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# Comparison of Lipid Peroxidation and Lipid Profile Among Apparently Healthy Abdominal and Generalized Obese Subjects in Sokoto.

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## Abstract

Globesity is a term that describes the escalating global epidemic of overweight and obesity which appears to be catching up with Sub-Saharan Africa. Obesity has been defined as having a Body Mass Index (BMI) of  $\geq 30$ Kg/m<sup>2</sup>. Several researches have reported increased morbidity and mortality in obese subjects. In the current study lipid profile, lipid peroxidation and anthropometric parameters were determined in sixty apparently healthy nonobese subjects as controls, and abdominal and generalized obese subjects of comparable age and social status. The results showed significant (p<0.05) high levels in MDA, LDL-C, AIX, BMI and hip circumference and lower HDL-C and insignificant (p>0.05) levels in TC and VLDL-C in generalized obesity compared to abdominal obesity. The results further revealed positive correlation of BMI and MDA, TC, TG, LDL-C, VLDL-C and AIX which negatively correlated with HDL-C. Conclusively, results showed increased risks of cardiovascular and oxidative stress complications in generalized obesity compared to abdominal obesity.

*Key words:* BMI, Globesity, lipid profile and lipid peroxidation

## Introduction

Obesity is a worldwide problem of great public health importance affecting many developing and developed nations, and is being defined in terms of Body Mass Index (BMI) Ratio of weight (Kg)/height ( $M^2$ ) of about  $\geq 30$  Kg/m<sup>2</sup> (WHO, 1990). A BMI of  $\leq 25$  is considered normal; 25 to 30 is overweight, and greater than 30, obese. Obesity is indeed life-threatening. It is caused by a complex interplay of environmental and genetic factors (Kumar and Clark, 2012). Environmental factor deals with the availability of food consumption, humidity, alcoholism and smoking. Whereas, genetic contribution is due to defective leptin gene (Greek leptos, "thin") that is produced in adipocytes and moves through the blood to the brain, where it acts on receptors in the hypothalamus to curtail appetite. Phenotypically, symptoms seen are elevated serum cortisol levels; they are unable to stay warm, they grow abnormally, do not reproduce, and exhibit unrestrained appetite (Lehninger et al., 2005). Obesity is the result of taking in more calories in the diet than are expended by the body's energy-consuming activities. In obese individuals, two distinct phenotypes are apparent: generalized obesity and abdominal obesity. These subgroups have different clinical problems and health risks (Davidson, 2002). Generalized obesity is associated with increased fats deposit across the whole body but mainly on the hips and thighs, depicted as pear-shaped, while, abdominal obesity is recognized by increased waist circumference or waist: hip ratio (apple-shaped). Abdominal obesity is a strong indicator for development of coronary artery disease and associated with insulin resistance, type 2 diabetes mellitus, hypertension, stroke and pro-inflammatory states (Glew and Rosenthal, 2007). The prevalence of this complex of co-morbidities associated with obesity, now referred to as the metabolic syndrome, is reaching epidemic proportion (Grundy et al., 2004; Roth et



*al.*, 2002). The strategies for the management of obesity include: exercise diet monitoring, drugs and surgery (AAFP, 2013).

Lipid peroxidation is a complex process known to occur in both plants and animals. It involves the formation and propagation of lipid radicals, the uptake of oxygen, a rearrangement of the double bonds in unsaturated lipids and the eventual destruction of membrane lipids, producing a variety of breakdown products, including alcohols, ketones, aldehydes, and ethers (El-Beltagi and Mohamed, 2013). Plasma lipid profile is very useful for assessing the health of an individual. The total plasma lipid level is 400-600mg/dl. Out of this, 40% is cholesterol; 30% is phospholipids; 20% is triglycerides. Being hydrophobic in nature, they are always in combination of proteins as carriers (lipoproteins) (Satyanarayana, 2010).

The aim of this study was to compare lipid peroxidation and lipid profile in apparently healthy abdominal and generalized obese individuals in Sokoto.

## Materials and Methods Study Site

The study area selected for this research was Sokoto metropolis and its environs located Northwestern Nigeria. It shares borders with Niger-Republic to the North, Kebbi State to the South-West and Zamfara State to the East (SSBD, 2007). The city lies between longitude 05°42`` to 22`` East and latitude 12° 15 to 29`` North and covers an area of 60.33km (Collins Maps, 2017).

## Participants

Participants for this case-control study included 180 individuals made up of 60 subjects each were used as abdominal obese and generalized obese while 60 non-obese subjects were used as controls. Each individual's weight, height, waist and hip circumference were measured. Written informed consent was sought for and obtained from all participants. Ethical Committee approval was also obtained from the Ethical Review Committee of Sokoto State Ministry of Hospital.

