THE RELATIONSHIP BETWEEN SMOKING STATUS AND METABOLIC SYNDROME: A CROSS SECTIONAL STUDY AMONG EMPLOYEES AT A UNIVERSITY IN LEBANON

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Abstract

Background:

Tobacco use is the leading cause of preventable death worldwide. The prevalence of smoking and metabolic syndrome (MetS) is increasing globally and in Lebanon. The objectives of this study are to assess the prevalence of tobacco use (waterpipe, cigarette, cigar, pipe, smokeless tobacco products) and to examine the independent associations between tobacco and MetS/ MetS components (low high-density lipoprotein, elevated triglyceride, hypertension, impaired fasting glucose and abdominal obesity) among a sample of Lebanese adults.

Methods:

A cross-sectional study was carried out on Notre Dame University (NDU) employees. A total of 316 participants aged between 20 and 74 years old were included. Sociodemographic and lifestyle characteristics, including smoking status, were gathered via a questionnaire. Anthropometric and biochemical measures were also assessed. MetS was defined according to the third Report of the National Cholesterol Education Program (NCEP- ATP III). The data were analyzed using the SPSS version 22. P < 0.05 was considered statistically significant. Logistic regression was used to examine the association between smoking and MetS/ MetS components. The prevalence of MetS was found to be significantly higher among smokers (63.2%), as compared to former smokers (5.9%) and people who never smoked (30.9%), p=0.002. Among MetS components, the prevalence of high blood pressure, high triglyceride levels, low high-density lipoprotein levels and abdominal obesity was higher among current smokers (53.2%, 56.6%, 63.7% and 56.2%, respectively) as compared to former smokers (6.5%, 6.1%, 3.9% and 3.2%, respectively) and people who never smoked (40.3%, 37.4%, 32.5% and 40.6%, respectively). After controlling for the effects of age, gender, income, level of education, physical activity level, marital status, clinical diagnosis of hypertension, diabetes and chronic conditions other than, diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, family history of diabetes, hypertension, obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, body fat percentage (BF %) and CRP, smoking was significantly associated with MetS (OR=4.12; 95% CI: [1.52-11.56], p=0.006), low high-density lipoprotein cholesterol (OR=2.56; 95% CI: [1.29-5.19], p=0.007), hypertriglyceridemia (OR=15.72; 95% CI: [1.07-231.19], p=0.045), and abdominal obesity (OR=2.63; 95% CI: [1.15-6.01], p=0.022).

Conclusion:

Smoking was found to be significantly associated with increased risk of metabolic syndrome and some of its components (low high-density lipoprotein, high triglyceride and abdominal obesity). However, no significant association was found between smoking and hypertension or impaired fasting glucose.

Introduction:

Globally, 8 million deaths are associated with tobacco smoking each year, with more than 7 million deaths associated with direct tobacco use and around 1.2 million deaths associated with second-hand smoking (WHO, 2019). Tobacco use is considered to be one of the top three causes of mortality and morbidity around the world, making it a global health epidemic (Lim SS et al., 2012). In 2015, more than 1.1 billion people smoked tobacco with much more males than females (WHO, 2015). Even though the prevalence of tobacco smoking is decreasing in many developed countries due to increased awareness on the effects of tobacco, it seems to be increasing in Eastern Mediterranean and African countries, with Lebanon being ranked among the top 10 countries with the highest smoking rates (WHO, 2015). Tobacco use is the leading cause of preventable death worldwide and is responsible for the economic costs of hundreds of billions of dollars each year (Belin I et al., 2012). The causes of death due to tobacco are mainly related to cancer, cardiovascular diseases and chronic obstructive pulmonary disease (Ezzati M & Lopez AD, 2000). Tobacco is consumed in many forms including cigarettes, waterpipe or hookah, cigars, pipes and smokeless tobacco products, with all forms being harmful regardless of the level of exposure. Tobacco prevalence is affected by many factors including the socioeconomic factors, with more than 80% of smokers living in low- to middle- income countries, and culture where smoking is part of social acceptance especially for males (WHO, 2020). The prevalence of smoking tends to be higher in Southeast Asian countries and Eastern Europe while it is lower in Western Europe and America. Serious actions have been applied to decrease the use of tobacco products around the world and smoking rate has decreased in some countries such as the United Kingdom where smoking prevalence

has decreased from 38% to 19.5%. However, the prevalence of smoking is still high in some countries including Lebanon which has a smoking prevalence rate of 42.6%, with higher percentage of male smokers than female smokers (WHO, 2019).

Studies have shown that smoking increases the risks of type 2 diabetes mellitus, cardiovascular disease, chronic obstructive pulmonary disease, and different types of cancer such as lung, colorectal, prostate and breast cancer (Nakanishi N et al., 2005; Taghizadeh N et al., 2016; Yeh HC et al., 2010, El-zaatari ZM et al., 2015; Mohammad Y et al., 2008; El-Setouhy M et al., 2009). This is due to the amount of toxic and carcinogenic substances found in tobacco, which are directly absorbed by the mucosal tissues in the mouth and lungs, entering the blood circulation, and then into all body organs and tissues (American Cancer Society, 2014). A growing body of evidence is suggesting an association between tobacco use and metabolic syndrome (MetS) (Sun K et al., 2012). MetS is a cluster of metabolic abnormalities, including abdominal obesity, dyslipidemia, hypertension and glucose intolerance, that increase the risk of developing cardiovascular disease and type 2 diabetes (Calo W et al., 2013). MetS is associated with increasing the risk of type 2 diabetes by 3 to 4 times and the risk of cardiovascular disease by 1.4-fold (Ford ES et al., 2008; Mottilo S et al., 2010). Several potential mechanisms that lie behind the association between smoking and MetS have been suggested. One of these pathways might be the negative effect of smoking on insulin resistance (Chiolero A et al., 2008). Other suggestions include that smoking might lead to lipoproteins metabolism alteration and endothelial dysfunction (Jia WP, 2013). The International Diabetes Federation (IDF) estimates that one-quarter of the world's population has metabolic syndrome (Alberti KGMM et al., 2009). Lebanon has one of the highest prevalence rates of metabolic

syndrome (31.2%) among adults in the region (Sibai A et al., 2008). Several epidemiological studies have suggested an association between smoking and metabolic syndrome (MetS), however these studies had inconsistent and controversial results (Sun K et al., 2012). A cross-sectional study, which used data collected in the Korean National Health and Nutrition Examination Surveys, was done on 11559 participants (5358 men and 6201 women) aged 19 years old and older to assess the association between cotinineverified smoking status and MetS. This study showed that the risks of MetS and high triglyceride (TG) level were significantly higher in cotinine-verified smokers than in nonsmokers in both men and women (Mets: OR men=1.26; 95% CI: [1.04-1.53], p=0.02) and OR women = 1.32; 95% CI: [1.01–1.73] p=0.05) (TG: OR men =1.30; 95% CI: [1.12–1.51], p<0.01) and OR women =1.45; 95% CI: [1.13–1.85], p<0.01). The risk of abdominal obesity was significantly higher in women smokers compared to women non-smoker, but not significantly higher in men smokers compared to men non-smokers (abdominal obesity: OR men =1.52; 95% CI: [1.03–2.23], p=0.07 and OR women =1.25; 95% CI: [0.99–1.58] respectively, p=0.03) after adjusting for age, body mass index, marital state, education, alcohol intake, physical activity, total fat intake, and total fiber intake. In addition, men smokers had a higher risk of low HDL cholesterol than men non-smokers, (OR = 1.40; 95% CI: [1.16–1.68], p<0.01) and a lower risk of high blood pressure (OR=0.81; 95% CI: [0.68– (0.96], p<0.01), however these association were insignificant among women smokers compared to women non-smokers (OR= 1.16; 95% CI: [0.95-1.42], p=0.15 and OR= 0.83 95% CI: [0.63-1.09], p=0.18 for HDL and blood pressure respectively). No significant association was found between smoking and impaired fasting glucose in both men and women (OR men = 1.00; 95% CI: [0.84–1.20], p=0.97 and OR women = 1.00; 95% CI: [0.77–

1.31], p=0.98. However, smokers in this study used only cigarettes as tobacco products thus, findings cannot be generalized to smokers of other tobacco products. (Kang JH and Song YM, 2015). Other studies were done on only one tobacco product as well. An example of this is cross-sectional study done on 856 individuals aged between 21 and 79 years old from Puerto Rico showed that the prevalence of MetS was significantly higher in former cigarette smokers (48.4%) than in current cigarette (42.7%) and never smokers (40.0%), p<0.001. After adjusting for age, sex, education, health insurance coverage, alcohol consumption, physical activity and BMI, the prevalence of MetS was higher in both current and former cigarette smokers compared to never smokers (OR=2.24; 95% CI: [1.00-4.99] and OR=1.36; 95% CI: [0.88-2.10], respectively). No significant associations were found between smoking and each of the metabolic syndrome components except for TG and HDL levels; higher TG levels and lower HDL cholesterol levels were found among the group of current heavy smokers (≥ 20 cigarettes per day) compared to non-smokers. $(OR_{TG}=2.22; 95\% \text{ CI: } [1.12-4.38] \text{ and } OR_{HDL}=2.49; 95\% \text{ CI: } [1.28-4.86], \text{ respectively})$ (Calo W et al.; 2013). A cross-sectional study done on 2032 healthy individuals aged between 30 to 75 years (51.1% males and 48.9% females) living in rural region in Pakistan to examine the association between metabolic syndrome and waterpipe smoking in the total sample and different sex groups. After adjusting for age, the prevalence of metabolic syndrome was significantly higher among current waterpipe smokers (33.1%) compared with non-smokers (14.8%), p<0.01. After adjusting for age, sex and social class, waterpipe smokers were three times more likely to have metabolic syndrome than non-smokers (OR: 3.21 (95% CI: [2.38-4.33]). Waterpipe smokers were significantly more likely to have hypertriglyceridemia (OR=1.63, 95% CI: [1.25-2.10]), hyperglycemia (OR=1.82, 95% CI:

[1.37-2.41]), hypertension (OR=1.95, 95% CI: [1.51-2.51]) and abdominal obesity (OR=1.93, 95% CI: [1.52-2.45]) than non-smokers, with no significant differences in HDL level found between waterpipe smokers and non-smokers. In addition, sex-stratified analysis showed that male waterpipe smokers were significantly more likely to have a low HDL level (OR=1.75, 95% CI: [1.11-2.78]), hypertriglyceridemia (OR=1.60, 95% CI: [1.06-2.41]) and hyperglycemia (OR=1.88, 95% CI: [1.22-2.89]) than male non-smokers, while no differences in hypertension and central obesity were seen between the two male groups. Female waterpipe smokers were more likely to have hypertriglyceridemia, hypertension and obesity than female non-smokers, while no differences in HDL levels and glucose levels were seen between the two male groups. The first limitation of this study is that they used only waterpipe as tobacco product. The second limitation is that the study included participants from only one province of Pakistan so the findings of the study cannot be generalized. The third limitation is that the lack of evaluation of dietary regimen and physical activity may have confounded the relationship between waterpipe smoking and metabolic syndrome. (Shafique K et al., 2012). A study was done on 9840 Iranian participants aged between 35 and 65 years from the Mashhad stroke and heart atherosclerotic disorder (MASHAD) cohort study to investigate the effects of both cigarette and waterpipe smoking on cardiovascular disease development and biochemical measurements. Participants were divided into five groups: non-smokers (n=6742), exsmokers (n=976), cigarette smokers (n=864), waterpipe smokers (n=1067), and combined cigarette and waterpipe smokers (n=41). After adjusting for age and sex, the prevalence of metabolic syndrome was significantly the highest among waterpipe smokers as compared to combined waterpipe and cigarette smokers, non-smokers and cigarettes smokers (46.8%,

