www.sciencedirect.com/journal/journal-of-vascular-nursing

Metabolic differences between men and women who are long-term users of the water pipe: The Irbid WiHi project

Mahmoud A. Alomari, PhD, Omar F. Khabour, PhD, and Karem H. Alzoubi, PhD

Smoking tobacco and metabolic disorders are global epidemics associated with cardiovascular, immune, respiratory, and metabolic diseases. Cigarette smoking seems to affect metabolic disorders. However, the effect of water pipe (Wp), also called hookah, smoking duration on obesity and lipid profile is still a sparse. The present study examined the relationship of smoking Wp with body weight (Bw), body mass index (BMI), total cholesterol, low-density lipoprotein cholesterol (LDLc), high-density lipoprotein cholesterol (HDLc), total cholesterol/HDLc, LDLc/HDLc, and triglycerides. Obesity, lipid profile, and smoking status were obtained from a total of 291 participants of which 147 smoked Wp. Smokers were divided into 3 groups: smoked <10 years (Wp1) (n = 72), smoked 10–20 years (Wp2) (n = 43), and smoked >20 years (Wp3) (n = 30). The ANCOVA revealed greater BMI and Bw and lipid profile measures in individuals smoking Wp vs never (P < .01). Additional analysis revealed that Bw and BMI were greater in the women (n = 11) with longer Wp smoking history (P < .05) but not the men (n = 19). In addition, Tc, LDLc, Tc/HDLc, and LDLc/HDLc levels were greater among the men smoking Wp for longer time (P < .05), but not the women. In conclusion, the study found greater obesity and lipid profile in the adults smoked Wp vs never, especially the ones smoked for longer time. (J Vasc Nurs 2020;38:18-24)

INTRODUCTION

Tobacco smoking is a social behavior with a prevalence soaring worldwide, particularly in developing countries with current use rates of about 30%.[1,2] It is a devastating public health dilemma associated with cardiovascular and respiratory diseases, stroke, cancer, and diabetes, the leading causes of global hospitalization, morbidity, and mortality.[3] Water pipe (Wp) smoking is a tobacco consumption style that involves inhaling smoke from a charcoal-burned tobacco through a bowl of water with a hose into the smoker mouth.[4] It has aggressively reemerged as a favorable smoking style among all population segments across the globe. On the contrary to cigarette smoking, this global phenomenon is spiraling also in developed countries, including Europe, the United States, and Australia.[5] Acceptance, peer pressure, gathering, publicity, accessibility, and affordability are some of the social factors alluring people to smoke Wp.[6,7] Similar to cigarette smoking, however, Wp smoking is associated with the most horrendous diseases, including cardiovascular and respiratory diseases, stroke, cancer, and metabolic syndrome.[8]

Obesity is another proliferating global epidemic associated with greater hospitalization, morbidity, and mortality. It is a complex metabolic disorder linked to increased risk of developing diseases of many body systems, including the cardiovascular, respiratory, metabolic, musculoskeletal, and immune systems. In fact, obesity has recently been classified as a disease.[9] Based on a WHO report, global obesity has nearly tripled during the last 4 decades.[10] Currently, global adult obesity/overweight has exceeded 2.5 billion (~52%)[10] and projected to reach ~3.3 billion in 2030.[11] Most of this future increase is in low- and middle-income countries (ie, the Middle East and North Africa, MENA region),[12] but some developed countries are no exception (ie, North America).[10] This disproportionate increase in global obesity has led to the introduction of "globesity" term.[9]

Similarly, dyslipidemia is prevalent and contribute to most serious illnesses, ischemic heart, and cerebral diseases. Circulatory lipid and cholesterol have been implicated in atherogenesis and subsequent arterial narrowing, blood flow restriction, and heart and cerebral ischemia.[13] According to WHO data (~147 000 000 individuals), the mean prevalence of dyslipidemia is 33% (range: 19.2–61.6%). The report was from the United States, Mexico, Japan, Thailand, England, Germany, Scotland and Jordan, a sample of developed and underdeveloped countries.[14]

The causes of obesity and dyslipidemia are multifaceted, that include genetic and environmental factors. However, the

From the Department of Physical Education, Qatar University, Doha, Qatar; Division of Physical Therapy, Department of Rehabilitation Sciences, Jordan University of Science and Technology, Irbid, Jordan; Department of Medical Laboratory Sciences, Jordan University of Science and Technology, Irbid, Jordan; Department of Clinical Pharmacy, Jordan University of Science and Technology, Irbid, Jordan.

