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Prevalence and associations of behavioural risk factors with blood lipids profile in Lebanese adults: findings from WHO STEPwise NCD cross-sectional survey

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ABSTRACT

Objective To examine associations of behavioural risk factors, namely cigarette smoking, physical activity, dietary intakes and alcohol consumption, with blood lipids profile.

Design and participants Data drawn from a cross-sectional study involving participants aged 18 years and over (n=363) from the nationwide WHO STEPwise Nutrition and Non-communicable Disease Risk Factor survey in Lebanon.

Measures Demographic characteristics, behaviours and medical history were obtained from participants by questionnaire. Dietary assessment was performed using a 61-item Culture-Specific Food Frequency Questionnaire that measured food intake over the past year. Lipid levels were measured by the analysis of fasting blood samples (serum total cholesterol (TC), triglycerides (TG), very low-density lipoprotein (VLDL), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C)).

Results Current cigarette smoking, alcohol consumption and low physical activity were prevalent among 33.3%, 39.7% and 41.6% of the sample, respectively. The contributions of fat and saturated fat to daily energy intake were high, estimated at 36.5% and 11.4%, respectively. Abnormal levels of TC, TG, VLDL, LDL-C and HDL-C were observed for 55.4%, 31.4%, 29.2%, 47.5% and 21.8% of participants, respectively. Adjusting for potential confounders, cigarette smoking was positively associated with higher odds of TG and VLDL (OR=4.27; 95% CI 1.69 to 10.77; and 3.26; 95% CI 1.33 to 8.03, respectively) with a significant dose–response relationship (p value for trend=0.010 and 0.030, respectively). Alcohol drinking and high saturated fat intake (≥10% energy intake) were associated with higher odds of LDL-C (OR=1.68; 95% CI 1.01 to 2.82 and OR= 1.73; 95% CI 1.02 to 2.93). Physical activity did not associate significantly with any blood lipid parameter.

Conclusion The demonstrated positive associations between smoking, alcohol drinking and high saturated fat intake with adverse lipoprotein levels lay further evidence for clinical practitioners, public health professionals and dietitians in the development of preventive strategies among subjects with a high risk of cardiovascular diseases in Lebanon and other neighbouring countries with similar epidemiological profile.

Strengths and limitations of this study

- A nationwide survey administered in Lebanon, following WHO STEPwise approach to surveillance guidelines, thus allowing for comparison with international literature.
- Low response rate (27.3%) for those who consented and gave fasting blood samples. Yet, differences between responders and non-responders were not significant on a number of sociodemographic characteristics.
- While data on smoking, physical activity and dietary intakes were sufficiently detailed, measures of exposure to alcohol consumption lacked information on intensity and type and may be prone to misclassification bias.
- Attention to associations between behavioural factors and dietary intakes with intermediary metabolic conditions along the causal pathway for cardiovascular diseases informs work towards the development of tailored preventive strategies for high-risk individuals.

INTRODUCTION

The prevalence of cardiovascular diseases (CVD) is growing worldwide, and has reached epidemic levels, affecting both developed and developing countries.[1] The Middle East and North Africa countries represent a region which is now facing a fast rate of development and urbanisation, with rates of chronic diseases increasing at an alarming rate and exceeding at times those of developed countries.[2] In Lebanon, a small middle-income country at the Eastern Mediterranean shore, data from WHO indicate that the proportional mortality from CVD alone is 45%,

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making it the highest among all non-communicable diseases (NCDs).[3] With a population estimate of around 4.2 million and a gross domestic product of close to US$8520per capita, Lebanon is characterised by a high urbanisation rate (87%), a growing trend towards survival in later life, coupled with westernisation and modernisation in diet and lifestyle and higher uptake of NCD risk factors.[4]