34.1%, 38.8% and 26.2%, respectively, p<0.001). After multivariate analysis, the presence of metabolic syndrome was significantly lower among cigarette smokers than non-smokers (OR=0.82; 95% CI: [0.69-0.97], p<0.05) yet significantly higher among waterpipe smokers than non-smokers (OR=1.29; 95% CI: [1.12-1.48], p<0.001)). The major limitation of this study was that only age and sex were adjusted for in the multivariate analyses. Another limitation was that hypertension (one of the metabolic syndrome components) was not studied. (Sofflaei S et al., 2018). Other studies showed an association between smoking and MetS but participants of these studies were only males, thus limiting the generalizability of the study findings to females. A cross-sectional study done on 6032 men aged more than 19 years old from the Korea National Health and Nutrition Examination Survey (KNHANES) showed that the risks of MetS, lower HDL cholesterol and higher triglyceride levels increased with the increase of tobacco use in current smokers (Mets: RR 10 to 20 packs per year =1.36; 95% CI: [1.06-1.74], RR 20 to 30 packs per year =1.69; 95% CI: [1.30-2.19] and RR $_{>30 \text{ packs per year}}$ =1.79; 95% CI: [1.39-2.29]); (HDL: RR $_{\leq 10 \text{ packs per year}}$ =1.33;95% CI:[1.06-1.67], RR 10 to 20 packs per year =1.35; 95% CI:[1.09-1.68], RR 20 to 30 packs per year =1.44; 95% CI: [1.14-1.82] and RR > 30 packs per year =1.39; 95% CI: [1.11-1.75]); (TG: RR ≤ 10 packs per year = 1.32; 95% CI:[1.06-1.64], RR 10 to 20 packs per year = 1.78; 95% CI: [1.44-2.19], RR_{20 to 30 packs per year} =1.75; 95% CI: [1.39-2.20] and RR_{> 30 packs per year} =2.05; 95% CI: [1.63-2.57] (Shin HS et al., 2018). As for the other components of Mets, the risk was dependent on the number of packs smoked per year. For of impaired fasting glucose, the risk was significantly higher among participants who smoke more than 30 packs per year (RR= 1.33; 95% CI: [1.07-1.67]) and for hypertension, the risk was significantly lower among participants who smoke less than 10 packs per year (RR = 0.74; 95% CI: [0.600.92]). In former smokers, the risk of MetS, higher waist circumference, higher triglyceride and higher fasting blood glucose was significantly higher among participants who smoke more than 30 packs per year (Mets: RR > 30 packs per year =1.72; 95% CI: [1.29-2.29]); WC: RR>30 packs per year =1.69; 95% CI: [1.20-2.37]; (TG: RR>30 packs per year =1.56; 95% CI: [1.18-2.05]; FBG: RR> 30 packs per year =1.52; 95% CI: [1.17-1.98]). The increased risk of higher blood pressure was only significant among former smokers who smoked 10 packs or less per year (BP: RR> 30 packs per year =1.34; 95% CI: [1.10-1.61]). No significant association was found between former smokers and higher fasting blood glucose regardless of the number of packs per year. In addition, the risk of MetS increased with the increase of tobacco use in former smokers with under 20 years of smoking cessation (RR=1.49; 95% CI: [1.03-2.16], p<0.05 for smoking cessation less than 20 years). (Shin HS et al., 2018). Another cross-sectional study was done on 1215 male Japanese workers aged between 20 and 67 years old. Crude analysis did not show an association between smoking and MetS, however after adjusting for age, previous coronary artery disease, exercise, insomnia and stress perception, smoking was found to be associated with increased odds of MetS (OR=1.4; 95% CI: [1.1-2.1]), waist circumference (OR=1.5; 95% CI: [1.2-1.9]), high triglyceride level (OR=1.9; 95% CI: [1.4-2.4]) and low HDL cholesterol (OR=1.7; 95% CI: [1.1-2.7]). (Takeuchi T et al., 2009). A cohort study was done on 4542 male participants from South Korea with a mean age of 42 years old. Participants were followed up for an average period of 2.9 years. The prevalence of MetS was 8% in non-smokers, 7.1% in new smokers (who never smoked at baseline but were currently smoking at follow up), 17.1% in ex-smokers (who smoked at baseline but quit smoking by follow up) and 13.9% in sustained smokers (smokers who smoked continuously from baseline to follow up) with a p value of less than 0.001. After adjusting for age, baseline weight, alcohol consumption, exercise, and baseline number of metabolic syndrome components, sustained and ex- smoking were significantly associated with Mets (OR=1.68; 95% CI: [1.33-2.12] and OR=2.43; 95% CI: [1.80-3.29], respectively) as compared to non-smokers. In addition, after adjustment for the abovementioned confounding factors and for LDL, CRP, uric acid, homeostasis model assessment of insulin resistance, ex-smokers had significantly increased odds of having MetS compared to sustained smokers (OR=1.45; 95% CI: [1.06-1.98] and OR=1.44; 95% CI: 1.04-2.00] respectively), however, after adjusting for weight change, this association was no longer significant (OR=1.22; 95% CI: [0.89-1.68]. (Kim BJ et al., 2009). One crosssectional study done on 5913 participants from the Multi-Ethnic Study of Atherosclerosis (MESA) aged between 45 and 84 years old showed that smoking was significantly associated with low HDL cholesterol and high triglyceride level, with and without adjustment for BMI (HDL: OR adjusted =1.3; 95% CI: [1.2-1.6], p=0.001 and OR unadjusted =1.5; 95% CI: [1.3-1.8], p<0.001; TG: OR adjusted =1.1; 95% CI: [1.1-1.6], p=0.009 and OR unadjusted =1.4; 95% CI: [1.2-1.7], p<0.001). On the other hand, a significant positive association between smoking and MetS and waist circumference (WC) was found only after adjustment for BMI (Mets: OR adjusted=1.4; 95% CI: [1.1-1.7], p=0.03 vs. OR unadjusted =1.0; 95% CI: [0.8-1.1]; WC: (OR adjusted =1.6; 95% CI: [1.2-2.1], p=0.004 vs. OR unadjusted =1.1; 95% CI: [0.9-1.3].) and no significant association was found between smoking and elevated blood pressure and impaired fasting glucose, with and without BMI adjustment. One limitation of this study was that the study population was limited to men and women aged between 45 to 84 years old which limits the generalizability of the findings to people younger than 45 because people older than 45 years old are at higher risk of developing

MetS. (Berlin I et al., 2012). Several other studies have shown an association between smoking and MetS but did not adjust for all possibly relevant risk factors that may have influenced the results. One study done on 24389 men and 35078 women aged between 18 and 80 years old who participated in the LifeLines cohort study in Netherland, showed an association between current smoking and a higher risk of having MetS, low HDL level, high TG level and large WC in both men and women, independent of age and BMI (p<0.001) No significant association was found between smoking and impaired fasting glucose and hypertension. However, all models were only adjusted for age and stratified by sex and BMI. (Slagter S et al., 2013). In Lebanon, only one retrospective cross-sectional study was done to assess the association of waterpipe smoking with the reason for catheterization (myocardial infraction, unstable angina and presence of ischemia during exercise stress testing) among Lebanese catheterized patients-, as well as explore its association with determinants of metabolic syndrome, specifically, diabetes, hypertension and hyperlipidemia. A total of 7,594 Lebanese patients (67% males) undergoing cardiac catheterization were enrolled in this study. After adjusting for age and sex, of the three studied metabolic syndrome conditions, only diabetes showed a significant positive association with waterpipe smoking among patients with MI (OR=1.66, 95%CI: [1.04– 2.63]; p=0.032). A major limitation of this study was that it was done on patients who were undergoing cardiac catherization thus findings cannot be generalized to the general population. (Platt D et al., 2017). One cohort study reported a protective effect of cigarette smoking against MetS. This study was done on 3385 participants in Turkey (1674 men and 1711 women), aged 28 years and older. After adjustment for age, baseline family income and physical activity level, smoking was found to be significantly negatively associated

with MetS in women (RR=0.50; 95% CI: [0.26-0.94]) (Onat A et al., 2007). In this study, smokers had smaller waist circumference (WC= 89.6±0.43 in smokers vs 93.9±0.3 in never smokers, p < 0.005) which might have caused the lower incidence of MetS among the group of smokers. In addition, this study was done on one type of tobacco product (cigarette smoking) which may limit generalization of the study findings to populations who use different types of tobacco products. Another example of this is a cohort study done in Slovakia on 125 participants (75 men and 50 women) with an average age of 57.3 years showed an inverse association between smoking and MetS. Participants were divided into two groups according to their smoking status (smokers (n=59) and non-smokers (n=66)) and were followed for 5 years. Lower incidence of MetS was found in the smokers' group (40.7% of smokers had MetS versus 68.2% of non-smokers had MetS. The lower prevalence of MetS among smokers was related to the fact that smokers had smaller waist circumference and lower levels of arterial blood pressure. Waist circumference was smaller among male smokers compared to male non-smokers by 5.2 cm and smaller among female smokers compared to female non-smokers by 3.3 cm. The incidence of arterial hypertension among smokers was 62.7% and among non-smokers 68.2%. The incidence of lower values of HDL cholesterol was 36% in men smokers compared to 42.3% in men non-smokers and 50% in women smokers compared to 20% in women non-smokers. The incidence of triglycerides was the same among the smokers and non-smokers groups. Smokers had a high percentage of impaired fasting glucose (66.1%) (Dedinska I et al., 2014). In this study, smokers also had smaller waist circumference which might have caused the lower prevalence of MetS among this group. When analyzing the results, there was no control for any possible confounding factor. In addition, this study was done on one

type of tobacco product (cigarette smoking) which may limit generalization of the study findings to other populations who use different types of tobacco products.

Given that 1) Lebanon has one of the highest prevalence rates of smoking and metabolic syndrome and 2) lack of /scarcity of evidence on the association between smoking in general and metabolic syndrome in healthy adults 3) characterization of the existing literature with major limitations such as lack of evaluation of major confounders in the relationship between smoking and metabolic syndrome and restriction of study participation to a particular subgroup of the population (particular sex group, patients,...), limiting the generalizability of the study findings , it becomes important to conduct a study to address the existing gaps in the literature. Among a sample of Notre Dame employees, the objectives of our study are 1) to assess the prevalence of smoking (waterpipe, cigarette, cigar, pipe) in the total sample 2) to examine the independent associations between smoking (waterpipe, cigarette, cigar, pipe) and MetS/ MetS components.

Methods:

Study design and recruitment methods

A cross-sectional study was carried out on Notre Dame University (NDU) employees at the main campus (Zouk Mosbeh) and its two regional campuses (North, and Shouf). The protocol was approved by the Institutional Review Board of NDU and the study was done in accordance with the ethical standards placed in the 1964 Declaration of Helsinki and its later amendments. At the beginning, all NDU employees (N=600) were invited to join the study by email. Those who showed interest in participation (n=360) were then contacted by the study researchers to schedule appointments with them. Nutritionists then visited those employees in their offices to screen them for eligibility. Employees were excluded if they were pregnant, lactating, or have a pacemaker or metal pieces in their body. Employees who were found to be eligible were then asked to read and sign a consent form. A total of 316 participants aged between 20 and 74 years old were included in the study. Participants were interviewed to complete the study questionnaires in a 30 minutes faceto-face interview. At the end of the interview, participants were given appointments to come to the Nutrition lab at the campus where the employee works for anthropometric measurements, body composition assessment and a blood draw. Participants were instructed to avoid food and drink after 12 midnight (NPO after 12 midnight).

Data Collection

Socio-demographic and lifestyle variables

The interview involved completion of two questionnaires: a background questionnaire and the short-form of the International Physical Activity Questionnaire (IPAQ). An identification number was assigned for each participant at the beginning of the interview which was used to label the participant's questionnaire, blood sample and blood test results.

The background questionnaire was divided into three parts. The first part included questions on socio-demographic characteristics such as age, gender, marital status, education level, and income. The second part included questions on lifestyle habits: Smoking status was determined by: "How often do you smoke cigarettes?" (daily, occasional, former daily, former occasional, never smoked), "How often do you smoke waterpipe?" (daily, occasional, former daily, former occasional, never smoked), "How often do you smoke cigar?" (daily, occasional, former daily, former occasional, never smoked), "How often do you use smokeless tobacco products (chewing gums, pouches...)" (daily, occasional, former daily, former occasional, never smoked), "do you consider yourself to be a passive smoker at work?" (Yes, No), "do you consider yourself to be a passive smoker at work?" (Yes, No). The third part included questions on health status such as existing medical conditions and intake of medications.

Physical activity (PA) level of participants was assessed using the IPAQ-Short Form, a 7item self-administered questionnaire that is used to assess PA level among adults (Canadian Nutrient File, 2018). IPAQ include three specific types of activities: walking, moderate and vigorous physical activities and time spent by an individual (Booth M, 2000). Separate scores were provided for each of these activities. Four continuous scores were calculated using the following values: Walking = 3.3 METs, Moderate PA = 4.0 METs and Vigorous PA = 8.0 METs. These scores were then added to calculate the total physical activity score. Low-level, moderate-level and high-level PA were defined by scores of less than 600 MET-minutes per week, between 600 to less than 3000 MET-minutes per week, and of 3000 or more MET-minutes per week, respectively.

