

## Research Article

# The Effect of Cigarette Smoking on Fasting Lipid Profile: A Single Center Study

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## Abstract

**Background:** Cigarette smoking is one of the leading causes of preventable death globally. Smoking causes dyslipidemia prone to atherosclerosis that is associated with cardiovascular disease (CVD) risk.

**Objectives:** The aim of this study was to assess the effect of cigarette smoking on lipid profile in healthy adult male smokers.

**Methods:** This cross sectional study was conducted at the Department of Physiology, Rajshahi Medical College, Rajshahi, Bangladesh, from July 2017 to June 2018. A total of 160 healthy male subjects aged above 30 years were selected; among them 80 were smokers and 80 were age matched apparently healthy non smokers for comparison. The smokers were regularly smoking for at least 1 year. Fasting blood samples from all study subjects were collected and analyzed for lipid profile [Total cholesterol (TC), High Density Lipoprotein-cholesterol (HDL- C), Low Density Lipoprotein-cholesterol (LDL- C), and Triglyceride (TG)] using enzymatic-colorimetric method.

**Results:** The smokers had significantly increased levels of total cholesterol, triglyceride, LDL- C and decreased HDL- C level in comparison to non-smokers ( $<0.05$ ). It was observed that total cholesterol, serum triglycerides and LDL- C levels were highest in heavy smokers, less in moderate smokers and least in mild smokers, but HDL- C levels were highest in mild smokers less in moderate smokers and least in heavy smokers.

**Conclusion:** Continuous cigarette smoking is associated with dyslipidemia. Smoking increases levels of total cholesterol, triglyceride, LDL- C and decrease HDL- C level. All components of lipid profile are affected by the heaviness of smoking.

**Keywords:** Cigarette Smoking; Dyslipidemia; Lipid Profile; Non-Smoker; Smoker

## 1. Introduction

Cigarette smoking is addictive and harmful to health. Cigarette smoke contains a variety of compound including nicotine, oxidants and free radicals that are capable of irritating or promoting oxidative damage leading to various degenerative pulmonary and cardiovascular diseases as well as cancer [1-2]. Smoking has severe adverse effect on blood lipoprotein level and associated with a greater risk for developing atherosclerosis, IHD, hypertension, stroke and peripheral vascular disease [3-5].

Cigarette smoking is one of the most potent and prevalent addictive influencing behavior of the human beings for over four centuries [6]. Smoking continues to be the second major cause of death in the world [7]. It is estimated that globally smoking kill nearly six million people each year [7]. Smoking is one of the modifiable risk factors of many chronic diseases such as cardiovascular disease, chronic obstructive lung disease, asthma and cancer [8]. A smoker is a person who smokes any tobacco product either daily or occasionally. Daily smoker is a person, who smokes any tobacco product at least once a day

and occasional smoker is a person, who smokes, but not every day. Nonsmokers are those who do not smoke at all [9]. Cigarette smoking has been reported to be very high among males as compared to females [10]. Cigarette smoking is a common problem in Bangladesh and also a major public health problem associated with morbidity and mortality [11].

During recent years a considerable body of evidence emerges suggesting that cigarette smoking cause dyslipidemia prone to atherosclerosis [12-15]. Both smoking and different component of serum lipids play an important role in functional as well as structural changes in arterial system which ultimately leads to cardiovascular disease (CVD) [4]. The changes become more marked with increased duration and number of cigarette smoked per day [1, 15]. Smokers had higher fasting triglycerides (TG) and lower high density lipoprotein cholesterol (HDL-C) levels and an increased proportion of small dense low density lipoprotein particles [12]. Craig *et al.* found that, smoker had higher level of serum total cholesterol (TC) and very low density lipoprotein cholesterol (VLDL-C) and lower levels of high density lipoprotein cholesterol (HDL-C) than non-smoker [13]. Lee *et al.* suggested that, nicotine which is the main pharmacologically active component of cigarette stimulates sympathetic nerve activity and causes release of catecholamine leading to lipolysis which increases the plasma concentration of free fatty acid and decrease plasma HDL- Cholesterol fraction [14]. It has been proposed that smoking leads to dyslipidemia in the form of increased serum total and low-density lipoprotein cholesterol levels, triglycerides level and decreased high density lipoprotein cholesterol level [15-16]. There is scarce evidence to assess the impact of cigarette smoking on lipid profile. In this present study, attempt was made

to determine the effect of smoking on lipid profile among smokers.

