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Altered Phenotypic Parameters, Obesity Risk Factors and Co-Morbidities in a Population of Southwestern Nigeria

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ABSTRACT

Background: Obesity is a disease with multifactorial etiology with co-morbidities occurring in epidemic proportions in Nigeria, thereby constituting significant economic and health burdens. This study investigates altered biophysical parameters and likelihood model fits for predicting the risk of co-morbidities in obese and non–obese subjects in Southwest Nigeria.

Methods: Three thousand subjects (1500 obese and 1500 non-obese) aged 18 years and above were recruited from Osun and Oyo States, Nigeria communities. Blood samples were collected into appropriate bottles for biochemical analyses using standard procedures. Phenotypic parameters (height, weight, BMI, waist and hip circumferences) were also assessed. Multivariate regression analyses and statistical models were designed to determine the association between obesity risk factors and co-morbidities.

Results: Mean ages of obese (60.4± 13.8years) and non-obese (58.6± 15.7years) subjects were significantly different. Obesity showed a male preponderance. Mean BMI, waist-hip ratio, and waist and hip circumferences were higher ($p<0.05$) among obese subjects but increased with age in both groups ($P<0.05$). Mean triglyceride, fasting glucose levels, arterial blood pressure, pulse pressure, and mean heart rate increased in the obese group compared to the non-obese group. Adjusted Odds of Obesity (WHO) showed that stress, family history of cardiovascular disease, and diabetes were significantly associated with obesity.

Conclusion: Increasing age, male gender, and hypertension were all significantly associated with the development of obesity among Nigerians of Southwestern extraction.

Keywords: Obesity, Hypertension, Comorbidities, Nigerian.

1.0 INTRODUCTION

Obesity, characterized by an increase in body weight resulting in excessive fat accumulation, is a major underlying factor in the pathogenesis of several diseases. Obesity can cause increased fatty acid deposition in the myocardium, causing ventricular dysfunction. [1, 2, 3]. Several works of literature have emerged showing that overweight and obesity are major causes of co-morbidities such as type 2 diabetes mellitus, cardiovascular disease, and various cancers, thereby increasing their prevalence and mortality [4, 5, 6, 7].

It was recently documented that over 1.9 billion adults, 18 years and older, were overweight; of these, over 650 million were obese, their prevalence varying with geographical location and lifestyle. Obesity has an enormous healthcare burden exceeding 700 billion USD annually [8].

Obesity is often associated with chronic increases in pro-inflammatory cytokines and C-reactive protein [9, 10]. Activating the innate immune system in adipose tissue promotes pro-inflammatory status and oxidative stress, triggering a systemic acute-phase response. It was hypothesized that inflammation of adipose tissue in obese patients plays a critical role in the pathogenesis of obesity -related complications [11, 12].

Obesity and its comorbidities have a devastating effect on vascular function, promoting conditions that favor cardiovascular diseases via mechanisms including ectopic lipid deposition, hyperglycemia, and developing a procoagulant state [13]. A 10% reduction in body weight was observed to reduce the occurrence of clinical diabetes mellitus in obese individuals [7, 10].

It has been established that modifiable risk factors such as cardiovascular disease, diet, lifestyle, physical activity, and stress contribute significantly to the occurrence of obesity [5, 6]; this study, therefore, characterizes the presentation as well as the association of anthropometric and phenotypic parameters with other comorbidities in obese and non – obese subjects in Oyo and Osun States Nigeria to give further insight into alleviating obesity risk and associated complications.

2.0 METHODOLOGY

2.1 Study Population

This is a community-based case-control study spanning a period from 2015 to 2018. A multistage sampling technique was employed in the selection of participants. The sample size was determined using QUANTO version 1.2.4 software. A sample size of 3000 subjects was sufficient to estimate the population prevalence of diabetes with a 95% confidence interval (CI) of less than 1%.

A total of 3000 subjects made up of 1500 obese and 1500 non-obese individuals were recruited from communities in Osun and Oyo States through the Community Medicine Department of LAUTECH Teaching Hospitals (Osogbo and Ogbomoso), Osun State Hospital Management Board, Asubiaro, Osun State and Bowen University Teaching Hospital, Ogbomoso, Oyo State. Obesity was defined as BMI≥30kg/m2 [8].

