

BMJ Open

BMJ Open is committed to open peer review. As part of this commitment we make the peer review history of every article we publish publicly available.

When an article is published we post the peer reviewers' comments and the authors' responses online. We also post the versions of the paper that were used during peer review. These are the versions that the peer review comments apply to.

The versions of the paper that follow are the versions that were submitted during the peer review process. They are not the versions of record or the final published versions. They should not be cited or distributed as the published version of this manuscript.

BMJ Open is an open access journal and the full, final, typeset and author-corrected version of record of the manuscript is available on our site with no access controls, subscription charges or pay-per-view fees (<http://bmjopen.bmj.com>).

If you have any questions on BMJ Open's open peer review process please email info.bmjopen@bmj.com

BMJ Open

Prevalence and associations of behavioral risk factors with blood lipids profile in Lebanese adults: findings from the WHO STEPwise NCD Survey

Journal:	<i>BMJ Open</i>
Manuscript ID	bmjopen-2018-026148
Article Type:	Research
Date Submitted by the Author:	20-Aug-2018
Complete List of Authors:	Mansour, Megali; American University of Beirut Faculty of Health Sciences, EPHD Tamim, Hani; American University of Beirut, Biostatistics Unit, Clinical Research Institute EL Khoury, Christelle; Tufts University School of Medicine Hwalla, Nahla; American University of Beirut Chaaya, Monique; American University of Beirut Farhat, Antoine ; Notre Dame University Sibai, Abla; American University of Beirut, Department of Epidemiology and Population Health
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cigarette Smoking, Blood lipids

SCHOLARONE™
Manuscripts

1
2
3 **Prevalence and associations of behavioral risk factors with blood lipids profile in**
4
5 **Lebanese adults: findings from the WHO STEPwise NCD Survey**
6
7

8 Magali Mansour^{1†}, Hani Tamim^{2†}, Christelle El Khoury³, Nahla Hwalla⁴, Monique Chaaya¹,
9 Antoine Farhat⁵, Abla M. Sibai^{1*}
10
11

12
13 **Author affiliations**

14 ¹ Department of Epidemiology and Population Health, Faculty of Health Sciences, American
15 University of Beirut, P.O.Box 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon
16

17 ² Department of Internal Medicine , American University of Beirut-Medical Center, P.O.Box
18 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon
19

20 [†] Equal Contribution

21 ³ School of Medicine Department of Public Health, Tufts University, 136 Harrison Avenue,
22 Boston, MA 02111, US
23

24 ⁴ Faculty of Agriculture and Food Sciences, American University of Beirut, P.O.Box 11-0236,
25 Riad El-Solh, Beirut 1107 2020, Lebanon
26

27 ⁵ Faculty of Nursing and Health Sciences, Notre Dame University, Lebanon
28
29
30
31
32

33 ***Corresponding author:**

34 **Abla M. Sibai**
35
36

37 Epidemiology and Population Health
38 Faculty of Health Sciences
39 American University of Beirut
40 PO Box 11-0236
41 Beirut-Lebanon
42 Tel: +961-1-350000 ext. 4647
43 Fax: +961-1-744470
44 Email: am00@aub.edu.lb
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

1
2
3 **Keywords:** Cardiovascular disease, cigarette smoking, alcohol consumption, physical
4
5 inactivity, blood lipids, Lebanon
6
7

8
9 **Word count (excluding title page, references, tables and figures):** 2358
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

Abstract

Objective: To examine associations of behavioral risk factors, including cigarette smoking, physical activity 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 national WHO Stepwise Nutrition and Non-communicable Disease Risk Factor survey in Lebanon.

Measures: Demographic characteristics, behaviors and medical history were obtained from participants by questionnaire. Lipid levels were measured by 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%, 15.4%, and 41.6% of the sample, respectively. Abnormal levels of total cholesterol, triglyceride (TG), very low density lipoprotein (VLDL), low density lipoprotein (LDL-C), and high density lipoprotein were observed for 54.4%, 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=3.82; 95% CI 1.50-9.71; and 4.75; 95% CI 1.10-6.72) with a significant dose-response relationship (p-value for trend=0.019 and 0.039, respectively). Physical activity and alcohol intake did not associate significantly with any blood lipid parameter. **Conclusion:** The demonstrated positive association between smoking and adverse lipoprotein levels in this study population in Lebanon lays further evidence for clinical practitioners, public health professionals and dieticians in the development of preventive strategies among subjects with high risk of CVDs

Strengths and limitations

- A nationwide survey administered in Lebanon with a population distribution similar to that in national records.
- Attention to associations between behavioral factors and intermediary metabolic conditions along the causal pathway for CVD informs work towards development of tailored preventive strategies for high risk individuals.
- Whilst data on smoking and physical activity were sufficiently detailed, measures of exposure to alcohol consumption lacked information on intensity and type, and may be prone to misclassification bias.

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 (MENA) countries represent a region which is now facing a fast rate of development and urbanization, 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 the World Health Organization (WHO) indicate that the proportional mortality from CVD alone is 45%, 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 (GDP) of 7,315 US Dollars per capita, Lebanon is characterized by a high urbanization rate (87%), a growing trend towards survival in later life, coupled with westernization and modernization in 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 behaviors that are amenable to intervention, namely, cigarette smoking, physical inactivity 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 nonsmokers.⁵ and the number 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

1
2
3 with weight reduction has a large beneficial impact on the lipoproteins profile of adult men
4 and women,⁹ by increasing plasma volume, decreasing blood thickness, and thus reducing
5 LDL-C concentrations.¹⁰ Also, systematic reviews and meta-analysis of intervention studies
6
7
8 have shown that heavy alcohol drinking results in an elevation in triglyceride levels, while
9 moderate consumption increases circulating levels of HDL-C.¹¹
10
11
12

13 Much of the above evidence comes from studies conducted in North American and
14 European countries and to a lesser extent from the Far East, mostly among Japanese and
15 Korean population. Arab populations including the Lebanese have quite different risk
16 behaviors, varied dietary habits and risk profile,¹² and studies evaluating the association
17 between behaviors and intermediary variables along the causal pathway of CVD
18 including lipid levels remain scarce in the region. Compared with other neighboring
19 countries, Lebanon was shown to have one of the highest prevalence estimates of
20 metabolic syndrome,¹³ and has been witnessing high rates of smoking, among both
21 adult men and women aged 18 years and over (42.9% and 27.5%, respectively).¹⁴ Using
22 data from a nation-wide population-based survey of Lebanese adults, this study aims to
23 examine the relation between behavioral risk factors including cigarette smoking,
24 physical activity and alcohol consumption with serum lipids and lipoproteins, while
25 taking into account several potential confounding factors. Findings from this study
26 inform prevention strategies among subjects with high risk of CVD in the country.
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Methods

Study design and participants

The data presented in this study are derived from the WHO Nutrition and Non-Communicable Diseases Risk Factor (NNCD-RF) cross-sectional household survey conducted in Lebanon in 2009.¹⁵ Using multi-stage 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.¹⁶ Pregnant and lactating women and individuals with mental disabilities were excluded. Survey participants aged 18 years and above and free from known history of hyperlipidemia and diabetes in the first phase of the study (n = 1,331) 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 The study protocol was approved by the Institutional Review Board of the American University of Beirut (AUB), and informed consent was taken from all participants.

Data collection

The data collection procedure followed the WHO STEPwise approach to Surveillance,¹⁷ and included the following three steps: Step 1 Questionnaire, whereby information about socio-demographic characteristics, NCDs and NCD risk factors were collected through face-to-face interviews; Step 2 in which anthropometric and blood pressure measurements were taken using standardized techniques and calibrated equipment; and finally, Step 3 in which biochemical analysis for assessment of the blood lipid profile was performed on blood samples collected after an overnight fast. Serum was centrifuged on site and shipped on dry ice to the AUB Laboratory.

Measures of blood lipids

Levels of blood lipids including TC, TG, very low density lipoprotein (VLDL), LDL-C and HDL-C were analyzed using the Vitros 350 analyzer, an enzymatic spectrophotometric technique. The inter-assay 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 utilized in the analysis. Based on the Adult Treatment Panel III guidelines,¹⁸ the cutoff points used for the definition of risk levels of TC, TG, VLDL, LDL-C and HDL-C were ≥ 200 , ≥ 150 , ≥ 30 , ≥ 130 and ≤ 40 mg/dl, respectively.

Behavioral risk factors and other measures

Behavioral 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 categorized into three levels according to number of cigarettes/day (1 to 19, 20 to 39 and ≥ 40). The short version of the International Physical Activity Questionnaire was used to assess physical activity among participants.¹⁹ 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 consumption was assessed as a dichotomous variable (yes/no) based on consumption of at least 1 drink/week in the year prior to the survey. Covariates of interest included total daily caloric intake, which was estimated based on the food frequency questionnaire and divided into tertiles. 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 the WHO STEPwise approach to NCD risk factor surveillance,¹⁷ that did not directly involve patients or the public in outcome development or conduct of the study. However, we are engaging with stakeholders to disseminate the findings on the burden of tobacco consumption and its association with various health-related outcomes to the public at large.

Statistical analysis

Means, standard deviations (SD) and frequencies were used to describe the various socio-demographic, behavioral, nutritional and clinical characteristics of the participants. The associations between each of the three behavioral risk factors and levels of the different blood lipids were examined using multiple logistic regression analysis. Unadjusted and adjusted odds ratios (ORs) and their 95% confidence intervals (CIs) controlling for age, gender, education, marital status and caloric intake were estimated. Test for trend with increasing number of daily amount of cigarette smoked was also conducted, and a two-sided p-value < 0.05 was considered significant. The Statistical Package for the Social Sciences 22.0.1 (SPSS Inc., Chicago, IL, USA) was used for all computations.

Results

The socio-demographic and health-related characteristics of the study sample are summarized in Table 1. The sample was equally divided by gender (49.9% females and 50.1% males), with a mean age of 39.2 ± 15.2 years (range 18 to 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 an almost equal distribution across the various educational levels. Ever smokers constituted 37.4% of the sample. 41.6% were classified as being engaged in low-intensity

1
2
3 physical activity, and 15.4% reported current alcohol consumption at least once per week.

4
5 The sample consumed 2656 ± 1249 Kcal/day on average. Abnormal levels of TC, TG,
6
7 VLDL, LDL-C, and HDL-C were observed for 55.4%, 31.4%, 29.2%, 47.5% and 21.8% of
8
9 the participants, respectively.

10
11
12 Insert table 1 around here

13
14
15
16 Tables 2a and 2b show the unadjusted and adjusted ORs for the association between
17
18 behavioral risk factors and blood lipids levels. Out of all the relationships examined, only
19
20 cigarette smoking showed a significant association with blood lipids. Associations were
21
22 significant for those consuming more than 40 cigarette/day compared to non-smokers, with
23
24 unadjusted ORs of 5.03 for TG, 4.09 for VLDL and 3.02 for HDL-C. Adjusting for potential
25
26 confounders, the associations maintained statistical significance for TG and VLDL, with an
27
28 adjusted OR 3.82 (95% CI 1.50- 9.71) and 4.75 (95% CI 1.10-6.72), respectively. Results
29
30 showed a dose-response relationship with increasing number of cigarettes consumed for (p-
31
32 value for trend = 0.019 and 0.039, respectively). Physical activity and alcohol intake were not
33
34 associated with any of the blood lipid parameters.
35
36
37
38
39
40
41
42
43
44
45

46
47
48
49
50
51
52
53
54
55
56
57
58
59
60
Insert tables 2a and 2b around here

Discussion

Our results showed that heavy cigarette smoking exceeding 40 cigarettes/day is associated with increased levels of TG and VLDL, with findings showing significant dose-response relationships with increasing number of cigarettes smoked per day. However, there were no consistent associations between physical activity and alcohol consumption with fasting blood

1
2
3 lipids profile. To our knowledge, this is the first study in Lebanon to explore these
4
5 associations, based on objectively measured lipid parameters and using standardized
6
7 laboratory techniques and tools, while taking into consideration the effect of potential
8
9 confounders.

10
11
12 There are strong indicators in the literature that the deleterious effect of cigarette smoking on
13
14 heart disease and atherosclerosis is partially explained by the effect of smoking on the
15
16 concentration of blood lipids and lipoproteins. Our study confirms the results of earlier cross-
17
18 sectional studies showing that the association between smoking and lipoproteins are observed
19
20 in the levels of TG, VLDL and HDL-C, ^{6,20,21} with the impact on lipid levels increasing with
21
22 increase in the number of cigarettes smoked/day in a dose dependent relationship. ^{22,23} Our
23
24 observation of the largest effect of cigarette smoking on lipid parameters being seen in those
25
26 who smoked > 40 cigarettes/day is also consistent with Chen et al. study comprising of 1,164
27
28 men in Taiwan. ²⁴ One popular mechanism by which smoking affects lipoproteins is that
29
30 cigarette particulate matter alters catecholamine release—and thus free fatty acid release,
31
32 which in turn contributes to the accumulation of the LDL-C concentrations and to lower
33
34 levels of HDL-C in the blood. ²⁵ The association between cigarette smoking and the increase
35
36 in TG can also be explained by the decrease in the activity of the lipoprotein lipase among
37
38 smokers thus disrupting lipid and lipoprotein metabolism. Furthermore, smoking cessation
39
40 was found to improve lipid and lipoprotein levels in observational studies and randomized
41
42 clinical trials. ^{7,26} Taken together, the totality of evidence from these studies and our data-
43
44 including consistency upon replication across various studies, the dose-response relationship,
45
46 the magnitude and significance of association, biological plausibility as well as effects of
47
48 smoking cessation on lipoprotein levels, supports a strong relationship between smoking and
49
50 lipid profiles.
51
52
53
54
55
56
57
58
59
60

1
2
3 In our study, physical activity and alcohol consumption did not appear to be associated with
4 lipid parameters. The literature suggests that physical activity increases the level of HDL-C
5 and has beneficial effects on lipoprotein particle size and number.²⁷ Also, moderate intensity
6 exercise is known to have a more favorable effect on blood lipids since it allows the use of
7 lipids as a fuel source which implies an increase in the uptake and oxidation of the lipids in
8 the skeletal muscle.²⁷ As for the relationship between alcohol consumption and lipid
9 parameters in the general population, findings have been less consistent in the literature, with
10 some but not all showing an effect on higher HDL-C levels and lower non-HDL-C levels.^{28,29}
11 Indeed, the effect of alcohol on CVD including coronary artery disease, stroke and
12 myocardial infarction appears to be biphasic showing a J or U-shaped relationship based on
13 the amount of alcohol consumed,^{29,30} with lower CVD mortality risks by light to moderate
14 alcohol consumption and increased risk by heavy alcohol intake.³¹ The discrepancy between
15 our results with those in the literature may be due to differences in race/ethnicity and genetic
16 variations known to play a significant role in lipid metabolism,³² or alternatively, owing to
17 measurement error in our assessment of alcohol consumption, based on self-reports of a
18 dichotomous variable. Lipid profiles are influenced by the type of alcohol consumed and by
19 drinking frequency and patterns.

20 Some other limitations should be taken into consideration when analyzing the results of our
21 study. Because the study used a cross-sectional design, the results only imply associations
22 and findings cannot establish causal relationships. The number of participants who agreed to
23 give blood samples was relatively small; however, these responders were comparable to
24 non-responders on a number of socio-demographic characteristics except for marital status
25 (61% of responders vs. 50% of non-responders were married). As mentioned earlier,
26 measures of exposure were self-reported which can introduce some misclassification error.
27 Whilst this may have been problematic in case of alcohol consumption that lacked detailed

1
2
3 data on intensity and type, measurement of cigarette smoking and physical activity were more
4
5 detailed and reliable. Information on cigarette smoking in our study included the amount of
6
7 cigarettes smoked and the standardized IPAQ was used to assess physical activity.
8

9 To conclude, our results suggest that smoking is associated with adverse levels of
10
11 lipoprotein, notably TGs and VLDL among Lebanese men and women. The role of
12
13 lipoproteins in atherogenesis has been clearly defined, and controlling for blood lipid levels
14
15 decreases the risk of heart and many other chronic diseases. Our findings lay further evidence
16
17 for clinical practitioners, public health professionals and dieticians regarding the potential
18
19 benefits of smoking cessation in their pursuit to curb the burden of hyperlipidemia and CVD
20
21 at the individual and population level. The overall high rate of smoking behavior in Lebanon
22
23 among both men and women, coupled with lack of dedicated funds and weak implementing
24
25 power for the enforcement of the tobacco control law, are likely to adversely impact on the
26
27 healthcare bill in the country. Further studies with larger sample size that examine the
28
29 association of combination patterns of poor lifestyle factors on lipid profile among Lebanese
30
31 adults are warranted.
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Contributors

AMS conceived the study. MM did the analyses and wrote first draft of the paper. HT and AMS supervised the conduct of analyses and contributed substantially to the write-up of the paper. CK, NH, MC and AF provided statistical advice and contributed to the paper revision. All authors read and approved the final manuscript.

Funding statement

The original study conduct was funded by the World Health Organization (WHO) - Lebanon.

Data sharing statement

The database set was available for all authors of the study, and will be available for other non-commercial researchers on request.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

REFERENCES

1. World Health Organization. Cardiovascular diseases. Fact sheet N°317. 2015.
2. Sibai AM, Nasreddine L, Mokdad AH, Adra N, Tabet M, Hwalla N. Nutrition transition and cardiovascular disease risk factors in Middle East and North Africa countries: reviewing the evidence. *Annals of Nutrition and Metabolism*. 2010;57:193-203.
3. World Health Organization. Non-communicable Diseases Country Profiles 2014.
4. Naja F, Nasreddine L, Itani L, Adra N, Sibai A, Hwalla N. Association between dietary patterns and the risk of metabolic syndrome among Lebanese adults. *European Journal of Nutrition*. 2013;52:97-105.
5. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Atlanta (GA): The health consequences of smoking—50 years of progress: a report of the Surgeon General: Centers for Disease Control and Prevention (US), National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
6. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. *Journal of Clinical Lipidology*. 2009;3:372-8.
7. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. *American Heart Journal*. 2011;161:145-51.
8. [8] Abramson JL, Vaccarino V. Relationship between physical activity and inflammation among apparently healthy middle-aged and older US adults. *Archives of Internal Medicine*. 2002;162:1286-92.
9. [9] Barengo NC, Kastarinen M, Lakka T, Nissinen A, Tuomilehto J. Different forms of physical activity and cardiovascular risk factors among 24–64-year-old men and women in Finland. *European Journal of Cardiovascular Prevention & Rehabilitation*. 2006;13:51-9.
10. [10] Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Medicine and Science in Sports and Exercise*. 2001;33:S438-45.
11. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. *BMJ*. 2011;342:d636.
12. Naja F, Nasreddine L, Itani L, Chamieh MC, Adra N, Sibai AM, et al. Dietary patterns and their association with obesity and sociodemographic factors in a national sample of Lebanese adults. *Public Health Nutrition*. 2011;14:1570-8.
13. Sibai AM, Obeid O, Batal M, Adra N, El Khoury D, Hwalla N. Prevalence and correlates of metabolic syndrome in an adult Lebanese population. *CVD Prevention & Control*. 2008;3:83-90.

