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Effect of exclusive cigarette smoking and in combination with waterpipe smoking on lipoproteins

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ABSTRACT

Objective: A significant increase in total cholesterol and LDL-C is well shown in tobacco users, as compared to non-tobacco users. The additive effects of waterpipe and cigarette smoking on LDL levels have not been studied. The study's objective was to assess the correlation between cigarette smoking and LDL levels in exclusive cigarette smokers and to check the interaction effect of waterpipe and cigarette smoking on LDL levels.

Methods: This cross-sectional study was conducted between October 2016 and February 2017 in 4 different laboratories, enrolling 308 patients (188 non-smokers, 105 cigarette smokers, 15 previous smokers).

Results: Current cigarette smoking (Beta = 25.57; p < 0.0001) was significantly associated with higher LDL levels and higher total cholesterol levels (Beta = 53.29; p < 0.0001) in exclusive cigarette smokers. Among current cigarette smokers who were current waterpipe smokers, a significant increase in LDL level was observed relative to current cigarette smokers who were not waterpipe smokers (Beta = 66.64 vs Beta = 37.37; p < 0.0001).

Conclusion: Among Lebanese current cigarette smokers, LDL levels increased relative to nonsmokers, consistent with findings in other populations. In addition, among Lebanese current cigarette smokers, current waterpipe smoking might increase adverse lipid profiles associated with adverse coronary effects more than cigarette smoking alone. The direct cause responsible for these observed variations in our study remains unidentified, with the hope that future research will reveal it.

1. Introduction

Coronary heart disease (CHD) is the single largest cause of death in the developed countries and is one of the leading causes of disease burden in developing countries as well [1]. Smoking was responsible for 16.3% of cancer deaths, 17.2% of years of potential life lost and 21% of the cost of productivity in Iran (2012) [2]. Cigarette smoking may be an important factor in potential changes in lipid profile already in young healthy people [3,4]. A significant increase in total cholesterol and LDL-C is well shown in tobacco users, as compared to non-tobacco users [5–7]. Thus, smokers have less favorable lipid profiles, even after accounting for current and lifetime smoking history and other CVD risk factors [8].

There is a significant increase in levels of total cholesterol, triglycerides, low density lipoprotein (LDL), very low density lipoprotein (VLDL) and reduced levels of high density lipoprotein (HDL) among smokers [9]. Maternal smoking during pregnancy is associated with an increased rise in total cholesterol levels and a tendency towards an adverse lipoprotein profile in the offspring.
Even, maternal environmental tobacco smoking exposure affects milk lipids which are essential for infant growth [11].

In addition, smoking is associated with an increased prevalence of metabolic syndrome, independent of sex and BMI class, mainly related to lower HDL cholesterol, and higher triglycerides and waist circumference [12]. Stress and depression were also significantly correlated with an increase in cholesterol levels [13]. Moreover, smoking was associated with unfavorable changes in apoA1 and apoB, and in lipoprotein particle size [12]. Data suggested a synergistic effect between the apoE allele epsilon4 and smoking and apoB, and in lipoprotein particle size [12]. Data suggested a synergistic effect between the apoE allele epsilon4 and smoking and apoB, and in lipoprotein particle size [12].

Clinical characteristics and outcomes of acute coronary syndrome patients depend on the tobacco modality used [16]. In fact, overall tobacco users (cigarettes and waterpipe) tended to have dyslipidemia compared to previous smokers or non-smokers [16]. In addition, waterpipe and cigarette smoking was significantly associated with dyslipidemia [17]. However, the additive effect of waterpipe and cigarette smoking on LDL levels has not been studied to the best of our knowledge. Furthermore, no studies have assessed the impact of cigarette smoking on the LDL in the Lebanese population. Therefore, our primary objective was to assess the correlation between cigarette smoking and LDL levels in the Lebanese cigarette smokers and to check the interaction effect of waterpipe and cigarette smoking on LDL levels. Secondary objectives were to assess its correlation with other cardiovascular risk factors (total cholesterol), taking into account known comorbidities (high blood pressure and obesity status), food habits, stress and physical activity.