All participants were interviewed upon enrollment to obtain their demographic information with a questionnaire. Both groups underwent health examinations to confirm obesity. The Ethical Committees of LAUTECH Teaching Hospital, Ogbomoso (Ref: LTH/OGB/EC/2015/083) and LAUTECH College of Health Sciences, Osogbo (Ref: LAU/CHS/DEAN/ETHICAL/.052) approved the study protocol. Informed consent was sought and obtained from all participants.

2.2 Blood collection

The skin at the site of blood collection was disinfected using alcohol swaps. 5 mL Overnight fasting venous blood was aliquoted into fluoride oxalate and plain bottles for fasting blood glucose estimation and lipid analysis, respectively. A 2.5 mL blood sample was after that drawn for 2 hours postprandial. The enzymatic method was used to determine blood glucose levels, while serum total cholesterol and triglyceride were determined by enzymatic hydrolysis and oxidation, as previously described [14].

2.3 Anthropometric Measurements

All measurements were made by trained personnel as previously described with modifications [15]. Briefly, blood pressure (BP) was measured twice to the nearest 2mmHg with a mercury sphygmomanometer. Two readings were taken on each subject's left and right arms in a sitting position after a 5-min rest between readings, and the average of systolic and diastolic BPs was determined. Arterial pulse pressure was estimated by subtracting the resting diastolic from systolic BP values. The weighing scale was standardized, and weight was measured based on internationally acceptable standards. Weighing scales

were calibrated each day before use.

The weight of each subject was measured using HANA mechanical bathroom scale and recorded to the nearest kilogram. Height was measured using a meter rule with the individual standing erect with heels, buttocks, upper back, and occipital against the meter rule and recorded to the nearest 1cm. BMI was computed using the standard formula: weight(kg)/height(m^2)

2.4 Statistical Analysis

Statistical analyses were performed using SAS (version 9.4) and R statistical programs (version 3.4.2). Frequencies were presented as percentages, continuous variables as Mean ± SD. Analysis of variance was used to compare groups, χ^2 test to examine differences in age and other variables among the subjects.

3.0 RESULTS

Of the 3,000 subjects enrolled in the study, 76.8% had co-morbidities. Obese individuals were older than non-obese (60.4± 13.8 vs. 58.6± 15.7 years). Obesity had a male preponderance (50.4%). The proportion of obese subjects with co-morbidities -hypertension, dyslipidemia, and diabetes - was significantly higher. The frequency of stress, physical inactivity, and tobacco use in the last twelve months was higher among obese subjects. (Table 1).

Table 2 showed that mean BMI, waist-hip ratio, waist, and hip circumferences were higher ($p<0.05$) among obese subjects than non-obese. In addition, a higher percentage of subjects with obesity had SBP> 140mmHg, raised mean triglyceride, fasting glucose levels, arterial blood pressure, pulse pressure, and mean heart rate compared to the non-obese group.

A positive family history of hypertension, diabetes, cardiovascular diseases, and stroke was more likely among obese individuals than the non-obese group ($p>0.05$). The consumption of green leafy vegetables, sugar, and meat was similar in the two groups (Table 3).

3.1 Adjusted Odds of Obesity

Table 4 shows the factors associated with Obesity based on the WHO definition. Model 0 represents the association with demographic characteristics in which patients with age > 50 years and average monthly income >100 USD per month were more likely to have obesity. Males were more likely to have obesity compared to females.

Table 1. Demographic and Clinical Characteristics of all Study Participants

Characteristics		Non Obese (n=1500)	Obese (n=1500)	p-value
Age (years)	<50	390 (26.8)	314 (20.9)	<0.001
	>50	1098 (73.2)	1186 (79.1)	
		58.6±15.7	60.4±13.8	<0.001
Sex	Male	1137 (75.8)	756 (50.4)	<0.001
	Female	363 (24.3)	744 (49.6)	
Education	No education	248 (16.5)	294 (19.6)	0.035
	Some education	1252 (83.5)	1206 (80.4)	
Income	≤ 100USD	723 (48.2)	678 (45.2)	0.11
	>100USD	777 (51.8)	822 (54.8)	
Cardio-vascular disease risk factors	Hypertension (yes)	1344 (89.6)	1465 (97.7)	<0.001
	Dyslipidemia (yes)	533 (35.5)	1322 (88.1)	<0.001
	Diabetes (yes)	290 (19.3)	651 (43.4)	<0.001
	Cardiac Disease (yes)	161(10.7)	177 (11.8)	0.391
	Depression (yes)	110 (7.3)	108 (7.2)	0.719
	Stress (yes)	233 (15.5)	305 (20.3)	0.002
	Physical inactivity (yes)	1451 (96.7)	1419 (94.6)	0.014
	Tobacco Use (past 12 months)	75 (5.4)	39 (2.6)	<0.001