Corresponding author: Mahmoud A. Alomari, PhD, Division of Physical Education, Department of Educational Sciences, Qatar University, Doha 2713, Qatar (E-mail: Malomari@qu.edu.qa).

Conflict of interest: The authors declare no conflict of interest or financial interest.

1062-0303/$36.00

https://doi.org/10.1016/j.jvn.2019.12.004

underlining causes have been attributed to lifestyle, mainly physical inactivity and poor diet.[9,13]

The effect of smoking cigarettes on obesity and lipid profile is well studied. Reduced body mass index (BMI),[15] greater abdominal fatness,[16] and dyslipidemia[17] have been reported in individuals smoking cigarettes. A limited number of studies, however, examined the effect of smoking Wp on obesity[18–20] and blood lipids.[21] Few studies reported that smoking Wp is associated with increased obesity (ie, BMI), 2 in adults[19,20] and one in adolescents.[18] Similarly, one study revealed greater total cholesterol (Tc) and low-density lipoprotein cholesterol (LDLc) in individuals smoking Wp only vs never[22] and in concurrent Wp/cigarette smokers compared with never and cigarette smokers only.[21] However, these studies did not report the effect of the duration of Wp smoking history on obesity and lipid. Therefore, the present study examined the relationship of smoking Wp duration with obesity and lipid. Obesity measures were body weight (Bw) and BMI. Lipid profile included Tc, high-density lipoprotein cholesterol (HDLc), LDLc, Tc/HDLc, LDLc/HDLc, and triglycerides (Trg). The present study is particularly important, given the spiral increase in Wp use. In addition, it can further verify the health effects of smoking, especially when combined with elevated obesity[23] and lipid,[24] among individuals smoking Wp.

MATERIALS AND METHOD

Design and recruitment

Data presented in the present study were extracted from the "Water pipe and Health in Irbid (Irbid WiHi)" project. Irbid is a city located in Northern Jordan, the second largest municipality in Jordan with population of around 1,911,600.[25] The aim of the project was to assess the health effect of Wp tobacco smoking among young and old adults. Individuals who smoked Wp exclusively were recruited for the study. The present study is descriptive, comparative, and cross-sectional, designed to investigate the possible association between the long-term use Wp and measures of lipid profile men and women. The status of tobacco consumption and lipid profile indices were acquired from eligible individuals.

Adults, who are apparently healthy, were recruited to the study from the greater governorate of Irbid. The Wp cafes and shops, malls, and parks were recruitment sites for the study. Recruitment was achieved after participants were screened for eligibility to the study. Excluded from the study were smokers who reported acute hypertension, hyperglycemia, hypercholesterolemia, hyperlipidemia, cancer, psychiatric and stress-related mood disorders, or patients on medications that could influence lipid profile. The study protocol was approved by the institutional review board of the University and as in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Written informed consents were singed by all study participants. The participant primary physician was notified in case abnormal lipid results were found. The study conduct and dissemination were in compliance with the STROBE guidelines for observational studies.[26]

Anthropometric measurements

A standard weighing scale was used to measure the Bw. A standard measuring tape was used to determine the height (Ht). The BMI was calculated as Bw/Ht^2. The participants were divided according to the following criteria[27]:

- Underweight: BMI is less than 18.5
- Healthy weight: BMI is 18.5 to 24.9
- Overweight: BMI is 25 to 29.9
- Obese: BMI is 30 or higher

Smoking status

Participants were asked about age, gender, smoking, and socioeconomic status. Moreover, years of Wp smoking were obtained.[2,28] Individuals who reported a tobacco use history other than Wp were excluded from the study. The control group for this study was age-matched, gender-matched, geographical distribution–matched, and exclusion criteria–matched individuals who never smoked any form of tobacco products. The participants were further divided into 4 groups according to smoking history. These groups include do not smoke (never), smoked <10 years (Wp1), smoked 10–20 years (Wp2), and smoked >20 years (Wp3).

