



## RESEARCH ARTICLE

## Determinants of Glucose Imbalance and its Effects on Developing Undiagnosed Type 2 Diabetes Mellitus in an Urban and Rural Ghanaian Community

Shani Issah<sup>1\*</sup> , Obed Akwaa Harrison<sup>1</sup>, Angelina O Danquah<sup>2</sup>, Kamal Sumani Mumuni<sup>3</sup>, Iddrisu Salifu<sup>1</sup>, Mutala Abdulai<sup>4</sup> and Matilda Steiner Aseidu<sup>1</sup>

<sup>1</sup>Department of Nutrition and Food Science, University of Ghana, Ghana

<sup>2</sup>Department of Family and Consumer Sciences, University of Ghana, Ghana

<sup>3</sup>School of Public Health, University of Allied and Health Sciences, Ghana

<sup>4</sup>Family Health Division, Ghana Health Service, Ghana

\*Corresponding author: Shani Issah, Department of Nutrition and Food Science, University of Ghana, Ghana



### Abstract

**Background and Objectives:** Glucose imbalance or impaired fasting glycemia refers to a condition where there is imbalance in blood sugar levels in an individual. This is usually influenced by the inability of the kidneys to properly convert excess sugar into a form that can easily be eliminated from the body. Accumulation of excess sugar in the body leads to the occurrence of Type 2 diabetes mellitus with its associated complications. In this study, we determined the prevalence of impaired fasting glycaemia (IFG), factors associated with its occurrence and its effects on developing undiagnosed Type 2 diabetes mellitus (UT2DM) (Hyperglycaemia) in an urban and rural Ghanaian community.

**Methods:** To carry out this study. A period of four months, from January to May, 2018 was used to collect both socio-demographic and biochemical data. The sampling of communities for this study was guided by enumeration principles of the Ghana Statistical Service. Random sampling technique was used to select households and individual participants in selected communities to form the study population.

**Results:** Mean age of study participants was  $34.4 \pm 10.6$  years. Male participants had a lower mean age,  $32.0 \pm 10.4$  years than that of female participants,  $36.4 \pm 10.3$  years with a significant difference between their mean ages ( $p < 0.01$ ). The mean fasting blood glucose of study participants was  $4.92 \pm 1.19$  mmol/L. Males had a mean fasting blood glucose of  $4.72 \pm 0.89$  mmol/L, which was lower than a mean fasting blood glucose of  $5.08 \pm 1.36$  mmol/L among

females with a significant difference between the two means ( $p < 0.01$ ).

Our study found an impaired fasting blood glucose (Glucose imbalance in blood) prevalence of 26.7% in our study participants. This consisted of 9.0% of hypoglycaemia (FBG  $< 3.9$  mmol/L), 13.0% of pre-diabetes (FBG from 5.6-6.9 mmol/L) and 4.7% of hyperglycaemia (FBG  $> 7$  mmol/L). Pre-diabetes was more common in females, 14.5% than in males, 11.2%. IFG was more prevalent in participants with positive history of pre-diabetes 87.5%. A total mean cholesterol of  $4.62 \pm 1.43$  mmol/L was observed among study participants. This was high among females  $4.80 \pm 1.45$  mmol/L than in males  $4.41 \pm 1.38$  mmol/L. Female participants were 6.2 times more likely to have IFG compared to their male counterparts; OR = 6.2; CI = (1.02-1.80);  $P = 0.03$ . Obese people were 5.5 times more likely to develop IFG than people with normal weight; OR = 5.5; CI = (1.17-2.76);  $P < 0.01$ . Also, residents who had stayed in the metropolis beyond 20 years had 2.5 times risk of having IFG and an increased risk of developing UT2DM; OR = 2.5, CI (1.02-2.43);  $P = 0.04$ .

**Conclusion:** IFG was more prevalent among females and urban residents. Predisposing factors such as family history of diabetes, age, dietary behaviours and residential status predicted IFG. Lifestyle changes that will promote healthy dietary behaviours among females and urban residents is needed in order to reduce the risk of developing IFG and UT2DM respectively.

## Keywords

Tamale Metropolis and Hyperglycemia

## Introduction

Impaired fasting blood glucose (IFG) has been identified as a major predictor of Type 2 diabetes mellitus which continuous to be one of the World's most difficult chronic disease to manage. This is because, it is usually characterized by factors that forms part of an individual's routine daily activities, mostly undesirable dietary practices [1]. Recent reports from the World Health Organization, shows an estimated 422 million people living with Type 2 diabetes mellitus globally and an estimated 1.9 million people lost their lives in 2020 as a result of Type 2 diabetes mellitus usually precipitated by IFG [2].

Existing literature suggest that genetic factors predisposes an individual to impaired fasting blood glucose (IFG) [1]. However, recent studies points to the fact that the major causes of IFG are mostly dietary practices which leads to abnormal accumulation of glucose in blood and subsequent clinical manifestation of Type 2 diabetes mellitus [3]. Appropriate dietary practices are shown to reduce the occurrence of IFG and hence mitigate the onset of Type 2 diabetes mellitus [4]. As also indicated by [5], healthy dietary practices significantly reduces IFG in exposed individuals and by extension reduces the onset and complications of Type 2 diabetes mellitus. Based on this, the International diabetes federation (IDF) and the American diabetes association (ADA) have recommended appropriate dietary practices as the first steps towards minimizing exposures and occurrence of IFG, and providing a healthy living for people who might have been affected by Type 2 diabetes mellitus [6] [7].

This work demonstrates how the burden of Type 2 diabetes mellitus and its associated complications on both an individual and the country can be well managed by controlling the occurrence of IFG. We examined critical aspects of lifestyle and dietary behaviours that exposes an individual to IFG, a major predictor of Type 2 diabetes mellitus in Ghanaian society.

