Research Article

Lipid Profile of Cigarette Smokers in an Ancient City

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Abstract: Reports have shown that sudden death is 2-4 times more often in heavy cigarette smokers than non smokers. Cigarettes smoke has been confirmed to contain toxicants that can disrupt normal metabolic processes. This study was carried out to assess the status of lipid fractions in smokers in a population in South West Nigeria. 25 males who were smoking 5 – 8 sticks of cigarette per day aged 20 – 45 years were selected as the study group, while 20 aged matched males who never smoked cigarette were selected as the control group. The concentrations of total cholesterol (TC), High density lipoprotein - cholesterol (HDL-C), triglyceride (TG), were determined using standard enzymatic colorimetric methods and low density lipoprotein - cholesterol (LDL-C) was calculated using Friedewalds formula. The concentrations of all the lipid fractions were significantly higher (p<0.05) in smokers than that of non smokers except HDL-C which was otherwise. The various ratios of LDL/HDL, TC/HDL, and TG/HDL were all higher in the smokers than in the non smokers. The percentage of LDL in total cholesterol was higher in smokers than non-smokers, but reverse was the case with HDL. There were significant and direct association between TG and LDL (r=0.902, p<0.01), TC(r=0.931, p<0.01) but inverse relationships were observed between TG and HDL (r=0.839, p<0.01). There was no significant difference between the BMI of the smokes and non smokers. The results of this study show that smokers are at much greater risk of developing atherosclerotic plaques and different heart diseases than non-smokers.
Keywords: Smokers, Cigarette, Lipoprotein, non-smokers, risk factor.

INTRODUCTION

Tobacco smoking is one of the most potent and prevalent addictive, influencing behavior of human beings for over four centuries. Smoking is now increasing rapidly throughout the developing world and is one of the biggest threats to current and future health [1]. Tobacco continues to be the second major cause of death in the world. By 2030, if current trends continue, smoking will kill over 9million people annually [2]. Smoking is an important preventable cause of mortality worldwide. The prevalence of pulmonary and cardiovascular disease, cataracts and some cancers is higher in smokers. Tobacco smoke contains some deadly carcinogenic chemicals formed from natural components of the tobacco plants and leads to the uptake of many hazardous compounds such as heavy metals and N-nitrosocompound [3]. Apart from natural constituents of tobacco, many substances are added to cigarette by manufacturers to enhance the flavor or to make smoking more pleasant. Some of the compounds found in tobacco smoke include ammonia, tar and carbon monoxide. Exactly what effects these substances have on the cigarettes smokers' health is unknown, but there is no evidence that lowering the tar content of cigarette lower the health risk [4].

Cigarette smoking leads to the uptake of many hazardous compounds and their metabolites extracted from burning tobacco. These substances may be electrophilic and react with biological molecules, give rise to oxidative stress through the formation of reactive species or the initiation of lipid peroxidation chain reactions in the membrane [5]. Cigarette smoking has been found to alter the lipoprotein levels [6].

Several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile [7-9]. However, in spite of all these information, there is still much controversy about which part or parts in the lipid profile are mainly altered in response to cigarette smoking, and whether those lipid profile components influence other parts directly and vice versa. Different results have been obtained by various investigators. For example, Siekmeier et al. concluded that HDL-C levels were same for smokers and non-smokers [10], whereas some other

investigators obtained conflicting results wherein significant variations (low levels of HDL-C in cigarette smokers) were obtained [11, 12]. Currently, there is paucity of data on the status of lipid profile in our environment. This study was conducted to investigate the lipid profile status in smokers in our community.

MATERIALS AND METHODS

This study was carried out among the smokers and non-smokers that gave their consent of participation. The study group comprised of 25 males with age range between 20 and 45years (average 32.5years) and 20 age matched males as controls. The smokers in this study were those smoking 5 – 8 cigarette/day with an average of 6.5 cigarette/day and duration of smoking habit was 1 – 3years with average of 2.5 years.

Before the collection of blood samples from smokers and non-smokers, questionnaires were administered to provide the details about their smoking habits. The age, body weight, height and other physical measurements were obtained. All the recruited subjects were neither alcoholic nor having any form of diseases or ailment. Hence, all male subjects included in the present study were apparently normal healthy individual. About 5ml of venous blood samples was collected from each of the subjects into lithium heparin bottle after 8 - 12hrs overnight fast with individuals being on their normal diet prior to the test. The samples were spurned and the plasma separated within 2hrs of collection and analyzed immediately using standard enzymatic methods for each of the parameters investigated. The LDL was calculated from the values of triglyceride and cholesterol using the Friedewalds formula [13].