Lipid profile

After an overnight fasting, blood samples were collected from study participants in EDTA tubes. The blood samples were centrifuged to obtain plasma samples, which were stored in aliquots at −30°C until further analysis. The Tc, HDL, LDL, and TG were determined using the Roche Analyzer and Roche reagents (Roche Diagnostics, Basel, Switzerland). A grant from JUST was used to pay for the participants' time and transportation as well as to determine blood lipid profile.

Statistics

Statistical analyses were completed with SPSS software for Windows (version 22.0; Chicago, IL). Data are expressed as means ± SD, and α was preset at $P < .05$.

Two-way (4 smoking groups* 2 gender) ANCOVA, while covariating for age, was used to compare obesity and lipid measures between never, group 1 Wp, group 2 Wp, and group 3 Wp in the men vs women. Obesity included Bw and BMI, whereas lipid profile included Tc, LDLc, HDLc, Tc/HDLc, LDLc/HDLc, and Trg. Additional subsequent comparisons were used to determine the differences between specific groups.[29]

RESULTS

Participant characteristics

A total of 291 men (n = 141) and women (n = 150) agreed to participate in the study, of which 147 smoked Wp. Among the participants, a total of 70 men and 74 women did not smoke any type of tobacco. As in Table 1, age, Bw, Ht, and BMI ranges were 20–85 years, 41–130 kg, 140–195 cm, and 11.9–46.3 kg/m^2, respectively.

Metabolic differences with water pipe smoking

The obesity and lipid profile comparisons between never and groups Wp1, Wp2, and Wp3 smokers are shown in Table 2. The two-way ANCOVA revealed a main effect of smoking history ($P < .0001$) and gender ($P < .0001$) without ($P > .3$) interaction effect for Bw after adjusting for age. Additional comparisons showed that Bw was greater ($P < .05$) in the women in Wp2 and Wp3 vs never smokers and in Wp2 and Wp3 vs ($P < .05$) Wp1. No differences ($P > .5$) in Bw were found between Wp1

TABLE 1

THE PARTICIPANTS' DEMOGRAPHIC CHARACTERISTICS (N = 291)

Gender (%)	
Male	48.9
Female	51.1
Age (y, mean [SD])	39.7 (13.2)
Weight (kg, mean [SD])	76.2 (16.3)
Height (cm, mean [SD])	169.2 (8.1)
BMI (kg/m^2, mean [SD])	26.5 (5.0)
Smoking status (%)	
Never smoked	49.48
Wp smokers	50.51
Years of smoking (y, mean [SD])	12.46 (7.3)
Dependence score	7.15 (8.4)

BMI = body mass index; Wp = water pipe.

smokers and never or between Wp2 and Wp3. No differences ($P > .5$) were found in Bw between the men groups.

Another two-way ANCOVA showed a main effect of smoking history ($P < .005$) without gender ($P < .3$) and ($P > .12$) interaction effect for BMI after adjusting for age. Subsequent comparisons in the women showed that BMI was greater ($P < .05$) in Wp2 and Wp3 vs never smokers and in Wp2 and Wp3 vs Wp1 smokers, without differences ($P > .5$) between Wp1 and never or between Wp2 and Wp3. In addition, no differences ($P > .5$) were found in BMI between the men groups.

A separate two-way ANCOVA revealed a main effect of smoking history ($P < .04$) and gender ($P < .02$) without interaction effect ($P > .3$) for Tc after adjusting for age. Subsequent comparisons showed that Tc was greater ($P < .05$) in the men in Wp3 vs never and Wp2 smoking. No differences ($P > .05$) were found in Tc in the men in Wp2 vs never and Wp1 or between Wp1 never and Wp3, as well as between the women groups.

With respect to Trg, no effect for smoking history ($P > .20$), gender ($P > .8$), or interaction ($P > .9$) after adjusting for age was detected. A different two-way ANCOVA revealed a main effect of gender ($P < .0001$) but not of smoking history ($P > .15$) and interaction ($P > .9$) for HDLc after adjusting for age. Subsequent comparisons showed no differences ($P > .5$) in HDLc between the men groups or the women groups.