In this study, we determined the prevalence of impaired fasting blood glucose, factors that influence its occurrence and its impact on the development of undiagnosed Type 2 diabetes mellitus among rural and urban Ghanaian communities. This is necessary to help in health care planning and to promote good health care delivery. Our findings revealed high prevalence of IFG in the study area. Several factors were found to contribute significantly to the occurrence of impaired fasting glycaemia and thus propels the onset of Type 2 diabetes mellitus among Ghanaians. Notable among these factors were family history of diabetes, length of stay in a particular area, gender and pre-diabetes

status prior to the study. However, we observed that these factors vary from one geographical location to the other in the study area and even within the country at large as also shown in previous studies [8]. This could be due to host of factors including cultural practices, socio-economic status and urbanization characterized by the consumption of more processed and fatty food and poor dietary habits of individual Ghanaians.

The findings of this study showed that people living in urban Ghanaian communities are at high risk of having impaired fasting glycaemia and thus increases their risk of developing Undiagnosed Type 2 diabetes mellitus compared to those in rural communities. Inappropriate dietary practices characterized by the consumption of more processed foods and more fatty fats could partly explain this observation [4]. High socio-economic status coupled with poor knowledge on proper dietary practices was more common among urban communities and could also be a major contributory factor to the above observation. This observation was also made by [9] when they studied knowledge and practices of primary caregivers of Type 2 diabetes mellitus in Nigeria.

Our findings also show that most of the factors associated with impaired fasting glycaemia and the occurrence of undiagnosed Type 2 diabetes mellitus among Ghanaians are modifiable (Overweight/Obesity, Lipid Imbalance, Length of stay in a community and general poor dietary practices) with some few been non-modifiable (Gender, Ethnicity and family history of diabetes and hypertension). This therefore implies that more efforts that will ensure that people pay much attention to their dietary habits is needed in both rural and urban Ghanaian communities. As noted by Pollakova and the colleagues when they studied the impact of vegan diet on Type 2 diabetes, this will help break the transgenerational nature of the disease by reducing the prevalence of the modifiable risk factors associated with impaired fasting glycaemia and Type 2 diabetes mellitus [10]. This will then foster proper health care and improve the overall health status of both rural and urban Ghanaians who are exposed to the risk associated with IFG and Type 2 diabetes mellitus.

## Methodology

### Research design

A cross-sectional survey approach was adopted and data collection was done from January to May 2018 in the Tamale Metropolis.

### Study area and setting

Tamale Metropolis of the Northern Region of Ghana was the study area. The Metropolis had a total population of 293,881 as of December 2018 and majority are urban inhabitants [11]. It lies between latitude 9°16 and 9°34 North and longitudes 0°36 and 0°57 West. The Metropolis lies within the savannah woodland zone in

the country, it is about 180 m above sea level and has only one raining season [12].

### Study population

The study population was made up of adult residents of the Metropolis aged 18-50 years. The occurrence of Type 2 diabetes mellitus influenced by impaired fasting blood glucose appears to be most prevalent among this age group [13]. Hence the decision to select this age bracket to constitute the study population.

### Sample size determination

A total of 300 adults aged 18-50 years-old were used for this study. We estimated the sample size using Ralph. B. Formula [14], on crude prevalence of 16.4% of impaired fasting glycaemia among patients with impaired fasting glycaemia in Ghana as determined by Asamoah [15], at an alpha level of 1.96, an error margin of  $\pm 0.03$  and at a confidence interval of 95%. The formula yielded a sample size of 272. We also factored in a non-response rate of 10% and rounded the sample size to 300 to increase precision of the prevalence and factors estimates.

### Sampling

The metropolis has a total of 115 enumeration areas (EA's) [16]. This was grouped into five main enumerations sites, namely; Tamale North (Site A), Tamale South (Site B), Tamale East (Site C), Tamale Central (Site D) and Tamale West (Site E) using Ghana Statistical Service numeration guidelines. In each of the sites, four (4) communities were randomly selected consisting of two (2) rural and two (2) urban communities respectively, except for site D which consisted of only urban communities. Hence all four (4) communities selected were urban in site D. In each community, a first household was randomly selected, thereafter, a sampling frame of 5 was obtained and used to select the remaining 4 households. Five (5) households were selected from each community and three (3) participants randomly selected from each household. In all, a total of fifteen (15) participants were sampled from each community to give a total sample size of 300 participants.

### Inclusion and exclusion criteria

Adult residents of Tamale Metropolis aged 18-50 years, who gave consent to participate were recruited into the study. Individuals who were not residents in the Metropolis, severely-ill adults and non-consented people were excluded from the study.

### Training of data collectors

Four data collectors consisting of 2 enumerators and 2 phlebotomists were trained and used to collect all data for this study. The training was aimed at refreshing their knowledge on the specific data to be collected for the study and the necessary precautions to be put in place to ensure accurate data collection.

### Pretesting of data collection instruments

All data collection instruments (study questionnaire, scales, MUAC tapes and Height measuring boards) were pretested before been subsequently used for data collection on the field. Data precision depends on the accuracy of instruments used and the precautionary measures undertaken by data collectors [17].

### Data collection

A pre-tested questionnaire designed by the research team was used to collect both socio-demographic and biochemical data. The study was approved by the ethics committee of the college of basic and applied sciences, University of Ghana.

### Socio-demographic characteristics

A structured questionnaire was administered. This was on the participant's age, gender, educational background, family history of diabetes, hypertension, participants knowledge of diabetes and determinants of impaired fasting glycemia, IFG and its effects on the occurrence of Undiagnosed Type 2 Diabetes Mellitus (UT2DM).

