Original Article

Prevalence and Determinants of Glucose Intolerance in a Northern Nigerian Population: Role of Insulin Resistance

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Abstract

Background: Insulin resistance is a major factor in the pathogenesis of type 2 diabetes mellitus. This study is aimed at determining the prevalence of insulin resistance in a Northern Nigerian population. Other objectives include determining the interrelationship between body mass index (BMI), waist circumference (WC), waist–hip ratio (WHR), insulin resistance, and plasma glucose levels; and establishing whether insulin resistance is a significant determinant of glucose intolerance in the studied population. **Materials and Methods:** In total, 400 subjects were recruited using cluster sampling from their respective communities after due consent. Relevant biodata were documented and appropriate examinations including anthropometric measurements were carried out. Oral glucose tolerance test was carried out and fasting plasma insulin levels were also measured. Insulin resistance was calculated using homeostatic model assessment of insulin resistance. **Results:** The prevalence of insulin resistance was 36.6%, whereas that of glucose intolerance (prediabetes 17.2% and diabetes 9.3%) was 26.5%. Logistic regression analysis showed that insulin resistance, BMI, WC, waist–height ratio, and WHR were significant determinants of glucose intolerance. **Conclusions:** The prevalence of insulin resistance and glucose intolerance were high. Markers of insulin resistance and some key markers of obesity were important determinants of glucose intolerance in this study.

Keywords: Diabetes mellitus, glucose intolerance, insulin resistance, oral glucose tolerance test

Introduction

Insulin resistance is one of the major pathogenetic mechanisms in the development of type 2 diabetes mellitus (DM).[1-3] It is a state in which a given concentration of insulin produces a less-than-expected biological effect.[3] There is reduction in peripheral glucose uptake and local storage of glucose as glycogen and triglycerides in muscle and fat cells, respectively. In addition, reduced glycogen synthesis and storage and a failure to suppress glucose production and release into the blood occur in liver cells.[3]

In a study involving different segments of the Nigerian population, Bakari *et al.*,[4] Gezawa *et al.*,[5] and Awofisoye *et al.*[6] reported predominant insulin resistance among type 2 DM patients. Insulin resistance has been implicated by several other authors as the predominant defect in obese subjects with or without type 2 DM.[7-15] Varied prevalence figures of insulin resistance have been recorded in different parts of the world. Gezawa *et al.*[5] reported a prevalence of 25% in a Northeast Nigerian population, whereas Young *et al.*[7] reported a prevalence of 45.3% in a southeast Nigeria population, which is similar to the 46.3% prevalence recorded by Yin *et al.*[8] in a Chinese population. Another study of insulin resistance among some Indian subjects by Roopa *et al.*[9] showed a prevalence of 20%. These high-prevalence figures were linked to increasing adoption of western lifestyles and obesity. The increasing implication of insulin resistance as a pathogenetic factor underlying type 2 DM among Africans and other developing countries is not unconnected with the urbanization of rural areas. This includes increasing adoption of western lifestyles, viz., consumption of high energy, refined fast foods/drinks, sedentary lifestyle, smoking, etc.[1-3]

Access this article online

Quick Response Code:

Website: www.njbcs.net

DOI: 10.4103/njbcs.njbcs_36_18

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Received : 14-Dec-18 **Revised :** 01-Mar-19
Accepted : 06-Mar-19 **Published Online :** 19-Nov-19

How to cite this article: Lawal Y, Bello F, Anumah FE, Bakari AG. Prevalence and determinants of glucose intolerance in a Northern Nigerian population: Role of insulin resistance. Niger J Basic Clin Sci 2019;16:83-9.

This study was aimed at determining the prevalence of insulin resistance and its relationship with key markers of obesity and glucose intolerance in a Northern Nigerian population. Lifestyle and pharmacological therapy can, therefore, be targeted against insulin resistance irrespective of glycemic status, thereby reducing the overall incidence and prevalence of type 2 DM.[16-20] Furthermore, there are not much studies on insulin resistance (homeostasis model assessment for insulin resistance [HOMA-IR]) that have been carried out in Northern Nigeria further justifying the need for this research.