Another two-way ANCOVA revealed a main effect of smoking history ($P < .03$) and interaction ($P < .03$) without gender effect ($P > .3$) for LDLc after adjusting for age. Subsequent comparisons showed that LDLc was greater ($P < .05$) in the men Wp3, Wp2, and Wp1 vs never smoking. No differences ($P > .5$) were found between the Wp3, Wp2, and Wp1 smoking group. In addition, no differences ($P > .5$) were found in LDLc between the women groups.

As in Tables 2 and 3, a separate two-way ANCOVA revealed a main effect of smoking history ($P < .004$) and gender ($P < .03$) without interaction effect ($P > .5$) for Tc/HDLc and LDLc/HDLc after adjusting for age. Subsequent comparisons showed that Tc/HDLc and LDLc/HDLc was greater ($P < .05$) in the men Wp3, Wp2, and Wp1 vs never smoking. No differences ($P > .5$) were found between the Wp3, Wp2, and Wp1 smoking groups. However, no differences ($P > .5$) were found in Tc/HDLc and LDLc/HDLc between the women groups.

TABLE 2

TWO-WAY ANCOVA IN MEN AND WOMEN WATER PIPE SMOKERS

	Men (n = 141)		*Women (n = 150)*	
	Water pipe	***Never***	***Water pipe***	***Never***
Weight (kg)	85.2 ± 15.3*	78.3 ± 15.6	71.2 ± 15.5	68.9 ± 13.4
BMI (kg/m^2)	27.8 ± 4.6*	25.9 ± 4.6	26.1 ± 5.4	26.9 ± 4.9
Cholesterol (mmol/L)	4.6 ± 1.0*	4.0 ± 0.8	4.8 ± 1.1	4.9 ± 1.0
Triglyceride (mmol/L)	2.1 ± 1.3	1.7 ± 1.3	1.9 ± 1.1	1.7 ± 1.3
HDLc (mmol/L)	1.0 ± 0.2	1.0 ± 0.2	1.2 ± 0.3	1.2 ± 0.2
LDLc (mmol/L)	3.4 ± 0.8*	2.7 ± 0.6	3.3 ± 0.9	3.5 ± 0.8
Cholesterol/HDLc	4.6 ± 0.9*	4.0 ± 1.0	4.1 ± 0.9	4.0 ± 0.8
LDLc/HDLc	3.3 ± 0.7*	2.7 ± 0.8	2.9 ± 0.8	2.8 ± 0.70

BMI = body mass index.

*$P < .05$ vs never smoker counterpart.

TABLE 3

TWO-WAY ANCOVA FOR YEARS OF SMOKING IN MEN AND WOMEN SMOKING WATER PIPE

Men	*Never (n = 70)*	*Water pipe 1 (n = 24)*	*Water pipe 2 (n = 28)*	*Water pipe 3 (n = 19)*
Weight (kg)	79.3 ± 16.2	82.1 ± 17.2	83.2 ± 14.9	78.9 ± 13.5
BMI (kg/m^2)	26.6 ± 5.1	26.9 ± 5.2	27.5 ± 4.6	28.2 ± 4.9
Cholesterol (mmol/L)	4.2 ± 0.9	4.4 ± 1.1	4.4 ± 0.9	5.1 ± 1.0*,‡
Triglyceride (mmol/L)	1.7 ± 1.2	2.0 ± 1.4	1.7 ± 1.4	2.2 ± 1.1
HDLc (mmol/L)	1.0 ± 0.2	1.2 ± 0.3	1.2 ± 0.3	1.2 ± 0.2
LDLc (mmol/L)	2.8 ± 0.7	3.3 ± 0.9*	3.2 ± 0.7*	3.7 ± 0.9*
Tc/HDLc	4.0 ± 1.0	4.3 ± 1.0*	4.6 ± 0.9*	4.7 ± 0.9*
LDLc/HDLc	2.7 ± 0.8	3.2 ± 0.9*	3.3 ± 0.7*	3.4 ± 0.8*
Women	Never (n = 74)	Water pipe 1 (n = 48)	Water pipe 2 (n = 15)	Water pipe 3 (n = 11)
Weight (kg)	67.1 ± 11.8	68.3 ± 13.9	77.4 ± 15.7*,†	78.9 ± 18.0*,†
BMI (kg/m^2)	25.5 ± 4.2	24.9 ± 5.2	28.5 ± 5.6*,†	28.9 ± 6.4*,†
Cholesterol (mmol/L)	4.8 ± 0.9	4.7 ± 1.2	4.6 ± 1.1	5.4 ± 0.5
Triglyceride (mmol/L)	1.7 ± 1.2	2.0 ± 1.1	1.8 ± 1.0	2.3 ± 1.1
HDLc (mmol/L)	1.2 ± 0.2	1.2 ± 0.3	1.1 ± 0.3	1.2 ± 0.2
LDLc (mmol/L)	3.4 ± 0.7	3.3 ± 1.0	3.2 ± 0.9	3.7 ± 0.4
Tc/HDLc	4.0 ± 0.7	4.1 ± 1.0	4.2 ± 1.0	4.0 ± 0.6
LDLc/HDLc	2.8 ± 0.7	2.8 ± 0.9	2.9 ± 0.9	3.1 ± 0.5

