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# Clinical profile and risk factors for type-2 diabetes – A cross-sectional study

Peace N. Okoro[1], Kola Olarinoye[1], Arinola Esan[2]

[1]Department of Internal Medicine, University of Ilorin Teaching Hospital, University street, Ilorin, Kwara, [2]Department of Medicine, University College Hospital, Ibadan, Nigeria.

***Corresponding author:**
Dr. Peace N. Okoro, Department of Internal Medicine, University of Ilorin Teaching Hospital, University street, Ilorin, Kwara, Nigeria.

okorop41@gmail.com

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## ABSTRACT

**Objectives:** Type 2 diabetes is a chronic metabolic disorder characterised by elevated blood glucose levels due to insulin resistance or insufficient insulin production. Understanding the prevalence, characteristics, and markers of this disease is essential for effective prevention, management and treatment. The study aims to explore the association between type 2 diabetes and its prevalence, characteristics and markers.

**Material and Methods**: Adults with type 2 diabetes and matched healthy controls were enrolled in the study. Statistical calculations were used to establish the sample size. Information on demographics, physical examinations and lab tests were collected. We assessed plasma glucose, glycated haemoglobin (HbA1c), serum lipids and serum DPP4.

**Results**: The mean age (SD) of the population under research was 55.7 (6.12) years for the study subjects and 55.7 (6.11) years for the controls. A positive family history of diabetes was present in 34 (or 33%) of the diabetes patients compared to 11 (or 11%) of the non-diabetic patients (p 0.001). The mean HbA1c in the diabetic group was substantially greater than that of the non-diabetic controls (5.47 1.89%), as expected (7.23 2.69%), p 0.001. It's interesting to note that total cholesterol was markedly higher in the diabetes participants (5.59 2.24 mmol/L) than in the non-diabetic controls (6.48 1.54 mmol/L), p = 0.001.

**Conclusion**: Due to common risk factors and underlying mechanisms, type 2 diabetes and hypertension may be related, as suggested by the high prevalence of hypertension in the diabetic group. Type 2 diabetes was substantially related to elevated HbA1c and fasting plasma glucose levels, indicating poor glycaemic control. The higher mean serum Dipeptidyl peptidase-4 (DPP4) level in the group with type 2 diabetes shows a link between high DPP4 levels and the disease, which may have an effect on incretin hormone activity, insulin resistance and therapeutic options.

**Keywords:** Type 2 diabetes, HbA1c, Fasting plasma glucose levels, DPP4, Prevalence

## INTRODUCTION

Type 2 diabetes is now a significant public health concern due to its rising incidence around the globe.[1] The necessity for focused treatments and healthcare resources is highlighted by the prevalence study's insights on the size and scope of the issue.[2] In addition, identifying sensitive populations and potential risk factors can be accomplished by looking into the socio-demographic traits of people with type 2 diabetes.[3]

Understanding the course and effects of type 2 diabetes depends heavily on the traits of those who have it.[4] To ascertain whether there are any sex-based differences in the prevalence or susceptibility, gender distribution is frequently investigated.[5] Age is another crucial factor

because it's well known that as people get older, they have a higher risk of acquiring type 2 diabetes.[6] The development and management of type 2 diabetes may also be influenced by additional sociodemographic factors, such as lifestyle choices, family history and socioeconomic position.[7]

For the purposes of diagnosis, observation and therapy, it is essential to identify the indicators connected to type 2 diabetes.[8] The glycated haemoglobin (HbA1c) level, which measures long-term blood glucose control, is used to diagnose diabetes.[9] Higher HbA1c levels are associated with worse glycaemic control and a higher risk of complications.[10] Another sign used to identify type 2 diabetes is fasting plasma glucose (FPG) levels, which are assessed following an overnight fast. Elevated FPG levels are associated with insulin resistance and decreased control of fasting glucose.[11]

Examining additional markers, such as Dipeptidyl peptidase-4 (DPP4) levels, can further shed light on type 2 diabetes' underlying mechanisms. DPP4, an enzyme implicated in the control of incretin hormones and glucose metabolism, may contribute to the pathogenesis of the illness.[12] The development of tailored treatments and interventions can benefit from knowledge of the link between increased DPP4 levels and type 2 diabetes.[13]

This study intends to increase our understanding of the condition, discover potential risk factors, refine diagnostic procedures and promote more efficient management techniques by examining the prevalence, traits and indicators connected with type 2 diabetes. This information may ultimately help to lessen the burden of type 2 diabetes and enhance the general health and well-being of those who are affected.