### Anthropometric measurements

Bodyweight (kg), height (cm), and waist circumference (cm) measurements were done using modified World Health Organization standards [13]. Body mass indices (BMI's) of participants were calculated using the weight and height obtained as  $\text{weight/height}^2$  ( $\text{kg/m}^2$ ). This was then used to classify respondents into underweight, normal weight and overweight/obesity [18]. This criterion of measurement is categorized as follows: Normal weight = BMI of (18.5-24.9)  $\text{Kg/m}^2$ ; Overweight = BMI of (25-29.9)  $\text{Kg/m}^2$ ; Obesity = BMI  $\geq 30$   $\text{Kg/m}^2$ . In using waist circumference (WC), central obesity was classified as follows: Normal central body mass = WC < 104 cm for males, WC = < 88 cm for females, Central obesity = WC =  $\geq 104$  cm for males and WC  $\geq 88$  cm for females.

### Blood draw and processing

A 2 ml of venous blood was taken from each participant and placed in EPDM test tubes. This was after the participants had done an overnight fasting (approximately between 10-12 hours). The samples were deposited in a vaccine carrier containing ice packs to provide appropriate temperature. Samples were transported to Tamale Teaching Hospital laboratory for analysis. They were centrifuged at 3000 rpm for 10 minutes at room temperature using normal standard centrifuging. Blood sera were separated into plain separator tubes and used for clinical determination of impaired fasting glycaemia and Type 2 diabetes. The levels of total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) were determined using



an automated chemistry analyser with model number URIT-8021A.

### **Categorization of blood glucose among study participants**

Blood glucose concentrations were measured using a Glucometer (model number SN-2806405E) on fresh capillary blood samples drawn from each participant. Blood samples were dropped on strips and inserted into the glucometer to give a fasting blood glucose (FBG) reading for each participant. The serum glucose results were categorized and used to define the various indices in this study using the ADA classification [7]. Hypoglycaemia was defined as fasting blood glucose levels of  $< 3.9$  mmol/L; Normal glycaemia (Normoglycemia) was defined as fasting blood glucose of 3.9-5.5 mmol/L. Pre-diabetes was defined as fasting blood glucose of 5.6-6.9 mmol/L while Hyperglycaemia (Type 2 diabetes mellitus) was defined as fasting blood glucose  $\geq 7$  mmol/L [19].

### **Determination of glucose imbalance and undiagnosed type 2 diabetes mellitus in study participants**

Undiagnosed Type 2 diabetes mellitus in this study refers to diabetes of participants with no knowledge of their diabetes status prior to the study. This was determined using ADA classification [20], where fasting blood glucose  $\geq 7$  mmol/L in a patient defines Type 2 diabetes presence. IFG in this study referred to glucose imbalance in participants. Impaired fasting glucose was 26.7% and Undiagnosed Type 2 Diabetes Mellitus was 4.7% among adult Ghanaians in the Tamale Metropolis. Gestational diabetes which refers to fasting blood glucose  $\geq 5.6$  mmol/L in pregnant women was determined. Impaired fasting blood glucose in this study was defined to consist of both hypoglycaemia and hyperglycaemia [21].

### **Quality control**

The scales used to measure body weight of participants were standardized with a known weight on each day the research team took body weight measurements on the field. In order to ascertain the reliability of the glucometer, a certified reference blood sample with known FBG level was used every day and the reading checked to reflect accurate measurement. For the chemistry analyser, normal and abnormal reference control sera were used to determine the performance of both the reagent and the instrument in the determination process of total blood cholesterol, triglycerides, HDL-C and LDL-C.

### **Data analyses**

The IBM Statistical Package for Social Sciences (SPSS) version 23.0 for windows was used for data entry and for all statistical analyses except for anthropometric values that were first entered into WHO Anthropometric

software and later exported into SPSS. For continuous variables, means ( $\bar{n}$ ) and standard deviations (SD) were calculated, while for categorical variables, proportions were determined. Differences between means were assessed by independent t-sample test analysis and chi-square was used to determine differences between categorical variables. Simple linear regression was used to determine the association between potential risk factors and impaired fasting blood glucose among study participants. Binary logistic regression analysis was used to determine association between potential risk factors and the development of undiagnosed Type 2 diabetes mellitus among study participants. A p-value of  $< 0.05$  was considered statistically significant.

### **Ethical approval**

Inform consent was sought from participants before they were enrolled into the study. Those who were enrolled signed the consent form and others thumb-printed the form to indicate their consent. Ethical approval of this research was granted by the Ethics Committee of the College of Basic and Applied Sciences, ECBAS, University of Ghana, Legon (ECBAS 017/17-18).

## **Results**

### **Descriptive statistics on socio-demographic characteristics of participants**

Three hundred (300) participants were recruited into the study and their socio-demographic characteristics are shown in [Table 1](#). This consisted of 44.7% males and 55.3% females. The mean age of our study participants was  $34.4 \pm 10.6$  years. Mean age among males was lower than that among females ( $32.0 \pm 10.4$  years and  $36.4 \pm 10.3$  years respectively). There was a significant difference between the two mean age groups ( $p < 0.01$ ). Most of our study participants (83.7%) had low socio-economic status with females mostly affected (88.6%). Dagomba's were the most dominant ethnic group (90%) among the study participants. There was an observed significant difference in ethnicity in our study population ( $P < 0.01$ ). Also, there was significant difference between participant's educational status ( $P < 0.01$ ).

### **Dietary behaviour of study participants**

Most of the participants (85.0%) consumed carbohydrate base foods with only few (10.4%) consuming fruits and vegetables. A lot of the participants, (53.0%) consumed their staple foods of the day in the evening. Most of the participants (82.3%) could afford the usual three daily meals, however their choice of food was mainly influenced by the availability and cost of the food (56.0%) ([Table 2](#)). Health benefits of food were the last thing to consider when it comes to the choice of food among study participants.

