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Cardiovascular Disease risk Factors in Male Cigarette Smokers in Calabar, Southern Nigeria

Iya Eze Bassey[1], Uwem Okon Akpan[*1], Ifenyinwa Maryann Okafor[1], Imeobong Joseph Inyang[1], Oko Eze[2]

[1]*Department of Medical Laboratory Science, University of Calabar, PMB 1115 Calabar, Cross River State, Nigeria*

[2]*Department of Chemical Pathology, University of Abuja Teaching Hospital, Gwagwalada, Abuja, Nigeria*

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KEYWORDS

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ABSTRACT: Cigarette smoking has been linked to atherosclerosis, coronary artery disease, peripheral vascular disorders and various malignancies. However, the mechanism by which smoking increases the risk of cardiovascular diseases is still controversial. One hundred and forty one apparently healthy male cigarette smokers and sixty apparently healthy non-smokers aged 18 to 65 years were enrolled into the study. The smokers were sub-divided into light (<8 pack years), moderate (8-30 pack years) and heavy (>30 pack years) smokers. Anthropometric indices and blood pressure were measured. Fasting plasma glucose, total cholesterol (TC), triglycerides and High density lipoprotein-cholesterol (HDL-C) were estimated using colorimetric test methods while low density lipoprotein-cholesterol (LDL-C) and very low density lipoprotein were calculated using Friedewald's equation. Serum insulin was estimated using ELISA. Data was analyzed using SPSS version 20.0; level of significance was set at $p<0.05$. The smokers had significantly higher diastolic BP ($p=0.0001$), TC ($p= 0.008$) and LDL-C ($p = 0.0001$) and significantly lower HDL-C ($p = 0.0001$) compared to the non-smokers. There was a significant higher fasting plasma glucose in the light smokers ($p=0.001$) than in the moderate and heavy smokers whereas, serum TC and LDL-C levels were significantly increased in heavy smokers ($p=0.001$) than in the light and moderate smokers. There was a negative correlation between BMI and smoking pack years. Dyslipidaemia was observed to be the most prevalent cardiovascular disease risk factor. It is concluded that the alterations in lipid profile and blood pressure observed in this study may contribute to higher risk of cardiovascular disease.

INTRODUCTION

Cigarette smoking is among the leading causes of preventable death in the world today and is linked to high mortality and morbidity. Smoking harms almost every organ or tissue in the body and greatly reduces both the quality of life and life expectancy [1].

Tobacco use kills over 7 million persons annually. This is expected to rise to more than 8 million annually by 2030 [2]. About 80% of the 1.1 billion smokers in the world reside in low and middle-income countries including Nigeria [3].

In Nigeria, the use of tobacco products will soon fast becoming a major etiological agent of early death and other health related conditions, unless strict measures are employed to discourage young adults from tobacco usage

*Corresponding author: akpauwem11@gmail.com (U. O. Akpan)
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and prevent them from smoking. In 2008, the Nigeria Demographic and Health Survey reported that less than 1% of females and 11.5% of males aged 14-49 years use tobacco products and 9% of them smokes cigarette [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].

Cigarette smoking is an important and independent modifiable risk factor of hypertension, atherosclerosis, coronary artery disease, peripheral vascular disorders, emphysema and various types of malignancies [2]. The mechanism by which tobacco smoking increases the risk of cardiovascular diseases is still controversial. These cardiovascular risk factors include hypertension, dyslipidaemia, diabetes mellitus, overweight and obesity, smoking, excessive alcohol intake, physical inactivity. Other cardiovascular risk factors are non-modifiable and include age, gender and family history [6]. The clustering together of the individual risk factors in a significant pattern tends to increase an individual's total cardiovascular risk [6]. Abnormality of cholesterol metabolism may lead to cardiovascular accidents and heart attacks [7]. However, studies have shown incomplete, inconclusive or conflicting results about smoking and its relationship with lipids and lipoproteins [8]. Cigarette smoking is also known to increase the generation of free radicals which oxidizes low density lipoprotein (LDL)-cholesterol. Oxidized LDL-C promotes the risk of atherosclerosis, one of the major components of metabolic syndrome [9]. There are several evidences that smoking enhances the risk of diabetes. However, the appropriate mechanism on how tobacco smoking enhances the risk of diabetes and diminished glucose homeostasis is not fully understood, but existing data shows that smoking elevates insulin resistance. Studies have also shown that smoking reduced insulin mediated glucose uptake in smokers compared with non-smokers [10] and smokers have a significantly elevated HOMA-IR index one hour after tobacco smoking [11].