14. Sibai AM, Iskandarani M, Darzi A, Nakkash R, Saleh S, Fares S, et al. Cigarette smoking in a Middle Eastern country and its association with hospitalisation use: a nationwide cross-sectional study. *BMJ open*. 2016;6:e009881.
15. Sibai AM, Hwalla N. WHO STEPS Chronic Disease Risk Factor Surveillance: Data Book for Lebanon. In: Beirut AUo, editor. 2010.
16. Lavrakas PJ. *Encyclopedia of survey research methods*: Sage Publications; 2008.
17. World Health Organization (WHO). Noncommunicable diseases and their risk factors- STEPS Manual. <http://www.who.int/ncds/surveillance/steps/manual/en/>
18. National Cholesterol Education Program Adult Treatment Panel III. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-421.
19. IPAQ Research Committee. Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)—short and long forms. 2005.
20. Yu M, Xu C-X, Zhu H-H, Hu R-Y, Zhang J, Wang H, et al. Associations of cigarette smoking and alcohol consumption with metabolic syndrome in a male Chinese population: a cross-sectional study. *Journal of Epidemiology*. 2014;24:361-9.
21. Berlin I, Lin S, Lima JA, Bertoni AG. Smoking status and metabolic syndrome in the multi-ethnic study of atherosclerosis. A cross-sectional study. *Tobacco Induced Diseases*. 2012;10:9-16.
22. Bišanović S, Mehić B, Sivić S. Status of lipids and the frequency diseases of cardiovascular origin in smokers according to the length period of smoking and a number of cigarettes smoked daily. *Bosnian Journal of Basic Medical Sciences*. 2011;11:46-51.
23. Slagter SN, van Vliet-Ostapchouk JV, Vonk JM, Boezen HM, Dullaart RP, Koblod ACM, et al. Associations between smoking, components of metabolic syndrome and lipoprotein particle size. *BMC Medicine*. 2013;11:195-209.
24. Chen C-C, Li T-C, Chang P-C, Liu C-S, Lin W-Y, Wu M-T, et al. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. *Metabolism*. 2008;57:544-8.
25. Campbell SC, Moffatt RJ, Stamford BA. Smoking and smoking cessation—the relationship between cardiovascular disease and lipoprotein metabolism: a review. *Atherosclerosis*. 2008;201:225-35.
26. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: a meta-analysis. *Preventive Medicine*. 2003;37:283-90.
27. Slentz CA, Houmard JA, Johnson JL, Bateman LA, Tanner CJ, McCartney JS, et al. Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a randomized, controlled study of exercise intensity and amount. *Journal of Applied Physiology*. 2007;103:432-42.
28. Ellison RC, Zhang Y, Qureshi MM, Knox S, Arnett DK, Province MA. Lifestyle determinants of high-density lipoprotein cholesterol: The national heart, lung, and blood institute family heart study. *American Heart Journal*. 2004;147:529-35.
29. Park H, Kim K. Association of alcohol consumption with lipid profile in hypertensive men. *Alcohol and Alcoholism*. 2012;47:282-7.

- 1
- 2
- 3 30. Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary
- 4 heart disease: a meta-analysis. *Addiction*. 2000;95:1505-23.
- 5 31. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol
- 6 consumption with selected cardiovascular disease outcomes: a systematic review and
- 7 meta-analysis. *BMJ*. 2011;342:d671.
- 8 32. López EP, Rice C, Weddle DO, Rahill GJ. The relationship among cardiovascular risk
- 9 factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years
- 10 and older. *Journal of the American Dietetic Association*. 2008;108:248-56.
- 11
- 12
- 13
- 14
- 15
- 16
- 17
- 18
- 19
- 20
- 21
- 22
- 23
- 24
- 25
- 26
- 27
- 28
- 29
- 30
- 31
- 32
- 33
- 34
- 35
- 36
- 37
- 38
- 39
- 40
- 41
- 42
- 43
- 44
- 45
- 46
- 47
- 48
- 49
- 50
- 51
- 52
- 53
- 54
- 55
- 56
- 57
- 58
- 59
- 60

For peer review only

Table 1 Socio-demographic and health-related characteristics of the study population.

	n	%
Gender (% female)	181	49.9
Age (mean \pm SD, years)	39.2 \pm 15.2	
18-29	113	31.1
30-39	106	29.2
40-49	67	18.5
50-59	32	8.8
\geq 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	4.1
Current smoker	121	33.3
Number of cigarettes smoked/day		
0	227	62.5
1-19	43	11.8
20-39	63	17.4
\geq 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 (\geq once/week)	56	15.4
Total caloric intake (mean \pm SD, Kcal/day)	2656 \pm 1249	
Total Cholesterol (mean \pm SD, mg/dl)	210 \pm 45	
% Elevated total cholesterol	201	55.4
Triglycerides (mean \pm SD, mg/dl)	138 \pm 78	
% Elevated triglycerides	114	31.4
VLDL* (mean \pm SD, mg/dl)	27 \pm 15	
% Elevated VLDL	106	29.2
LDL-C* (mean \pm SD, mg/dl)	131 \pm 39	
% Elevated LDL-C	172	47.5
HDL-C* (mean \pm SD, mg/dl)	51 \pm 14	
% Reduced HDL-C	79	21.8

*Very Low Density Lipoprotein (VLDL); Low Density Lipoprotein cholesterol (LDL-C); High Density Lipoprotein cholesterol (HDL-C)

Table 2a Logistic regression analysis: associations of behavioral 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 (0.60-2.25)	0.77 (0.35-1.67)	30.2	1.26 (0.62-2.58)	1.00 (0.44-2.26)
	20-35	63.5	1.60 (0.90-2.85)	1.03 (0.51-2.07)	38.1	1.79 (0.99-3.23)	1.25 (0.64-2.46)
	≥40	63.3	1.59 (0.73-3.50)	1.08 (0.41-2.84)	63.3	5.03 (2.26-11.2)	3.82 (1.50-9.71)
	p-trend		0.070	0.916		<0.001	0.019
Physical activity	High	55.7	1.00		25.0	1.00	
	Moderate	56.2	1.02 (0.59-1.77)	0.85 (0.45- 1.63)	32.2	1.43 (0.77-2.64)	1.34 (0.67-2.66)
	Low	55.6	0.99 (0.59-1.69)	0.97 (0.52-1.78)	35.1	1.62 (0.90-2.92)	1.42 (0.75-2.68)
Alcohol intake	No	54.7	1.00	1.00	30.6	1.00	1.00
	Yes	58.9	1.19 (0.67-2.12)	1.37 (0.67-2.77)	35.7	1.26 (0.69-2.29)	1.05 (0.52-2.10)

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

Table 2b Logistic regression analysis: associations of behavioral 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		43.6	1.00		18.1	1.00	
	1-19	27.9	1.21 (0.58-2.52)	0.96 (0.41-2.20)	53.5	1.49 (0.77-2.86)	1.09 (0.51-2.34)	18.6	1.04 (0.45-2.40)	0.94 (0.35-2.54)
	20-39	35.5	1.72 (0.94-3.14)	1.21 (0.61-2.42)	54.8	1.57 (0.89-2.76)	1.00 (0.51-1.97)	28.6	1.81 (0.95-3.45)	1.67 (0.77-3.63)
	≥40	56.7	4.09 (1.87-8.95)	4.75 (1.10-6.72)	53.3	1.48 (0.69-3.17)	1.78 (0.37-2.37)	40.0	3.02 (1.35-6.76)	1.61 (0.62-4.24)
	p-trend		<0.001	0.039		0.083	0.688		0.003	0.745
Physical activity	High	26.1	1.00		48.9	1.00		27.3	1.00	
	Moderate	28.9	1.15 (0.62-2.13)	1.00 (0.50-1.99)	49.6	1.02 (0.59-1.78)	0.87 (0.46-1.64)	19.0	0.62 (0.32-1.20)	0.78 (0.36-1.68)
	Low	32.0	1.33 (0.74-2.39)	1.11 (0.59-2.09)	46.0	0.89 (0.53-1.51)	0.85 (0.47-1.53)	21.2	0.72 (0.40-1.32)	0.62 (0.31-1.25)
Alcohol Intake	No	28.7	1.00		46.6	1.00		21.2	1.00	
	Yes	32.7	1.21 (0.65-2.24)	0.93 (0.46-1.90)	52.7	1.28 (0.72-2.27)	1.36 (0.68-2.73)	25.0	1.24 (0.64-2.41)	0.75 (0.35-1.61)

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

BMJ Open

Prevalence and associations of behavioral risk factors with blood lipids profile in Lebanese adults: findings from the WHO STEPwise NCD Survey

Journal:	<i>BMJ Open</i>
Manuscript ID	bmjopen-2018-026148.R1
Article Type:	Research
Date Submitted by the Author:	07-Mar-2019
Complete List of Authors:	Mansour, Megali; American University of Beirut Faculty of Health Sciences, EPHD Tamim, Hani; American University of Beirut, Biostatistics Unit, Clinical Research Institute Nasreddine, Lara; American University of Beirut EL Khoury, Christelle; Tufts University School of Medicine Hwalla, Nahla; American University of Beirut Chaaya, Monique; American University of Beirut Farhat, Antoine ; Notre Dame University Sibai, Abla; American University of Beirut, EPHD
Primary Subject Heading:	Public health
Secondary Subject Heading:	Epidemiology, Nutrition and metabolism, Smoking and tobacco, Public health
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cigarette Smoking, Blood lipids

SCHOLARONE™
Manuscripts

1
2
3 **Prevalence and associations of behavioral risk factors with blood lipids profile in**
4
5 **Lebanese adults: findings from the WHO STEPwise NCD Survey**
6
7

8
9 Magali Mansour^{1†}, Hani Tamim^{2†}, Lara Nasreddine³, Christelle El Khoury⁴, Nahla Hwalla³,
10 Monique Chaaya¹, Antoine Farhat⁵, Abla M. Sibai^{1*}
11
12

13
14 **Author affiliations**

15
16 ¹ Department of Epidemiology and Population Health, Faculty of Health Sciences, American
17 University of Beirut, P.O.Box 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon

18
19 ² Department of Internal Medicine , American University of Beirut-Medical Center, P.O.Box
20 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon

21
22 † Equal Contribution

23
24 ³ Faculty of Agricultural and Food Sciences, American University of Beirut, P.O.Box 11-
25 0236, Riad El-Solh, Beirut 1107 2020, Lebanon

26
27 ⁴ School of Medicine Department of Public Health, Tufts University, 136 Harrison Avenue,
28 Boston, MA 02111, US

29
30 ⁵ Faculty of Nursing and Health Sciences, Notre Dame University, Lebanon
31
32
33
34

35 ***Corresponding author:**

36 **Abla M. Sibai**
37
38

39
40 Epidemiology and Population Health
41 Faculty of Health Sciences
42 American University of Beirut
43 PO Box 11-0236
44 Beirut-Lebanon
45 Tel: +961-1-350000 ext. 4647
46 Fax: +961-1-744470
47 Email: am00@aub.edu.lb
48
49
50
51
52
53
54
55
56
57
58
59
60

1
2
3 **Keywords:** Cardiovascular disease, smoking, alcohol consumption, physical inactivity,
4 dietary intake, blood lipids, Lebanon
5
6
7
8
9

10 **Word count (excluding title page, references, tables and figures):** 3047
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

Abstract

Objective: To examine associations of behavioral 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, behaviors 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 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 1.69-10.77; and 3.26; 95% CI 1.33-8.03) with a significant dose-response relationship (p-value for trend=0.010 and 0.030, respectively). Alcohol drinking and high saturated fat intake ($\geq 10\%$ energy intake) were associated with higher odds of LDL-C (OR=1.68; 95% CI: 1.01-2.82 and OR= 1.73; 95% CI: 1.02-2.93). Physical activity did not associate significantly with any blood lipid parameter.

Conclusion: The demonstrated positive association between smoking, alcohol drinking and high saturated fat intake with adverse lipoprotein levels lays further evidence for clinical

1
2
3 practitioners, public health professionals and dietitians in the development of preventive
4
5 strategies among subjects with high risk of CVDs in Lebanon.
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

Strengths and limitations

- A nationwide survey administered in Lebanon, following the 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 socio-demographic characteristics.
- Whilst 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 behavioral factors and dietary intakes with intermediary metabolic conditions along the causal pathway for CVD informs work towards 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 urbanization, 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 the World Health Organization (WHO) indicate that the proportional mortality from CVD alone is 45%, 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 (GDP) of close to 8,520 US Dollars per capita, Lebanon is characterized by a high urbanization rate (87%), a growing trend towards survival in later life, coupled with westernization and modernization 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 behaviors 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 nonsmokers⁵ 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

1
2
3 Regular physical activity with weight reduction has a large beneficial impact on the
4 lipoproteins profile of adult men and women,⁹ by increasing plasma volume, decreasing
5 blood thickness, and thus reducing LDL-C concentrations.¹⁰ Also, systematic reviews and
6 meta-analysis of intervention studies have shown that heavy alcohol drinking results in an
7 elevation in triglyceride levels, while moderate consumption increases circulating levels of
8 HDL-C.¹¹ Similarly, diet is recognized as a modifiable risk factor that can make a
9 substantial contribution to the risk of CVD.^{12 13} Energy intake, the types of fatty acids
10 consumed and the level of sugar ingestion may impact the lipid and cardiometabolic
11 profile.¹⁴ The 2017 Presidential Advisory from the American Heart Association indicated that
12 replacement of saturated fat with unsaturated fatty acids decreases LDL-C levels and CVD
13 risk, while replacing it with refined carbohydrates and sugar, yields no significant benefits to
14 cardiovascular health.¹⁴

15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60
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
behaviors, varied dietary habits and risk profile,¹⁵ and studies evaluating the association
between behaviors and intermediary variables along the causal pathway of CVD
including lipid levels remain scarce in the region. Compared with other neighboring
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 behavioral 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

1
2
3 from this study inform prevention strategies among subjects with high risk of CVD in
4
5 the country.
6
7
8
9

10 **Methods**

11 **Study design and participants**

12
13
14
15
16 The data presented in this study are derived from the WHO Nutrition and Non-
17
18 Communicable Diseases Risk Factor (NNCD-RF) cross-sectional household survey
19
20 conducted in Lebanon in 2009.¹⁸ Using multi-stage stratified cluster study design, the
21
22 sampling was based on the age-sex distribution of the Lebanese population as provided by the
23
24 Central Administration for Statistics. One adult was randomly selected from each household
25
26 using Kish methodology.¹⁹ Pregnant and lactating women and individuals with mental
27
28 disabilities were excluded. With a non-response rate of 10% at the individual level, this
29
30 yielded a sample of 2668 survey participants aged 18 years and above. Those free from
31
32 known history of hyperlipidemia and diabetes in the first phase of the study (n = 1,331) were
33
34 approached to undergo a biochemical assessment, of which 363 provided written consent and
35
36 gave fasting blood samples. Further details on the design and sample of the survey are
37
38 published elsewhere.⁴ The study protocol was approved by the Institutional Review Board of
39
40 the American University of Beirut (AUB), and informed consent was taken from all
41
42
43
44
45
46
47
48
49 participants.

50 **Data collection**

51
52 The data collection procedure followed the WHO STEPwise approach to Surveillance,²⁰ and
53
54 included the following three steps: Step 1 Questionnaire, whereby information about socio-
55
56 demographic characteristics, NCDs and NCD risk factors, including dietary intake, were
57
58 collected through face-to-face interviews; Step 2 in which anthropometric and blood pressure
59
60

1
2
3 measurements were taken using standardized techniques and calibrated equipment; and
4
5 finally, Step 3 in which biochemical analysis for assessment of the blood lipid profile was
6
7 performed on blood samples collected after an overnight fast of at least 8 hours. Serum was
8
9 centrifuged on site and shipped on dry ice to the AUB Laboratory.
10
11
12
13

14 **Measures of blood lipids**

15
16 Levels of blood lipids including TC, TG, very low density lipoprotein (VLDL), LDL-C and
17
18 HDL-C were analyzed using the Vitros 350 analyzer, an enzymatic spectrophotometric
19
20 technique. The inter-assay variation of measurements did not exceed 4%. Quality control was
21
22 performed within each run using standard performance verifier solutions provided by Ortho-
23
24 Clinical Diagnostics. Analyses were conducted in duplicates, and the average value was
25
26 utilized in the analysis. Based on the Adult Treatment Panel III guidelines,²¹ the cutoff points
27
28 used for the definition of risk levels of TC, TG, VLDL, LDL-C and HDL-C were ≥ 200 ,
29
30 ≥ 150 , ≥ 30 , ≥ 130 and ≤ 40 mg/dl, respectively.
31
32
33
34
35
36
37

38 **Behavioral risk factors and other measures**

39
40 Behavioral risk factors examined in this study included cigarette smoking, physical activity,
41
42 and alcohol consumption. Cigarette smoking status (never, past and current) and intensity
43
44 (number of cigarettes smoked/day) were assessed. Intensity was later categorized into three
45
46 levels according to number of cigarettes/day (1 to 19, 20 to 39 and ≥ 40). The short version of
47
48 the International Physical Activity Questionnaire was used to assess physical activity among
49
50 participants.²² Three categories of physical activity (low, moderate, and high) were assigned
51
52 based on METs-min/week (MET-min being the product of the resting metabolic rate for an
53
54 activity and the number of minutes taken to perform it). Alcohol-related behavior was
55
56 assessed as a dichotomous variable (ever vs never). Dietary assessment was performed using
57
58
59
60

1
2
3 a 61-item culture-specific food frequency questionnaire that measured food intake over the
4
5 past year.^{4, 15} Intakes of energy and macronutrients were estimated using the food
6
7 composition database of the Nutritionist IV software, and the food composition table for local
8
9 and traditional Middle-Eastern foods.^{23,24} Intakes of carbohydrates, fat and protein were
10
11 compared to cut-offs within the Acceptable Macronutrient Distribution Range,²⁵ and intakes
12
13 of saturated fat and sugar were compared to the recommendations of the WHO.^{26, 27}
14
15
16

17
18
19 Covariates of interest included total daily caloric intake and body mass index (BMI),
20
21 measured as the ratio of weight (kilograms) to the square of height (meters). In addition,
22
23 gender, age (18-29, 30-39, 40-49, 50-59, and ≥ 60), marital status (single, married,
24
25 divorced/widowed), education level (complementary or less, secondary/technical, university
26
27 and above), and occupational status (student/volunteer, working, does not
28
29 work/housewife/retired) were considered as potential covariates.
30
31
32