2. Methods

2.1. Study design and included population

A cross-sectional study was conducted between October 2016 and February 2017 in 4 laboratories, chosen from 4 different districts in Lebanon. All patients coming for a regular blood test to the laboratory and who were 18 years old and above, were included in the study. However, patients treated with a statin were excluded since statins are established in the primary and secondary prevention of coronary artery disease [18]. Furthermore, patients having thyroid disorders at the time of the study were also excluded for hyper- and hypothyroidism can affect lipid levels and thus change the risk estimation of coronary heart disease [19]. Exclusion criteria also included individuals with a history of cardiovascular, endocrine, dementia or gastrointestinal disorders.

2.2. Sample size calculation

Using the Gpower 3.1.9.2 program for the calculation of the minimal sample size needed for our study, with a 1-β = 0.95, a proportion p2 = 0.05, according to the study of Neki [20] that showed a mean LDL of 87 ± 17.80 mg/dL in non-smokers versus 103.7 ± 29.16 mg/dL in smokers, and considering a ratio of 1 control for every case, the results showed that we need 47 cases versus 47 controls [21].

2.3. Data collection

The questionnaire was administered in Arabic, the native language in Lebanon. A first part of the data was collected via a face-to-face interview and included the following variables: demographics information (age, gender, geographic region, marital status, occupation, educational level, monthly salary per house divided into three levels (low (<1000 USD), intermediate (1000–2000 USD), high (>2000 USD) based on the total income of the household, history of medical illness (hypertension, asthma, chronic obstructive pulmonary disease (COPD), diabetes, epilepsy) and the medications intake at the time of the study. The social habits of the participants were assessed; we asked about the frequency of cigarette smoking (number of cigarettes smoked per day), the number of alcoholic glasses drunk per week and the number of coffee cups drunk per day.

In the second part of the questionnaire, participants were asked about the total number of hours of no activity during weekdays and weekends, taking into consideration the average hours of sleep, rest, occupational, and extracurricular activities over a typical 24-h period. Information about the physical activity was also collected. In order to test the effect of each activity on the cardiovascular risk, we categorized separately the activities in a dichotomous variable (yes/no), with a yes answer meaning a daily activity of 30 min or more.

We chose a validated scale in Lebanon, the Beirut Distress Scale 22 (BDS22) [22], to measure the level of stress in these patients. The BDS 22 is an Arabic scale, composed of 22 questions that determine six factors, reflecting: depressive symptoms, demotivation, psychosomatic symptoms, mood deterioration, intellectual inhibition and anxiety in these patients. Participants were asked to rate symptoms of stress by indicating how often they have experienced each symptom during the past week on a Likert-type scale that ranges from 0 (not at all) to 3 (all of the time). Possible scores range from 0 to 66 for the BDS22, with higher scores indicating higher levels of stress.

2.4. Dietary intake assessment

The self-administered questionnaire used in this study included numerous questions related to the socio-demographic background of our participants and a short food frequency questionnaire (FFQ) to assess the usual dietary intake of Lebanese patients. The FFQ was composed of 16 semi-quantitative questions covering different food categories (including the five basic food categories typically consumed by the Lebanese population) [23]. The FFQ used in this study was adapted from the questionnaire earlier administered in a sample of a Lebanese population [23]. The FFQ used in this study was adapted from the questionnaire earlier administered in a sample of a Lebanese population [23]. The BDS22 is an Arabic scale, composed of 22 questions that determine six factors, reflecting: depressive symptoms, demotivation, psychosomatic symptoms, mood deterioration, intellectual inhibition and anxiety in these patients. Participants were asked to rate symptoms of stress by indicating how often they have experienced each symptom during the past week on a Likert-type scale that ranges from 0 (not at all) to 3 (all of the time). Possible scores range from 0 to 66 for the BDS22, with higher scores indicating higher levels of stress.