Among cardiovascular parameters, blood pressure is greatly influenced by smoking by a mechanism not fully elucidated [12]. Also, it is still controversial whether smoking exposure leads to an elevated or reduced blood pressure hence, the relationship between cigarette smoking and blood pressure in epidemiological studies still remains contradictory despite scientific evidence that smoking increases blood pressure [13]. This study investigated lipid profile, insulin, fasting plasma glucose, HOMA-IR, blood pressure and anthropometric indices in male cigarette smokers in Calabar.

MATERIALS AND METHODS

Study area

The study was carried out within Calabar South and Calabar Municipality Local Government Areas of Cross River State, Southern 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. A written informed consent and ethical clearance with certificate number ***RP/REC/2015/311*** were obtained for the study. A structured questionnaire was used to obtain information about the socio-demographic parameters of study participants. The study was carried out between October 2015 and April 2016.

Calculation of sample size

The sample size calculation was done using the formula described below [14].

$N = Z^2pq / d^2$

where N = sample size

Z = the Z score (1.96 at 95% confidence interval)
p = sample proportion
q = (1-p) proportion of non-occurrence
d= precision (allowable error margin- 5%)
when p = 9% i.e. 0.09 [15],

we have

$N = 1.96^2 \times 0.09(1 - 0.09) / (0.05)^2 =$ **125**

Inclusion and exclusion criteria

The test participants were males who smoked cigarette at least once every day for one month or more and were asymptomatic of any disease (apparently healthy). The control participants had never smoked cigarette in their life. Smokers with any smoking-associated disease (including lung carcinoma, coronary heart disease), diabetes mellitus, hypertension, terminal disease or on medication were exempted from participation**.**

Sample collection

Venous blood samples were collected from participants between 7a.m and 10a.m after fasting overnight. Six millilitres (6ml) of blood was aseptically collected by venipuncture via the median cubital vein with a well tied tourniquet. Two millilitres (2ml) were dispensed into fluoride oxalate bottle for fasting plasma glucose determination while four millilitres (4ml) were dispensed into a plain bottle and allowed to clot and retract. After clotting, the blood was centrifuged at 4000 revolutions per minute for 5 minutes and the serum extracted was stored frozen and later used for lipid profile and insulin studies.

Measurement of blood pressure

The measurement of blood pressure was done in a well-seated and relaxed position after resting about 10 minutes, using a Digital blood pressure monitor from OMRON HEALTHCARE LTD, United Kingdom. Two readings were taken from each subject and the systolic and diastolic blood pressures were recorded after computing the average of the two readings.

Measurement of weight and height

The measurement of weight was achieved using a bathroom weighing scale. The participants were made to stand erect bare-footed and facing front. The values were read to the nearest 0.1Kg. A stadiometer was used in the measurement of height. Each participant was instructed to stand erect, without any shoes and cap against a wall. Their heights were read in metres.

Measurement of waist and hip circumferences

A stretch resistant tape was used in measuring the waist circumference. The value was obtained by calculating the mean of two measurements taken, one before inspiration and the other after expiration with the tape placed at midway between the base of the rib cage and the apex of the iliac crest. Measurement of hip circumference was achieved by placing a stretch resistant tape horizontally around the widest part of the hips and buttocks while the participants were standing erect and relaxed with their feet as close together as possible.

Determination of body mass index and waist-hip ratio

Body Mass Index (BMI) was obtained using the formula below;

$$BMI = \text{Weight (Kg)} / \text{Height (m}^2\text{)}$$

This is expressed in Kg/m^2

Waist-Hip ratio was obtained by dividing the values of waist circumference (cm) by the hip circumference (cm)

W/H ratio = Waist circumference (cm) / Hip circumference (cm)

Determination of fasting plasma glucose

The glucose oxidase-peroxidase method was used to determine the plasma glucose concentration colorimetrically. Kits were obtained from Giesse Diagnostics, Italy.

Determination of serum insulin

Serum insulin was analyzed using a solid phase two-site enzyme immunoassay insulin ELISA kit by CALBIOTECH Inc, United States.

Assessment of fasting lipid profile

The lipids assessed were serum triglycerides, total cholesterol and high density lipoprotein-cholesterol (HDL-C). Serum triglycerides and total cholesterol and were determined using the enzymatic colorimetric method and HDL-cholesterol by the precipitation/cholesterol determination method. Serum total cholesterol and triglycerides kits were obtained from ELITech Clinical Systems, SAS, France. HDL-C kits were obtained from Giesse Diagnostics, Italy. Very low density lipoprotein-cholesterol (VLDL-C) and Low density lipoprotein-cholesterol (LDL-C) were calculated using Friedewald's equation.

