ORIGINAL ARTICLE

Biomarkers of metabolic syndrome in male cigarette smokers in Calabar, Southern Nigeria

Uwem Okon Akpan and Iya Eze Bassey

ABSTRACT

Background: Metabolic syndrome has recently attracted much attention due to increasing knowledge of its relationship with cardiovascular mortality, morbidity and other conditions.

Objective: This study assessed the biomarkers and frequency of metabolic syndrome in adult male smokers.

Methods: 141 apparently healthy male cigarette smokers and 60 apparently healthy non-smokers aged 18 to 65 years were recruited for the study. The smokers were classified as light, moderate or heavy smokers. Anthropometric indices, blood pressure, fasting plasma glucose, lipid profile and serum insulin were measured. Insulin resistance was calculated using homeostasis model assessment of insulin resistance (HOMA-IR). Data was analysed using SPSS version 20.0; $p<0.05$ was considered statistically significant.

Results: The smokers had significantly higher diastolic blood pressure (p=0.0001), Total cholesterol (p=0.008) and LDL-C (p=0.0001) and significantly lower HDL-C (p = 0.0001), compared to the controls. The frequency of smokers with metabolic syndrome was significantly higher than non-smokers using the Adult Treatment Panel III (p=0.032) criteria with dyslipidemia being the most prevalent metabolic risk factor.

Conclusions: The unfavorable changes in the lipid profile and blood pressure observed in this study may predispose smokers to a higher risk of cardiovascular disease and there is a higher frequency of metabolic syndrome among smokers in Calabar compared to the non-smokers.

Keywords: Metabolic syndrome; cigarette smoking; dyslipidemia.

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INTRODUCTION

Tobacco smoking is one of the leading causes of preventable death in the world today and is associated with high morbidity and mortality. Smoking harms almost every organ or tissue in the body and greatly reduces both the quality of life and life expectancy (1).

Tobacco kills more than 7 million people each year. More than 6 million of those deaths occur as a result of direct tobacco use while around 890,000 deaths result from non-smokers exposure to second-hand smoke. This is expected to rise to more than 8 million annually by 2030 (2). Around 80% of the world's 1.1 billion smokers live in low and middle-income countries, including Nigeria (3). According to the 2008 Nigeria Demographic and Health Survey, less than 1% of women aged 15-49 and 11.5% of men aged 15-49 used tobacco products with those who smoked cigarettes constituting 9% (4). By the year 2018, it is expected that about 8.5% of Nigerian males aged 15 years and above will smoke cigarettes with 7.1% daily smokers (2).

Metabolic syndrome may be defined as a collection of inter-related metabolic risk factors that directly increase the risk of coronary heart disease, other forms of cardiovascular atherosclerotic diseases and type 2 diabetes mellitus (5). The components of metabolic syndrome are obesity, insulin resistance, high blood pressure and dyslipidemia but, with the upsurge of new findings, the list keeps increasing. The components now include hyperinsulinemia, insulin resistance, high blood pressure, central obesity and atherogenic dyslipidemia (increase in LDL-C, plasma triglycerides and decrease in HDL-C), endothelial dysfunction, genetic susceptibility, prothrombotic state, pro-inflammatory state and chronic stress (6), but, the diagnosis and recognition of metabolic syndrome depends on the particular criterion used. Many factors, including physical inactivity, obesity, excessive alcohol consumption and unhealthy diet have been identified as important modifiable metabolic syndrome risk factors and its consequences but the mechanisms underlying the onset are not fully elucidated (7).

Presently, the prevalence of metabolic syndrome globally is multiplying and reports from several countries have comparable prevalence rates ranging between 10-20%. This is evident in a study conducted in Hong Kong with a prevalence of 13.1% using the WHO criteria and Asian criterion of BMI (25kg/m^2 and above) and waist circumference (>90cm for men), and 9.6% using the NCEP criteria (8).

In African populations the prevalence of metabolic syndrome ranges from as low as 0% to as high as around 50%, or even greater, depending on the study population and design (9). In a study of adults in semi-urban and rural areas in Enugu state, South-East Nigeria, the metabolic syndrome prevalence was reported to be 18% (10). Another study in Benin, Nigeria using the three diagnostic tools - World Health Organization (WHO), Adult Treatment Panel (ATP III) and International Diabetes Federation (IDF) criteria, revealed a prevalence of 33.4%, 22.6% and 30.9% respectively (11).