The primary goal in the prevention and management of CVD is to identify and modify the underlying risk behaviours that are amenable to intervention, namely, cigarette smoking, physical inactivity, dietary intakes and alcohol consumption. Associations between these factors and CVD risk through their effect on blood lipid levels have been widely examined in the western literature. Studies have shown that smokers are 2–4 times more at risk of developing heart disease than non-smokers[5] and the number of cigarettes smoked/day independently predicts higher levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG).[6] Also, smoking cessation has been shown to improve high-density lipoprotein cholesterol (HDL-C) levels.[7] Cigarette smoking is described as a strong inflammation mediator and a key promoter in the atherosclerotic process. Similarly, the anti-inflammatory effect of frequent physical activity has been noted to be the reason behind reduced heart disease risk among physically active individuals.[8] Regular physical activity with weight reduction has a large beneficial impact on the lipoproteins profile of adult men and women,[9] by increasing plasma volume, decreasing blood thickness, and thus reducing LDL-C concentrations.[10] Also, systematic reviews and meta-analysis of intervention studies have shown that heavy alcohol drinking results in an elevation in TG levels, while moderate consumption increases circulating levels of HDL-C.[11] Similarly, diet is recognised as a modifiable risk factor that can make a substantial contribution to the risk of CVD.[12 13] Energy intake, the types of fatty acids consumed and the level of sugar ingestion, may impact the lipid and cardiometabolic profile.[14] The 2017 Presidential Advisory from the American Heart Association (AHA) indicated that the replacement of saturated fat with unsaturated fatty acids decreases LDL-C levels and CVD risk, while replacing it with refined carbohydrates and sugar yields no significant benefits to cardiovascular health.[14]

Much of the above evidence comes from studies conducted in North American and European countries and to a lesser extent from the Far East, mostly among Japanese and Korean population. Arab populations including the Lebanese have quite different risk behaviours, varied dietary habits and risk profile,[15] and studies evaluating the association between behaviours and intermediary variables along the causal pathway of CVD including lipid levels remain scarce in the region. Compared with other neighbouring countries, Lebanon was shown to have one of the highest prevalence estimates of metabolic syndrome,[16] and has been witnessing high rates of smoking, among both adult men and women aged 18 years and over (42.9% and 27.5%, respectively).[17] Using data from a nationwide population-based survey of Lebanese adults, this study aims to examine the relation between behavioural risk factors including cigarette smoking, physical activity, dietary intakes and alcohol consumption with serum lipids and lipoproteins, while taking into account several potential confounding factors. Findings from this study inform prevention strategies among subjects with a high risk of CVD in the country.

METHODS

Study design and participants

The data presented in this study are derived from WHO Nutrition and Non-Communicable Diseases Risk Factor cross-sectional household survey conducted in Lebanon in 2009.[18] Using multistage stratified cluster study design, the sampling was based on the age–sex distribution of the Lebanese population as provided by the Central Administration for Statistics. One adult was randomly selected from each household using Kish methodology.[19] Pregnant and lactating women and individuals with mental disabilities were excluded. With a non-response rate of 10% at the individual level, this yielded a sample of 2668 survey participants aged 18 years and above. Those free from known history of hyperlipidaemia and diabetes in the first phase of the study (n=1331) were approached to undergo a biochemical assessment, of which 363 provided written consent and gave fasting blood samples. Further details on the design and sample of the survey are published elsewhere.[4]

Data collection

The data collection procedure followed WHO STEPwise approach to Surveillance,[20] and included the following three steps: step 1 questionnaire, whereby information about sociodemographic characteristics, NCDs and NCD risk factors, including dietary intake, were collected through face-to-face interviews; step 2 in which anthropometric and blood pressure measurements were taken using standardised techniques and calibrated equipment; and finally, step 3 in which the biochemical analysis for the assessment of the blood lipid profile was performed on blood samples collected after an overnight fast of at least 8 hours. Serum was centrifuged on site and shipped on dry ice to the American University of Beirut Laboratory.