*$P < .05$ vs never smokers.
†$P < .05$ vs water pipe 1 smokers.
‡$P < .05$ vs water pipe 2 smokers.

DISCUSSION

Smoking Wp is a behavior spiraling throughout the globe across all society segments. Many social factors have contributed to this increase in Wp use including acceptance, peer pressure, gathering, publicity, accessibility, and affordability.[6,7] The study examined the relationship of Wp smoking with obesity and lipid profile. The results revealed greater obesity and lipid profile measures in individuals smoking Wp vs never. Additional analysis revealed that weight and BMI were greater in the women with longer Wp smoking history, but not the men. Weight in the women smoked >20 years packed extra 10 kg as compared with never smokers and the ones smoked <10 years, whereas BMI was 3–4 units greater. Similarly, Tc, LDLc, Tc/HDLc, and LDLc/HDLc levels were greater among the men smoking Wp for longer time but not the women. Tc, LDLc, Tc/HDLc, and LDLc/HDLc were ∼25%, ∼35%, ∼17%, and ∼26%, respectively, greater in the men Wp smokers vs never smokers. The results are unprecedented and suggest that the relationship of Wp smoking with obesity and lipid profile is affected by gender and duration of smoking. In addition, the study further confirms the adverse health effects of Wp smoking.

The causes of obesity and adverse circulatory lipid profile are multifactorial, which include genetic and environmental factors. However, the underlining causes are lifestyle-related, a positive mismatch between caloric consumption and expenditure. Poor diet, physical inactivity, or a combination of both often results in metabolic disarray, obesity, and dyslipidemia.[9,30,31]

Several studies have shown inconsistent relationship of smoking tobacco with obesity, depending on the obesity measure used. The "popular wisdom," although, smoking cigarettes is associated with lower obesity, whereas smoking cessation usually results in weight gains. In fact, smoking has been used as a strategy for weight control.[15] However, although smoking is associated with increased food intake, these discrepancies in caloric intake were not sufficient to explain these differences in obesity.[32]

In one study, BMI was similar in never compared with ex-smokers and greater than current smokers.[33] BMI increased after smoking cessation[34] and was not explained by food and alcohol intake.[33] Interestingly, this relationship seems to be age-dependent[35] and smoking status–dependent.[34] Lower BMI was found in younger but not old adult smokers[35] and among individuals

smoked 6–10 cigarettes but not heavy smokers.[34] Conversely, another study showed that BMI was positively related to the number of smoked cigarettes.[36] The results of these studies indicate a dose-response relationship of smoking with BMI.[34] The relationship of smoking with abdominal obesity seems direct and smoking duration–related, age-related, and gender-related. Waist circumference was greater among smokers, especially older adults and females,[35] and increased with the number of cigarettes smoked per day.[16] In addition, adolescent cigarette smoking was associated with abdominal obesity during adulthood in both genders and in overweight women, especially heavy smokers.[37]

However, the gender and smoking duration effects on obesity is still equivocal in individuals smoking Wp. Among the few studies examining the relationship of Wp smoking with obesity, one study found that Wp smokers are more likely to have abdominal obesity compared with never.[19] The relationship was dose-dependent as obesity was greater in heavy Wp users, whereas it was similar in mild and former Wp compared with never users.[20] Similarly, BMI and weight in the current sample were greater in individuals smoked Wp longer time in the women but not in the men.