The current pattern of increase in the metabolic syndrome prevalence is mostly, but not completely, attributed to embracing a western lifestyle, which is characterised by increased physical inactivity and replacement of the traditional African diet rich in fresh vegetables and fruits for the more calorie-laden foods (12). However, documented studies on metabolic syndrome in Nigerian cigarette smokers are scarce (13).

We therefore assessed the risk of metabolic syndrome and biomarkers of metabolic syndrome in male cigarette smokers and to determine any relationship between cigarette smoking and metabolic syndrome in male cigarette smokers in Calabar.

METHODS

Study area

The study was carried out within the Calabar South and Calabar Municipality Local Government Areas of Cross River State, Nigeria.

Study design/subject selection

A case control study design was used for the study. One hundred and forty-one (141) apparently healthy active male cigarette smokers and sixty (60) apparently healthy non-smokers aged 18 to 65 years were consecutively recruited for this study. They were all residents of Calabar. Each participant was duly informed on the objectives of the study and their informed consent was obtained. Participation was voluntary and confidentiality of participants' data was maintained. A structured questionnaire was administered to obtain information from the participants about their age, family and medical history, and dietary and physical activity and lifestyle. Ethical approval was obtained from the Health Research and Ethics Committee of the Cross River State Ministry of Health, Nigeria.

Inclusion and exclusion criteria

The test participants included males who smoked cigarettes at least once every day for one month or more and were asymptomatic of any disease (apparently healthy). The control participants had never smoked cigarettes in their life. Smokers who were diagnosed of any smoking-related disease (such as lung cancer, coronary heart disease), diabetes mellitus, hypertension, terminal disease or on drugs, were excluded from participation.

Sample collection

Venous blood samples were obtained from participants between 7a.m and 10a.m after an overnight fast. Six milliliters of blood was aseptically collected by venipuncture via the median cubital vein with a well tied tourniquet. Two milliliters were dispensed into a fluoride oxalate bottle for fasting plasma glucose determination while four milliliters were dispensed into a plain bottle and allowed to clot and retract at room temperature for about 45 minutes. After clotting, the blood was centrifuged at 4000 rpm for 5 minutes and the serum extracted was stored frozen at -20 ºC prior to lipid profile and insulin studies. All blood samples were free from haemolysis.

Calculation of sample size

A sample size of 125 was obtained using the formula described by Daniel with a prevalence rate of 9% (14) and 95% confidence limit (15).

Assessment of metabolic syndrome

Metabolic syndrome was assessed using the National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III) criteria. Metabolic syndrome for men according to the (NCEP-ATP III) criteria is the presence of three or more of the following; fasting plasma glucose level ≥5.6mmol/L, triglycerides level ≥1.7mmol/L, HDL-C level <1.03mmol/L, waist circumference ≥102cm and blood pressure ≥130/85 mmHg (16).

Measurement of blood pressure and anthropometric indices

The measurement of blood pressure was done using a digital blood pressure monitor (Omron Healthcare Ltd, UK). Weight and height of participants was measured in Kg and metres respectively using appropriate equipment. Body mass index (expressed in Kg/m^2) and waist/hip ratio were calculated using appropriate formulae (17,18).

Laboratory analyses

Serum insulin was measured using ELISA kits (Calbiotech Inc., California, USA). Plasma glucose and HDL-C levels were measured using enzymatic colorimetric methods (Giesse Diagnostics, Italy). Total cholesterol and triglycerides were measured using enzymatic colorimetric methods (ELITech Clinical Systems, SAS, France). Very low density lipoprotein-cholesterol (VLDL-C) and low density lipoprotein-cholesterol (LDL-C) were calculated using the Friedewald equation (19). Insulin resistance was determined using the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) formula (20).

Smoking pack years

Smoking pack years was calculated as the product of the number of packs of cigarettes smoked in a day and the number of years an individual has smoked. For example, 1 pack year implies smoking 1 pack of cigarettes (20 sticks) per day for one year. Smokers were classified as light smokers (<8 pack years), moderate smokers (8-30 pack years) or heavy smokers (>30 pack years) (21, 22).