Anthropometric measurements and body composition assessment

Participants' anthropometric measurements (height, waist circumference and body composition assessment) and blood draws were performed at the nutrition laboratory at each campus after an overnight fast. Height was measured by nutritionists to the nearest

0.1 cm according to the following protocol: no shoes, heels together and head touching the stadiometer's ruler aligned horizontally. Waist circumference was also measured by nutritionists with a non-stretchable tailor measuring tape placed around the bare abdomen just above the hip bone and parallel to the floor. Participants were asked to exhale, and measurement was taken to the nearest centimeter at the midpoint between the bottom of the rib cage and above the top of the iliac crest during minimal respiration (Nishida C et al., 2008). Waist circumference values were classified as high risk using the World Health Organization (WHO) cutoffs for men > 102 cm and women > 88 cm (WHO, 2008). Weight and body composition were obtained using the bioelectrical impedance analysis (BIA) machine InBody 720 (Biospace, Seoul, Korea). BIA is commonly used in research as it is quick, safe, and inexpensive (Shantavasincul PC et al., 2015). BIA works by impedance and measures body water, and then estimates fat mass and fat-free mass (Baracos V et al., 2012). The BIA machine was transported to different campuses for data collection and it was calibrated prior to its use. Participants were asked to arrive on an empty bladder and stomach. Prior to use of BIA, subjects were asked to wipe the palm of their hands and soles using a specific Biospace Electrolyte tissue to increase electrical conductivity. The participants were asked to stand on the machine barefooted, without wearing any metal or jewelry. Body Mass Index (BMI) was calculated based on the measured weight and height as: Weight (kg)/ Height (m2). Underweight was defined as BMI < 18.5 kg/m2, normal weight: 18.5-24.9 kg/m2, overweight: 25-29.9 kg/m2, and obese $\geq 30 \text{ kg/m2}$ (WHO, 2008).

Metabolic Syndrome and its components

During the visit to the Nutrition lab, blood pressure (BP) was measured by a nurse according to the National High Blood Pressure Education Program's guidance on optimal BP measurement techniques. Each patient was seated comfortably, with back supported, legs uncrossed, and upper arm bared and his/her arm supported at heart level. Cuff bladder encircled 80% or more of the patient's arm circumference. Mercury column was deflated at 2 to 3 mm per second. The first and fifth audible Korotkoff sounds were recorded as systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. Three BP measurements were obtained at an interval of ten minutes each, the measurements were given to the nearest 2 mmHg and the mean was calculated. The nurse then collected a fasting sample of blood for assessment of triglyceride (TG), high density lipoprotein and fasting blood glucose (FBG). Samples collected at the regional campuses were transported to the Zouk Mosbeh campus on ice on a daily basis. They were stored in the Biology lab at the main campus at -20 °C for a maximum period of 6 weeks before analysis. Serum TG, HDL and FBG were measured using a dry chemistry analyzer Vitros 250 (Ortho Clinical Diagnostic, Raritan, New Jersey, USA) available at the same laboratory.

The definition of the Third Report of the National Cholesterol Education Program (NCEP-ATP III) was used to identify individuals who had metabolic syndrome. Participants who met three or more of the following criteria were considered as having metabolic syndrome: WC ≥ 102 cm in men or ≥ 88 cm in women, serum triglyceride level ≥ 150 mg/dL, HDLcholesterol levels< 40 mg/dL in men or < 50 mg/ dL in women, FBG ≥ 100 mg/dL or on antidiabetic treatment and BP $\geq 130/85$ mmHg or on anti-hypertensive medication (Parikh R & Mohan V, 2012)

Pilot testing

The questionnaires were pre-tested during the month of September 2016. The draft questionnaires were tried out on a random sample of 30 NDU staff and faculty members (14% of the calculated sample size). Pilot testing was performed to measure how much time it takes to complete each questionnaire, and clarify question wording, or response categories when necessary and then questionnaires were revised as needed prior to the launching of the study. This sample was not included in data analyses.

Statistical analysis

Assuming that the prevalence rate of metabolic syndrome in Lebanese adults was 31%, the sample size was calculated to be 316 individuals, with a power of 87% using G*Power Version 3.1.3.2 software (Program written, concept and design by Franz, Universitat Kiel, Germany) (Sibai A et al., 2008; Faul F et al., 2007). Quantitative and qualitative measurements were summarized as mean ± standard deviation and n (%), respectively. Participant characteristics were compared according to metabolic syndrome status and smoking status using chi-square/Fisher's Exact tests for categorical variables and Independent two-sample t, ANOVA–/Mann-Whitney U test, Kruskal Wallis test for continuous variables. Binary logistic regression analyses were used to examine the independent associations between smoking and metabolic syndrome/MeTs components (Hypertriglyceridemia, low HDL levels, risky WC, hypertension and impaired fasting glucose). In addition to age and gender, independent variables with a p-value < 0.05 in the bivariate analysis involving MeTs' component as the dependent variable were included in the logistic regression model. Gender- smoking status interaction terms were included in

the logistic regression model. The data were analyzed using the Statistical Package for Social Sciences software (SPSS) version 22. A P-value of less than 0.05 was considered an indication of statistical significance.

Results:

There was a total of 316 study participants with a mean age of 42.37 ± 11.45 years. The majority of the study participants were females (51.3%), married (65.2%), lived in urban areas (61.7%), had children (61.1%), with a mean number of 4.87 ± 3.37 children, had a university degree (78.7%) with a monthly income of at least 1250\$ (67.0%), never smoked or were former smokers (51.9%), were not passive smokers (70.6%), did not drink alcohol (74.4%), did not have family history of diabetes (59.2%), obesity (95.3%), heart disease (63.6%), hypercholesterolemia (63.6%) and hypertriglyceridemia (98.1%). Therefore and as expected the majority of the study participants had normal creatinine levels (0.81 mg /dl), did not have recent diagnosis of chronic diseases (60.8%) and take medications (69.6%), were found not to have metabolic syndrome (78.5%), high blood pressure (80.4%), impaired fasting blood glucose levels (82%), hypertriglyceridemia (68.7%), low HDL levels (75.6%), abdominal obesity (50.9%), high cholesterol levels (64.9%). However, the majority of the study participants were found to be overweight/obese with low physical activity level (64.6%), high LDL (62.3%) and CRP levels (58.9%).

Daily smokers were found to be significantly older than occasional smokers and people who never smoked (Daily smokers: 45.97 ± 11.29 vs occasional smokers: 39.65 ± 9.24 vs people who never smoked: 41.22 ± 11.84 , p daily vs occasional=0.004, p daily vs people who never smoked=0.010). A significantly higher percentage of males (M: 55.2% vs F:

41.7%, p=0.045, individuals who are married (married: 51.4% vs single: 41.8%, p=0.028) and have children (Y: 53.9% vs N: 39.0%, p=0.002) reported to be daily and occasional smokers, with occasional smokers and people who never smoked reporting to have greater number of children than daily smokers (daily: 3.76±2.90 vs occasional: 5.18±3.48 vs people who never smoked: 5.35 ± 3.48 , p daily vs occasional =0.044, p daily vs people who never smoked =0.002). A significantly higher percentage of individuals who have less than high school degree and high school degree (less than high school: 66.7% vs high school: 73.5% vs undergraduate: 48.2% vs graduate: 38.6%, p=0.001), drink alcohol (Y: 63.0% vs N: 43.0%, p=0.011), had a recent diagnosis of cardiovascular disease (Y: 83.3 vs N: 47.4%, p=0.001), hypertension (Y: 51.3% vs N: 47.7%, p=0.049), chronic conditions other than, diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, (Y: 64.9% vs N:44.4%, p=0.022) reported to be daily and occasional smokers. In addition, a significantly higher percentage of individuals who were found to have metabolic syndrome (Y: 63.2% vs N: 43.9%, p=0.002), high blood pressure (Y: 53.2% vs N: 46.8%, p=0.026), high triglyceride levels (Y: 56.6% vs N: 44.2%, p=0.004), low HDL levels (low: 63.7% vs normal: 43.1%, p=0.003), abdominal obesity (Y: 56.2% vs N: 40.4%, p=0.022) and high CRP levels (high: 54.3% vs moderate: 39.3%, p= 0.022) reported to be daily and occasional smokers (Table 1).

There was a total of 68 out of 316 participants with metabolic syndrome (MeTs) who were significantly older thank those without MeTs (Y: 49.06 ± 9.11 vs N: 40.54 ± 11.36 , p=0.000). A significantly higher percentage of individuals with MeTs were males (Y: 72.1% vs N: 42.3%, p=0.000), had less than high school or high school degree (Y: 39.7% vs N: 16.1%,

p=0.000) with a monthly income of less than 1250\$ (Y: 50.0% vs N: 28.2%, p=0.003). As expected, A significantly higher percentage of individuals with MeTs had a recent diagnosis of chronic diseases (Y: 63.2% vs N: 32.7%, p=0.000), diabetes (Y: 16.2% vs N: 2.4%, p= 0.000) and hypertension (Y: 22.1% vs N: 8.9%, p= 0.005), were on medications for chronic conditions (Y: 50.0% vs N: 25.0%, p=0.000) and had a family history of diabetes (Y: 55.9% vs N: 36.7%, p=0.007). In addition, a significantly higher percentage of individuals with MeTs were found to have abdominal obesity (Y: 92.6% vs N: 37.1%, p=0.000), obesity (Y: 63.2% vs N: 13.7%, p=0.000), higher percentage of body fat (Y: 34.81 \pm 7.16 vs N: 29.67 \pm 7.84, p= 0.000), higher mean creatinine levels (Y: 0.87 \pm 0.36 vs N: 0.80 \pm 0.48, p=0.001) and high CRP levels (high: 83.8% vs moderate: 52.0%, p=0.000).

There was a total of 62 out 316 participants with high blood pressure who were significantly older than those without raised blood pressure (Y: 50.19 ± 10.51 vs N: 40.46 ± 10.86 , p=0.000). A significantly higher percentage of Individuals with raised blood pressure were males (Y: 74.2% vs N: 42.5 %, p=0.000), married (married: 79.0% vs N: 61.8%, p=0.016), had less than high school or high school degree (Y: 37.1% vs N: 17.3%, p=0.009). As predicted, A significantly higher percentage of individuals with raised blood pressure had a recent diagnosis of chronic diseases (Y: 61.3% vs N: 33.9%, p= 0.000), diabetes (Y: 12.9% vs N: 3.5%, p= 0.008) and hypertension (Y: 32.3% vs N: 6.7%, p= 0.000), were on medications for chronic conditions (Y: 50.0% vs N: 25.6%, p=0.000), were found to have abdominal obesity (Y: 69.4% vs N: 44.1%, p=0.001), obesity (Y: 50.0% vs N: 18.1%, p=0.000), higher percentage of body fat (Y: 32.88\pm8.33 vs N: 30.26\pm7.81, p= 0.021) and higher mean creatinine levels (Y: 0.83\pm0.18 vs N: 0.81\pm0.51, p=0.039).

There was a total of 57 out of 316 participants with elevated blood glucose level who were significantly older than those without elevated blood glucose level (Y: 50.54 ± 11.05 vs 40.58 ± 10.75 , p=0.000). A significantly higher percentage of individuals with elevated blood glucose level were males (Y: 75.4% vs N: 42.9%, p=0.000), had less than high school or high school degree (Y: 29.8% vs N: 19.3%, p=0.045) with a monthly income of less than 1250\$ (Y: 45.6% vs N: 30.1%, p=0.010). As anticipated, A significantly higher percentage of individuals with elevated blood glucose level had a recent diagnosis of chronic diseases (Y: 71.9% vs N: 32.0%, p=0.000), diabetes (Y: 24.6% vs N: 1.2%, p= 0.000) and hypertension (Y: 28.1% vs N: 8.1%, p= 0.000), chronic diseases other than, diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, (Y: 31.6% vs N: 15.1%, p=(0.006), were on medications on chronic conditions (Y: 61.4% vs N: 23.6%, p= 0.000), were found to have abdominal obesity (Y: 78.9% vs N: 42.5%, p=0.000), overweight/obesity (Y: 91.2% vs N: 54.4%, p=0.000), higher percentage of body fat (Y: 33.41 ± 81.84 vs N: 30.19±7.66, p= 0.006), higher mean creatinine levels (Y: 0.91±0.39 vs N: 0.79±0.47, p=0.000) and high CRP levels (high: 73.3% vs moderate: 55.6%, p=0.018).

