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## Effect of Chronic Cigarette Smoking on lipid profile – A Pilot study in North-Eastern Nigeria.

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### Abstract

Cigarette smoking is a major risk factor of atherosclerosis and coronary artery disease. It has been linked with alterations in lipid profile level but which fraction of lipids profile is affected by cigarette smoking remains controversial. In this study we evaluated the lipids profile among smokers in North-Eastern Nigerian. Total Cholesterol (Tc), High Density Lipoprotein Cholesterol (HDL-c) and Triglycerides (TG) were determined colorimetrically but Low Density Lipoprotein Cholesterol (LDL-c) was calculated. A total of One hundred and forty (140) current smokers, Twelve (12) Ex-smokers and Eighty four (84) controls were recruited. The current smokers were also categorized based on daily cigarette consumption; Light (<5 sticks/Day), Moderate (6-10 sticks/day) and Heavy (>10 sticks/day), and duration of smoking; Short (5-10 years), Medium (11-20years) and Long (>20 years). Clinical history, daily cigarette consumption and duration of smoking were obtained from a semi-structured questionnaire. TC, LDL-c, and TG were all significantly elevated in current smokers compared with control ( $p < 0.05$ ) with a significant decrease in HDL level ( $p < 0.05$ ). They all correlated with duration and quantity of daily cigarette consumption. There was no significant difference between ex-smokers and control, except for the significant increase in TG level observed. The results indicated an increased risk of dyslipidaemia in cigarette smokers which is pro-atherogenic and can potentially put smokers at a greater risk of cardiovascular diseases.

**Key words:** Atherogenic indices, Atherosclerosis, Cholesterol; Lipids; Cigarette smoking; Cardiovascular Disease.

### Introduction

Tobacco smoking is one of the most potent and prevalent addictive habits, influencing behaviour of human beings for more than four centuries. While the prevalence of tobacco smoking has declined in the developed world, it is increasing rapidly throughout the developing world and is a major threat to current and future global health (Edwards,

2004). It has been observed that 47.5% of men and 10.3% of women worldwide are current smokers (Dilyara *et al.*, 2007). In Nigeria, it has been reported that 7.2% of men and 0.3% of women are current smokers (GATS, 2012) while in North-Eastern Nigeria, 45.3% of men and 18.4% of women were reported as current smokers (Desalu *et al.*, 2006). Tobacco continues to be a major cause of mortality in the world. By 2030, if current trends continue, smoking will kill more than nine million people annually (World Health Report, 2002). The adverse health effects from cigarette smoking account for an estimated 443,000 deaths or nearly 1 of every 5 deaths, each year in the United States (CDC, 2008 and 2009).

More deaths are caused each year by tobacco use than by all deaths from human immunodeficiency virus (HIV), illegal drug use, alcohol use, motor vehicle injuries, suicides, and murders combined (CDC, 2008).

Lipids play an important role in many aspects of biological life which includes serving as hormones or hormone precursor, providing energy, storage function and metabolic fuels, and forming insulation to allow nerve conduction or to prevent heat loss (Adedeji and Etukudo, 2006). Cigarette smoking has been reported to be a major risk factor for peripheral, coronary and cerebral atherosclerotic vascular disease (Heitzer *et al.*, 1996; Raya *et al.*, 2009). Several reports have reported that cigarette smoking is associated with altered lipids profile but these reports have been consistent with which fraction of lipids profile is affected. Elevated levels of total cholesterol, triglycerides and LDL-C and a reduced level of HDL-C (Raya *et al.*, 2009; Kavita *et al.*, 2013; Naisargi *et al.*, 2013). Others reported no significant change in lipids profile between cigarette

smokers and control (Sirisali *et al.*, 1992) while Adedeji and Etukudo, reported that only HDL-C and total cholesterol level were altered by cigarette smoking.

Altered lipids profile is a major risk factor of atherosclerotic vascular diseases (Shai *et al.*, 2004; Raya *et al.*, 2009). There is still paucity of data on effect of cigarette smoking in North-Eastern Nigeria with such a high population of cigarette smokers. Thus, this study was undertaken to investigate further the effect of cigarette smoking on lipid profile level in North-Eastern Nigeria.

