ATLANTIS PRESS

Journal of Epidemiology and Global Health

ISSN (Online): 2210-6014 **ISSN (Print): 2210-6006**
Journal Home Page: https://www.atlantis-press.com/journals/jegh

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To cite this article: Souheil Hallit, Marouan Zoghbi, Rabih Hallit, Lara Youssef, Rachelle Costantine, Nelly Kheir, Pascale Salameh (2017) Effect of exclusive cigarette smoking and in combination with waterpipe smoking on lipoproteins, Journal of Epidemiology and Global Health 7:4, 269–275, DOI: https://doi.org/10.1016/j.jegh.2017.08.006

To link to this article: https://doi.org/10.1016/j.jegh.2017.08.006

Published online: 16 April 2019

Journal of Epidemiology and Global Health 7 (2017) 269–275

Contents lists available at ScienceDirect

Journal of Epidemiology and Global Health

journal homepage: www.elsevier.com/locate/jegh

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

Souheil Hallit [a,b,c,d,e,*], Marouan Zoghbi [d,f], Rabih Hallit [c], Lara Youssef [g], Rachelle Costantine [h], Nelly Kheir [i], Pascale Salameh [a,j]

[a] Lebanese University, Faculty of Pharmacy, Beirut, Lebanon
[b] Saint-Joseph University, Faculty of Pharmacy, Beirut, Lebanon
[c] Holy Spirit University of Kaslik, Faculty of Medicine and Medical Sciences, Kaslik, Lebanon
[d] Research Department, Psychiatric Hospital of the Cross, P.O. Box 60096, Jal Eddib, Lebanon
[e] Occupational Health Environment Research Team, U1219 BPH Bordeaux Population Health Research Center Inserm – Université de Bordeaux, France
[f] Saint-Joseph University, Faculty of Medicine, Beirut, Lebanon
[g] Department of Nursing and Health Sciences, Notre-Dame University, Zouk, Lebanon
[h] Holy Spirit University of Kaslik, Department of Agronomic Sciences, Kaslik, Lebanon
[i] Lebanese University, Faculty of Sciences 2, Fanar, Lebanon
[j] Lebanese University, Faculty of Medicine, Beirut, Lebanon

ARTICLE INFO

Article history:
Received 13 July 2017
Received in revised form 19 August 2017
Accepted 23 August 2017
Available online 31 August 2017

Keywords:
Waterpipe
Cigarette
LDL
Total cholesterol

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 Lebanese 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

Peer review under responsibility of Ministry of Health, Saudi Arabia.

* Corresponding author at: Street 8, Building 560, 1st Floor, Biakout, Mount Lebanon, Lebanon.
E-mail address: souheilhallit@hotmail.com (S. Hallit).

http://dx.doi.org/10.1016/j.jegh.2017.08.006
2210-6006/

[10]. 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 on carotid atherosclerosis [14], as well as to insulin resistance phenomenon [15].

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 $1\text{-}\beta = 0.95$, 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 (>2.000 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] and the CDC Global School Health Survey [24]; the finally used items were vegetables, fruits, olive oil, fish and sea food, meats (including cooked meats, poultry, ham, and hotdog), pasta, sweets (cake, ice cream, chocolate), carbonated beverages, fruit, vegetables, fast food (hamburger, pizza, Lebanese pizza (known as Mankouche with thyme or cheese or yogurt based kechek), fried potatoes and chips. We omitted to ask questions about eggs and dairy products as separate items because they would have been confusing to the participants to record in the FFQ given that these food items are frequently consumed in Lebanon within composite dishes (eggs, cheese, and yogurt within cooked dishes), and fast food meals. 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 meter. 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 ($\geq$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 χ^2 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 $\times$ 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 ± 25.26 in exclusive waterpipe smokers and 165.02 ± 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).

Table 1
Sociodemographic and socioeconomic characteristics of the participants.

