INFLUENCE OF CIGARETTE SMOKING ON LIPID PROFILE IN MALE UNIVERSITY STUDENTS

WAHEEB D. M. ALHARBI
Department of Physiology, Faculty of Medicine, Umm Al-Qura University, Makkah, Saudi Arabia

ABSTRACT
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. The study comprised Group 1 (3-5 cigarettes/day) and Group 2 (lower and higher frequency of smoking). Plasma LDL-C, HDL-C and triglycerides in Group 1 were found significantly increased for smokers compared to the healthy control non-smoking subjects. Group 2 comparisons showed significantly increased levels of triglycerides at 1-5 cigarettes per day frequency, HDL-C and triglycerides for 6-10 and 10-15 cigarettes per day, and all components of lipid profile studied for > 15 cigarettes per day frequency. The present study is hence, helpful for future studies in elucidating the underlying mechanism causing series of changes influenced by smoking activity.

Keywords: Cigarette smoking, lipid profile, cholesterol, HDL-C, LDL-C, triglycerides.

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 (Cuesta et al., 1989; Guedes et al., 2007; Arslan et al., 2008). However, inspite of all that information, there is still much controversy about which part or parts 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. Differing results were obtained by various investigators, for example, Siekmeier et al. (1996) concludes that HDL-C levels are same for smokers and non-smokers. Whereas other investigators obtained conflicting results wherein significant variations (low levels of HDL-C in cigarette smokers) were obtained (Criqui et al., 1980; Siekmeier et al., 1994; Ito et al., 1995).

Ingredients of cigarette smoke such as nicotine and carbon monoxide have been found to be involved in causing hypoxia (Yokode et al., 1988), and increased susceptibility of LDL to be oxidized (Scheffler et al., 1992). However, the precise mechanism of the harmful involvement of cigarette smoking is not clear (Moriguchi et al., 1990).

Significantly higher serum concentrations of total cholesterol were obtained in smokers (Cuesta et al., 1989; Guedes et al., 2007). The HDL-C is a significant part of total cholesterol (TC), but the serum level of this part has been reported to have no or only a weak relationship to the TC concentration. Whereas other investigators suggest that the HDL-C level and the level of TC have a firm relationship. Furthermore, being an indicator of serum lipid patterns, the ratio of HDL-C to TC (HDL-C/TC) is considered quite important (Koyama et al., 1990). Smoking has been documented to be associated with low levels of HDL cholesterol (Criqui and Wallace, 1980; Thelle et al., 1983;
Influence of Cigarette Smoking on Lipid Profile

Guedes et al., 2007; Arslan et al., 2008). Another report shows lower but non-sigificant HDL cholesterol (HDL-C) contents in smokers (Lopes et al., 2004). The LDL-C has been found to be significantly higher in smokers (Cuesta et al., 1989; Guedes et al., 2007). Other important component in lipid profile is triglycerides that has been obtained higher in smokers (Cuesta et al., 1989; Guedes et al., 2007; Arslan et al., 2008).

In view of conflicting results obtained in various investigations, it seemed important to study the involvement of cigarette smoking at various frequencies of its usage. For that purpose this study was undertaken to clarify further the influence of daily cigarette smoking on the components of lipid profile. However, further studies are still required to be conducted to check and assess the interrelated influences leading to abnormal alterations in cholesterol/ lipid metabolism in individuals with varied levels of cigarette smoking activity.

MATERIALS AND METHODS

The present work comprised two groups of subjects. These subjects (cigarette smoking and non-smoking) were consulted in Umm Al-Qura University and related institutions in Makkah, Saudi Arabia. Group 1 contained 38 smoking and 32 non-smoking male subjects. Age range of these subjects was 17-26 years (mean: 23 years). The smokers in this group were those smoking 3-5 cigarettes/ day with an average of 4.5 cigarettes/ day; and duration of the smoking habit was 1-3 years with average of 2.5 years.

Another group (Group 2) comprised 80 smoking subjects (Age range: 18-27 years; Mean: 24.5 years; duration of the smoking habit: 1-4 years with average 3 years) accompanied by the normal controls from the group 1.

The male Subjects in group 1 were categorized as smokers and non-smokers. However, the subjects in group 2 were categorized as those having habit of smoking 1-5, 6-10, 11-15 and > 15 cigarettes per day as average. The numbers of subjects in these categories were also represented as % of total smoking male subjects, and were compared for various estimations.

Before the collection of blood for the estimation of serum cholesterol, LDL-C, HDL-C and triglycerides by routine methods in smoking and non-smoking male subjects, each subject provided the details about his smoking habits, physical/ physiological measurements and other information in the form of a standard questionnaire. The age, blood pressure, BBT, body weight, body height and other physical measurements were performed. The criteria of the selection of subjects (either smoking or non-smoking) was that no one should have any medical complication such as hypertension, ischemic heart disease, stroke, diabetes or such other disorder. Hence, all male subjects included in the present study are the normal healthy subjects.

The data was collected and analyzed statistically using student t test and correlation coefficient ‘r’ of the regression line where required. The significance values (p) for various comparisons were found and the results were interpreted.