The FFQ asked how often each food item, group, or beverage was usually consumed with five possible answers for each of the food categories: (1) never, (2) two times or less per week, (3) three to six times per week, (4) at least one time per day, and (5) at all meals. These five response categories were later merged into four categories for analysis purposes, namely: (1) never, (2) once or twice per week (3) three to six times per week, and (4) consumption on daily basis. We also asked the patient if he eats more when stressed, with the answers dichotomized as yes/no.

2.5. Laboratory analysis

Blood samples were drawn from the antecubital vein between 6:00 am and 7:00 am after an overnight fasting (12 hours fasting) in order to screen for dyslipidemia and check the fasting blood glu-
cose levels. Total cholesterol, High Density Lipoproteins (HDL cholesterol), Low-Density Lipoproteins (LDL cholesterol), triglycerides (TG) and fasting blood glucose were measured by each laboratory.

2.6. Anthropometric measures

On the same day where the blood samples were collected, we measured the participants’ height, weight and waist circumference using the Detecto® model 339 balance and a tape measure. This measure was taken by one technician designated in each laboratory. All technicians received appropriate training prior to the beginning of the data collection.

The Body Mass Index (BMI) was calculated, using the formula: body weight (in kilograms) divided by the square of the height (in meters), and classified according to the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS) guidelines 2011 and WHO: Underweight (<18.5), Normal (18.5–24.9), Overweight (25.0–29.9) and Obese (>30.0) [25,26].

The waist circumference (WC) was considered normal, according to NCEP/ATP III guidelines for definition of metabolic syndrome [27], if the value was less than 102 cm for men and less than 88 cm for women.

The blood pressure (BP) was measured using a sphygmomanometer (ALP-K2 a professional traditional aneroid blood pressure). According to the JNC8 (Eighth Joint National Committee) guidelines [28], two BP measurements were made in seated position at the upper arm, at 1–2 min interval.

2.7. Data analysis

Data entry and analysis were performed on Statistical Package for the Social Sciences (SPSS) software version 23. The independent-sample t-test was used to compare means between two groups. Pearson correlation coefficient was used to correlate between quantitative variables. For categorical variables, the χ² and Fisher exact tests were used when applicable. The ANOVA test was used to compare means between multiple groups.

A multivariable analysis linear regression was carried out using the LDL-C as the dependent variable, and taking the independent variables that showed a p < 0.2 in the bivariate analysis [29,30]. Potential confounders may be eliminated only if p > 0.2, in order to protect against residual confounding [31]. Additional linear regressions were carried out using triglycerides and total cholesterol as dependent variables. We also conducted linear regressions on the same dependent variables but taking the cumulative cigarette smoking (number of cigarettes smoked per week × number of years of cigarette smoking) as independent variables, to confirm the presence of a dose effect relationship. Two additional linear regressions were conducted, comparing the increase in LDL levels in cigarette and waterpipe smokers, compared to cigarette smokers alone. The independent variables that were entered in the model were eating mankoushe, fast food, French fries, olive oil, lentils, fish, meat/chicken, hotdog, white and brown bread, rice, legumes, fruits, desserts, full fat and diet milk, carbonated beverages, marital status, educational level, monthly salary, alcohol drinking, Body Mass Index (BMI) and cigarette smoking. Significance was defined as a p-value less than 0.05.

3. Results

3.1. Sensitivity analysis

We ran a sensitivity analysis (data not shown) to check for a difference between the principal model results to alternative choices of the set of subjects analyzed in each laboratory separately; there was no difference detected. Thus, results were shown on patients from all laboratories as one set.