Smoking pack years

A pack year is a quantification of cigarette smoking. It is a tool used in determining the quantity of cigarette smoked by an individual over a period of time. It is calculated by the product of the number of cigarette packs smoked in a day and total number of years the person has smoked. For example, 1 pack year implies smoking 1 pack of cigarette (20 sticks) per day for one year, or 2 packs (40 sticks) per day for 6 months, and so on.

Smokers were therefore classified as light smokers (<8 pack years), moderate smokers (8-30 pack years) or heavy smokers (>30 pack years) [16].

Statistical analysis

The data obtained were analyzed on Microsoft Excel (MS office 2010) for Windows and 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. Probability value, $p<0.05$ was considered statistically significant

Ethical consideration

Ethical clearance was obtained from the Health Research and Ethics committee of the Cross River State Ministry of Health, Nigeria with certificate number *RP/REC/2015/311*.

Each participant was duly informed on the objectives of the study and their consent was obtained. All experiments and procedures were done following the ethical guidelines of 1964 Declaration of Helsinki.

RESULTS

The results show no significant difference ($p>0.05$) in the age, Body mass index (BMI), waist circumference (WC), waist-hip ratio, systolic blood pressure (SBP), fasting plasma glucose (FPG), fasting insulin (FI), Homeostasis model assessment of Insulin resistance (HOMA-IR) and Triglyceride (TG) and very low density lipoprotein-cholesterol (LDL-C) levels in the smokers than the non-smokers. On the other hand, the diastolic blood pressure (DBP), total cholesterol (TC) ($p=0.008$) and LDL-C ($p=0.0001$) were significantly increased ($p=0.0001$) in the smokers than in the non-smokers while high density lipoprotein-cholesterol (HDL-C) ($p=0.0001$) was significantly reduced in the smokers compared to the non-smokers (Table 1).

Table 2 shows a significant variation in FPG ($P=0.0001$), TG ($p=0.054$), TC ($p=0.009$) and LDL-C ($p=0.023$) levels in light, moderate and heavy smokers. Other parameters showed no significant variation ($p>0.05$).

Table 3 shows the comparison of BMI, FPG, TG, TC, LDL-C and VLDL-C in the smokers based on smoking pack years using LSD post-hoc analysis. BMI was significantly lower in heavy smokers than in the light ($p=0.041$) and moderate smokers ($p=0.029$). Fasting plasma glucose was significantly higher in the light smokers than in the moderate ($p=0.004$) and heavy smokers ($p=0.001$). Serum TG levels were significantly higher ($p=0.016$) in the heavy smokers than in the moderate smokers while TC levels were significantly higher in the heavy smokers than in the light ($p=0.016$) and moderate smokers ($p=0.003$). Serum LDL-C were also significantly higher in the heavy smokers than in the light ($p=0.036$) and moderate smokers ($p=0.006$). However, VLDL-C levels were significantly lower ($p=0.018$) in the moderate smokers than in the heavy smokers.

Table 4 shows the cardiovascular disease risk factors in smokers and non-smokers. It was observed that dyslipidemia [elevated LDL-C (p=0.0001) and reduced HDL-C (p=0.0001)] was the most predominant risk factor of metabolic syndrome in the smokers compared to non-smokers. Additionally, other risk factors were higher in the smokers than in non-smokers though not statistically significant (p>0.05). Figure 1 shows a significant negative correlation between smoking pack years and BMI (r = -0.254, p=0.002).

Table 1. Age, blood pressure, anthropometric indices, Fasting plasma glucose, Insulin, HOMA-IR and Lipid Profile in smokers and non-smokers**.**

Parameter	Smokers, n=141	Non-smokers, n=60	t-cal	P-value
Age (years)	32.99 ± 9.21	30.87 ± 9.93	1.414	0.160
BMI (Kg/m^2)	23.04 ± 4.30	23.79 ± 2.65	1.504	0.134
WC (cm)	78.40 ± 8.29	79.65 ± 7.43	1.050	0.296
W/H	0.86 ± 0.07	0.85 ± 0.07	0.835	0.406
Systolic BP (mmHg)	129.96 ± 15.99	128.65 ± 12.22	0.634	0.527
Diastolic BP (mmHg)	82.55 ± 11.97	75.92 ± 9.34	4.222	0.0001*
FPG (mmol/L)	4.77 ± 0.96	4.91 ± 1.00	0.940	0.349
FI (µIU/ml)	1.98 ± 2.18	2.49 ± 1.86	1.697	0.092
HOMA-IR	0.42 ± 0.51	0.52 ± 0.40	1.573	0.118
TG (mmol/L)	0.99 ± 0.50	0.90 ± 0.36	1.446	0.150
TC (mmol/L)	4.68 ± 1.40	4.38 ± 1.03	2.683	0.008*
HDL-C (mmol/L)	0.53 ± 0.17	1.43 ± 0.38	17.544	0.0001*
LDL-C (mmol/L)	3.88 ± 1.39	2.67 ± 0.95	7.153	0.0001*
VLDL-C (mmol/L)	0.45 ± 0.23	0.41 ± 0.17	1.459	0.147