Measures of blood lipids

Levels of blood lipids including TC, TG, very low density lipoprotein (VLDL), LDL-C and HDL-C were analysed using the Vitros 350 analyzer, an enzymatic spectrophotometric technique. The interassay variation of measurements did not exceed 4%. Quality control was performed within each run using standard performance verifier solutions provided by Ortho-Clinical Diagnostics. Analyses were conducted in duplicates, and the average value was used in the analysis. Based on the Adult Treatment Panel III guidelines,[21] the cut-off points used for the definition

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of risk levels of TC, TG, VLDL, LDL-C and HDL-C were ≥200, ≥150, ≥30, ≥130 and ≤40 mg/dL, respectively.

Behavioural risk factors and other measures

Behavioural risk factors examined in this study included cigarette smoking, physical activity and alcohol consumption. Cigarette smoking status (never, past and current) and intensity (number of cigarettes smoked/day) were assessed. Intensity was later categorised into three levels according to number of cigarettes/day (1–19, 20–39 and ≥40). The short version of the International Physical Activity Questionnaire (IPAQ) was used to assess physical activity among participants.[22] Three categories of physical activity (low, moderate and high) were assigned based on METs-min/week (MET-min being the product of the resting metabolic rate for an activity and the number of minutes taken to perform it). Alcohol-related behaviour was assessed as a dichotomous variable (ever vs never). Dietary assessment was performed using a 61-item Culture-Specific Food Frequency Questionnaire that measured food intake over the past year.[4 15] Intakes of energy and macronutrients were estimated using the food composition database of the Nutritionist IV software, and the food composition table for local and traditional Middle-Eastern foods.[23 24] Intakes of carbohydrates, fat and protein were compared with cut-offs within the Acceptable Macronutrient Distribution Range,[25] and intakes of saturated fat and sugar were compared with the recommendations of WHO.[26 27]

Covariates of interest included total daily caloric intake and body mass index (BMI), measured as the ratio of weight (kilograms) to the square of height (metres). In addition, gender, age (18–29, 30–39, 40–49, 50–59 and ≥60), marital status (single, married, divorced/widowed), education level (complementary or less, secondary/technical, university and above) and occupational status (student/volunteer, working, does not work/housewife/retired) were considered as potential covariates.

Patient and public involvement

This study is based on secondary data analyses. The original data collection tool was adapted from WHO STEPwise approach to NCD risk factor surveillance,[20] that did not directly involve patients or the public in outcome development or conduct of the study. However, we have been engaging with stakeholders to disseminate the findings on NCD risk factors, including tobacco consumption and dietary intakes, and on associations with various health-related outcomes to the public at large.

Statistical analysis

Means, SD and frequencies were used to describe the various sociodemographic, behavioural, nutritional and clinical characteristics of the participants. The associations between each of the risk factors and levels of the different blood lipids were examined using multiple logistic regression analysis. Unadjusted and adjusted ORs and their 95% CIs controlling for age, gender, education, marital status, caloric intake and BMI were estimated. Test for trend with increasing number of daily amount of cigarettes smoked was also conducted, and a two-sided $p<0.05$ was considered significant. The Statistical Package for the Social Sciences V.22.0.1 (SPSS) was used for all computations.

RESULTS

The sociodemographic and health-related characteristics of the study sample are summarised in table 1. The sample was equally divided by gender (49.9% women and 50.1% men), with a mean age of 39.2±15.2 years (range 18–92 years). The majority were married (60.6%), and close to half of the participants were employed (52.3%) at the time of the study, with a high percentage having less than complementary education (39.7%). Ever smokers constituted 37.5% of the sample, 41.6% were classified as being engaged in low-intensity physical activity and 39.7% reported ever alcohol drinkers. Average daily energy intake was estimated at 2656±1249 kcal/day, with 36.5% of caloric intake from fat, 11.4%% from saturated fat, 49.1% from carbohydrates, 5.7% from sugar and 15.2% from protein. Abnormal levels of TC, TG, VLDL, LDL-C and HDL-C were observed for 55.4%, 31.4%, 29.2%, 47.5% and 21.8% of the participants, respectively.