### Methodology:

A total of one hundred and forty (140) male current non-alcoholic cigarette smokers and twelve (12) ex-smoker were recruited from Maiduguri, Damaturu and Potiskum, Nigeria. Eighty-four (84) age-matched apparently healthy non-smokers were also recruited from the same metropolitan areas to serve as control. All participants in the study were included after obtaining written informed consent from them. Subjects (current smokers) were aged 25-60 years with mean age of  $46 \pm 9.2$  years and have history of cigarette smoking spanning for at least five (5) years. Those with medical conditions such as diabetes, hypertension, tuberculosis and those on medication known to affect biomarkers of cardiovascular, renal and hepatic diseases were excluded from the study. Clinical examination was done by a Medical Practitioner. Information on demographic, anthropometric and life style factors of each participant was obtained through a well-structured questionnaire.

### Grouping of subjects

The subjects were grouped as follows:

**Group 1:** Control

**Group 2:** Smokers

**Group 3i:** Ex-smokers

The smokers were further grouped according to intensity of smoking (number of Cigarette smoked per day)

**Group 2A:** Light smokers (1-5 sticks of cigarette daily)

**Group 2B:** Moderate smokers (6-10 sticks of cigarette daily)

**Group 2C :** Heavy smokers ( More than ten(10) sticks of cigarette daily)

And also according to the duration of smoking, the subjects were grouped into 3 groups;

**Group 2D :** Short Term duration (5-10 years of smoking) smokers

**Group 2E:** Medium Term Duration (11-20 years of smoking) smokers

**Group 2F:** Long Term Duration (> 20 years of Smoking) smokers

### Collection of blood sample

The blood sample from the subjects was collected after an overnight fast. The subjects were asked to have a light, fat free diet prior to the day of collection. The venepuncture was done in the cubital fossa. A tourniquet was used but was released just before collecting the blood sample to avoid artificial increase in concentration of serum lipids and protein. Fasting blood sample (15 ml) was aseptically collected from each participant into a plain bottle and left undisturbed for an hour. The blood sample was then centrifuged at 4000Xg for 10 minutes and serum collected was stored at  $-20^{\circ}\text{C}$ . Analysis of biochemical parameters was done within two weeks.

**Lipid profile determination:** Total cholesterol, triglyceride, HDL-cholesterol and LDL-cholesterol levels were measured spectrophotometrically using reagent kit obtained from Agappe Diagnostics, India.

