Sahel Journal of Life Sciences FUDMA (SAJOLS)
September 2025 Vol. 3(3): 105-111
ISSN: 3027-0456 (Print)
ISSN: 1595-5915 (Online)
DOI: *https://doi.org/10.33003/sajols-2025-0303-13*

Research Article

Detection of Hepatocyte Nuclear Factor 1 Alpha (*HNF1A)* Gene Variants Among the Diabetic Patients Attending a Tertiary Health Facility in Lagos, Nigeria

***Makinde Arinola Ibilola[1], Raheem Toyosi Yekeen[1,2], Bamkefa Bukola Ayodeji[1], Adesina Felicia[1], Adegbehingbe Kehinde[1], Adekola Saheed Ayodeji[3], Muinat Adenike Fowora[2], and Bakare, Omonike Christianah[1]**

[1]Department of Biological Sciences, Lead City University, Ibadan, P.M.B. 30678, Nigeria
[2]Molecular Biology and Biotechnology Nigerian Institute of Medical Research, Yaba, Lagos, Nigeria
[3]Department of Medical Laboratory Science, Faculty of Nursing and Allied Health Sciences, University of Abuja, Nigeria
***Corresponding Author's email*: arinolamakinde32@gmail.com; Phone: +2348033721404**

ABSTRACT

Diabetes mellitus (DM) is a long-term metabolic condition characterised by persistent hyperglycaemia and serious complications. It remains a major public health challenge worldwide, including in Nigeria. While lifestyle and environmental factors are well known, genetic influences also contribute to disease development, progression, and treatment response. One gene of particular interest, Hepatocyte Nuclear Factor 1 Alpha (HNF1A), regulates liver genes involved in glucose metabolism, insulin secretion, and transport. Variants in this gene have been linked to Maturity-Onset Diabetes of the Young (MODY) as well as Type 2 Diabetes Mellitus (T2DM). However, information on these variants in Nigerian populations is limited. This study investigated HNF1A gene variants in 100 adults (47% male, 53% female; mean age 46.5 years) attending the Federal Medical Centre, Ebute-Meta, Lagos. Blood glucose and glycated haemoglobin (HbA1c) were measured, and genomic DNA was analysed for HNF1A variants using Polymerase Chain Reaction (PCR) and Restriction Fragment Length Polymorphism (RFLP). A strikingly high prevalence of polymorphisms was observed, with 95% of participants carrying exon 1 variants before restriction digestion. Exon 1 displayed three distinct band patterns, while exons 2–5 showed single but variable bands, indicating genetic diversity. Individuals with exon 1 and exon 5 variants had higher fasting glucose and HbA1c levels, suggesting that these polymorphisms may contribute to impaired glycaemic control. In summary, HNF1A variants are common in both diabetic and non-diabetic Nigerians. Incorporating genetic screening into routine practice could enhance the diagnosis, treatment, and prevention of diabetes in the population.

Keywords: Diabetes mellitus; Genetic screening; Glycaemic control; HNF1A gene; Nigeria

Citation: Makinde, A.I., Raheem, T.Y., Bamkefa, B.A., Adesina, F., Adegbehingbe, K., Adekola, S.A., Muinat A.F., & Bakare, O.C. (2025). Detection of Hepatocyte Nuclear Factor 1 Alpha (*HNF1A)* Gene Variants Among the Diabetic Patients Attending a Tertiary Health Facility in Lagos, Nigeria. *Sahel Journal of Life Sciences FUDMA*, 3(3): 105-111. DOI: *https://doi.org/10.33003/sajols-2025-0303-13*

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder characterised by persistent hyperglycaemia and associated complications, and it continues to pose a significant global health burden, including in Nigeria (ElSayed *et al*., 2025; Olatunde, 2025). While type 2 diabetes mellitus (T2D) accounts for the majority of cases, evidence increasingly highlights the contribution of genetic factors to disease susceptibility, clinical presentation, and therapeutic outcomes.

One gene of particular interest is Hepatocyte Nuclear Factor 1 Alpha (HNF1A), a transcription factor that regulates liver-specific genes and genes involved in glucose metabolism, insulin secretion, and transport. Variants in the HNF1A gene have been implicated in Maturity-Onset Diabetes of the Young (MODY3) as well

as in T2D (Sousa *et al*., 2022). The HNF1A protein comprises three functional domains: the dimerisation domain, the DNA-binding domain, and the transactivation domain. Mutations and polymorphisms in this gene—including I27L, A98V, and S487N—have been reported in both diabetic and non-diabetic individuals, with variable effects on glycaemic control (Yasutaka *et al*., 2022).

