case group who had the CC (wildtype homozygote) genotype were 17 people (56.7%), and the TT (homozygote mutant) genotype was 1 person (3.3%), and the CT (heterozygote mutant) genotype was 12 people (40%). In the control group, the CC genotype (wildtype homozygote) was found in 4 participants (13.3%), and the CT genotype (heterozygote mutant) was found in 26 participants (86.7%).

Frequency Distribution of Alleles

The frequency distribution of alleles in the PPARG2 rs3856806 gene polymorphism consisted of allele C and allele T as seen in Table 3.

Table 3. *Frequency distribution of alleles*

Allele	Case		Control	
	F	%	F	%
C	46	76.7%	34	56.7%
T	14	23.3%	26	43.3%
Total	60	100%	60	100%

The table above shows that the allele distribution of alleles for the PPARG2 rs3856806 polymorphism in the case group consisted of 46 C allele (76.7%) and 14 T allele (23.3%). In the control group, there were 34 allele C (56.6%) and 26 allele T (43.3%).

Frequency Distribution of Polymorphisms

The frequency distribution of polymorphisms in the PPARG2 rs3856806 gene was obtained through PCR-RFLP examination. Subjects were considered to have a polymorphism in the PPARG2 rs3856806 gene if they had the TT and CT genotypes. The frequency distribution of polymorphisms in the PPARG2 rs3856806 gene in the case group showed that 13 subjects (43.3%) had a polymorphism, and 17 subjects (56.7%) had no polymorphism. In the control group, 26 subjects (86.7%) were detected with a polymorphism, and 4 subjects (13.3%) who did not have a polymorphism.

Table 4. Frequency distribution of polymorphisms

Polymorphism	Case		Control	
	F	%	F	%
Yes	13	43.3%	26	86.7%
No	17	56.7%	4	13.3%
Total	30	100%	30	100%

Crosstab

The correlation between type 2 diabetes and the PPARG2 rs3856806 gene polymorphism, as determined using PCR-RFLP analysis, was examined using bivariate analysis. Subjects were classified as having polymorphisms if they had 3 bands at 200bp, 120bp, and 80bp or 1 band at 200bp on gel electrophoresis visualization. The chi-square test was used in statistical tests. Table 5 displays the bivariate analysis results.

Table 5. Crosstab

Polymorphism	Case		Control		P	OR	CI (95%)
	F	%	F	%			
Yes	13	43.3%	26	86.7%	0.001	0.118	0.033.
No	17	56.7%	4	13.3%			0.422
Total	30	100%	30	100%			

According to the findings of the bivariate analysis, 13 individuals (43.3%) in the case group have polymorphisms, while 17 individuals (56.7%) did not. In the control group, there were 26 subjects (86.7%) who had polymorphisms and 4 subjects (13.3%) who did not have polymorphisms. After correction (Continuity Correction), the value (p = 0.001) was acquired from the Chi-Square Test findings (OR = 0.118) (CI = 0.033-0.422). These findings indicate that the PPARG2 rs3856806 polymorphism is significantly associated with a reduced risk of Type 2 Diabetes Mellitus in the Cirebon population. The recessive model (TT vs CC+CT) was not further analyzed due to the very low frequency of the TT genotype.

4. Discussion

Type 2 diabetes is the result of interactions between genetic and environmental factors. These environmental factors influence the development of diabetes, but their effects vary among individuals. Even with comparable environmental exposures, some people are more susceptible to diabetes than others, and this increased risk appears to be influenced by genetic inheritance. Both hereditary and environmental factors contribute to the risk of developing diabetes. SNPs are variations found at single nucleotide locations in the DNA genome sequence that contribute to the development of complex diseases (Ali, 2013; Shawki et al., 2022).

The rs3856806 is an SNP suggested to have stronger predictive value for insulin resistance than several others, a mutation located in exon 6. Another designation for this SNP is C1431T, which indicates that at nucleotide 1431, T has been substituted for C. This silent mutation may influence PPARG2 expression through alterations in mRNA processing, potentially affecting adipocyte differentiation (Tiongco et al., 2023). Biologically, it has been proposed that the rs3856806 polymorphism may influence PPARG2 transcriptional activity, potentially affecting adipocyte differentiation and lipid metabolism. Improved lipid storage efficiency and reduced lipotoxicity could hypothetically enhance insulin sensitivity. However, this mechanistic explanation remains hypothetical, as the rs3856806 variant represents a silent mutation and its its functional impact has not been fully established. Further functional and longitudinal studies are needed to clarify the biological relevance of this association.