RESULTS AND DISCUSSION

Influence of cigarette smoking on lipid profile in healthy male university students was studied initially in a group (Group:1) of 38 smoking subjects compared with 32 non-smoking subjects. The mean ± SD values for serum cholesterol, LDL-C, HDL-C and triglycerides are given in Table 1. The comparison for serum cholesterol (mg/dl) showed non-significant variations. However, all other components of lipid profile studied (LDL-C, HDL-C and triglycerides) were found significantly increased for smokers compared to the healthy control non-smoking subjects. The values of significance for various comparisons are given in Table 1.
The above given data provided a basic concept about the general influence of cigarette smoking in healthy male subjects. However, to have further information of the influence of daily cigarette smoking (as per number of cigarettes per day) on lipid profile, 80 male smokers were studied (Group 2; Table 2). In this set of data, the subjects were categorized according to average number of cigarettes (1-5; 6-10; 10-15; and above 15) smoked daily by each subject. These subjects were compared with the data of the normal healthy controls (n: 32). There was no significant variation found for any of the component of lipid profile for the subjects smoking at the lowest frequency (1-5 cigarettes per day) in the current study except triglycerides that showed significant difference (Table 2; p=0.0394). The LDL-C and triglycerides in the next higher frequency (6-10 cigarettes/day) however, were found significantly increased for smoking subjects while compared with the normal healthy males in Group 1 (Table 2).

In the second highest frequency of cigarette smoking (10-15 per day; Table 2), HDL-C and triglycerides increased highly significantly (p=0.0006 and <0.0001 respectively). The most interesting results were obtained for the highest frequency of cigarette smoking (>15/ day; 31.25 % subjects) in the present study, where all components of lipid profile studied presently increased significantly especially the HDL-C and triglycerides that vary highly significantly (p<0.0001).

The results for serum cholesterol in the present study are quite similar to the findings of Cuesta et al. (1989) and Guedes et al. (2007) who found increased levels of total

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**Table 1**

Lipids profile in smoking and non-smoking healthy male students

<table>
<thead>
<tr>
<th>Blood levels</th>
<th>Non-smoking (n: 32)</th>
<th>Smoking (n: 38)</th>
<th>Significance (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mg/dl)</td>
<td>205.23 ± 48.05</td>
<td>210.52 ± 35.34</td>
<td>(p=0.5980) ns</td>
</tr>
<tr>
<td>LDL-C (mg/dl)</td>
<td>107.40 ± 39.24</td>
<td>126.87 ± 39.09</td>
<td>(p=0.0420)</td>
</tr>
<tr>
<td>HDL-C (mg/dl)</td>
<td>68.82 ± 32.22</td>
<td>51.95 ± 28.13</td>
<td>(p=0.0223)</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>145.22 ± 23.24</td>
<td>163.61 ± 20.01</td>
<td>(p=0.0007)</td>
</tr>
</tbody>
</table>

The values are mean ± SD; n: number of subjects; ns: non-significant.

**Table 2**

Influence of daily cigarette smoking on lipid profile in healthy male students

<table>
<thead>
<tr>
<th>Cigarette smoking (Cigarettes / day)</th>
<th>Subjects</th>
<th>Lipid profile</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>Cholesterol (mg/dl)</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>±49.68</td>
</tr>
<tr>
<td>1-5</td>
<td>12</td>
<td>207.11</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±46.81</td>
</tr>
<tr>
<td>6-10</td>
<td>23</td>
<td>205.39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±48.66</td>
</tr>
<tr>
<td>10-15</td>
<td>20</td>
<td>209.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±49.95</td>
</tr>
<tr>
<td>&gt;15</td>
<td>25</td>
<td>228.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±32.27*</td>
</tr>
</tbody>
</table>

The values are mean± SD; n: number of subjects; *: p< 0.0416, *a  p<0.0488, *b p=0.0431, *c p=0.0006, *d p<0.0001, *e p=0.0394, *f p=0.0167, *g p<0.0001
cholesterol in smokers. Furthermore, the levels of HDL-C were found lower in the present study. Hence, these results are similar to those obtained by other investigators (Criqui et al., 1980; Siekmeier et al., 1994; Ito et al., 1995), who documented low levels of HDL-C in smokers too. However, the investigations in the current report could not interpret the results obtained by Siekmeier et al. (1996) wherein the HDL-C levels are same for smokers and non-smokers. Moreover, the present results differ from another report where smokers had lower but non-significant HDL cholesterol (HDL-C) contents (Lopes et al., 2004). Whereas the findings for LDL-C and triglycerides in the present report are similar to the investigations of Cuesta et al. (1989), Guedes et al. (2007 and Arslan et al. (2008).

The present study provides opportunity to explain the physiological consequences of the cigarette smoking activity. As the current report concerns solely to the study of lipid profile in normal healthy controls and smoking male subjects, the results of this study can be correlated with other biochemical, physiological and clinical aspects. This approach is hence, helpful for future studies in understanding the underlying mechanism causing series of changes influenced by smoking activity.

REFERENCES