## MATERIAL AND METHODS

A cross-sectional study was conducted at the University of Ilorin Teaching Hospital in Ilorin, Kwara State, Nigeria. The study involved the diabetes clinic, medical outpatient department and medical wards of the University of Ilorin Teaching Hospital (UITH). Ilorin, the state capital of Kwara State, Nigeria, houses the 650-bed tertiary health facility known as UITH. The hospital serves a diverse population, primarily composed of Yoruba, Fulani, Hausa, Baruba and Igbo individuals. The diabetic clinic, managed by three consultant endocrinologists and three resident doctors, attends to 40 Type 2 Diabetes Mellitus (T2DM) patients weekly from Kwara state and neighbouring states such as Osun, Oyo, Ekiti, Kogi and Niger.

Adults with T2DM visiting the UITH, Ilorin Diabetes Clinic, were included in the study, along with age- and sex-matched healthy controls sourced from hospital staff members or patients' relatives. This resulted in two distinct groups: individuals with diabetes and those without. The diagnostic criteria for diabetes followed the WHO guidelines (2020). Individuals were considered diabetic if they exhibited hyperglycaemic symptoms with a random plasma glucose level exceeding 11.1 mmol/L or 200 mg/dl, or a FPG level above 7 mmol/L on multiple occasions after 8 hours of fasting. In addition, individuals with T2DM under insulin or glucose-lowering medications for at least 3 months were included. Patients with T2DM visiting the Diabetes Clinic were enrolled, and age- and sex-matched healthy controls were recruited from hospital staff through convenient sampling.

### Inclusion and exclusion criteria

The inclusion and exclusion criteria were carefully established to ensure the study's relevance and the safety of participants. Inclusion criteria for the diabetes group encompassed adults aged 18–65 diagnosed with T2DM who willingly consented to participate. On the other hand, the non-diabetes group included hospital employees and non-diabetic family members aged 18–65, with FPG within the normal range (3.9–5.6 mmol/L), and who provided informed consent.

Exclusion criteria for the diabetes group aimed at excluding patients with complications such as bilateral amputations, trauma to arterial vasculature, diabetic pregnant patients, Takayasu patients with peripheral arterial disease (PAD) from a different aetiology and those who had undergone arterial graft surgeries. Similarly, exclusion criteria for the non-diabetes group aimed at excluding individuals with FPG levels indicating pre-diabetes (5.6–6.9 mmol/L) and diabetes (7 mmol/L), those below 18 or above 65 years of age and those who declined consent.

These criteria were thoughtfully designed to ensure homogeneity within the groups and minimise confounding factors.

### Sample size calculation

The sample size calculation followed Fisher's statistical formula and the reference article by Goyal *et al*., (2020)[14] in Oyo state (prevalence of diabetes: 4.6%) was used as the basis. The precision used for the calculation was in line with standard statistical practices to ensure the reliability of the results.

### Data analysis

IBM USA, Amonk, NY 10504 provided the social science statistical program IBM SPSS StatisticsR 2012 version 21.0 for Windows for the analysis of the data. Using frequency, percentages and proportions, the cases' clinical and demographic data were gathered.

Means ± standard deviation was used to express normally distributed continuous variables such as measured DPP4

levels and FPG. For variables that weren't distributed normally, the median and interquartile ranges were used. Fisher's exact formula was used to compare categorical variable proportions, while the student's t-test was used to evaluate continuous variable averages. The standard for comparison was the constant value of P.

### Ethical consideration

Ethical approval was obtained from the Ethics and Research Committee of the Faculty of Basic Clinical Sciences University of Ilorin Teaching Hospital with approval number: COHS/FCSERC/2022/04/007.

## RESULTS

### Sociodemographic characteristics of participants

Table 1 displays the socio-demographic details of the participants and the controls. There were 79 men and 121 women in all, or 60.5% of the group. Both the diabetes group and the non-diabetic group had the same numbers, that is, 39 males and 61 females. The mean age (SD) of the population under research was 55.7 (6.12) years for the study subjects and 55.7 (6.11) years for the controls. The majority of participants in both groups (subjects 29% and controls 40%) were people with tertiary education. In the T2DM group, 86 (86%) of the individuals were married, 11 (11%) were widowed, 2 (2%) were single and 1 (1%) was divorced, whereas in the control group, 85 (85%) of the individuals were married, 7 (7%) were widowed, 6 (6%) were single and 2 (2%) were divorced. Yoruba people made up the majority of the study's participants in both the subject and control groups (95% and 93%, respectively).