Statistical analysis

Data was analysed using Microsoft Excel (MS office 2010) for Windows and Statistical Package for the Social Sciences (SPSS), version 20.0. Student's t-test, Analysis of Variance (ANOVA), Statistics calculator, Pearson's correlation and least significant difference (LSD) post hoc analysis were all employed in data analysis. Statistical significance was set at the p 0.05 level.

RESULTS

Diastolic blood pressure was significantly higher in smokers compared to non-smokers. There was, however, no significant difference in age, body mass index, waist circumference, waist/hip ratio or systolic blood pressure in smokers compared to non-smokers (Table 1).

Total cholesterol and LDL-C levels were significantly higher in the smokers compared to non-smokers while HDL-C was significantly lower in smokers compared to non-smokers. However, fasting plasma glucose, HOMA-IR, triglycerides and VLDL-C levels showed no significant difference between the two groups (Table 2).

Table 3 shows no significant differences in blood pressure or anthropometric indices in smokers based on their smoking pack years. Table 4 shows that there was a significant difference in the levels of fasting plasma glucose, triglycerides, TC and LDL-C among the three categories of smokers. Other parameters showed no significant differences.

Using LSD post-hoc analysis, BMI was significantly lower in heavy smokers than in the light or moderate smokers. Fasting plasma glucose was significantly higher in the light smokers than in the moderate and heavy smokers. Serum triglyceride levels were significantly higher in heavy smokers than in moderate smokers while triglycerides levels were significantly higher in heavy smokers than in light or moderate smokers. Serum LDL-C levels were also significantly higher in heavy smokers compared to light or moderate smokers. However, VLDL-C levels were significantly lower in moderate smokers compared to heavy smokers (Table 5).

Table 6 shows the cardio-metabolic risk factors in smokers and non-smokers. It was observed that dyslipidemia (increased LDL-C levels and decreased HDL-C) was the most predominant risk factor of metabolic syndrome in smokers compared to non-smokers. Additionally, some other risk factors were higher in smokers compared to non-smokers, though not statistically significant.

Table 7 shows the percentage of smokers and non-smokers with metabolic syndrome using the NCEP-ATP III criteria. The percentage of smokers with metabolic syndrome was significantly higher than that of the non-smokers.

Table 1. Age, blood pressure and anthropometric indices in male smokers and non-smokers.

Parameter	Smokers N=141	Non-smokers N=60	P
Age (years)	32.99 ± 9.21	30.87 ± 9.93	0.160
BMI (Kg/m^2)	23.04 ± 4.30	23.79 ± 2.65	0.134
WC (cm)	78.40 ± 8.29	79.65 ± 7.43	0.296
W/H	0.86 ± 0.07	0.85 ± 0.07	0.406
Systolic BP (mmHg)	129.96 ± 15.99	128.65 ± 12.22	0.527
Diastolic BP (mmHg)	82.55 ± 11.97	75.92 ± 9.34	0.0001

Values are expressed as mean ± SD.

BMI=body mass index; WC=waist circumference; W/H=waist/hip ratio; BP=blood pressure.

Table 2. Fasting plasma glucose, insulin, HOMA-IR and lipid profile in smokers and non-smokers.

Parameter	Smokers N=141	Non-smokers N=60	P
Glucose (mmol/L)	4.77 ± 0.96	4.91 ± 1.00	0.349
Insulin (µIU/ml)	1.98 ± 2.18	2.49 ± 1.86	0.092
HOMA-IR	0.42 ± 0.51	0.52 ± 0.40	0.118
TG (mmol/L)	0.99 ± 0.50	0.90 ± 0.36	0.150
TC (mmol/L)	4.68 ± 1.40	4.38 ± 1.03	0.008
HDL-C (mmol/L)	0.53 ± 0.17	1.43 ± 0.38	0.0001
LDL-C (mmol/L)	3.88 ± 1.39	2.67 ± 0.95	0.0001
VLDL-C (mmol/L)	0.45 ± 0.23	0.41 ± 0.17	0.147

Values are expressed as mean ± SD.

HOMA-IR=homeostasis model assessment of insulin resistance; TG=triglycerides; TC-total cholesterol; HDL-C=high density lipoprotein; LDL-C=low density lipoprotein,; VLDL-C=very low density lipoprotein.

Table 3. Blood pressure and anthropometric indices of male smokers based on smoking intensity.