There was a total of 77 out of 316 participants with low HDL level. A significantly higher percentage of individuals with low HDL levels had less than high school or high school degree (Y: 29.9% vs N: 18.4%, p=0.025), a family history of obesity (Y: 10.4% vs N: 2.9%, p= 0.013), were found to have abdominal obesity (Y: 62.3% vs N: 44.8%, p= 0.011), obesity (Y: 36.4% vs N: 20.5%, p= 0.019) and high CRP levels (Y: 77.9% vs N: 52.7%, p= 0.000).

There was a total of 99 out of 316 participants with elevated triglyceride level who were significantly older than those without elevated triglyceride level (Y: 48.06 ± 10.72 vs N: 39.78 ± 10.83 , p=0.000). A significantly higher percentage of individuals with elevated triglyceride level were males (Y: 74.7% vs N: 36.9%, p=0.000), had less than high school or high school degree (Y: 32.3% vs N: 16.1%, p=0.000) with a monthly income of less than 1250\$ (Y: 45.5% vs N: 27.2%, p=0.001). As expected, a significantly higher percentage of individuals with elevated triglyceride level had a recent diagnosis of chronic diseases (Y: 58.6% vs N: 30.4%, p= 0.000), diabetes (Y: 10.1% vs N: 3.2%, p=0.025), hypertension (Y: 20.2% vs N: 7.8%, p=0.003), and chronic diseases other than, diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, (Y: 27.3 vs N: 13.8%, p=0.006), had a family history of diabetes (Y: 50.5% vs N: 36.4%, p=0.025), obesity (Y: 9.1% vs N: 2.8%, p=0.021) and heart disease (Y: 46.5% vs N: 31.8%, p= 0.017) and were on medications for chronic conditions (Y: 46.5% vs N: 23.0%, p= 0.000). In addition, a significantly higher percentage of individuals with elevated triglyceride level were found to have abdominal obesity (Y: 69.7% vs N: 39.6%, p= 0.000), overweight /obesity (Y: 89.9% vs N: 50.2%, p=0.000, higher percentage of body fat (Y: 32.76±7.66 vs N: 29.88±7.97, p=0.003), higher mean creatinine levels (Y: 0.87 ± 0.31 vs N: 0.79 ± 0.51 , p= 0.000), and high CRP levels (high: 80.8% vs N: 48.8%, p=0.000).

There was a total of 155 out of 316 participants with abdominal obesity who were significantly older than those without abdominal obesity (Y: 44.94 ± 11.14 vs 39.90 ± 11.23 , p=0.000). A significantly higher percentage of individuals with abdominal obesity had less than high school or high school degree (Y: 28.4% vs N: 14.3%, p=0.000), with a monthly

income of less than 1250\$ (Y: 41.3% vs N: 24.8%, p= 0.002). As expected, a significantly higher percentage of individuals with abdominal obesity had a recent diagnosis of chronic diseases (Y: 47.7% vs N: 31.1%, p= 0.003), diabetes (Y: 9.0% vs N: 1.9%, p=0.010) and hypertension (Y: 19.4% vs N: 4.3%, p=0.000), had a family history of diabetes (Y: 48.4% vs N: 33.5%, p= 0.010) and hypertension (Y: 58.1% vs N: 42.2%, p=0.007), were on medications for chronic conditions (Y: 37.4% vs N: 23.6%, p= 0.011). In addition, a significantly higher percentage of individuals with abdominal obesity were found to have overweight /obesity (Y: 87.7% vs N: 38.5%, p= 0.000), higher percentage of body fat (Y: 35.22 \pm 7.26.51 vs N: 26.51 \pm 6.06, p= 0.000), and high CRP levels (high: 69.7% vs moderate: 48.4%, p= 0.000) (Table 2).

After controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension, diabetes and chronic conditions other than, diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, family history of diabetes, hypertension, obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, body fat percentage (BF %) and CRP, smoking was found to be significantly associated with metabolic syndrome. Specifically, the odds of having metabolic syndrome were found to be four times higher for occasional smokers as compared to individuals who never smoked. In addition, the odds of having metabolic syndrome were found to be 17% higher for a 1% increase in body fat percentage, 90 % lower for females as compared to males and 85%, 86% and 82% lower for individuals with high school degree, bachelor's degree and graduate degree, respectively, as compared to individuals with less than high school degree.

After controlling for the effects of age, gender, income, level of education, physical activity level, marital status, clinical diagnosis of hypertension and diabetes, family history of hypertension, obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake and abdominal obesity, smoking was not found to be significantly associated with hypertension. However, the odds of having hypertension were found to be 5% higher for a 1-year increase in age, ~ 3.5 times/2 times higher for individuals with a clinical diagnosis of hypertension / abdominal obesity, respectively, as compared to their counterparts and~ 67% lower for females as compared to males.

After controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension, diabetes, and chronic conditions other than diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, family history of obesity and heart disease, medication intake, abdominal obesity and CRP, smoking was not found to be significantly associated with impaired fasting glucose. However, the odds of having impaired fasting glucose were found to be 4% higher for a 1-year increase in age, ~ 14 times/and 4 times higher for individuals with a clinical diagnosis of diabetes/ abdominal obesity, respectively, as compared to their counterparts and ~ 74% lower for females as compared to males.

After controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension and diabetes, family history of obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, abdominal obesity and CRP, smoking was found to be significantly associated with low HDL cholesterol. Specifically, the odds of having low HDL cholesterol were found to be ~ 2.6

times and 5.7 times higher for daily smokers and former smoker, respectively, as compared to individuals who never smoked. In addition, the odds of having low HDL cholesterol were found to be ~3.6 times higher, ~2.7 times higher and 69.5% lower for individuals with a family history of obesity, high CRP levels and a clinical diagnosis of hypertension as compared to individuals without a family history of obesity, with moderate CRP levels and without a clinical diagnosis of hypertension, respectively.

After controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension, diabetes and chronic conditions other than diabetes, cardiovascular disease, stroke, hypertension, asthma, cancer, neurological disease, kidney disease, liver cirrhosis, thyroid gland disorder, family history of diabetes, obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, abdominal obesity and CRP, smoking was found to be significantly associated with hypertriglyceridemia. Specifically, the odds of having hypertriglyceridemia were found to be ~sixteen times higher for former smokers as compared to individuals who never smoked. In addition, the odds of having hypertriglyceridemia were found to be 5% higher for a 1year increase in age, 7 times, ~ 2.7 times and ~ 3 times higher for individuals with a family history of obesity, abdominal obesity and high CRP levels as compared to individuals without a family history of obesity, without abdominal obesity and with moderate CRP levels, respectively. On the other hand, the odds of having hypertriglyceridemia were found to be ~ 81% lower for females as compared to males, 88.5% and $\sim 82\%$ lower for individuals with high school degree and bachelor's degree, respectively, as compared to individuals with less than high school degree. After controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension and diabetes, family history of diabetes, hypertension, obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, body fat percentage (BF %) and CRP, smoking was found to be significantly associated with abdominal obesity. Specifically, the odds of having abdominal obesity were found to be ~2.6 times higher for occasional smokers as compared to individuals who never smoked. In addition, the odds of having abdominal obesity were found to be ~30% higher for a 1% increase in body fat percentage, ~5 times higher for individuals with a clinical diagnosis of hypertension, ~71 % lower for females as compared to males and ~ 86% lower for individuals with a graduate degree as compared to individuals with less than high school degree. (Table 3)

Discussion:

In this study, the prevalence of MetS was found to be significantly higher among smokerscigarettes, waterpipe, cigar, pipe and smokeless tobacco products- (daily and occasional 63.2%), as compared to former smokers (5.9%) and people who never smoked (30.9%), p=0.002. This finding has been supported by findings of several studies. In a study done on 856 persons from the San Juan Metropolitan area, aged between 21 and 79 years, MetS was significantly more prevalent in current cigarettes smokers (42.7%) compared to never smokers (40.0%), p<0.001. (Calo W et al., 2013). However, this association has not been supported by other studies. A study done on 125 participants with a mean age of 57.3, showed a lower incidence of MetS in the cigarette smokers' group (40.7%) as compared to the never smokers' group (68.2%). This finding might be due to the fact that smokers in this study had a lower waist circumference (average waist circumference was 100.6 for men and 95.4 for women) (Dedinska et al., 2014), whereas in our study the percentage of people having an unhealthy waist circumference was found to be higher in the "smokers" group (56.2%) as compared to the "never smokers' group (40.6%), p=0.022. In addition, this study studied the effect of one type of tobacco product only (cigarette smoking or waterpipe smoking) on Mets whereas in our study we studied the combined effect of different tobacco products including cigarettes, waterpipe, cigar, pipe and smokeless tobacco products on Mets.

The significant association between daily and occasional smoking- cigarettes, waterpipe, cigar, pipe and smokeless tobacco products and MetS in our study remained even after controlling for several important confounding variables. This finding was supported by the findings of other studies. In a large cross-sectional study, done on 59,467 individuals aged between 18 and 80, a significant positive association was found between smoking and MetS after adjustment for age, sex and BMI. Information about smoking was obtained via a self-administered questionnaire and included different tobacco types (cigarette, cigarillo, cigar, pipe, or a mixture of different kinds). The prevalence of MetS was significantly higher among male and female smokers within each BMI group compared to non-smokers. However, this study did not include waterpipe smoking as in our study (Slagter S et al., 2013). In another cross-sectional study done on 5913 participants from the Multi-Ethnic Study of Atherosclerosis (MESA) aged 45 years or more, a significant positive association was found between cigarette smoking and MetS after adjustment for BMI (OR=1.4; 95% CI:[1.1-1.7], p=0.03) (Berlin et al., 2012). A study done on cigarette and waterpipe smoking also reported, after controlling for sex and age, that the prevalence of MetS was significantly higher in the waterpipe smokers' group (46.8%) compared to non-smokers (38.8%) and the cigarettes smokers' group (26.2%), p<0.001(Sofflaei S et al., 2018).

However, this finding was not supported by findings of other studies. In a study done on 3385 Turkish people, aged 28 years and older, cigarette smoking was found to be protective against Mets, after controlling for age, sex, physical activity grade and family income (RR=0.69; 95% CI: [0.53-0.93]). This was due to the lower waist circumference of current smokers (89.6±0.43 cm) as compared to non-smokers (93.9±0.3 cm), p<0.005. (Onat A et al., 2007).

The possible mechanism behind the association between smoking and MetS is the fact that increased adipocytes lead to an increase in the secretions of pro-inflammatory cytokines and chemokines which in turn lead to an increase in tissues inflammation, increase insulin resistance and thus increase the risk of MetS (Botella-Carretero JI et al., 2007).

When studying the association between MetS components and smoking, the percentage of individuals who were found to have high blood pressure was significantly higher among smokers- cigarettes, waterpipe, cigar, pipe and smokeless tobacco products (daily and occasional smokers 53.2%) as compared to former smokers (6.5%) and people who never smoked (40.3%), p=0.026. Findings related to the association between smoking and high blood pressure have been controversial in previous studies. Some studies supported our finding. A study done on waterpipe smoking, reported a significant higher percentage of individuals having high blood pressure among smokers (systolic 21.9% and diastolic 9.9%) as compared to non-smokers (systolic 19.7% and diastolic 9.6%), p<0.01 (Shafique K et al., 2012). Other studies, however, reported lower blood pressure among cigarette smokers (62.7% and 35.6 % in each study respectively) as compared to non-smokers (68.2% and 60.0% in each study respectively). A possible explanation for having a lower blood

pressure among smokers may be their lower mean BMI compared to non-smokers (29.7 kg/m² and 26.4 kg/m² compared to 30.2 kg/m² and 28.2 kg/m², respectively). (Dedinska I et al., 2014; Onat A et al., 2007). After we adjusted for confounding variables, no significant association was found between smoking and high blood pressure. Several studies supported our finding and did not find a significant association after controlling for confounding factors. A study done on 59,467 individuals aged between 18 and 80 to examine the effect of smoking (cigarettes, cigar, cigarillo, pipe, or a mixture of different kinds) on Mets and its components did not find a significant association between smoking and raised blood pressure in all BMI classes and among both men and women (Slagter S et al., 2013). Another study showed no significant association between cigarette smoking and high blood pressure after controlling for BMI (Berlin I et al., 2012). Findings of several studies however did not support this finding. One study done on Turkish individuals found a significant association between cigarettes smoking and lower blood pressure, even after controlling for confounding factors; in this study, however, researchers failed to control for the effect of BMI (Onat A et al., 2007). Moreover, another study done on 1215 Japanese male workers showed a significantly lower blood pressure level among cigarettes smokers after controlling for possible confounding factors (OR=0.8; 95% CI:[0.6-1.1] and OR=0.7; 95% CI:[0.5-0.9] for systolic and diastolic blood pressure respectively). However, this study was done on males only. (Takeuchi T et al., 2009). In another study done, the association between waterpipe smoking and high blood pressure was found to be significant even after controlling for confounding factors (OR: 1.95; 95% CI: [1.51–2.51]), yet this study only studied the effect waterpipe smoking on blood pressure and they only controlled for age, sex and social class without taking into account other factors such as

BMI or body fat percentage which might have affect the results. (Shafique K et al., 2012). It's worth to mention that a study that compared the ambulatory blood pressure monitoring with office blood pressure showed that 24 hours blood pressure monitoring was significantly higher among smokers as compared to non-smokers (145.0 ± 22.2 mmHg vs 140.4 ± 16.4 mm Hg, p< 0.05) whereas office blood pressure was differences between the two groups was not significant. (Mann SJ et al., 1991). This proposes that ambulatory blood pressure is more accurate than office blood pressure when the association with blood pressure is in question and this could explain the discrepancies in findings of our study and those of other studies. In addition, the studies that contradicted our findings were done on one type of tobacco product only (cigarettes smoking or waterpipe smoking) while in our study we included all tobacco products (cigarettes, waterpipe, cigar, pipe, smokeless tobacco products).