33 34 35 **Patient and Public Involvement**

36
37 This study is based on secondary data analyses. The original data collection tool was adapted
38
39 from the WHO STEPwise approach to NCD risk factor surveillance,²⁰ that did not directly
40
41 involve patients or the public in outcome development or conduct of the study. However, we
42
43 have been engaging with stakeholders to disseminate the findings on NCD risk factors,
44
45 including tobacco consumption and dietary intakes, and on associations with various health-
46
47 related outcomes to the public at large.
48
49
50

51 52 53 **Statistical analysis**

54
55 Means, standard deviations (SD) and frequencies were used to describe the various socio-
56
57 demographic, behavioral, nutritional and clinical characteristics of the participants. The
58
59
60

1
2
3 associations between each of the risk factors and levels of the different blood lipids were
4
5 examined using multiple logistic regression analysis. Unadjusted and adjusted odds ratios
6
7 (ORs) and their 95% confidence intervals (CIs) controlling for age, gender, education, marital
8
9 status, caloric intake and BMI were estimated. Test for trend with increasing number of daily
10
11 amount of cigarettes smoked was also conducted, and a two-sided p-value < 0.05 was
12
13 considered significant. The Statistical Package for the Social Sciences 22.0.1 (SPSS Inc.,
14
15 Chicago, IL, USA) was used for all computations.
16
17
18
19
20
21
22
23

24 **Results**

25
26 The socio-demographic and health-related characteristics of the study sample are summarized
27
28 in Table 1. The sample was equally divided by gender (49.9% females and 50.1% males),
29
30 with a mean age of 39.2 ± 15.2 years (range 18 to 92 years). The majority were married
31
32 (60.6%), and close to half of the participants were employed (52.3%) at the time of the study,
33
34 with a high percentage having less than complementary education (39.7%). Ever smokers
35
36 constituted 37.5% of the sample, 41.6% were classified as being engaged in low-intensity
37
38 physical activity, and 39.7% reported ever alcohol drinkers. Average daily energy intake was
39
40 estimated at 2656 ± 1249 Kcal/day, with 36.5% of caloric intake from fat, 11.4% from
41
42 saturated fat, 49.1% from carbohydrates, 5.7% from sugar, and 15.2% from protein.
43
44 Abnormal levels of TC, TG, VLDL, LDL-C, and HDL-C were observed for 55.4%, 31.4%,
45
46 29.2%, 47.5% and 21.8% of the participants, respectively.
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Insert table 1 around here

1
2
3 Tables 2a and 2b show the unadjusted and adjusted ORs for the association between
4 behavioral risk factors and dietary variables with blood lipids levels. Associations with
5 cigarette smoking were positive for most outcomes but significant for those consuming more
6 than 40 cigarettes/day, compared to non-smokers, in the case of TG (unadjusted OR = 5.03)
7 and VLDL (OR = 4.09) and HDL-C OR = 3.02). Adjusting for potential confounders, the
8 associations maintained statistical significance for TG and VLDL, with an adjusted OR of
9 4.27 (95% CI 1.69- 10.77) and 3.26 (95% CI 1.33-8.03), respectively. Results showed a dose-
10 response relationship with increasing number of cigarettes consumed for (p-value for trend =
11 0.010 and 0.030, respectively). In addition, a statistically significant association was observed
12 between ever alcohol drinking and LDL-C (OR=1.53). This association retained statistical
13 significance even after adjustment for potential confounders with an OR of 1.68 (95% CI
14 1.01-2.82). Out of all the dietary variables examined, only saturated fat was associated with
15 blood lipids, namely TC and LDL-C, with an adjusted OR of 1.73 for both lipid abnormalities
16 (95% CI 1.02-2.94 and 1.02-2.93, respectively). Physical activity was not associated with any
17 of the blood lipid parameters.
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49

Insert tables 2a and 2b around here

50 Discussion

51 Our results showed that heavy cigarette smoking is associated with increased levels of TG
52 and VLDL, with findings showing significant dose-response relationships with increasing
53 number of cigarettes smoked per day. The study also showed that alcohol drinking and high
54 saturated fat intake are significantly associated with higher levels of LDL-C. However, there
55 were no consistent associations between physical activity and fasting blood lipids profile. To
56
57
58
59
60

1
2
3 our knowledge, this is the first study in Lebanon to explore these associations, based on
4 objectively measured lipid parameters and using standardized laboratory techniques and
5
6
7
8 tools, while taking into consideration the effect of potential confounders.
9

10
11 There are strong indicators in the literature that the deleterious effect of cigarette smoking on
12 heart disease and atherosclerosis is partially explained by the effect of smoking on the
13 concentration of blood lipids and lipoproteins. Our study confirms the results of earlier cross-
14 sectional studies showing that the association between smoking and lipoproteins are observed
15 in the levels of TG, VLDL and HDL-C,^{6, 28, 29} with the impact on lipid levels increasing with
16 increase in the number of cigarettes smoked/day in a dose dependent relationship.^{30, 31} Our
17 observation of the largest effect of cigarette smoking on lipid parameters being seen in those
18 who smoked more than two packs a day is also consistent with Chen et al. study comprising
19 of 1,164 men in Taiwan.³² One popular mechanism by which smoking affects lipoproteins is
20 that cigarette particulate matter alters catecholamine release—and thus free fatty acid release,
21 which in turn contributes to the accumulation of the LDL-C concentrations and to lower
22 levels of HDL-C in the blood.³³ The association between cigarette smoking and the increase
23 in TG can also be explained by the decrease in the activity of the lipoprotein lipase among
24 smokers thus disrupting lipid and lipoprotein metabolism. Furthermore, smoking cessation
25 was found to improve lipid and lipoprotein levels in observational studies and randomized
26 clinical trials.^{7, 34} Taken together, the totality of evidence from these studies and our data-
27 including consistency upon replication across various studies, the dose-response relationship,
28 the magnitude and significance of association, biological plausibility as well as effects of
29 smoking cessation on lipoprotein levels, supports a strong relationship between smoking and
30 lipid profiles.
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

1
2
3 Our study showed that alcohol drinking was associated with higher LDL-C levels in
4
5 Lebanese adults, but was not associated with other lipid parameters. Available evidence on
6
7 the impact of alcohol on LDL-C is conflicting with recent studies suggesting that the
8
9 association of alcohol and LDL-C levels may be population-specific.³⁵ For instance, studies
10
11 conducted amongst Danish adults reported an inverse association between alcohol intake and
12
13 LDL-C,³⁶ while studies conducted in Spanish and Italian populations found that higher
14
15 alcohol consumption was associated with increased LDL-C.^{37,38} According to a review by
16
17 Brinton (2012), these inconsistent findings on the association between alcohol intake and
18
19 LDL-C, may be explained by allele-specific genetic effects, such as the Apo E4 and Apo A5
20
21 genes.³⁹ In our study, alcohol drinking was not associated with HDL-C levels. Findings on
22
23 the relationship between alcohol and HDL-C have in fact been less consistent in the
24
25 literature, with some but not all showing an effect on higher HDL-C levels.^{40,41} Indeed, the
26
27 effect of alcohol on CVD including coronary artery disease, stroke and myocardial infarction
28
29 appears to be biphasic showing a J or U-shaped relationship based on the amount of alcohol
30
31 consumed,^{41,42} with lower CVD mortality risks by light to moderate alcohol consumption
32
33 and increased risk by heavy alcohol intake.⁴³ The discrepancy between our results with those
34
35 in the literature may be due to differences in race/ethnicity and genetic variations,⁴⁴ or
36
37 alternatively, owing to measurement error in our assessment of alcohol consumption, based
38
39 on self-reports of a dichotomous variable. Lipid profiles are influenced by the type of alcohol
40
41 consumed and by drinking frequency and patterns.

42
43 Dietary intakes are amongst the modifiable risk factors that may modulate plasma lipids and
44
45 the risk of CVD. In our study, saturated fat was the only dietary factor that was significantly
46
47 associated with lipid parameters, and specifically with TC and LDL-C. Despite the
48
49 increasing controversy around the relationship between saturated fat and blood cholesterol
50
51 levels, an increasing body of evidence highlights the strong atherogenicity of saturated fatty
52
53
54
55
56
57
58
59
60

1
2
3 acids through their impact on LDL-C. ¹⁴ A systematic review and meta-regression analysis
4 published in 2016 showed that a decrease in saturated fats of 1% of daily energy intake
5 coupled with an increase of 1% in polyunsaturated fat, lowered LDL cholesterol by 2.1
6 mg/dL.⁴⁵ In 2017, a Presidential Advisory from the American Heart Association (AHA)
7 concluded that available evidence strengthens the long-standing AHA recommendations to
8 decrease saturated fat intake and replace it with unsaturated fats. The AHA Advisory
9 highlighted that the shift from saturated to unsaturated fats should occur in the context of an
10 overall healthful dietary pattern such as the DASH or Mediterranean patterns. ¹⁴
11
12
13
14
15
16
17
18
19
20
21
22

23 In our study, physical activity did not appear to be associated with lipid parameters. The
24 literature suggests that physical activity increases the level of HDL-C and has beneficial
25 effects on lipoprotein particle size and number. ⁴⁶ Also, moderate intensity exercise is known
26 to have a more favorable effect on blood lipids since it allows the use of lipids as a fuel
27 source which implies an increase in the uptake and oxidation of the lipids in the skeletal
28 muscle. ⁴⁶
29
30
31
32
33
34
35
36
37

38 Some other limitations should be taken into consideration when interpreting the results of our
39 study. Because the study used a cross-sectional design, findings only imply associations and
40 causal relationships cannot be established. The proportion of participants who gave fasting
41 blood samples (of at least 8 hours) was relatively small (27.3%); however, responders were
42 comparable to non-responders on a number of socio-demographic characteristics except for
43 marital status (61% of responders vs. 50% of non-responders were married). Also, we had
44 earlier documented comparable dietary data between respondents and non-respondents based
45 on factor loading matrices on patterns of food groups intake. ⁴ As mentioned earlier,
46 measures of exposure were self-reported which can introduce some misclassification error.
47
48
49
50
51
52
53
54
55
56
57
58
59
60 Whilst this may have been problematic in the case of alcohol consumption that lacked

1
2
3 detailed data on type of alcohol consumed, measurement of cigarette smoking and physical
4 activity were more detailed and reliable. Information on cigarette smoking in our study
5 included the amount of cigarettes smoked and the standardized IPAQ was used to assess
6 physical activity.
7
8
9
10
11
12

13 To conclude, our results suggest that smoking, alcohol drinking and high saturated fat are
14 associated with adverse levels of lipoprotein, among Lebanese men and women. The role of
15 lipoproteins in atherogenesis has been clearly defined, and controlling for blood lipid levels
16 decreases the risk of heart and many other chronic diseases. Our findings lay further evidence
17 for clinical practitioners, public health professionals and dieticians regarding the potential
18 benefits of lifestyle and dietary modifications in their pursuit to curb the burden of
19 hyperlipidemia and CVD at the individual and population level. The overall high rate of
20 smoking behavior in Lebanon among both men and women, coupled with the shift in dietary
21 patterns towards high fat energy dense foods,^{4, 15} are likely to adversely impact on the
22 healthcare bill in the country. Further studies with larger sample size that examine the
23 association of combination patterns of poor lifestyle factors on lipid profile among Lebanese
24 adults are warranted.
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Contributors

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

Funding statement

The original study conduct was funded by the World Health Organization (WHO) - Lebanon. However, no funds were available for this secondary analysis.

Data sharing statement

The dataset was available for all authors of the study and will be available for other non-commercial researchers upon request.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

REFERENCES

1. Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G et al. Global, Regional, and National Burden of Cardiovascular Diseases for 10 Causes, 1990 to 2015. *Journal of the American College of Cardiology*. 2017;70:1-25.
2. Sibai AM, Nasreddine L, Mokdad AH, Adra N, Tabet M, Hwalla N. Nutrition transition and cardiovascular disease risk factors in Middle East and North Africa countries: reviewing the evidence. *Annals of Nutrition and Metabolism*. 2010;57:193-203.
3. World Health Organization. Non-communicable Diseases Country Profiles 2014.
4. Naja F, Nasreddine L, Itani L, Adra N, Sibai A, Hwalla N. Association between dietary patterns and the risk of metabolic syndrome among Lebanese adults. *European Journal of Nutrition*. 2013;52:97-105.
5. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Atlanta (GA): The health consequences of smoking—50 years of progress: a report of the Surgeon General: Centers for Disease Control and Prevention (US), National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
6. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. *Journal of Clinical Lipidology*. 2009;3:372-8.
7. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. *American Heart Journal*. 2011;161:145-51.
8. Abramson JL, Vaccarino V. Relationship between physical activity and inflammation among apparently healthy middle-aged and older US adults. *Archives of Internal Medicine*. 2002;162:1286-92.
9. Barengo NC, Kastarinen M, Lakka T, Nissinen A, Tuomilehto J. Different forms of physical activity and cardiovascular risk factors among 24–64-year-old men and women in Finland. *European Journal of Cardiovascular Prevention & Rehabilitation*. 2006;13:51-9.
10. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Medicine and Science in Sports and Exercise*. 2001;33:S438-45.
11. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. *BMJ*. 2011;342:d636.
12. Wang Q, Afshin A, Yakoob MY, Singh GM, Rehm CD, Khatibzadeh S, on behalf of the Global Burden of Diseases. Nutrition and Chronic Diseases Expert Group (NutriCoDE). Impact of Nonoptimal Intakes of Saturated, Polyunsaturated, and Trans Fat on Global Burdens of Coronary Heart Disease. *J Am Heart Assoc*. 2016;5:e002891

13. Stone NJ, Robinson JG, Lichtenstein AH, Noel Bairey Merz C, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*. 2014;63:2889-934.
14. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, et al. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation*. 2017;13:e1-e23.
15. Naja F, Nasreddine L, Itani L, Chamieh MC, Adra N, Sibai AM, et al. Dietary patterns and their association with obesity and sociodemographic factors in a national sample of Lebanese adults. *Public Health Nutrition*. 2011;14:1570-8.
16. Sibai AM, Obeid O, Batal M, Adra N, El Khoury D, Hwalla N. Prevalence and correlates of metabolic syndrome in an adult Lebanese population. *CVD Prevention & Control*. 2008;3:83-90.
17. Sibai AM, Iskandarani M, Darzi A, Nakkash R, Saleh S, Fares S, et al. Cigarette smoking in a Middle Eastern country and its association with hospitalisation use: a nationwide cross-sectional study. *BMJ Open*. 2016;6:e009881.
18. Sibai AM, Hwalla N. WHO STEPS Chronic Disease Risk Factor Surveillance: Data Book for Lebanon. 2010.
https://www.who.int/ncds/surveillance/steps/2008_STEPS_Lebanon.pdf
19. Lavrakas PJ. *Encyclopedia of survey research methods*: Sage Publications; 2008.
20. World Health Organization (WHO). *Noncommunicable diseases and their risk factors- STEPS Manual*. <http://www.who.int/ncds/surveillance/steps/manual/en/>
21. National Cholesterol Education Program Adult Treatment Panel III. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-421.
22. IPAQ Research Committee. *Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)—short and long forms*. 2005.
23. Nutritionist IV. *N-squared computing*: Silverton; 1998. 31.
24. Pellet P, Shadarevian S. Food composition. Tables for use in the Middle East. In: *Food composition tables for use in the Middle East*. 2nd ed. 1970.
25. Institute of Medicine. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: The National Academies Press. 2005; <https://doi.org/10.17226/10490>.
26. Food and Agriculture Organization/World Health Organization. *Interim Summary of Conclusions and Dietary Recommendations on Total Fat & Fatty Acids*. 2008; https://www.who.int/nutrition/topics/FFA_summary_rec_conclusion.pdf?ua=1.
27. World Health Organization. *Guideline: Sugars intake for adults and children*. 2015; http://apps.who.int/iris/bitstream/handle/10665/149782/9789241549028_eng.pdf;jsessionid=2A114891D4D08F644AA154370830829B?sequence=1.
28. Yu M, Xu C-X, Zhu H-H, Hu R-Y, Zhang J, Wang H, et al. Associations of cigarette smoking and alcohol consumption with metabolic syndrome in a male Chinese population: a cross-sectional study. *Journal of Epidemiology*. 2014;24:361-9.

- 1
 - 2
 - 3
 - 4
 - 5
 - 6
 - 7
 - 8
 - 9
 - 10
 - 11
 - 12
 - 13
 - 14
 - 15
 - 16
 - 17
 - 18
 - 19
 - 20
 - 21
 - 22
 - 23
 - 24
 - 25
 - 26
 - 27
 - 28
 - 29
 - 30
 - 31
 - 32
 - 33
 - 34
 - 35
 - 36
 - 37
 - 38
 - 39
 - 40
 - 41
 - 42
 - 43
 - 44
 - 45
 - 46
 - 47
 - 48
 - 49
 - 50
 - 51
 - 52
 - 53
 - 54
 - 55
 - 56
 - 57
 - 58
 - 59
 - 60
29. Berlin I, Lin S, Lima JA, Bertoni AG. Smoking status and metabolic syndrome in the multi-ethnic study of atherosclerosis. A cross-sectional study. *Tobacco Induced Diseases*. 2012;10:9-16.
30. Bišanović S, Mehić B, Sivić S. Status of lipids and the frequency diseases of cardiovascular origin in smokers according to the length period of smoking and number of cigarettes smoked daily. *Bosnian Journal of Basic Medical Sciences*. 2011;11:46-51.
31. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Koblold ACM, et al. Associations between smoking, components of metabolic syndrome and lipoprotein particle size. *BMC Medicine*. 2013;11:195-209.
32. Chen C-C, Li T-C, Chang P-C, Liu C-S, Lin W-Y, Wu M-T, et al. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. *Metabolism*. 2008;57:544-8.
33. Campbell SC, Moffatt RJ, Stamford BA. Smoking and smoking cessation—the relationship between cardiovascular disease and lipoprotein metabolism: a review. *Atherosclerosis*. 2008;201:225-35.
34. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: a meta-analysis. *Preventive Medicine*. 2003;37:283-90.
35. Matsumoto C, Miedema MD, Ofman P, Gaziano JM, Sesso HD. An expanding knowledge of the mechanisms and effects of alcohol consumption on cardiovascular disease. *Journal of Cardiopulmonary Rehabilitation and Prevention*. 2014;34:159-71.
36. Tolstrup JS, Gronbaek M, Nordestgaard BRG. Alcohol intake, myocardial infarction, biochemical risk factors, and alcohol dehydrogenase genotypes. *Circulation: Cardiovascular Genetics*. 2009;2:507-14.
37. Corella D, Portolés O, Arriola L, Chirlaque MD, Barricarte A, Francés F, et al. Saturated fat intake and alcohol consumption modulate the association between the APOE polymorphism and risk of future coronary heart disease: a nested case-control study in the Spanish EPIC cohort. *The Journal of Nutritional Biochemistry*. 2011;22:487-94.
38. Perissinotto E, Buja A, Maggi S, Enzi G, Manzato E, Scafato E, et al. Alcohol consumption and cardiovascular risk factors in older lifelong wine drinkers: the Italian Longitudinal Study on Aging. *Nutrition, Metabolism and Cardiovascular Diseases*. 2010;20:647-55.
39. Brinton EA. Effects of ethanol intake on lipoproteins. *Current Atherosclerosis Reports*. 2012;14:108-14.
40. Ellison RC, Zhang Y, Qureshi MM, Knox S, Arnett DK, Province MA. Lifestyle determinants of high-density lipoprotein cholesterol: The national heart, lung, and blood institute family heart study. *American Heart Journal*. 2004;147:529-35.
41. Park H, Kim K. Association of alcohol consumption with lipid profile in hypertensive men. *Alcohol and Alcoholism*. 2012;47:282-7.
42. Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction*. 2000;95:1505-23.

