



RESEARCH ARTICLE



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Conflict of Interest: None Declared !

## Effect of Cigarette Smoking on Lipid Profile In Male at Collage of Police and Low Khartoum, Sudan

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### Abstract

#### Background:

Smoking is one of the environmental factors which can alter normal lipid profile. It is one of the major risk factors in the genesis of coronary atherosclerosis and development of coronary heart disease. The present study aimed to compare the lipid profile between smoker and non smoker and to evaluate the durational significance on lipid profile in the smokers and to clarify the influence of daily cigarette smoking on the components of lipid profile in Sudanese youth males.

#### Materials and Methods:

Out of 200 apparently healthy male subjects, 100 were smokers [26.2±5.2Years] and 100 were non smokers [24.0±4.6Years]. The subjects were asked to fast overnight and early morning blood samples collected and analyzed for lipid profile [Cholesterol, Triglyceride, High density lipoprotein and Low density lipoprotein] by using Cobas integra 400 plus Roche analyser.

#### Results:

A significantly increased of serum Cholesterol, Triglyceride and Low density lipoprotein with significant decreased in serum High density lipoprotein level in smokers as compared to non smokers and same results were found in smoker group with > 15 cigarettes smoked per day while with increase duration of smoking the TC & LDL-C were increased, TG showed no difference while the HDL-C was decreased showing greater risk of these persons to atherosclerosis and coronary heart disease.

#### Conclusions:

This study concluded that smoking causes alteration in lipid profile. Increased duration of smoking and number of smoked cigarettes / day causes more dyslipidaemia .This smoking might be related in the alteration in serum lipid levels increases risk for coronary artery disease.

#### Key words:

Cigarette Smoking, Coronary Heart Disease, Dyslipidaemia, Lipid profile and Tobacco.

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## 1. INTRODUCTION

Cigarette smoking is generally considered as associated with increased risk of a variety of medical disorders. Several studies provide the evidence that tobacco is strongly associated with altering the normal status of the lipid profile [1]. Cigarette smoking is now acknowledged to be one of the leading causes of preventable morbidity and mortality and is one of the largest single preventable causes of ill health in the world [2].

Nicotine and other toxic substances from tobacco smoke are absorbed through the lungs into the blood stream and are circulated throughout the body. These substances narrow or damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a nonsmoker [3]. Nicotine and other toxic substances from Tobacco smoke are absorbed through the lungs into the blood stream and are circulated throughout the body. These substances damage the blood vessel walls, which allow plaques to form at a faster rate than they would in a non smoker [4]. Nicotine increases the amount of bad fats (total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG)) circulating in the blood vessels and decreases the amount of good fat (high-density lipoprotein cholesterol (HDL-C) availability [4]. Nicotine induces oxidative stress, generates free radicals that attack on the membrane lipids resulting in the formation of malondialdehyde (MDA), which causes peroxidative, tissue damage [5].

Lipoprotein oxidation is presumed to occur in the artery that may generate superoxide radicals, hydrogen peroxide or lipid peroxides outside the cell may contribute to the oxidation of LDL [6]. These silent effects begin immediately and greatly which increase the risk for heart disease and stroke [7]. An increased level of MDA has been documented in smokers by several authors [8, 9], an evidence of intensification of lipid peroxidation processes which may cause chronic stress for endothelial cells. On the other hand, it can also re-orientate enzymatic systems of the arachidonic acid cascade towards intensified TXA<sub>2</sub> synthesis [8, 9]. In this way cigarette smoking substantially hastening the risk of coronary heart disease and ischemic stroke [10, 11], to date, no statistical data are available on tobacco smoking among Sudanese people. Moreover, there have been no studies showing the relationships of cigarettes smoking according to the number of cigarettes smoked per day and the duration of smoking and their influence on serum lipid profile. Globally there is much controversy about which components in the lipid profile are mainly altered in response to cigarette smoking, and whether those lipid profile components influence other parts directly or indirectly

and vice versa. The present study was undertaken to clarify the influence of daily cigarette smoking on the components of lipid profile.

## 2. MATERIALS & METHODS:

The study was conducted from January 2013 to September 2013 at Sudan collage of police and low. A total of two hundred healthily male were enrolled in this study in the age of 18 – 32 Years, 100 were smokers [26.2±5.2Years] and 100 were non smokers [24.0±4.6Years].

The local ethics committee approved the study. Before participation, volunteers were fully informed of the nature and purpose of the study and written consent was obtained from each.

The smoker group was sub classified according to smoking number of cigarette / day into two group smoking less than 15 cigarettes/ day and smoking more than 15 cigarettes/ day, and according to the duration of smoking sub classified to smoking for less than 10 and smoking for more than 10 years.

Blood samples were obtained following an overnight fasting. Samples were withdrawn from a cubital vein into blood tubes. The serum was then separated from the cells by centrifugation at 3000 r/min for 10 min and immediately stored on ice at 4°C.

Serum Cholesterol, Triglyceride, HDLC and LDLC was measured by using the enzymatic method using Cobas integra 400 plus -Roche, the reference value are [TC < 200mg/dl, TG < 200mg/dl, HDLC > 55mg/dl and LDL 49-172mg/dl].

## 3. STATISTICAL ANALYSIS:

Data were expressed as mean ± standard deviation (SD). The means were compared using Independent sample t.test. Analysis was two-tailed and a p-value ≤ 0.05 was considered as statistically significant.

## 4. RESULTS:

Baseline characteristics of the 200 participants (male youth), 50% of them was non smoker (n = 100) aged 24.06±4.6 years and 50% were Cigarettes smoker and they were 26.18±5.2 years of age.