Similar to the finding pertaining to blood pressure, no significant association was found between impaired fasting glucose and smoking in this study after adjusting for possible confounding factors. This finding is in line with the findings of many studies (Berlin I et al., 2013; Slagter S et al., 2013; Takeuchi T et al., 2009; Kang JH & Song YM, 2015). One study, however, found an association between smoking 30 packs of cigarettes per year and impaired fasting glucose after adjustment for confounders (OR=1.33; 95% CI: [1.07-1.67], p<0.0001) (Shin HS et al., 2018). Another study also found that smoking more than 16 to 20 cigarettes per day increases the risk of developing type 2 diabetes mellitus (RR: 1.61; 95% CI: [1.43-1.80]) (Willi C, 2007), suggesting a dose-response relationship between smoking and development of type 2 diabetes mellitus. Other studies reported a higher prevalence of impaired fasting glucose in men smokers compared to non-smokers (100±24 vs 97 ± 16 , p<0.01) and used cut-off values for elevated blood glucose of 110 mg/dl) (Ishizaka N et al., 2007) different than that used in our study (100 mg/dl). The discrepancies in results between our study and the other studies could, therefore, be attributed to the restriction of study participation to one sex group, use of different cut-off values for elevated fasting glucose, and lack of information on tobacco dosage smoked.

In this study, smoking was found to be significantly associated with both low HDL and high TG levels. This association comes in line with different studies (Slagter S et al., 2013; Berlin I et al., 2012; Nakashita Y et al., 2010; Kang JH & Song YM, 2015). In addition, some studies showed that even though current cigarettes smokers were found to have a lower body weight, smoking was still associated with lower HDL levels and higher triglyceride levels (Berlin I et al., 2012, Onat A et al., 2007). This shows that despite the advertising claim that smoking helps in controlling body weight (Clair C et al., 2011) and despite the protective effect, mediated by lower body weight, of smoking on MetS found in a Turkish study (Onat A et al., 2007), lower BMI does not protect against dyslipidemia caused by smoking.

After controlling for possible confounding factors, the odds of having high TG were found to be significantly higher in former smokers compared to never smokers. This finding is in line with different studies that found a significant association between former smokers who used to be heavy smokers and high triglyceride levels (Calo W et al., 2013; Shin HS et al., 2018)

Least but not last, our study shows a significant association between smoking and larger waist circumference before and after adjustment for confounders. This finding is in line with several previous studies (Slagter S et al., 2013; Shafique K et al., 2012). Other studies

showed an inverse association between cigarette smoking and waist circumference before adjustment for confounding factors (Onat A et al., 2007; Dedinska I et al., 2014). This may be mediated by the lower BMI among smokers. However, in one study, after controlling for BMI, cigarettes smoking was found to be significantly positively associated with waist circumference (OR= 1.6; 95% CI: [1.2-2.1], p<0.004) (Berlin I et al., 2012). Another possible explanation for the controversial results among studies is the dose-dependent relationship (which was not assessed in our study) since the amount of tobacco smoked can modify the association between smoking and abdominal obesity. In a cross-sectional study done on 6032 men, the association between cigarette smoking and high waist circumference was only significant in individuals who smoke more than 30 packs per year (OR=1.77; 95% CI: [1.29-2.44], p<0.0001)(Shin HS et al., 2018). It is well known that smokers have higher blood cortisol levels as compared to non-smokers. Moreover, smoking is suggested to cause an increase in hypothalamic pituitary-adrenal (HPA) axis activation. The increase in cortisol production and activation of HPA may be the reason behind abdominal obesity caused by smoking. (Rohleder N & Kirschbaum C, 2006; Pasquali R & Vicennati V, 2000).

This study has several strengths. To the best of our knowledge, this was the first study to assess the association between smoking all kind of tobacco products (cigarettes, waterpipe, cigar, pipe and smokeless tobacco product) and metabolic syndrome and its components. In Lebanon, this is the first study to assess the relationship between smoking all kind of tobacco products (cigarettes, waterpipe, cigar, pipe and smokeless tobacco product) and metabolic syndrome in healthy individuals.

However, this study has some limitations. First of all, this was a cross-sectional study so a temporal relationship between smoking and MetS and its components cannot be established. The second limitation is that smoking was based on self-reporting which could have resulted in inaccurate data (Kang JH & Song YM; 2015). A third limitation is that the study participants were not asked to specify frequency and amount of tobacco smoked per day thus, we couldn't assess the dose-dependent relationship between smoking and Mets/ MetS components in which, in turn, might have masked the associations between smoking and Mets/ Mets components. In addition, we had few people in some of the categories for the variable" type of tobacco product smoked" which did not allow us to examine the individual effect of each product separately.

Conclusion

This study showed a significant association between smoking and MetS even after controlling for the effects of age, gender, income, level of education, physical activity level, clinical diagnosis of hypertension and diabetes, family history of obesity, heart disease, hypercholesterolemia, hypertriglyceridemia, medication intake, abdominal obesity and CRP. This positive association is mainly related to the significant association we found between smoking and low HDL, hypertriglyceridemia and abdominal obesity. However, we could not find a significant association between smoking status and blood pressure or impaired fasting glucose. In all cases, tobacco imposes many health risks and is considered to be a global health epidemic. Therefore, a plan for prevention and intervention for tobacco control is necessary in all countries, especially in Lebanon in which laws for tobacco control are not being applied. In addition, educational programs that highlight the harmful

effects of smoking on health and the importance of adopting a healthy lifestyle (smoking cessation, good dietary habits, physical activity) are highly recommended.

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Characteristics						
	Total (n=)	Daily smokers (n=)	Occasional smokers (n=)	Former smokers (n=)	Never smoked (n=)	P-value =
	Mean \pm SD	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
	Or n (%)	Or n (%)	Or n (%)	Or n (%)	Or n (%)	
Age (years)	42.37±11.45	45.97±11.29	39.65±9.24	48.63±12.28	41.22±11.84	0.004* 0.010**
Gender						
Male	154 (48.7)	46 (29.9)	39 (25.3)	5 (3.2)	64 (41.6)	0.045
Female	162 (51.3)	40 (24.7)	27 (16.7)	3 (1.9)	92 (56.8)	
Residence						
Urban	195 (61.7)	50 (25.6)	40 (20.5)	5 (2.6)	100 (51.3)	0.823
Rural	121 (38.3)	36 (29.8)	26 (21.5)	3 (2.5)	56 (46.3)	
Marital status						
Single/Separated/ Divorced	110 (34.8)	19 (17.3)	27 (24.5)	3 (2.7)	61 (55.5)	0.028
Married	206 (65.2)	67 (32.5)	39 (18.9)	5 (2.4)	95 (46.1)	
Children						
No	123 (38.9)	19 (15.4)	29 (23.6)	3 (2.4)	72 (58.5)	0.001
Yes	193 (61.1)	67 (34.7)	37 (19.2)	5 (2.6)	84 (43.5)	
Number of children	4.87±3.37	3.76±2.90	5.18±3.48	4.88±3.52	5.35±3.48	0.002* 0.044**
Monthly income (\$)						
<1,250	104 (32.9)	37 (35.6)	23 (22.1)	4 (3.8)	40 (38.5)	0.058
1,250-4,000	82 (25.9)	20 (24.4)	21 (25.6)	1 (1.2)	40 (48.8)	
≥4,000	130 (41.1)	29 (22.3)	22 (16.9)	3 (2.3)	76 (58.5)	
Educational level						
Less than high school	18 (5.7)	10 (55.6)	2 (11.1)	0 (0.0)	6 (33.3)	0.001
High school or equivalent	49 (15.5)	20 (40.8)	16 (32.7)	1 (2.0)	12 (24.5)	
Bachelor's degree	81 (25.6)	23 (28.4)	16 (19.8)	1 (1.2)	41 (50.6)	
Graduate degree	168 (53.1)	33 (19.6)	32 (19.0)	6 (3.6)	97 (57.7)	
Alcohol drinking						
No	234 (74.4)	54 (23.0)	47 (20.0)	7 (3.0)	127 (54.0)	0.011
Yes	81 (25.6)	32 (39.5)	19 (23.5)	1 (1.2)	29 (35.8)	
Passive smoking						
No	223 (70.6)	59 (26.5)	46 (20.6)	5 (2.2)	133 (50.7)	0.844
Yes	93 (29.4)	27 (29.0)	20 (21.5)	3 (3.2)	43 (46.2)	
Physical activity level						
Low	204 (64.6)	59 (28.9)	49 (24.0)	4 (2.0)	92 (45.1)	0.095
Moderate/ high	112 (35.4)	27 (24.1)	17 (15.2)	4 (3.6)	64 (57.1)	1
Medical morbidity						
No	192 (60.8)	49 (25.5)	43 (22.4)	5 (2.6)	95 (49.5)	0.773
Yes	124 (39.2)	37 (29.8)	23 (18.5)	3 (2.4)	61 (49.2)	1
Diabetes	, , , , , , , , , , , , , , , , , , ,		<u>`</u>	, <i>,</i> ,	, , , , , , , , , , , , , , , , , , ,	
No	299 (94.6)	81 (27.1)	63 (21.1)	8 (2.7)	147 (49.2)	1.000
Yes	17 (5.4)	5 (29.4)	3 (17.6)	0 (0.0)	9 (52.9)	1

Cardiovascular disease						
No	310 (98.1)	81 (26.1)	66 (21.3)	7 (2.3)	156 (50.3)	0.001
Yes	6 (1.9)	5 (83.3)	0 (0.0)	1 (16.7)	0 (0.0)	
Stroke				- ()		
No	310 (100.0)	86 (27.2)	66 (20.9)	8 (2.5)	156 (49.4)	
Yes	0 (0)			0 (10)		
Hypertension						
No	279 (88.3)	70 (25.1)	63 (22.6)	7 (2.5)	139 (49.8)	0.049
Yes	37 (11.7)	16 (43.2)	3 (8.1)	1 (2.7)	17 (45.9)	
Asthma						
No	311 (98.4)	85 (27.3)	66 (21.2)	8 (2.6)	152 (48.9)	0.570
Yes	5 (1.6)	1 (20.0)	0 (0.0)	0 (0.0)	4 (80.0)	
Cancer						
No	316 (100.0)	86 (27.2)	66 (20.9)	8 (2.5)	156 (49.4)	
Yes	0 (0)					
Neurological disease						
No	316 (100.0)	86 (27.2)	66 (20.9)	8 (2.5)	156 (49.4)	
Yes	0 (0)					
Kidney disease						
No	315 (99.7)	85 (27.0)	66 (21.0)	8 (2.5)	156 (49.5)	0.506
Yes	1 (0.3)	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	
Liver cirrhosis						
No	316 (100.0)	86 (27.2)	66 (20.9)	8 (2.5)	156 (49.4)	
Yes	0 (0)					
Thyroid gland disorders						
No	306 (96.8)	82 (26.8)	66 (21.6)	8 (2.5)	150 (49.0)	0.315
Yes	10 (3.2)	4 (40.0)	0 (0.0)	0 (0.0)	6 (3.8)	0.010
Other medical	10 (0.2)	. (1010)		0 (0.0)		
condition						
No	259 (82.0)	68 (26.3)	47 (18.1)	7 (2.7)	137 (52.9)	0.022
Yes	57 (18.0)	18 (31.6)	19 (33.3)	1 (1.8)	19 (33.3)	
Family history						
Diabetes	ſ	1	Ī			
No	187 (59.2)	55 (29.4)	37 (19.8)	2 (1.1)	93 (49.7)	0.184
Yes	129 (40.8)	31 (24.0)	23 (22.5)	6 (4.7)	64 (48.8)	
Hypertension						
No	158 (50)	46 (29.1)	31 (19.6)	4 (2.5)	77 (48.7)	0.880
Yes	158 (50)	40 (25.3)	35 (22.2)	4 (2.5)	79 (50.0)	1
Obesity						
No	301 (95.3)	81 (26.9)	65 (21.6)	8 (2.7)	147 (48.8)	0.571
Yes	15 (4.7)	5 (33.3)	1 (6.7)	0 (0.0)	9 (60.0)	
Heart disease						
No	201 (63.6)	57 (28.4)	46 (22.9)	4 (2.0)	94 (46.8)	0.427
Yes	115 (36.4)	29 (25.2)	20 (17.4)	4 (3.5)	62 (53.9)	
Hypercholesterolemia						
No	201 (63.6)	55 (27.4)	37 (18.4)	5 (2.5)	104 (51.7)	0.516
Yes	115 (36.4)	31 (27.0)	29 (25.2)	3 (2.6)	52 (45.2)	7