Materials and Methods

This was a cross-sectional observational community-based study carried out in Zaria, Northwest Nigeria, in 2014. Zaria is a major city in Kaduna State of Northern Nigeria, whose population is mainly of Hausa–Fulani ethnic group. The predominant occupation of the men include farming and trading, whereas the women are mainly housewives who engage in domestic works.[21]

Approval for the study was sought from and granted by the Research and Ethical committee of Ahmadu Bello University Teaching Hospital, Zaria. In total, 400 subjects who satisfied the inclusion criteria were enrolled. Inclusion criteria included subjects who were not previously known to have diabetes within the age of 18–70 years, whereas exclusion criteria included subjects known to have DM, pregnant women, ill subjects, those that cannot stand or are wheelchair bound, subjects on medications that affect glucose metabolism such as steroids, thiazides, beta blockers or HIV protease inhibitors, and subjects who declined consent.

Sample selection was achieved by cluster random sampling from 10 communities with each community representing a cluster. The communities included Shika, Milgoma, Bomo, Samaru, and Hayin Dogo. Other recruitment areas for the study were Zangon Shanu, Hayin Mallam, Hayin Dogo, and Hanwa communities. About 40 subjects were selected from each community by simple random sampling. Blinding was achieved by writing numbers concealed in small pieces of paper properly mixed and subjects asked to pick. Those that pick the numbers marked as subjects were subsequently enrolled into the study.

The patient information sheets were distributed to selected participants who were literate, and read/explained to those who were not literate. Written and duly signed or thumb-printed consent was obtained from each prospective participant and consecutively enrolled.

The World Health Organisation (WHO)-3-step pro forma[22] modified to suit the peculiarities of this study was used for the collection of data. These included history (age, sex, ethnic group, occupation, marital status; medical, medication, social history; and family history of diabetes, hypertension, obesity); physical examination (some anthropometric indices, systemic examinations); and results of laboratory investigation (FPG, 2hrPPG, fasting insulin level). Study participants were diagnosed as having impaired fasting glucose, impaired glucose tolerance, or DM, if they satisfy the WHO/International Diabetes Federation (IDF) criteria.[1,2] The pro forma was filled for each participant by the researcher with the help of a trained assistant.

Physical examinations including anthropometric measurements were carried out by the researcher with the help of a trained assistant and in the presence of a chaperone.

Standing height was measured using a Stadiometer (Seca 213 portable Stadiometer, Seca North America, Chino, CA) with a fixed vertical backboard and an adjustable head piece. Participants were asked to remove any hair ornaments, jewelry, buns, or braids from the top of their heads.[23] Each participant's head was positioned in the Frankfort horizontal plane (i.e., when the horizontal line from the ear canal to the lower border of the orbit is parallel to the floor and perpendicular to the vertical backboard), and then the height was measured to the nearest 0.1 cm.[23]

Study participants were then weighed in kilograms using a beam balance (Seca 700 series, Seca North America). They were asked to wear only light clothings/undergarments and stand in the center of the scale platform facing the recorder, hands at the sides, looking straight ahead, and weight evenly distributed. The weight was then recorded in kilograms to the nearest 0.5 kg.[23] Body mass index (BMI) was calculated by dividing weight (in kilograms) by the square of height (in meters) and expressed in kg/m^2. Cut-off values included underweight (<18 kg/m^2), normal (18–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥30 kg/m^2).