**Table 1**: Sociodemographic characteristics of study subjects and controls.

| Socio-demographic characteristics | Diabetic subjects n=100 (%) | Non-diabetic controls n=100 (%) | $\chi^2$ | p-value |
|---|---|---|---|---|
| **Mean Age (years) ± SD** | 55.72 ± 6.12* | 55.7 ± 6.11* | 0.023¶ | 0.962 |
| **Age group (years)** | | | 0.104 | 1 |
| 45–49 | 22 (22) | 22 (22) | | |
| 50–54 | 19 (19) | 19 (19) | | |
| 55–59 | 20 (20) | 20 (20) | | |
| 60–64 | 35 (35) | 35 (35) | | |
| 65–69 | 4 (4) | 4 (4) | | |
| **Sex** | | | 0.021 | 0.885 |
| Male | 39 (39) | 39 (39) | | |
| Female | 61 (61) | 61 (61) | | |
| **Education** | | | 9.083 | 0.058 |
| None | 18 (18) | 17 (17) | | |
| Primary | 24 (24) | 9 (9) | | |
| Secondary | 21 (21) | 25 (25) | | |
| Tertiary | 29 (29) | 40 (40) | | |
| Postgraduate | 8 (8) | 9 (9) | | |
| **Marital status** | | | 3.188 | 0.375 |
| Single | 2 (2) | 6 (6) | | |
| Married | 86 (86) | 85 (85) | | |
| Divorced | 1 (1) | 2 (2) | | |
| Widowed | 11 (11) | 7 (7) | | |
| **Occupation** | | | 11.524 | 0.06 |
| Trader | 49 (49) | 41 (41) | | |
| Civil servant | 35 (35) | 46 (46) | | |
| Caterer | 1 (1) | 0 | | |
| Retiree | 7 (7) | 1 (1) | | |
| Unemployed | 1 (1) | 0 | | |
| Farmer | 4 (4) | 9 (9) | | |
| Driver | 1 (1) | 0 | | |
| Clergy | 2 (2) | 3 (3) | | |
| **Ethnicity** | | | 1.876 | 0.705 |
| Yoruba | 95 (95) | 93 (93) | | |
| Hausa | 1 (1) | 2 (2) | | |
| Igbo | 1 (1) | 0 | | |
| Others | 3 (3) | 5 (5) | | |
| **Religion** | | | 3.596 | 0.199 |
| Islam | 48 (48) | 38 (38) | | |
| Christianity | 52 (52) | 60 (60) | | |
| Traditional | 0 | 1 (1) | | |
| Others | 0 | 1 (1) | | |

The statistical test of significance was done using Fisher's exact test.
¶ Statistical test of significance was done using the independent t-test.
* Represents mean ± standard deviation (SD)

### Clinical characteristics of participants

The clinical features of the research population were displayed in Table 2. In the diabetes group (66%) compared to the controls (37%), there was a statistically significant difference in the history of hypertension (HTN) (P = 0.001). A positive family history of diabetes was present in 34 (or 33%) of the diabetes patients compared to 11 (or 11%) of the non-diabetic patients (p = 0.001). The diabetes group's median diabetes mellitus (DM) duration was 4.5 (2–8) years. There were 21 (21%) people in the diabetes group who had a history of diabetic foot ulceration (DMFU), but none of the controls did (p = 0.001).

### Spectrum of drug prescription in the participants

The range of individuals' pharmacological prescriptions was displayed in Table 3. Antihypertensive medications were taken by 63 (63%) participants in the diabetes group and 34 (34%) participants in the non-diabetic group (p 0.001). A total of 49 (49%) people in the diabetes group were taking statins, compared to 2 (2%) in the non-diabetic group (p = 0.001). In the diabetic group, metformin was taken by

**Table 2**: Clinical characteristics of the study population.

| Clinical characteristics of respondents | Diabetic subjects n=100 (%) | Non-diabetic controls n=100 (%) | $\chi^2$ | p-value |
|---|---|---|---|---|
| **History of HTN** | | | 16.835 | <0.001** |
| Present | 66 (66) | 37 (37) | | |
| Absent | 34 (34) | 63 (63) | | |
| *Median duration of HTN (yrs)** | *4 (0–7)* | *5 (1.5–6)* | 15.241 | <0.001** |
| **Median duration of DM (yrs)*** | 4.5 (2–8) * | 0 | | |
| **Family history of DM** | | | 16.710 | <0.001** |
| Present | 34 (34) | 11 (11) | | |
| Absent | 66 (66) | 89 (89) | | |
| **History of stroke** | | | 5.128 | 0.059 |
| Present | 5 (5) | 0 | | |
| Absent | 95 (95) | 100 (100) | | |
| **History of myocardial infarction** | | | 1.005 | 1 |
| Present | 1 (1) | 0 | | |
| Absent | 99 (99) | 100 (100) | | |
| **History of amputation** | | | 1.005 | 1 |
| Present | 1 (1) | 0 | | |
| Absent | 99 (99) | 100 (100) | | |
| **History of cigarette smoking** | | | 0.205 | 1 |
| Present | 2 (2) | 3 (3) | | |
| Absent | 98 (98) | 97 (97) | | |
| *Median duration of smoking (yrs)** | *4 (0–4)* | *5 (3–5)* | | |
| **History of alcohol intake** | | | 1.047 | 0.498 |
| Present | 6 (6) | 3 (3) | | |
| Absent | 94 (94) | 97 (97) | | |
| *Median quantity of alcohol consumed (gram/week)** | *120 (105–135)* | *240 (120–240)* | | |
| **History of exercise** | | | 1.229 | 0.342 |
| Present | 24 (24) | 31 (31) | | |
| Absent | 76 (76) | 69 (69) | | |
| *Median number of exercise days per week** | *3 (2-3)* | *2 (1-3)* | | |
| **History of intermittent claudicating** | | | 4.031 | 0.082 |
| Present | 10 (10) | 3 (3) | | |
| Absent | 90 (90) | 97 (97) | | |
| **History of DMFU** | | | 23.464 | <0.001** |
| Present | 21 (21) | 0 | | |
| Absent | 79 (79) | 100 (100) | | |