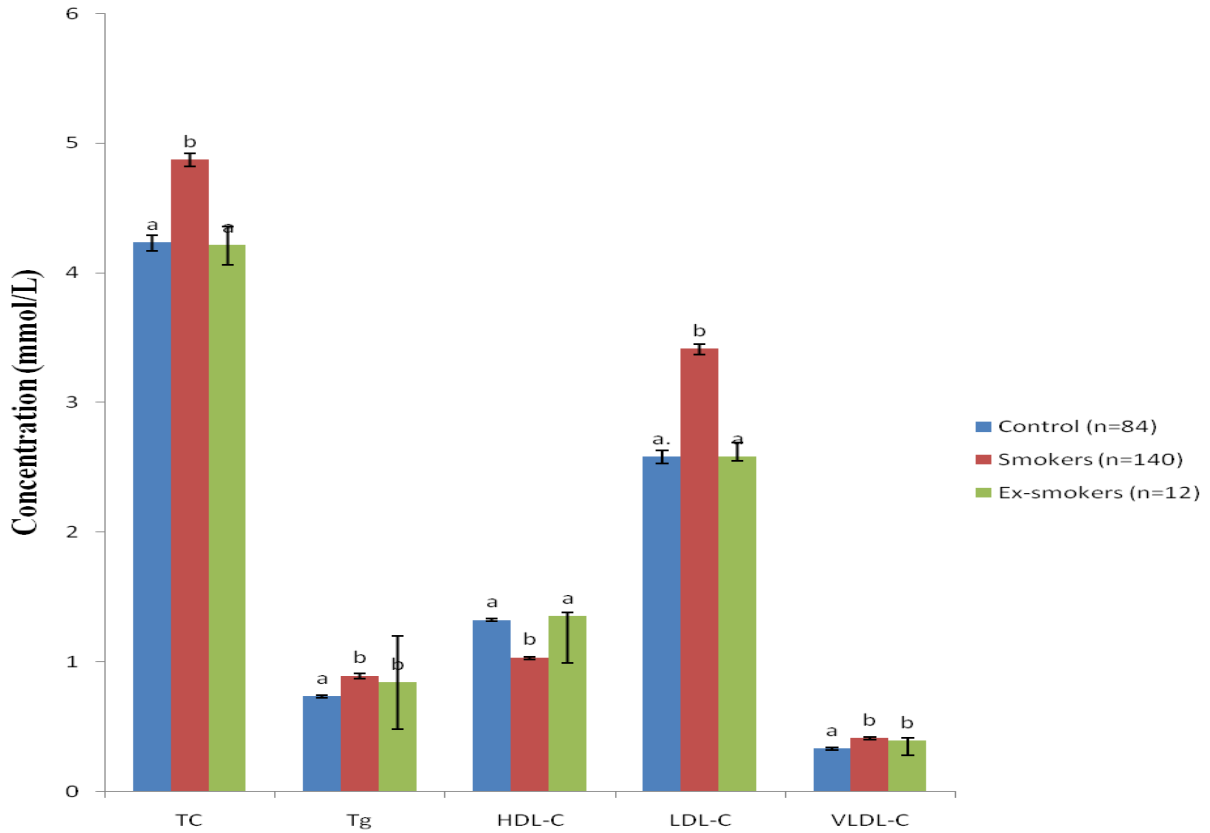
**Statistical analysis:** Data were presented as mean  $\pm$  standard Error of Mean (SEM). Data were subjected to One-Way Analysis of Variance (ANOVA) followed by test of significance. The level of statistical significance was taken at 5% confidence level.

### Results

Total cholesterol, triglycerides, low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C) concentrations were all significantly elevated ( $p < 0.05$ ) with a significantly decrease ( $p < 0.05$ ) in high density lipoprotein cholesterol (HDL-C) levels in smokers when compared to control. The total cholesterol, HDL-C and LDL-C concentrations in ex-smokers were not significantly different ( $p > 0.05$ ) when compared with that of controls. However, the concentration of triglycerides and VLDL-C were significantly elevated in ex-smokers when compared to control (Figure 1). There was a significant dosage dependent increase ( $p < 0.05$ ) in the levels of plasma total cholesterol and LDL-C of smokers. Triglycerides and VLDL-C concentrations though significantly elevated ( $p < 0.05$ ) when compared to control was not significantly different ( $p > 0.05$ ) between light and moderate smokers. High density

