



ISSN: 0975-833X

RESEARCH ARTICLE

THE INFLUENCE OF CIGARETTE SMOKING ON LIPID PROFILE

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ARTICLE INFO

Article History:

Received 09<sup>th</sup> March, 2015  
Received in revised form  
17<sup>th</sup> April, 2015  
Accepted 05<sup>th</sup> May, 2015  
Published online 27<sup>th</sup> June, 2015

Key words:

Cigarette smoking,  
Total cholesterol,  
High density lipoprotein,  
Low density lipoprotein,  
Triglycerides

ABSTRACT

Cigarette smoking remains the leading cause of preventable premature morbidity and mortality in many countries around the world. It has been established that one of the constituents of tobacco i.e Nicotine has considerable influence on increasing the lipid levels in blood. Derangement of cholesterol metabolism leads to increased triglyceride concentration in blood. This study was undertaken in Thanjavur medical college to evaluate lipid profile in young cigarette/bidi smokers in Thanjavur, and compare it with nonsmokers in fasting state. It rules out whether any correlation exists between chronic smokers and lipid level. This study was conducted on 40 healthy male cigarette/bidi smokers and compared with 40 nonsmokers. The smokers must have smoked minimum of daily 3-5 cigarette/bidi for 2 years duration. The study group includes male smokers within the age group of 25-35 years. Age and weight matched non-obese, nonsmokers served as control. The lipid profile parameters include serum Total cholesterol, LDL, HDL and Triglycerides. The tests were carried out in a semiautoanalyzer. Alterations in the lipid profile are statistically significant in chronic smokers. They have significant increase in serum level of Total cholesterol, LDL, TG and decrease in HDL. So chronic cigarette smoking makes definite change in lipid profile.

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**Citation:** Dr. S. Sudha, Dr. R. Vindoha and Dr. R. Girija, 2015. "The influence of cigarette smoking on lipid profile", *International Journal of Current Research*, 7, (6), 16968-16970.

INTRODUCTION

Lipids play essential role in virtually all aspects of biological life. One of the major determinants of atherosclerosis and coronary heart disease is variation in lipid profile. Dietary and environmental factors influence the lipid profile (John camm, 2002). Smoking is one of the environmental factor, which alter normal lipid profile (Antonio *et al.*, 2005). Cigarette smoke contains toxicants that can disrupt normal metabolic processes. Smoking of cigarettes/bidi leads to increase in the concentration of serum total cholesterol, triglycerides, LDL-Cholesterol, and fall in the level of antiatherogenic HDL - Cholesterol as reported by various workers (Yadav *et al.*, 2005). Smoking creates a state of permanent inflammation and an imbalance in the lipid profile that leads to lipid accumulation in liver and blood vessels of heart and aorta. Smoking increases the Acetyl COA and cholesterol synthesis. Smoking initiates mechanism that generates superoxide radicals and hydrogen peroxides (Palanisamy Pasupathi *et al.*, 2009). It may contribute to oxidation of LDL and generate potent pro-atherogenic mediators like oxidized LDL. Neski *et al.* (2002) has done the study in persons, who smoke

less than 20 cigarette per day for 8 years, and they have recorded mean serum total cholesterol 181 mg/dl in smokers in comparison with control 164mg/dl. In (Pairat Saengdith, 2008) study lipid profile was measured in priest in Bangkok to asses the effect of smoking. He found the serum HDL values are comparatively lower in smokers than non smokers. This study was undertaken to evaluate lipid profile in young cigarette /bidi smokers in Thanjavur and compare it with non smokers in fasting state.

MATERIALS AND METHODS

This study was undertaken to prove the influence of cigarette smoking on lipid profile. The study done here is cross sectional study of lipid profile among smokers and non smokers. The subjects were selected through convenient non probability sampling. The study was conducted during May 2009 to July 2010. Subjects in this study were selected in Thanjavur Town, Tamil Nadu, India. The following criteria were used in selection of cases for study group.

Inclusion criteria

History of smoking cigarette/bidi  
Smokers must have smoked minimum of three cigarettes /bidi for two years duration.

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Age group of subjects between 25 to 35 years.  
Only males were included in the study.

### Exclusion Criteria

Persons with obesity, Diabetes Mellitus, Hypertension, Renal Failure and hepatic impairment.  
History of Alcoholism.  
Persons on treatment for any major illness.  
History of drug intake like B- blockers, lipid lowering drugs, and Thiazide diuretics.

### Control group

Forty subjects with age and sex matched normal healthy individuals with no history of smoking were selected as control. They must be free from Diabetes Mellitus and other chronic diseases.