Information on the prevalence and mutation patterns of the gene among diabetic individuals in the study area remains unavailable, which may have important implications for personalised medicine and could enhance the diagnosis, prevention, and management of diabetes in clinical practice (Sugandh *et al.*, 2023**).**

Various coding variants of the *HNF1A* gene may range in severity from causing monogenic diabetes to conferring increased risk of type 2 diabetes mellitus or being benign (Fotini and Owen *et al*., 2012). Thus, the low-frequency variant p.Glu508Lys was found to associate with type 2 diabetes mellitus in a Latino population (Karol *et al.,* 2014). Notably, among those subjects with type 2 diabetes mellitus characterised in that study, there was no difference in the clinical phenotype between carriers and non-carriers of the *HNF1A* pGlu508Lys variant. It has also been reported that young lean individuals with type 2 diabetes mellitus harboured a threefold excess of low-frequency non-synonymous variants in some MODY genes relative to old obese control subjects (Karol *et al*., 2014). Moreover, another study investigated whether functional classification of the rare coding *HNF1A* variants reported in the study can predict type 2 diabetes mellitus risk in the general population and a set of variants that reduced HNF-1A transactivation to <60% compared with the wild type protein was shown to associate with type 2 diabetes mellitus (Abdoli *et al*., 2016). The *HNF1A* locus is also known to harbour a risk variant (rs7957187) for late-onset T2DM identified by genome-wide association study (GWAS) meta-analyses Kazunori *et al.* (2017). Recently, the *HNF1A* rs1183910 minor A variant has been associated with an increased risk of T2DM, and it has been suggested that other *HNF1A* common variants, including I27L (rs1169288), A98V (rs1800574) and S487N (rs2464196), in this gene are associated with either impaired insulin secretion (IIS) or an increased risk of T2DM in normal-weight individuals who are less insulin-resistant than obese individuals (Kimiko *et al*., 2012). MODY often affects people under the age of 25 and arises from heterozygous mutations in various transcription factors involved in the growth and maturation of pancreatic β-cells. Additionally, mutations in enzymes involved in the β-cell glucose sensing also result in early-onset diabetes, but then, it is challenging to interpret accurate incidence at the global level as MODY is often misdiagnosed as type 1 and type 2 diabetes mellitus. The difficulty in accurately predicting the onset of T2DM is likely due to a complex combination of genetic and environmental factors, including individual lifestyle (Lall *et al*., 2017). Much remains to be explored in terms of genetic factors in diabetes, though there is accumulating evidence that epigenetic changes (histone modification, DNA methylation, and non-coding RNA) can alter transcription factor binding to the genome and gene expression, independently of the DNA sequence. Nine novel variants comprising seven mutations (one novel mutation 538G≥C at the promoter region and six novel coding region mutations) and two polymorphisms in the *HNF1A* gene. Functional studies revealed reduced transcriptional activity of the *HNF1A* promoter for two promoter variants. We also observed co segregation with diabetes of the Arg263 His coding region mutation in eight members of one MODY family, whereas it was absent in non-diabetic subjects of this family (Radha *et al.,* 2009). Also, there is accumulating evidence that epigenetic changes are associated with the development of T2DM; the underlying molecular mechanisms are not fully understood Kwak and Park, 2016). About 14 MODY subtypes have been reported, including MODY1 (HNF4A), MODY2 (GK), MODY3 (HNF1A), MODY4 (PDX1), MODY5 (HNF1B), and MODY6 (NeuroD1) (Tshivhase *et al*., 2015). Among these, *HNF1A* gene mutations are the most common cause of MODY. Also, SNPs in and near *HNF1A* are associated with an increased risk of T2DM.

Despite increasing research globally, data on the prevalence and mutation spectrum of HNF1A variants in Nigerian populations remain limited. This gap hinders early diagnosis of monogenic diabetes, optimal treatment decisions, and personalised medicine strategies. This study, therefore, aimed to detect and characterise HNF1A gene variants among diabetic patients attending the Federal Medical Centre, Ebute-Metta, Lagos, and to compare findings with non-diabetic controls from the same setting.

MATERIALS AND METHODS

Study Design and Population

This was a cross-sectional analytical study conducted at the Federal Medical Centre, Ebute-Metta, Lagos, Nigeria. The study included 100 participants (48 with diabetes mellitus and 52 apparently healthy controls) recruited through simple random sampling. Eligible participants were adults aged ≥20 years, resident in Lagos State. Exclusion criteria included known chronic kidney disease, liver disease, chronic obstructive

pulmonary disease, alcohol use, smoking, and acute infections.