Tables 2a and 2b show the unadjusted and adjusted ORs for the association between behavioural risk factors and dietary variables with blood lipids levels. Associations with cigarette smoking were positive for most outcomes but significant for those consuming more than 40 cigarettes/day, compared with non-smokers, in the case of TG (unadjusted OR=5.03), VLDL (OR=4.09) and HDL-C (OR=3.02). Adjusting for potential confounders, the associations maintained statistical significance for TG and VLDL, with an adjusted OR of 4.27 (95% CI 1.69 to 10.77) and 3.26 (95% CI 1.33 to 8.03), respectively. Results showed a dose–response relationship with increasing number of cigarettes consumed for (p value for trend=0.010 and 0.030, respectively). In addition, a statistically significant association was observed between ever alcohol drinking and LDL-C (OR=1.53). This association retained statistical significance even after adjustment for potential confounders with an OR of 1.68 (95% CI 1.01 to 2.82). Out of all the dietary variables examined, only saturated fat was associated with blood lipids, namely TC and LDL-C, with an adjusted OR of 1.73 for both lipid abnormalities (95% CI 1.02 to 2.94 and 1.02 to 2.93, respectively). Physical activity was not associated with any of the blood lipid parameters.

DISCUSSION

Our results showed that heavy cigarette smoking is associated with increased levels of TG and VLDL, with findings showing significant dose–response relationships with increasing number of cigarettes smoked per day. The study also showed that alcohol drinking and high saturated fat

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Table 1 Distribution of sociodemographic characteristics, behavioural factors and lipid profile of the study population

	n	%
Gender (% female)	181	49.9
Age (mean±SD, years)	39.2±15.2	
18–29	113	31.1
30–39	106	29.2
40–49	67	18.5
50–59	32	8.8
≥60	45	12.4
Marital status		
Single	128	35.3
Married	220	60.6
Divorced/widowed	15	4.1
Work status		
Student or volunteer	31	8.5
Employed	190	52.3
Does not work/housewife/ retired	142	39.1
Educational level		
Complementary or less	144	39.7
Secondary or technical	99	27.3
University and above	120	33.1
Cigarette smoking		
Never smoked	227	62.5
Past smoker	15	4.2
Current smoker	121	33.3
Number of cigarettes smoked/ day		
0	227	62.5
1–19	43	11.8
20–39	63	17.4
≥40	30	8.3
Physical activity		
Low-intensity activity	151	41.6
Moderate-intensity activity	121	33.3
High-intensity activity	88	24.2
Alcohol consumption (ever drinker)	144	39.7
Total caloric intake (mean±SD, kcal/day)	2656±1249	
Fat intake (% of total energy)*	36.5±6.89	
≥30†	275	81.6
Saturated fat intake (% of total energy)*	11.4±2.92	
≥10‡	224	66.5
Carbohydrates intake (% of total energy)*	49.1±7.03	
≥55†	71	21.1
Sugar (% of total energy)*	5.7±4.64	
≥10§	56	16.6
Proteins intake (% of total energy)*	152±2.91	
<15†	143	42.4
Total cholesterol (mean±SD, mg/ dL)	210±45	
% Elevated total cholesterol (≥200 mg/dL)¶	201	55.4
Triglycerides (mean±SD, mg/dL)	138±78	
% Elevated triglycerides (≥150 mg/dL)¶	114	31.4
VLDL (mean±SD, mg/dL)	27±15	
% Elevated VLDL (≥30 mg/ dL)¶	106	29.2
LDL-C (mean±SD, mg/dL)	131±39	
% Elevated LDL-C (≥130 mg/ dL) ¶	172	47.5
HDL-C (mean±SD, mg/dL)	51±14	
% Reduced HDL-C (≤40 mg/ dL) ¶	79	21.8