## **Blood Samples**

A total volume of five milliliters (5mL) of blood samples from each subject was taken intravenously, 3 mL was delivered into clean dry tubes and allowed to clot at room temperature. The samples were centrifuged at 3000 rpm for 5 minutes using a bench top centrifuge and the serum harvested and kept in a labeled sample bottles at -20°C until required for analysis. The remaining 2 mL of blood were put into fluoride oxalate containers for analysis.

## **Chemicals and Reagents**

Analytical grade chemicals and reagents were used for this research. Reagent kit for the assay of fasting blood glucose and lipid profile assays were purchased from Spectrum Diagnostics, Cairo, Egypt.

## **Statistical Analysis**

The data obtained was analyzed using Microsoft Office Excel 2007 and SPSS Statistical Software Version 23. The results were expressed as mean  $\pm$  SD. Group comparisons were carried out using Analysis of Variance (ANOVA) Test, and p-value of equal to or less than 0.05 was considered as significant.

## Results

Table 1.0 shows the baseline characteristics of apparently healthy obese subjects. Fasting plasma glucose and blood pressure of apparently healthy obese subjects was presented in Table 2.0. There was no significant difference of the analytes among groups. Table 3.0 shows lipid profile and MDA of apparently healthy obese subjects. The result showed significant higher levels in MDA, LDL-C, AIX and lower level in HDL-C and insignificant high (P>0.005) levels in TC and VLDL-C in generalized obesity compared to abdominal obese subjects. BMI and Waist/Hip ratio of apparently healthy obese subjects are presented in Table 4.0. The results showed significant levels in BMI and Hip circumference in generalized obesity compared to abdominal obesity subjects. Table 5.0 showed the correlation coefficient (r) of BMI with lipid profile and MDA. There was weak to moderate positive correlation between obesity and lipid profile parameters except for HDL-Cholesterol which had weak negative coefficient using Pearson regression analysis at p < 0.01. Additionally, lipid peroxidation had a positive weak correlation with obesity.

Variable	Abdominal obesity (n=60)	Generalized obesity(n=60)	Controls (n=60)
Age	40.68±7.19	35.23±11.76	25.57±5.07
Gender			
Male	30	27	43
Female	30	32	17
Tribe			
Hausa	36	36	24
Yoruba	8	6	15
Igbo	6	7	7
Others	10	12	12

Table 1.0: Baseline characteristics of apparently healthy obese subjects

 $n = number of participants; age expressed as mean \pm SD$ 

Variable	Abdominal obesity (n=60)	Generalized obesity (n=60)	Controls (n=60)
FBG (mmol/L)	5.47±0.66a	$5.04\pm0.94a$	5.08±0.56a
SBP (mmHg)	127.12±8.41a	129.57±10.22a	123.98±10.60a
DBP (mmHg)	$82.40 \pm 11.32a$	$84.92 \pm 8.18a$	82.48±8.10a

Data is expressed as mean  $\pm$  SD. FBG= Fasting Blood Glucose, SBP =Systolic Blood Pressure, DBP =Diastolic Blood Pressure. Values bearing the same superscript on row show no significant different (P >0.05) using one way analysis of variance (ANOVA).

Table 3.0: Lipid peroxidation	and lipid profile of	f apparently health	v obese subjects
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Variable	Abdominal obesity	Generalized	Controls (n=60)	
variable	(n=60)	obesity(n=60)	Controls (n=60)	
MDA (µmol/mL)	1.22 ±0.65a	2.74±1.00b	1.85±0.80c	
TC (mmol/L)	4.95±0.85a	5.23±0.77a	4.14±0.77b	
TG (mmol/L)	1.20±0.42a	1.20±0.42a	$0.86 \pm 0.45b$	
HDL-C(mmol/L)	1.89±0.53b	1.58±0.56a	1.91±0.62b	
LDL-C(mmol/L)	2.52±1.14a	3.12±0.97b	1.82±0.95c	
VLDL-C(mmol/L)	0.55±0.18a	0.54±0.19a	$0.41 \pm 0.13a$	
AIX	3.06±1.68a	3.79±1.82b	2.37± 0.91a	

Data expressed as mean  $\pm$  SD. Values bearing different superscripts on the row differ significantly (*P*<0.05), while ones with the same superscripts indicate no significant different (*P*> 0.05) using ANOVA.

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Variable	Abdominal obesity	Generalized obesity	Controls (n=60)	
variable	(n=60)	(n=60)		
$BMI(Kg/m^2)$	$32.21 \pm 4.37a$	$35.47\pm5.44b$	$22.01 \pm 2.42c$	
Waist circ.(cm)	101.32± 9.75a	$102.85 \pm 11.30a$	$75.53\pm6.46b$	
Hip circ.(cm)	$106\pm0.42a$	$116.12 \pm 0.42b$	$93.07\pm8.57c$	
W/H ratio	$0.95\pm0.07a$	$0.88 \pm 0.08 \mathrm{c}$	$0.81\pm0.05a$	

Table 4.0: BMI and Waist-Hip ratio of apparently healthy obese subjects

Data expressed as mean  $\pm$  SD. Values bearing different superscripts on the row differ significantly (*P*<0.05), while ones with the same superscripts indicate no significant different (*P*> 0.05) using ANOVA.

