Original Article

Correlation between HDL-C and Smoking in Teachers Residing in Shiraz, Iran

M Zamiriam[1], MJ Zibaeenezhad[1], R Mozaffari[1], ST Heydari[2], F Abtahi[1], Sh Khosropanah[1], AR Moaref[1], MA BabaeeBigi[1], K Aghasadeghi[1]

[1]Cardiovascular Research Center, [2]Health Policy Research Center, Shiraz University of Medical Sciences, Shiraz, Iran

Background: It has been established that serum HDL-C is a main predictor for cardiovascular diseases. The aim of this study was to assess the relationship of HDL-C to smoking in teachers residing in Shiraz in order to improve the health status of the group under study and community as a whole.
Methods: The present study comprised a total of 3115 teachers working in Shiraz recruited in a prospective cohort study. Of these, two groups of 235 smokers and 235 non-smokers were randomly selected for evaluation of cardiovascular risk factors, and to determine any association between serum HDL-C, LDL, triglyceride (TG), systolic and diastolic blood pressure with smoking, gender and BMI. One way ANOVA, Pearson correlation and independent sample t tests by SPSS version 16 were used for statistical analysis and all means were followed by SD.
Results: Mean age of subjects was 45.5±6.7 years. Of smokers 4.7% were females. Among smokers males had a significantly higher mean of HDL (P=0.002) compared to females (42 vs. 32.4 mg/dl). HDL level was significantly lower (P<0.001) in heavy smokers compared to non-smokers and light smokers (36.5 vs 41.7 and 43.5). However, light smokers had a higher mean of HDL than non-smokers, although the difference was not statistically significant (43.5 vs. 41.7 mg/dl, P=0.131). Serum HDL was not affected by other factors.
Conclusion: Smoking was associated with a low serum HDL in the subjects under study who were intellectual class of the population. It is thus warranted to take preventive measures to combat cardiovascular diseases in this sector of community.

Keywords: Smoking, Serum HDL-C, Serum LDL

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Introduction

Epidemiologic studies have firmly established that low-density lipoproteins (LDLs) and high-density lipoproteins (HDLs) are independent risk factors for the development of cardiovascular diseases (CVD).[1,2] Over the past two decades, clinical trials in low and high risk patients have shown that a decrease in plasma LDL level is associated with a 25% to 45% decrease in cardiac events.[3-8]

Despite this reduction in risk, there remains a significant residual risk for cardiovascular events in patients treated with statins.[9] Evidence from both experimental and clinical studies suggests that increasing HDL will be associated with a decrease in CVD risk. Although limited in number, human clinical trials have supported the concept that increasing HDL may decrease clinical events.

HDL-C has quickly evolved as one of the "traditional" risk factors used by clinicians to predict risk of incident CHD.[10] An estimated 1 mg/dl higher HDL-C is associated with a 2% lower risk of CHD for men and a 3% lower risk for women[11]. As a result, there has been increasing interest in HDL-C as a therapeutic target.[12]

The prevalence of low levels of HDL-C (e.g.,

Correspondence:
R Mozaffari
Cardiovascular Research Center, Shiraz University of Medical Sciences, Shiraz, Iran
Tel&Fax: +98-711-2343529
Email: mozaffarir@gmail.com

_40 mg/dl) varies geographically[13] and is particularly high in Latin American countries, where its prevalence is as high as 46% in men.[14] Over a 7-year period, this prevalence has continued to rise in Mexico by 2% to 3%.[15] Approximately 25% of men in the United Kingdom have low levels of HDL-C.[16] A more recent Pan-European survey suggests that the prevalence of HDL-C throughout Europe is similar to that in the U.S., with low HDL-C levels in nearly one-half of the Dutch.[17] Although a significant number in China have low HDL-C levels, the prevalence is much lower than in North America, with 7% of males and<2% of females having low levels.[18] The international study evaluating risk factors for acute myocardial infarction known as the INTERHEART study, has recently shown that there is specifically a higher levels in prevalence of low HDL-C South Asians compared with other Asians.[19]

Observational and interventional studies have assessed the relationship between different factors and circulating HDL-C levels and other factors like lifestyle, physical activity, diet and smoking.