- 1
2
3 43. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol
4 consumption with selected cardiovascular disease outcomes: a systematic review and
5 meta-analysis. *BMJ*. 2011;342:d671.
6
7 44. López EP, Rice C, Weddle DO, Rahill GJ. The relationship among cardiovascular risk
8 factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years
9 and older. *Journal of the American Dietetic Association*. 2008;108:248-56.
10
11 45. Mensink RP. Effects of Saturated Fatty Acids on Serum Lipids and Lipoproteins: A
12 Systematic Review and Regression Analysis. Geneva, Switzerland: World Health
13 Organization; 2016.
14
15 46. Slentz CA, Houmard JA, Johnson JL, Bateman LA, Tanner CJ, McCartney JS, et al.
16 Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a
17 randomized, controlled study of exercise intensity and amount. *Journal of Applied
18 Physiology*. 2007;103:432-42.
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

Table 1 Distribution of socio-demographic characteristics, behavioral factors and lipid profile of the study population.

	n	%
Gender (% female)	181	49.9
Age (mean \pm SD, years)	39.2 \pm 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 \pm SD, Kcal/day)	2656 \pm 1249	
Fat intake (% of total energy)^a	36.5 \pm 6.89	
≥ 30 ^b	275	81.6
Saturated fat intake (% of total energy)^a	11.4 \pm 2.92	
≥ 10 ^c	224	66.5
Carbohydrates intake (% of total energy)^a	49.1 \pm 7.03	
≥ 55 ^b	71	21.1
Sugar (% of total energy)^a	5.7 \pm 4.64	
≥ 10 ^d	56	16.6
Proteins intake (% of total energy)^a	152 \pm 2.91	
< 15 ^b	143	42.4
Total Cholesterol (mean \pm SD, mg/dl)	210 \pm 45	
% Elevated total cholesterol (≥ 200 mg/dl) ^e	201	55.4
Triglycerides (mean \pm SD, mg/dl)	138 \pm 78	
% Elevated triglycerides (≥ 150 mg/dl) ^e	114	31.4

VLDL (mean ± SD, mg/dl)	27 ± 15	
% Elevated VLDL (≥30 mg/dl) ^e	106	29.2
LDL-C (mean ± SD, mg/dl)	131 ± 39	
% Elevated LDL-C (≥130 mg/dl) ^e	172	47.5
HDL-C (mean ± SD, mg/dl)	51 ± 14	
% Reduced HDL-C (≤40 mg/dl) ^e	79	21.8

^a Dietary variables are based on a sample of 337 subjects owing to missing data

^b Macronutrient cutoffs are within the Acceptable Macronutrient Distribution Range (AMDR)²⁵

^c Saturated fat cutoff based on the WHO recommendations²⁶

^d Sugar intake cutoff based on the WHO recommendations²⁷

^e Lipid cutoff values based on the Adult Treatment Panel III guidelines²¹; Very Low Density Lipoprotein (VLDL); Low Density Lipoprotein cholesterol (LDL-C); High Density Lipoprotein cholesterol (HDL-C)

For peer review only

Table 2a Logistic regression analysis: associations of behavioral 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)
	0	52.0	1.00	1.00	25.6	1.00	1.00
Number of Cigarettes	1-19	55.8	1.17 (0.60-2.25)	0.85 (0.40-1.77)	30.2	1.26 (0.62-2.58)	1.06 (0.48-2.33)
	20-35	63.5	1.60 (0.90-2.85)	1.01 (0.51-2.00)	38.1	1.79 (0.99-3.23)	1.30 (0.67-2.53)
	≥40	63.3	1.59 (0.73-3.50)	1.24 (0.48-3.18)	63.3	5.03 (2.26-11.2)	4.27 (1.69-10.77)
	p-trend		0.070	0.783		<0.001	0.010
Physical activity	High	55.7	1.00	1	25.0	1.00	1
	Moderate	56.2	1.02 (0.59-1.77)	0.89 (0.48-1.67)	32.2	1.43 (0.77-2.64)	1.64 (0.83-3.25)
	Low	55.6	0.99 (0.59-1.69)	1.11 (0.31-2.00)	35.1	1.62 (0.90-2.92)	1.75 (0.93-3.29)
Alcohol intake	No	53	1	1	31.5	1	1
	Yes	59	1.28 (0.84-1.96)	1.52 (0.90-2.55)	31.3	0.99 (0.63-1.56)	0.98 (0.57-1.69)
Total fat intake (% energy)	<30	54.8	1	1.00	30.6	1	1
	≥30	54.9	1.03 (0.57-1.74)	1.15 (0.62-2.13)	31.3	1.03 (0.57-1.87)	1.22 (0.34-2.35)
Saturated Fat (% energy)	<10	52.2	1	1	31	1	1
	≥10	56.3	1.17 (0.75-1.85)	1.73 (1.02-2.94)	31.3	1.01 (0.62-1.65)	1.01 (0.59-1.74)
Carbohydrates (% energy)	<55	53.8	1	1	30.8	1	1
	≥55	59.2	1.25 (0.73-2.12)	1.07 (0.59-1.93)	32.4	1.07 (0.61-1.88)	0.96 (0.52-1.76)
Sugar intake (% energy)	<10	54.8	1	1	32	1	1
	≥10	55.4	1.02 (0.57-1.82)	1.12 (0.59-2.16)	26.8	0.78 (0.41-1.48)	0.86 (0.43-1.73)
Protein intake (% energy)	<15	56.6	1	1	36.4	1	1
	≥15	53.6	0.88 (0.57-1.37)	0.83 (0.50-1.37)	27.3	0.66 (0.41-1.05)	0.79 (0.47-1.32)

*Controlling for age, gender, education, marital status, caloric intake and BMI

Table 2b Logistic regression analysis: associations of behavioral 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 (0.58-2.52)	1.01 (0.45-2.25)	53.5	1.49 (0.77-2.86)	1.12 (0.54-2.33)	18.6	1.04 (0.45-2.40)	1.01 (0.39-2.63)
	20-39	35.5	1.72 (0.94-3.14)	1.27 (0.64-2.51)	54.8	1.57 (0.89-2.76)	0.97 (0.50-1.89)	28.6	1.81 (0.95-3.45)	1.61 (0.75-3.45)
	≥40	56.7	4.09 (1.87-8.95)	3.26 (1.33-8.03)	53.3	1.48 (0.69-3.17)	0.96 (0.39-2.40)	40.0	3.02 (1.35-6.76)	1.98 (0.77-5.08)
	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 (0.62-2.13)	1.27 (0.54-2.52)	49.6	1.02 (0.59-1.78)	0.89 (0.48-1.65)	19.0	0.62 (0.32-1.20)	0.93 (0.43-2.01)
	Low	32.0	1.33 (0.74-2.39)	1.44 (0.76-2.71)	46.0	0.89 (0.53-1.51)	0.93 (0.52-1.65)	21.2	0.72 (0.40-1.32)	0.69 (0.34-1.39)
Alcohol intake	No	28.3	1	1	43.4	1	1	20.5	1	1
	Yes	30.8	1.12 (0.71-1.79)	1.12 (0.65-1.93)	53.8	1.53 (1.00-2.33)	1.68 (1.01-2.82)	23.6	1.19 (0.72-1.98)	0.98 (0.54-1.78)
Total fat intake (% energy)	<30	29.0	1	1	45.2	1	1	25.8	1	1
	≥ 30	28.8	0.99 (0.54-1.82)	1.15 (0.59-2.22)	46.7	1.06 (0.61-1.85)	1.26 (0.69-2.33)	21.8	0.80 (0.42-1.52)	0.81 (0.39-1.65)
Saturated Fat (% energy)	<10	29.2	1	1	44.2	1	1	20.4	1	1
	≥ 10 ^s	28.7	0.98 (0.59-1.61)	0.96 (0.56-1.66)	47.5	1.15 (0.72-1.80)	1.73 (1.02-2.93)	23.7	1.21 (0.70-2.16)	1.16 (0.62-2.17)
Carbohydrates (% energy)	<55	28.7	1	1	45.7	1	1	22.2	1	1
	≥55	29.6	1.04 (0.59-1.86)	0.96 (0.52-1.79)	49.3	1.16 (0.68-1.95)	0.93 (0.52-1.67)	23.9	1.10 (0.60-2.05)	1.18 (0.59-2.36)
Sugar intake (% energy)	<10	29.6	1	1	47.1	1	1	23.8	1	1
	≥10	25.0	0.79 (0.41-1.53)	0.89 (0.44-1.82)	42.9	0.84 (0.47-1.50)	0.89 (0.47-1.71)	16.1	0.61 (0.28-1.31)	0.62 (0.26-1.45)
Protein intake (% energy)	<15	32.9	1	1	48.3	1	1	21	1	1
	≥15	25.9	0.71(0.44-1.15)	0.85 (0.50-1.44)	45.1	0.88 (0.57-1.36)	0.75 (0.46-1.24)	23.7	1.17 (0.59-1.97)	1.49 (0.82-2.72)

*Controlling for age, gender, education, marital status, caloric intake and BMI

BMJ Open

Prevalence and associations of behavioral risk factors with blood lipids profile in Lebanese adults: findings from the WHO STEPwise NCD Survey

Journal:	<i>BMJ Open</i>
Manuscript ID	bmjopen-2018-026148.R2
Article Type:	Research
Date Submitted by the Author:	13-May-2019
Complete List of Authors:	Mansour, Megali; American University of Beirut Faculty of Health Sciences, EPHD Tamim, Hani; American University of Beirut, Biostatistics Unit, Clinical Research Institute Nasreddine, Lara; American University of Beirut EL Khoury, Christelle; Tufts University School of Medicine Hwalla, Nahla; American University of Beirut Chaaya, Monique; American University of Beirut Farhat, Antoine ; Notre Dame University Sibai, Abla; American University of Beirut, EPHD
Primary Subject Heading:	Public health
Secondary Subject Heading:	Epidemiology, Nutrition and metabolism, Smoking and tobacco, Public health
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cigarette Smoking, Blood lipids

SCHOLARONE™
Manuscripts

1
2
3 **Prevalence and associations of behavioral risk factors with blood lipids profile in**
4
5 **Lebanese adults: findings from the WHO STEPwise NCD Survey**
6
7
8

9 Magali Mansour^{1†}, Hani Tamim^{2†}, Lara Nasreddine³, Christelle El Khoury⁴, Nahla Hwalla³,
10 Monique Chaaya¹, Antoine Farhat⁵, Abla M. Sibai^{1*}
11
12

13
14 **Author affiliations**

15 ¹ Department of Epidemiology and Population Health, Faculty of Health Sciences, American
16 University of Beirut, P.O.Box 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon

17 ² Department of Internal Medicine , American University of Beirut-Medical Center, P.O.Box
18 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon
19

20 [†] Equal Contribution
21

22 ³ Faculty of Agricultural and Food Sciences, American University of Beirut, P.O.Box 11-
23 0236, Riad El-Solh, Beirut 1107 2020, Lebanon
24

25 ⁴ School of Medicine Department of Public Health, Tufts University, 136 Harrison Avenue,
26 Boston, MA 02111, US
27

28 ⁵ Faculty of Nursing and Health Sciences, Notre Dame University, Lebanon
29
30
31
32
33
34

35 ***Corresponding author:**

36 **Abla M. Sibai**
37
38

39 Epidemiology and Population Health
40 Faculty of Health Sciences
41 American University of Beirut
42 PO Box 11-0236
43 Beirut-Lebanon
44 Tel: +961-1-350000 ext. 4647
45 Fax: +961-1-744470
46 Email: am00@aub.edu.lb
47
48
49
50
51
52
53
54
55
56
57
58
59
60

1
2
3 **Keywords:** Cardiovascular disease, smoking, alcohol consumption, physical inactivity,
4
5 dietary intake, blood lipids, Lebanon
6
7
8
9

10 **Word count (excluding title page, references, tables and figures):** 3141
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

Abstract

Objective: To examine associations of behavioral 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, behaviors 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 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 1.69-10.77; and 3.26; 95% CI 1.33-8.03, respectively) with a significant dose-response relationship (p-value for trend=0.010 and 0.030, respectively). Alcohol drinking and high saturated fat intake ($\geq 10\%$ energy intake) were associated with higher odds of LDL-C (OR=1.68; 95% CI: 1.01-2.82 and OR= 1.73; 95% CI: 1.02-2.93). 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

1
2
3 practitioners, public health professionals and dietitians in the development of preventive
4
5 strategies among subjects with high risk of CVDs in Lebanon.
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

Strengths and limitations

- A nationwide survey administered in Lebanon, following the 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 socio-demographic characteristics.
- Whilst 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 behavioral factors and dietary intakes with intermediary metabolic conditions along the causal pathway for CVD informs work towards 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 urbanization, 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 the World Health Organization (WHO) indicate that the proportional mortality from CVD alone is 45%, 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 (GDP) of close to 8,520 US Dollars per capita, Lebanon is characterized by a high urbanization rate (87%), a growing trend towards survival in later life, coupled with westernization and modernization 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 behaviors 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 nonsmokers⁵ 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

1
2
3 Regular physical activity with weight reduction has a large beneficial impact on the
4 lipoproteins profile of adult men and women,⁹ by increasing plasma volume, decreasing
5 blood thickness, and thus reducing LDL-C concentrations.¹⁰ Also, systematic reviews and
6 meta-analysis of intervention studies have shown that heavy alcohol drinking results in an
7 elevation in triglyceride levels, while moderate consumption increases circulating levels of
8 HDL-C.¹¹ Similarly, diet is recognized as a modifiable risk factor that can make a
9 substantial contribution to the risk of CVD.^{12 13} Energy intake, the types of fatty acids
10 consumed and the level of sugar ingestion may impact the lipid and cardiometabolic
11 profile.¹⁴ The 2017 Presidential Advisory from the American Heart Association indicated that
12 replacement of saturated fat with unsaturated fatty acids decreases LDL-C levels and CVD
13 risk, while replacing it with refined carbohydrates and sugar, yields no significant benefits to
14 cardiovascular health.¹⁴

15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60
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
behaviors, varied dietary habits and risk profile,¹⁵ and studies evaluating the association
between behaviors and intermediary variables along the causal pathway of CVD
including lipid levels remain scarce in the region. Compared with other neighboring
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 behavioral 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

1
2
3 from this study inform prevention strategies among subjects with high risk of CVD in
4
5 the country.
6
7
8
9

10 **Methods**

11 **Study design and participants**

12
13
14
15
16 The data presented in this study are derived from the WHO Nutrition and Non-
17
18 Communicable Diseases Risk Factor (NNCD-RF) cross-sectional household survey
19
20 conducted in Lebanon in 2009.¹⁸ Using multi-stage stratified cluster study design, the
21
22 sampling was based on the age-sex distribution of the Lebanese population as provided by the
23
24 Central Administration for Statistics. One adult was randomly selected from each household
25
26 using Kish methodology.¹⁹ Pregnant and lactating women and individuals with mental
27
28 disabilities were excluded. With a non-response rate of 10% at the individual level, this
29
30 yielded a sample of 2668 survey participants aged 18 years and above. Those free from
31
32 known history of hyperlipidemia and diabetes in the first phase of the study (n = 1,331) were
33
34 approached to undergo a biochemical assessment, of which 363 provided written consent and
35
36 gave fasting blood samples. Further details on the design and sample of the survey are
37
38 published elsewhere.⁴ The study protocol was approved by the Institutional Review Board of
39
40 the American University of Beirut (AUB), and informed consent was taken from all
41
42
43
44
45
46
47
48
49 participants.