### **Physical activity and anthropometric characteristics of participants**

Our findings reveal that a lot of people in the study area engaged in low physical activities (49.7%). Only

**Table 1:** Socio-demographic characteristics of study participants.

Variables	Male (n = 134) <sup>2</sup> n (%)	Female (n = 166) n (%)	Total (N = 300) N (%)	p-value <sup>1</sup>
<b>Age in yrs.</b>				
(Mean ± SD) <sup>3</sup>	32.0 ± 10.4	36.4 ± 10.3	34.4 ± 10.6	< 0.01**
18-30	70 (52.2)	59 (35.5)	129 (43.0)	
31-50	64 (47.8)	107 (64.5)	171 (57.0)	
<b>Ethnic groups</b>				
Dagomba's	127 (94.8)	143 (86.1)	270 (90.0)	0.01**
Gonja's	1 (0.7)	11 (6.7)	12 (4.0)	
Mamprusi's	2 (1.5)	6 (3.6)	8 (2.7)	
Akan's	2 (1.5)	1 (0.6)	3 (1.0)	
Grusi's	2 (1.5)	4 (2.4)	6 (2.0)	
Mou's	0 (0.0)	1 (0.6)	1 (0.3)	
<b>Educational status</b>				
No formal Education	56 (41.8)	108 (65.1)	164 (54.7)	< 0.01**
Primary	7 (5.2)	20 (12.0)	27 (9.0)	
J.H. S	0 (0.0)	4 (2.4)	4 (1.3)	
S.H.S	47 (35.1)	25 (15.1)	72 (24.0)	
Tertiary	24 (17.9)	9 (5.4)	33 (11.0)	
<b>Occupation</b>				
Farmers	52 (38.8)	64 (38.6)	116 (38.7)	0.78
Civil servants	14 (10.4)	11 (6.6)	25 (8.3)	
Traders/Business personnel	32 (23.9)	63 (38.0)	95 (31.7)	
Students	18 (13.4)	4 (2.4)	22 (7.3)	
Others	18 (13.4)	24 (14.5)	42 (14.0)	
<b>Socio-economic status</b>				
Low	104 (77.6)	147 (88.6)	251 (83.7)	0.05
Middle	14 (10.4)	12 (7.2)	26 (8.7)	
High	16 (12.0)	7 (4.2)	23 (7.6)	

SD: Standard Deviation; <sup>1</sup>Significance based on independent T-test for continuous variables and chi-square for categorical variables; <sup>2</sup>n (%) = proportions; <sup>3</sup>Mean ± standard deviation; \*\*p-values significant at < 0.05.

**Table 2:** Dietary behaviour/characteristics of study participants.

Variables	Male (n = 134) <sup>2</sup> n (%)	Female (n = 166) n (%)	Total (N = 300) N (%)	p -value <sup>1</sup>
<b>Staple foods consumed</b>				
Touzafi	113 (84.3)	142 (85.6)	255 (85.0)	0.85
Banku	5 (3.7)	5 (5.0)	10 (3.3)	
Kenkey	3 (2.2)	1 (0.6)	4 (1.3)	
Rice	11 (8.2)	17 (10.2)	28 (9.4)	
Fufu	1 (0.7)	1 (0.6)	2 (0.7)	
Yam	1 (0.7)	0 (0.0)	1 (0.3)	
<b>Other foods consumed</b>				
Porridge	77 (57.5)	82 (49.4)	159 (53.0)	0.11
Tea	35 (26.1)	35 (21.1)	70 (23.3)	
Rice porridge	3 (2.2)	21 (12.7)	24 (8.0)	
Fruit base foods	11 (8.2)	20 (12.0)	31 (10.4)	
<b>Time of food consumption</b>				
Morning	32 (23.9)	56 (33.7)	88 (29.3)	0.03**

Afternoon	29 (21.6)	24 (14.5)	53 (17.7)	
Evening	73 (54.5)	86 (51.8)	159 (53.0)	
<b>Frequency of food consumed</b>				
< 3 a day	29 (21.6)	24 (14.5)	53 (17.7)	0.01**
≥ 3 a day	105 (78.4)	142 (85.5)	247 (82.3)	
<b>Choice of food</b>				
Availability	81 (60.4)	87 (52.4)	168 (56.0)	0.15
Cost of food	48 (35.8)	65 (39.2)	113 (37.7)	
Health benefits	5 (3.8)	14 (8.4)	19 (6.3)	

<sup>1</sup>Significance based on Pearson's chi-square test for categorical variables; <sup>2</sup>n (%) = proportions; \*\*p-values significant at < 0.05.

**Table 3:** Physical activity and anthropometric profile of participants by gender.

Variables	Males (n = 134) <sup>2</sup> n (%)	Females (n = 166) n (%)	Total (N = 300) N (%)	p-value <sup>1</sup>
<b>Physical Activity</b>				
Low	54 (40.3)	95 (57.2)	149 (49.7)	0.06
Moderate	42 (31.3)	57 (34.3)	99 (33.0)	
High	38 (28.4)	14 (8.4)	52 (17.3)	
<b>BMI (Kg/m<sup>2</sup>)</b>				
(Mean ± SD) <sup>3</sup>	22.4 ± 3.7	23.8 ± 4.7	23.2 ± 4.3	< 0.01**
< 18.5	21 (15.7)	14 (8.4)	35 (11.7)	
18.5-24.9	85 (63.4)	92 (55.4)	177 (59.0)	
25-29.9	23 (17.2)	47 (28.3)	70 (23.3)	
> 30	5 (3.7)	13 (7.8)	18 (6.0)	
<b>WC (cm)</b>				
(Mean ± SD)	77.8 ± 10.6	84.3 ± 11.1	81.4 ± 11.4.	< 0.01**
< 60	1 (0.7)	0 (0.0)	1 (0.3)	
60-87	112 (83.6)	107 (64.5)	219 (73.0)	
88-104	18 (13.4)	52 (31.3)	70 (23.3)	
> 104	3 (2.2)	7 (4.2)	10 (3.3)	

WC: Waist Circumference; BMI: Body Mass Index, SD: Standard Deviation, <sup>1</sup>Significance based on independent T-test for continuous variables and chi-square test for categorical variables; <sup>2</sup>proportions; <sup>3</sup>Mean ± standard deviation; \*\*p-values significant at < 0.05.