Parameter	Light smokers (<8 pack yrs) N=94	Moderate smokers (8-30 pack yrs) N=27	Heavy smokers (>30 pack yrs) N=20	P
BMI (Kg/m^2)	23.27 ± 4.02	23.83 ± 5.83	21.07 ± 2.36	0.070
WC (cm)	78.84 ± 7.38	79.19 ± 10.51	75.30 ± 8.79	0.192
W/H	0.86 ± 0.07	0.87 ± 0.06	0.85 ± 0.09	0.723
Systolic BP (mmHg)	129.64 ± 15.11	134.33 ± 18.89	125.60 ± 15.21	0.170
Diastolic BP (mmHg)	81.22 ± 11.54	85.33 ± 11.97	85.55 ± 11.97	0.175

Values are expressed as mean ± SD.

BMI=body mass index; WC=waist circumference; W/H=waist-Hip ratio; BP=blood pressure.

Table 4. Fasting plasma glucose, insulin, HOMA-IR and lipid profile in smokers based on smoking pack years.

Parameter	Light smokers (<8 pack yrs) N=94	Moderate smokers (8-30 pack yrs) N=27	Heavy smokers (>30 pack yrs) N=20	P
Glucose (mmol/L)	5.0 ± 0.83	4.4 ± 1.21	4.2 ± 0.82	0.0001
Insulin(µIU/ml)	1.8 ± 2.27	2.1 ± 2.25	2.5 ± 1.57	0.475
HOMA-IR	0.39 ± 0.50	0.45 ± 0.64	0.48 ± 0.37	0.717
TG (mmol/L)	0.99 ± 0.55	0.85 ± 0.36	1.20 ± 0.34	0.054
TC (mmol/L)	4.82 ± 1.49	4.41 ± 0.97	5.65 ± 1.18	0.009
HDL-C (mmol/L)	0.52 ± 0.18	0.56 ± 0.15	0.55 ± 0.17	0.624
LDL-C (mmol/L)	3.86 ± 1.49	3.45 ± 0.97	4.56 ± 1.14	0.023

Values are expressed as mean ± SD.

HOMA-IR=homeostasis model assessment of insulin resistance; TG=triglycerides; TC=total cholesterol; HDL-C=high density lipoprotein; LDL-C=low density lipoprotein; VLDL-C=very low density lipoprotein.

Table 5. Glucose, TG, TC, LDL-C and VLDL-C in light, moderate and heavy smokers using LSD post hoc.

Parameter	Groups		Mean diff.	Std. error	P-value
	Light smokers (n=94)	**Moderate smokers (n=27)**			
BMI	23.23±4.02	23.83±5.83	-0.604	0.927	0.516
Glucose	5.0±0.83	4.4±1.21	0.590	0.199	0.004
TG	0.99±0.55	0.85±0.36	0.141	0.107	0.191
TC	4.82±1.49	4.41±0.97	0.416	0.297	0.164
LDL-C	3.86±1.49	3.45±0.97	0.402	0.296	0.178
VLDL-C	0.45±0.25	0.39±0.17	0.062	0.049	0.207
	Light smokers (n=94)	**Heavy smokers (n=20)**			
BMI	23.23±4.02	21.07±2.36	2.156	1.046	0.041
Glucose	5.0±0.83	4.2±0.82	0.741	0.225	0.001
TG	0.99±0.55	1.20±0.34	-0.213	0.121	0.082
TC	4.82±1.49	5.65±1.18	-0.822	0.335	0.016
LDL-C	3.86±1.49	4.56±1.14	-0.708	0.334	0.036
VLDL-C	0.45±0.25	0.55±0.15	-0.096	0.055	0.084
	Moderate smokers (n=27)	**Heavy smokers (n=20)**			
BMI	23.83±5.83	21.07±2.36	-2.760	1.253	0.029
Glucose	4.4±1.21	4.2±0.82	0.151	0.270	0.576
TG	0.85±0.36	1.20±0.34	-3.354	0.145	0.016
TC	4.41±0.97	5.65±1.18	-1.238	0.402	0.003
LDL-C	3.45±0.97	4.56±1.14	-1.109	0.401	0.006
VLDL-C	0.39±0.17	0.55±0.15	-0.158	0.066	0.018

Table 6. Metabolic abnormalities in male cigarette smokers and non-smokers.