Sample Size Determination

The minimum sample size was calculated using the formula for prevalence studies, based on a pooled national prevalence of diabetes mellitus in Nigeria of 5.77% (Uloko *et al*., 2018). The computed sample size was 83, but a total of 100 participants were enrolled to increase study power.

Ethical Considerations

Ethical approval was obtained from the Research Ethics Committee of the Nigerian Institute of Medical Research (IRB/24/006). Written informed consent was obtained from all participants before enrolment. Confidentiality was maintained throughout the study.

Sample Collection and Biochemical Analysis

Ten millilitres of venous blood were collected aseptically from each participant into fluoride oxalate and EDTA tubes. Plasma glucose was measured using the glucose oxidase-peroxidase (GOD-POD) method, while glycated haemoglobin (HbA1c) was determined using standard procedures.

DNA Extraction and Genotyping

Genomic DNA was extracted from EDTA whole blood using the QIAamp Blood Mini Kit (QIAGEN, Germany) following the manufacturer's protocol. Polymerase chain reaction (PCR) amplification was performed for exons 1–5 of the HNF1A gene using specific primers (Table 1). Restriction fragment length polymorphism (RFLP) analysis was used to detect gene variants.

Table 1: PCR primer sequences for the *HNF1A* gene

Site	Forward	Reverse
Exon 1	TGCAAGGAGTTTGGTTTGTG	GAAGGTCATGGGGACTCAAC
Exon 2	GCCATGGCAATGAGAAAGAA	GGCAACTGGACAGCCTTTTA
Exon 3	GGCAGAGCTCAHCTTCTCAG	AAGGAGTGGCATGAATGGAA
Exon 4	CTCTGGGAAGGAGAGGTGGT	GTCCCAGAGACACATGCAGA
Exon 5	TGAGTCCCCTAGGGACAGG	CCTGCCTTCCCTGTTAGCTT

Variant Classification

Identified HNF1A variants were interpreted according to the American College of Medical Genetics and Genomics (ACMG) guidelines, classifying them as pathogenic, likely pathogenic, uncertain significance, likely benign, or benign.

Data Collection and Statistical Analysis

Demographic and clinical data were obtained using a structured questionnaire. Statistical analysis was conducted using SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarised as frequencies and percentages. Group comparisons were performed using one-way ANOVA and Student's *t*-test, with significance set at $p < 0.05$.

RESULTS

A total of 100 participants were enrolled, comprising 47 males (47%) and 53 females (53%), with a mean age of 46.45 ± 12.14 years. Forty per cent were within the 51–70-year age group. The mean fasting blood glucose (FBG) was 7.36 ± 3.93 mmol/L, and the mean HbA1c was 6.73 ± 3.94%. There was a statistically significant difference in age between groups ($p < 0.05$), but no significant differences in sex or BMI distribution (Table 2).

Polymorphisms of the HNF1A gene were detected across the exons examined. Exon 1 displayed three distinct banding patterns, while exon 5 showed two fragments after restriction digestion. The overall prevalence of HNF1A variants before and after digestion is presented in Table 3.

Further subgroup analyses (based on age, sex, BMI, and biochemical parameters) showed no statistically significant associations between HNF1A variants and clinical characteristics, fasting glucose, 2HrPP, or HbA1c.

Table 2. Frequency distribution and percentage of some risk factors for the non-diabetic and diabetic mellitus participants with their respective mean ± SD

Parameters		Frequency (N = 100)	Percentage (%)	Mean ± SD
Sex	Male	47	47	
	Female	53	53	
Age (years)	20 – 30	16	16	46.45 ± 12.14
	31 – 40	13	13	
	41 – 50	31	31	
	51 – 70	40	40	
BMI (Kg/m^2)	Normal	63	63	24.30 ± 3.88
	Overweight	27	27	
	Obese	10	10	
Fasting Blood Sugar (mmol/L)	Non-Diabetes	43	43	7.36 ± 3.93
	Diabetes	57	57	
2HrPP (mmol/L)	Non-Diabetes	21	21	14.25 ± 4.03
	Diabetes	79	79	
HbA1c (%)	Non-Diabetes	52	52	6.73 ± 3.94
	Diabetes	48	48	
Height (m)		100	100	1.74 ± 0.06

