

# NUTRITION OF



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# **Lipid Profile of Cigarette Smokers in Calabar Municipality**

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Abstract: Since it has been proven that high levels of LDL-Cholesterol predispose individuals to the risk of coronary heart disease, and that cigarette smoke contains toxicants that can disrupt normal metabolic processes, the present work was carried out to assess the lipid levels of smokers in Calabar, Nigeria, Total Cholesterol and triglyceride estimations were carried out using the enzymatic end point kit method. HDL-Cholesterol was estimated by precipitation of non-HDL lipoproteins, and estimations done, using the supernatant. Both LDL and VLDL were estimated by Calculation. Fifty (50) smokers and twenty (20) nonsmokers were recruited for the study. The findings showed that the difference in mean total cholesterol level between smokers (5.26±0.93mmol/L) and non smokers (4.06±0.38mmol/L) was statistically significant (p<0.05). The mean HDL - Cholesterol level of Smokers was (1.45±0.54mmol/L) and that for non-smokers (1.15±0.03 mmol/L). There was no statistically significant difference in the levels of LDL-Cholesterol between the Smokers (3.27±0.42mmol/L) and non smokers (2.37±0.15mmol/L) (p <0.05]. The VLDL-Cholesterol and total triglyceride values were (0.57±0.07mmol/L) and (1.22±0.27mmol/l) respectively for smokers and (0.57±0.02mmol/L) and (1.25±0.09 mmol/L) respectively for non-smokers. Among the smokers, the VLDL-Cholesterol + LDL-Cholesterol Value of (3.84±0.49mmol/L) was found to be statistically significant when compared with the (2.94±0.17mmol/L) value obtained for the non-smokers (P<0.05). These findings indicate that the risk of coronary heart disease may exist in smokers even when HDL-cholesterol levels are within the normal range and as such public enlightenment programmes should be stepped up to create awareness on the dangers of cigarette smoking.

Key words: Lipid profile, coronary heart disease, LDL-cholesterol, VLDL-cholesterol, HDL-cholesterol

#### Introduction

Lipids play essential roles in virtually all aspects of biological life. Some of these roles include serving as hormones or hormone precursors, helping digestion, providing energy, storage and metabolic fuels, acting as functional and structural components in biomembranes and forming insulation to allow nerve conduction or to prevent heat loss.

One of the major determinants of atherogenesis and coronary heart disease (CHD) is the plasma lipoprotein or lipid profile. In particular, the higher concentrations of low density lipo proteins (LDL), very low density lipo proteins (VLDL), triglycerides (TG) and lower concentration of high density lipoproteins correlate positively with development of severe and premature atherogenesis (Gordon et al., 1977; Castelli et al., 1977). There is overwhelming evidence from cohort studies that plasma cholesterol concentration is associated with increased risk of Coronary Heart Disease (CHD) and that decreasing plasma cholesterol concentration decreases the risk of CHD. It has been estimated that each 1% increase in plasma cholesterol concentration is associated with a 2.7% increase in risk (Law et al., 1994).

Cigarette smoking, hypertension, hyperlipdiaemia Obesity and Physical inactivity have long been recognized as environmental risk factors for CHD. Family history is also a powerful CHD risk factor, through a number of pathways among which is an increased oxidation of LDL particles that increase their atherogenecity (Fick *et al.*, 1996). Relatively few studies have been carried out on the level of cholesterol and lipoproteins in African groups. These include those on Nigerian Subjects (Taylor and Agbedana, 1983; Afia, 1988).

Since dietary and environmental factors influence lipid profile, the lipid profile of cigarette smokers in Calabar Municipality has been determined to see if variations occur in smokers.

# **Materials and Methods**

Fasting venous blood samples were collected from subjects, aseptically by venepuncture into plain sample containers. The blood was allowed to clot. The sample was spun at 3,000g and serum collected for analysis. Total cholesterol, triglyceride and HDL-Cholesterol were estimated using the enzymatic endpoint (kit) method. Both LDL and VLDL were estimated by Calculation.

### Results

Table 1 shows the mean Total Cholesterol values, HDL-Cholesterol, LDL-Cholesterol, VLDL-Cholesterol, and Total Triglyceride Values, of both smokers and non smokers.

Table 1: Mean Cholesterol and Triglyceride Values for Smokers and Non-Smokers

Cholesterol Fractions	Smokers (Mean±SD)	Non-Smokers (Mean±SD)	,b,	
Total Cholesterol	5.26±0.93 4.06±0.38		P< 0.05	
HDL - Cholesterol	1.45±0.54	1.15±0.03	P> 0.05	
LDL - Cholesterol	3.27±0.42	2.37±0.15	P< 0.05	
VLDL- Cholesterol	0.57±0.07	0.57±0.02	P> 0.05	
VLDL + LDL Cholesterol	3.84±0.49	2.94±0.17	P> 0.05	
Total triglyceride	1.22±0.27	1.25±0.09	P> 0.05	
n=	50	20		

n = number of subjects studied

The mean Total cholesterol values between Smokers (5.26 $\pm$ 0.93mmol/l) was statistically significant when compared with that of non smokers (4.06 $\pm$ 0.38mmol/L) P < 0.05.

The mean HDL-Cholesterol Level of Smokers (1.45±054mmol/L) was not statistically different from that of the non-smokers (1.15±0.03mmol/L) (P>0.05).

There was a statistically significant difference in the LDL-Cholesterol value between the Smokers (3.27±0.42mmol/L) and non-smokers (2.37±0.15 mmol/L) (P<0.05).

There were no significant differences in the levels of VLDL-Cholesterol and Triglyceride between the smokers and non smokers (P>0.05).

The Value of VLDL-Cholesterol+LDL-Cholesterol of (3.84±0.49 mmol/L) among smokers was found to be statistically significant when compared with 2.94±0.17mmol/L value for the non-smokers P<0.05.

## Discussion

The statistically different levels of mean total cholesterol in smokers when compared with that of non-smokers indicates that the smokers have an increased Serum Concentration of Cholesterol than non-smokers.

It is interesting to note in this study that although the HDL-Cholesterol for both groups seemed to be identical as the difference between their mean values were not found to be statistically different, there were striking differences in the total Cholesterol and Cholesterol Composition of the other lipid fractions (Table 1). The LDL-Cholesterol fraction for instance was 62% of the total cholesterol in Smokers; Suggesting that there is increased LDL- Cholesterol synthesis in smokers.

The percentage distribution of HDL, LDL, VLDL cholesterol in the non-smokers are as follows:

HDL - Cholesterol	-	28%	
LDL - Cholesterol	-	58.4%	
VLDL - Cholesterol	-	14%	

This suggests that in non smokers, VLDL and LDL Cholesterol made up to 72.4% of the total plasma cholesterol.

In contrast, however, in the smokers, the bulk of the Cholesterol, 62.2% appears to be from LDL-Cholesterol. Cigarette Smoking, Obesity, hypertension and increased level of cholesterol have been previously implicated as risk factors associated with atherosclerotic plaque formation (Brown and Goldstein, 1983). From this present study of our subjects from Calabar, Cross River State, one can deduce from our results that smokers are at a much greater risk of developing atherosclerotic plaques, than non-smokers.

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