## 2. Methodology

This cross sectional study was conducted at the Department of Physiology, Rajshahi Medical College, Rajshahi, Bangladesh from July 2017 to June 2018. This study was approved by the Ethical Review Committee Rajshahi Medical College, Rajshahi, Bangladesh. A total of 160 male subjects were selected according to the selection criteria. Among them 80 persons were smoker and rest 80 persons were nonsmoker. All individuals were apparently healthy, non-alcoholic and well nourished. Male subjects age 30 years and above having history of smoking at least 1 stick every day for last 1 year were included in smoker group. Age and body mass index (BMI) matched participants having no current or past history of smoking were considered as control group. Smokers were divided into three groups (categories) according to the number of cigarette sticks smoked per day. Subjects who smoked 10 or less than 10 sticks per day was consider as mild smokers, those who smoked more than 10 sticks but less than 20 sticks per day were consider as moderate smokers and persons smoked more than 20 sticks per day were categorized as heavy smokers.

### 2.1. Collection, processing and analysis of blood samples

The healthy adults who fulfilled the inclusion criteria were enrolled in this study. After taking informed written consent, complete history of each participant was taken and their relevant physical examination were done accordingly, all data were recorded in a data collection sheet. Then, following an overnight fasting (10-12 hours), 3 ml of venous blood from each study subject was drawn into a plane test tube

(from the antecubital space of their forearm) by venipuncture with taking all aseptic precautions. Each test tube then kept in vertical position for 30 minutes to allow coagulation. Serum was separated by centrifugation at 3000 rpm for 10 minutes in room temperature (22°C-25°C). Thereafter, serum was utilized for estimation of fasting lipid profile [Total cholesterol (TC), High Density Lipoprotein-cholesterol (HDL- C) and Triglyceride (TG)] using semi auto analyzer (EMP-168 Biochemical Analyzer) and that was measured by enzymatic-colorimetric method. Low Density Lipoprotein-cholesterol (LDL- C) was estimated by Friedewald equation as- total cholesterol minus high density lipoprotein-cholesterol (HDL- C) minus triglycerides divided by five [17]. The biochemical tests were performed at the Biochemistry Laboratory, Department of Biochemistry, Rajshahi Medical College, Rajshahi, Bangladesh.

## 2.2. Data processing and statistical analysis

Data was analyzed by computer based software program Statistical Package for Social Sciences

(SPSS) for windows version 20. Results of the analysis were expressed as mean  $\pm$  standard deviation and frequency with percentage. Student's unpaired t-test and ANOVA test were performed for the statistical analysis of data. Probability value less than 0.05 was considered as the significant value.

## 3. Results

A total of 160 healthy adult male subjects were participated in this study. Among them 80 male smokers were taken as a study group and 80 nonsmokers male healthy subjects were taken as control group. Among the study subjects; mean( $\pm$ SD) age (years), weight (kg), height (cm) and body mass index [BMI (kg/m<sup>2</sup>)] of the smokers were 46.20 $\pm$ 9.14 years, 64.77 $\pm$ 7.60 kg, 161.48 $\pm$ 8.37 cm and 21.80 $\pm$ 2.82 kg/m<sup>2</sup> and that of nonsmokers were 45.91 $\pm$ 9.47 years, 66.58 $\pm$ 9.45 kg, 163.09 $\pm$ 5.60 cm and 22.42 $\pm$ 2.29 kg/m<sup>2</sup>. There was no statistically significant difference between the groups ( $p > 0.5$ ) (Table- 1).

**Table- 1:** Distribution of basic health parameters of the study subjects (N=160)

Parameters	Smoker (n=80) (mean $\pm$ SD)	Non-smoker (n=80) (mean $\pm$ SD)	p-value
Age (yrs)	46.20 $\pm$ 9.14	45.91 $\pm$ 9.47	0.673 <sup>ns</sup>
Weight (Kg)	64.77 $\pm$ 7.60	66.58 $\pm$ 9.45	0.184 <sup>ns</sup>
Height (cm)	161.48 $\pm$ 8.37	163.09 $\pm$ 5.60	0.158 <sup>ns</sup>
BMI (Kg/m <sup>2</sup> )	21.80 $\pm$ 2.82	22.42 $\pm$ 2.29	0.128 <sup>ns</sup>