The statistical test of significance was done using Fisher's exact test.
**Represents significant p-value.
*Represents median Interquartile range (IQR). The respective continuous variables were not uniformly distributed and they thus are summarised in **median (IQR)** instead of using **mean ± SD** (Standard deviation).
HTN: Hypertension; DMFU: Diabetic foot ulcer; DM: Diabetes mellitus.

**Table 3**: Spectrum of drug prescription in the respondents.

| Spectrum of drug prescription in the respondents | Diabetic subjects n=100 (%) | Non-diabetic controls n=100 (%) | $\chi^2$ | p-value |
|---|---|---|---|---|
| Antihypertensive | 63 (63) | 34 (34) | 16.835 | <0.001** |
| Statins | 49 (49) | 2 (2) | 58.139 | <0.001** |
| Metformin | 91 (91) | | | |
| Sulphonylureas | 49 (49) | | | |
| DPP4 inhibitors | 16 (16) | | | |
| Alpha glucosidase | 5 (5) | | | |
| Sodium-glucose co-transporter-2 (SGLT2) | 1 (1) | | | |
| Premixed insulin | 3 (3) | | | |
| Insulin glargine | 25 (25) | | | |

The statistical test of significance was done using Fisher's exact test.
**Represents significant p-value.
DPP4: Dipeptidyl peptidase-4

91 (91%) individuals, sulphonylureas by 49 (49%) people, insulin glargine by 25 (25%) participants, DPP4 inhibitors by 16 (16%) participants, glucosidase inhibitors by 5 (5%), premixed insulin by 3% participants and Sodium-glucose co-transporter-2 (SGLT2) inhibitors by 1% participants.

### Laboratory parameters in the participants

The laboratory parameters for the subjects are shown in Table 4. The mean HbA1c in the diabetic group was substantially greater than that of the non-diabetic controls (5.47 1.89%), as expected (7.23 2.69%), p 0.001. It's interesting to note that total cholesterol was markedly higher in the diabetes participants (5.59 2.24 mmol/L) than in the non-diabetic controls (6.48 1.54 mmol/L), p = 0.001. Though the High Density Lipoprotein (HDL) was greater in the diabetes group (1.97 1.87 mmol/L) than in the non-diabetic controls (1.88 1.25 mmo/L) (p = 0.687), it was also higher in the controls overall. In addition, the non-diabetic group's mean low density lipoprotein (LDL) was considerably higher (2.96 1.1 mmol/L) than that of the diabetic group (2.37 1.57 mmol/L). As expected, the mean FPG was substantially greater in the diabetes group (6.07 1.27mmol/L) than in the non-diabetic participants (4.86 0.90mmol/L) (p = 0.001).

### Serum DPP4 levels of participants

The subjects' mean serum DPP4 levels are displayed in Table 4. With a p-value of 0.01, the mean serum DPP4 level in the diabetes group was considerably higher (31.71 7.09 ng/mL) than in the non-diabetic control subjects (22.78 7.63 ng/mL).

### Serum DPP4 levels and glucose control among participants

Table 4 demonstrates that, with a p-value of less than 0.05, the mean serum DPP4 level in the diabetes group was substantially higher than that in the non-diabetic control subjects (31.71 7.09 ng/mL vs. 22.78 7.63 ng/mL). The mean HbA1c in the diabetic group was substantially greater than that of the non-diabetic controls (5.47 1.89%), as expected (7.23 2.69%), p 0.001. As expected, the mean FPG was substantially greater in the diabetes group (6.07 1.27mmol/L) than in the non-diabetic participants (4.86 0.90mmol/L) (p = 0.001).