3.2. Sociodemographic characteristics

Out of 400 questionnaires distributed to laboratories, 308 (77%) were collected back from the patients. There were missing values in our results since not all questions were answered by all participants. Table 1 summarizes the bivariate analysis of sociodemographic and socioeconomic factors and the cigarette smoking status. The results showed that there was a significant difference between the 3 groups (non cigarette smokers, current cigarette and previous cigarette smokers) concerning the monthly salary (p < 0.0001), ever drunk alcohol (p = 0.044), ever drunk coffee (p = 0.001), waist circumference (p < 0.0001), heart rate (p < 0.0001), HDL-C (p = 0.008), LDL-C (p < 0.0001), triglycerides (p = 0.002), BMI (p = 0.001) and regular sports (p < 0.0001). The mean LDL-C level in controls was 93.08 ± 21.84, 127.79 ± 20.54 in exclusive cigarette smokers, 164.08 ± 33.12 in both cigarettes and waterpipe smokers.

3.3. Bivariate analysis

Systolic and diastolic blood pressures, heart rate, total cholesterol, and triglycerides were all significantly and positively correlated with the LDL score in non-waterpipe smokers, while HDL-C was significantly but negatively correlated with the LDL score in the same group (Table 2). Moreover, systolic and diastolic blood pressures, total cholesterol and the body mass index were all significantly and positively correlated with the LDL score in waterpipe smokers. The ANOVA test showed that there was a significant difference between all types of food and LDL-C (p < 0.05 for all variables) (data not shown). Similar results were obtained for the total cholesterol taken as the dependent variable.

3.4. Multivariable analyses

The results of the first multivariate analysis taking the LDL levels as the dependent variable showed that current cigarette smoking (p < 0.0001), eating olive oil once daily (p = 0.014) and eating meat/chicken 3–6 times weekly (p = 0.034) were significantly associated with higher LDL levels.

A second multivariate analysis taking the total cholesterol as the dependent variable, showed that current cigarette smoking (p < 0.0001), eating more when stressed (p = 0.001), eating hotdog 3–6 times weekly (p < 0.0001), eating meat/chicken 3–6 times weekly (p = 0.006), eating desserts at every meal and eating rice once daily (p < 0.0001 for both variables) were significantly associated with higher total cholesterol levels respectively. Furthermore, drinking full fat milk once daily (p = 0.025), the BMI (p = 0.021), the intermediate and the high socioeconomic status (p < 0.0001 for both variables) were significantly associated with higher total cholesterol levels respectively. However, drinking fruit juice 3–6 times weekly and eating brown bread once daily (p < 0.0001 for both variables) were significantly associated with lower total cholesterol levels respectively.

A third and fourth linear regression, taking the LDL cholesterol as the dependent variable, and comparing between non-waterpipe and waterpipe smokers, showed that among current cigarette smokers who were current waterpipe smokers, a significant increase in LDL level was observed relative to current cigarette smokers who were not waterpipe smokers (Beta = 66.64 vs Beta = 37.37; p < 0.0001) (Table 3).
4. Discussion

4.1. Summary

Our results suggest a clear association between cigarette smoking and higher LDL and total cholesterol levels. This association persisted after adjustment for suggested confounding factors. Furthermore, LDL levels would be higher in patients smoking waterpipe and cigarettes compared to cigarettes alone.

4.2. Comparison with existing literature

This study showed a clear absolute difference in LDL levels between non-smokers and smokers. An absolute LDL difference
of more than 30 mg/dl would have a big impact on the overall risk profile and would reduce the risk of coronary heart disease by approximately 30% [32]. Both LDL and smoking are well established independent risk factors for cardiovascular disease [33]. The effect of smoking on the LDL quality was also shown in the literature; smoking reduces serum antioxidant defense, induces lipid peroxidation and leads to LDL modifications toward more atherogenic forms [34–39]. Atherogenic forms are associated with higher rates of cardiovascular outcomes [32]. The effect of smoking on LDL particles size is another contributor to the atherogenicity of these particles [40]. We can hypothesize that the effect of smoking on LDL is both qualitative and quantitative. Both effects go in the same direction, in agreement with the study of Stanhope et al. [47]. Eating olive oil once daily would significantly increase the LDL cholesterol level. The study of Marrugat et al. [49] showed the same results and proved that an olive oil-rich diet results in higher concentrations of LDL cholesterol. This increased level of LDL due to a possible potentiation effect of waterpipe and cigarette needs to be further studied.