*Dietary variables are based on a sample of 337 subjects owing to missing data.
†Macronutrient cut-offs are within the acceptable macronutrient distribution range.[25]
‡Saturated fat cut-off based on WHO recommendations.[26]
§Sugar intake cut-off based on WHO recommendations.[27]
¶Lipid cut-off values based on the Adult Treatment Panel III guidelines.[21]
HDL-C, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; VLDL, Very low density lipoprotein.

intake are significantly associated with higher levels of LDL-C. However, there were no consistent associations between physical activity and fasting blood lipids profile. To our knowledge, this is the first study in Lebanon to explore these associations, based on objectively measured lipid parameters and using standardised laboratory techniques and tools, while taking into consideration the effect of potential confounders.

There are strong indicators in the literature that the deleterious effect of cigarette smoking on heart disease and atherosclerosis is partially explained by the effect of smoking on the concentration of blood lipids and lipoproteins. Our study confirms the results of earlier cross-sectional studies showing that the association between smoking and lipoproteins is observed in the levels of TG, VLDL and HDL-C,[6 28 29] with the impact on lipid levels increasing with increase in the number of cigarettes smoked/day in a dose-dependent relationship.[30 31] Our observation of the largest effect of cigarette smoking on lipid parameters being seen in those who smoked more than two packs a day is also consistent with Chen *et al*

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Table 2a Logistic regression analysis: associations of behavioural risks with total cholesterol (TC) and triglycerides (TG)

	TC			TG		
	%≥200 mg/dL	Crude OR (CI)	Adjusted* OR (CI)	%≥150 mg/dL	Crude OR (CI)	Adjusted* OR (CI)
Number of cigarettes						
0	52.0	1.00	1.00	25.6	1.00	1.00
1–19	55.8	1.17 (0.60 to 2.25)	0.85 (0.40 to 1.77)	30.2	1.26 (0.62 to 2.58)	1.06 (0.48 to 2.33)
20–35	63.5	1.60 (0.90 to 2.85)	1.01 (0.51 to 2.00)	38.1	1.79 (0.99 to 3.23)	1.30 (0.67 to 2.53)
≥40	63.3	1.59 (0.73 to 3.50)	1.24 (0.48 to 3.18)	63.3	5.03 (2.26 to 11.2)	4.27 (1.69 to 10.77)
P trend		0.070	00.783		<0.001	0.010
Physical activity						
High	55.7	1.00	1	25.0	1.00	1
Moderate	56.2	1.02 (0.59 to 1.77)	0.89 (0.48 to 1.67)	32.2	1.43 (0.77 to 2.64)	1.64 (0.83 to 3.25)
Low	55.6	0.99 (0.59 to 1.69)	1.11 (0.31 to 2.00)	35.1	1.62 (0.90 to 2.92)	1.75 (0.93 to 3.29)
Alcohol intake						
No	53	1	1	31.5	1	1
Yes	59	1.28 (0.84 to 1.96)	1.52 (0.90 to 2.55)	31.3	0.99 (0.63 to 1.56)	0.98 (0.57 to 1.69)
Total fat intake (% energy)						
<30	54.8	1	1.00	30.6	1	1
≥30	54.9	1.03 (0.57 to 1.74)	1.15 (0.62 to 2.13)	31.3	1.03 (0.57 to 1.87)	1.22 (0.34 to 2.35)
Saturated fat (% energy)						
<10	52.2	1	1	31	1	1
≥10	56.3	1.17 (0.75 to 1.85)	1.73 (1.02 to 2.94)	31.3	1.01 (0.62 to 1.65)	1.01 (0.59 to 1.74)
Carbohydrates (% energy)						
<55	53.8	1	1	30.8	1	1
≥55	59.2	1.25 (0.73 to 2.12)	1.07 (0.59 to 1.93)	32.4	1.07 (0.61 to 1.88)	0.96 (0.52 to 1.76)
Sugar intake (% energy)						
<10	54.8	1	1	32	1	1
≥10	55.4	1.02 (0.57 to 1.82)	1.12 (0.59 to 2.16)	26.8	0.78 (0.41 to 1.48)	0.86 (0.43 to 1.73)
Protein intake (% energy)						
<15	56.6	1	1	36.4	1	1
≥15	53.6	0.88 (0.57 to 1.37)	0.83 (0.50 to 1.37)	27.3	0.66 (0.41 to 1.05)	0.79 (0.47 to 1.32)