### Procedures for Investigations

Informed consent was obtained from all subjects prior to enrolment into the study. A detailed history of both groups was obtained. A proforma with detailed history of smoking of subjects were filled. Height, Weight and Body mass index were measured and taken into account. Under general examination, CVS, RS, Abdomen and CNS were examined. Blood pressure and pulse rate of all subjects were recorded. Routine blood investigations were done for all subjects. They were subjected to Laboratory investigations including Blood glucose, Hemoglobin estimation, RBC Count and Total count of WBC were done for all subjects.

### Bio Chemical analysis

Collection of blood samples for biochemical assays was done after fasting for at least 12 hours. Blood samples were collected in morning. 5 ml of blood from ante-cubital vein from each subject was collected aseptically without prolonged venous stasis, in disposable sterile 10 ml syringes and was allowed to clot and stored. Samples were processed within 1 hour for quantitative lipoprotein cholesterol measurements using the vertical spin centrifugation technique. Serum was obtained by centrifugation for 4 min at 3000 rpm and was then transferred into properly labeled sterile vials and stored at -20°C till the performance of lipid profile. Serum TC, serum TG and HDL-C tests were evaluated by standard enzymatic kits methods, whereas LDL-C was calculated according to Friedwald *et al.* (1972).

Total cholesterol was estimated using CHOD-PAP method enzymatic assay. Serum Triglycerides was estimated employing standard GPO-POD Method of enzymatic assay.

HDL Cholesterol was estimated by PTA method using autokit-Roche.

LDL Cholesterol was calculated by the Friedwald and Fredickson's Formula.

LDL- Total cholesterol – (Triglycerides /5+ HDL cholesterol)

These tests were carried out in a semi Auto analyzer.

## RESULTS

80 subjects took part in this study. Out of 80 subjects, 40 were under study group. They are chronic smokers of minimum 3-5 cigarettes per day for two years. Control group has remaining 40 subjects who are non smokers. Their age varied from 25 to 35 years. All the data was statistically evaluated according to steel and Torrie by using statistical package for social sciences version 10. Quantitative variables were represented as mean, SD, whereas frequencies and percentages were applied for qualitative variables. Independent sample t test and chi-square test were carried out to assess the significance of parameters like serum total cholesterol, Triglycerides, LDL and HDL Cholesterol.

**Table 1. Observation of parameters by independent sample t test**

Variables	Group	N	Mean	Std. Deviation	P value
AGE	Smokers	40	31.9500	2.5008	0.0005
	Non smokers	40	30.9750	3.3930	
TC	Smokers	40	227.0250	40.5655	0.0005
	Non smokers	40	169.7800	30.1817	
TGL	Smokers	40	192.2150	45.1124	0.0005
	Non smokers	40	119.5650	29.9078	
HDL	Smokers	40	37.7625	3.5275	0.0005
	Non smokers	40	44.6775	5.2937	
LDL	Smokers	40	147.9875	35.4774	0.0005
	Non smokers	40	106.9875	20.7483	

(P value < 0.05 is taken as significant)

The serum total cholesterol, Triglycerides and LDL values are higher in smokers than control group with significant p value of <0.05. HDL cholesterol was lower in smokers than non smokers. The p value is <0.05. Table 1

## DISCUSSION

The results of present study indicate that the persons, who smoke 3 to 5 cigarettes/Bidi for minimum of two years duration shows significant, change in lipid profile than non smokers. By applying independent sample t test, significant differences were observed in serum TC, TG, LDL-C and HDL-C. By applying chi-square ( $\chi^2$ ) test, significant association of lipid profile was found among two groups. From the results of the present study, it may be concluded that, chronic cigarette smoking induce alteration in serum lipid level in the direction of increased risk for coronary artery disease. Mammias *et al*<sup>6</sup> done their study in medical students in Greece. They found raised serum TC in smokers. Similar results were observed in our study. Craig *et al.* (2004) done an analysis of published data about relation between cigarette smoking and serum lipid profile. He found that in overall studies smokers had

significantly higher serum TG which is similar to our present study. Abdulla abbassi<sup>1</sup> done his study in 102 persons, Among them 72 were smokers and 32 were non smokers. He recorded that serum LDL-C are significantly increased in smokers, which is in conformity with our study. In Chandra sekkarpur<sup>4</sup> study, they found serum HDL values are lower in smoking group than non smoking counterparts. This study correlates with the result of present study. The constellation of these altered lipoproteins suggests that smokers are at a high risk for the development of coronary heart disease. These risk profiles may be helpful in developing preventive cardiovascular strategies for smokers.

### Conclusion

Alterations in the lipid profile are statistically significant in chronic smokers. There is statistically significant increase in serum total cholesterol, LDL and Triglyceride level. The reduction in serum –HDL level is significant in study group.

### Abbreviations used in this study

LDL – Low Density Lipoproteins  
 HDL- High Density Lipoproteins  
 TC – Total Cholesterol  
 TG – Triglycerides

### Acknowledgement

We thank our Dean, our subjects who gave us full cooperation for conducting this work.

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