50 **Data collection**

51
52 The data collection procedure followed the WHO STEPwise approach to Surveillance,²⁰ and
53
54 included the following three steps: Step 1 Questionnaire, whereby information about socio-
55
56 demographic characteristics, NCDs and NCD risk factors, including dietary intake, were
57
58 collected through face-to-face interviews; Step 2 in which anthropometric and blood pressure
59
60

1
2
3 measurements were taken using standardized techniques and calibrated equipment; and
4
5 finally, Step 3 in which biochemical analysis for assessment of the blood lipid profile was
6
7 performed on blood samples collected after an overnight fast of at least 8 hours. Serum was
8
9 centrifuged on site and shipped on dry ice to the AUB Laboratory.
10
11
12
13

14 **Measures of blood lipids**

15
16 Levels of blood lipids including TC, TG, very low density lipoprotein (VLDL), LDL-C and
17
18 HDL-C were analyzed using the Vitros 350 analyzer, an enzymatic spectrophotometric
19
20 technique. The inter-assay variation of measurements did not exceed 4%. Quality control was
21
22 performed within each run using standard performance verifier solutions provided by Ortho-
23
24 Clinical Diagnostics. Analyses were conducted in duplicates, and the average value was
25
26 utilized in the analysis. Based on the Adult Treatment Panel III guidelines,²¹ the cutoff points
27
28 used for the definition of risk levels of TC, TG, VLDL, LDL-C and HDL-C were ≥ 200 ,
29
30 ≥ 150 , ≥ 30 , ≥ 130 and ≤ 40 mg/dl, respectively.
31
32
33
34
35
36
37

38 **Behavioral risk factors and other measures**

39
40 Behavioral risk factors examined in this study included cigarette smoking, physical activity,
41
42 and alcohol consumption. Cigarette smoking status (never, past and current) and intensity
43
44 (number of cigarettes smoked/day) were assessed. Intensity was later categorized into three
45
46 levels according to number of cigarettes/day (1 to 19, 20 to 39 and ≥ 40). The short version of
47
48 the International Physical Activity Questionnaire was used to assess physical activity among
49
50 participants.²² Three categories of physical activity (low, moderate, and high) were assigned
51
52 based on METs-min/week (MET-min being the product of the resting metabolic rate for an
53
54 activity and the number of minutes taken to perform it). Alcohol-related behavior was
55
56 assessed as a dichotomous variable (ever vs never). Dietary assessment was performed using
57
58
59
60

1
2
3 a 61-item culture-specific food frequency questionnaire that measured food intake over the
4
5 past year.^{4, 15} Intakes of energy and macronutrients were estimated using the food
6
7 composition database of the Nutritionist IV software, and the food composition table for local
8
9 and traditional Middle-Eastern foods.^{23,24} Intakes of carbohydrates, fat and protein were
10
11 compared to cut-offs within the Acceptable Macronutrient Distribution Range,²⁵ and intakes
12
13 of saturated fat and sugar were compared to the recommendations of the WHO.^{26, 27}
14
15
16
17
18

19
20 Covariates of interest included total daily caloric intake and body mass index (BMI),
21
22 measured as the ratio of weight (kilograms) to the square of height (meters). In addition,
23
24 gender, age (18-29, 30-39, 40-49, 50-59, and ≥ 60), marital status (single, married,
25
26 divorced/widowed), education level (complementary or less, secondary/technical, university
27
28 and above), and occupational status (student/volunteer, working, does not
29
30 work/housewife/retired) were considered as potential covariates.
31
32
33
34

35 **Patient and Public Involvement**

36
37 This study is based on secondary data analyses. The original data collection tool was adapted
38
39 from the WHO STEPwise approach to NCD risk factor surveillance,²⁰ that did not directly
40
41 involve patients or the public in outcome development or conduct of the study. However, we
42
43 have been engaging with stakeholders to disseminate the findings on NCD risk factors,
44
45 including tobacco consumption and dietary intakes, and on associations with various health-
46
47 related outcomes to the public at large.
48
49
50
51
52

53 **Statistical analysis**

54
55 Means, standard deviations (SD) and frequencies were used to describe the various socio-
56
57 demographic, behavioral, nutritional and clinical characteristics of the participants. The
58
59
60

1
2
3 associations between each of the risk factors and levels of the different blood lipids were
4
5 examined using multiple logistic regression analysis. Unadjusted and adjusted odds ratios
6
7 (ORs) and their 95% confidence intervals (CIs) controlling for age, gender, education, marital
8
9 status, caloric intake and BMI were estimated. Test for trend with increasing number of daily
10
11 amount of cigarettes smoked was also conducted, and a two-sided p-value < 0.05 was
12
13 considered significant. The Statistical Package for the Social Sciences 22.0.1 (SPSS Inc.,
14
15 Chicago, IL, USA) was used for all computations.
16
17
18
19
20
21
22
23

24 **Results**

25
26
27 The socio-demographic and health-related characteristics of the study sample are summarized
28
29 in Table 1. The sample was equally divided by gender (49.9% females and 50.1% males),
30
31 with a mean age of 39.2 ± 15.2 years (range 18 to 92 years). The majority were married
32
33 (60.6%), and close to half of the participants were employed (52.3%) at the time of the study,
34
35 with a high percentage having less than complementary education (39.7%). Ever smokers
36
37 constituted 37.5% of the sample, 41.6% were classified as being engaged in low-intensity
38
39 physical activity, and 39.7% reported ever alcohol drinkers. Average daily energy intake was
40
41 estimated at 2656 ± 1249 Kcal/day, with 36.5% of caloric intake from fat, 11.4% from
42
43 saturated fat, 49.1% from carbohydrates, 5.7% from sugar, and 15.2% from protein.
44
45 Abnormal levels of TC, TG, VLDL, LDL-C, and HDL-C were observed for 55.4%, 31.4%,
46
47 29.2%, 47.5% and 21.8% of the participants, respectively.
48
49
50
51
52
53
54
55
56
57
58
59
60

Insert table 1 around here

1
2
3 Tables 2a and 2b show the unadjusted and adjusted ORs for the association between
4 behavioral risk factors and dietary variables with blood lipids levels. Associations with
5 cigarette smoking were positive for most outcomes but significant for those consuming more
6 than 40 cigarettes/day, compared to non-smokers, in the case of TG (unadjusted OR = 5.03)
7 and VLDL (OR = 4.09) and HDL-C OR = 3.02). Adjusting for potential confounders, the
8 associations maintained statistical significance for TG and VLDL, with an adjusted OR of
9 4.27 (95% CI 1.69- 10.77) and 3.26 (95% CI 1.33-8.03), respectively. Results showed a dose-
10 response relationship with increasing number of cigarettes consumed for (p-value for trend =
11 0.010 and 0.030, respectively). In addition, a statistically significant association was observed
12 between ever alcohol drinking and LDL-C (OR=1.53). This association retained statistical
13 significance even after adjustment for potential confounders with an OR of 1.68 (95% CI
14 1.01-2.82). Out of all the dietary variables examined, only saturated fat was associated with
15 blood lipids, namely TC and LDL-C, with an adjusted OR of 1.73 for both lipid abnormalities
16 (95% CI 1.02-2.94 and 1.02-2.93, respectively). Physical activity was not associated with any
17 of the blood lipid parameters.
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49

Insert tables 2a and 2b around here

50 Discussion

51 Our results showed that heavy cigarette smoking is associated with increased levels of TG
52 and VLDL, with findings showing significant dose-response relationships with increasing
53 number of cigarettes smoked per day. The study also showed that alcohol drinking and high
54 saturated fat intake are significantly associated with higher levels of LDL-C. However, there
55 were no consistent associations between physical activity and fasting blood lipids profile. To
56
57
58
59
60

1
2
3 our knowledge, this is the first study in Lebanon to explore these associations, based on
4 objectively measured lipid parameters and using standardized laboratory techniques and
5
6 tools, while taking into consideration the effect of potential confounders.
7
8
9

10
11 There are strong indicators in the literature that the deleterious effect of cigarette smoking on
12 heart disease and atherosclerosis is partially explained by the effect of smoking on the
13 concentration of blood lipids and lipoproteins. Our study confirms the results of earlier cross-
14 sectional studies showing that the association between smoking and lipoproteins are observed
15 in the levels of TG, VLDL and HDL-C, ^{6, 28, 29} with the impact on lipid levels increasing with
16 increase in the number of cigarettes smoked/day in a dose dependent relationship. ^{30, 31} Our
17 observation of the largest effect of cigarette smoking on lipid parameters being seen in those
18 who smoked more than two packs a day is also consistent with Chen et al. study comprising
19 of 1,164 men in Taiwan. ³² One popular mechanism by which smoking affects lipoproteins is
20 that cigarette particulate matter alters catecholamine release—and thus free fatty acid release,
21 which in turn contributes to the accumulation of the LDL-C concentrations and to lower
22 levels of HDL-C in the blood. ³³ The association between cigarette smoking and the increase
23 in TG can also be explained by the decrease in the activity of the lipoprotein lipase among
24 smokers thus disrupting lipid and lipoprotein metabolism. Furthermore, smoking cessation
25 was found to improve lipid and lipoprotein levels in observational studies and randomized
26 clinical trials. ^{7, 34} Taken together, the totality of evidence from these studies and our data-
27 including consistency upon replication across various studies, the dose-response relationship,
28 the magnitude and significance of association, biological plausibility as well as effects of
29 smoking cessation on lipoprotein levels, supports a strong relationship between smoking and
30 lipid profiles.
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

1
2
3 Our study showed that alcohol drinking was associated with higher LDL-C levels in
4
5 Lebanese adults but was not associated with other lipid parameters. Available evidence on
6
7 the impact of alcohol on LDL-C is conflicting with recent studies suggesting that the
8
9 association of alcohol and LDL-C levels may be population-specific.³⁵ For instance, studies
10
11 conducted amongst Danish adults reported an inverse association between alcohol intake and
12
13 LDL-C,³⁶ while studies conducted in Spanish and Italian populations found that higher
14
15 alcohol consumption was associated with increased LDL-C.^{37,38} According to a review by
16
17 Brinton (2012), these inconsistent findings on the association between alcohol intake and
18
19 LDL-C, may be explained by allele-specific genetic effects, such as the Apo E4 and Apo A5
20
21 genes.³⁹ In our study, alcohol drinking was not associated with HDL-C levels. Findings on
22
23 the relationship between alcohol and HDL-C have in fact been less consistent in the
24
25 literature, with some but not all showing an effect on higher HDL-C levels.^{40,41} Indeed, the
26
27 effect of alcohol on CVD including coronary artery disease, stroke and myocardial infarction
28
29 appears to be biphasic showing a J or U-shaped relationship based on the amount of alcohol
30
31 consumed,^{41,42} with lower CVD mortality risks by light to moderate alcohol consumption
32
33 and increased risk by heavy alcohol intake.⁴³ The discrepancy between our results with those
34
35 in the literature may be due to differences in race/ethnicity and genetic variations,⁴⁴
36
37 Alternatively, this discrepancy may be due to the definition of alcohol exposure adopted in
38
39 our study which was based on a dichotomous variable (ever vs never), and thus does not
40
41 capture alcohol intake in terms of frequency, intensity, types and pattern of alcohol
42
43 consumed. It is important to also acknowledge that alcohol consumption may be subject to
44
45 reporting bias in the Lebanese society due to cultural or religious norms. The observed
46
47 association between alcohol and lipid profile should therefore be interpreted with caution, as
48
49 it may have been the artifact of other social or lifestyle factors that were not measured in our
50
51 study.
52
53
54
55
56
57
58
59
60

1
2
3 Dietary intakes are amongst the modifiable risk factors that may modulate plasma lipids and
4 the risk of CVD. In our study, saturated fat was the only dietary factor that was significantly
5 associated with lipid parameters, and specifically with TC and LDL-C. Despite the
6 increasing controversy around the relationship between saturated fat and blood cholesterol
7 levels, an increasing body of evidence highlights the strong atherogenicity of saturated fatty
8 acids through their impact on LDL-C.¹⁴ A systematic review and meta-regression analysis
9 published in 2016 showed that a decrease in saturated fats of 1% of daily energy intake
10 coupled with an increase of 1% in polyunsaturated fat, lowered LDL cholesterol by 2.1
11 mg/dL.⁴⁵ In 2017, a Presidential Advisory from the American Heart Association (AHA)
12 concluded that available evidence strengthens the long-standing AHA recommendations to
13 decrease saturated fat intake and replace it with unsaturated fats. The AHA Advisory
14 highlighted that the shift from saturated to unsaturated fats should occur in the context of an
15 overall healthful dietary pattern such as the DASH or Mediterranean patterns.¹⁴

16
17
18 In our study, physical activity did not appear to be associated with lipid parameters. The
19 literature suggests that physical activity increases the level of HDL-C and has beneficial
20 effects on lipoprotein particle size and number.⁴⁶ Also, moderate intensity exercise is known
21 to have a more favorable effect on blood lipids since it allows the use of lipids as a fuel
22 source which implies an increase in the uptake and oxidation of the lipids in the skeletal
23 muscle.⁴⁶

24
25
26 Some other limitations should be taken into consideration when interpreting the results of our
27 study. Because the study used a cross-sectional design, findings only imply associations and
28 causal relationships cannot be established. The proportion of participants who gave fasting
29 blood samples (of at least 8 hours) was relatively small (27.3%); however, responders were
30 comparable to non-responders on a number of socio-demographic characteristics except for

1
2
3 marital status (61% of responders vs. 50% of non-responders were married). Also, we had
4
5 earlier documented comparable dietary data between respondents and non-respondents based
6
7 on factor loading matrices on patterns of food groups intake.⁴ As mentioned earlier,
8
9 measures of exposure were self-reported which can introduce some misclassification error.
10
11 This may have been particularly problematic in the case of alcohol consumption, given that
12
13 our definition based on a dichotomous variable of “ever” vs “never” does not allow for the
14
15 assessment of drinking frequency and patterns or the type of alcohol consumed. In contrast,
16
17 our measurement of cigarette smoking and physical activity were more detailed and reliable.
18
19 Information on cigarette smoking in our study included the amount of cigarettes smoked and
20
21 the standardized IPAQ was used to assess physical activity.
22
23
24
25
26

27 To conclude, our results suggest that smoking, alcohol drinking and high saturated fat are
28
29 associated with adverse levels of lipoprotein, among Lebanese men and women. There is
30
31 enough evidence in the literature indicating the role of lipoproteins in atherogenesis and that
32
33 controlling for blood lipid levels decreases the risk of heart and many other chronic diseases.
34
35 Our findings lay further evidence for clinical practitioners, public health professionals and
36
37 dieticians regarding the potential benefits of lifestyle and dietary modifications in their
38
39 pursuit to curb the burden of hyperlipidemia and CVD at the individual and population level.
40
41 The overall high rate of smoking behavior in Lebanon among both men and women, coupled
42
43 with the shift in dietary patterns towards high fat energy dense foods,^{7, 15} are likely to
44
45 adversely impact on the healthcare bill in the country. Further studies with larger sample size
46
47 that examine the association of combination patterns of poor lifestyle factors on lipid profile
48
49 among Lebanese adults are warranted.
50
51
52
53
54
55
56
57
58
59
60

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. CK, NH, MC and AF provided statistical advice and contributed to the paper revisions. AMS coordinated original study conduct, conceived and finalized the analyses, and contributed substantially to the write-up and revisions. All authors read and approved the final version of the manuscript.

Funding statement

The original study conduct was funded by the World Health Organization (WHO) - Lebanon. However, no funds were available for this secondary analysis.

Data sharing statement

The dataset was available for all authors of the study and will be available for other non-commercial researchers upon request.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

REFERENCES

1. Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G et al. Global, Regional, and National Burden of Cardiovascular Diseases for 10 Causes, 1990 to 2015. *Journal of the American College of Cardiology*. 2017;70:1-25.
2. Sibai AM, Nasreddine L, Mokdad AH, Adra N, Tabet M, Hwalla N. Nutrition transition and cardiovascular disease risk factors in Middle East and North Africa countries: reviewing the evidence. *Annals of Nutrition and Metabolism*. 2010;57:193-203.
3. World Health Organization. Non-communicable Diseases Country Profiles 2014.
4. Naja F, Nasreddine L, Itani L, Adra N, Sibai A, Hwalla N. Association between dietary patterns and the risk of metabolic syndrome among Lebanese adults. *European Journal of Nutrition*. 2013;52:97-105.
5. US Department of Health and Human Services. The health consequences of smoking—50 years of progress: a report of the Surgeon General. Atlanta (GA): The health consequences of smoking—50 years of progress: a report of the Surgeon General: Centers for Disease Control and Prevention (US), National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014.
6. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking intensity and lipoprotein abnormalities in active smokers. *Journal of Clinical Lipidology*. 2009;3:372-8.
7. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of smoking and smoking cessation on lipids and lipoproteins: outcomes from a randomized clinical trial. *American Heart Journal*. 2011;161:145-51.
8. Abramson JL, Vaccarino V. Relationship between physical activity and inflammation among apparently healthy middle-aged and older US adults. *Archives of Internal Medicine*. 2002;162:1286-92.
9. Barengo NC, Kastarinen M, Lakka T, Nissinen A, Tuomilehto J. Different forms of physical activity and cardiovascular risk factors among 24–64-year-old men and women in Finland. *European Journal of Cardiovascular Prevention & Rehabilitation*. 2006;13:51-9.
10. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The acute versus the chronic response to exercise. *Medicine and Science in Sports and Exercise*. 2001;33:S438-45.
11. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. *BMJ*. 2011;342:d636.
12. Wang Q, Afshin A, Yakoob MY, Singh GM, Rehm CD, Khatibzadeh S, on behalf of the Global Burden of Diseases. Nutrition and Chronic Diseases Expert Group (NutriCoDE). Impact of Nonoptimal Intakes of Saturated, Polyunsaturated, and Trans Fat on Global Burdens of Coronary Heart Disease. *J Am Heart Assoc*. 2016;5:e002891

13. Stone NJ, Robinson JG, Lichtenstein AH, Noel Bairey Merz C, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*. 2014;63:2889-934.
14. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, et al. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation*. 2017;13:e1-e23.
15. Naja F, Nasreddine L, Itani L, Chamieh MC, Adra N, Sibai AM, et al. Dietary patterns and their association with obesity and sociodemographic factors in a national sample of Lebanese adults. *Public Health Nutrition*. 2011;14:1570-8.
16. Sibai AM, Obeid O, Batal M, Adra N, El Khoury D, Hwalla N. Prevalence and correlates of metabolic syndrome in an adult Lebanese population. *CVD Prevention & Control*. 2008;3:83-90.
17. Sibai AM, Iskandarani M, Darzi A, Nakkash R, Saleh S, Fares S, et al. Cigarette smoking in a Middle Eastern country and its association with hospitalisation use: a nationwide cross-sectional study. *BMJ Open*. 2016;6:e009881.
18. Sibai AM, Hwalla N. WHO STEPS Chronic Disease Risk Factor Surveillance: Data Book for Lebanon. 2010.
https://www.who.int/ncds/surveillance/steps/2008_STEPS_Lebanon.pdf
19. Lavrakas PJ. *Encyclopedia of survey research methods*: Sage Publications; 2008.
20. World Health Organization (WHO). *Noncommunicable diseases and their risk factors- STEPS Manual*. <http://www.who.int/ncds/surveillance/steps/manual/en/>
21. National Cholesterol Education Program Adult Treatment Panel III. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-421.
22. IPAQ Research Committee. *Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)—short and long forms*. 2005.
23. Nutritionist IV. *N-squared computing*: Silverton; 1998. 31.
24. Pellet P, Shadarevian S. *Food composition. Tables for use in the Middle East*. In: *Food composition tables for use in the Middle East*. 2nd ed. 1970.
25. Institute of Medicine. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: The National Academies Press. 2005; <https://doi.org/10.17226/10490>.
26. Food and Agriculture Organization/World Health Organization. *Interim Summary of Conclusions and Dietary Recommendations on Total Fat & Fatty Acids*. 2008; https://www.who.int/nutrition/topics/FFA_summary_rec_conclusion.pdf?ua=1.
27. World Health Organization. *Guideline: Sugars intake for adults and children*. 2015; http://apps.who.int/iris/bitstream/handle/10665/149782/9789241549028_eng.pdf;jsessionid=2A114891D4D08F644AA154370830829B?sequence=1.
28. Yu M, Xu C-X, Zhu H-H, Hu R-Y, Zhang J, Wang H, et al. Associations of cigarette smoking and alcohol consumption with metabolic syndrome in a male Chinese population: a cross-sectional study. *Journal of Epidemiology*. 2014;24:361-9.