17.3% of the participants engaged in high physical activities. Male participants had low BMI (22.4 ± 3.78 kg/m<sup>2</sup>) than that of females (23.8 ± 4.7 kg/m<sup>2</sup>) and their difference was statistically significant (p < 0.01). This is shown in Table 2 and Table 3 respectively.

We observed that 11.7% of the participants had low weight with respect to their age (BMI < 18 Kg/m<sup>2</sup>). Obesity which has become an issue of public health concern in Ghana was higher (6.0%) than anticipated among study participants (BMI ≥ 30 Kg/m<sup>2</sup>). Males had higher mean waist circumference (77.8 ± 10.6 cm) than that of females (84.3 ± 11.1 cm) with significant mean difference (p < 0.01). However central obesity was more prevalent among females (35.5%) than males (2.2%) with WC > 104 cm. Central obesity was high among urban women (24.2%) than women living in rural areas (21.7%). However central obesity among both rural and

urban men was same (3.3%). Overall central obesity was quite high among our study participants (20.7%).

Urban residents had low weight (12.8%) compared to that of the rural participants (10.0%). As many as 30.6% of urban participants were overweight while only 12.5% of the rural participants were overweight. Obesity was more prevalent among urban residents (7.2%) than rural residents (4.2%). This predisposed urban residents to developing impaired fasting blood glucose and exposes them to UT2DM in the study area.

### Fasting blood glucose and biochemical characteristics of participants

Fasting blood glucose in our study participants ranged from 3.2 to 14.2 mmol/L, with a mean of 4.92 ± 1.19 mmol/L (Table 4). Mean FBG among males (4.72 ± 0.89 mmol/L) was lower than among females (5.08 ± 1.36

**Table 4:** Physical activity and anthropometric profile of participants by setting.

Variables	Rural (n = 120) <sup>2</sup> n (%)	Urban (n = 180) n (%)	Total (N = 300) N (%)	p -value <sup>1</sup>
<b>Physical Activity</b>				
Low	47 (39.2)	102 (56.7)	149 (49.7)	< 0.01**
Moderate	45 (37.5)	54 (30.0)	99 (33.0)	
High	28 (23.3)	24 (13.3)	52 (17.3)	
<b>BMI (Kg/m<sup>2</sup>)</b>				
(Mean ± SD) <sup>3</sup>	22.09 ± 3.89	23.90 ± 4.46	23.2 ± 4.3	< 0.01**
< 18.5	12 (10.0)	23 (12.8)	35 (11.7)	
18.5-24.9	88 (73.3)	89 (49.4)	177 (59.0)	
25-29.9	15 (12.5)	55 (30.6)	70 (23.3)	
> 30	5 (4.2)	13 (7.2)	18 (6.0)	
<b>WC (cm)</b>				
(Mean ± SD)	81.50 ± 10.51	81.30 ± 11.93	81.41 ± 11.40	0.88
< 60	0 (0.0)	1 (0.3)	1 (0.3)	
60 - 87	90 (75)	129 (71.7)	219 (73)	
88 - 104	26 (21.7)	44 (24.4)	70 (23.3)	
> 104	4 (3.3)	6 (3.3)	10 (3.3)	

BMI: Body Mass Index; WC: Waist Circumference, <sup>1</sup>Significance based on Pearson's chi-square for categorical variables and independent sample t-test for continuous variables, <sup>2</sup>proportions; <sup>3</sup>Mean ± SD indicates mean ± standard deviation; \*\*p-values significant at p < 0.05.

mmol/L) and their difference was statistically significant (p < 0.01). We observed that 9.0% of the participants had low fasting blood glucose (hypoglycemia) (FBG < 3.9 mmol/L) and 13.0% had pre-diabetes (FBG from 5.6-6.9 mmol/L) which comprised of 11.2% males and 14.5% females. We found an impaired fasting blood glucose, IFG prevalence of 26.7% and Undiagnosed Type 2 diabetes mellitus, UT2DM (hyperglycemia) prevalence of 4.7% respectively among our study participants.

Mean total cholesterol in study participants was 4.62 ± 1.43 mmol/L. Total blood cholesterol ranged from 0.68 mmol/L to 9.68 mmol/L in males and 0.72 mmol/L to 8.62 mmol/L in females. We observed that mean total cholesterol among females (4.80 ± 1.45 mmol/L) was higher than that among males (4.41 ± 1.38 mmol/L) and their difference was statistically significant (p = 0.02). Lowest and highest blood triglycerides were 0.29 mmol/L and 7.08 mmol/L respectively with a mean of 1.60 ± 0.83 mmol/L. Mean triglycerides in males and females were 1.58 ± 0.75 mmol/L and 1.61 ± 0.89 mmol/L respectively with ranges of 0.33 mmol/L to 7.08 mmol/L in males and 0.29 mmol/L to 7.08 mmol/L in females. Hypertriglyceridemia was more prevalent among females (13.9%) than among males (11.2%). There was no significant difference between mean hypertriglyceridemia in males and that of females (p = 0.69). We observed that 12.7% of our study participants had hypertriglyceridemia (triglyceride levels ≥ 2.3 mmol/L).