Waist circumference (WC) was measured using a measuring tape (NON 171330, 72″, Medline Industries Inc., Northfield, IL, USA) that is 1 cm in width and made of a material that does not stretch. Each study participant was instructed to gather his or her gown or shirt above the waist, lower the pants and underclothing to slightly below the waist, cross the arms, and place the hands on opposite shoulders. From the right side of each study participant, the waist was located at a level mid-way between the iliac crest and the costal margin along the mid-axillary line, and then the WC was measured to the nearest 0.1 cm at the end of normal expiration. IDF cut-off values of >94 cm (male subjects) and >80 cm (female subjects) were used to define abdominal obesity.[23]

In order to measure the hip circumference, each study participant was asked to stand up with feet together and weight evenly distributed on both feet. The measuring tape was placed around the hip at the level of the greater trochanters and measurement taken to the nearest 0.1 cm. If the greater trochanter was not palpable, the largest horizontal girth around the buttocks was used.[23] Waist–hip ratio (WHR) was calculated by dividing WC (cm) by hip circumference (cm). WHO cut-off values of 0.9 (men)

and 0.85 (women) were used to define abdominal obesity. In addition, waist–height ratio (WHtR) was calculated by dividing WC (cm) by height (cm) and value ≥ 0.5 was defined as obesity for both men and women.[24]

Subsequently, oral glucose tolerance test was performed for each study participant. They were asked to take normal diet with no carbohydrate restriction 72 h prior to test. They then fasted for 8–12 h overnight prior to test day (water was allowed). At 0900 h, blood sample for fasting plasma glucose and fasting insulin levels were drawn from each subject's antecubital vein.

Each study participant was then given a 75-g anhydrous glucose load in 250 mL of water to drink within 5 min. Two hours after the oral glucose load, venous blood from each subject was again drawn for plasma glucose determination using the glucose oxidase method. The WHO criteria was used in the diagnosis of impaired fasting glucose (FPG ≥ 6.1 mmol/L), impaired glucose tolerance (2hrPPG ≥ 7.8 mmol/L), and DM (FPG ≥ 7.0 mmol/L and/or 2hrPPG ≥ 11.1 mmol/L).

Fasting insulin level was determined using insulin Elisa kit (Cortez Diagnostics, 2013 model, ref no 1606-15, Woodland Hills, CA, United States, with detection range of 0–600 U/L, sensitivity 4.6 U/mL) and microplate reader (Bio Infotech, Prem Nagar, Delhi, India, 2013 model at wavelength of 450 nm). HOMA-IR was calculated as thus: HOMA-IR = [fasting plasma insulin (mIU/L) × fasting plasma glucose (mmol/L)] divided by 22.5. A value of <2.2 was taken as normal and >2.2 as insulin resistance.[14]

Statistical analysis

Data were entered into Microsoft excel and then analyzed using Statistical Package for Social Sciences (SPSS) version 19 released by IBM SPSS company in 2011. Results were expressed as means ± standard deviation at 95% confidence interval.

Student's *t*-test was used to compare continuous variables and Chi-square for categorical variables. Pearson's correlation was used to test for association between FPG/2hrPPG, HOMA-IR, BMI, WC, and WHR. Logistic regression was used to assess whether insulin resistance and some obesity indices were significant determinants of glucose intolerance. *P* value was taken as significant when ≤0.05.

Results

This study was carried out to determine the prevalence of insulin resistance and to determine if insulin resistance and some obesity indices are significant determinants of glucose intolerance in a northern Nigerian population. At the beginning of this study, 400 participants were enrolled, out of which 4 respondents were excluded from the analysis due to incomplete biodata and laboratory results. Response rate was 99% with a male:female ratio of 1 to 1.1 and peak age of 31–50 years [Table 1].