- 1
 - 2
 - 3
 - 4
 - 5
 - 6
 - 7
 - 8
 - 9
 - 10
 - 11
 - 12
 - 13
 - 14
 - 15
 - 16
 - 17
 - 18
 - 19
 - 20
 - 21
 - 22
 - 23
 - 24
 - 25
 - 26
 - 27
 - 28
 - 29
 - 30
 - 31
 - 32
 - 33
 - 34
 - 35
 - 36
 - 37
 - 38
 - 39
 - 40
 - 41
 - 42
 - 43
 - 44
 - 45
 - 46
 - 47
 - 48
 - 49
 - 50
 - 51
 - 52
 - 53
 - 54
 - 55
 - 56
 - 57
 - 58
 - 59
 - 60
29. Berlin I, Lin S, Lima JA, Bertoni AG. Smoking status and metabolic syndrome in the multi-ethnic study of atherosclerosis. A cross-sectional study. *Tobacco Induced Diseases*. 2012;10:9-16.
30. Bišanović S, Mehić B, Sivić S. Status of lipids and the frequency diseases of cardiovascular origin in smokers according to the length period of smoking and number of cigarettes smoked daily. *Bosnian Journal of Basic Medical Sciences*. 2011;11:46-51.
31. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Koblold ACM, et al. Associations between smoking, components of metabolic syndrome and lipoprotein particle size. *BMC Medicine*. 2013;11:195-209.
32. Chen C-C, Li T-C, Chang P-C, Liu C-S, Lin W-Y, Wu M-T, et al. Association among cigarette smoking, metabolic syndrome, and its individual components: the metabolic syndrome study in Taiwan. *Metabolism*. 2008;57:544-8.
33. Campbell SC, Moffatt RJ, Stamford BA. Smoking and smoking cessation—the relationship between cardiovascular disease and lipoprotein metabolism: a review. *Atherosclerosis*. 2008;201:225-35.
34. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the lipid and lipoprotein profiles: a meta-analysis. *Preventive Medicine*. 2003;37:283-90.
35. Matsumoto C, Miedema MD, Ofman P, Gaziano JM, Sesso HD. An expanding knowledge of the mechanisms and effects of alcohol consumption on cardiovascular disease. *Journal of Cardiopulmonary Rehabilitation and Prevention*. 2014;34:159-71.
36. Tolstrup JS, Gronbaek M, Nordestgaard BRG. Alcohol intake, myocardial infarction, biochemical risk factors, and alcohol dehydrogenase genotypes. *Circulation: Cardiovascular Genetics*. 2009;2:507-14.
37. Corella D, Portolés O, Arriola L, Chirlaque MD, Barricarte A, Francés F, et al. Saturated fat intake and alcohol consumption modulate the association between the APOE polymorphism and risk of future coronary heart disease: a nested case-control study in the Spanish EPIC cohort. *The Journal of Nutritional Biochemistry*. 2011;22:487-94.
38. Perissinotto E, Buja A, Maggi S, Enzi G, Manzato E, Scafato E, et al. Alcohol consumption and cardiovascular risk factors in older lifelong wine drinkers: the Italian Longitudinal Study on Aging. *Nutrition, Metabolism and Cardiovascular Diseases*. 2010;20:647-55.
39. Brinton EA. Effects of ethanol intake on lipoproteins. *Current Atherosclerosis Reports*. 2012;14:108-14.
40. Ellison RC, Zhang Y, Qureshi MM, Knox S, Arnett DK, Province MA. Lifestyle determinants of high-density lipoprotein cholesterol: The national heart, lung, and blood institute family heart study. *American Heart Journal*. 2004;147:529-35.
41. Park H, Kim K. Association of alcohol consumption with lipid profile in hypertensive men. *Alcohol and Alcoholism*. 2012;47:282-7.
42. Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary heart disease: a meta-analysis. *Addiction*. 2000;95:1505-23.

- 1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60
43. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. *BMJ*. 2011;342:d671.
44. López EP, Rice C, Weddle DO, Rahill GJ. The relationship among cardiovascular risk factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years and older. *Journal of the American Dietetic Association*. 2008;108:248-56.
45. Mensink RP. *Effects of Saturated Fatty Acids on Serum Lipids and Lipoproteins: A Systematic Review and Regression Analysis*. Geneva, Switzerland: World Health Organization; 2016.
46. Slentz CA, Houmard JA, Johnson JL, Bateman LA, Tanner CJ, McCartney JS, et al. Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a randomized, controlled study of exercise intensity and amount. *Journal of Applied Physiology*. 2007;103:432-42.

Table 1 Distribution of socio-demographic characteristics, behavioral factors and lipid profile of the study population.

	n	%
Gender (% female)	181	49.9
Age (mean \pm SD, years)	39.2 \pm 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 \pm SD, Kcal/day)	2656 \pm 1249	
Fat intake (% of total energy)^a	36.5 \pm 6.89	
≥ 30 ^b	275	81.6
Saturated fat intake (% of total energy)^a	11.4 \pm 2.92	
≥ 10 ^c	224	66.5
Carbohydrates intake (% of total energy)^a	49.1 \pm 7.03	
≥ 55 ^b	71	21.1
Sugar (% of total energy)^a	5.7 \pm 4.64	
≥ 10 ^d	56	16.6
Proteins intake (% of total energy)^a	152 \pm 2.91	
< 15 ^b	143	42.4
Total Cholesterol (mean \pm SD, mg/dl)	210 \pm 45	
% Elevated total cholesterol (≥ 200 mg/dl) ^e	201	55.4
Triglycerides (mean \pm SD, mg/dl)	138 \pm 78	
% Elevated triglycerides (≥ 150 mg/dl) ^e	114	31.4

VLDL (mean ± SD, mg/dl)	27 ± 15	
% Elevated VLDL (≥30 mg/dl) ^e	106	29.2
LDL-C (mean ± SD, mg/dl)	131 ± 39	
% Elevated LDL-C (≥130 mg/dl) ^e	172	47.5
HDL-C (mean ± SD, mg/dl)	51 ± 14	
% Reduced HDL-C (≤40 mg/dl) ^e	79	21.8

^a Dietary variables are based on a sample of 337 subjects owing to missing data

^b Macronutrient cutoffs are within the Acceptable Macronutrient Distribution Range (AMDR)²⁵

^c Saturated fat cutoff based on the WHO recommendations²⁶

^d Sugar intake cutoff based on the WHO recommendations²⁷

^e Lipid cutoff values based on the Adult Treatment Panel III guidelines²¹; Very Low Density Lipoprotein (VLDL); Low Density Lipoprotein cholesterol (LDL-C); High Density Lipoprotein cholesterol (HDL-C)

For peer review only

Table 2a Logistic regression analysis: associations of behavioral 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)
	0	52.0	1.00	1.00	25.6	1.00	1.00
Number of Cigarettes	1-19	55.8	1.17 (0.60-2.25)	0.85 (0.40-1.77)	30.2	1.26 (0.62-2.58)	1.06 (0.48-2.33)
	20-35	63.5	1.60 (0.90-2.85)	1.01 (0.51-2.00)	38.1	1.79 (0.99-3.23)	1.30 (0.67-2.53)
	≥40	63.3	1.59 (0.73-3.50)	1.24 (0.48-3.18)	63.3	5.03 (2.26-11.2)	4.27 (1.69-10.77)
	p-trend		0.070	0.783		<0.001	0.010
Physical activity	High	55.7	1.00	1	25.0	1.00	1
	Moderate	56.2	1.02 (0.59-1.77)	0.89 (0.48-1.67)	32.2	1.43 (0.77-2.64)	1.64 (0.83-3.25)
	Low	55.6	0.99 (0.59-1.69)	1.11 (0.31-2.00)	35.1	1.62 (0.90-2.92)	1.75 (0.93-3.29)
Alcohol intake	No	53	1	1	31.5	1	1
	Yes	59	1.28 (0.84-1.96)	1.52 (0.90-2.55)	31.3	0.99 (0.63-1.56)	0.98 (0.57-1.69)
Total fat intake (% energy)	<30	54.8	1	1.00	30.6	1	1
	≥30	54.9	1.03 (0.57-1.74)	1.15 (0.62-2.13)	31.3	1.03 (0.57-1.87)	1.22 (0.34-2.35)
Saturated Fat (% energy)	<10	52.2	1	1	31	1	1
	≥10	56.3	1.17 (0.75-1.85)	1.73 (1.02-2.94)	31.3	1.01 (0.62-1.65)	1.01 (0.59-1.74)
Carbohydrates (% energy)	<55	53.8	1	1	30.8	1	1
	≥55	59.2	1.25 (0.73-2.12)	1.07 (0.59-1.93)	32.4	1.07 (0.61-1.88)	0.96 (0.52-1.76)
Sugar intake (% energy)	<10	54.8	1	1	32	1	1
	≥10	55.4	1.02 (0.57-1.82)	1.12 (0.59-2.16)	26.8	0.78 (0.41-1.48)	0.86 (0.43-1.73)
Protein intake (% energy)	<15	56.6	1	1	36.4	1	1
	≥15	53.6	0.88 (0.57-1.37)	0.83 (0.50-1.37)	27.3	0.66 (0.41-1.05)	0.79 (0.47-1.32)

*Controlling for age, gender, education, marital status, caloric intake and BMI

Table 2b Logistic regression analysis: associations of behavioral 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 (0.58-2.52)	1.01 (0.45-2.25)	53.5	1.49 (0.77-2.86)	1.12 (0.54-2.33)	18.6	1.04 (0.45-2.40)	1.01 (0.39-2.63)
	20-39	35.5	1.72 (0.94-3.14)	1.27 (0.64-2.51)	54.8	1.57 (0.89-2.76)	0.97 (0.50-1.89)	28.6	1.81 (0.95-3.45)	1.61 (0.75-3.45)
	≥40	56.7	4.09 (1.87-8.95)	3.26 (1.33-8.03)	53.3	1.48 (0.69-3.17)	0.96 (0.39-2.40)	40.0	3.02 (1.35-6.76)	1.98 (0.77-5.08)
	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 (0.62-2.13)	1.27 (0.54-2.52)	49.6	1.02 (0.59-1.78)	0.89 (0.48-1.65)	19.0	0.62 (0.32-1.20)	0.93 (0.43-2.01)
	Low	32.0	1.33 (0.74-2.39)	1.44 (0.76-2.71)	46.0	0.89 (0.53-1.51)	0.93 (0.52-1.65)	21.2	0.72 (0.40-1.32)	0.69 (0.34-1.39)
Alcohol intake	No	28.3	1	1	43.4	1	1	20.5	1	1
	Yes	30.8	1.12 (0.71-1.79)	1.12 (0.65-1.93)	53.8	1.53 (1.00-2.33)	1.68 (1.01-2.82)	23.6	1.19 (0.72-1.98)	0.98 (0.54-1.78)
Total fat intake (% energy)	<30	29.0	1	1	45.2	1	1	25.8	1	1
	≥ 30	28.8	0.99 (0.54-1.82)	1.15 (0.59-2.22)	46.7	1.06 (0.61-1.85)	1.26 (0.69-2.33)	21.8	0.80 (0.42-1.52)	0.81 (0.39-1.65)
Saturated Fat (% energy)	<10	29.2	1	1	44.2	1	1	20.4	1	1
	≥ 10 ^s	28.7	0.98 (0.59-1.61)	0.96 (0.56-1.66)	47.5	1.15 (0.72-1.80)	1.73 (1.02-2.93)	23.7	1.21 (0.70-2.16)	1.16 (0.62-2.17)
Carbohydrates (% energy)	<55	28.7	1	1	45.7	1	1	22.2	1	1
	≥55	29.6	1.04 (0.59-1.86)	0.96 (0.52-1.79)	49.3	1.16 (0.68-1.95)	0.93 (0.52-1.67)	23.9	1.10 (0.60-2.05)	1.18 (0.59-2.36)
Sugar intake (% energy)	<10	29.6	1	1	47.1	1	1	23.8	1	1
	≥10	25.0	0.79 (0.41-1.53)	0.89 (0.44-1.82)	42.9	0.84 (0.47-1.50)	0.89 (0.47-1.71)	16.1	0.61 (0.28-1.31)	0.62 (0.26-1.45)
Protein intake (% energy)	<15	32.9	1	1	48.3	1	1	21	1	1
	≥15	25.9	0.71(0.44-1.15)	0.85 (0.50-1.44)	45.1	0.88 (0.57-1.36)	0.75 (0.46-1.24)	23.7	1.17 (0.59-1.97)	1.49 (0.82-2.72)

*Controlling for age, gender, education, marital status, caloric intake and BMI

BMJ Open

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

Journal:	<i>BMJ Open</i>
Manuscript ID	bmjopen-2018-026148.R3
Article Type:	Research
Date Submitted by the Author:	04-Jul-2019
Complete List of Authors:	Mansour, Megali; American University of Beirut Faculty of Health Sciences, EPHD Tamim, Hani; American University of Beirut, Biostatistics Unit, Clinical Research Institute Nasreddine, Lara; American University of Beirut EL Khoury, Christelle; Tufts University School of Medicine Hwalla, Nahla; American University of Beirut Chaaya, Monique; American University of Beirut Farhat, Antoine ; Notre Dame University Sibai, Abla; American University of Beirut, EPHD
Primary Subject Heading:	Public health
Secondary Subject Heading:	Epidemiology, Nutrition and metabolism, Smoking and tobacco, Public health
Keywords:	PUBLIC HEALTH, EPIDEMIOLOGY, Cigarette Smoking, Blood lipids

SCHOLARONE™
Manuscripts

1
2
3 **1 Prevalence and associations of behavioral risk factors with blood lipids profile in**
4
5 **2 Lebanese adults: findings from the WHO STEPwise NCD cross-sectional Survey**
6
7

8 3

9 4 Magali Mansour^{1†}, Hani Tamim^{2†}, Lara Nasreddine³, Christelle El Khoury⁴, Nahla Hwalla³,
10 5 Monique Chaaya¹, Antoine Farhat⁵, Abla M. Sibai^{1*}
11
12 6

13
14 **7 Author affiliations**

15 8 ¹ Department of Epidemiology and Population Health, Faculty of Health Sciences, American
16 9 University of Beirut, P.O.Box 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon

17 10 ² Department of Internal Medicine , American University of Beirut-Medical Center, P.O.Box
18 11 11-0236, Riad El-Solh, Beirut 1107 2020, Lebanon

19 12 † Equal Contribution

20 13 ³Faculty of Agricultural and Food Sciences, American University of Beirut, P.O.Box 11-
21 14 0236, Riad El-Solh, Beirut 1107 2020, Lebanon

22 15 ⁴School of Medicine Department of Public Health, Tufts University,136 Harrison Avenue,
23 16 Boston, MA 02111, US

24 17 ⁵Faculty of Nursing and Health Sciences, Notre Dame University, Lebanon
25 18
26 19
27 20

28
29
30
31
32
33
34
35 **21 *Corresponding author:**

36 **22 Abla M. Sibai**

37
38
39 23
40 24 Epidemiology and Population Health
41 25 Faculty of Health Sciences
42 26 American University of Beirut
43 27 PO Box 11-0236
44 28 Beirut-Lebanon
45 29 Tel: +961-1-350000 ext. 4647
46 30 Fax: +961-1-744470
47 31 Email: am00@aub.edu.lb
48
49 32
50
51 33
52
53 34
54
55
56
57
58
59
60

1
2
3 1 **Keywords:** Cardiovascular disease, smoking, alcohol consumption, physical inactivity,
4
5 2 dietary intake, blood lipids, Lebanon
6
7
8 3
9

10 4 **Word count (excluding title page, abstract, references, and tables):** 3143
11
12 5
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

1 **Abstract**

2 **Objective:** To examine associations of behavioral risk factors, namely cigarette smoking,
3 physical activity, dietary intakes and alcohol consumption, with blood lipids profile.

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

7 **Measures:** Demographic characteristics, behaviors and medical history were obtained from
8 participants by questionnaire. Dietary assessment was performed using a 61-item culture-
9 specific food frequency questionnaire that measured food intake over the past year. Lipid
10 levels were measured by analysis of fasting blood samples (serum total cholesterol-TC,
11 triglycerides-TG, very low-density lipoprotein-VLDL, low-density lipoprotein cholesterol-
12 LDL-C and high-density lipoprotein cholesterol-HDL-C).

13 **Results:** Current cigarette smoking, alcohol consumption and low physical activity were
14 prevalent among 33.3%, 39.7%, and 41.6% of the sample, respectively. The contributions of
15 fat and saturated fat to daily energy intake were high, estimated at 36.5% and 11.4%,
16 respectively. Abnormal levels of TC, TG, VLDL, LDL-C, and HDL-C were observed for
17 55.4%, 31.4%, 29.2%, 47.5% and 21.8% of participants, respectively. Adjusting for potential
18 confounders, cigarette smoking was positively associated with higher odds of TG and VLDL
19 (OR=4.27; 95% CI 1.69-10.77; and 3.26; 95% CI 1.33-8.03, respectively) with a significant
20 dose-response relationship (p-value for trend=0.010 and 0.030, respectively). Alcohol
21 drinking and high saturated fat intake ($\geq 10\%$ energy intake) were associated with higher odds
22 of LDL-C (OR=1.68; 95% CI: 1.01-2.82 and OR= 1.73; 95% CI: 1.02-2.93). Physical
23 activity did not associate significantly with any blood lipid parameter.