The test significance was calculated using unpaired t-test; ns= Not significant

Table 2 shows the distribution of cardiovascular parameters among the study groups. It was observed that the mean( $\pm$ SD) pulse rate of smokers

(78.75 $\pm$ 6.67 beats/min) was comparatively higher than that of non-smokers (76.82 $\pm$ 6.72 beats/min). The mean( $\pm$ SD) systolic blood pressure (BP) of the

smokers was significantly higher than non-smokers ((130.62±13.57 mm Hg versus 122.76±15.65 mm Hg, p=0.001). While mean(±SD) diastolic blood

pressure (BP) of the smokers was also significantly higher than that of non-smokers (84.77±9.47 mm Hg versus 79.46±10.26 mm Hg, p=0.001) (Table- 2).

**Table- 2:** Distribution of cardiovascular parameters of the study groups

Variables	Smoker (n=80) (mean±SD)	Non-smoker (n=80) (mean±SD)	p-value
Pulse (beats/min)	78.75±6.74	76.82±6.72	0.073 <sup>ns</sup>
Systolic BP (mm Hg)	130.62±11.40	122.76±15.65	0.001 <sup>s</sup>
Diastolic BP (mm Hg)	84.77±9.47	79.46±10.26	0.001 <sup>s</sup>

The test significance was calculated using unpaired t-test; s= Significant; ns= Not significant.

In this study, the number of subjects in mild smoker group were 16(20%), the number of subjects in moderate smoker group were 37(46.2%) and the

number of subjects in heavy smoker group were 27(33.8%) among total 80 smokers subjects (Table- 3).

**Table- 3:** Distribution of smokers based on number of cigarettes smoked per day (n=80)

Smokers group	Number of subjects (n)	Percentage (%)
1-10 (mild smoker)	16	20.0%
11-20 (moderate smoker)	37	46.2%
>20 (Heavy smoker)	27	33.8%
Total	80	100.0%

Data analysis revealed that mean(±SD) serum total cholesterol (TC), serum triglyceride (TG) and serum low density lipoprotein cholesterol (LDL-C) levels were significantly higher among smokers compared to non-smokers (205.56±51.58mg/dl, 179.11±68.19 mg/dl and 127.79±48.50 mg/dl versus 172.05±29.76

mg/dl, 121.05±44.19 mg/dl and 111.05±31.57 mg/dl, p<0.001). On the other hand, smokers had significantly lower level of mean(±SD) serum high density lipoprotein cholesterol (HDL- C) than non-smokers (38.07±4.25 mg/dl versus 40.82±3.92 mg/dl, p<0.001) (Table- 4).

**Table- 4:** Distribution of the fasting lipid profile among study groups [N=160]

Lipid profile (mg/dl)	Smokers (n=80)	Non smokers (n=80)	p value
Total cholesterol	205.56±51.58	172.05±29.76	<0.001 <sup>s</sup>
Serum triglycerides	179.11±68.19	121.05±44.19	<0.001 <sup>s</sup>
Serum LDL-C	127.79±48.50	111.05±31.57	<0.001 <sup>s</sup>
Serum HDL- C	38.07±4.25	40.82±3.92	<0.001 <sup>s</sup>

The test significance was calculated using unpaired t-test; s= significant

It was found that, total cholesterol, serum triglycerides and LDL- C levels were highest in heavy smokers, less in moderate smokers and least in mild smokers, but HDL- C levels were highest in

mild smokers less in moderate smokers and least in heavy smokers. The difference of these values with non-smokers was found statistically significant ( $p < 0.05$ ) (Table- 5).