Variable	Coefficient	<b>P-Value</b>	Remark	
MDA	0.193	0.009**	S	
Total cholesterol	0.475	0.000**	S	
Triglyceride	0.269	0.000**	S	
HDL-cholesterol	-0.198	0.008**	S	
LDL-cholesterol	0.437	0.000**	S	
VLDL	0.142	0.057*	S	
AIX	0.339	0.000**	S	

Table 5.0: Correlation Coefficient (r) of BMI with MDA and lipid profile

\*Correlation is significant at the 0.05 level \*\*Correlation is significant at the 0.01 level S= statistically significant NS=insignificant.

## Discussion

From the baseline characteristics of the study population, it was revealed that: age, gender and tribe had no significance with the phenotypes of obesity. This was simply because obesity had genetic predisposition as stated by Lehninger *et al.* (2005) or acquired due to environmental factors as reported by Nwoka *et al.* (2014). While ensuring that only the apparently healthy participants were included, we attempted to determine fasting blood glucose and blood pressure in order to rule out diabetes mellitus and hypertension, respectively. Results obtained confirmed their fitness for study since all values were within normal range.

Moreso, comparing the lipid peroxidation-MDA of the two phenotypes, it indicated significant difference, MDA (p<0.05) of the abdominal obesity compared to generalized obesity. This finding is consistent with previous reports

(Crook, 2012; Sankhla et al., 2012) on metabolic syndrome and obesity. TC, TG and VLDL of both abdominal and generalized obesity were insignificantly different (p>0.05) as previously reported (Davidson, 2002; Hirani et al., 2011). This was simply due to the fact that all excess calories were converted to fatty acid for storage. This contradicted earlier literature and other research papers which stated that abdominal obesity was associated with cardio-metabolic risks higher than generalized obesity. HDL-C of abdominal obesity subjects was higher than that of generalized obesity (p<0.05). However, this was in contrast to previous reports (Thakur et al., 2017; Kaees et al., 2014) which indicated that HDL-C was low in abdominal obesity. LDL-C and AIX of generalized obesity are higher than that of abdominal obesity. The result also differed from the work of previous researches as it was reported that LDL-C is a bad cholesterol that ferries FFA to peripheral tissues. Thus, making an individual more prone to metabolic syndrome. This was however in contrast to the report of Thakur et al. (2017) and Alberti et al. (2009).



Moreover, the BMI of abdominal obesity and that of generalized obesity was significantly different (p<0.05). This finding is consistent with previous report by Sultan et al. (2014) who obtained significant difference between the phenotypes. However, the waist circumference of the result indicated no statistical difference which is contrary to the report of Sultan et al. (2014). Additionally, there was a significant difference in hip circumference between the two phenotypes. This is simply because abdominal obese subjects are having fats at the viscera whereas generalized obese subjects have across the body surface area as stated by Davidson *et al.* (2002). The waist-hip ratio has insignificant difference which is contrary to WHO (1990) classification of obesity and International Diabetes Federation (IDF, 2008).

Furthermore, lipid peroxidation-MDA had a weak positive correlation with BMI. And the value has an increasing constant (0.18)depending on the class of obesity. This is in accordance with research done in London by Lima et al. (2004); Vesilbursa et al. (2005) and Amirkhizr et al. (2008). This could be backed up from physiological perspective as the more surface area and cells the more the wearing and repairs thus generating oxidative free radicals and oxidative stress (Sembulingam and Sembulingam, 2013). Plasma lipids and AIX have weak to moderate correlation with BMI. However, HDL-C has negative correlation. These findings are in harmony with a research conducted on apparently healthy obese civil servants in Abakaliki by Ugwuja et al. (2013). Our finding is also in agreement with previous reports (Kumar and Clark, 2012; Harpers, 2003) on obese subjects.

## Conclusion

In conclusion, this study has shown that there are alterations in lipid profile and lipid peroxidation of the various phenotypes of the obesity. MDA and AIX values increased in generalized obesity. TC, TG and VLDL-C showed insignificant difference in the abdominal and generalized obesity. However, LDL-Chol showed significant increase in generalized obesity compared to abdominal obesity.

## Recommendation

Assessment of lipids profile is highly recommended in obese subjects as this will help to prevent cardio-metabolic risk and other related diseases. It is important to estimate antioxidant enzymes and vitamins so as to checkmate the oxidative stress biomarkers in obese individuals.

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