

Effect of Chronic Cigarette Smoking on Lipid Profile

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ABSTRACT

This study is designed to observe the effects of chronic cigarette smoking on lipid profile including triglycerides, total cholesterol, HDL-C, and LDL-C. A total of 102 (30 non-smokers and 72 smokers) apparently healthy male subjects with similar dietary habits and socioeconomic conditions were selected for this study. Total cholesterol, triglyceride and LDL-C were significantly increased in smokers (group B), when compared with non-smokers (Group A), while the level of HDL-C was significantly decreased in smokers as compared to the non-smokers. Total cholesterol, triglycerides and LDL-C were significantly increased in heavy smokers (Group B2) as compared to the moderate smokers (Group B1) and that of HDL-C was significantly decreased in heavy smokers as compared to moderate smokers.

Key Words: Triglycerides, HDL-C, LDL-C, Smokers

INTRODUCTION

Cigarette smoking is a major risk factor for cardiovascular disease, cancer, chronic obstructive pulmonary disease and cerebrovascular disease^{1,2,3,4}. Cigarette smoking induces a wide variety of physiological responses. Some of which appear likely to be involved in accelerating atherogenesis or increasing the probability of thrombosis⁵. These responses include reduction in plasma HDL cholesterol followed by elevation of LDL cholesterol and triglycerides⁶. Therefore, proposed study was designed to assess the effect of chronic cigarette smoking on lipid profile.

METHODOLOGY

This study was carried out in the Department of physiology, Basic Medical Sciences Institute, JPMC, Karachi. A total of 102 apparently healthy male subjects of age ranging between 25-35 years, were selected. These were divided into different groups:
Group A=Non Smokers (n = 30)

Group B=Smokers with h/o smoking for >5 years (n=72)

Group B1=Moderate smokers who consumed < 20 cigarettes/day (n = 36).

Group B2=Heavy smokers who consumed > 20 cigarettes/day (n = 36)

Subjects, who were taking multivitamin supplementation or suffering from any acute or chronic diseases, excluded from the study. Fasting blood sample (5 ml) was taken from all the selected subjects. Serum total cholesterol, triglycerides and HDL-C were determined by the enzymatic – colorimetric method and LDL-C was calculated according to Friedwald formula.

RESULTS

Comparison of total cholesterol, triglycerides, HDL-c and LDL-c in non-smokers (Group A), smokers (Group B), moderate smokers (Group B1) and heavy smokers (Group B2) was given in tables 1 & 2.

Table 1: Comparison of Lipid Profile in Non Smokers (Group A) and Smokers (Group B)

Parameters (mg/dl)	Non-smokers (Group A) (n=30)	Smokers (Group B) (n=72)	Level of Significance (A vs B)
Total cholesterol	173.27±1.70	190.82±1.75	HS
Triglycerides	115.17±2.99	161.82±1.95	HS
HDL-C	41.23±0.95	33.25±0.28	HS
LDL-C	109.00±2.13	125.21±1.80	HS

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Table 2: Comparison of S-Cholesterol, Triglycerides, HDL-C and LDL-C in Non-Smokers (Group A), Moderate Smokers (Group B1) and Heavy Smokers (Group B2)

Parameters (mg/dl)	Non-smokers (Group A) (n=30)	Moderate Smokers (Group B1) (n=36)	Heavy smokers (Group B2) (n = 36)	Level of Significance		
				A Vs B1	A Vs B2	B1 Vs B2
Total cholesterol	173.27 ± 1.70	184.36 ± 2.17	197.28 ± 2.32	HS	HS	HS
Triglycerides	115.17 ± 2.99	151.42 ± 2.21	172.22 ± 2.11	HS	HS	HS
HDL-C	41.23 ± 0.95	34.44 ± 0.4	32.06 ± 0.26	HS	HS	S
LDL-C	109.00 ± 2.13	119.63 ± 2.37	130.80 ± 2.40	HS	HS	HS

DISCUSSION

Cigarette smoking adversely affects the lipid profile⁵. It is said that smoking acts in two ways. First by altering the concentration of serum lipids and secondly by modifying the lipids chemically⁹. Total cholesterol in smokers was found to be higher than those of non-smokers and difference was highly significant statistically. A dose dependent increase was also observed among the smokers. Similar findings were reported by Craig et al (1989)⁷ and Hallfrish et al (1994)⁸.

Serum triglycerides in smokers were found to be elevated as compared to non-smokers and difference was highly significant statistically. These findings are consistent with the findings of Willett et al (1983)¹⁰, Craig et al (1989)⁷, Facchini et al. (1992)¹¹ and Marangon et al. (1998)¹². HDL-C in smokers was found to be significantly lower than those of non-smokers. Same findings were reported by Mjos (1988)¹³, Craig et al. (1989)⁷, Facchini et al (1992)¹¹ and Hallfrisch et al (1994)⁸. HDL-c provides protection against atherosclerosis by competing with the LDL-c thus mobilizing cholesterol away from the atherosclerotic lesion^{13,14}. Serum LDL-C was significantly elevated in smokers as compared to non-smokers and difference was highly significant statistically. Similar findings were reported by Craig et al (1989)⁹ Hughes et al (1993)¹⁵ and Sniderman et al (1997)¹⁶. The most abundant class of atherogenic lipoproteins in human plasma is low-density lipoproteins (LDL), which transports cholesterol from liver to tissues¹⁷. We know that nicotine stimulates the release of adrenaline, which in turn raises plasma concentrations of free fatty acids (FFAs) through enhanced lipolysis and FFA mobilization from adipose tissue¹³. Free fatty acids stimulate the hepatic secretion of VLDLs, triglycerides and also cholesterol⁷. An inhibition of lipoprotein lipase activity by smoking has been thought to increase the levels of triglycerides and LDL-C¹⁸.

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