The results of this multivariate analysis showed that eating hotdog 2 to 6 times weekly is significantly associated with higher total cholesterol level. The study of S.R. Baggio [46] showed the same correlation between the consumption of hotdogs, salami and other processed meats and the elevation of the lipid parameters especially total cholesterol level. Processed meats contain high amounts of dietary fat and many other harmful chemicals and their regular consumption is linked to increased risk of several diseases like heart disease and cancer.

In addition, this multivariate analysis has shown that eating desserts on every meal significantly increase total cholesterol. Processed meats and the elevation of the lipid parameters especially total cholesterol level. Processed meats contain high amounts of dietary fat and many other harmful chemicals and their regular consumption is linked to increased risk of several diseases like heart disease and cancer.

Moreover, the results showed that drinking full fat milk 3–6 times weekly significantly increased the total cholesterol. Switching to skimmed milk to decrease these concentrations and improve lipid profile should be investigated further [48].

Furthermore, this study showed that eating olive oil once daily is positively correlated with LDL cholesterol level. The study of Marrugat et al. [49] showed the same results and proved that an olive oil-rich diet results in higher concentrations of LDL cholesterol. This is controversial with many studies that showed that olive oil contains monosaturated fats that help lower the LDL cholesterol and increase HDL cholesterol [50]. However, our results of the multivariate analysis showed that eating hotdog 2 to 6 times weekly is significantly associated with higher total cholesterol level. The study of S.R. Baggio [46] showed the same correlation between the consumption of hotdogs, salami and other processed meats and the elevation of the lipid parameters especially total cholesterol level. Processed meats contain high amounts of dietary fat and many other harmful chemicals and their regular consumption is linked to increased risk of several diseases like heart disease and cancer.

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Table 3
Multivariable analysis.