Smoking cigarettes is related to dyslipidemia, including increased Tc, LDLc, and Tg and LDLc and diminished HDLc.[38] In fact, heparin lipoprotein, hepatic lipase, cholesterol ester transfer protein, and lecithin cholesterol acyltransferase, essential for lipid metabolism, release, and function are altered with cigarette smokers.[30] This relationship is smoking duration dependent.[17] In the present study, smoking Wp was related to adverse circulatory lipid profile, including Tc, LDLc, Tc/HDLc, and LDLc/HDLc. Interestingly, these circulatory lipids were altered in the men but not in the women. These findings are of clinical significance and can be useful for the health care providers for proper management of altered lipid profile associated with cardiovascular diseases among Wp smokers.

Nicotine mobilizes free fatty acids to circulation, subsequent to the release of catecholamines, cortisol, and growth hormones. In turn, free fatty acids stimulate the production of very-low-density lipoprotein and Trg.[39] The proatherogenic response to smoking seems to contribute to the formation, and subsequent release of lipid into the circulation has also been implicated.[40] In addition, the clustering of multiple risky behaviors (ie, physical inactivity and poor diet) with smoking should not be dismissed.[41] This is particularly important given that Wp, unlike cigarette smoking, is usually practiced while sitting and around food,[42] both of which are risky behaviors associated with obesity and dyslipidemia.[9,30,31] However, these are mere postulation that require verification in future longitudinal and experimental studies.

In terms of clinical implications, smoking, obesity, and dyslipidemia are global epidemics associated with the most divesting diseases, cardiovascular, immune, respiratory, and metabolic diseases, to name a few. According to the current results, lipid profile is elevated in the men and obesity is elevated in the women smoking Wp vs never smoked, especially among the ones smoked for extended periods. Given the rapid proliferation[43] and the health consequences[8] of Wp smoking, cessation programs are desperately needed. The focus of these programs is to offset the widespread and the adverse health effect of Wp smoking, such dyslipidemia and obesity. They also should be especially designed to accommodate the unique features of Wp smoking. These features include gender differences, smoking duration, cultural significance, social environment, religious context, motivations, and intermittent use pattern.[44]

Few limitations can be realized in the present study. Importantly, causal inferences are confined to the cross-sectional design. In addition, the relatively small sample size can limit the generalizability of the study conclusions. Smoking information was self-reported, which might be associated with misreporting. However, the Arabic version of the questionnaire is validated and reliable.[2,28] Despite the fairly similar risk factors, attitudes, behaviors, and trends of Wp smoking, especially across the MENA region,[45] the study population was from Irbid, the second largest governorate in Jordan, which might constrain the generalizability of the findings. The extrapolation of the results, thus, should be with cautious in other countries/regions. Therefore, studies examining the relationship of Wp smoking with obesity and lipid profile on a larger sample from other regions/countries are warranted. In addition, objective measures for smoking information using longitudinal/experimental design are essential.

Collectively, in the present study, the relationship of Wp smoking with obesity and lipid profile is affected by gender and smoking duration. Obesity was greater in the women, whereas lipid profile was greater in the men, with longer Wp smoking use. Weight in the women smoked >20 years packed extra 10 kg as compared with never smokers and the ones smoked <10 years, while BMI was 3–4 units greater. Similarly, Tc, LDLc, Tc/HDLc, and LDLc/HDLc were ∼25%, ∼35%, ∼17%, and ∼26% greater in the men smoking Wp vs never smoked.

ACKNOWLEDGMENTS

The current manuscript was supported by Deanship of Scientific Research at Jordan University of Science and Technology, Jordan with fund no: OK/16/2016.