Table 1: Distribution of the study participants by age and sex

			Sex		Total
			F	M	
Age range (year)	18-30	Count	39	31	70
		% within age range	55.7	44.3	100.0
		% within sex	18.8	16.4	17.7
		% of Total	9.8	7.8	17.7
	31-40	Count	54	88	142
		% within age range	38.0	62.0	100.0
		% within sex	26.1	46.6	35.9
		% of Total	13.6	22.2	35.9
	41-50	Count	73	43	116
		% within age range	62.9	37.1	100.0
		% within sex	35.3	22.8	29.3
		% of Total	18.4	10.9	29.3
	51-60	Count	28	24	52
		% within age range	53.8	46.2	100.0
		% within sex	13.5	12.7	13.1
		% of Total	7.1	6.1	13.1
	61-70	Count	13	3	16
		% within age range	81.3	18.8	100.0
		% within sex	6.3	1.6	4.0
		% of Total	3.3	0.8	4.0
Total		Count	207	189	396
		% within age range	52.3	47.7	100.0
		% within sex	100.0	100.0	100.0
		% of Total	52.3	47.7	100.0

F: Female, M: Male

The mean age of the study participants was found to be 40.4 ± 10.4 years with mean BMI of 27.0 ± 5.9 kg/m^2. The mean WC, WHR, and WHtR were 87.9 ± 12.3 cm, 0.93 ± 0.16, and 0.56 ± 0.09, respectively, whereas the mean FPG and 2hrPPG were 5.17 ± 2.1 and 6.51 ± 3.41 mmol/L, respectively. In addition, the mean value of HOMA-IR was 3.62 ± 1.57 unit [Table 2].

Using the BMI criterion, 244 subjects (61.6%) were found to have obesity, out of which female and male subjects constituted 132 (33.3%) and 112 (28.3%), respectively. WC criterion showed a total of 215 (54.3%) obese subjects [157 females (39.6%) and 58 males (14.6%)]. Analysis of WHR revealed 223 subjects (56.3%) as obese with female and male subjects constituting 113 (28.5%) and 110 (27.8%), respectively, whereas WHtR data showed 281 (71.0%) obese subjects [156 females (39.4%) and 125 males (31.6%)]. Though all obesity indices showed higher number of obese female than male subjects, WC and WHtR revealed significant sex difference [Table 3].

The insulin resistance–age curve shows a logarithmic increase in HOMA-IR with age [Figure 1].

The prevalence of glucose intolerance (prediabetes 17.2% and diabetes 9.3%) was 26.5% and that of insulin resistance was 36.6% out of which 20.2% was reported for the females and 16.4% for the males [Table 4].

Table 2: Mean values of clinical and laboratory parameters of the study participants by sex

Variables	Females			Males			Total		
	n	Mean	SD	*n*	Mean	SD	*n*	Mean	SD
Age (year)	207	41.2560	11.16936	189	39.5397	9.41393	396	40.4369	10.39126
BMI	207	27.3328	5.92744	189	26.7744	5.86558	396	27.0663	5.89715
WC	207	88.0870	11.63264	189	87.7201	12.98479	396	87.9119	12.28219
WHR	207	0.8978	0.14649	189	0.9709	0.15862	396	0.9327	0.15653
WHtR	207	0.5635	0.08583	189	0.5511	0.09860	396	0.5576	0.09223
FPG	207	5.2198	2.26978	189	5.1175	2.09779	396	5.1710	2.18722
2HrPPG	207	6.6019	3.61612	189	6.4169	3.17938	396	6.5136	3.41163
HOMA-IR	207	2.5636	3.36471	189	4.7769	22.35994	396	3.6199	15.65530

n: Number of subjects, SD: Standard deviation, BMI: Body mass index, WC: Waist circumference, WHR: Waist–hip ratio, WHtR: Waist–height ratio, HOMA-IR: Homeostasis model assessment of insulin resistance, FPG: Fasting plasma glucose, 2HrPPG: Two-hour post-prandial plasma glucose

Table 3: Distribution of obesity among the study participants by sex

		BMI			WC			WHR			WHtR		
		F	M	Total	F	M	Total	F	M	Total	F	M	Total
Normal	No	12	10	22	50	131	181	94	79	173	51	64	115
	% of Total	3.0	2.5	5.6	12.6	33.1	45.7	23.7	19.9	43.6	12.9	16.2	29.1
Obese	No	132	112	244	157	58	215	113	110	223	156	125	281
	% of Total	33.3	28.3	61.6	39.6	14.6	54.3	28.5	27.8	56.3	39.4	31.6	71.0
Overweight	No	63	67	130									
	% of Total	15.9	16.9	32.8									