## DISCUSSION

The findings give a quick glimpse of the socio-demographic details of the cases and controls as well as the prevalence of diabetes. Both the diabetes and non-diabetic groups had roughly similar representations of males and females in terms of gender, indicating that gender may not be a key determinant of diabetes prevalence. The fact that the mean age was the same in both groups suggests that, in this particular study, age may not be a significant factor in the occurrence of diabetes. Additional investigation is needed to comprehend the effects of these sociodemographic factors on the occurrence of type 2 diabetes. In addition to the sociodemographic information, other aspects must be taken into account[15,16] such as dietary habits, levels of physical activity, family history of diabetes, socioeconomic position and other potential risk factors.

In comparison to the controls (37%), the diabetic group (66%) showed a significantly greater prevalence of hypertension. The high prevalence of hypertension in diabetics raises the possibility that the two disorders are related.[17] People with hypertension may acquire type 2 diabetes.[18] Type 2 diabetes and high blood pressure typically co-occur and have similar risk factors and underlying biological mechanisms.[19]

The shared underlying processes that underlie insulin resistance[20] obesity, inflammation,[21] endothelial dysfunction[22] and common genetic and lifestyle factors [23] have been linked to the relationship between type 2 diabetes

**Table 4**: Laboratory parameters and serum DPP4 levels in participants, with a focus on glucose control.

| Laboratory parameters in the respondents | Diabetic subjects n=100 (mean ± SD) | Non-diabetic controls n=100 (mean ± SD) | T | p-value |
|---|---|---|---|---|
| Glycated haemoglobin (HbA1c) (%) | 7.23 ± 2.69 | 5.47 ± 1.89 | 5.367 | <0.001** |
| Total cholesterol (mmol/L) | 5.59 ± 2.24 | 6.48 ± 1.54 | –3.265 | 0.001** |
| High Density Lipoprotein (HDL) (mmo/L) | 1.97 ± 1.87 | 1.88 ± 1.25 | 0.404 | 0.687 |
| Low Density Lipoprotein (LDL) (mmol/L) | 2.37 ± 1.57 | 2.96 ± 1.1 | –3.123 | 0.002** |
| Triglyceride (TG) (mmol/L) | 1.62 ± 1.86 | 1.62 ± 0.47 | –0.042 | 0.967 |
| Fasting Blood Sugar (FBS) (mmol/L) | 6.07 ± 1.27 | 4.86 ± 0.90 | 7.819 | <0.001** |
| Serum DPP4 levels (ng/mL) | 31.71 ± 7.09 | 22.78 ± 7.63 | 4.425 | <0.001** |
| HbA1c (%) | 7.23 ± 2.69 | 5.47 ± 1.89 | 5.367 | <0.001** |
| DPP4 levels (ng/mL) | 31.38 ± 11.89 | 24.71 ± 9.26 | 4.425 | <0.001** |
| Fasting Plasma Glucose (FPG) (mmol/l) | 6.08 ± 1.27 | 4.86 ± 0.90 | 7.819 | <0.001** |

The statistical test of significance was done using the independent sample t-test.
**Represents significant p-value (P-Value < 0.001), DPP4: Dipeptidyl peptidase-4, SD: Standard deviation.

and hypertension. As their coexistence can increase the risk of cardiovascular problems and other negative health outcomes, it is crucial to manage both illnesses thoroughly.

Compared to the controls, 21% of participants in the diabetes group had a history of DMFU. A major side effect of diabetes is diabetic foot ulcers frequently develop as a result of impaired blood flow and nerve damage.[24] This clinical condition's association with type 2 diabetes is further supported by the diabetes group's increased prevalence of DMFU. Peripheral neuropathy[25] peripheral vascular disease,[26] decreased immunological response[27] and the combined effects of these variables [28] are the main causes of the relationship between DMFU and type 2 diabetes. For those with diabetes, good foot hygiene, regular monitoring and early detection of foot issues are critical to preventing the development of foot ulcers and minimising the difficulties that come with them.[29]

The capacity of HbA1c (glycated haemoglobin) levels to represent long-term blood glucose control makes them related with type 2 diabetes.[30] An assessment of the average blood glucose levels over the previous two to three months is provided by HbA1c.[31] It displays how much glucose has bonded to red blood cell haemoglobin molecules. Red blood cells normally only have a lifespan of 120 days, and the HbA1c test measures the average blood glucose levels throughout that time. Poorer glycaemic control is indicated by higher HbA1c values, which show that blood glucose levels have been gradually increasing over time.[32] Consequently, increased HbA1c values are a sign of diabetes. HbA1c is now accepted as a diagnostic indicator for diabetes by the World Health Organization (WHO) and the American Diabetes Association (ADA).[19] At a HbA1c result of 6.5% or higher, the ADA deems diabetes to be diagnosable.[33]

