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# Serum Lipids and Vitamin C Levels of Male Cigarette Smokers in Asaba, Delta State, Nigeria

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**Received 28 October 2013** **Accepted 17 November 2013**

## Abstract

Two hundred male subjects were selected randomly in parts of Delta State out of which 100 were cigarette smokers and another 100 non-smokers which served as control. Serum ascorbic acid level (AA), High Density Lipoprotein Cholesterol (HDL-C), total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein cholesterol (VLDL- C) were determined in all the samples taken from the subjects. Results were compared using the T-test at $p<0.001$. The serum level of vitamin C in smokers (0.81 ± 0.06 mg/dl) was found to be significantly lower than for non-smokers (1.10 ± 0.05 mg/dl); HDL-C level in smokers (30.82 ± 3..08 mg/dl) was found to be significantly decreased in smokers as against non-smokers (47.48 ± 2.85mg/dl); TC level in smokers (274.84 ± 11.16mg/dl) was significantly higher than non-smokers (166.68 ± 6.78mg/dl); TG level in smokers (250.38 ± 6.79mg/dl) was significantly higher than in non-smokers (149.39 ± 4.15mg/dl); LDL-C level in smokers (194.02 ± 11.13mg/dl) was significantly higher than in non-smokers (88.99 ± 7.56mg/dl); VLDL- C level of smokers (50.07 ± 1.35 mg/dl) was significantly increased in smokers as against non-smokers (29.86 ± 0.82mg/dl). The data obtained are useful for the control of cigarette smoking which causes heart diseases and reduction in anti-oxidation capacity of the body

**Key Words:** Serum Lipids, Vitamin C, Cardiovascular disease, Cigarette Smoking.

## Introduction

Cigarette smokers have been reported to be susceptible to coronary heart diseases which have also been associated with changes in lipids and lipoprotein levels in serum [1, 2]. Some other workers [3], in their separate works agreed that incidence of developing coronary heart disease is directly related to the number of cigarette sticks smoked and that sudden death is 2-4 times more in heavy smoker than in non-smokers. As many as 30% of all coronary heart disease deaths in the United States each year are attributed to cigarette smoking [3]. It has been estimated that each 1% increase in plasma cholesterol concentration is associated with a 2.7% increase in risk [4]. In particular, the higher concentrations of low density lipoproteins (LDL-C), very low density lipoproteins (VLDL-C), triglycerides (TG) and the lower concentrations of high density lipoproteins correlate positively with development of severe and premature atherogenesis [5]. Cigarette smokers are reported to be susceptible to develop coronary heart diseases [1]. This is because cigarette smoking causes oxidative stress and results in the release of free radicals. Cigarette smoke is a complex mixture of thousands of compounds containing

relatively high concentrations of oxidants or pro-oxidants [5], which include: nicotine, carbon monoxide, cadmium etc.[6].

Some workers [7] reported an inverse association between cigarette smoking and vitamin C. Others [8, 9] have also found an association between cigarette smoking, alcohol consumption and serum lipid profile. These reports point to the need to investigate the changes in the level of serum lipids and their ascorbic acid in serum from both cigarette smokers and non-smokers, with a view to bring to light the deleterious health effects of cigarette smoking in Delta State, Nigeria.

## Materials And Methods

The study was carried out in Ministry of Health laboratories, Asaba, Delta State. Two hundred participants were randomly selected from Agbor, Warri and Ugheli, which represents 3 senatorial districts in Delta State. The participants which were all males supplied questionnaire data including self-reported age, history of cigarette smoking including duration of smoking and average number of sticks per day, intake of vitamins and fruits, intake of alcohol and other drugs. Serum obtained from fasting blood samples were used for analysis of ascorbic acid (AA), total cholesterol (TC), triglyceride (TG), HDL-C, low density lipoprotein LDL-C, and VLDL-C. The calculations were done using the Freidewald's formula [12]. Subjects taking ascorbic acid and other drugs likely to interfere with the investigation were excluded. Also excluded were those suffering from maladsorbtion syndrome and those who declined to participate. Ethical clearance was obtained from the Ethical Committee of the Delta State Ministry of Health for this study.

Ascorbic acid was determined in serum using the 2, 6-dichlorophenolindophenol method of Harris and Ray [13]. Serum total cholesterol was estimated using the enzymatic method of Richmond [35]The serum triglycerides level was estimated using glycerol-3-phosphate oxidase -PAP method of Trinder [14]. The determination of HDL – cholesterol involved two stages: the first is the precipitation stage while the second is the estimation of the cholesterol using the enzymatic end – point method for cholesterol [15]. The VLDL-cholesterol and the LDL – C were calculated using the Friedwald formula [12].