BMI: Body mass index, WC: Waist circumference, WHR: Waist–hip ratio, WHtR: Waist–height ratio, F: Female, M: Male, No: Number of participants

Table 4: Distribution of insulin resistance among the study participants by sex

			HOMA-IR		Total
			Normal	Resistant	
Sex	F	Count	127	80	207
		% within sex	61.4	38.6	100.0
		% of Total	32.1	20.2	52.3
	M	Count	124	65	189
		% within sex	65.6	34.4	100.0
		% of Total	31.3	16.4	47.7
Total		Count	251	145	396
		% of Total	63.4	36.6	100.0

HOMA-IR: Homeostasis model assessment of insulin resistance, F: Female, M: Male, normal: No insulin resistance, resistant: Presence of insulin resistance

There were significant correlations between HOMA-IR and BMI ($P < 0.001$), WC ($P < 0.001$), WHR ($P = 0.001$), WHtR ($P < 0.001$), FPG ($P < 0.001$), and2hrPPG ($P = 0.001$) [Table 5].

Following logistic regression analysis, significant determinants of glucose intolerance were BMI ($P = 0.017$), WC ($P = 0.001$), WHR ($P < 0.001$), WHtR ($P < 0,001$), and HOMA-IR ($P < 0.001$) [Table 6].

Following sex-specific logistic regression analysis, significant determinants of glucose intolerance among male subjects were BMI ($P = 0.013$), WHR ($P = 0.048$), WHtR ($P = 0.002$), and HOMA-IR ($P < 0.001$), whereas

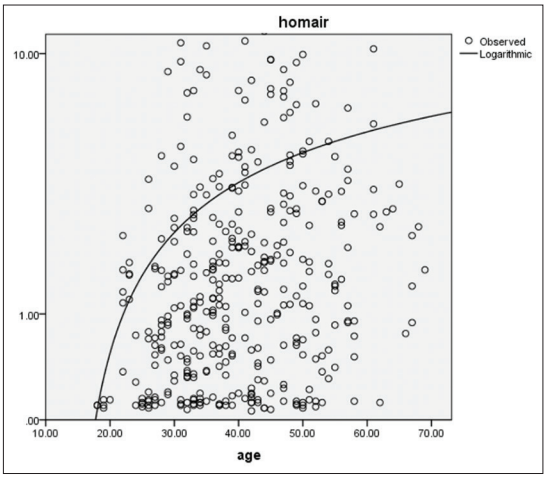

Figure 1: The logarithmic trend of HOMA-IR with age. HOMA-IR = homeostasis model of insulin resistance, age (in years)

WC ($P = 0.493$) was not a significant independent determinant of glucose intolerance among the male subjects in this study [Table 7].

Following sex-specific logistic regression analysis, significant determinants of glucose intolerance among female subjects were BMI ($P = 0.001$), WC ($P = 0.006$), WHtR ($P < 0.001$), and HOMA-IR ($P < 0.001$), whereas WHR ($P = 0.749$) was not a significant independent determinant of glucose intolerance among the female subjects in this study [Table 8].

Table 5: Pearson's correlation between HOMA-IR and other variables

Variables	BMI		WC		WHR		WHtR		FPG		2hrPPG		HOMA-IR	
	R	*P*	*R*	*P*	*R*	*P*	*R*	*P*	*R*	*P*	*R*	*P*	*R*	*P*
BMI	1		0.291	<.001	0.257	<.001	0.412	<.001	0.212	0.003	0.188	0.008	0.422	<.001
WC	0.291	<.001	1		0.780	<.001	0.914	<.001	0.195	<.001	0.189	<.001	0.512	<.001
WHR	0.257	<.001	0.780	<.001	1		0.699	<.001	0.304	0.004	0.252	0.007	0.383	0.001
WHtR	0.412	<.001	0.914	<.001	0.699	<.001	1		0.380	<.001	0.326	<.001	0.391	<.001
FPG	0.212	0.003	0.195	0.006	0.304	0.004	0.380	<.001	1		0.942	<.001	0.405	<.001
2HPG	0.188	0.008	0.189	0.008	0.252	0.007	0.326	<.001	0.942	<.001	1		0.371	0.001
HOMA-IR	0.422	<.001	0.512	<.001	0.383	0.001	0.391	<.001	0.405	<.001	0.371	0.001	1	