*Controlling for age, gender, education, marital status, caloric intake and body mass index.

Table 2b Logistic regression analysis: associations of behavioural risks with very low density lipoprotein (VLDL), low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C)

	VLDL			LDL-C			HDL-C		
	%≥30 mg/dL	Crude OR (CI)	Adjusted* OR (CI)	%≥130 mg/dL	Crude OR (CI)	Adjusted* OR (CI)	%<40 mg/dL	Crude OR (CI)	Adjusted OR (CI)
Number of cigarettes									
0	24.2	1.00	1	43.6	1.00	1	18.1	1.00	1.00
1–19	27.9	1.21 (0.58 to 2.52)	1.01 (0.45 to 2.25)	53.5	1.49 (0.77 to 2.86)	1.12 (0.54 to 2.33)	18.6	1.04 (0.45 to 2.40)	1.01 (0.39 to 2.63)
20–39	35.5	1.72 (0.94 to 3.14)	1.27 (0.64 to 2.51)	54.8	1.57 (0.89 to 2.76)	0.97 (0.50 to 1.89)	28.6	1.81 (0.95 to 3.45)	1.61 (0.75 to 3.45)
≥40	56.7	4.09 (1.87 to 8.95)	3.26 (1.33 to 8.03)	53.3	1.48 (0.69 to 3.17)	0.96 (0.39 to 2.40)	40.0	3.02 (1.35 to 6.76)	1.98 (0.77 to 5.08)

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Table 2b Continued

	VLDL			LDL-C			HDL-C		
	%≥30 mg/dL	Crude OR (CI)	Adjusted* OR (CI)	%≥130 mg/dL	Crude OR (CI)	Adjusted* OR (CI)	%<40 mg/dL	Crude OR (CI)	Adjusted OR (CI)
P trend		<0.001	0.030		0.078	0.946		<0.001	0.106
Physical activity									
High	26.1	1.00	1	48.9	1.00	1	27.3	1.00	1
Moderate	28.9	1.15 (0.62 to 2.13)	1.27 (0.54 to 2.52)	49.6	1.02 (0.59 to 1.78)	0.89 (0.48 to 1.65)	19.0	0.62 (0.32 to 1.20)	0.93 (0.43 to 2.01)
Low	32.0	1.33 (0.74 to 2.39)	1.44 (0.76 to 2.71)	46.0	0.89 (0.53 to 1.51)	0.93 (0.52 to 1.65)	21.2	0.72 (0.40 to 1.32)	0.69 (0.34 to 1.39)
Alcohol intake									
No	28.3	1	1	43.4	1	1	20.5	1	1
Yes	30.8	1.12 (0.71 to 1.79)	1.12 (0.65 to 1.93)	53.8	1.53 (1.00 to 2.33)	1.68 (1.01 to 2.82)	23.6	1.19 (0.72 to 1.98)	0.98 (0.54 to 1.78)
Total fat intake (% energy)									
<30	29.0	1	1	45.2	1	1	25.8	1	1
≥30	28.8	0.99 (0.54 to 1.82)	1.15 (0.59 to 2.22)	46.7	1.06 (0.61 to 1.85)	1.26 (0.69 to 2.33)	21.8	0.80 (0.42 to 1.52)	0.81 (0.39 to 1.65)
Saturated fat (% energy)									
<10	29.2	1	1	44.2	1	1	20.4	1	1
≥10	28.7	0.98 (0.59 to 1.61)	0.96 (0.56 to 1.66)	47.5	1.15 (0.72 to 1.80)	1.73 (1.02 to 2.93)	23.7	1.21 (0.70 to 2.16)	1.16 (0.62 to 2.17)
Carbohydrates (% energy)									
<55	28.7	1	1	45.7	1	1	22.2	1	1
≥55	29.6	1.04 (0.59 to 1.86)	0.96 (0.52 to 1.79)	49.3	1.16 (0.68 to 1.95)	0.93 (0.52 to 1.67)	23.9	1.10 (0.60 to 2.05)	1.18 (0.59 to 2.36)
Sugar intake (% energy)									
<10	29.6	1	1	47.1	1	1	23.8	1	
≥10	25.0	0.79 (0.41 to 1.53)	0.89 (0.44 to 1.82)	42.9	0.84 (0.47 to 1.50)	0.89 (0.47 to 1.71)	16.1	0.61 (0.28 to 1.31)	0.62 (0.26 to 1.45)
Protein intake (% energy)									
<15	32.9	1	1	48.3	1	1	21	1	1
≥15	25.9	0.71 (0.44 to 1.15)	0.85 (0.50 to 1.44)	45.1	0.88 (0.57 to 1.36)	0.75 (0.46 to 1.24)	23.7	1.17 (0.59 to 1.97)	1.49 (0.82 to 2.72)