**Table- 5:** Comparison of fasting lipid profile among smokers group according to the degree of smoking

Lipid profile (mg/dl)	Nonsmokers (n=80)	Mild smokers (n=16)	Moderate smokers (n=37)	Heavy smokers (n=27)	p value
Serum total cholesterol	172.05±29.76	189.68±45.55	201.85±50.99	215.13±53.67	<0.05 <sup>s</sup>
Serum triglycerides	121.05±44.19	135.31±63.33	188.33±63.29	191.32±67.61	<0.05 <sup>s</sup>
Serum HDL- C	40.82±3.92	39.00±7.00	37.89±1.55	30.93±1.34	<0.05 <sup>s</sup>
Serum LDL-C	111.05±31.57	122.43±46.15	123.00±50.78	133.60±48.44	<0.05 <sup>s</sup>

The significance of difference was calculated using ANOVA test; s=significance

## 4. Discussion

Cigarette smoking is a global escalating public health problem that estimated to kill 6 million people and causes over hundred billion dollars of economic damage worldwide each year [18]. It was documented that cigarette smoking is an independent risk factor for atherosclerosis, coronary artery disease and peripheral vascular disorders [19]. Smoking also increases the incidence of non-insulin dependent diabetes mellitus, dyslipidemia and insulin resistance [19].

The present study was carried out to assess the effect of cigarette smoking on serum lipid profile [total cholesterol, triglyceride, HDL- cholesterol, LDL- cholesterol] among 80 adult male cigarette smokers and 80 adult male nonsmokers. Age and body mass index (BMI) was matched among smokers and non-smokers. They all were non diabetic and non-alcoholic healthy males. The mean(±SD) age of the study subjects was 46.20±9.14 years in smokers and that was 45.91±9.47 years in non-smokers.

### 4.1 Association between smoking and lipid profile

#### 4.1.1 Serum total cholesterol (TC)

In this study, mean(±SD) serum total cholesterol was significantly higher in smokers in comparison to non-smokers (205.05±51.58 versus 172.05±29.76,  $p < 0.001$ ). This observation was compatible with a previous study which revealed higher cholesterol concentration among smokers than that of non-smokers [20]. Similar result was obtained from the study of Joshi *et al.*, where mean total cholesterol of study group (smokers) was significantly higher than control group (nonsmokers) [21]. On the contrary, Gupta V *et al.* found no significant difference in cholesterol concentration among smokers and nonsmokers [22]. This may be due to variation in sample selection criteria, different food habit of the study subjects, comparatively younger study group etc. Moreover, the duration of smoking was not prolonged in this study.

The total cholesterol value in mild smokers was 189 mg/dl and those in moderate smokers was 201mg/dl and in heavy smokers was 215 mg/dl, these

differences were statistically significant ( $p < 0.05$ ). These findings were in accordance with a related previous study [21]. Increase cholesterol levels and coronary heart disease (CHD) are observed in cigarette smokers [1-2]. Higher level of cholesterol is associated with CHD [2]. Cigarettes smoking substantially increase the risk of coronary heart disease and ischemic stroke [3-5]. The result of total cholesterol of this study was different from other studies. There are many factors that are involved in the different results, such as- diet habit, daily physical activity, and genetic/ethnic diversity.

#### 4.1.2 Serum Triglyceride (TG)

In this study, mean( $\pm$ SD) serum triglyceride was significantly higher in smokers than non-smokers ((179.11 $\pm$ 68.19 versus 121.05 $\pm$ 44.19,  $p < 0.001$ ). This finding was consistent with a couple of previous study and reported that smokers had higher value of serum triglyceride compared with non-smokers [15, 23]. The mean value of serum triglyceride was significantly higher in heavy smokers than moderate and mild smokers ( $p < 0.05$ ). This finding was supported by related previous studies [21, 24]. However these results were inconsistent with another study as showed smokers had lower level of serum triglyceride compared with non-smokers [25].

Studies have suggested that triglyceride level was one of the important factor leading to cardiovascular disease (CHD) [1-2]. Although triglyceride is a risk factor for developing CHD has been suggested by various research workers [3-5]. The reduce lipoprotein lipase activity in smokers as observed by Freeman *et al.* may explain impaired triglyceride metabolism and higher triglyceride levels [26].

#### 4.1.3 Serum low density lipoprotein-cholesterol (LDL- C)

In this study, mean( $\pm$ SD) serum LDL- C was significantly higher in smokers (127.79 $\pm$ 48.50) in comparison to non smokers (111.05 $\pm$ 31.57), which was statistically significant ( $< 0.001$ ). This finding was similar with a previous study and found that smokers had higher value of LDL- C than non-smokers [4]. Contrary report was also documented that there was no significant difference of LDL- C between smokers and non-smokers [27]. Furthermore, this study found that the subjects smoking more than 20 cigarettes per day had significantly higher LDL- C (133.60 mg/dl) as compared to those who smoked 11-20 cigarettes per day (123 mg/dl) and in those who smoked 1-10 cigarettes per day (122.43 mg/dl).