	Non cigarette smokers (N = 188)	Cigarette smokers (N = 105)	Previous cigarette smokers (N = 15)	p-value
Factor				
Gender				0.979
Male	98 (52.1%)	56 (53.3%)	8 (53.3%)	
Female	90 (47.9%)	49 (46.7%)	7 (46.7%)	
District				0.438
Beirut	14 (7.4%)	9 (8.6%)	2 (13.3%)	
Mount Lebanon	56 (29.8%)	42 (40%)	5 (33.3%)	
North	78 (41.5%)	38 (36.2%)	4 (26.7%)	
South	40 (21.3%)	15 (14.3%)	4 (26.7%)	
Bekaa	0 (0%)	1 (0.7%)	0 (0%)	
Marital status				<0.0001
Single	81 (43.1%)	22 (21%)	12 (80%)	
Married	101 (53.7%)	71 (67.6%)	3 (20%)	
Widowed	5 (2.7%)	11 (10.5%)	0 (0%)	
Divorced	1 (0.5%)	1 (1%)	0 (0%)	
Educational level				0.110
Illiterate	4 (2.1%)	2 (1.9%)	0 (0%)	
Primary level	14 (7.4%)	16 (15.2%)	0 (0%)	
Complementary & Secondary levels	31 (16.5%)	25 (23.8%)	3 (20%)	
University level	139 (73.9%)	62 (59%)	12 (80%)	
Monthly salary				0.119
<1000 USD	82 (48.2%)	30 (29.7%)	7 (46.7%)	
1000–2000 USD	54 (31.8%)	38 (37.6%)	3 (20%)	
>2000 USD	33 (19.4%)	31 (30.7%)	5 (33.3%)0	
Ever drunk alcohol				0.044
No	77 (41%)	28 (26.7%)	2 (13.3%)	
Yes and still drinking	106 (56.4%)	72 (68.6%)	12 (80%)	
Previous drinker	5 (2.7%)	5 (4.8%)	1 (6.7%)	
Ever drunk coffee				0.001
No	39 (20.7%)	7 (6.7%)	0 (0%)	
Yes and still drinking	146 (77.7%)	91 (86.7%)	15 (100%)	
Previous drinker	3 (1.6%)	7 (6.7%)	0 (0%)	
Regular sports/activities				<0.0001
No	97 (55.4%)	79 (79.8%)	7 (50%)	
Yes	78 (44.6%)	20 (20.2%)	7 (50%)	
Age	40.85 ± 14.76	43.14 ± 12.80	38.93 ± 13.49	0.315
Waist circumference	87.04 ± 15.01	96.30 ± 14.04	91.66 ± 12.11	<0.001
Systolic Blood Pressure	119.30 ± 14.15	120.39 ± 14.65	118.33 ± 17.49	0.808
Diastolic Blood Pressure	71.59 ± 11.84	68.29 ± 10.76	67.66 ± 14.16	0.07
Heart rate	76.91 ± 10.77	84.98 ± 18.03	80 ± 14.35	<0.001
Total cholesterol	197.48 ± 46.70	210.72 ± 55.09	189.03 ± 51.35	0.059
HDL	46.46 ± 16.24	41.49 ± 9.04	40.56 ± 7.40	0.008
LDL	124.88 ± 42.46	149.16 ± 33.55	141.84 ± 30.89	<0.0001
Triglycerides	159.66 ± 69.92	188.67 ± 73.35	196.75 ± 78.59	0.002
BMI	25.25 ± 3.61	26.33 ± 4.86	29.22 ± 5.70	0.001
Stress score	10.49 ± 8.70	11.41 ± 8.48	12.86 ± 4.51	0.444

Table 2
Correlation of factors with LDL-C.

Factor	Non-waterpipe smokers		Waterpipe smokers	
	Correlation factor	p-value	Correlation factor	p-value
Waist circumference	0.121	0.254	0.144	0.129
Systolic Blood Pressure	0.177	0.042	0.32	<0.0001
Diastolic blood pressure	0.198	0.022	0.346	<0.0001
Heart rate	0.264	0.005	0.156	0.093
Total cholesterol	0.406	<0.0001	0.173	0.036
HDL	−0.187	0.019	−0.138	0.094
Triglycerides	0.397	<0.0001	0.121	0.143
Glucose	0.150	0.061	0.078	0.35
Body Mass Index	0.163	0.055	0.263	0.002
Stress	0.097	0.227	0.12	0.943

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

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
Eat 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	0.002	−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

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 increased risk direction. Knowing the major role of LDL in cardiovascular risk [41,42], we hypothesize that the 30–60 mg/dl difference observed would suggest a dual benefit of smoking cessation on this risk.

The effect of smoking on LDL seems constant through different periods of social and economic changes. A study published in 1979 showed an increase in LDL with cigarette and coffee drinking [43]. The reproducibility of this effect across time and countries from different geographic, ethnic and genetic backgrounds is of notable importance.

The positive association needs further evaluation to explore the possible physiologic mechanism that would explain this association.

Smoking cessation recommendation is a cornerstone for cardiovascular risk reduction but the available evidence does not allow us to conclude to a positive effect on LDL levels. A study conducted in 2011 did not detect any significant effect of smoking cessation on LDL levels [26]. An older meta-analysis did not equally show any benefit on LDL from smoking cessation [44].

Current cigarettes smoking would significantly increase the LDL levels more in waterpipe smokers as compared to non-waterpipe smokers. A possible explanation of our findings is that waterpipe smoking may lower serum vitamin C levels, thus leading to significant increases in LDL and apolipoprotein B levels, while decreasing HDL cholesterol [45]. 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 the total cholesterol level, in agreement with the study of Stanhope et al. [47]. Eating refined sugar, fructose and glucose enriched foods were positively correlated to high cholesterol level and this can increase the risk of developing heart disease.

Moreover, the results showed that drinking full fat milk 3–6 times weekly significantly increased the total cholesterol. Saturated and transfat found in full fat milk are the main causes of increased cholesterol level. 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

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

Funding sources

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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