Hypertriglyceridemia						
No	310 (98.1)	85 (27.4)	64 (20.6)	8 (2.6)	153 (49.4)	0.784
Yes	6 (1.9)	1 (16.7)	2 (33.3)	0 (0.0)	3 (50.0)	
Medications		- ()				
No	220 (69.6)	55 (25.0)	50 (22.7)	5 (2.3)	110 (50.0)	0.407
Yes	96 (30.4)	31 (32.3)	16 (16.7)	3 (3.1)	46 (47.9)	
Number of stressful	0.56±0.76	0.70±0.87	0.45±0.71	0.25±0.46	0.54±0.74	0.185
life events						
Anxiety score	1.90±1.95	1.72±1.75	2.42±2.18	1.00±1.77	1.83±1.93	0.087
Mental illness (other						
than depression)						
No	305 (96.5)	82 (26.9)	65 (21.3)	8 (2.6)	150 (49.4)	0.706
Yes	11 (3.5)	4 (36.4)	1 (9.1)	0 (0.0)	6 (50.0)	
Medications for						
mental illness						
No	310 (98.1)	83 (26.8)	66 (21.3)	8 (2.6)	153 (98.1)	0.498
Yes	6 (1.9)	3 (50.0)	0 (0.0)	0 (0.0)	3 (1.9)	
Family member with						
mental illness or						
depression						
No	295 (93.4)	80 (27.1)	60 (20.3)	8 (2.7)	147 (49.8)	0.791
Yes	21 (6.6)	6 (28.6)	6 (28.6)	0 (0.0)	9 (42.9)	
Depression						
No	304 (96.2)	82 (27.0)	66 (21.7)	8 (2.6)	148 (48.7)	0.269
Yes	12 (3.8)	4 (33.3)	0 (0.0)	0 (0.0)	8 (66.7)	
Medication for						
depression						
No	300 (94.9)	79 (26.3)	66 (22.0)	8 (2.7)	147 (49.0)	0.093
Yes	16 (5.1)	7 (43.8)	0 (0.0)	0 (0.0)	9 (56.2)	
Stressful life event						
No	186 (58.9)	44 (23.7)	44 (23.7)	6 (3.2)	92 (49.5)	0.206
Yes	130 (41.1)	42 (32.3)	22 (16.9)	2 (1.5)	64 (49.2)	
Anxious						
No	214 (67.7)	61 (28.5)	39 (18.2)	7 (3.3)	107 (50.0)	0.342
Yes	101 (32.0)	25 (24.8)	26 (25.7)	1 (1.0)	49 (48.5)	
Creatinine levels	0.81±0.46	0.82±0.34	0.79±0.17	0.83±0.15	0.82±0.60	0.799
(mg/dL)						
Vitamin D status						
Optimal	122 (38.6)	38 (31.1)	22 (18.0)	3 (2.5)	59 (48.4)	0.566
Suboptimal	191 (60.4)	47 (24.6)	44 (23.0)	5 (2.6)	95 (49.7)	
Metabolic syndrome						
No	248 (78.5)	63 (25.4)	46 (18.5)	4 (1.6)	135 (54.4)	0.002
Yes	68 (21.5)	23 (33.8)	20 (29.4)	4 (5.9)	21 (30.9)	
High BP (mmHg)						
No	254 (80.4)	63 (24.8)	56 (22.0)	4 (1.6)	131 (51.6)	0.026
Yes	62 (19.6)	23 (37.1)	10 (16.1)	4 (6.5)	25 (40.3)	
High IFG (mg/dL)						
No	259 (82.0)	69 (26.6)	53 (20.5)	5 (1.9)	132 (51.0)	0.319
Yes	57 (18.0)	17 (29.8)	13 (22.8)	3 (5.3)	24 (42.1)	

High TG (mg/dL)						
No	217 (68.7)	55 (25.3)	41 (18.9)	2 (0.9)	119 (54.8)	0.004
Yes	99 (31.3)	31 (31.3)	25 (25.3)	6 (6.1)	37 (37.4)	
HDL c (mg/dL)						
Normal	239 (75.6)	55 (23.0)	48 (20.1)	5 (2.1)	131 (54.8)	0.003
Low	77 (24.4)	31 (40.3)	18 (23.4)	3 (3.9)	25 (32.5)	
WC (cm)						
Normal	161 (50.9)	38 (23.6)	27 (16.8)	3 (1.9)	93 (57.8)	0.022
Abdominal obesity	155 (49.1)	48 (31.0)	39 (25.2)	5 (3.2)	63 (40.6)	
Body composition	30.77±7.97	31.01±7.43	31.21±6.82	31.73±9.47	30.42±8.66	0.910
(total body fat %)						
BMI (kg/m ²)						
Underweight	2 (0.6)	0 (0.0)	0 (0.0)	0 (0.0)	2 (100.0)	0.128
Normal weight	116 (36.7)	26 (22.4)	19 (16.4)	2 (1.7)	69 (59.5)	
Overweight	121 (38.3)	34 (28.1)	26 (21.5)	4 (2.3)	57 (47.1)	
Obese	77 (24.4)	26 (33.8)	21 (27.3)	2 (2.6)	28 (36.4)	
Hypercholesterolemia						
(mg/dL)						
No	205 (64.9)	54 (26.3)	43 (21.0)	5 (2.4)	103 (50.2)	0.962
Yes	111 (35.1)	32 (28.8)	23 (20.7)	3 (2.7)	53 (47.7)	
LDL c (mg/dL)						
Normal	116 (36.7)	31 (26.7)	26 (22.4)	2 (1.7)	57 (49.1)	0.921
High	197 (62.3)	54 (27.4)	40 (20.3)	6 (3.0)	97 (49.2)	
CRP ³ (mg/L)						
Moderate	130 (41.1)	30 (23.1)	21 (16.2)	5 (3.8)	74 (23.4)	0.048
High	186 (58.9)	56 (30.1)	45 (24.2)	3 (1.6)	82 (25.9)	

<u>Table 1:</u> Association between smoking status and sociodemographic characteristics, biochemical measurements, metabolic syndrome and its components

* Daily smokers Vs Occasional smokers

** Daily smokers Vs Never smokers

BP, blood pressure; high blood pressure as defined by systolic/diastolic BP ${\geq}130/85~mmHg$

IFG, impaired fasting glucose; IFG as defined by FG \geq 100 mg/dl

TG, triglyceride; high TG as defined by TG≥150 mg/dl

HDLc, high density lipoprotein cholesterol; low HDLc as defined by: <40 mg/dl in men and <50 mg/dl in women

WC, waist circumference; high WC as defined by ≥ 102 cm in men and ≥ 88 cm in women LDLc, low density lipoprotein cholesterol; high LDLc as defined by LDL ≥ 100 mg/dl BMI, body mass index; underweight as defined BMI <18.5 kg/m², normal weight as defined by BMI between 18.5 and 24.9 kg/m², overweight as defined by BMI between 25 and 29.9 kg/m², obese as defined by BMI >30 kg/m²

CRP, c-reactive protein; low as defined by CRP <1 mg/l, moderate as defined by CRP between 1 and 3 mg/l, high as defined by CRP >3 mg/l

Cha ract						Met	aboli	ic syr	ndro	ome a	and i	its c	omj	pone	ents			
erist ics																		
	Metal syndr (n=)		p- val ue	Raise (n=)	d BP	p - v a 1 u e	d		p - v a 1 u e	Lov HD (n=	L	p - v a 1 u e	Ele ate TC (n=	ed G	p - v a 1 u e	Ab min obe y	nal	p - v a 1 u e
	Mean SD Or n			Mean Or n ((%)		Mea SD Or 1 (%)			Mea ± S Or 1 (%)	D n		Mo n = SE Or (%	⊧) n		Me ± S Or (%)	D n	
	No	Yes		No	Yes		N o	Y es		N o	Y es		N o	Y e s		N o	Y es	
Age (yea rs)	40.5 4±1 1.36	49. 06± 9.1 1	0.0 00	40.4 6±1 0.86	50.19 ±10.5 1	0.0000000000000000000000000000000000000	40 .5 8± 10 .7 5	$50 \\ .5 \\ 4\pm \\ 11 \\ .0 \\ 5$	0.0000000000000000000000000000000000000	42 .1 8± 11 .6 3	4 2. 9 6 ± 1 0. 9 3	0 6 0 5	3 9 7 8 ± 1 0 8 3	$\begin{array}{c} 4 \\ 8 \\ . \\ 0 \\ 6 \\ \pm \\ 1 \\ 0 \\ . \\ 7 \\ 2 \end{array}$	0 0 0 0	$3 \\ 9. \\ 9 \\ 0 \\ \pm \\ 1 \\ 1. \\ 2 \\ 3$	4 4. 9 4 ± 1 1. 1 4	0.0000000000000000000000000000000000000
Gen der Mal e	105 (42. 3)	49 (72. 1)	0.0 00	108 (42. 5)	46 (74.2)	0 0 0 0	11 1 (4 2. 9)	43 (7 5. 4)	0 0 0	11 4 (4 7. 7)	4 0 (5 1. 9)	0 6 0 5	8 0 (3 6	7 4 (7 4	0 0 0 0	7 9 (4 9. 1)	8 2 (5 0. 9)	0 9 9 3

Fem ale	143 (57. 7)	19 (27. 9)		146 (57. 5)	16 (25.8)		14 8 (5 7. 1)	14 (2 4. 6)		12 5 (5 2. 3)	3 7 (4 8. 1)		9) 1 3 7 (6 3 1)	7) 2 5 (2 5 3)		7 5 (4 8. 4)	8 0 (5 1. 6)	
Resi den ce Urb	157	38	0.3	159	36	0	16	31	0	14	4	0	1	5	0	1	9	0
an	(63. 3)	58 (55. 9)	30	(62. 6)	(58.1)	6 0 8	10 4 (6 3. 3)	(5 4. 4)	0 2 6 9	14 8 (6 1. 9)	4 7 (6 1. 0)	9 9 7	1 3 6 (6 3 6)	3 7 (5 7 6)	0 3 7 0	1 0 1 (6 2. 7)	9 4 (6 0. 6)	7 9 0
Rura 1	91 (36. 7)	30 (44. 1)		95 (37. 4)	26 (41.9)		95 (3 6. 7)	26 (4 5. 6)		91 (3 8. 1)	3 0 (3 9. 0)		7 9 (3 6 4)	4 2 (4 2 4)		6 0 (3 7. 3)	6 1 (3 9. 4)	
Mar ital stat us																		
Sing le/S epar ated/ Div orce d	91 (36. 7)	19 (27. 9)	0.2 31	97 (38. 2)	13 (21.0)	0 0 1 6	95 (3 6. 7)	15 (2 6. 3)	0 1 8 2	89 (3 7. 2)	2 1 (2 7. 3)	0 1 4 7	8 3 (3 8 2)	2 7 (2 7 3)	0 0 7 6	5 3 (3 2. 9)	5 7 (3 6. 8)	0 5 4 8