As a result, the relationship between elevated HbA1c levels and type 2 diabetes is supported by the considerably higher HbA1c values in the diabetic group (7.23 2.69%) compared to the non-diabetic controls (5.47 1.89%). It's vital to remember that variables other than diabetes, such as certain medical illnesses or haemoglobin variations, may affect HbA1c values.[34] The much higher HbA1c levels in the diabetic group compared to the non-diabetic controls, however, demonstrate its relationship with type 2 diabetes and its usefulness as a marker of glycaemic control in diabetic persons in the context of the information supplied.

Because FPG levels are used to measure and identify high blood glucose levels, they are linked to type 2 diabetes.[35] Blood glucose levels are frequently assessed using FPG after an overnight fast.[36] It provides information on the blood glucose level at a specific time, typically in the morning, before any food or liquid is consumed.[37] FPG levels are a reflection of the body's ability to regulate blood sugar levels in the absence of a recent meal.[38] FPG readings are also widely used as a type 2 diabetes diagnostic reference.[39] Diagnosing diabetes is advised by the ADA when the FPG is 126 mg/dL (7.0 mmol/L) or above on two separate occasions.

The significantly higher FPG levels in the diabetic group (6.07 1.27 mmol/L) compared to the non-diabetic subjects (4.86 0.90 mmol/L) confirm the association between elevated FPG levels and type 2 diabetes. High blood glucose levels occur in type 2 diabetes because the body either generates insufficient insulin or becomes resistant to its effects.[40] Fasting hyperglycaemia, characterised by elevated FPG readings, is one feature that sets type 2 diabetes apart. The elevated FPG levels in the diabetic population are indicative of insufficient fasting glucose management and may potentially be a sign of impaired insulin sensitivity or insufficient insulin production.[41]

The study found that the mean serum DPP4 (Dipeptidyl Peptidase-4) level in the diabetes group was significantly higher than that of the control group. DPP4 is an enzyme that controls glucose metabolism as well as the incretin hormones' glucagon-like peptide-1 (GLP-1) and gastric inhibitory polypeptide (GIP) [42]. These hormones regulate blood sugar levels, insulin secretion and feelings of fullness. DPP4 breaks down these hormones in order to lessen their bioactivity.[43].

The significantly higher mean serum DPP4 levels in the diabetic group (31.71 7.09 ng/mL) compared to the non-diabetic control participants (22.78 7.63 ng/mL) show a link between elevated DPP4 levels and type 2 diabetes. This outcome is in line with past research that showed higher DPP4 levels in type 2 diabetics.[44]

The greater DPP4 levels in the group of people with diabetes may mean numerous things: (a) Elevated DPP4 levels can accelerate GLP-1 and GIP breakdown, decreasing its bioactivity.[45] These incretin hormones promote glucose regulation by increasing insulin production from pancreatic beta cells, inhibiting glucagon release and delaying gastric emptying.[46] Elevated DPP4 levels may cause incretin hormone activity to decline, making it more difficult for the body to control blood glucose levels.[47] DPP4 has been linked to insulin resistance, a crucial aspect of type 2 diabetes, in (b). When the body's cells lose their receptivity to the effects of insulin, it results in insulin resistance, which raises blood sugar levels. Increased DPP4 levels may cause insulin resistance through as-yet-unidentified mechanisms [48,49], and (c) the link between increased DPP4 levels and type 2 diabetes points to DPP4 as a possible therapeutic target. Type 2 diabetes is treated with DPP4 inhibitors, also referred to as gliptins or DPP4 inhibitors.[50] These drugs improve glucose regulation by inhibiting DPP4 and increasing the bioavailability of incretin hormones.[51]

The specific processes behind the link between elevated DPP4 levels and type 2 diabetes must be further studied, as it is crucial to emphasise. DPP4 levels may also be impacted by individual differences and other factors.[52] The considerable difference in mean serum DPP4 levels between the diabetic group and the non-diabetic control participants, however, raises the possibility that DPP4 plays a component in the pathogenesis of type 2 diabetes.

## CONCLUSION

This study reveals socio-demographic traits and diabetes prevalence, with age and gender showing no significant variations. The correlation between type 2 diabetes and hypertension is suggested by a higher prevalence of hypertension and diabetic foot ulcers in the diabetic group. Elevated HbA1c, FPG and serum DPP4 levels further highlight links and potential impacts on therapeutic options.