The mean ± SD values for serum cholesterol, triglycerides, LDL-C and HDL-C are given in Table 1. All the components of lipid profile studied (Cholesterol, triglycerides and LDL-C) were found significantly increased for smokers compared to the healthy control non-smoking subjects, while the HDL-C were decreased in smoker group compared to the non smoker group. The values of significance for various comparisons are given in Table 1.

Table 2 In this set of data, the subjects were categorized according to average number of cigarettes [Less than 15 and above 15 cigarettes / day] table 2 showed that there was significant increased in the

mean levels of cholesterol, Triglyceride and LDL while HDLC was significantly decreased among smoking group [ $>15$  cigarettes/day versus  $< 15$  cigarettes/day.

Table 3 In this set of data, the subjects were categorized according to the duration of smoking [Less than 10 and above 10 Years] table 3 showed that the mean levels of total cholesterol and LDLC were increased with increase in the duration of smoking, HDLC shows a decreased with the increase duration of smoking, while the duration of smoking has no effect on serum Triglyceride.

Serum level	Smoker [n=100] Mean±SD	Non smoker [n=100] Mean±SD	P value
Cholesterol [mg/dl]	191.29±46.3	133.3±24.5	0.000*
Triglyceride [mg/dl]	147.8±62.4	100.3±21.7	0.000*
HDLC [mg/dl]	43.2±7.4	55.9±10.8	0.000*
LDL [mg/dl]	117.6±41.9	56.6±18.6	0.000*

The table shows the mean ± SD and probability (P). T-test was used for comparison. P value ≤ 0.05 was considered significant \*\*indicates highly significant ≤ 0.01

**Table1: Comparison of lipid profile between smoker and non smoker**

Serum level	Smoking >15 cig/ day [n=39] Mean±SD	Smoking <15 cig/ day [n=61] Mean±SD	P Value
Cholesterol [mg/dl]	202.19±48.9	174.23±36.3	0.003*
Triglyceride [mg/dl]	162.86±69.9	124.36±38.7	0.002*
HDLC [mg/dl]	41.75±7.3	45.6±7.1	0.010*
LDL [mg/dl]	126.5±42.8	103.8±36.7	0.008*

The table shows the mean ± SD and probability (P). T-test was used for comparison. P value ≤ 0.05 was considered significant \*indicates highly significant ≤ 0.01

**Table2: Influence of daily number of smoking cigarettes on lipid profile**

Serum level	Smoking >10 years [n=51] Mean±SD	Smoking <10 years [n=49] Mean±SD	P value
Cholesterol [mg/dl]	204.7±52.2	177.3±34.7	0.003*
Triglyceride [mg/dl]	152.1±72.1	143.4±50.7	0.489
HDLC [mg/dl]	39.7±5.5	46.9±7.5	0.000*
LDL [mg/dl]	133.5±44.8	101.2±31.3	0.000*

The table shows the mean ± SD and probability (P). T-test was used for comparison. P value ≤ 0.05 was considered significant \*indicates highly significant ≤ 0.01

**Table 3 Influence of duration of smoking on lipid profile**

## 5. DISCUSSION:

Cigarette smoking is one of the leading causes of preventable morbidity and mortality that usually starts in adolescence and continues into adult life [1]. In the developing world, tobacco consumption is rising especially among youth [2]. The tobacco smoking is responsible for premature development

of CAD Cardiovascular

disease [3] and abnormal levels of risk factors such as serum lipid and lipoprotein levels, hypertension, and smoking are related to the earliest stages of atherosclerotic CAD [4].

Various mechanisms leading to lipid alteration by smoking are: (a) nicotine stimulates sympathetic adrenal system leading to increase secretion of catecholamines resulting in increased lipolysis and increased concentration of plasma free fatty acids (FFA) which further result in increased secretion of hepatic FFAs and hepatic triglycerides along with VLDL in the blood stream [12] (b) Fall in oestrogen levels occurs due to smoking which further leads to decreased HDL [13]; (c) Presence of hyperinsulinaemia in smokers leads to increased cholesterol, LDL and TG due to decreased activity of lipoprotein lipase[14].

We conducted this study to assess the impact of smoking on lipid profile. In our study all subjects in both smokers and non smokers group were apparently healthy adult males. In our study Serum TC, TG and LDL were significantly higher in smokers as compared to non-smokers and the serum HDL level was significantly lower in smokers as compared to non-smokers. Our findings are in accordance with the findings of many research workers. The change in the serum cholesterol & lipoprotein levels became more marked with the number of cigarettes smoked per day and duration of smoking in years. This finding has been substantiated by Imamura et al.[5], N S Neki[6] Contrary to the above findings Diricana M et al[7] did not find significant differences in serum TC, TG, LDL HDL levels between smokers and nonsmokers. Nesje LA et al [8] also found no significant difference between smokers and non-smokers concerning triglycerides and total cholesterol. These differences may due to ethnic's variation in population in previous studies.

Dyslipidemia is a well-established risk factor for the development of coronary artery disease. Our study demonstrated presence of dyslipidemia in chronic smokers. The main limitation of this study is that important factors which may contribute to the cardiovascular risk factors among youth such as dietary habits, physical activity and genetics were not included.

## 6. CONCLUSION:

These findings suggest that smoking might be related in the alteration in lipid profile adversely causing dyslipidemia in smokers and the changes become more marked with increased duration / Years and number of cigarette/day smoked. Smoking plays the key role for atherosclerotic process and with coronary artery disease.

## 7. RECOMMENDATIONS:

It is strongly recommended to avoid smoking for the benefit of cardiac health. It is important to establish a visible and audible communication aids and through schools and colleges explaining risks of smoking on the cardiovascular system and other systems.

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