Metabolic abnormalities	Smokers N=141 (%)	Non-smokers N=60 (%)	P
Hypertension[a]	16 (11.3)	3 (5)	0.163
Diabetes[b]	3 (2.1)	1 (1.7)	0.852
High LDL-C[c]	61 (43.3)	5 (8.3)	0.0001
Low HDL-C[d]	139 (98.6)	4 (6.7)	0.0001
Hypertriglyceridemia[e]	14 (9.9)	4 (6.7)	0.468
Central obesity[f]	11 (7.8)	5 (8.3)	0.904
High BMI	8 (5.7)	1 (1.7)	0.212
High Waist circumference	8 (5.7)	4 (6.7)	0.785
Dyslipidemia[g]	139 (98.6)	8 (13.3)	0.0001

a. Defined as blood pressure ≥140/90 mmHg.
b. Defined as fasting plasma glucose ≥7.0mmol/L.
c. Defined as LDL ≥4.0mmol/L.
d. Defined as HDL<0.9mmol/L.
e. Defined as triglycerides ≥1.70mmol/L.
f. Defined as waist circumference ≥94cm and/or BMI ≥30Kg/m^2.
g. Defined as triglycerides ≥1.70mmol/L and/or HDL<0.9mmol/L.

Table 7. Percentage of smokers and non-smokers with metabolic syndrome based on NCEP-ATP III criteria.

Metabolic Syndrome	N	NCEP-ATP III N (%)
Smokers	141	19 (13.5)
Non-smokers	60	2 (3.3)
t-test		2.163
P		0.032*

DISCUSSION

Cigarette smoking could predispose an individual to a variety of diseases, including metabolic syndrome (23). The findings from this study showed that the smokers and non-smokers have a comparable BMI with values within the normal range (18-25Kg/m^2) (24). This observation agrees with a study in Delta State, Nigeria which reported that smokers have a comparable BMI with non-smokers (13). However, while a study by Zbikowski *et al.* reported no significant association between smoking status and BMI (25), another study reported that BMI was significantly lower in smokers than in non-smokers (26). Our study showed a negative and significant relationship between smoking pack years and BMI. This implies that with increase in smoking pack years, there is loss in weight. This may be due to the adverse effects of smoking on food consumption such as loss of appetite, increased olfactory and gustatory receptor insensitivity, increase in energy expenditure (via cortisol enhanced lipolysis) and increase in metabolic rate (27). This finding is in line with a study by Pragti and Sunil (28).

Fasting plasma glucose, insulin and HOMA-IR were comparable in both smokers and non-smokers. The association between cigarette smoking and blood glucose levels remains controversial. Our observation of fasting blood glucose agrees with the findings of Nakanishi *et al.* (29) but, disagrees with the study of Oli *et al.* (30) who reported a transient increase in plasma glucose concentration in smokers. However, another study reported decreased fasting blood glucose levels in smokers compared to non-smokers (13). Nicotine has been linked to hyperglycemia. In small concentrations, it increases the activity of nicotinic acetylcholine receptors which provokes an increase in catecholamines (epinephrine and norepinephrine) as well as cortisol production. These hormones impair insulin action by stimulating hepatic glycogenolysis and gluconeogenesis leading an increase in plasma glucose levels (31). However, the decrease in plasma glucose levels with increased smoking intensity found in our study could be as a result of poor feeding habits of the smokers and fake satiety usually experienced by cigarette smokers in our community.

In our study, HOMA IR, a measure of insulin resistance, was not associated with smoking status. This is in agreement with a study by Berlin *et al.* (32). It has been observed that metabolic syndrome plays a key role between cigarette smoking and cardiovascular disease (33). This suggestion is mainly based on short-term human laboratory studies (34) and on the observations that smoking may result in reduced blood flow to skeletal muscles (increased peripheral resistance), vascular changes and central obesity, all potentially associated with decreased insulin-mediated glucose uptake and increased insulin insensitivity. However, another study showed that insulin levels and HOMA-IR were significantly higher in cigarette smokers than in non-smokers (29), which is in contrast with the findings of our study. Other studies have reported an increase in insulin resistance and a significant increase in HOMA-IR in cigarette smokers after an hour of smoking (35,36). Dietary lifestyle might contribute to the comparable results observed in both smokers and non-smokers. Future studies are, therefore, necessary to explore specifically the relationship between smoking and insulin resistance in this community.