### Ethical approval

Ethical Approval was obtained from University of Ilorin Teaching Hospital Ethics Review Committee with approval number NHREC/02/05/2010.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

### Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

## REFERENCES

1. International Diabetes Federation. IDF Diabetes Atlas, 9th Edition. [Internet]. 2019 [cited 2023 Jan 30]. Available from: https://www.diabetesatlas.org
2. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27:1047–53.
3. Agardh EE, Ahlbom A, Andersson T, Efendic S, Grill V, Hallqvist J, *et al*. Socioeconomic position at three points in life in association with type 2 diabetes and impaired glucose tolerance in middle-aged Swedish men and women. Int J Epidemiol 2011;40:804–12.
4. Schöttker B, Rathmann W, Herder C. Heterogeneity in Type 2 Diabetes: Predisposition, Complications, and Mortality. Diabetes Spectrum 2013;26:235–41.
5. Bae JP, Lage MJ, Mo D. Gender Differences in Type 2 Diabetes Mellitus and Factors Related to Patient Delays in Seeking Care. Gender Medicine 2017;14:115–27.
6. Murea M, Ma L, Freedman BI, Parks JS. Diabetes Associated with a Novel Mutation in the KCNJ11 Gene: Extending the Clinical Spectrum of Mutations to Transient Neonatal Diabetes and Beyond. Diabetes. 2018 Jun;67(Supplement 1):221-OR.
7. Tancredi M, Rosengren A, Svensson AM, Kosiborod M, Pivodic A, Gudbjörnsdottir S, *et al*. Excess mortality among persons with type 2 diabetes. N Engl J Med 2015;373:1720–32.
8. American Diabetes Association. Standards of Medical Care in Diabetes—2020 Abridged for Primary Care Providers. Clin Diabetes 2020;38:10–38.
9. American Diabetes Association. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2019. Diabetes Care 2019;42:S13–S28.
10. Gerstein HC, Pogue J, Mann JF, Lonn E, Dagenais GR, McQueen M, *et al*. The relationship between dysglycaemia and cardiovascular and renal risk in diabetic and non-diabetic participants in the HOPE study: a prospective epidemiological analysis. Diabetologia 2005;48:1749–55.
11. American Diabetes Association. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes—2019. Diabetes Care 2019;42:S13–S28.
12. Drucker DJ. Enhancing incretin action for the treatment of type 2 diabetes. Diabetes Care 2006;29:1289–301.
13. Aroda VR, Edelstein SL, Goldberg RB, Knowler WC, Marcovina SM, Orchard TJ, *et al*. Long-term metformin use and vitamin B12 deficiency in the Diabetes Prevention Program Outcomes Study. J Clin Endocrinol Metab 2016;101:1754–61.
14. Goyal A, Gupta Y, Singla R, Kalra S, Tandon N. American diabetes association "standards of medical care—2020 for gestational diabetes mellitus": a critical Appraisal. Diabetes Therapy 2020;11:1639–44.
15. World Health Organization. Global report on diabetes. [Internet]. 2016 [cited 2023 Jan 30]. Available from: https://www.who.int/diabetes/global-report/en/
16. Centers for Disease Control and Prevention. National diabetes statistics report, 2020. [Internet]. 2021 [cited 2023 Jan 30]. Available from: https://www.cdc.gov/diabetes/pdfs/data/statistics/national-diabetes-statistics-report.pdf
17. Lee CM, Huxley RR, Wildman RP, Woodward M. Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: A meta-analysis. J Clin Epidemiol 2008;61:646–53.
18. Reaven GM. Relationships among insulin resistance, type 2 diabetes, essential hypertension, and cardiovascular disease: similarities and differences. J Clin Hypertens 2011;13:238–43.
19. de Boer IH, Bangalore S, Benetos A, Davis AM, Michos ED, Muntner P, *et al*. Diabetes and hypertension: a position statement by the American Diabetes Association. Diabetes Care 2017;40:1273–84.