Co-morbidities were added to Model 1and the results showed that the demographic variables retained their influence. Stress, family history of cardiovascular disease, and diabetes were significantly associated with Obesity. Model 2 controlled for demographic, comorbidities, and lifestyle characteristics. All previous parameters retained their effect as model 2, including an enhanced effect of the presence of cardiac disease. Meat consumption was significantly associated with Obesity. The area under the curve receiver operative characteristics (ROC) (AUCROC), which signifies the predictive power of the models 0, 1, and 2, were 0.66, 0.67, and 0.69, respectively.

3.2 Socio-demographic Model

Another set of models were fitted for Obesity according to WHO definition (Table 5). The socio-demographic model (Model 0) showed that age>50, income >100 USD

Table 2. Anthropometric and Biochemical Parameters of all Subjects

Parameters		Non-Obese (n=1500)	Obese (n=1500)	p-value
BMI		25.3±4.7	27.2±5.1	<0.001
Waist circumference		79.4±8.5	92.1±14.2	<0.001
Hip circumference		85.6±10.1	97.4±14.3	<0.001
Waist-to-hip ratio	Normal	196 (25.4)	432 (14.8)	<0.001
	Raised	576 (74.6)	2484 (85.2)	
Systolic BP (mmHg)		0.9±0.1	1.0±0.1	<0.001
	≤140	337 (39.1)	775 (25.5)	<0.001
	>140	524 (60.9)	2268 (74.5)	
Diastolic BP (mmHg)		152.6±33.3	160.2±30.0	<0.001
	≤90	369 (42.9)	1047 (34.4)	<0.001
	>90	492 (57.1)	1996 (65.6)	
Mean arterial Pressure		98.7±17.1	95.7±17.7	0.345
Pulse pressure		86.7±11.1	96.2±22.2	<0.001
Heart rate		53.7±17.9	64.2±19.9	<0.001
Total Cholesterol (mg/dl)		86.0±19.1	88.7±18.7	<0.001
	<200	369 (61.1)	1590 (58.4)	0.23
	≥200	235 (38.9)	1131 (41.6)	
Triglyceride (mg/dl)		191.5±51.6	190.7±58.7	0.758
	<150	581 (96.0)	1915 (70.2)	<0.001
	≥150	24 (4.0)	812 (29.8)	
FBG (mg/dl)		95.1±48.1	133.2±88.6	<0.001
	Normal;	206 (97.6)	934 (69.7)	<0.001
	High;	5 (2.4)	406 (30.3)	<0.001
		88.2±15.2	124.0±51.1	<0.001

Table 4. Multivariate Regression Analysis for the Association between Obesity and its Risk Factors.

	Adjusted OR (95%CI)		
	Model 0	Model1	Model 2
Socio-demographic			
Age; ≥ 50	1.41 (1.18, 1.68)	1.48 (1.23, 1.79)	1.46 (1.19,1.80)
Male gender	3.37 (2.83, 4.00)	3.56 (2.95,4.28)	4.06 (3.28,5.03)
Income; ≥100USD	1.42 (1.21, 1.66)	1.40 (1.18,1.65)	1.42 (1.17,1.71)
Co-morbidities			
CVD; Yes		1.04 (0.80,1.36)	1.29 (0.94,1.75)
Stress; Yes		1.39 (1.11,1.74)	1.46 (1.14,1.87)
Family history of CVD		1.10 (0.89,1.36)	1.01 (0.80,1.28)
Family history diabetes		1.14 (0.85,1.36)	1.20 (0.88,1.64)
Lifestyle/ Behavioral			
Physical activity; (Yes)			0.71 (0.44,1.16)
Tobacco; (Yes)			0.74 (0.48,1.16)
Salt intake; Some			0.73 (0.53,1.00)
Green vegetables; Some			0.96 (0.78,1.18)
Sugar consumption; Some			1.00 (0.82, 1.23)
Meat consumption; Some			1.11 (0.90, 1.38)
AUC (95% CI)	0.66 (0.64, 0.68)	0.67 (0.65, 0.70)	0.69 (0.67, 0.72)