Mean HDL-cholesterol among males (0.73 ± 0.22 mmol/L) was lower than that among females (0.82

± 0.26 mmol/L) and there were statistically different from each other (p < 0.01). Also mean LDL-cholesterol among males, 2.99 ± 1.18 mmol/L was lower than among females, 3.25 ± 1.99 mmol/L, however their difference was not statistically significant (p = 0.06). High LDL cholesterol (LDL ≥ 3.4 mmol/L) was more prevalent in females than in males (40.4% and 30.6% respectively). Surprisingly impaired fasting blood glucose was slightly more prevalent in males (26.9%) than in females (26.5%). The means of LDL-cholesterol between males and females was not statistically different from each other (p = 0.06).

#### Determinants of impaired fasting blood glucose among study participants

We observed a positive association between family history of diabetes mellitus among participants and the development of IFG among our study participants (hypoglycemia, pre-diabetes and hyperglycemia) (p < 0.01). Thus, impaired fasting blood glucose, IFG was more prevalent (54.8%) among study participants with positive family history of Type 2 diabetes mellitus.

We also found a significant association between family history of hypertension and impaired fasting blood glucose among study participants (p = 0.01). More impaired fasting blood glucose levels was found in participants with positive history of pre-diabetes and gestational diabetes (87.5% and 75.0% respectively). There was a positive association between pre-diabetes, gestational diabetes and the development of impaired fasting blood glucose among study participants.

( $p < 0.01$ ). There was also an association between participant's knowledge on diabetes mellitus and impaired fasting blood glucose among participants, however the association was not statistically significant ( $p = 0.09$ ).

We also found an association between engaging in physical activity and impaired fasting blood glucose among study participants, however this association

was not statistically significant at  $p = 0.48$ . A significant statistical association was found between gender and impaired fasting blood glucose among study participants ( $p = 0.03$ ). Residence status of study participants was also significantly associated with the development of impaired fasting blood glucose (hypoglycemia, pre-diabetes and hyperglycemia) among the study participants ( $p = 0.02$ ).

**Table 5:** Fasting blood glucose and lipid profile of participants by gender.

Variables (mmol/L)	Male (n = 134) <sup>2</sup> n (%)	Female (n = 166) n (%)	Total (N = 300) N (%)	p-value <sup>1</sup>
<b>FBG</b>				
(Mean $\pm$ SD) <sup>3</sup>	4.72 $\pm$ 0.89	5.08 $\pm$ 1.36	4.92 $\pm$ 1.19	0.01**
< 3.9	18 (13.4)	9 (5.4)	27 (9.0)	
3.9-5.5	98 (73.1)	122 (73.5)	220 (73.3)	
5.6-6.9	15 (11.2)	24 (14.5)	39 (13.0)	
$\geq 7$	3 (2.2)	11 (6.6)	14 (4.7)	
<b>Total cholesterol</b>				
(Mean $\pm$ SD)	4.41 $\pm$ 1.38	4.80 $\pm$ 1.45	4.62 $\pm$ 1.43	0.02**
< 5.2	104 (77.6)	100 (60.2)	204 (68.0)	
$\geq 5.2$	30 (22.4)	66 (39.8)	96 (32.0)	
<b>Triglycerides</b>				
(Mean $\pm$ SD)	1.58 $\pm$ 0.75	1.61 $\pm$ 0.89	1.60 $\pm$ 0.83	0.69
< 2.3	119 (88.8)	143 (86.1)	262 (87.3)	
$\geq 2.3$	15 (11.2)	23 (13.9)	38 (12.7)	
<b>HDL - cholesterol</b>				
(Mean $\pm$ SD)	0.73 $\pm$ 0.22	0.82 $\pm$ 0.26	0.78 $\pm$ 0.24	< 0.01**
< 1	122 (91.0)	130 (78.3)	252 (84.0)	
1-1.29	11 (8.2)	30 (18.1)	41 (13.7)	
$\geq 1.3$	1 (0.7)	6 (3.6)	7 (2.3)	
<b>LDL - cholesterol</b>				
(Mean $\pm$ SD)	2.99 $\pm$ 1.18	3.25 $\pm$ 1.19	3.14 $\pm$ 1.19	0.06
< 3.4	93 (69.4)	99 (59.6)	192 (64.0)	
$\geq 3.4$	41 (30.6)	67 (40.4)	108 (36.0)	

FBG: Fasting Blood Glucose; HDL: High Density Lipoprotein cholesterol; LDL: Low Density Lipoprotein cholesterol; mean  $\pm$  SD = Mean  $\pm$  standard deviation; <sup>1</sup>Significance based on independent t-test, <sup>2</sup>proportions; \*\*p-value significant at  $< 0.05$ .

**Table 6:** Fasting blood glucose and lipid profile of participants by setting.

Variables (mmol/L)	Rural (n = 120) <sup>2</sup> n (%)	Urban (n = 180) n (%)	Total (N = 300) N (%)	p -value <sup>1</sup>
<b>FBG</b>				
(Mean $\pm$ SD) <sup>3</sup>	4.76 $\pm$ 0.87	5.02 $\pm$ 1.35	4.92 $\pm$ 1.19	0.06
< 3.9	10 (8.4)	17 (9.4)	27 (9.0)	
3.9-5.5	97 (80.8)	123 (68.4)	220 (73.3)	
5.6-6.9	12 (10.0)	27 (15.0)	39 (13.0)	
$\geq 7$	1 (0.8)	13 (7.2)	14 (4.7)	
<b>Total cholesterol</b>				
(Mean $\pm$ SD)	4.11 $\pm$ 1.49	4.97 $\pm$ 1.29	4.62 $\pm$ 1.43	< 0.01**
< 5.2	94 (78.3)	110 (61.1)	204 (68.0)	
$\geq 5.2$	26 (21.7)	70 (38.9)	96 (32.0)	