The Framingham study group showed that cigarette smoking accounted for a drop in HDL-C by 4 mg/dl in men and 6 mg/dl in women.[20] A meta-analysis demonstrated that smokers generally have a 9% lower HDL-C level and a 6% lower apolipoprotein A-I level compared with matched nonsmokers.[21] This inverse association of cigarette smoking and HDL-C levels is dose dependent.[21-25] Increased Cholesteryl Ester Transfer Protein (CETP) activity and the resultant transfer of cholesterol esters from HDL to apolipoprotein B-containing particles mediate atherogenic lipoprotein changes caused by tobacco smoking.[26,27] Furthermore, in a meta-analysis of 27 studies, it was observed that when participants quit smoking, their HDL-C levels rose by 4 mg/dl without a significant effect on total cholesterol, LDL-C, or triglyceride levels[28]. Additionally, smoking cessation results in an increase in apolipoprotein A-I.[29]

These favorable effects on the lipid profile can be seen as early as 30 days after quitting smoking.[30] Beside acute unfavorable effects on blood pressure and sympathetic tone, and a reduction in myocardial oxygen supply, smoking affects atherothrombosis by several other mechanisms. In addition to accelerating atherosclerotic progression, long-term smoking may enhance oxidation of low-density lipoprotein (LDL) cholesterol and impair endothelium-dependent coronary artery vasodilation. The latter effect has been linked to dysfunctional endothelial nitric oxide biosynthesis following chronic as well as acute cigarette consumption. In addition, smoking has adverse hemostatic and inflammatory effects, including increased levels of CRP, soluble intercellular adhesion molecule-1 (ICAM-1), fibrinogen, and homocysteine.[31]

The aim of this study is to detect and determine the relationship between HDL-C to tobacco smoking in teachers residing in Shiraz to provide a clear guideline for improving the health status of the group under study and community as a whole.

Methods

This study was done in collaboration with the Ministry of Education, Shiraz branch, and Cardiovascular Research Center of Shiraz University of medical sciences.

Of a total of 3115 teachers working in Shiraz who participated in a prospective cohort study two groups of 235 smokers and 235 non-smokers were randomly selected for evaluation of cardiovascular risk factors. The sample was selected from those who were not under treatment by anti lipid drugs,anti glycemic agents or betablocker therapy. Systolic and diastolic blood pressures, weight and height of all subjects were recorded by trained staff. Level of TG, LDL and HDL were determined in all subjects. The weight of participants was determined with bare foot and light clothing by portable weight scale and the height accurately measured with inflexible bars. Based on the body mass index (BMI) subjects were divided into three groups of normal (BMI<25), overweight (BMI: 25-30) and obese (BMI>30). Blood pressures (BP) of both arms were measured by digital micro life sphygmanometer. The average of three measurements, at five minutes intervals was used for final BP analysis. Venous blood samplings were taken in sitting and relaxed position. About 5cc of venous blood was drawn from antecubital vein, kept at 5 Cº and transferred immediately to the laboratory of Fars province health center. Serum TC, LDL-c, HDL-c, and TG were measured by enzymatic methods.

Using SPSS version 16 the data were analyzed and level of significance was considered as 0 .05. One way ANOWA, Pearson correlation and independent sample tests were used for analysis of the data.

Results

The subjects aged from 21 to 68 years, with mean age of 45.5±6.7. Mean of systolic and diastolic blood pressures were 118.3±17.2 and 76.2±12.9. Mean weight was 68.7±11.4 and mean height was

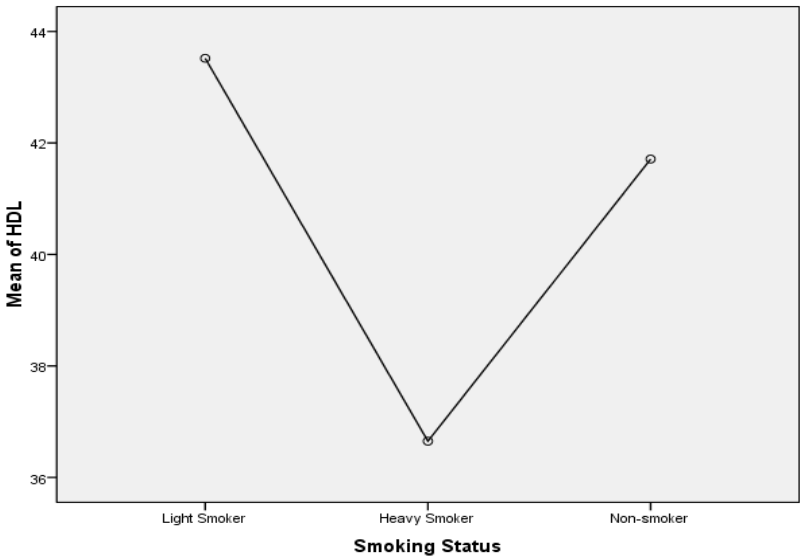

Figure 1. Mean of HDL in non-smokers, light smokers and heavy smokers

163.1±9.4. Mean LDL and HDL were 106.3±27.8 and 41.7±9.4 respectively. Mean TG level was 145.7±95.3. Among non-smokers, 46.8% were males and 53.2% females. Of smokers only 4.7% were females. Serum HDL was not affected by sex (P=0.734).