In this study, the CT genotype was more common in the control group (26 participants, 86.7%) compared to the case group (12 participants, 40%). CT is a heterozygous mutant genotype type with a weight of 200bp, 120bp, and 80bp. The CC genotype (wild-type homozygote) was less frequent in the control group, namely 4 samples (13.3%), and as many as 17 samples (56.7%) in the case group. The analysis results obtained a value (OR = 0.118), (C1 = 0.033-0.422) and (*p* value = *0.001)*. These findings suggest that the rs3856806 polymorphism may be associated with reduced risk of Type 2 Diabetes Mellitus (T2DM) in the Cirebon population. The protective effect of the PPARG2 rs3856806 polymorphism observed in the Cirebon population may reflect population-specific genetic architecture rather than a contradictory biological role of the variant itself (Tiongco et al., 2023). Genetic variants associated with complex metabolic diseases such as T2DM often show heterogeneous effects across ethnic groups due to differences in allele frequency and genetic background (Sarhangi et al., 2020).

Although this study obtained these results, there are studies reporting different findings. Shawki et al. (2022) conducted a study on the Egyptian population, using 140 T2DM cases and 120 controls. The study found substantial p-values and reported that the PPARG2 rs3856806 polymorphism may play a role in diabetes susceptibility (Shawki et al., 2022). In addition, Lv et al. (2017) conducted a study on the Han population in China, using 647 cases of type 2 diabetes mellitus and 650 control samples. This study discovered that carriers of the T allele of the polymorphism rs3856806 enhanced the incidence of type 2 diabetes in the Han population in China (Lv et al., 2017).

The PPARG2 rs3856806 exhibits ethnic variation in Minor Allele Frequency (MAF), with allele frequencies differing across populations. MAF represents the frequency of the less common allele, which can vary among populations or ethnic groups. MAF is used to describe the level of genetic diversity in an individual and population. In this study, within the Cirebon population, allele C (0.766) was the most common in the case group and (0.566) in the control group. Referring to dbSNP, data on the MAF for SNP rs3856806 in Europeans shows an allele C frequency (0.88) higher than allele T (0.12). These figures are similar to those reported in Asian populations, which show an allele C frequency (0.82) higher than allele A (0.18).

Although the dominant model analysis showed a strong protective association (OR = 0.118), caution is warranted when interpreting the effect's magnitude. The relatively small sample size may have led to overestimation of the true effect size, which is a common occurrence in early genetic association research. Furthermore, the wide confidence interval indicates statistical uncertainty. A single SNP is unlikely to exert a substantial independent protective effect due to the complicated and multifactorial nature of type 2 diabetes mellitus. Therefore, in order to reduce the likelihood of error, the current findings should be considered exploratory and require validation in larger, well-powered studies.

This is the first study in Indonesia to examine the PPARG2 rs3856806 polymorphism in relation to T2DM. Thus, this study may serve as an initial reference for future research on this topic. The results may differ across studies due to population differences and small sample sizes. Therefore, it is recommended for future researchers to conduct research with larger sample sizes.

Several suggestions for further research include using a larger and more representative sample size, as well as examining the influence of the PPARG2 rs3856806 polymorphism alongside other SNP variants, other related genes, and additional risk factors associated with T2DM. Considering that T2DM is a complex condition influenced by genetic–environmental interactions and individual lifestyle differences. These factors may cause variations in genetic effects across populations, and future studies are encouraged to involve diverse Indonesian populations and apply alternative research methods, including experimental designs, to obtain more comprehensive and scientifically valid results. Moreover, the current study's limitations, such as not assessing lifestyle factors, family history, BMI, or other potential contributors, as well as not evaluating genotype distribution or Hardy–Weinberg equilibrium due to sample-size limitations, highlight the need for more robust methodological approaches in subsequent research.

5. Conclusion

In conclusion, the findings of this study indicate that the PPARG2 rs3856806 polymorphism is associated with a reduced risk of type 2 diabetes mellitus in the Cirebon population. The genotype distribution showed that the CT heterozygous genotype was more frequently observed in the control group (86.7%) than in the case group (40.0%), whereas the CC wild-type genotype predominated among individuals with type 2 diabetes mellitus (56.7%) compared with controls (13.3%). The TT homozygous mutant genotype was rare and was identified only in the case group (3.3%). Statistical analysis demonstrated a significant association between the PPARG2 rs3856806 polymorphism and type 2 diabetes mellitus, with an odds ratio of 0.118 (95% CI: 0.033–0.422), indicating lower odds of type 2 diabetes mellitus in this population, however, the magnitude of this association should be interpreted with caution. These results suggest that the PPARG2 rs3856806 polymorphism may confer a population-specific protective effect in the pathogenesis of type 2 diabetes mellitus. However, given the relatively small sample size and the focus on a single genetic variant, these findings should be interpreted with caution. Future studies involving larger sample sizes, multiple genetic markers, and a comprehensive evaluation of environmental and metabolic factors are recommended to provide a more complete understanding of the genetic risk of type 2 diabetes mellitus in the Indonesian population.