REFERENCES

1. Jawad M, Charide R, Waziry R, et al. The prevalence and trends of waterpipe tobacco smoking: a systematic review. PLoS One 2018;13:e0192191.
2. Khabour OF, Alzoubi KH, Eissenberg T, et al. Waterpipe tobacco and cigarette smoking among university students in Jordan. Int J Tuberc Lung Dis 2012;16:986-92.
3. Adams TN, Morris J. Smoking (tobacco). Treasure Island, FL: StatPearls; 2019.
4. Singh SK, Enzhong L, Reidpath DD, et al. Shisha (waterpipe) smoking initiation among youth in Malaysia and global perspective: a scoping review (2006-2015). Public Health 2017;144:78-85.
5. Ward KD. The waterpipe: an emerging global epidemic in need of action. Tob Control 2015;24(Suppl 1):i1-2.
6. Momenabadi V, Hossein Kaveh Ph DM, Hashemi SY, et al. Factors affecting hookah smoking trend in the society: a review article. Addict Health 2016;8:123-35.

7. Sharma E, Clark PI, Sharp KE. Understanding psychosocial aspects of waterpipe smoking among college students. Am J Health Behav 2014;38:440-7.
8. Waziry R, Jawad M, Ballout RA, et al. The effects of waterpipe tobacco smoking on health outcomes: an updated systematic review and meta-analysis. Int J Epidemiol 2017;46:32-43.
9. Vasileva LV, Marchev AS, Georgiev MI. Causes and solutions to "globesity": The new fa(s)t alarming global epidemic. Food Chem Toxicol 2018;121:173-93.
10. Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. Lancet 2016;387:1377-96.
11. Kelly T, Yang W, Chen CS, et al. Global burden of obesity in 2005 and projections to 2030. Int J Obes (Lond) 2008;32:1431-7.
12. Ford ND, Patel SA, Narayan KM. Obesity in low- and middle-income countries: burden, drivers, and emerging challenges. Annu Rev Public Health 2017;38:145-64.
13. Kopin L, Lowenstein C. Dyslipidemia. Ann Intern Med 2017;167:ITC81-96.
14. Roth GA, Fihn SD, Mokdad AH, et al. High total serum cholesterol, medication coverage and therapeutic control: an analysis of national health examination survey data from eight countries. Bull World Health Organ 2011;89:92-101.
15. Tian J, Venn A, Otahal P, et al. The association between quitting smoking and weight gain: a systematic review and meta-analysis of prospective cohort studies. Obes Rev 2015;16:883-901.
16. Clair C, Chiolero A, Faeh D, et al. Dose-dependent positive association between cigarette smoking, abdominal obesity and body fat: cross-sectional data from a population-based survey. BMC Public Health 2011;11:23.
17. Shi J, Bai Y, Qiu S, et al. Classified status of smoking and quitting has different associations with dyslipidemia in residents in northeast China, Clinica chimica acta. Int J Clin Chem 2018;486:209-13.
18. Alomari MA, Al-Sheyab NA, Ward KD. adolescent waterpipe use is associated with greater body weight: the Irbid-TRY. Subst Use Misuse 2018;53:1194-202.
19. Shafique K, Mirza SS, Mughal MK, et al. Water-pipe smoking and metabolic syndrome: a population-based study. PLoS One 2012;7:e39734.
20. Ward KD, Ahn S, Mzayek F, et al. The relationship between waterpipe smoking and body weight: population-based findings from syria. Nicotine Tob Res 2015;17:34-40.
21. Hallit S, Zoghbi M, Hallit R, et al. Effect of exclusive cigarette smoking and in combination with waterpipe smoking on lipoproteins. J Epidemiol Glob Health 2017;7:269-75.
22. Hallit S, Hallit R, Haddad C, et al. Previous, current, and cumulative dose effect of waterpipe smoking on LDL and total cholesterol. Environ Sci Pollut Res Int 2019;26:8194-201.
23. Freedman DM, Sigurdson AJ, Rajaraman P, et al. The mortality risk of smoking and obesity combined. Am J Prev Med 2006;31:355-62.
24. Parizadeh D, Momenan AA, Amouzegar A, et al. Tobacco smoking: findings from 20 years of the tehran lipid and glucose study. Int J Endocrinol Metab 2018;16:e84738.