Values are expressed as Mean ± SD, *significant at P≤0.05; Key: BMI-Body mass index, WC-Waist circumference, W/H-Waist-Hip ratio, BP-Blood pressure, FPG-Fasting plasma glucose, FI-Fasting insulin, 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 2. Blood pressure, anthropometric indices, 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	F-cal	P-value
BMI (Kg/m^2)	23.27 ± 4.02	23.83 ± 5.83	21.07 ± 2.36	2.709	0.070
WC (cm)	78.84 ± 7.38	79.19 ± 10.51	75.30 ± 8.79	1.668	0.192
W/H	0.86 ± 0.07	0.87 ± 0.06	0.85 ± 0.09	0.325	0.723
Systolic BP (mmHg)	129.64 ± 15.11	134.33 ± 18.89	125.60 ± 15.21	1.792	0.170
Diastolic BP (mmHg)	81.22 ± 11.54	85.33 ± 11.97	85.55 ± 11.97	1.763	0.175
FPG (mmol/L)	5.0 ± 0.83	4.4 ± 1.21	4.2 ± 0.82	8.162	0.0001*
FI (µIU/ml)	1.8 ± 2.27	2.1 ± 2.25	2.5 ± 1.57	0.749	0.475
HOMA-IR	0.39 ± 0.50	0.45 ± 0.64	0.48 ± 0.37	0.333	0.717
TG (mmol/L)	0.99 ± 0.55	0.85 ± 0.36	1.20 ± 0.34	2.975	0.054*
TC (mmol/L)	4.82 ± 1.49	4.41 ± 0.97	5.65 ± 1.18	4.843	0.009*
HDL-C (mmol/L)	0.52 ± 0.18	0.56 ± 0.15	0.55 ± 0.17	0.473	0.624
LDL-C (mmol/L)	3.86 ± 1.49	3.45 ± 0.97	4.56 ± 1.14	3.876	0.023*
VLDL-C (mmol/L)	0.45 ± 0.25	0.39 ± 0.17	0.55 ± 0.15	2.870	0.060

Values are expressed as Mean ± SD

*=significant at P≤0.05, BMI-Body mass index, WC-Waist circumference, W/H-Waist-Hip ratio, BP-Blood pressure, FPG=Fasting plasma glucose, 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. BMI, FPG, 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
FPG	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*
FPG	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*
FPG	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*

*Significant at P≤0.05

Table 4. Cardiovascular disease risk factors in male cigarette smokers and non-smokers.

Risk factor	Smokers n=141(%)	Non-smokers n=60(%)	P-value
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
Dyslipidemia[g]	139(98.6)	8(13.3)	0.0001*

Key: *significant at P≤0.05

a)Defined as BP ≥140/90 mmHgb, b) Defined as FPG ≥7.0mmol/L, c)Defined as LDL ≥4.0mmol/L, d)Defined as HDL<0.9mmol/L, e) Defined as TG ≥1.70mmol/L, f)Defined as WC ≥94cm and/or BMI ≥30Kg/m^2, g)Defined as TG ≥1.70mmol/L and/or HDL<0.9mmol/L