### Statistical Methods

The distribution of the concentration of ascorbic acid as well as that of serumL total cholesterol, triglyceride, HDL-C, LDL-C and VLDL-C was examined. The association of ascorbic acid level with serum lipid and lipoprotein variables was examined. Analysis was performed using the SPFSS. Two – tailed p values of $\leq 0.01$ were considered to be statistically significant for the comparisons.

## Results

The findings in the study participants, with regard to the parameters investigated, including mean age, serum ascorbic acid, total cholesterol, triglycerides, HDL-C, LDL-C and VLDL-C are presented in Table 1. Table 2 shows the levels of ascorbic acid and the lipid profiles in non-smokers and the categories of smokers taken in batches of light smokers (B1), moderate smokers (B2), and heavy smokers (B3). Table 3 shows the ratios of mean±SD of VLDL-C/HDL-C, LDL-C/HDL-C, TC/HDL-C and TG/HDL-C. Table 4 shows the ratios of mean±SD of VLDL-C/HDL-C, LDL-C/HDL-C,

TC/HDL-C, and TG/HDL-C for the batches of smokers used (B1-B3). These Tables are presented below:

**Table 1** Showing result of analysis (Mean ± SD) of serum ascorbic acid, total cholesterol and various lipoproteins among cigarette smokers and non - smokers

| | AA | TC | TG | HDL-C | LDL-C | VLDL-C |
|---|---|---|---|---|---|---|
| **Smoker** | 0.81± | 274.84± | 250.38± | 30.82± | 194.03± | 50.08± |
| **n = 100** | 0.062 | 11.16 | 6.79 | 3.08 | 11.13 | 1.35 |
| **Non smokers** | 1.10± | 166.68± | 149.31± | 47.48± | 88.99± | 29.86± |
| **n = 100** | 0.050 | 6.78 | 4.15 | 2.85 | 7.56 | 0.82 |

Key: AA (Ascorbic acid), TC (Total cholesterol), TG (Triglycerides), HDL (High Identity Lipoprotein), LDL (low density lipoprotein), and VLDL (very low density lipoprotein).

**Table 2** Showing Mean ± SD of Ascorbic Acid and various Lipid profile in different categories of non –cigarette smokers

| PARAMETERS | NON SMOKERS ({A} N = 100) | LIGHT SMOKERS ({B1} N = 10) | MODERATE SMOKERS ({B2} N = 20) | HEAVY SMOKERS ({B3} N = 70) |
|---|---|---|---|---|
| **Ascorbic acid** | 1.1±0.05 | 0.90±0.01 | 0.82±0.04 | 0.80±0.06 |
| **Total Cholesterol** | 166.7±6.8 | 260±3.3 | 265±9.3 | 279±9.3 |
| **Triglycerides** | 149.3±4.14 | 246±8.6 | 249±8.2 | 252±5.5 |
| **VLDL – C** | 29.86±0.8 | 48±1.7 | 50±1.6 | 50.4±1.1 |
| **LDL – C** | 89.3±7.4 | 186.7±5.4 | 190.4±8.3 | 198.4±9.4 |
| **HDL – C** | 47.48±2.9 | 32.0±3.5 | 31.0±3.3 | 30±30 |

**Table 3** Showing mean ± SD of VLDL-C/HDL-C, LDL-C/HDL-C, LDL-C/ HDL-C, TC/HDL-C and TG/HDL-C ratios in cigarette smokers and non- smokers

| | VLDL-C/HDL-C | LDL-C/HDL-C | TC/HDL-C | TG/HDL-C |
|---|---|---|---|---|
| **Smokers n = 100** | 1.64±0.16 | 6.36±0.79 | 8.99±0.95 | 8.20±0.83 |
| **Non – smokers n = 100** | 0.63±0.04 | 1.89±0.23 | 3.52±0.26 | 3.15±0.20 |
| | P<0.001 | P<0.001 | P<0.001 | P<0.001 |

**Table 4** Showing Mean ± SD of VLDL-C/HDL-C, LDL-C/HDL-C, TC/HDL-C, TG/HDL-C in different categories of cigarette smokers and non -smokers

| PARAMETERS | NON SMOKERS ({A} N = 100) | LIGHT SMOKERS ({B1} N = 10) | MODERATE SMOKERS ({B2} N = 20) | HEAVY SMOKERS ({B3} N = 70) |
|---|---|---|---|---|
| **VLDL-C/HDL-C** | 0.62±0.04 | 1.48±0.16 | 1.57±0.16 | 1.66±0.18 |
| **LDL-C/HDL-C** | 1.88±0.23 | 5.93±0.69 | 6.1±1.0 | 6.6±0.74 |
| **TC/HDL-C** | 3.51±0.26 | 8.51±0.84 | 8.6.±0.9 | 9.2±9.0 |
| **TG/HDL-C** | 3.15±0.20 | 7.40±0.81 | 8.0±0.8 | 8.3±0.84 |