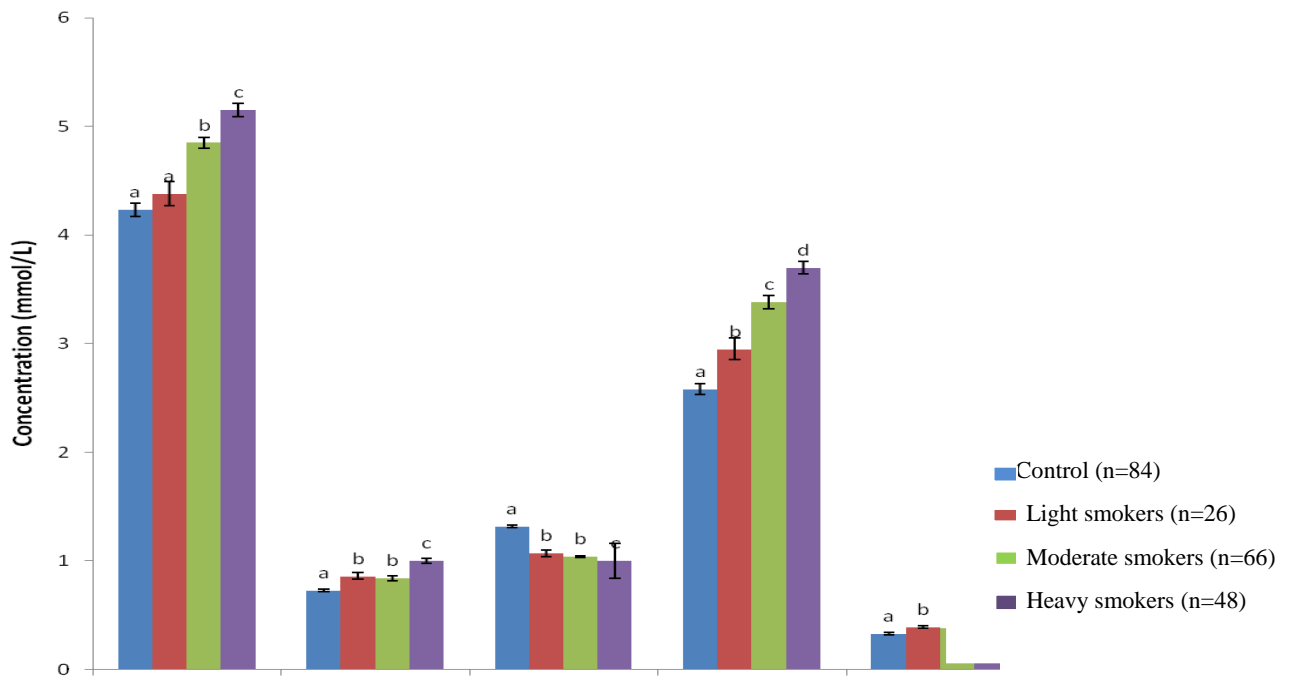
lipoprotein cholesterol (HDL-c) level decreases with the number of cigarette smoked per day (Figure 2). Total cholesterol, triglycerides, LDL-C and VLDL-C plasma concentration increase with the duration of

smoking. However, there was no statistically difference ( $p > 0.05$ ) between medium and long term smokers. In the same vein, HDL-C level decreased with duration of smoking (Figure 3).



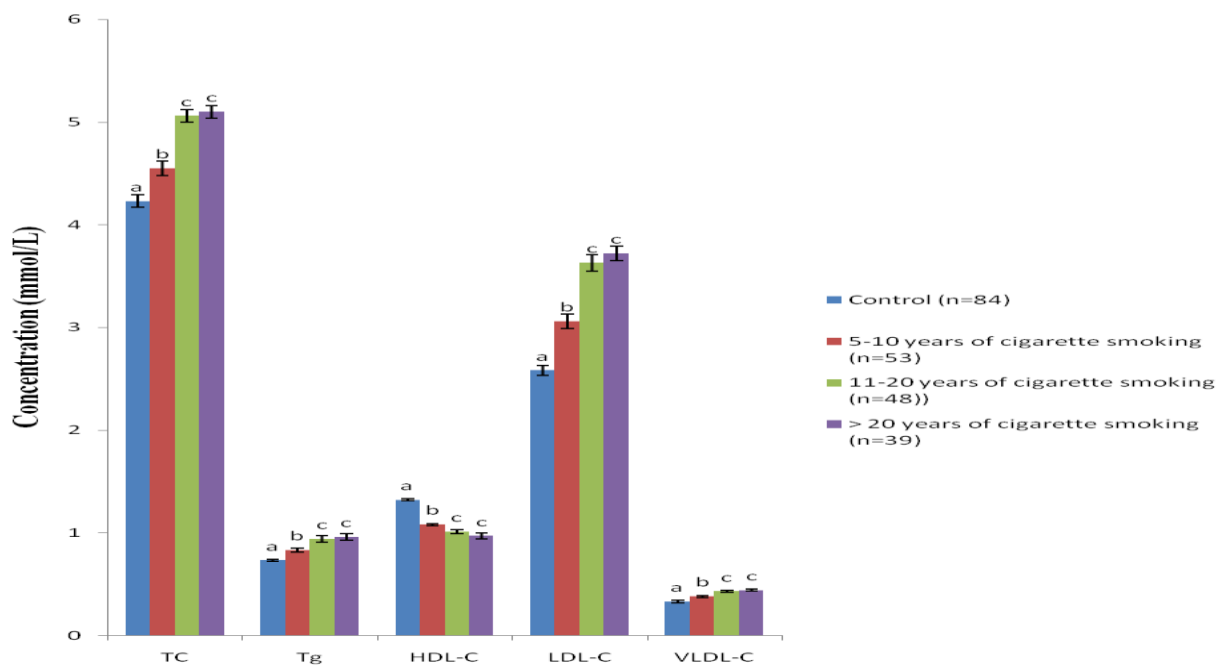
**Figure 1: Plasma lipids profile of smokers and ex-smokers**