Mar ried Chil dre	157 (63. 3)	49 (72. 1)		157 (61. 8)	49 (79.0)		16 4 (6 3. 3)	42 (7 3. 7)		15 0 (6 2. 8)	5 6 (7 2. 7)		1 3 4 (6 1 8)	7 2 (7 2 7)		1 0 8 (6 7. 1)	9 8 (6 3. 2)	
n No	99 (39. 9)	24 (35. 3)	0.5 81	106 (41. 7)	17 (27.4)	0 0 5 4	10 3 (3 9. 8)	20 (3 5. 1)	0 6 1 3	99 (4 1. 4)	2 4 (3 1. 2)	0 1 4 1	9 1 (4 1 9)	3 2 (3 2 3)	0 1 3 3	6 2 (3 8. 5)	6 1 (3 9. 4)	0 9 6 9
Yes	149 (60. 1)	44 (64. 7)		148 (58. 3)	45 (72.6)		15 6 (6 0. 2)	37 (6 4. 9)		14 0 (5 8. 6)	5 3 (6 8. 8)		1 2 6 (5 8 1)	6 7 (6 7 7)		9 9 (6 1. 5)	9 4 (6 0. 6)	
Nu mbe r of chil dre n	4.90 ±3.4 2	4.7 8±3 .21	0.6 44	5.01 ±3.4 4	4.29± 3.01	0 5 6 8	4. 89 ±3 .4 1	4. 77 ±3 .2 2	0 6 8 6	5. 00 ± 3 .4 3	4. 4 5 ± 3. 1 8	0 4 8 8	5 0 4 ± 3 4 4	$ \begin{array}{c} 4 \\ . \\ 5 \\ 1 \\ \pm \\ 3 \\ . \\ 2 \\ 0 \end{array} $	0 5 0 3	4. 8 4 ± 3. 3 7	4. 9 0 ± 3. 8	0 8 5 6
Mo nthl y inco																		

me (\$)																		
<1,2 50	70 (28. 2)	43 (50. 0)	0.0 03	78 (30. 7)	26 (41.9)	0 0 8 1	78 (3 0. 1)	26 (4 5. 6)	0 0 1 0	72 (3 0. 1)	3 2 (4 1. 6)	0 0 8 7	5 9 (2 7 2)	4 5 (4 5 5)	0 0 0 1	4 0 (2 4. 8)	6 4 (4 1. 3)	0 0 0 2
1,25 0- 4,00 0	71 (28. 6)	11 (16. 2)		72 (28. 3)	10 (16.1)		75 (2 9. 0)	7 (1 2. 3)		61 (2 5. 5)	2 1 (2 7. 3)		6 7 (3 0 9)	1 5 (1 5 2)		4 1 (2 5. 5)	4 1 (2 6. 5)	
≥4,0 00	107 (43. 1)	23 (33. 8)		104 (40. 9)	16 (41.9)		10 6 (4 0. 9)	24 (4 2. 1)		10 6 (4 4. 4)	2 4 (3 1. 2)		9 1 (4 1 9)	3 9 (3 9 4)		8 0 (4 9. 7)	5 0 (3 2. 3)	
Edu cati onal level																		
Less than high scho ol	6 (2.4)	12 (17. 6)	0.0 00	11 (4.3)	7 (11.3)	0 0 9	10 (3 .9)	8 (1 4. 0)	0 0 4 5	11 (4 .6)	7 (9 .1)	0 0 2 5	4 (1 8)	1 4 (1 4 1)	0 0 0	3 (1 .9)	1 5 (9 .7)	0 0 0 0
Hig h scho ol or equi	34 (13. 7)	15 (22. 1)		33 (13. 0)	16 (25.8)		40 (1 5. 4)	9 (1 5. 8)		33 (1 3. 8)	1 6 (2 0. 8)		3 1 (1 4	1 8 (1 8		2 0 (1 2. 4)	2 9 (1 8. 7)	

vale nt													3)	2)				
Bac helo r's degr ee	67 (27. 0)	14 (20. 6)		69 (26. 8)	13 (21.0)		67 (2 5. 9)	14 (2 4. 6)		57 (2 3. 8)	2 4 (3 1. 2)		6 0 (2 7 6)	2 1 (2 1 2)		3 4 (2 1. 1)	4 7 (3 0. 3)	
Gra duat e degr ee	141 (56. 9)	27 (39. 7)		142 (55. 9)	26 (41.9)		14 2 (5 4. 8)	26 (4 5. 6)		13 8 (5 7. 7)	3 0 (3 9. 0)		1 2 (5 6 2)	4 6 (4 6 5)		1 0 4 (6 4. 6)	6 4 (4 1. 3)	
Alco hol drin king																		
No	185 (74. 6)	50 (73. 5)	0.9 83	186 (73. 2)	49 (79.9)	0 4 3 8	19 4 (7 4. 9)	41 (7 1. 9)	0 7 6 6	17 9 (7 4. 9)	5 6 (7 2. 7)	0 8 1 9	1 1 6 (7 2 0)	1 9 (7 6 8)	0 4 0 5	1 1 6 (7 2. 0)	1 9 (7 6. 8)	0 4 0 5
Yes	63 (25. 4)	18 (26. 5)		68 (26. 8)	3 (21.0)		65 (2 5. 1)	16 (2 8. 1)		60 (2 5. 1)	2 1 (2 7. 3)		4 5 (2 8 0)	3 6 (2 3 2)		4 5 (2 8. 0)	3 6 (2 3. 2)	
Pass ive																		

smo king																		
No	176 (71. 2)	47 (69. 1)	0.8 84	176 (69. 3)	47 (75.8)	0 3 9 3	18 6 (7 1. 8)	37 (6 4. 9)	0 3 8 2	16 9 (7 0. 7)	5 4 (7 0. 1)	1 0 0 0	1 5 4 (7 1 0)	6 9 (6 9 7)	0 9 2 3	1 2 0 (7 4. 5)	1 0 3 (6 6. 5)	0 1 4 6
Yes	72 (29. 0)	21 (30. 9)		78 (30. 7)	15 (24.2)		73 (2 8. 2)	20 (3 5. 1)		70 (2 9. 3)	2 3 (2 9. 9)		6 3 (2 9 0)	3 0 (3 0 3)		4 1 (2 5. 5)	5 2 (3 3. 5)	
Phy sical acti vity level																		
Low	157 (63. 3)	47 (69. 1)	0.4 57	163 (64. 2)	41 (66.1)	0 8 8 8	16 7 (6 4. 5)	37 (6 4. 9)	1 0 0 0	14 7 (6 1. 5)	5 7 (7 4. 0)	0 0 6 3	1 3 6 (6 2 7)	6 8 (6 8 7)	0 3 6 3	9 7 (6 0. 2)	1 0 7 (6 9. 0)	0 1 3 0
Mod erate / high	91 (36. 7)	21 (30. 9)		91 (35. 8)	21 (33.9)		92 (3 5. 5)	20 (3 5. 1)		92 (3 8. 5)	2 0 (2 6. 0)		8 1 (3 7 3)	3 1 (3 1 3)		6 4 (3 9. 8)	4 8 (3 1. 0)	
Med ical																		

mor bidi ty																		
No	167 (67. 3)	25 (36. 8)	0.0 00	168 (66. 1)	24 (38.7)	0 0 0 0	17 6 (6 8. 0)	16 (2 8. 1)	0 0 0	14 8 (6 1. 9)	4 (5 7. 1)	0 5 4 0	1 5 1 (6 9 6)	4 1 (4 1 4)	0 0 0	1 1 (6 8. 9)	8 1 (5 2. 3)	0 0 0 3
Yes	81 (32. 7)	43 (63. 2)		86 (33. 9)	38 (61.3)		83 (3 2. 0)	41 (7 1. 9)		91 (3 8. 1)	3 3 (4 2. 9)		6 6 (3 0 4)	5 8 (5 8 6)		5 0 (3 1. 1)	7 4 (4 7. 7)	
Dia bete s																		
No	242 (97. 6)	57 (83. 8)	0.0 00	245 (96. 5)	54 (87.1	0 0 0 8	25 6 (9 8. 8)	43 (7 5. 4)	0 0 0	22 8 (9 5. 4)	7 1 (9 2. 2)	0 3 8 1	2 1 0 (9 6 8)	8 9 (8 9 9)	0 0 2 5	1 5 8 (9 8. 1)	1 4 1 (9 1. 0)	0 0 1 0
Yes	6 (2.4)	11 (16. 2)		9 (3.5)	8 (12.9)		3 (1 .2)	14 (2 4. 6)		11 (4 .6)	6 (7 .8)		7 (3 2)	1 0 (1 0 1)		3 (1 .9)	1 4 (9 .0)	
Car diov ascu																		

lar dise ase																		
No	245 (98. 8)	65 (95. 6)	0.1 16	251 (98. 8)	59 (95.2)	0 0 9 2	25 6 (9 8. 8)	54 (9 4. 7)	0 0 7 4	23 5 (9 8. 3)	7 5 (9 7. 4)	0 6 3 6	2 1 5 (9 9 1)	9 5 (9 6 0)	0 0 8 0	1 5 9 (9 8. 8)	1 5 1 (9 7. 4)	0 4 4 1
Yes	3 (1.2)	3 (4.4)		3 (1.2)	3 (4.8)		3 (1 .2)	3 (5 .3)		4 (1 .7)	2 (2 .6)		2 (0 9)	4 (4 0)		2 (1 .2)	4 (2 .6)	
Stro ke																		
No	248 (10 0.0)	68 (10 0.0)		254 (10 0.0)	52 (0.0)		25 9 (1 00 .0)	57 (1 00 .0)		23 9 (1 00 .0)	7 7 (1 0 0. 0)		2 1 7 (1 0 0 0)	9 9 (1 0 0 0)		1 6 1 (1 0 0. 0)	1 5 (1 0 0. 0)	
Yes	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)		0 (0 .0)	0 (0 .0)		0 (0 .0)	0 (0 .0)		0 (0 0)	0 (0 0)		(0 .0)	0 (0 .0)	
Hyp erte nsio n																		

No	226 (91. 1)	53 (77. 9)	0.0 05	237 (93. 3)	42 (67.7)	0 0 0 0	23 8 (9 1. 9)	41 (7 1. 9)	0 0 0	21 1 (8 8. 3)	6 8 (8 8. 3)	1 0 0 0	2 0 (9 2 2)	7 9 (7 9 8)	0 0 0 3	1 5 4 (9 5. 7)	1 2 5 (8 0. 6)	0 0 0
Yes	22 (8.9)	15 (22. 1)		17 (6.7)	20 (32.3)		21 (8 .1)	16 (2 8. 1)		28 (1 1. 7)	9 (1 1. 7)		1 7 (7 8)	2 0 (2 0 2)		7 (4 .3)	3 0 (1 9. 4)	
Ast hma																		
No	245 (98. 8)	66 (97. 1)	0.2 94	250 (98. 4)	61 (98.4)	1 0 0 0	25 5 (9 8. 5)	56 (9 8. 2)	1 0 0	23 6 (9 8. 7)	7 5 (9 7. 4)	0 5 9 9	2 1 5 (9 9 1)	9 6 9 7 0)	0 1 7 9	1 5 9 (9 8. 8)	1 5 2 (9 8. 1)	0 6 8 0
Yes	3 (1.2)	2 (2.9)		4 (1.6)	1 (1.6)		4 (1 .5)	1 (1 .8)		3 (1 .3)	2 (2 .6)		2 (0 9)	3 (3 0)		2 (1 .2)	3 (1 .9)	
Can cer																		
No	248 (10 0.0)	68 (10 0.0)		254 (10 0.0)	62 (100.0)		25 9 (1 00 .0)	57 (1 00 .0)		23 9 (1 00 .0)	7 7 (1 0 0. 0)		2 1 7 (1 0 0	9 9 (1 0 0		1 6 1 (1 0 0. 0)	1 5 (1 0 0. 0)	