24 **Conclusion:** The demonstrated positive associations between smoking, alcohol drinking and
25 high saturated fat intake with adverse lipoprotein levels lay further evidence for clinical

1
2
3 1 practitioners, public health professionals and dietitians in the development of preventive
4
5 2 strategies among subjects with high risk of CVDs in Lebanon and other neighboring
6
7 3 countries with similar epidemiological profile.
8
9
10 4
11
12 5
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

For peer review only

1 **Strengths and limitations**

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

1 Introduction

2 The prevalence of cardiovascular diseases (CVD) is growing worldwide, and has reached
3 epidemic levels, affecting both developed and developing countries.¹ The Middle East and
4 North Africa countries represent a region which is now facing a fast rate of development and
5 urbanization, with rates of chronic diseases increasing at an alarming rate and exceeding at
6 times those of developed countries.² In Lebanon, a small middle-income country at the
7 Eastern Mediterranean shore, data from the World Health Organization (WHO) indicate that
8 the proportional mortality from CVD alone is 45%, making it the highest among all non-
9 communicable diseases (NCDs).³ With a population estimate of around 4.2 million and a
10 Gross Domestic Product (GDP) of close to 8,520 US Dollars per capita, Lebanon is
11 characterized by a high urbanization rate (87%), a growing trend towards survival in later
12 life, coupled with westernization and modernization in diet and lifestyle and higher uptake of
13 NCD risk factors.⁴

14 The primary goal in the prevention and management of CVD is to identify and modify the
15 underlying risk behaviors that are amenable to intervention, namely, cigarette smoking,
16 physical inactivity, dietary intakes and alcohol consumption. Associations between these
17 factors and CVD risk through their effect on blood lipid levels have been widely examined in
18 the western literature. Studies have shown that smokers are 2-4 times more at risk of
19 developing heart disease than nonsmokers⁵ and the number of cigarettes smoked/day
20 independently predicts higher levels of Total Cholesterol (TC), Low Density Lipoprotein-
21 Cholesterol (LDL-C), and Triglycerides (TG).⁶ Also, smoking cessation has been shown to
22 improve High Density Lipoprotein-Cholesterol (HDL-C) levels.⁷ Cigarette smoking is
23 described as a strong inflammation mediator and a key promoter in the atherosclerotic
24 process. Similarly, the anti-inflammatory effect of frequent physical activity has been noted
25 to be the reason behind reduced heart disease risk among physically active individuals.⁸

1
2
3 1 Regular physical activity with weight reduction has a large beneficial impact on the
4
5 2 lipoproteins profile of adult men and women,⁹ by increasing plasma volume, decreasing
6
7 3 blood thickness, and thus reducing LDL-C concentrations.¹⁰ Also, systematic reviews and
8
9 4 meta-analysis of intervention studies have shown that heavy alcohol drinking results in an
10
11 5 elevation in triglyceride levels, while moderate consumption increases circulating levels of
12
13 6 HDL-C.¹¹ Similarly, diet is recognized as a modifiable risk factor that can make a
14
15 7 substantial contribution to the risk of CVD.^{12 13} Energy intake, the types of fatty acids
16
17 8 consumed and the level of sugar ingestion may impact the lipid and cardiometabolic
18
19 9 profile.¹⁴ The 2017 Presidential Advisory from the American Heart Association indicated that
20
21 10 replacement of saturated fat with unsaturated fatty acids decreases LDL-C levels and CVD
22
23 11 risk, while replacing it with refined carbohydrates and sugar, yields no significant benefits to
24
25 12 cardiovascular health.¹⁴

26
27 13 Much of the above evidence comes from studies conducted in North American and
28
29 14 European countries and to a lesser extent from the Far East, mostly among Japanese and
30
31 15 Korean population. Arab populations including the Lebanese have quite different risk
32
33 16 behaviors, varied dietary habits and risk profile,¹⁵ and studies evaluating the association
34
35 17 between behaviors and intermediary variables along the causal pathway of CVD
36
37 18 including lipid levels remain scarce in the region. Compared with other neighboring
38
39 19 countries, Lebanon was shown to have one of the highest prevalence estimates of
40
41 20 metabolic syndrome,¹⁶ and has been witnessing high rates of smoking, among both
42
43 21 adult men and women aged 18 years and over (42.9% and 27.5%, respectively).¹⁷ Using
44
45 22 data from a nationwide population-based survey of Lebanese adults, this study aims to
46
47 23 examine the relation between behavioral risk factors including cigarette smoking,
48
49 24 physical activity, dietary intakes and alcohol consumption with serum lipids and
50
51 25 lipoproteins, while taking into account several potential confounding factors. Findings
52
53
54
55
56
57
58
59
60

1 from this study inform prevention strategies among subjects with high risk of CVD in
2 the country.

3

4 **Methods**

5 **Study design and participants**

6 The data presented in this study are derived from the WHO Nutrition and Non-
7 Communicable Diseases Risk Factor (NNCD-RF) cross-sectional household survey
8 conducted in Lebanon in 2009.¹⁸ Using multi-stage stratified cluster study design, the
9 sampling was based on the age-sex distribution of the Lebanese population as provided by the
10 Central Administration for Statistics. One adult was randomly selected from each household
11 using Kish methodology.¹⁹ Pregnant and lactating women and individuals with mental
12 disabilities were excluded. With a non-response rate of 10% at the individual level, this
13 yielded a sample of 2668 survey participants aged 18 years and above. Those free from
14 known history of hyperlipidemia and diabetes in the first phase of the study (n = 1,331) were
15 approached to undergo a biochemical assessment, of which 363 provided written consent and
16 gave fasting blood samples. Further details on the design and sample of the survey are
17 published elsewhere.⁴ The study protocol was approved by the Institutional Review Board of
18 the American University of Beirut (AUB), and informed consent was taken from all
19 participants.

20 **Data collection**

21 The data collection procedure followed the WHO STEPwise approach to Surveillance,²⁰ and
22 included the following three steps: Step 1 Questionnaire, whereby information about socio-
23 demographic characteristics, NCDs and NCD risk factors, including dietary intake, were
24 collected through face-to-face interviews; Step 2 in which anthropometric and blood pressure

1 measurements were taken using standardized techniques and calibrated equipment; and
2
3 finally, Step 3 in which biochemical analysis for assessment of the blood lipid profile was
4
5 performed on blood samples collected after an overnight fast of at least 8 hours. Serum was
6
7 centrifuged on site and shipped on dry ice to the AUB Laboratory.
8
9
10
11
12
13

14 **Measures of blood lipids**

15
16 Levels of blood lipids including TC, TG, very low density lipoprotein (VLDL), LDL-C and
17
18 HDL-C were analyzed using the Vitros 350 analyzer, an enzymatic spectrophotometric
19
20 technique. The inter-assay variation of measurements did not exceed 4%. Quality control was
21
22 performed within each run using standard performance verifier solutions provided by Ortho-
23
24 Clinical Diagnostics. Analyses were conducted in duplicates, and the average value was
25
26 utilized in the analysis. Based on the Adult Treatment Panel III guidelines,²¹ the cutoff points
27
28 used for the definition of risk levels of TC, TG, VLDL, LDL-C and HDL-C were ≥ 200 ,
29
30 ≥ 150 , ≥ 30 , ≥ 130 and ≤ 40 mg/dl, respectively.
31
32
33
34
35
36
37

38 **Behavioral risk factors and other measures**

39
40 Behavioral risk factors examined in this study included cigarette smoking, physical activity,
41
42 and alcohol consumption. Cigarette smoking status (never, past and current) and intensity
43
44 (number of cigarettes smoked/day) were assessed. Intensity was later categorized into three
45
46 levels according to number of cigarettes/day (1 to 19, 20 to 39 and ≥ 40). The short version of
47
48 the International Physical Activity Questionnaire was used to assess physical activity among
49
50 participants.²² Three categories of physical activity (low, moderate, and high) were assigned
51
52 based on METs-min/week (MET-min being the product of the resting metabolic rate for an
53
54 activity and the number of minutes taken to perform it). Alcohol-related behavior was
55
56 assessed as a dichotomous variable (ever vs never). Dietary assessment was performed using
57
58
59
60

1 a 61-item culture-specific food frequency questionnaire that measured food intake over the
2 past year.^{4, 15} Intakes of energy and macronutrients were estimated using the food
3 composition database of the Nutritionist IV software, and the food composition table for local
4 and traditional Middle-Eastern foods.^{23,24} Intakes of carbohydrates, fat and protein were
5 compared to cut-offs within the Acceptable Macronutrient Distribution Range,²⁵ and intakes
6 of saturated fat and sugar were compared to the recommendations of the WHO.^{26, 27}

7
8 Covariates of interest included total daily caloric intake and body mass index (BMI),
9 measured as the ratio of weight (kilograms) to the square of height (meters). In addition,
10 gender, age (18-29, 30-39, 40-49, 50-59, and ≥ 60), marital status (single, married,
11 divorced/widowed), education level (complementary or less, secondary/technical, university
12 and above), and occupational status (student/volunteer, working, does not
13 work/housewife/retired) were considered as potential covariates.

15 **Patient and Public Involvement**

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

23 **Statistical analysis**

24 Means, standard deviations (SD) and frequencies were used to describe the various socio-
25 demographic, behavioral, nutritional and clinical characteristics of the participants. The

1 associations between each of the risk factors and levels of the different blood lipids were
2 examined using multiple logistic regression analysis. Unadjusted and adjusted odds ratios
3 (ORs) and their 95% confidence intervals (CIs) controlling for age, gender, education, marital
4 status, caloric intake and BMI were estimated. Test for trend with increasing number of daily
5 amount of cigarettes smoked was also conducted, and a two-sided p-value < 0.05 was
6 considered significant. The Statistical Package for the Social Sciences 22.0.1 (SPSS Inc.,
7 Chicago, IL, USA) was used for all computations.

10 Results

11 The socio-demographic and health-related characteristics of the study sample are summarized
12 in Table 1. The sample was equally divided by gender (49.9% females and 50.1% males),
13 with a mean age of 39.2 ± 15.2 years (range 18 to 92 years). The majority were married
14 (60.6%), and close to half of the participants were employed (52.3%) at the time of the study,
15 with a high percentage having less than complementary education (39.7%). Ever smokers
16 constituted 37.5% of the sample, 41.6% were classified as being engaged in low-intensity
17 physical activity, and 39.7% reported ever alcohol drinkers. Average daily energy intake was
18 estimated at 2656 ± 1249 Kcal/day, with 36.5% of caloric intake from fat, 11.4% from
19 saturated fat, 49.1% from carbohydrates, 5.7% from sugar, and 15.2% from protein.
20 Abnormal levels of TC, TG, VLDL, LDL-C, and HDL-C were observed for 55.4%, 31.4%,
21 29.2%, 47.5% and 21.8% of the participants, respectively.

23 Insert table 1 around here

1
2
3 1 Tables 2a and 2b show the unadjusted and adjusted ORs for the association between
4
5 2 behavioral risk factors and dietary variables with blood lipids levels. Associations with
6
7 3 cigarette smoking were positive for most outcomes but significant for those consuming more
8
9 4 than 40 cigarettes/day, compared to non-smokers, in the case of TG (unadjusted OR = 5.03)
10
11 5 and VLDL (OR = 4.09) and HDL-C OR = 3.02). Adjusting for potential confounders, the
12
13 6 associations maintained statistical significance for TG and VLDL, with an adjusted OR of
14
15 7 4.27 (95% CI 1.69- 10.77) and 3.26 (95% CI 1.33-8.03), respectively. Results showed a dose-
16
17 8 response relationship with increasing number of cigarettes consumed for (p-value for trend =
18
19 9 0.010 and 0.030, respectively). In addition, a statistically significant association was observed
20
21 10 between ever alcohol drinking and LDL-C (OR=1.53). This association retained statistical
22
23 11 significance even after adjustment for potential confounders with an OR of 1.68 (95% CI
24
25 12 1.01-2.82). Out of all the dietary variables examined, only saturated fat was associated with
26
27 13 blood lipids, namely TC and LDL-C, with an adjusted OR of 1.73 for both lipid abnormalities
28
29 14 (95% CI 1.02-2.94 and 1.02-2.93, respectively). Physical activity was not associated with any
30
31 15 of the blood lipid parameters.
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49

17 Insert tables 2a and 2b around here

20 Discussion

21 Our results showed that heavy cigarette smoking is associated with increased levels of TG
22 and VLDL, with findings showing significant dose-response relationships with increasing
23 number of cigarettes smoked per day. The study also showed that alcohol drinking and high
24 saturated fat intake are significantly associated with higher levels of LDL-C. However, there
25 were no consistent associations between physical activity and fasting blood lipids profile. To
60

1
2
3 1 our knowledge, this is the first study in Lebanon to explore these associations, based on
4
5 2 objectively measured lipid parameters and using standardized laboratory techniques and
6
7 3 tools, while taking into consideration the effect of potential confounders.
8
9

10
11 4 There are strong indicators in the literature that the deleterious effect of cigarette smoking on
12
13 5 heart disease and atherosclerosis is partially explained by the effect of smoking on the
14
15 6 concentration of blood lipids and lipoproteins. Our study confirms the results of earlier cross-
16
17 7 sectional studies showing that the association between smoking and lipoproteins are observed
18
19 8 in the levels of TG, VLDL and HDL-C, ^{6, 28, 29} with the impact on lipid levels increasing with
20
21 9 increase in the number of cigarettes smoked/day in a dose dependent relationship. ^{30, 31} Our
22
23 10 observation of the largest effect of cigarette smoking on lipid parameters being seen in those
24
25 11 who smoked more than two packs a day is also consistent with Chen et al. study comprising
26
27 12 of 1,164 men in Taiwan. ³² One popular mechanism by which smoking affects lipoproteins is
28
29 13 that cigarette particulate matter alters catecholamine release—and thus free fatty acid release,
30
31 14 which in turn contributes to the accumulation of the LDL-C concentrations and to lower
32
33 15 levels of HDL-C in the blood. ³³ The association between cigarette smoking and the increase
34
35 16 in TG can also be explained by the decrease in the activity of the lipoprotein lipase among
36
37 17 smokers thus disrupting lipid and lipoprotein metabolism. Furthermore, smoking cessation
38
39 18 was found to improve lipid and lipoprotein levels in observational studies and randomized
40
41 19 clinical trials. ^{7, 34} Taken together, the totality of evidence from these studies and our data-
42
43 20 including consistency upon replication across various studies, the dose-response relationship,
44
45 21 the magnitude and significance of association, biological plausibility as well as effects of
46
47 22 smoking cessation on lipoprotein levels, supports a strong relationship between smoking and
48
49 23 lipid profiles.
50
51
52
53
54
55
56
57
58
59
60

1
2
3 1 Our study showed that alcohol drinking was associated with higher LDL-C levels in
4
5 2 Lebanese adults but was not associated with other lipid parameters. Available evidence on
6
7 3 the impact of alcohol on LDL-C is conflicting with recent studies suggesting that the
8
9 4 association of alcohol and LDL-C levels may be population-specific.³⁵ For instance, studies
10
11 5 conducted amongst Danish adults reported an inverse association between alcohol intake and
12
13 6 LDL-C,³⁶ while studies conducted in Spanish and Italian populations found that higher
14
15 7 alcohol consumption was associated with increased LDL-C.^{37,38} According to a review by
16
17 8 Brinton (2012), these inconsistent findings on the association between alcohol intake and
18
19 9 LDL-C, may be explained by allele-specific genetic effects, such as the Apo E4 and Apo A5
20
21 10 genes.³⁹ In our study, alcohol drinking was not associated with HDL-C levels. Findings on
22
23 11 the relationship between alcohol and HDL-C have in fact been less consistent in the
24
25 12 literature, with some but not all showing an effect on higher HDL-C levels.^{40,41} Indeed, the
26
27 13 effect of alcohol on CVD including coronary artery disease, stroke and myocardial infarction
28
29 14 appears to be biphasic showing a J or U-shaped relationship based on the amount of alcohol
30
31 15 consumed,^{41,42} with lower CVD mortality risks by light to moderate alcohol consumption
32
33 16 and increased risk by heavy alcohol intake.⁴³ The discrepancy between our results with those
34
35 17 in the literature may be due to differences in race/ethnicity and genetic variations.⁴⁴
36
37 18 Alternatively, it may be due to the definition of alcohol exposure adopted in our study which
38
39 19 was based on a dichotomous variable (ever vs never), and thus does not capture alcohol
40
41 20 intake in terms of frequency, intensity, types and pattern of alcohol consumed. It is important
42
43 21 to also acknowledge that alcohol consumption may be subject to reporting bias in the
44
45 22 Lebanese society due to cultural or religious norms. The observed association between
46
47 23 alcohol and lipid profile should therefore be interpreted with caution, as it may have been the
48
49 24 artifact of other social or lifestyle factors that were not measured in our study.
50
51
52
53
54
55
56
57
58
59
60

1
2
3 1 Dietary intakes are amongst the modifiable risk factors that may modulate plasma lipids and
4
5 2 the risk of CVD. In our study, saturated fat was the only dietary factor that was significantly
6
7 3 associated with lipid parameters, and specifically with TC and LDL-C. Despite the
8
9
10 4 increasing controversy around the relationship between saturated fat and blood cholesterol
11
12 5 levels, an increasing body of evidence highlights the strong atherogenicity of saturated fatty
13
14 6 acids through their impact on LDL-C.¹⁴ A systematic review and meta-regression analysis
15
16 7 published in 2016 showed that a decrease in saturated fats of 1% of daily energy intake
17
18 8 coupled with an increase of 1% in polyunsaturated fat, lowered LDL cholesterol by 2.1
19
20 9 mg/dL.⁴⁵ In 2017, a Presidential Advisory from the American Heart Association (AHA)
21
22 10 concluded that available evidence strengthens the long-standing AHA recommendations to
23
24 11 decrease saturated fat intake and replace it with unsaturated fats. The AHA Advisory
25
26 12 highlighted that the shift from saturated to unsaturated fats should occur in the context of an
27
28 13 overall healthful dietary pattern such as the DASH or Mediterranean patterns.¹⁴
29
30
31
32
33
34 14 In our study, physical activity did not appear to be associated with lipid parameters. The
35
36 15 literature suggests that physical activity increases the level of HDL-C and has beneficial
37
38 16 effects on lipoprotein particle size and number.⁴⁶ Also, moderate intensity exercise is known
39
40 17 to have a more favorable effect on blood lipids since it allows the use of lipids as a fuel
41
42 18 source which implies an increase in the uptake and oxidation of the lipids in the skeletal
43
44 19 muscle.⁴⁶
45
46
47
48
49 20 Some other limitations should be taken into consideration when interpreting the results of our
50
51 21 study. Because the study used a cross-sectional design, findings only imply associations and
52
53 22 causal relationships cannot be established. The proportion of participants who gave fasting
54
55 23 blood samples (of at least 8 hours) was relatively small (27.3%); however, responders were
56
57 24 comparable to non-responders on a number of socio-demographic characteristics except for
58
59
60

1 marital status (61% of responders vs. 50% of non-responders were married). Also, we had
2 earlier documented comparable dietary data between respondents and non-respondents based
3 on factor loading matrices on patterns of food groups intake.⁴ As mentioned earlier,
4 measures of exposure were self-reported which can introduce some misclassification error.
5 This may have been particularly problematic in the case of alcohol consumption, given that
6 our definition based on a dichotomous variable of “ever” vs “never” does not allow for the
7 assessment of drinking frequency and patterns or the type of alcohol consumed. In contrast,
8 our measurement of cigarette smoking and physical activity were more detailed and reliable.
9 Information on cigarette smoking in our study included the amount of cigarettes smoked and
10 the standardized IPAQ was used to assess physical activity.

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

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. CK, NH, MC and AF provided statistical advice and contributed to the paper revisions. AMS coordinated original study conduct, conceived and finalized the analyses, and contributed substantially to the write-up and revisions. All authors read and approved the final version of the manuscript.

Funding statement

The original study conduct was funded by the World Health Organization (WHO) - Lebanon. However, no funds were available for this secondary analysis.

Data sharing statement

The dataset was available for all authors of the study and will be available for other non-commercial researchers upon request.

Declaration of conflicting interests

The authors declare that there is no conflict of interest.