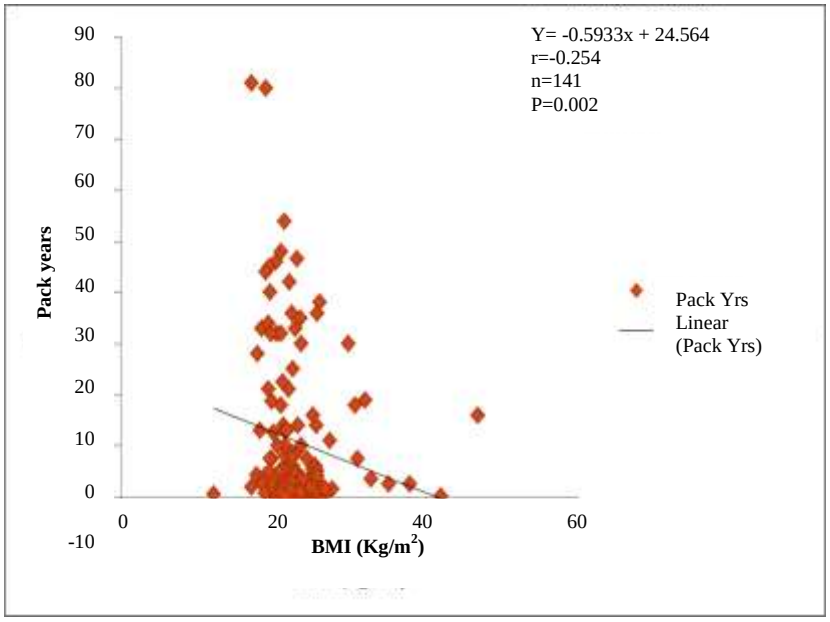

Figure 1. Correlation plot of Pack years against BMI in smokers.

DISCUSSION

Cigarette smoking has been implicated as one of the cardiovascular disease risk factors and other systemic anomalies [3]. 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) [17]. This observation agrees with the findings of previous studies [5, 18] which reported that smokers have a comparable BMI with non-smokers. Another study [19] also reported no significant relationship between BMI and smoking status but another study reported reduced BMI in cigarette smokers [20] while some studies in Europe showed a positive association between cigarette smoking and BMI/obesity [21, 22]. The correlation analysis 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 negative effects of tobacco 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 [22]. This finding is in line with a previous study [23].

Fasting plasma glucose, Insulin and HOMA-IR were comparable in both smokers and non-smokers. The relationship between blood glucose levels and cigarette smoking still remains controversial. Our observation on FPG agrees with the findings of [23] but, disagrees with the works of other researchers [24]. However, a study in Nigeria reported a decreased FPG in smokers compared to non-smokers [18]. 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 nonepinephrine) as well as cortisol production. These hormones impair insulin action by stimulating hepatic glycogenolysis and gluconeogenesis leading to increase plasma glucose levels. However, the decrease in plasma glucose levels with increased smoking intensity observed 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 this study, HOMA IR, a measure of insulin resistance was not associated with smoking status. This agrees with the findings of previous work [25] who reported that smoking did not have any effect on insulin resistance as measured by HOMA-IR. It has been observed that metabolic syndrome plays a key role between

cardiovascular disease and tobacco smoking [26]. This suggestion is mainly based on short-term human laboratory studies [27] 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, a study reported that Insulin and HOMA-IR were significantly higher in cigarette smokers than non-smokers [24] which are in contrast with the findings of the study. Other studies have reported high insulin resistance and a significant increase in HOMA-IR in cigarette smokers after an hour of smoking [11, 28]. 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 smokers.

A significantly higher diastolic blood pressure was observed in the smokers than in the controls. This finding is consistent with the observations of other researchers [5, 18]. This may be due to the resultant effect of rapid mobilization of catecholamines by nicotine during smoking, which is accompanied by high blood pressure and increase heart rate [29].

Smoking has been linked to increase synthesis and release of catecholamines, thereby resulting in an upsurge in circulating free fatty acids via lipolysis, which could be responsible for the high TC and LDL-C concentrations found in our study [30]. The dyslipidemia (high TC and LDL-C levels, low HDL-C levels) observed in this study is in line with findings of earlier investigators who reported that tobacco smoking is associated with high levels of TG, LDL-C and reduced levels of HDL-C [31, 32].

The mechanisms through which smoking reduces HDL-C are not completely understood but it has been linked to alteration in some important enzymes of lipid transport; by reducing Lecithin-cholesterol acyl transferase (LCAT) activity, lowering Cholesterol ester Transfer Protein (CETP) and hepatic lipase activity [33] as observed in this study. High density lipoprotein-cholesterol may also become vulnerable to oxidative changes by cigarette smoke thereby losing its atheroprotective function. Based on smoking pack years, this study demonstrated that TC and LDL levels were associated with increased intensity and duration of smoking. These observations are similar to the findings of previous studies [32] which found that high levels of atherogenic lipoproteins mainly IDL 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. A study also reported that heavy smokers had slightly higher LDL-C than the light and moderate smokers [34].

This study identifies dyslipidemia (high LDL-C and low HDL-C) as the major predominant risk factor of cardiovascular disease. This may be because the study was conducted in an urban area where physical activity is reduced as well as nutrition transition to refined, low fibre and calorie dense meals [35]. This is similar to previous research findings [18] which observed that dyslipidemia and hypertension were the predominant risk factors associated with cigarette smoking.

CONCLUSIONS

The unfavourable alterations in the lipid parameters and blood pressure found in this study may predispose smokers to high cardiovascular diseases risk compared to the non-smokers.

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Conflict of interest

All authors declare that there is no conflict of interest.

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