<b>Triglycerides</b>				
(Mean ± SD)	1.29 ± 0.75	1.80 ± 0.82	1.60 ± 0.83	< 0.01**
< 2.3	116 (96.7)	146 (81.1)	262 (87.3)	
≥ 2.3	4 (3.3)	34 (18.9)	38 (12.7)	
<b>HDL Cholesterol</b>				
(Mean ± SD)	0.72 ± 0.25	0.82 ± 0.23	0.78 ± 0.24	< 0.01**
< 1	106 (88.3)	146 (81.1)	252 (84.0)	
1-1.29	12 (10)	29 (16.1)	41 (13.7)	
≥ 1.3	2 (1.7)	5 (2.8)	7 (2.3)	
<b>LDL Cholesterol</b>				
(Mean ± SD)	2.82 ± 1.22	3.35 ± 1.15	3.14 ± 1.19	< 0.01**
< 3.4	84 (70.0)	108 (60.0)	192 (64.0)	
≥ 3.4	36 (30.0)	72 (40.0)	108 (36.0)	

FBG: Fasting Blood Glucose, HDL: High-density lipoprotein, LDL: Low-Density Lipoprotein, <sup>1</sup>Significance based on independent sample t-test, <sup>2</sup>proportions, <sup>3</sup>Mean±SD indicates mean ± standard deviation, \*\*p-values significant at p < 0.05.

**Table 7:** Factors associated with impaired fasting blood glucose among participants.

Variables	Impaired IFG <sup>2</sup> n (%)	Normoglycemia n (%)	Total N (%)	Odds Ratio	95% CI. Ref. Cat.	p-value <sup>1</sup>
<b>FDM</b>						
Yes	17 (54.8)	14 (45.2)	31 (10.3)	2.6	0.06-1.99	< 0.01**
No	63 (23.4)	206 (76.8)	269 (89.7)	1.0		
<b>FHPT</b>						
Yes	24 (38.7)	38 (61.3)	62 (20.7)	2.4	0.41-2.46	0.03**
No	56 (23.5)	182 (76.5)	238 (79.3)	1.0		
<b>HPD</b>						
Yes	7 (87.5)	1 (12.5)	8 (2.7)	1.5	0.67-1.13	< 0.01**
No	73 (25.0)	219 (75.0)	292 (97.3)	1.0		
<b>HGD</b>						
Yes	6 (75.0)	2 (25.0)	8 (4.8)	4.3	0.08-3.88	< 0.01**
No	38 (24.1)	120 (75.9)	158 (95.2)	1.0		
<b>KDM</b>						
Yes	42 (34.1)	81 (65.9)	123 (41.0)	0.3	0.04-0.29	0.09
No	38 (21.5)	139 (78.5)	177 (59.0)	1.0		
<b>VPA</b>						
Yes	44 (30.8)	99 (69.2)	143 (47.7)	0.1	0.09-12.20	0.48
No	36 (22.9)	121 (77.1)	157 (52.3)	1.0		
<b>Gender</b>						
Males	36 (26.9)	98 (73.1)	134 (44.7)	1.0	1.02-1.80	0.03**
Females	44 (26.5)	122 (73.5)	166 (55.3)	6.2		
<b>Residence</b>						
Rural	23 (19.2)	97 (80.8)	120 (40.0)	1.0	0.91-2.67	0.02**
Urban	57 (31.7)	123 (68.3)	180 (60.0)	2.8		
<b>BMI</b>						
Normal	52 (24.5)	160 (75.5)	212 (70.7)	1.0		
Overweight	20 (28.6)	50 (71.4)	70 (23.3)	3.4	1.17-2.76	< 0.01**
Obese	8 (44.4)	10 (55.6)	18 (6.0)	5.5		
<b>Age (yrs)</b>						
18-30	31 (24.0)	98 (76.0)	129 (43.0)	1.0	1.02-2.43	0.42
31-40	19 (22.6)	65 (77.4)	84 (28.0)	1.4		

41-50	30 (34.5)	57 (66.5)	87 (29.0)	2.2		
<b>LSTM (yrs)</b>						
< 10	14 (25.0)	42 (75.0)	56 (18.6)	1.0	1.02-2.43	0.04**
10-20	15 (27.8)	39 (72.2)	54 (18.0)	2.1		
> 20	51 (26.8)	139 (73.2)	190 (63.3)	2.5		
<b>Ethnicity</b>						
Dagomba's	65 (24.1)	205 (75.9)	270 (90.0)	1.0	1.03-1.67	< 0.01**
Gonja's	9 (75.0)	3 (25.0)	12 (4.0)	1.3		
Other's	6 (33.3)	12 (66.7)	18 (6.0)	1.9		

LSTM: Length of stay in Tamale Metropolis, CI: Confidence Interval, Others: Manprusi's, Grusi's, Akan's, Mou's. \*Significance based on Pearson's chi-square test for categorical variables, <sup>2</sup>proportions; \*\*p-values significant at  $p < 0.05$ .

Impaired fasting blood glucose was more prevalent among participants who were overweight and obese (28.6% and 44.4% respectively) (Table 5, Table 6 and Table 7). Statistically overweight/obesity among study participants was significantly associated with developing impaired fasting blood glucose among participants ( $p < 0.01$ ). Age was found to be significantly associated with developing impaired fasting blood glucose among study participants at  $p < 0.01$ . We also found a positive association between participant's length of stay in Tamale Metropolis and developing impaired fasting blood glucose. Impaired fasting blood glucose was more prevalent (26.8%) among participants who had stayed in the Metropolis for more than 20 years. Statistically however this association was not significant ( $p = 0.42$ ). Impaired fasting blood glucose was lower among Dagomba's (24.1%) than other ethnic groups. There was a significant statistical association between ethnicity and impaired fasting blood glucose at  $p = 0.04$ .