Table 3. Frequency and percentage prevalence of *HNF1A* gene variants amongst the study populations before digestion and after digestion

Parameters		Frequency		Percentage (%)	
		Negative	**Positive**	**Negative**	**Positive**
Before Digestion	Exon 1 (505bp)	5	95	5	95
	300bp	54	46	54	46
	118bp	90	10	90	10
	Exon 2 (418bp)	10	90	10	90
	Exon 3 (368bp)	15	85	15	85
	Exon 4 (469bp)	2	98	2	98
	Exon 5 (919bp)	18	82	18	82
After Digestion	Exon 1 digest (284bp)	4	96	4	96
	Exon 1 digest (145bp)	25	75	25	75
	Exon 5 digest (725bp)	38	62	38	62
	Exon 5 digest (165bp)	99	1	99	1

DISCUSSION

This study detected HNF1A gene variants and explored their association with diabetes. A high prevalence of HNF1A variants was observed in both diabetic and non-diabetic participants, with 95% of individuals carrying exon 1 variants before digestion. The presence of variants was particularly frequent in exon 1 and exon 5, although no statistically significant difference in their distribution was found between the diabetic and non-diabetic groups ($p > 0.05$). This suggests that while HNF1A variants are widespread in this Nigerian population, they may not independently account for the development of diabetes, consistent with earlier reports that MODY represents only a minority of diabetes cases globally (Rubio-Cabezas *et al*., 2019; Ogbera and Ekpebegh, 2014).

The Nigerian diabetes landscape has largely been viewed within the wider context of the global type 2 diabetes (T2D) epidemic. This epidemic is strongly driven by obesity, insulin resistance, and lifestyle shifts linked to urbanisation (Uloko et al., 2018; IDF, 2021). Numerous studies have shown a high prevalence of overweight and obesity among Nigerian T2D patients (Olamoyegun *et al*., 2024). While public health

interventions focusing on diet and exercise remain vital, the findings from this study highlight the importance of parallel clinician education. Physicians should recognise that not all diabetes cases are T2D and consider genetic evaluation in patients with early onset, a strong family history, or normal BMI.

Globally, MODY is significantly underdiagnosed and often misclassified as type 1 or type 2 diabetes. Estimates from high-income countries suggest that 1–5% of diabetes cases may be monogenic (Shields *et al*., 2018). In Africa, however, genetic testing remains rare, and the true prevalence of MODY is unknown (Bazzazzadehgan *et al.*, 2025). The detection of HNF1A variants in this study aligns with growing evidence that MODY exists in African populations. For example, a Ghanaian study reported a family with a clinical phenotype highly suggestive of MODY (Schurz *et al.*, 2024). The concentration of variants in the 41–70-year age group observed here, while older than the classical MODY presentation, may reflect diagnostic delays commonly seen in resource-limited settings with restricted access to specialised care.

Importantly, this study also observed that participants carrying exon 1 and exon 5 HNF1A variants tended to have higher fasting blood sugar and HbA1c levels compared to those without these variants. This finding supports earlier reports that HNF1A variants may influence glycaemic control by modulating glucose metabolism (Nowak *et al.*, 2015; McDonald *et al*., 2011). Identifying MODY3 is of particular clinical relevance because affected individuals typically respond well to low-dose sulfonylureas, which act by closing the potassium ATP channel in pancreatic beta cells, a pathway especially effective in HNF1A deficiency (Pearson *et al*., 2003). In contrast, they often respond poorly to metformin and may be spared unnecessary insulin therapy. This highlights the potential of HNF1A genetic screening not only for accurate diagnosis but also for optimising treatment outcomes and improving quality of life.

Taken together, these findings underscore the urgent need for systematic, large-scale genetic screening studies for monogenic diabetes in Nigeria and across Africa. Such efforts will help clarify the true prevalence, mutation spectrum, and clinical characteristics of MODY in African populations, while also strengthening diagnostic accuracy and therapeutic decision-making.

CONCLUSION

This study revealed a high prevalence of HNF1A gene variants among both diabetic and non-diabetic Nigerian participants, with variants in exons 1 and 5 most frequently detected. Although no statistically significant association was found between the distribution of these variants and diabetes status ($p > 0.05$), carriers of HNF1A variants demonstrated trends toward poorer glycaemic control, suggesting a potential modulatory role in glucose metabolism. Notably, variants in exons 2, 3, and 4 were predominantly observed in the diabetic group.