*Controlling for age, gender, education, marital status, caloric intake and body mass index.

study comprising 1164 men in Taiwan.[32] One popular mechanism by which smoking affects lipoproteins is that cigarette particulate matter alters catecholamine release—and thus free fatty acid release, which in turn contributes to the accumulation of the LDL-C concentrations and to lower levels of HDL-C in the blood.[33] The association between cigarette smoking and the increase in TG can also be explained by the decrease in the activity of the lipoprotein lipase among smokers thus disrupting lipid and lipoprotein metabolism. Furthermore, smoking cessation was found to improve lipid and lipoprotein levels in observational studies and randomised clinical trials.[7 34] Taken together, the totality of evidence from these studies and our data, including consistency on replication across various studies, the dose–response relationship, the magnitude and significance of association, biological plausibility as well as effects of smoking cessation on lipoprotein levels, supports a strong relationship between smoking and lipid profiles.

Our study showed that alcohol drinking was associated with higher LDL-C levels in Lebanese adults but was not associated with other lipid parameters. Available evidence on the impact of alcohol on LDL-C is conflicting with recent studies suggesting that the association of alcohol and LDL-C levels may be population specific.[35] For instance, studies conducted among Danish adults reported an inverse association between alcohol intake and LDL-C,[36] while studies conducted in Spanish and Italian populations found that higher alcohol consumption was associated with increased LDL-C.[37 38] According to a review by Brinton (2012), these inconsistent findings on the association between alcohol intake and LDL-C may be explained by allele-specific genetic effects, such as the Apo E4 and Apo A5 genes.[39] In our study, alcohol

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drinking was not associated with HDL-C levels. Findings on the relationship between alcohol and HDL-C have in fact been less consistent in the literature, with some but not all showing an effect on higher HDL-C levels.[40 41] Indeed, the effect of alcohol on CVD including coronary artery disease, stroke and myocardial infarction appears to be biphasic showing a J-shaped or U-shaped relationship based on the amount of alcohol consumed,[41 42] with lower CVD mortality risks by light-to-moderate alcohol consumption and increased risk by heavy alcohol intake.[43] The discrepancy between our results with those in the literature may be due to differences in race/ethnicity and genetic variations.[44] Alternatively, it may be due to the definition of alcohol exposure adopted in our study which was based on a dichotomous variable (ever vs never), and thus did not capture alcohol intake in terms of frequency, intensity, types and pattern of alcohol consumed. It is important to also acknowledge that alcohol consumption may be subject to reporting bias in the Lebanese society due to cultural or religious norms. The observed association between alcohol and lipid profile should therefore be interpreted with caution, as it may have been the artefact of other social or lifestyle factors that were not measured in our study.