20. Samuel VT, Shulman GI. Mechanisms for insulin resistance: common threads and missing links. Cell 2016;165:855–70.
21. Hotamisligil GS. Inflammation and metabolic disorders. Nature 2006;444:860–7.
22. Williams B, Lacy PS, Thom SM. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: principal results of the Conduit Artery Function Evaluation (CAFE) study. Circulation 2006;113:1213–25.
23. Ma RC, Chan JC. Type 2 diabetes in East Asians: similarities and differences with populations in Europe and the United States. Ann N Y Acad Sci 2013;1281:64–91.
24. Singh N, Armstrong DG, Lipsky BA. Preventing foot ulcers in patients with diabetes. JAMA 2005;293:217–28.
25. Boulton AJ, Vinik AI, Arezzo JC, Bril V, Feldman EL, Freeman R, *et al*. Diabetic neuropathies: a statement by the American Diabetes Association. Diabetes Care 2005;28:956–62.
26. Hirsch AT, Haskal ZJ, Hertzer NR, Bakal CW, Creager MA, Halperin JL, *et al*. ACC/AHA 2005 guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): executive summary a collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease). Circulation 2006;113:1474–547.
27. Pickup JC, Crook MA. Is type II diabetes mellitus a disease of the innate immune system?. Diabetologia 1998;41:1241–8.
28. Petrofsky JS, Bains GS, Prowse M. Reduced skin blood flow in type 2 diabetes mellitus. Arch Dermatol 2004;140:948–50.
29. Armstrong DG, Boulton AJ, Bus SA. Diabetic foot ulcers and their recurrence. N Engl J Med 2017;376:2367–75.
30. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33:S62–S69.
31. Nathan DM, Kuenen J. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008;31:1473–8.
32. Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA, *et al*. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. Bmj 2000;321:405–12.
33. World Health Organization. Use of glycated haemoglobin (HbA1c) in the diagnosis of diabetes mellitus: abbreviated report of a WHO consultation. [Internet]. 2011 [cited 2023 Jan 30]. Available from: https://apps.who.int/iris/bitstream/handle/10665/70523/WHO_NMH_CHP_CPM_11.1_eng.pdf
34. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33:S62–S69.
35. Cohen RM, Franco RS. Hemoglobin A1c: metabolism, pathophysiology, and clinical use. Curr Diabetes Rep 2012;12:342–6.
36. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33:S62–S69.
37. Foster DW. Diabetes mellitus. In: The Merck Manual of Diagnosis and Therapy. 17th ed. Merck Research Laboratories; 2004.
38. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, *et al*. Follow-up report on the diagnosis of diabetes mellitus. Diabetes Care 2003;26:3160–7.
39. Rodbard HW, Blonde L, Braithwaite SS, Brett EM, Cobin RH, Handelsman Y, *et al*. American Association of Clinical Endocrinologists medical guidelines for clinical practice for the management of diabetes mellitus. Endocr Pract 2009;15:1–116.
40. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care 2010;33:S62–S69.
41. DeFronzo RA, Abdul-Ghani M. Assessment and treatment of cardiovascular risk in prediabetes: impaired glucose tolerance and impaired fasting glucose. Am J Cardiol 2011;108:3B–24B.
42. American Diabetes Association. Standards of medical care in diabetes—2014. Diabetes Care 2014;37:S14–S80.
43. Ahren B. DPP-4 inhibitors. Best Pract Res Clin Endocrinol Metab 2007;21:517–33.
44. Deacon CF, Holst JJ. Dipeptidyl peptidase-4 inhibitors in the treatment of type 2 diabetes: a comparative review. Diabetes Obes Metab 2005;7:671–86.
45. Mentlein R, Gallwitz B, Schmidt WE. Dipeptidyl-peptidase IV hydrolyses gastric inhibitory polypeptide, glucagon-like peptide-1 (7-36) amide, peptide histidine methionine and is responsible for their degradation in human serum. Eur J Biochem 1993;214:829–35.
46. Ahren B. Incretin dysfunction in type 2 diabetes: Clinical impact and future perspectives. Diabetes Metab Res Rev 2004;20:S114–S118.
47. Drucker DJ. Enhancing incretin action for the treatment of type 2 diabetes. Diabetes Care 2006;29:1356–73.
48. Nauck MA, Meier J, Cavender MA. DPP-4 inhibitors in the treatment of type 2 diabetes. In: Incretin-Based Therapies. Springer; 2018. p. 199–227.
49. Kim D, MacConell L. Dipeptidyl peptidase-4 inhibitors in type 2 diabetes therapy—focus on alogliptin. Diabetes Metab Syndr Obes 2015;8:419–34.
50. Drucker DJ. The biology of incretin hormones. Cell Metab 2007;3:153–65.
51. Herman GA, Bergman A, Stevens C, Kotey P, Yi B, Zhao P, *et al*. Effect of single oral doses of sitagliptin, a dipeptidyl peptidase-4 inhibitor, on incretin and plasma glucose levels after an oral glucose tolerance test in patients with type 2 diabetes. J Clin Endocrinol Metab 2006;91:4612–9.
52. Nauck MA, Meininger G, Sheng D, Terranella L, Stein PP. Efficacy and safety of the dipeptidyl peptidase-4 inhibitor, sitagliptin, compared with the sulfonylurea, glipizide, in patients with type 2 diabetes inadequately controlled on metformin alone: a randomized, double-blind, non-inferiority trial. Diabetes Obes Metab 2007;9:194–205.


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