25. A. Department of Statistics, Jordan, estimated population of the kingdom by municipality and sex, at end-year 2018. 2018. Accessed December 2018; http://dosweb.dos.gov.jo/product-category/employment/. Accessed December 2018.
26. Vandenbroucke JP, von Elm E, Altman DG, et al. Strengthening the reporting of observational studies in epidemiology (STROBE): explanation and elaboration. Epidemiology 2007;18:805-35.
27. Alomari MA, Shqair DM, Khabour OF, et al. The clinical and nonclinical values of nonexercise estimation of cardiovascular endurance in young asymptomatic individuals. ScientificWorldJournal 2012;2012:958752.
28. Primack BA, Khabour OF, Alzoubi KH, et al. The LWDS-10J: reliability and validity of the Lebanon Waterpipe Dependence Scale among university students in Jordan. Nicotine Tob Res 2014;16:915-22.
29. Lu Y, Fang J-Q, Tian L, et al., editors. Advanced Medical Statistics. 2nd ed. World Scientific Pub Co Inc; 2015.
30. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American Association of Clinical Endocrinologists and American College of Endocrinology guidelines for management of dyslipidemia and prevention of cardiovascular disease: executive summary. Complete appendix to guidelines available at http://journals.aace.com. Endocr Pract 2017;23:479-97.
31. Serra-Majem L, Bautista-Castano I. Etiology of obesity: two "key issues" and other emerging factors. Nutr Hosp 2013;28(Suppl 5):32-43.
32. Wack JT, Rodin J. Smoking and its effects on body weight and the systems of caloric regulation. Am J Clin Nutr 1982;35:366-80.
33. Munafo MR, Tilling K, Ben-Shlomo Y. Smoking status and body mass index: a longitudinal study. Nicotine Tob Res 2009;11:765-71.
34. Sneve M, Jorde R. Cross-sectional study on the relationship between body mass index and smoking, and longitudinal changes in body mass index in relation to change in smoking status: the Tromso Study. Scand J Public Health 2008;36:397-407.
35. Akbartabartoori M, Lean ME, Hankey CR. Relationships between cigarette smoking, body size and body shape. Int J Obes (Lond) 2005;29:236-43.
36. Bamia C, Trichopoulou A, Lenas D, et al. Tobacco smoking in relation to body fat mass and distribution in a general population sample. Int J Obes Relat Metab Disord 2004;28:1091-6.
37. Saarni SE, Pietilainen K, Kantonen S, et al. Association of smoking in adolescence with abdominal obesity in adulthood: a follow-up study of 5 birth cohorts of Finnish twins. Am J Public Health 2009;99:348-54.
38. Mjos OD. Lipid effects of smoking. Am Heart J 1988;115:272-5.
39. Tweed JO, Hsia SH, Lutfy K, et al. The endocrine effects of nicotine and cigarette smoke. Trends Endocrinol Metab 2012;23:334-42.
40. Yeh KW, Lee CM, Chang CJ, et al. Lipid profiles alter from pro-atherogenic into less atherogenic and proinflammatory in juvenile idiopathic arthritis patients responding to anti TNF-alpha treatment. PLoS One 2014;9:e90757.

41. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the global burden of disease study 2015. Lancet 2016;388:1659-724.
42. Mugyenyi AEK, Haberer JE, O'Neil I. Pleasure and practice: a qualitative study of the individual and social underpinnings of shisha use in cafes among youth in the UK. BMJ Open 2018;8:e018989.
43. Alomari MA, Al-sheyab NA. Dual tobacco smoking is the new trend among adolescents: update from the Irbid-TRY. J Subst Use 2017;23:92-8.
44. Ward KD, Siddiqi K, Ahluwalia JS, et al. Waterpipe tobacco smoking: The critical need for cessation treatment. Drug Alcohol Depend 2015;153:14-21.
45. Kheirallah KA, Veeranki SP, Alzyoud S, et al. Collision of waterpipe and cigarette smoking epidemics among youth in Arab countries. J Subst Use 2016;21:530-6.