Table 3. Lifestyle Characteristics by Obesity Status

Parameters		Non-Obese	Obese	p-value
Family history	CVD (yes)	302 (34.6)	1143 (37.4)	0.119
	HTN (yes)	274 (29.5)	1023 (33.3)	0.03
	DM (yes)	96 (10.3)	409 (13.3)	0.017
	Stroke (yes)	133 (15.2)	446 (14.6)	0.654
Salt intake	Never	767 (91.4)	2751 (93.4)	0.043
	Very Often	72 (8.6)	193 (6.6)	
Green leafy vegetable	None	225 (28.8)	774 (27.4)	0.429
	Some	556 (71.2)	2053 (72.6)	
Sugar Consumption	None	536 (70.5)	1973 (70.9)	0.832
	Some	224 (29.5)	809 (29.1)	
Meat consumption	None	197 (24.9)	713 (25.0)	0.945
	Some	594 (75.1)	2136 (75.0)	

CVD- cardiovascular disease, HTN- hypertension, DM– *Diabetes mellitus*

and male gender were positively associated with Obesity. In Model 1, all of the factors remained statistically significant, as well as comorbidities such as cardiac diseases, stress, cardiovascular disease and family history of diabetes mellitus. In the final model, the factors associated with obesity were in model 2, including meat consumption. The AUCROC for the respective models were: model 0 (0.66), model 1 (0.67) and model 2 (0.69).

3.3 Adjusted Odds for Comorbidity

Table 5 shows that in model 0, the presence of Obesity was one of the most important associated factors with comorbidities followed by age >50 years old. In model 1, comorbidities were introduced, and the presence of obesity as diagnosed by WHO criteria and age >50 years retained its positive linear associations though not statistically significant. Obesity severity profile, especially when moderate and severe, has significant associations with comorbidity. In model 2, lifestyle and behavioral factors were introduced, with most of the former associations maintained. The AUCROC for the respective models were: model 0 (1.00), model 1(0.62) and model 2 (0.58).

In model 2, when lifestyle and behavioral factors were introduced, all the previous associations were maintained while additionally, physical inactivity was significantly associated with one-month fatality among stroke patients. The AUCROC for the respective models were: model 0 (1.00), model 1 (0.56) and model 2 (0.53).

Table 5. Multivariate Regression Analysis for the Association of Obesity with Phenotypic Parameters and other risk Factors

	Adjusted OR (95%CI)		
Risk factors	Model 0	Model 1	Model 2
Socio-			
Age; ≥ 50	1.22 (1.03,1.45)	1.27 (1.06,1.52)	1.25 (1.03,1.52)
Male gender	3.38 (2.92,3.91)	3.30 (2.84,3.85)	3.58 (3.02,4.23)
Income; ≥100USD	1.36 (1.18,1.57)	1.33 (1.14,1.55)	1.30 (1.10,1.54)
Co-morbidities			
CVD (Yes)		1.27	1.26
Stress (Yes)		1.48	1.48
Family history of CVD		1.22 (1.01,1.48)	1.21 (0.99,1.49)
Family history diabetes		1.34 (1.05,1.72)	1.38 (1.06,1.78)
Lifestyle/			
Physical inactivity			1.57
Tobacco (Yes)			1.00
Salt intake; Some			0.98
Green vegetables;			1.22
Sugar consumption; Some			0.96 (0.80,1.16)
Meat consump-			0.89
AUC (95% CI)	0.66 (0.64,0.68)	0.67 (0.65,0.69)	0.69 (0.67,0.71)

4.0 DISCUSSION

The incidence of overweight and obesity is rising rapidly in Africa in tandem with the demographic change in the population. Similar results were obtained for the association of phenotypic and atherogenic parameters with oxidative stress relative to overweight globally [5, 16]. Age remains the strongest irreversible risk factor for stroke, while modifiable factors including vascular disease, diet, lifestyle, physical activity, and stress, also contribute significantly to the burden of obesity, CVD, and diabetes in Africa [17, 18]. The strongest link to modifiable risk factors for stroke is hypertension. Additionally, stroke is attributed to cerebral small vessel diseases [19, 20]. The contribution of obesity risk factors is multiplicative, probably with genetic predisposition, and contributes to the high burden of stroke and its relatively poor prognosis in Africa [21, 22].