R: Pearson's correlation; BMI: Body mass index; WC: Waist circumference; WHR: Waist–hip ratio; WHtR: Waist–height ratio; FPG: Fasting plasma glucose; 2hrPPG: Two-hour post-prandial plasma glucose; HOMA-IR: Homeostasis model assessment of insulin resistance

Table 6: Regression analysis of the determinants of glucose intolerance

	B	SE	Wald	df	*P*	Exp(B)
BMI	0.079	0.033	5.716	1	0.017	1.924
WC	0.066	0.020	11.018	1	0.001	1.936
WHR	15.380	2.717	32.040	1	0.000	1.000
WHtR	20.595	6.167	36.152	1	0.000	1.972
HOMA-IR	1.404	0.318	19.503	1	0.000	1.245
CONSTANT	28.612	3.473	67.876	1	0.000	2.66E12

BMI: Body mass index; WC: Waist circumference; WHR: Waist–hip ratio; WHtR: Waist–height ratio; HOMA-IR: Homeostatic model assessment of insulin resistance; B: Regression coefficient; SE: Standard error; Wald: Statistics type used; df: Degree of freedom; *P*: Significant value, taken as ≤0.05, Exp (B): Odds ratio

Discussion

The prevalence of insulin resistance was 36.6%, whereas that of glucose intolerance (prediabetes 17.2% and diabetes 9.3%) was 26.5%. This prevalence of insulin resistance is slightly lower than the 45.3% reported in a Southeast Nigerian population by Young *et al.*[7] and the 46.3% prevalence reported in a Chinese population by Yin *et al.*[8] The higher prevalence of insulin resistance in southeast Nigeria may be adduced to the fact that the area of the study was more urban than the index area of the study; thus, more western lifestyle practices such as consumption of high fat and high calorie fast foods, smoking, sedentary lifestyles, and obesity were more prevalent. This cause–effect relationship between obesity and insulin resistance was supported by the strong significant correlation between insulin resistance and obesity indices such as BMI, WC, WHR, and WHtR found in the index study.

The higher prevalence of insulin resistance among the female subjects may be explained by their higher obesity (WC, WHtR, BMI, and WHR criteria) compared with that of the male subjects. Another factor that may contribute to the higher prevalence of insulin resistance among the females is the higher mean age and age distribution of the females compared with the males [Tables 1 and 2] since a positive trend of HOMA-IR with age was shown in Figure 1.

Additionally, most women in this community are mainly housewives who live sedentary lifestyle and thus prone to obesity which is a major factor in the development of insulin resistance. This was supported by Bakari *et al.*[4] in a report in Northwest Nigeria.

Furthermore, the fact that the prevalence of insulin resistance is higher than that of glucose intolerance shows that some of the subjects are already insulin resistant but yet to develop glucose intolerance. This can be explained by the fact that during the early period of insulin resistance, hyperinsulinemia helps to prevent hyperglycemia as a compensatory mechanism. However, as the beta-cell secretory response begins to decline, glucose intolerance manifests. This underscores the fact that insulin resistance is a major pathogenetic factor in the development of glucose intolerance.[1-6]

Logistic regression analysis showed that BMI, WC, WHR, WHtR, and insulin resistance were significant determinants of glucose intolerance. This cause–effect relationship between obesity indices and glucose intolerance has been espoused in various studies including that of Kahn *et al.*,[3] Shaban *et al.*,[16] and Peplies *et al.*[17] However, sex-specific regression analysis showed similar result except that WHR was a better determinant of glucose intolerance than WC among the male subjects, whereas WC was a better determinant among the female subjects. This may be explained by the higher hip circumference of the women thus making their WHR lower and thus unreflective of the magnitude of central obesity among them. However, WHtR was a significant determinant of glucose intolerance among the male and female subjects alike underscoring the fact that WHtR was a better index of obesity compared with WC and WHR.