Table 1: Lipid profile of smokers and non-smokers and their body mass index (BMI)

	BMI (Kg/m^2)	Total cholesterol (mmol/L)	Triglyceride (mmol/L)	HDLC (mmol/L)	LDLC (mmol/L)
Smokers (n=25)	22.76±1.40	6.54±0.49	2.96±0.30	1.03±0.10	4.18±0.40
Non Smokers (n=20)	22.99±1.23	4.23±0.53	1.10±0.11	1.49±0.13	2.22±0.51
P value	>0.05	>0.05	>0.05	>0.05	>0.05

Key: BMI- Body mass index; TG-Triglyceride; TC – Total Cholesterol; HDL-High density lipoprotein; LDL-Low density lipoprotein. Values were expressed in mean ± standard deviation (SD)

Table 2: Coronary heart disease markers and ischemic disease markers index in smokers and non-smokers.

	BMI (Kg/m^2)	LDL/HDL	TC/HDL	TG/HDL
Smokers (n=25)	22.76±1.40	4.07±0.49	6.37±0.56	2.87±0.30
Non Smokers (n=20)	22.99±1.23	1.50±0.35	2.84±0.50	0.74±0.08
P value	>0.05	<0.05	<.05	<0.05

Key: LDL/HDL= Coronary heart disease index, TC/HDL = Ischemic disease index, TG/HDL = Triglyceride/ High density lipoprotein

Table 3: Pearson Correlation of parameters of smoker lipid profiles

	BMI	TG	TC	HDL	LDL	LDL/HDL	TC/HDL	TG/HDL
BMI (Kg/m^2)	1	-0.006	-0.089	0.135	-0.173	-0.112	-0.228	0.367*
TG(mmol/l)	-0.006	1	0.931**	-0.839**	0.902**	0.878**	0.819**	-0.612**
TC (mmol/l)	-0.089	0.931**	1	-0.725**	0.973**	0.893**	0.800**	-0.726**
HDL (mmol/l)	-0.135	-0.839**	-0.725**	1	-0.775**	-0.840**	-0.810**	0.512**
LDL (mmol/l)	-0.173	0.902**	0.973**	-0.775**	1	0.902**	0.840**	-.0784**
LDL/HDL	-0.112	0.878**	0.893**	-0.840**	0.902**	1	0.672**	-0.727**
TC/HDL	-0.228	0.819**	0.800**	-0.810**	0.840**	0.672**	1	-0.688**
TG/DL	0.367*	-0.612**	-0.726**	0.512**	-0.784**	-0.727**	-0.688**	1

*Correlation is significant at the 0.05 level, ** Correlation is significant at the 0.01level

Table 4: Percentage distribution of HDL and LDL in Total cholesterol

	LDL-C (%)	HDL-C (%)
Smokers (n=25)	63.9	15.8
Non Smokers (n=20)	52.5	35.2

RESULTS

Lipid profiles of smokers and non-smokers were shown in table 1, together with their body mass index (BMI). Statistically significant difference were noticed in all the lipid fractions estimated in smokers when compared with non-smokers (p<0.05).The experimental difference seen in the BMI was not statistically significant (p>0.05). The profile estimated were Body mass index (BMI), Triglyceride (TG), Total cholesterol (TC), High density lipoprotein cholesterol (HDL) and Low density lipoprotein cholesterol (LDL).

Table 2 shows the ratio of lipid fractions between smokers and non smokers. There were significant differences in all the entire ratios (LDL/HDL 4.07±0.49, 1.50±0.35; TC/HDL 6.37±0.56, 2.84±0.05; TC/LDL 1.55±0.80, 2.03±0.45 for test and control) the p-values were <0.05, <0.05 <0.05 respectively.

The Pearson correlation of all parameters estimated in the smokers were show in table 3. Positive correlation that was statistically significant exist between BMI and TG/HDL (r=0.367* p<0.05), while non-significant negative correlation was observed with other parameters. The correlation that exist between TG and TC, LDL, LDL/HDL, TC/HDL were positive (r=0.931**, r=0.902**, r=0.878**, r=0.819**), and statistically significant (p<0.01, <0.01, <0.01, <0.01). Negative correlation exist between TG and HDL, TC/LDL (r=-0.839**, r=-0.612**), these were also statistically significant (p<0.01, <0.01). Negative correlation exist between TC and HDL, TG/HDL(r=-0.725**, r=-0.726**) and were statistically significant (p<0.01, <0.01). There were positive correlations between TC, LDL, LDL/HDL and TC/HDL (r=0.973**, r=0.893**, r=0.800**). HDL correlate negatively with LDL, LDL/HDL, TC/HDL (r=-0.775**, r=-0.840**, r=-0.810) and positively with TG/HDL (r=0.512**). The correlations were statistically significant (p<0.01, <0.01, <0.01, <0.01). Statistically significant positive and negative correlations exist between LDL and LDL/HDL, TC/HDL, TG/HDL (r=0.902**, r=0.840**, r=-0.784**)(p<0.01, <0.01, < 0.01). Both positive and negative correlation were observed between LDL/HDL and TC/HDL, (r=0.672**), TG/HDL (r=-0.727**) and these were statistically significant (p<0.01, <0.01), while the correlation between the TC/HDL and TC/LDL (r=-0.688**) was negative and statistically significant (p<0.01).