Conflict of Interest

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References

Ali, O. (2013). Genetics of type 2 diabetes. *World Journal of Diabetes*, *4*(4), 114. https://doi.org/10.4239/wjd.v4.i4.114

International Diabetes Federation. (2021). IDF Diabetes Atlas 10th edition. https://fmdiabetes.org/wp-content/uploads/2022/01/IDF_Atlas_10th_Edition_2021-comprimido.pdf

Kementrian Kesehatan RI. (2020). *Infodatin Pusat Data dan Informasi Kementrian Kesehatan RI. Tetap Produktif, Cegah, dan Atasi Diabetes Melitus*. 1–5. https://id.scribd.com/document/496433422/Infodatin-2020-Diabetes-Melitus

Ley, S. H., Schulze, M. B., Hivert, M. F., Meigs, J. B., Hu, F. B. (2018) Risk Factors for Type 2 Diabetes. In: Cowie CC, Casagrande SS, Menke A, Cissell MA, Eberhardt MS, Meigs JB, Gregg EW, Knowler WC, Barrett-Connor E, Becker DJ, Brancati FL, Boyko EJ, Herman WH, Howard BV, Narayan KMV, Rewers M, Fradkin JE, editors. Diabetes in America. 3rd ed. Bethesda (MD): National Institute of Diabetes and Digestive and Kidney Diseases (US); 2018 Aug. CHAPTER 13. PMID: 33651531. https://europepmc.org/article/nbk/nbk567966

Lv, X., Zhang, L., Sun, J., Cai, Z., Gu, Q., Zhang, R., & Shan, A. (2017). Interaction between peroxisome proliferator-activated receptor gamma polymorphism and obesity on type 2 diabetes in a Chinese Han population. *Diabetology and Metabolic Syndrome*, *9*(1), 1–6. https://doi.org/10.1186/s13098-017-0205-5

Medina-Gomez, G., Gray, S., & Vidal-Puig, A. (2007, October). Adipogenesis and lipotoxicity: Role of peroxisome proliferator-activated receptor γ (PPARγ) and PPARγcoactivator-1 (PGC1). *Public Health Nutrition*, Vol. 10, pp. 1132–1137. https://doi.org/10.1017/S1368980007000614

Perkumpulan Endokrinologi Indonesia. (2021). *Pedoman Pengelolaan dan Pencegahan Diabetes Melitus Tipe 2 Dewasa di Indonesia*. *PB PERKENI*. https://pbperkeni.or.id/wp-content/uploads/2021/06/Pedoman-Pengelolaan-DM-Tipe-2-Dewasa-di-Indonesia-eBook-PDF.pdf

Priya, S. S., Sankaran, R., Ramalingam, S., Sairam, T., & Somasundaram, L. (2016). Genotype phenotype correlation of genetic polymorphism of PPAR gamma gene and therapeutic response to pioglitazone in type 2 diabetes mellitus- a pilot study. *Journal of Clinical and Diagnostic Research*, *10*(2), FC11–FC14. https://doi.org/10.7860/JCDR/2016/16494.7331

Putri, A. S. P., Pratamawati, T. M., & Nauphar, D. (2025). Potassium Inwardly Rectifying Channel Subfamily J Member 11 (KCNJ11) RS5219 Gene Polymorphism as a Risk Factor for Type 2 Diabetes Mellitus in Indonesia: A Case Control Study. *GHMJ (Global Health Management Journal)*, *8*(1s), 135–143. https://doi.org/10.35898/ghmj-81s1160

Sapra, A., & Bhandari P. (2023). Diabetes. *In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing*. https://www.ncbi.nlm.nih.gov/books/NBK551501

Sarhangi, N., Sharifi, F., Hashemian, L., Hassani Doabsari, M., Heshmatzad, K., Rahbaran, M., Jamaldini, S.H., Aghaei Meybodi, H.R. & Hasanzad, M. (2020). PPARG (Pro12Ala) genetic variant and risk of T2DM: a systematic review and meta-analysis. *Scientific Reports*, *10*(1). https://doi.org/10.1038/s41598-020-69363-7

Shawki, H. A., Abo-hashem, E. M., Youssef, M. M., Shahin, M., & Elzehery, R. (2022). PPARɣ2, aldose reductase, and TCF7L2 gene polymorphisms: relation to diabetes mellitus. *Journal of Diabetes and Metabolic Disorders*, *21*(1), 241–250. https://doi.org/10.1007/s40200-021-00963-4

Tiongco, R. E., Basilio, H., Camacho, D. R., Ellorin, W. M., Sico, C. A., & Arceo, E. (2023). Association of the rs3856806 Polymorphism in the PPARG Gene with Type 2 Diabetes Mellitus: A Meta-Analysis of 11,811 Individuals. *Laboratory Medicine*, *54*(2), 193–198. https://doi.org/10.1093/labmed/lmac095

World Health Organization. (2023). Diabetes. https://www.who.int/news-room/fact-sheets/detail/diabetes

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