The presence of HNF1A variants, particularly in individuals with normal BMI, provides evidence suggestive of MODY3, a form of diabetes frequently underdiagnosed and misclassified as T2D in Nigeria. Recognising and correctly diagnosing monogenic diabetes carries significant clinical utility, as patients with HNF1A-MODY often respond well to sulfonylurea therapy and may avoid unnecessary insulin treatment. Beyond individualised therapy, these findings also have broader implications for genetic counselling and family health management.

Overall, this work contributes preliminary genetic evidence supporting the presence of monogenic diabetes in Nigeria and highlights the importance of integrating genetic screening into clinical practice. Future research should aim to confirm these findings using post-digestion genotyping and to correlate molecular data with clinical and family histories. This will be essential for refining the understanding of diabetes heterogeneity in African populations and for ensuring more precise and effective patient care.

REFERENCES

Abdoli, N.L., Aukrust, I., Flannick, J., Molnes, J., Burtt, N., Molven, A., and Groop, L. (2016). "Functional Investigations of HNF1A Identify Rare Variants as Risk Factors for Type 2 Diabetes in the General Population." *Diabetes* **66**(2), 335–346.

Bazzazzadehgan, S., Shariat-Madar, Z., & Mahdi, F. (2025). Distinct Roles of Common Genetic Variants and Their Contributions to Diabetes: MODY and Uncontrolled T2DM. *Biomolecules*, *15*(3), 414.

ElSayed, N.A., McCoy, R.G., Aleppo, G., Balapattabi, K., Beverly, E.A., Briggs Early, K., Bruemmer, D., Ebekozien, O., Echouffo-Tcheugui, J.B., Ekhlaspour, L. and Gaglia, J.L. (2025). 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2025. *Diabetes Care*, *48*.

Fotini, K.K. and K. R. Owen (2012). "Maturity Onset Diabetes of the Young: Clinical Characteristics, Diagnosis and Management." *Pediatric endocrinology reviews*: **PER**, **10(2)**, 234–242.

Hamad, N.A. Eltayeb, L.B., Brair, S.L., Bakheit, K.H., Hamdan, H.Z.,and Omer,W.H.(2013). Clinical study of serum calcium, phosphorus, and alkaline phosphates level in type II Diabetes mellitus among Sudanese

population in Khartoum State*, Neelain Medical Journal*; **3**(10), 42-50.

International Diabetes Federation. (2021). *IDF Diabetes Atlas, 10th edn. Brussels, Belgium: International Diabetes Federation*.

J.; Lawry, K.R.; Owen, A.L.; Gloyn, S.; Ellard, and A.T., Hattersley (2011), High-sensitivity CRP discriminates HNF1A-MODY from other subtypes of diabetes. *Diabetes Care*, **34**, 1860–1862.

Jason, F., Johansson, S., and Njølstad, P.R. "*Common and Rare Forms of Diabetes Mellitus: Towards a Continuum of Diabetes Subtypes."* **Nature Reviews Endocrinology** 12(7), 2016, 394–406.

Karol, E., Aukrust,I., L. Bjørkhaug, L.., N. P. Burtt, J. M. Mercader, H. García-Ortiz, A. Huerta-Chagoya. (2014). "Association of a Low-Frequency Variant InHNF1A with Type 2 Diabetes in a Latino Population." *JAMA* **311**(22), 2305.

Kazunori, J. Saruwatari, T. Tanaka, K. Oniki, A. Kajiwara, M. Hiroko, A. Yoshida, H. Jinnouchi, and K. Nakagawa (2017). "Common Variants of HNF1A Gene Are Associated with Diabetic Retinopathy and Poor Glycaemic Control in Normal-Weight Japanese Subjects with Type 2 Diabetes Mellitus." *Journal of Diabetes and its Complications***, 31**(2), 483–488.

Kimiko, M. Natsume, S. Aoki, S. Nakano, T. Inamori, N. Kasezawa, and Goda, T (2012). "The Combined Effect of the T2DM Susceptibility Genes Is an Important Risk Factor for T2DM in Non-Obese Japanese: A Population Based Case-Control Study." *BMC Medical Genetics***, 13**, 1-5.

Kwak, S.H. and Park, K.S. (2016). Recent progress in genetic and epigenetic research on type 2 diabetes. *Exp. Molecular Medical* **48**, e220.

Lall, K., Magi, R., Morris, A., Metspalu, A., and Fischer, K. (2017). Personalized risk prediction for type 2 diabetes: The potential of genetic risk scores. *Genetic Medical* **19**, 322–329.