| Factor                        | Unstandardized Beta | Standardized Beta | p-value  | Confidence Interval |
|-------------------------------|---------------------|-------------------|----------|---------------------|
| Linear regression 1 taking the LDL-C as the dependent variable in the whole sample. |                     |                   |          |                     |
| Current cigarette smoker      | 25.573              | 0.408             | <0.0001  | 13.794              | 37.353             |
| Eating olive oil once daily   | 12.514              | 0.221             | 0.014    | 2.552               | 22.476              |
| Eating meat/chicken 3 to 6 times weekly | 11.147               | 0.202             | 0.034    | 0.853               | 21.442              |
| Linear regression 2 taking the total cholesterol as the dependent variable in the whole sample. |                     |                   |          |                     |
| Current cigarette smoker      | 53.291              | 0.434             | <0.0001  | 38.998              | 67.584              |
| Eating more when stressed sometimes | 26.329               | 0.186             | 0.001    | 10.784              | 41.875              |
| Eating brown bread once daily | –56.476             | –0.377            | <0.0001  | –76.237             | –36.715             |
| Eating hotdog 3 to 6 times weekly | 46.325             | 0.372             | <0.0001  | 32.766              | 59.884              |
| Eating meat/chicken 3 to 6 times weekly | 19.155             | 0.176             | 0.006    | 5.707               | 32.603              |
| Eating desserts every meal    | 49.617              | –0.194            | <0.0001  | 10.398              | 74.512              |
| Eating rice once daily        | 41.801              | 0.260             | <0.0001  | 22.386              | 61.216              |
| Drinking full fat milk once daily | 32.386           | 0.141             | 0.025    | 4.103               | 60.669              |
| Drinking fruit juice 3 to 6 times weekly | –48.921          | –0.346            | <0.0001  | –66.053             | –31.789             |
| BMI                           | 1.965               | 0.124             | 0.021    | 0.306               | 3.624               |
| Intermediate socioeconomic level | 60.683          | 0.470             | <0.0001  | 47.400              | 73.965              |
| High socioeconomic level      | 51.221              | 0.396             | <0.0001  | 34.863              | 67.579              |
| Linear regression 3 taking LDL as the dependent variable in non-waterpipe smokers. |                     |                   |          |                     |
| Current cigarettes smoker     | 37.34               | 0.63              | <0.0001  | 29.22               | 45.47               |
| Eating legumes once daily     | –17.68              | –0.19             | 0.01     | –31.06              | –4.30               |
| Drinking coffee/tea once daily | 8.68                | 0.16              | 0.02     | 1.17                | 16.19               |
| Linear regression 4 taking LDL as the dependent variable in waterpipe smokers. |                     |                   |          |                     |
| Current cigarette smoking     | 66.641              | 0.759             | <0.0001  | 58.726              | 72.555              |
| Eating mankouche once daily   | 28.348              | 0.283             | <0.0001  | 15.563              | 41.134              |
| Eating brown bread once daily | –21.458             | –0.273            | <0.0001  | –32.330             | –10.570             |
| Eating tabbouleh once daily   | –13.024             | –0.227            | <0.0001  | –21.114             | –4.934              |
| Eating white bread 3–6 times weekly | 14.674           | 0.175             | 0.011    | 3.455               | 25.893              |
| Drinking fruit juice 3–6 times weekly | –9.032          | –0.125            | 0.079    | –19.128             | 1.064               |
| Drinking carbonated beverages 3–6 times weekly | 23.869           | 0.158             | 0.031    | 2.279               | 45.459              |
| BMI                           | 1.191               | 0.185             | 0.006    | 0.346               | 2.036               |

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and those of Marrugat showed that opposite results can be seen depending on the amount of olive consumed daily.

4.3. Implications for research and/or practice

Spreading awareness by health professionals (physicians, pharmacists) about the drawbacks of cigarette and waterpipe smoking and their possible deleterious effects, can help educate the patients prevent cardiovascular diseases.

4.4. Limitations and strengths

Our study has several limitations and strength points. The total sample size is acceptable, withdrawn from four districts in Lebanon, however, might not be representative of the whole population. The demographic data analysis would not reproduce the exact picture of the Lebanese population. A selection bias is still, however, possible because of the twenty-three percent refusal rate. The use of a questionnaire in participants may not always be accurate: problems in question understanding, recall deficiency and over or under evaluating symptoms, which can lead to a possible information bias. The amounts/frequencies of cigarette smoking, alcohol and coffee drinking, as well as the eating habits are subjective and could not be measured, which can lead to an information bias. This study was a cross-sectional study, therefore, no causation can be determined between the exposure of cigarette smoking and subsequent disease.

5. Conclusion

Our findings revealed the presence of acute measurable cardiovascular effects with current cigarette smoking, which might be even more potentiated with waterpipe smoking. The direct cause responsible for these observed variations in our study remains unidentified, with the hope that future research will reveal it. Our results would help involved persons implement new rules and educational courses to apprise youth about the dangerous and addictive effects of cigarette and waterpipe smoking, arrange awareness promotions to encourage smokers to follow health-promoting behaviors. Future research should confirm these findings in this and other populations.

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None received.

Conflicts of interest

The authors have nothing to disclose.

Compliance with Ethical Standards

The Lebanese University school of Pharmacy Institutional Review Board waived the need for an approval based on the facts that it was an observational study that respected participants’ autonomy and confidentiality and induced minimal harm to them. A written informed consent was obtained from all patients prior to distributing the questionnaire to them.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jegh.2017.08.006.

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