A previous work [23] reported that the highest prevalence (29.3%) of overweight and obesity was observed in the Southwestern population of Nigeria compared with other geopolitical zones. The high prevalence of obesity (31%) recorded among participants in this study adds credence to the assertion of multiplicative effects suggesting that obese subjects were at high risk of developing cardiovascular diseases, hypertension, diabetes, and stroke. This may highlight the rapid epidemiological transition underpinning the increased burden of cardiovascular disease. The burden of CVD in Africa further complicated the already endemic communicable diseases responsible for significant mortalities and morbidities across the continent [24, 25].

Some studies have shown associations between the general population's vascular risk factors such as hypertension, diabetes, dyslipidemia, heart disease, obesity, atrial fibrillation, smoking, and stroke [26, 27]. Increased blood levels of C-reactive protein, carotid intima-media thickness, obesity, older age, and gender are risk factors that independently increase cardiovascular disease,

stroke, and diabetes mellitus. Still, the outcome is multiplicative if they are present in clusters rather than as individual risks [21,22,27]. The role of inflammation and arterial stiffness has been demonstrated in hypertension, diabetes, acute ischemic stroke, and patients with obesity [26, 27, 28].

The risks of obesity and diabetes with increasing age and its strong association with the male gender were demonstrated in this study. A similar result has been documented from a study involving African American youths [22], contrasting another that revealed a significant association with the female gender [29]. The mechanism leading to a greater risk for obesity in men in this study may be linked to a higher prevalence of clusters of cardiovascular risk factors, stress, smoking, and alcohol intake. It could also be related to genetic associations and hormonal profiles [26, 27].

The clinical characteristics associated with obesity were reflected in the pattern of cardiovascular risk factors among obese participants with significantly higher body mass index, waist, and hip circumferences, waist-hip ratio, mean arterial blood pressure, and pulse pressure compared to non-obese. This could be attributed to increased oxidative stress, as hypothesized in some studies [27, 28]. In addition, a significant proportion of the obese subjects reported high-stress levels, meat consumption, and physical inactivity. These could be possible targets for intervention in primary and secondary stroke prevention [28]. Obese subjects in this study manifested higher family history of CVD and diabetes mellitus than those without obesity, suggesting the genetic linkage of obesity, thus accounting for its linear relationship with hypertension and stroke. This observation is supported by other findings where severe forms of obesity were linked to genetics with polygenic involvement [27, 28].

In the adjusted analysis, male gender, income >100 USD, presence of cardiac diseases, stress, family history of diabetes mellitus, and cardiovascular disease were significantly associated with increased odds of obesity. This is in line with previous reports [29, 30] that showed increased odds of obesity with hypertension in males as the most important predictive component of obesity. Recently, one study showed that only the hypertensive trait among obesity-related factors was associated with a significantly elevated risk of ischemic stroke [31]. A relatively higher prevalence of obesity among Nigerians than in other countries and regions of the world has been documented, implying a higher fatality rate for comorbidities [16]. It was hypothesized that targeting obesity might effectively prevent early mortality associated with cerebrovascular events [31]. Consistent with what has been described in other settings, our findings suggest that obesity is more prevalent and might predict poor outcomes in patients with hypertension and stroke [31, 32]. Conclusively, this study showed a strong association between obesity, hypertension, and cardiovascular disease risk. Thus some lifestyle and behavioral habits, including regular physical activity, reducing meat consumption, and stress should be encouraged to reduce the burden and mortality resulting from hypertension, diabetes, and stroke in South Western Nigeria, sub-Saharan Africa.

Limitation of the Study

Recruiting incident cases could introduce potential bias when assessing the association between behavioral, environmental, or biological determinants of obesity and comorbidities; however the relatively large sample size reduced such bias to a large extent.

Conflicts of Interest

The authors declare no competing interests.

Authors' Contributions

CAA contributed to data collection and manuscript writing. **ROA, DPO** contributed to data analysis tools. **BGA, MAA** contributed to data collection and analysis of data. **OGA** contributed to manuscript writing. **TAA** conceived and designed the study. All authors approved the final copy of the manuscript.

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