Diastolic blood pressure was significantly increased in the smokers compared to the non-smokers. This finding is consistent with the observations of others (37). This may be due to the resultant effect of rapid mobilisation of catecholamines by nicotine during smoking, which is accompanied by high blood pressure and increased heart rate (38).

Smoking has been linked to increased synthesis and release of catecholamines, thereby resulting in an upsurge in circulating free fatty acids via lipolysis, which could be responsible for the high triglyceride and LDL-C concentrations previously observed (39). The dyslipidemia observed in our study is in line with findings of others who reported that tobacco smoking is associated with high levels of TG, LDL-C and reduced levels of HDL-C (40,41). However, other investigators reported no significant difference in lipid profile pattern among smokers and non-smokers (42).

The mechanisms through which smoking reduces HDL-C are not completely understood but has been linked to alteration in some important enzymes of lipid transport; by reducing lecithin-cholesterol acyl transferase activity, lowering cholesterol ester transfer protein and hepatic lipase activity (43). High density lipoprotein-cholesterol may also become vulnerable to oxidative changes by cigarette smoke thereby losing its atheroprotective function. Based on smoking pack years, our study demonstrated that triglyceride and LDL levels were associated with increased intensity and duration of smoking. These observations are similar to the findings of Omar *et al.* who reported that high levels of atherogenic lipoproteins, mainly LDL and LDL, in relation to increased smoking intensity most likely result in production of high concentration of oxidized LDL via increased oxidative alterations in the LDL molecule (43,44).

Our study showed a high prevalence of metabolic syndrome among smokers than non-smokers in Calabar using the NCEP-ATP III classification. However, the relationship between smoking status and metabolic syndrome is still controversial. Metabolic syndrome is estimated to affect around 20-25% of the adult population globally (45). The current trend of high metabolic syndrome prevalence is mostly and generally attributed to adoption of sedentary lifestyle characterised by reduced physical activity and replacing the traditional African diet rich in vegetables and fruits with high calorie containing foods (12). Findings from the NHANES III survey indicated that smoking was associated with a high risk of metabolic syndrome in adult males and females when adjusted for modifiable lifestyle factors (46).

In Sub-Saharan Africa and in Nigeria, there is paucity of data on epidemiological characteristics and prevalence of metabolic syndrome in cigarette smokers. In the general population, the first reported metabolic syndrome study in Sub-Saharan Africa was carried out in Cameroon in the mid-90s (47) which found a metabolic syndrome prevalence of 1.3% among men living in urban areas using the IDF classification (9), though, HDL-C concentration was not measured in that study. A similar study carried out in Seychelles found a high metabolic syndrome prevalence which was evident in 25-30% of the study participants (48). In Ethiopia the metabolic syndrome prevalence among working men, based on the NCEP-ATP III and IDF criteria, were 10% and 14% respectively (49). In a study involving adults in semi-urban and rural areas in Enugu state, Nigeria, the metabolic syndrome prevalence was found to be 18% (10). Using the NCEP-ATP III criteria, we found a significant prevalence estimate of metabolic syndrome to be 13.5% in smokers as against 3.3% observed in non-smokers. In the Sokoto state, Nigeria, a 27.36% metabolic syndrome prevalence rate in men (non-smokers) was reported based on the NCEP-ATP III criteria and also identified hypertension and low HDL-C to be the most predominant components of the metabolic syndrome (50). However, our study identified dyslipidemia (high LDL-C and low HDL-C) as the major predominant risk factors of metabolic syndrome. This may be because our study was conducted in an urban area where physical activity is reduced as well as nutrition transition to refined, low fiber and calorie dense meals.

Smokers in the Delta state of Nigeria are at high risk of metabolic syndrome based on some components of the syndrome (dyslipidemia and hypertension) (13). The high prevalence of metabolic syndrome and its components found in our study may be partly due to the epidemiological and

nutritional transition that have occurred in the world, including Sub-Saharan Africa, where lifestyle modifications and behavioral changes, both products of urbanisation and modernization, have occurred (51). The unfavorable changes in the lipid profile and blood pressure observed in our study may predispose smokers to a higher risk of cardiovascular diseases and there is a high frequency of metabolic syndrome among cigarette smokers in Calabar compared to the non-smokers.

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