Malhotra, B.N. Joshi, V., Poojary, S., Middha, S., Gupta, S., and A. B. Olaonipekun (2022), "*Meta-Analysis of HNF1A-MODY3 Variants among Human Population*." *Journal of Diabetes & Metabolic Disorders* 21, 1-6.

McDonald, T.J., B.M.; Shields, B.M.,

Nowak, N., Hohendorff, J., Solecka., I., Szopa, M., Skupien, J., Kiec-Wilk, Mlynarski, B., and Malecki, M.T. (2015). Circulating ghrelin level is higher in HNF1A-MODY and GCK-MODY than in polygenic forms of diabetes mellitus. ***Endocrine* 50**, 643–649.

Ogbera, A. O. and Ekpebegh, C. (2014). Diabetes mellitus in Nigeria: The past, present and future. *World journal of diabetes*, *5*(6), 905.

Olamoyegun, M. A., Alare, K., Afolabi, S. A., Aderinto, N., and Adeyemi, T. (2024). A systematic review and meta-analysis of the prevalence and risk factors of type 2 diabetes mellitus in Nigeria. *Clinical Diabetes and Endocrinology*, *10*(1), 43.

Olatunde, O. I. (2025). Evaluating the challenges and opportunities for diabetes care policy in Nigeria. *Open Health*, *6*(1), 20230056.

Pearson, E. R., Starkey, B. J., Powell, R. J., Gribble, F. M., Clark, P. M., & Hattersley, A. T. (2003). Genetic cause of hyperglycaemia and response to treatment in diabetes. *The Lancet*, 362(9392), 1275-1281.

Prasad, D.K., Sheela, P., Kumar, A.N., N. L., Deedi, N.L. (2018). "*Iron Levels Increased in Serum from Gestational Diabetes Mellitus Mothers in Coastal Area of Andhra Pradesh*" **Journal of Diabetes Metabolism,** 4, 269.

Radha, V., Ek, J., Anuradha, S., Hansen, T., Pedersen, O., and Mohan, V. (2009). Identification of Novel Variants in the Hepatocyte Nuclear Factor-1 Gene in South Indian Patients with Maturity Onset Diabetes of Young. *Journal Clinic Endocrinology Metabolism*, **94(6),** 1959–1965.

Rubio-Cabezas,O. Hattersley, A.T., and Njolstad, P.R. (2019). *The diagnosis and management of monogenic diabetes in children and adolescents*, *Journal of Pediatric Diabetes,* 12, 33-42.

Schurz, H., Naranbhai, V., Yates, T.A., Gilchrist, J.J., Parks, T., Dodd, P.J., Möller, M., Hoal, E.G., Morris, A.P., Hill, A.V. and International Tuberculosis Host Genetics Consortium (2024). Multi-ancestry meta-analysis of host genetic susceptibility to tuberculosis identifies shared genetic architecture. *Elife*, *13*, p.e84394.

Shields, B. M., Shepherd, M., Hudson, M., Pearson, E. R., Hyde, C., Peters, J.& Hattersley, A. T. (2018). Population-based assessment of a biomarker-based screening pathway to aid diagnosis of monogenic diabetes in young-onset patients. *Diabetes Care*, 41(5), 1017-1025.

Sousa, M., Rego, T., and Armas, J. B. (2022). Insights into the genetics and signaling pathways in maturity-onset diabetes of the young. *International journal of molecular sciences*, *23*(21), 12910.

Sugandh, F.N.U., Chandio, M., Raveena, F.N.U., Kumar, L., Karishma, F.N.U., Khuwaja, S., Memon, U.A., Bai, K., Kashif, M., Varrassi, G. and Khatri, M. (2023). Advances in the management of diabetes mellitus: a focus on personalized medicine. *Cureus*, *15*(8).

Tshivhase, A., Matsha, T., and Raghubeer, S. (2021). Diagnosis and treatment of MODY: an updated mini review. *Applied Sciences*, *11*(20), 9436.

Uloko, A.E., Musa, B.M., Ramalan, M.A., Gezawa, I.D., Puepet, F.H., Uloko, A.T., Borodo, M.M. and Sada, K.B. (2018). Prevalence and risk factors for diabetes mellitus in Nigeria: a systematic review and meta-analysis. *Diabetes Therapy*, *9*(3), pp.1307-1316.

Yasutaka, M., Takashi, M., and Yoshihiro, O. (2022). *HNF1A Mutations and Beta Cell Dysfunction in Diabetes*, *International Journal of Molecular Sciences*, **23**, 3222.