Dietary intakes are among the modifiable risk factors that may modulate plasma lipids and the risk of CVD. In our study, saturated fat was the only dietary factor that was significantly associated with lipid parameters, and specifically with TC and LDL-C. Despite the increasing controversy around the relationship between saturated fat and blood cholesterol levels, an increasing body of evidence highlights the strong atherogenicity of saturated fatty acids through their impact on LDL-C.[14] A systematic review and metaregression analysis published in 2016 showed that a decrease in saturated fats of 1% of daily energy intake coupled with an increase of 1% in polyunsaturated fat lowered LDL cholesterol by 2.1 mg/dL.[45] In 2017, a Presidential Advisory from the AHA concluded that available evidence strengthens the long-standing AHA recommendations to decrease saturated fat intake and replace it with unsaturated fats. The AHA Advisory highlighted that the shift from saturated to unsaturated fats should occur in the context of an overall healthful dietary pattern such as the Dietary Approaches to Stop Hypertension (DASH) or Mediterranean patterns.[14]

In our study, physical activity did not appear to be associated with lipid parameters. The literature suggests that physical activity increases the level of HDL-C and has beneficial effects on lipoprotein particle size and number.[46] Also, moderate intensity exercise is known to have a more favourable effect on blood lipids since it allows the use of lipids as a fuel source which implies an increase in the uptake and oxidation of the lipids in the skeletal muscle.[46]

Some other limitations should be taken into consideration when interpreting the results of our study. Because the study used a cross-sectional design, findings only imply associations and causal relationships cannot be established. The proportion of participants who gave fasting blood samples (of at least 8 hours) was relatively small (27.3%); however, responders were comparable to non-responders on a number of sociodemographic characteristics except for marital status (61% of responders vs 50% of non-responders were married). Also, we had earlier documented comparable dietary data between respondents and non-respondents based on factor loading matrices on patterns of food groups intake.[4] As mentioned earlier, measures of exposure were self-reported which can introduce some misclassification error. This may have been particularly problematic in the case of alcohol consumption, given that our definition based on a dichotomous variable of 'ever' versus 'never' does not allow for the assessment of drinking frequency and patterns or the type of alcohol consumed. In contrast, our measurement of cigarette smoking and physical activity was more detailed and reliable. Information on cigarette smoking in our study included the amount of cigarettes smoked and the standardised IPAQ was used to assess physical activity.

To conclude, our results suggest that smoking, alcohol drinking and high saturated fat are associated with adverse levels of lipoprotein, among Lebanese men and women. There is enough evidence in the literature indicating the role of lipoproteins in atherogenesis and that controlling for blood lipid levels decreases the risk of heart and many other chronic diseases. Our findings lay further evidence for clinical practitioners, public health professionals and dieticians regarding the potential benefits of lifestyle and dietary modifications in their pursuit to curb the burden of hyperlipidaemia and CVD at the individual and population level. The overall high rate of smoking behaviour in Lebanon among both men and women, coupled with the shift in dietary patterns towards high fat energy dense foods,[7 15] is likely to adversely impact on the healthcare bill in the country. Further studies with larger sample size that examine the association of combination patterns of poor lifestyle factors on lipid profile among Lebanese adults are warranted.

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Contributors MM conducted initial analyses and wrote the first draft, in partial fulfillment of her MSc in epidemiology. HT supervised the conduct of analyses and contributed to the write-up. LN conducted the dietary analyses and contributed to the write-up and revisions. CEK, NH, MC and AF provided statistical advice and contributed to the paper revisions. AMS coordinated original study conduct, conceived and finalised the analyses, and contributed substantially to the write-up and revisions. All authors read and approved the final version of the manuscript.

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