Based on the number of smoking per day, smokers were divided into two groups: light (1-5 cig/day) and heavy (above 5 cig/day). Descriptive statistics of study variables for three groups of non-smokers, light smokers and heavy smokers are compared and shown in Table 1. Mean TG for nonsmokers, light smokers and heavy smokers were 141.6, 144 and 165.3 mg/dl, respectively**.** Although, heavy smokers have a higher mean of TG it was not statistically significant (P=0.208). The three groups did not have a significantly different mean of weight, height, and systolic and diastolic blood pressure (P>0.5). Serum level of LDL was also not different between three groups.

The result showed that HDL level was significantly lower in heavy smokers compared to non-smokers and light smokers (36.5 vs 41.7 and 43.5 P<0.001). However, as shown in Fig.1 light smokers had a higher mean of HDL than non smokers, although the difference was not statistically significant (41.7 vs 43.5 mg/dl, P=0.131). Result of Pearson's correlation test showed that numbers of pack year of smoking was inversely associated with HDL level (r = 0.414, P=0.0001). Compared to heavy smokers light smokers had a lower mean of LDL (104 vs 110.5, P=0.13). BMI was not significantly associated with serum HDL. However, overweight subjects (BMI: 25-30) had a significantly higher LDL level compared to those with a normal (<25) BMI (111 vs 101, P=0.001). The LDL level of overweight and obese individuals was not significantly different. As for comparison between light with heavy smokers, the only significant difference was found in HDL level.

Discussion

The Results of our study showed that only HDL was affected by smoking. Obviously, heavy smokers were more likely to have a lower serum HDL compared to light smokers and non smokers. This is consistent with the results of other studies.[10,11,20,21] However, this is in contrast with the result of another study,[31] in which the very small sample size accounted for no association between serum HDL and smoking. Contrary to other reports[31-33] the results of our study did not show any association between serum LDL and smoking. Although, the mean of serum TG was higher in heavy smokers, it did not reach a statistically significant level. We did not find any relationship between gender and serum HDL. This may be due to the small number of females among smokers.[10] Among smokers males had a significantly higher mean of HDL compared to that of females. Other studies found that women were more likely to have a higher serum HDL.[14,19]

In the present study, BMI, systolic and diastolic blood pressures were not associated with smoking. Results of a meta-analysis showed that level of LDL, TG and total cholesterol considerably increased in smokers compared to non-smokers and this rise was also dependent on severity of smok-

Table 1: Distribution of variables in three groups based on smoking status

Smoking Status (Total Number)		Minimum	Maximum	Mean	SD
Light Smoker (169)	Age (year)	25	61	44.80	6.34
	Systol BP (mmHg)	70	170	118.79	16.86
	Diastol BP (mmHg)	40	119	75.60	13.83
	Weight (kg)	44	98	67.93	10.30
	Height (cm)	141	183	162.17	8.31
	LDL (mg/dl)	47	186	104.22	27.96
	HDL (mg/dl)	21	74	43.52	9.55
	TG (mg/dl)	40	882	144.07	108.93
Heavy Smoker	Age (year)	37	59	48.08	5.07
(65)	Systol BP (mmHg)	90	160	117.11	14.90
	Diastol BP (mmHg)	50	101	76.02	12.04
	Weight (kg)	47	97	66.65	9.96
	Height (cm)	110	182	160.06	10.88
	LDL (mg/dl)	51	236	110.52	28.73
	HDL (mg/dl)	18	84	36.65	8.74
	TG (mg/dl)	52	734	165.33	121.63
Non-smoker (235)	Age (year)	21	62	45.35	7.20
	Systol BP (mmHg)	80	200	118.22	18.01
	Diastol BP (mmHg)	45	110	76.76	12.46
	Weight (kg)	45	130	69.83	12.34
	Height (cm)	145	189	164.61	9.53
	LDL (mg/dl)	50	189	106.66	27.45
	HDL (mg/dl)	23	78	41.71	9.07
	TG (mg/dl)	31	456	141.67	74.81

ing. In addition serum HDL was inversely associated with smoking.[21]

Azizi et al in a study conducted in Tehran reported a similar result; however the severity of smoking was not taken into account in their study.[34] A study in India could not find any relationship between smoking and HDL and TG.[31] The observed difference found between our findings and the results of other studies could be due to genetic and dietary factors.

One limitation of the present study was its cross-sectional design. Although, in some cases no differences were detected between smokers and nonsmokers, they did not reach a statistically significant level. Further studies with prospective designs are needed to resolve and reach a better understanding of this relationship

In conclusion, as smoking was associated with a low serum HDL in the subjects under study who were intellectual class of the population, preventive measures should be taken to combat cardiovascular diseases in this sector of community.

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