1
2
3 **1 REFERENCES**
4

- 5
6 2 1. Roth GA, Johnson C, Abajobir A, Abd-Allah F, Abera SF, Abyu G et al. Global,
7 3 Regional, and National Burden of Cardiovascular Diseases for 10 Causes, 1990 to
8 4 2015. *Journal of the American College of Cardiology*. 2017;7 0:1-25.
- 9 5 2. Sibai AM, Nasreddine L, Mokdad AH, Adra N, Tabet M, Hwalla N. Nutrition
10 6 transition and cardiovascular disease risk factors in Middle East and North Africa
11 7 countries: reviewing the evidence. *Annals of Nutrition and Metabolism*. 2010;57:193-
12 8 203.
- 13 9 3. World Health Organization. Non-communicable Diseases Country Profiles 2014.
- 14 10 4. Naja F, Nasreddine L, Itani L, Adra N, Sibai A, Hwalla N. Association between
15 11 dietary patterns and the risk of metabolic syndrome among Lebanese adults. *European*
16 12 *Journal of Nutrition*. 2013;52:97-105.
- 17 13 5. US Department of Health and Human Services. The health consequences of
18 14 smoking—50 years of progress: a report of the Surgeon General. Atlanta (GA): The
19 15 health consequences of smoking—50 years of progress: a report of the Surgeon
20 16 General: Centers for Disease Control and Prevention (US), National Center for
21 17 Chronic Disease Prevention and Health Promotion, Office on Smoking and Health;
22 18 2014.
- 23 19 6. Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH. Smoking
24 20 intensity and lipoprotein abnormalities in active smokers. *Journal of Clinical*
25 21 *Lipidology*. 2009;3:372-8.
- 26 22 7. Gepner AD, Piper ME, Johnson HM, Fiore MC, Baker TB, Stein JH. Effects of
27 23 smoking and smoking cessation on lipids and lipoproteins: outcomes from a
28 24 randomized clinical trial. *American Heart Journal*. 2011;161:145-51.
- 29 25 8. Abramson JL, Vaccarino V. Relationship between physical activity and inflammation
30 26 among apparently healthy middle-aged and older US adults. *Archives of Internal*
31 27 *Medicine*. 2002;162:1286-92.
- 32 28 9. Barengo NC, Kastarinen M, Lakka T, Nissinen A, Tuomilehto J. Different forms of
33 29 physical activity and cardiovascular risk factors among 24–64-year-old men and
34 30 women in Finland. *European Journal of Cardiovascular Prevention & Rehabilitation*.
35 31 2006;13:51-9.
- 36 32 10. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N, Pescatello L. The
37 33 acute versus the chronic response to exercise. *Medicine and Science in Sports and*
38 34 *Exercise*. 2001;33:S438-45.
- 39 35 11. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol
40 36 consumption on biological markers associated with risk of coronary heart disease:
41 37 systematic review and meta-analysis of interventional studies. *BMJ*. 2011;342:d636.
- 42 38 12. Wang Q, Afshin A, Yakoob MY, Singh GM, Rehm CD, Khatibzadeh S, on behalf of
43 39 the Global Burden of Diseases. Nutrition and Chronic Diseases Expert Group
44 40 (NutriCoDE). Impact of Nonoptimal Intakes of Saturated, Polyunsaturated, and Trans
45 41 Fat on Global Burdens of Coronary Heart Disease. *J Am Heart Assoc*.
46 42 2016;5:e002891

13. Stone NJ, Robinson JG, Lichtenstein AH, Noel Bairey Merz C, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*. 2014;63:2889-934.
14. Sacks FM, Lichtenstein AH, Wu JH, Appel LJ, Creager MA, Kris-Etherton PM, et al. Dietary fats and cardiovascular disease: a presidential advisory from the American Heart Association. *Circulation*. 2017;13:e1-e23.
15. Naja F, Nasreddine L, Itani L, Chamieh MC, Adra N, Sibai AM, et al. Dietary patterns and their association with obesity and sociodemographic factors in a national sample of Lebanese adults. *Public Health Nutrition*. 2011;14:1570-8.
16. Sibai AM, Obeid O, Batal M, Adra N, El Khoury D, Hwalla N. Prevalence and correlates of metabolic syndrome in an adult Lebanese population. *CVD Prevention & Control*. 2008;3:83-90.
17. Sibai AM, Iskandarani M, Darzi A, Nakkash R, Saleh S, Fares S, et al. Cigarette smoking in a Middle Eastern country and its association with hospitalisation use: a nationwide cross-sectional study. *BMJ Open*. 2016;6:e009881.
18. Sibai AM, Hwalla N. WHO STEPS Chronic Disease Risk Factor Surveillance: Data Book for Lebanon. 2010.
https://www.who.int/ncds/surveillance/steps/2008_STEPS_Lebanon.pdf
19. Lavrakas PJ. *Encyclopedia of survey research methods*: Sage Publications; 2008.
20. World Health Organization (WHO). *Noncommunicable diseases and their risk factors- STEPS Manual*. <http://www.who.int/ncds/surveillance/steps/manual/en/>
21. National Cholesterol Education Program Adult Treatment Panel III. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-421.
22. IPAQ Research Committee. *Guidelines for data processing and analysis of the International Physical Activity Questionnaire (IPAQ)—short and long forms*. 2005.
23. Nutritionist IV. *N-squared computing*: Silverton; 1998. 31.
24. Pellet P, Shadarevian S. Food composition. Tables for use in the Middle East. In: *Food composition tables for use in the Middle East*. 2nd ed. 1970.
25. Institute of Medicine. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: The National Academies Press. 2005; <https://doi.org/10.17226/10490>.
26. Food and Agriculture Organization/World Health Organization. *Interim Summary of Conclusions and Dietary Recommendations on Total Fat & Fatty Acids*. 2008; https://www.who.int/nutrition/topics/FFA_summary_rec_conclusion.pdf?ua=1.
27. World Health Organization. *Guideline: Sugars intake for adults and children*. 2015; http://apps.who.int/iris/bitstream/handle/10665/149782/9789241549028_eng.pdf;jssessionid=2A114891D4D08F644AA154370830829B?sequence=1.
28. Yu M, Xu C-X, Zhu H-H, Hu R-Y, Zhang J, Wang H, et al. Associations of cigarette smoking and alcohol consumption with metabolic syndrome in a male Chinese population: a cross-sectional study. *Journal of Epidemiology*. 2014;24:361-9.

- 1
2
3 1 29. Berlin I, Lin S, Lima JA, Bertoni AG. Smoking status and metabolic syndrome in the
4 2 multi-ethnic study of atherosclerosis. A cross-sectional study. *Tobacco Induced*
5 3 *Diseases*. 2012;10:9-16.
- 6 4 30. Bišanović S, Mehić B, Sivić S. Status of lipids and the frequency diseases of
7 5 cardiovascular origin in smokers according to the length period of smoking and
8 6 number of cigarettes smoked daily. *Bosnian Journal of Basic Medical Sciences*.
9 7 2011;11:46-51.
- 10 8 31. Slagter SN, van Vliet-Ostapchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold
11 9 ACM, et al. Associations between smoking, components of metabolic syndrome and
12 10 lipoprotein particle size. *BMC Medicine*. 2013;11:195-209.
- 13 11 32. Chen C-C, Li T-C, Chang P-C, Liu C-S, Lin W-Y, Wu M-T, et al. Association among
14 12 cigarette smoking, metabolic syndrome, and its individual components: the metabolic
15 13 syndrome study in Taiwan. *Metabolism*. 2008;57:544-8.
- 16 14 33. Campbell SC, Moffatt RJ, Stamford BA. Smoking and smoking cessation—the
17 15 relationship between cardiovascular disease and lipoprotein metabolism: a review.
18 16 *Atherosclerosis*. 2008;201:225-35.
- 19 17 34. Maeda K, Noguchi Y, Fukui T. The effects of cessation from cigarette smoking on the
20 18 lipid and lipoprotein profiles: a meta-analysis. *Preventive Medicine*. 2003;37:283-90.
- 21 19 35. Matsumoto C, Miedema MD, Ofman P, Gaziano JM, Sesso HD. An expanding
22 20 knowledge of the mechanisms and effects of alcohol consumption on cardiovascular
23 21 disease. *Journal of Cardiopulmonary Rehabilitation and Prevention*. 2014;34:159-71.
- 24 22 36. Tolstrup JS, Gronbaek M, Nordestgaard BRG. Alcohol intake, myocardial infarction,
25 23 biochemical risk factors, and alcohol dehydrogenase genotypes. *Circulation:
26 24 Cardiovascular Genetics*. 2009;2:507-14.
- 27 25 37. Corella D, Portolés O, Arriola L, Chirlaque MD, Barricarte A, Francés F, et al.
28 26 Saturated fat intake and alcohol consumption modulate the association between the
29 27 APOE polymorphism and risk of future coronary heart disease: a nested case-control
30 28 study in the Spanish EPIC cohort. *The Journal of Nutritional Biochemistry*.
31 29 2011;22:487-94.
- 32 30 38. Perissinotto E, Buja A, Maggi S, Enzi G, Manzato E, Scafato E, et al. Alcohol
33 31 consumption and cardiovascular risk factors in older lifelong wine drinkers: the
34 32 Italian Longitudinal Study on Aging. *Nutrition, Metabolism and Cardiovascular
35 33 Diseases*. 2010;20:647-55.
- 36 34 39. Brinton EA. Effects of ethanol intake on lipoproteins. *Current Atherosclerosis
37 35 Reports*. 2012;14:108-14.
- 38 36 40. Ellison RC, Zhang Y, Qureshi MM, Knox S, Arnett DK, Province MA. Lifestyle
39 37 determinants of high-density lipoprotein cholesterol: The national heart, lung, and
40 38 blood institute family heart study. *American Heart Journal*. 2004;147:529-35.
- 41 39 41. Park H, Kim K. Association of alcohol consumption with lipid profile in hypertensive
42 40 men. *Alcohol and Alcoholism*. 2012;47:282-7.
- 43 41 42. Corrao G, Rubbiati L, Bagnardi V, Zambon A, Poikolainen K. Alcohol and coronary
44 42 heart disease: a meta-analysis. *Addiction*. 2000;95:1505-23.

1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22
23
24
25
26
27
28
29
30
31
32
33
34
35
36
37
38
39
40
41
42
43
44
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

- 1 43. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol
2 consumption with selected cardiovascular disease outcomes: a systematic review and
3 meta-analysis. *BMJ*. 2011;342:d671.
- 4 44. López EP, Rice C, Weddle DO, Rahill GJ. The relationship among cardiovascular risk
5 factors, diet patterns, alcohol consumption, and ethnicity among women aged 50 years
6 and older. *Journal of the American Dietetic Association*. 2008;108:248-56.
- 7 45. Mensink RP. Effects of Saturated Fatty Acids on Serum Lipids and Lipoproteins: A
8 Systematic Review and Regression Analysis. Geneva, Switzerland: World Health
9 Organization; 2016.
- 10 46. Slentz CA, Houmard JA, Johnson JL, Bateman LA, Tanner CJ, McCartney JS, et al.
11 Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a
12 randomized, controlled study of exercise intensity and amount. *Journal of Applied
13 Physiology*. 2007;103:432-42.

Table 1 Distribution of socio-demographic characteristics, behavioral factors and lipid profile of the study population.

	n	%
Gender (% female)	181	49.9
Age (mean \pm SD, years)	39.2 \pm 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 \pm SD, Kcal/day)	2656 \pm 1249	
Fat intake (% of total energy)^a	36.5 \pm 6.89	
≥ 30 ^b	275	81.6
Saturated fat intake (% of total energy)^a	11.4 \pm 2.92	
≥ 10 ^c	224	66.5
Carbohydrates intake (% of total energy)^a	49.1 \pm 7.03	
≥ 55 ^b	71	21.1
Sugar (% of total energy)^a	5.7 \pm 4.64	
≥ 10 ^d	56	16.6
Proteins intake (% of total energy)^a	152 \pm 2.91	
< 15 ^b	143	42.4
Total Cholesterol (mean \pm SD, mg/dl)	210 \pm 45	
% Elevated total cholesterol (≥ 200 mg/dl) ^e	201	55.4
Triglycerides (mean \pm SD, mg/dl)	138 \pm 78	
% Elevated triglycerides (≥ 150 mg/dl) ^e	114	31.4

VLDL (mean ± SD, mg/dl)	27 ± 15	
% Elevated VLDL (≥30 mg/dl) ^e	106	29.2
LDL-C (mean ± SD, mg/dl)	131 ± 39	
% Elevated LDL-C (≥130 mg/dl) ^e	172	47.5
HDL-C (mean ± SD, mg/dl)	51 ± 14	
% Reduced HDL-C (≤40 mg/dl) ^e	79	21.8

^a Dietary variables are based on a sample of 337 subjects owing to missing data

^b Macronutrient cutoffs are within the Acceptable Macronutrient Distribution Range (AMDR) ²⁵

^c Saturated fat cutoff based on the WHO recommendations ²⁶

^d Sugar intake cutoff based on the WHO recommendations ²⁷

^e Lipid cutoff values based on the Adult Treatment Panel III guidelines ²¹; Very Low Density Lipoprotein (VLDL); Low Density Lipoprotein cholesterol (LDL-C); High Density Lipoprotein cholesterol (HDL-C)

For peer review only

Table 2a Logistic regression analysis: associations of behavioral 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)
	0	52.0	1.00	1.00	25.6	1.00	1.00
Number of Cigarettes	1-19	55.8	1.17 (0.60-2.25)	0.85 (0.40-1.77)	30.2	1.26 (0.62-2.58)	1.06 (0.48-2.33)
	20-35	63.5	1.60 (0.90-2.85)	1.01 (0.51-2.00)	38.1	1.79 (0.99-3.23)	1.30 (0.67-2.53)
	≥40	63.3	1.59 (0.73-3.50)	1.24 (0.48-3.18)	63.3	5.03 (2.26-11.2)	4.27 (1.69-10.77)
	p-trend		0.070	0.783		<0.001	0.010
Physical activity	High	55.7	1.00	1	25.0	1.00	1
	Moderate	56.2	1.02 (0.59-1.77)	0.89 (0.48-1.67)	32.2	1.43 (0.77-2.64)	1.64 (0.83-3.25)
	Low	55.6	0.99 (0.59-1.69)	1.11 (0.31-2.00)	35.1	1.62 (0.90-2.92)	1.75 (0.93-3.29)
Alcohol intake	No	53	1	1	31.5	1	1
	Yes	59	1.28 (0.84-1.96)	1.52 (0.90-2.55)	31.3	0.99 (0.63-1.56)	0.98 (0.57-1.69)
Total fat intake (% energy)	<30	54.8	1	1.00	30.6	1	1
	≥30	54.9	1.03 (0.57-1.74)	1.15 (0.62-2.13)	31.3	1.03 (0.57-1.87)	1.22 (0.34-2.35)
Saturated Fat (% energy)	<10	52.2	1	1	31	1	1
	≥10	56.3	1.17 (0.75-1.85)	1.73 (1.02-2.94)	31.3	1.01 (0.62-1.65)	1.01 (0.59-1.74)
Carbohydrates (% energy)	<55	53.8	1	1	30.8	1	1
	≥55	59.2	1.25 (0.73-2.12)	1.07 (0.59-1.93)	32.4	1.07 (0.61-1.88)	0.96 (0.52-1.76)
Sugar intake (% energy)	<10	54.8	1	1	32	1	1
	≥10	55.4	1.02 (0.57-1.82)	1.12 (0.59-2.16)	26.8	0.78 (0.41-1.48)	0.86 (0.43-1.73)
Protein intake (% energy)	<15	56.6	1	1	36.4	1	1
	≥15	53.6	0.88 (0.57-1.37)	0.83 (0.50-1.37)	27.3	0.66 (0.41-1.05)	0.79 (0.47-1.32)

*Controlling for age, gender, education, marital status, caloric intake and BMI

Table 2b Logistic regression analysis: associations of behavioral 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 (0.58-2.52)	1.01 (0.45-2.25)	53.5	1.49 (0.77-2.86)	1.12 (0.54-2.33)	18.6	1.04 (0.45-2.40)	1.01 (0.39-2.63)
	20-39	35.5	1.72 (0.94-3.14)	1.27 (0.64-2.51)	54.8	1.57 (0.89-2.76)	0.97 (0.50-1.89)	28.6	1.81 (0.95-3.45)	1.61 (0.75-3.45)
	≥40	56.7	4.09 (1.87-8.95)	3.26 (1.33-8.03)	53.3	1.48 (0.69-3.17)	0.96 (0.39-2.40)	40.0	3.02 (1.35-6.76)	1.98 (0.77-5.08)
	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 (0.62-2.13)	1.27 (0.54-2.52)	49.6	1.02 (0.59-1.78)	0.89 (0.48-1.65)	19.0	0.62 (0.32-1.20)	0.93 (0.43-2.01)
	Low	32.0	1.33 (0.74-2.39)	1.44 (0.76-2.71)	46.0	0.89 (0.53-1.51)	0.93 (0.52-1.65)	21.2	0.72 (0.40-1.32)	0.69 (0.34-1.39)
Alcohol intake	No	28.3	1	1	43.4	1	1	20.5	1	1
	Yes	30.8	1.12 (0.71-1.79)	1.12 (0.65-1.93)	53.8	1.53 (1.00-2.33)	1.68 (1.01-2.82)	23.6	1.19 (0.72-1.98)	0.98 (0.54-1.78)
Total fat intake (% energy)	<30	29.0	1	1	45.2	1	1	25.8	1	1
	≥ 30	28.8	0.99 (0.54-1.82)	1.15 (0.59-2.22)	46.7	1.06 (0.61-1.85)	1.26 (0.69-2.33)	21.8	0.80 (0.42-1.52)	0.81 (0.39-1.65)
Saturated Fat (% energy)	<10	29.2	1	1	44.2	1	1	20.4	1	1
	≥ 10 ^s	28.7	0.98 (0.59-1.61)	0.96 (0.56-1.66)	47.5	1.15 (0.72-1.80)	1.73 (1.02-2.93)	23.7	1.21 (0.70-2.16)	1.16 (0.62-2.17)
Carbohydrates (% energy)	<55	28.7	1	1	45.7	1	1	22.2	1	1
	≥55	29.6	1.04 (0.59-1.86)	0.96 (0.52-1.79)	49.3	1.16 (0.68-1.95)	0.93 (0.52-1.67)	23.9	1.10 (0.60-2.05)	1.18 (0.59-2.36)
Sugar intake (% energy)	<10	29.6	1	1	47.1	1	1	23.8	1	1
	≥10	25.0	0.79 (0.41-1.53)	0.89 (0.44-1.82)	42.9	0.84 (0.47-1.50)	0.89 (0.47-1.71)	16.1	0.61 (0.28-1.31)	0.62 (0.26-1.45)
Protein intake (% energy)	<15	32.9	1	1	48.3	1	1	21	1	1
	≥15	25.9	0.71(0.44-1.15)	0.85 (0.50-1.44)	45.1	0.88 (0.57-1.36)	0.75 (0.46-1.24)	23.7	1.17 (0.59-1.97)	1.49 (0.82-2.72)

*Controlling for age, gender, education, marital status, caloric intake and BMI