The percentage distribution of HDL and LDL in total cholesterol is shown in table 4. The total cholesterol in smokers has up to 63.9% of LDL while that of non-smoker was 52.5%. The percentage of HDL in smokers was 15.8%, and that of non smokers was 35.2%.

DISCUSSION

It has long been established that one of the major constituents of tobacco i.e nicotine has a considerable influence in increasing the lipid levels in blood. Lipid have important roles In virtually all aspect of life, serving as hormones or hormones precursors, aiding in digestion, providing energy storage metabolic fuel, acting as functional and structural component in cell membranes and forming insulation to allow nerve conduction or to prevent heat lost [14], but their excessive concentrations are associated with various metabolic disorders.

The result of this work showed a statistically significant different in the total cholesterol level of smokers (p<0.05) when compared with non-smokers, this indicate that the cigarette smokers have increased serum concentration of cholesterol than non-smokers. The result of this work is in line with work of Adedeji and Etukudo, where high concentration of cholesterol was recorded in smokers when compared with the non-smokers [15]. In contrast Waheed and Alharbi in their work, which was on the influence of cigarette smoking on lipid profile in male university students recorded non significant result in total cholesterol in smokers when compared with non-smokers [16]. The increase in the total cholesterol level seen in the smokers was as a result of increase in the activity of hepatic HMG-CoA reductase [17]. and [18] reported that hepatic HMG CoA reductase, the main rate limiting enzyme in cholesterol synthesis is subject to induction and repression by several hormones, dietary factors and drugs one of which is nicotine. Increased cholesterol is a causative factor in the etiology of atherosclerotic disease [19]. The rise in blood cholesterol levels in smokers may be through catecholamine and adenyl cyclase axis including tissue lipolysis [20].

The values of triglycerides of smokers in this study were significantly increased compared to that of non-smokers. The increase in the value of triglyceride is due to induction of lipogenic enzyme by nicotine as reported by [21] and [22] where they established that there is induction of both glycerolkinase and glycerol- 3 – phosphate acyl transferase by nicotine. The result of this study is in line with the work of [15] and [16] where both recorded an increase in the triglyceride of smokers compared with non-smokers. It has also been documented that nicotine stimulates the release of adrenaline from the adrenal cortex leading to increased serum concentration of free fatty acids (FFA) which further stimulates hepatic synthesis and secretion of cholesterol [23] as well as hepatic secretion of very low density lipoprotein (VLDL) and hence increased TG [24].

High density lipoprotein of smokers was significant lower when compared with non-smokers' in this work. The low level recorded in this work might not be unconnected to the increase in the hepatic lipase and LCAT activity by nicotine. This result is in line

with the report of [25] that HDL concentration varies directly with the activity of hepatic lipase as well as LCAT. [26] also affirmed that hepatic lipase and LCAT are both nicotine inducible enzymes. A decreased HDL cholesterol concentration is associated with coronary heart disease [27]. This shows that smokers are predisposed to developing coronary heart disease earlier than their non-smoking counterpart.

Carl and Edward reported that clinically increase in LDL cholesterol is associated with increased risk of coronary heart disease [19]. The findings of this present work revealed high level of LDL cholesterol in smokers when compared with non-smokers. This finding is in consonance with the work of [21] where it was reported that increase in LDL level in cigarette smokers was due to the down regulation of LDL receptors and failure of receptor mediated endocytosis by metabolite of cigarette. [6] specifically attributed the down regulation of LDL receptor to inhibiting action of smoke allylamine and nicotine. [15] also reported high level of LDL in smokers, suggesting that there is increased LDL-Cholestarol synthesis in smokers which is dangerous to their health. LDL/HDL ratio was significantly higher in smokers as compared to that of controls. The result agrees with that of [28]. This ratio is an index of possibility of developing coronary heart disease (CHD) in smokers. In addition, the TG/HDL and TC/HDL ratio were significantly higher in smokers (p<0.05) than in non smokers. These ratios are useful as quick summary of disease risk. The TC/HDL ratio is of very high significance as values higher than accepted dangerous limit of >4.5 require intervention and indicate very high risk of CHD [29]. This shows that smokers are predisposed to developing CHD prematurely. The percentage distribution of LDL and HDL in the smokers as well as the control showed that the concentrations of these fractions are grossly altered in smokers.LDL was 63.9% of the total cholesterol in smokers as against 52.5% in non-smokers. HDL distribution is more affected; it's percentage (15.8%) in total cholesterol in smokers is less than half of that found in non-smokers (35.2%).This finding indicates that smoking habit predisposes individuals involved to various deleterious effects associated with increased LDL and reduced HDL concentrations. This result is in consonance with that obtained by [15]. Cigarette smoking, obesity, hypertension and increased cholesterol have been previously implicated as risk factors associated with atherosclerotic plaque formation [30].

CONCLUSION
The results of this study show that smokers are at much greater risk of developing atherosclerotic plaques and different heart diseases than non-smokers.

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