TC- Total cholesterol, Tg-Triglyceride, HDL-C- High density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol, VLDL-C- Very low density lipoprotein cholesterol. Values with different superscripts are significantly different at  $p < 0.05$ . Values are mean  $\pm$  Standard errors of mean



**Figure 2 : Effects of level of cigarette smoking on plasma lipid profile**

TC- Total cholesterol, TG- Triglyceride, HDL-C- High density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol, VLDL-C- Very low density lipoprotein cholesterol. Values with different superscripts are significantly different at  $p < 0.05$ . Values are mean  $\pm$  Standard errors of mean



**Figure 3: Effect of cigarette smoking on plasma lipid levels of smokers**

TC- Total cholesterol, TG- Triglyceride, HDL-C- High density lipoprotein cholesterol, LDL-C- Low density lipoprotein cholesterol, VLDL-C- Very low density lipoprotein cholesterol. Values with different superscripts are significantly different at  $p < 0.05$ . Values are mean  $\pm$  Standard errors of mean

## Discussion

Cigarette smoking is an important and independent risk factor of atherosclerosis, coronary artery disease and peripheral vascular disorders. Several possible explanations have been offered for this relationship such as impaired integrity of the arterial walls, changes in blood lipids and lipoprotein concentration (Kshitish and Ksitish, 2010). However, studies to date have revealed inconclusive or conflicting report on the association between cigarette smoking and lipids profile (Devaranavadgi *et al.*, 2012). The elevated levels of plasma total cholesterol, low density lipoprotein cholesterol, triglycerides with a reduced high density lipoprotein cholesterol observed in this present study is suggestive of impairment in lipid profile as a result of cigarette smoking.

Elevation in plasma total cholesterol, low density lipoprotein cholesterol and a decreased high density lipoprotein cholesterol, is a risk factor of peripheral, coronary and cerebral atherosclerotic vascular disease (Meenakshiundaram *et al.*, 2010; Yan-ling *et al.*, 2012). These results are consistent with reports (Verkasetan *et al.*, 2006; Alharbi, 2011). It has been reported that the stimulation of the sympathetic adrenal system caused by nicotine leads to an increase in catecholamines secretion, resulting in increased lipolysis and increased concentration of plasma free fatty acids. This further results in increased secretion of hepatic triglycerides and VLDL cholesterol in the blood stream as seen in this study (Kshitish *et al.*, 2010). Cigarette smoking increases insulin resistance and thus causes hyperinsulinaemia. Hyperinsulinaemia decreased the activity of lipoprotein lipase resulting in elevation of LDL-C, VLDL-C, and triglycerides due to impair clearance of VLDL-C (Reaven, 1988; Meenakshilndaram, 2011).

The reduced plasma HDL-C levels observed among smokers in this study may be due to an increased catabolism and inactivation of the lecithin cholesterol acyl transferase (LCAT) System (Venerander *et al.*, 2011). Cessation of smoking reversed the low levels of HDL-C in smokers which is also cardio-protective. The reduced level of HDL-C in cigarette smokers and the increased exposure of vascular endothelium to potentially cholesterol rich lipoproteins may provide a mechanism whereby smoking predisposes to greater risk of developing atherosclerosis.

**Conclusion:** This study shows that cigarette smoking is associated with raised levels of LDL-C,

triglycerides and total cholesterol with a decreased HDL-C that is dependent on duration and intensity of cigarette smoking. These findings can potentially predispose smokers to dyslipidaemia which is an underlying risk factor of cardiovascular disorders.

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