Impaired fasting blood glucose was more prevalent in participants who had no hypercholesterolemia (27.5%). Statistically, however there was no significant association between hypercholesterolemia and impaired fasting blood glucose ( $p = 0.05$ ). Impaired fasting blood glucose was more prevalent among participants who had hypertriglyceridemia (39.5%) (Table 7). The association between hypertriglyceridemia and impaired fasting blood glucose was statistically significant ( $p < 0.01$ ). We further observed that low high-density lipoproteins and high low-density lipoproteins in blood were both significantly associated with impaired fasting blood glucose in our study participants ( $p < 0.01$  and  $p = 0.01$  respectively).

## Discussion

In this study, we determined the prevalence of impaired fasting glucose, factors that influence its occurrence and its influence on developing undiagnosed Type 2 diabetes mellitus (UT2DM). Our findings revealed varied number of factors influencing the occurrence of high impaired fasting blood glucose among rural and urban Ghanaians and its effects on developing undiagnosed Type 2 diabetes mellitus.

We found an impaired fasting blood glucose prevalence of 26.7% among our study participants with significant impact on the development of undiagnosed Type 2 diabetes mellitus. This observation is consistent with an IFG prevalence of 26.4% among adult Nigerians in a study conducted by [18] which influenced Type 2 diabetes mellitus among study participants. The prevalence of IFG in the present study has significant consequences on undiagnosed Type 2 diabetes and its un-detection earlier could lead to other diseases such as cardiovascular diseases, kidney retinopathy and visual deterioration [22]. However the observed prevalence is lower than a prevalence of 57.0% reported among adult Ghanaians by [23]. Impaired fasting blood glucose was more prevalent among participants who had positive family history of diabetes mellitus, influencing Type 2 diabetes mellitus (Table 7) and the findings are similar to those found among Cameroonian adults by [24]. The high prevalence of IFG may also be due to the high levels of obesity and hyperlipidaemia in the present study. Similar observations were made by [18] and [25] in separate Nigerian Studies that looked at the geographical prevalence of diabetes mellitus which influences Type 2 diabetes positively.

Our findings showed a pre-diabetes prevalence of 13.0% and this is similar to what was reported by [26] in Cote d'Ivoire. Prediabetes was more prevalent among urban participants (15.0%) than rural participants (10.0%) and this agrees with findings of 15.2% and 9.8% among urban and rural adults respectively in Saudi Arabia as reported by [4]. It was not surprising that impaired fasting blood glucose and undiagnosed Type 2 diabetes was more prevalent among urban residents. Sedentary lifestyles and unhealthy dietary practices leading to high risks among urban dwellers could be a contributory factor to this observation. Similar conclusions were made by [27] in a study that looked at factors influencing IFG and diabetes occurrence in Saudi Arabia.

Being a female predicted IFG among our participants. This may be due to the fact that female participants may have a history of gestational diabetes which may not have been fully resolved prior to our study [28]. Available

data shows that Ghanaian females are more obese than men which predisposes them to diabetes mellitus and IFG [29]. Obesity predicted the development of IFG in the current study [30], consistent with a Cameroonian study which found that obese subjects were about 2-4 times at risk of developing IFG. In a separate Ivorian studies, Oyensu and Odegard also found obese participants to have 6 folds risk of developing IFG [10,26]. This in turn increases the risk of developing Type 2 diabetes by 4 folds. In a South African study carried out in the West province, Podoa also reported 6.5 times risk of developing IFG among obese subjects and increased risk of Type 2 diabetes [31]. It is believed that obesity leads to insulin resistance in the peripheral tissue and insulin secretory defect of the beta cell which predisposes an individual to diabetes mellitus and IFG [24] and [32].

Living in urban Tamale Metropolis predicted IFG and increased the risk of developing UT2DM. This may be due to physical inactivity and poor dietary intake which increases an individual chance of developing IFG and hyperglycaemia. Our findings are consistent with a comparative study by [9] and [33], whose findings showed that the prevalence of IFG increases with urbanization, with those migrating from rural to urban communities having 4 folds increased risk. In an Arabian and Nigerian studies respectively, Alotabi and Ogbera found environmental factors to be associated with IFG and Type 2 diabetes mellitus [25,27]. Our findings also revealed that family history of diabetes increases risk of IFG and UT2DM irrespective of the residency status of participants. This was also observed by [34], whose studies was among siblings of patients with Type 2 diabetes with high IFG. Subjects in the study had 12 fold risk of IFG and developing Type 2 diabetes. The findings of our study is also consistent with that of [35] who found a six-fold risk of developing IFG among siblings with Type 2 diabetes mellitus in a Saudi Arabia study.

### Study Limitations

Despite the robust nature of the study design employed for this study, it has some weaknesses. The inability to verify the diabetes status of respondents prior to their enrolment may have an influence on our findings. We did not perform some important diabetes test such as oral glucose test to confirm our results. We are also unable to establish causation but rather associations with the risk factors of IFG since this was a cross-sectional study. Despite these few limitations, our findings are still valid and accurate in estimating the true state of IFG and UT2DM in the study area.

### Conclusion

Our findings revealed significant prevalence of both impaired fasting blood glucose and Undiagnosed Type 2 diabetes mellitus. We observed that several factors increase the risk of developing IFG among residents of the study area which thus increases their chances of

having severe Undiagnosed Type 2 diabetes mellitus. Most of these factors were modifiable and hence proper attention especially healthy dietary behaviour change is needed to ensure that people do not expose themselves to the risk associated with both IFG and UT2DM.

### Results

Tables (Table 1, Table 2, Table 3, Table 4, Table 5, Table 6 and Table 7).

### Declaration

We confirm that the manuscript has been read and approved by all named authors and that there are no other persons who satisfied the criteria for authorship but are not listed. We further confirm that the order of authors listed in the manuscript has been approved by all of us. We confirm that all authors have contributed significantly to the work and attest to the validity and legitimacy of the data and its interpretation and agrees to its submission.

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### Conflicts of Interests

The authors declare that they have no conflict of interest.

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