## Discussion

The results of this study show that cigarette smokers had significantly ($P < 0.001$) lower level of vitamin C (ascorbic acid) than non-cigarette smokers who had never smoked (Table 4.1). This is in accordance with earlier findings of some workers [7, 10, 11, 12], who did similar work on cigarette smokers and found low level of vitamin C in them. Also heavy smokers had lower levels of vitamin C (Ascorbic acid) but not statistically significant ($P < 0.001$) when compared with moderate and light cigarette smokers (Table 2). This is in contrast with the reports of other worker [5, 7] who attributed this hypovitaminosis C in cigarette smokers to either impaired vitamin C absorption or increased metabolism. The increase metabolism may result from the depletion of body stock of vitamin C due to adverse effect of chemical substances in cigarette smoke such as nicotine [13]. The processes leading to this loss have been stated by some other worker [14]. The presence or absence of malabsorbtion was not investigated in this study.

The findings that total serum cholesterol levels of cigarette smokers was significantly ($P < 0.001$) higher when compared with the apparently healthy non cigarette smokers (Table 4.1), is in agreement with the work of some workers in their separate studies [15, 16, 17] in which elevated levels of total cholesterol among cigarette smokers were found. Also, the serum total cholesterol levels of heavy cigarette smokers was found in this study to be significantly higher ($P < 0.001$) when compared with moderate and light smokers (Table 4.2). This is in accordance with the work of some workers [9,18,19] who did similar work on smokers. A plausible explanation for the observed effect of cigarette smoke on serum cholesterol may be due to decreased activation of the enzyme 7 $\propto$- hydroxylase by the low level of vitamin C. This enzyme enhances the conversion of plasma cholesterol into bile acids. A decrease in vitamin C level as found in smokers causes a block in the synthesis of bile acids [14]. This leads to accumulation of cholesterol in serum as found in this study

A higher triglyceride level was observed in smokers when compared with apparently healthy non – smokers (Table .2). This finding is in conformity with similar works on cigarette smokers [15, 20, 17], though other workers [16] had a contrasting view that there is no significant

difference between the mean of triglycerides levels in cigarette smokers and non-cigarette smokers. Also, heavy cigarette smokers have higher triglycerides but not statistically significant when compared with moderate and light cigarette smokers (Table 2). This is in contrast with the findings of some workers (18, 9), who did similar work on smokers. Hypertriglyceridemia is generally due to an imbalance between synthesis and clearance of VLDL-C in the circulation [21]. It is presumed that nicotine stimulates sympathetic adrenal system leading to increased secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma fatty acids, which further results in increased synthesis of hepatic triglycerides, along with VLDL-C in the blood stream [22]. The increase in triglycerides found in this study may also be due to the effect of cigarette smoke. This can be deduced from the findings of some workers [23, 2 ]. The same workers [23], reported that cigarette smoking increases plasma concentration of lactate, insulin and growth hormone. It is known that insulin and growth hormone promote the production of VLDL-C by increasing the production of Apo E and Apo B – 48 and by stimulating lipolysis in the adipose tissues and triglyceride synthesis in the liver.

The presence of hyperinsulinaemia in smokers leads to increased triglycerides, LDL-C and VLDL-C due to decreased activity of lipoprotein lipase [24]. This may be the resultant effect of hypertriglyceridemia as observed in smokers in our study.

This study also shows decreased HDL-C levels in cigarette smokers when compared with non-cigarette smokers. This is in consonance with the work the report of some workers [15, 17, 20] who in their separate works found lower levels of HDL-C in cigarette smokers. However, this report is in contrast to the findings of other workers [16], which showed in their work that there is no significant difference between the mean of cigarette smokers and non- cigarette smokers. Also, there is a lower HDL-C level in heavy cigarette smokers but not statistically significant when compared with moderate and light cigarette smokers (Table .3). This finding is at variance with the work of other workers [29, 9)] who found significant difference between different groups based on the number of cigarette sticks smoked per day.