Insulin resistance as a strong determinant of glucose intolerance was supported by Bakari *et al.*,[4] Gezawa *et al.*,[5] and Awofisoye *et al.*[6] in different segments of the Nigerian population. Insulin resistance was also implicated by several other studies as the predominant defect among obese patients with or without type 2 DM.[7-13] The increasing implication of insulin resistance as a pathogenetic factor underlying type 2 DM among Africans is not unconnected with the urbanization of rural Africa. This includes increasing adoption of western lifestyle, viz., consumption

Table 7: Regression analysis of the determinants of glucose intolerance among male participants

	B	SE	Wald	df	*P*	Exp (B)	95% CI for EXP (B)	
							Lower	Upper
BMI	0.104	0.042	6.174	1	0.013	1.901	1.830	1.978
WC	0.020	0.030	0.470	1	0.493	1.080	0.924	1.039
WHR	2.702	2.337	1.336	1	0.048	1.967	1.001	6.550
WHtR	3.102	1.992	4.651	1	0.002	3.176	1.862	11.053
HOMA-IR	3.303	0.509	42.042	1	0.000	27.195	10.020	73.809
Constant	6.829	1.852	13.597	1	0.000	924.428		

BMI: Body mass index, WC: Waist circumference, WHR: Waist–hip ratio, WHtR: Waist–height ratio, HOMA-IR: Homeostatic model assessment of insulin resistance, B: Regression coefficient, SE: Standard error, Wald: Statistics type used, df: Degree of freedom, *P*: Significant value, taken as ≤0.05, Exp (B): Odds ratio

Table 8: Regression analysis of the determinants of glucose intolerance among female participants

	B	S.E.	Wald	df	*P*	Exp (B)	95% CI for EXP (B)	
							Lower	Upper
BMI	0.162	0.048	11.523	1	0.001	1.850	1.775	1.934
WC	0.079	0.029	7.616	1	0.006	1.924	1.873	1.977
WHR	0.675	2.105	0.103	1	0.749	1.263	0.032	1.647
WHtR	1.364	0.652	16.613	1	0.000	3.173	2.013	6.293
HOMA-IR	3.334	0.556	35.995	1	0.000	28.051	9.439	83.361
Constant	10.739	2.251	22.765	1	0.000	46103.170		

BMI: Body mass index, WC: Waist circumference, WHR: Waist–hip ratio, WHtR: Waist–height ratio, HOMA-IR: Homeostatic model assessment of insulin resistance, B: Regression coefficient, SE: Standard error, Wald: Statistics type used, df: Degree of freedom, *P*: Significant value, taken as ≤0.05, Exp (B): Odds ratio

of high energy, refined fast foods/drinks, sedentary lifestyle, smoking, etc.[1-3,6-12] Limitations of this study included not assaying for plasma lipids which could also affect the insulin resistance. A larger sample size would also have produced better results.

Conclusion

From our study, the prevalence of insulin resistance was high and it was present in those with or without glucose intolerance. Indices of obesity and insulin resistance were found to be significant determinants of glucose intolerance in the studied population, though WHtR was found to be a better determinant of glucose intolerance compared with other obesity indices in this study.

Although the demographic characteristics of Northern Nigerian populations are similar, the cross-sectional design of the study makes generalization of the findings difficult. Finally, further research is required in the efficacy of targeting insulin resistance irrespective of glycemic status by therapeutic lifestyle measures and even pharmacological agents in order to reduce the number of people that will eventually develop type 2 diabetes.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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