Similar finding have been reported in a couple of previous study [21, 28]. It has been described that nicotine contained in cigarette increased the circulatory pool of atherogenic LDL- C through accelerated transfer of lipids from HDL and impaired clearance of LDL particles from plasma compartment and hence LDL- C in the arterial wall increased [21, 28]. Smoking results an increase in oxidized LDL- C level which plays a key role in the development of atherosclerosis that also raising the cardiovascular disease (CVD) risk [3-5].

#### 4.1.4 Serum high density lipoprotein-cholesterol (HDL- C)

Studies reported that there is an inverse relationship between smoking and serum HDL- C, also this inverse relationship is dose dependent [24, 29]. In this study, mean( $\pm$ SD) serum HDL- C was significantly higher in non-smokers (40.82 $\pm$ 3.92) in comparison to smokers (38.07 $\pm$ 4.25), which was

statistically significant ( $<0.001$ ). This finding was supported by similar previous studies which reported that there was a fall in HDL- C level by 3-5 mg/dl in smokers and that was a strong cardiovascular risk factor [3, 30-31].

However no significant difference between smokers and non-smokers in HDL- C level was found in a separate study [32]. Moreover we observed a significant inverse association between serum HDL- C level with severity of smoking (mild smokers  $39.00 \pm 7.00$  mg/dl, moderate smokers  $37.89 \pm 1.55$  mg/dl and heavy smokers  $36.93 \pm 1.34$  mg/dl,  $p < 0.05$ ). Similar finding have been reported in a couple of previous study [24, 29]. While, according to Rashan *et al.* there was no significant difference in the level of HDL- C accompanied with increased smoking intensity [3].

This decreased level of HDL- C in smokers may be explain by smoking induce increase catecholamine cause increase very low density lipoprotein-cholesterol (VLDL- C) and decrease in HDL- C concentration, thus smoking promotes coronary heart disease (CHD) and atherosclerosis by lowering the anti- atherogenic factor like- HDL- C [4].

Several studies demonstrated dose related increases in total cholesterol, LDL- C and TG as well decrease in HDL- C among mild moderated and heavy smokers [20, 21, 24, 28, 29]. This current study also observed dose-dependent relationships of smoking with lipoprotein. Several studies have shown an association between cigarette smoking and altered serum lipid and lipoprotein concentration, but many of these have lacked enough statistical power to establish a firm association. By combining the results of individual studies in the present analysis we have

shown conclusively that smoking is associated with significantly higher serum concentration of total cholesterol (TC), triglycerides (TG) and low density lipoprotein cholesterol (LDL- C) while lower serum concentrations of high density lipoprotein cholesterol (HDL- C) and this association is dose dependent.

Recent epidemiological studies have shown that cigarette smoking is an independent risk factor for cardiovascular disease [1-5, 8]. One of the strength of our study was that we included only healthy adults who were absolutely free from any disease. So it was possible for us to detect any minute change in lipid profile due to cigarette smoking. Our study proves that smoking affects the lipid profiles even before onset of disease process. Further extensive studies are needed to improve our understanding about pathogenesis of disease by smoking and to strengthen our knowledge to depend detrimental effect of smoking.

## 5. Conclusion

This study concluded that serum total cholesterol (TC), triglyceride (TG) and low density lipoprotein cholesterol (LDL- C) levels were found significantly higher in smokers than non-smokers. Contrary to this, serum high density lipoprotein cholesterol (HDL- C) level was found significantly higher in non-smokers than smokers. It was also observed that there was a direct relation of increasing the value of LDL- C, TG, and TC with number of cigarette smoked per day. Opposite phenomenon, that is gradual decrease in the level of HDL- C was noted with number of cigarette smoked per day.

## Limitation of the study

There were several limitations in this study. Firstly, it was a single center study and a relatively small



sample size. Second, duration of smoking exposure was not monitored. Third, only male subjects and young adults were included in this study. Fourth, effect of exposure to active and passive smoking were not observed separately. Moreover, other health related abnormalities were ignored related with smoking.

### Recommendation

A long term large population based multicenter study needed to confirm the results of this current study.

### Conflict of interest

The authors declare that they have no conflicts of interest regarding the publication of this paper.

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