The findings in this study can be evaluated from an observation (15) that low levels of HDL-C in cigarette smokers and concluded that it is a threat to the development of atherosclerosis and increased coronary heart disease. Direct relationship of smoking towards coronary heart disease has been observed by Multiple Risk Factor Intervention Trail Research Group (MRFIT) [26], which reported that increase in HDL-C level by 1mg/dl was associated with decrease in the risk of coronary heart disease by 3%. Cigarette Smoking which contains nicotine influences the lipid levels by decreasing lipoprotein lipase activity, increasing hepatic lipase and decreasing cecithin cholesterol acyltransferase (LCAT) activity [27]. Also, cigarette smoke decrease oestrogen levels which further leads to decreased HDL-C [22] .This may be a plausible explanation for the low level of HDL -C observed in our results.

Our results also showed an elevated serum LDL-C in cigarette smokers n compared with apparently healthy non-smokers (Table 1). This is in consonance with earlier reports [15, 16 and 20]. There is also a high level of serum LDL-C in heavy cigarette smokers but was not statistically

significant when compared with moderate and light cigarette smokers (Table 2). This is contrast with the report of other workers [9]. Some workers [28] have reported that LDL-C is the carrier of 70% of total cholesterol and it transports cholesterol to tissues. Cigarette smoking is known to produce free oxygen radicals in our body [29] and these free radicals reduce amount of reactive oxygen species (ROS) scavengers and reduce oxidative damage. Free radicals also oxidize LDL- C which increases atherosclerosis. An excess of free oxygen radicals production due to lack of antioxidant, may however increase the risk of heart disease as found in cigarette smokers.

The VLDL -C of cigarette smokers was found to be significantly higher when compared with apparently healthy non cigarette smokers (Table .2). This is in conformity with an earlier report [15] of a similar work on cigarette smokers which found a high level of VLDL-C in them. Other workers [16] have a contrasting report which says that there is no significant difference observed between cigarette smokers and non-cigarette ‘smokers when their mean were compared. The VLDL-C level of heavy cigarette smokers was observed to be higher but not significant when compared with mean of moderate and light cigarette smokers (Table 2). This is in contrast with another report [9] which observed a significant difference (P< 0.001) among the different stratum of cigarette smokers. Presence of hyperinsulinaernia in cigarette smokers leads to increased cholesterol, LDL-C, VLDL-C and triglycerides due to decreased activity of lipoprotein lipase [30].

The Atherogenic Index (AI) which is TC/HDL-C, Coronary Risk Index (CRI) which is LDL-C/HDL-C and TG/HDL-C ratio (Table 3) are significantly higher (P < 0.001) in cigarette smokers when compared with that of non-cigarette smokers. There are also high ratios of Atherogenic Index, Coronary Risk Index and TG/HDL in heavy cigarette smokers which were not statistically higher when compared with moderate and light cigarette smokers (Table 4). These findings are in agreement with an earlier report [16]. In their work they observed that, with increase in these ratios, risk of developing coronary heart disease also increases proportionately. Coronary risk index ratio has been proven to be a reliable predictor of cardiovascular risk and is better than LDL-C alone or HDL-C alone [30].The ratio can also be used to monitor the effectiveness of lipid lowering therapies [31]. In addition to these, TG/HDL-C ratio and Atherogenic Index are useful as quick summary of disease risk in cigarette smokers.

Atherogenic and Coronary Risk Indices are of great significance as values higher than accepted dangerous limit of > 4.5 require intervention and indicate very high risk of coronary heart disease [32]. Atherogenic and Coronary Risk Indices estimates the net effect of two way traffic of cholesterol in and out of tissues. These indices have been suggested to be the most important predictor of premature development of coronary heart disease [33]. Our results show that smokers have Coronary Risk Index and Atherogenic Index of 6.36 and 1.89, respectively as against the 8.99 and 3.52 of non-smokers. Also the TG/HDL-C ratio of cigarette smokers was found to be 8.20 while non-cigarette smokers were found to be 3.15. These data have confirmed that smokers are persons at higher risk of coronary heart disease. Heavy cigarette smokers have a CRl and Al of 6.6 and 9.2 respectively as against moderate

cigarette smokers (6.1 and 8.6), Light cigarette smokers (5.9 and 8.5) and Non cigarette smokers (1.88 and 3.51). Also the TG/HDL ratio of smokers (8.2) is above the recommended value of 8.0 [33].These ratio has been demonstrated to be an independent predictor of incident CHD, [34].

## Conclusion

This study has established serum lipid profile and vitamin C level among Nigerian male cigarette smokers. It has also affirmed that cigarette smokers experience increases in some lipid fractions principally LDL-C which predisposes to coronary heart disease. Also, ascorbic acid which is an antioxidant was found to be lower in cigarette smokers indicating greater depletion of body's stock in smokers. This study has also revealed that number of cigarette sticks smoked per day does not significantly increase the vulnerability of the smoker to a greater impact of the effect of cigarette smoking.

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