Yes	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)		0 (0 .0)	0 (0 .0)		0 (0 .0)	0 (0 .0)		0) 0 (0 0)	0) 0 (0 0)		0 (0 .0)	0 (0 .0)	
Neu rolo gica l dise ase	240			254			25				-							
No	248 (10 0.0)	68 (10 0.0)		254 (10 0.0)	62 (100.0)		25 7 (1 00 .0)	57 (1 00 .0)		23 9 (1 00 .0)	7 7 (1 0 0. 0)		2 1 7 (1 0 0 0)	9 (1 0 0)		1 6 1 (1 0 0. 0)	1 5 (1 0 0. 0)	
Yes	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)		0 (0 .0)	0 (0 .0)		0 (0 .0)	0 (0 .0)		0 (0 0)	0 (0 0)		0 (0 .0)	0 (0 .0)	
Kid ney dise ase																		
No	248 (10 0.0)	67 (98. 5)	0.2 15	254 (10 0.0)	61 (98.4)	0 1 9 6	25 7 (1 00 .0)	56 (9 8. 2)	0 1 8 0	23 8 (9 9. 6)	7 7 (1 0 0. 0)	1 0 0 0	2 1 7 (1 0 0	9 8 (9 9 0)	0 3 1 3	1 6 1 (1 0 0. 0)	1 5 4 (9 9. 4)	0 4 9 1

Yes Live r cirr hosi s	0 (0.0)	1 (1.5)		0 (0.0)	1 (1.6)		0 (0 .0)	1 (1 .8)		1 (0 .4)	0 (0 .0)		0) 0 (0 0)	1 (1 0)		0 (0 .0)	1 (0 .6)	
No	248 (10 0.0)	68 (10 0.0)		254 (10 0.0)	62 (100.0)		25 9 (1 00 .0)	57 (1 00 .0)		23 9 (1 00 .0)	7 7 (1 0 0. 0)		2 1 7 (1 0 0 0)	9 9 (1 0 0 0)		1 6 1 (1 0 0. 0)	1 5 5 (1 0 0. 0)	
Yes	0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)		0 (0 .0)	0 (0 .0)		0 (0 .0)	0 (0 .0)		0 (0 0)	0 (0 0)		0 (0 .0)	0 (0 .0)	
roid glan d diso rder s																		
No	241 (97. 2)	65 (95. 6)	0.4 53	246 (96. 9)	60 (96.8)	1 0 0 0	25 2 (9 7. 3)	54 (9 4. 7)	0 3 9 5	23 2 (9 7. 1)	7 4 (9 6. 1)	0 7 1 0	2 0 9 (9 6	9 7 (9 8	0 7 3 0	1 5 6 (9 6. 9)	1 5 0 (9 6. 8)	1 0 0 0

													3)	0)				
Yes	7 (2.8)	3 (4.4)		8 (3.1)	2 (3.2)		7 (2 .7)	3 (5 .3)		7 (2 .9)	3 (3 .9)	•	8 (3 7)	2 (2 0)		5 (3 .1)	5 (3 .2)	
Oth er med ical con ditio n																		
No	209 (84. 3)	50 (73. 5)	0.0 62	212 (83. 5)	47 (75.8)	0 2 2 2	22 0 (8 4. 9)	39 (6 8. 4)	0 0 6	20 1 (8 4. 1)	5 8 (7 5. 3)	0 1 1 6	1 8 7 (8 6 2)	7 2 (7 2 7)	0 0 0 6	1 3 8 (8 5. 7)	1 2 1 (7 8. 1)	0 1 0 5
Yes	39 (15. 7)	18 (26. 5)		42 (16. 5)	15 (24.2)		39 (1 5. 1)	18 (3 1. 6)		38 (1 5. 9)	1 9 (2 4. 7)		3 0 (1 3 8)	2 7 (2 7 3)		2 3 (1 4. 3)	3 4 (2 1. 9)	
Fam ily hist ory																		
Dia bete s																		
No	157 (63. 3)	30 (44. 1)	0.0 07	154 (60. 6)	33 (53.2)	0 3	16 0 (6	27 (4	0 0	14 1 (5	4 6 (5	1 0	1 3 8 (4 9 (4	0 0	1 0 7 (6	8 0 (5	0 0

Yes	91 (36. 7)	38 (55. 9)		100 (39. 4)	29 (46.8)	5 8	1. 8) 99 (3 8. 2)	7. 4) 30 (5 2. 6)	64	9. 0) 98 (4 1. 0)	9. 7) 3 1 (4 0. 3)	00	6 3 6) 7 9 (3 6 4)	9 5) 5 0 (5 0 5)	25	6. 5) 5 4 (3 3. 5)	1. 6) 7 5 (4 8. 4)	1 0
Hyp erte nsio n																		
No	126 (50. 8)	32 (47. 1)	0.6 81	129 (50. 8)	29 (46.8)	0 6 7 1	13 4 (5 1. 7)	24 (4 2. 1)	0 2 4 2	12 1 (5 0. 6)	3 7 (4 8. 1)	0 7 9 3	1 1 0 (5 0 7)	4 8 (4 8 5)	0 8 0 8	9 3 (5 7. 8)	6 5 (4 1. 9)	0 0 0 7
Yes	122 (49. 2)	36 (52. 9)		125 (49. 2)	33 (53.2)		12 5 (4 8. 3)	33 (5 7. 9)		11 8 (4 9. 4)	4 0 (5 1. 9)		1 0 7 (4 9 3)	5 1 (5 1 5)		6 8 (4 2. 2)	9 0 (5 8. 1)	
Obe sity																		
No	238 (96. 0)	63 (92. 6)	0.3 30	242 (95. 3)	59 (95.2)	1 0 0 0	24 9 (9 6. 1)	52 (9 1. 2)	0 1 5 9	23 2 (9 7. 1)	6 9 (8 9. 6)	0 0 1 3	2 1 (9 7	9 0 (9 0	0 0 2 1	1 5 3 (9 5. 0)	1 4 8 (9 5. 5)	1 0 0 0

Yes Hea rt dise ase	10 (4.0)	5 (7.4)		12 (4.7)	3 (4.8)		10 (3 .9)	5 (8 .8)		7 (2 .9)	8 (1 0. 4)		· 2) 6 (2 8)	9) 9 (9 1)		8 (5 .0)	7 (4 .5)	
No	163 (65. 7) 85 (34. 3)	38 (55. 9) 30 (44. 1)	0.1 76	167 (65. 7) 87 (34. 3)	34 (54.8) 28 (45.2)	0.146	16 8 (6 4. 9) 91 (3 5.	33 (5 7. 9) 24 (4 2.	0.402	15 1 (6 3. 2) 88 (3 6.	5 0 (6 4. 9) 2 7 (3	0 8 8 7	1 4 8 (6 8 2) 6 9 (5 3 (5 3 5) 4 6 (0.0177	1 1 0 (6 8. 3) 5 1 (3	9 1 (5 8. 7) 6 4 (4	0.0977
Hyp erch olest erol emi a			0.4		40		1)	1)		8)	5. 1)	0	3 1 8)	4 6 5)		1. 7)	1. 3)	
No	161 (64. 9)	40 (58. 8)	0.4 33	161 (63. 4)	40 (64.5)	0 9 8 5	16 2 (6 2. 5)	39 (6 8. 4)	0 4 9 5	15 8 (6 6. 1)	4 3 (5 5. 8)	0 1 3 6	1 4 3 (6 5	5 8 (5 8	0 2 6 0	1 1 2 (6 9. 6)	8 9 (5 7. 4)	0 3 3

Yes	87 (35. 1)	28 (41. 2)		93 (36. 6)	22 (35.5)		97 (3 7. 5)	18 (3 1. 6)		81 (3 3. 9)	3 4 (4 4. 2)		9) 7 4 (3 4 1)	6) 4 1 (4 1 4)		4 9 (3 0. 4)	6 6 (4 2. 6)	
Hyp ertri glyc erid emi a																		
No	245 (98. 8)	65 (95. 6)	0.1 16	250 (98. 4)	60 (96.8)	0 3 5	25 5 (9 8. 5)	55 (9 6. 5)	0 .2 9 6	23 5 (9 8. 3)	7 5 (9 7. 4)	0 6 3 6	2 1 4 (9 8 6)	9 6 (9 7 0)	0 3 8 2	1 5 8 (9 8. 1)	1 5 2 (9 8. 1)	1 0 0
Yes	3 (1.2)	3 (4.4)		4 (1.6)	3 (3.2)		4 (1 .5)	2 (3 .5)		4 (1 .7)	2 (2 .6)		3 (1 4)	3 (3 0)		3 (1 .9)	3 (1 .9)	
Med icati ons																		
No	186 (75. 0)	34 (50. 0)	0.0 00	189 (74. 4)	31 (50.0)	0 0 0	19 8 (7 6. 4)	22 (3 8. 6)	0 0 0	17 2 (7 2. 0)	4 8 (6 2. 3)	0 1 4 6	1 6 7 (7 0)	5 3 (5 3 5)	0 0 0	1 2 3 (7 6. 4)	9 7 (6 2. 6)	0 0 1 1

Yes	62 (25. 0)	34 (50. 0)		65 (25. 6)	31 (50.0)		61 (2 3. 6)	35 (6 1. 4)		67 (2 8. 0)	2 9 (3 7. 7)		5 0 (2 3 0)	4 6 (4 6 5)		3 8 (2 3. 6)	5 8 (3 7. 4)	
Nu mbe r of stre ssful life even ts	0.56 ±0.7 6	0.5 7±0 .82	0.9 42	0.57 ±0.7 7	0.52± 0.76	0 5 4 0	0. 53 ±0 .7 3	0. 72 ±0 .9 2	0 1 7 9	0. 52 ± 0. 75	0. 6 8 ± 0. 8 2	0 1 2 7	$egin{array}{ccc} 0 & . & . & . & . & . & . & . & . & . &$	0 5 7 ± 0 7 7	0 9 3 4	$\begin{array}{c} 0. \\ 5 \\ 3 \\ \pm \\ 0. \\ 7 \\ 3 \end{array}$	$\begin{array}{c} 0. \\ 5 \\ 9 \\ \pm \\ 0. \\ 8 \\ 0 \end{array}$	0 6 7 4
Anx iety scor e	1.96 ±1.9 3	1.6 6±2 .01	0.1 41	1.92 ±1.9 3	1.82± 2.04	0 5 7 2	1. 97 ±1 .9 8	1. 59 ±1 .7 7	0 2 4 1	1. 92 ± 1. 99	$ \begin{array}{c} 1. \\ 8 \\ 3 \\ \pm \\ 1. \\ 8 \\ 1 \end{array} $	0 9 8 8	1 9 6 ± 1 9 7	$ \begin{array}{c} 1 \\ . \\ 7 \\ \pm \\ 1 \\ . \\ 9 \\ 0 \end{array} $	0 4 3 1	1. 8 ± 1. 9 9	$ \begin{array}{c} 1. \\ 9 \\ 2 \\ \pm \\ 1. \\ 9 \\ 2 \end{array} $	0 7 2 2
Men tal illne ss (oth er than dep ressi on)												<u> </u>						
No	237 (95. 6)	68 (10 0.0)	0.1 69	243 (95. 7)	62 (100.0)	0 1	25 0 (9	55 (9	1 0	23 2 (9	7 3 (9	0 4	2 0 8 (9 7 (9	0 5	1 5 5 (9	1 5 0 (9	1 0

Yes	11 (4.4)	0 (0.0)	-	11 (4.3)	0 (0.0)	3 0	6. 5) 9 (3 .5)	6. 5) 2 (3 .5)	000	7. 1) 7 (2 .9)	4. 8) 4 (5 .2)	72	9 5 9) 9 (4 1)	8 0)) 2 (2 0)	1 2	6. 3) 6 (3 .7)	6. 8) 5 (3 .2)	000
Med icati ons for men tal illne ss																		
No	242 (97. 6)	68 (10 0.0)	0.3 47	248 (97. 6)	62 (100.0)	0 6 0 2	25 4 (9 8. 1)	56 (9 8. 2)	1 0 0	23 5 (9 8. 3)	7 5 (9 7. 4)	0 6 3 6	2 1 1 (9 7 2)	9 (1 0 0 0)	0 2 8 2	1 5 7 (9 7. 5)	1 5 3 (9 8. 7)	0 6 8 5
Yes	6 (2.4)	0 (0.0)		6 (2.4)	0 (0.0)		5 (1 .9)	1 (1 .8)		4 (1 .7)	2 (2 .6)		6 (2 8)) (0 0)		4 (2 .5)	2 (1 .3)	
Fam ily me mbe r with men tal illne ss																		

or dep ressi on																		
No	231 (93. 1)	64 (94. 1)	1.0 00	235 (92. 5)	60 (96.8)	0 3 9 1	24 1 (9 3. 1)	54 (9 4. 7)	0 7 7 7	22 2 (9 2. 9)	7 3 (9 4. 8)	0 7 4 5	2 0 6 (9 4 9)	8 9 (8 9 9)	0 1 5 5	1 5 1 (9 3. 8)	1 4 (9 2. 9)	0 9 2 8
Yes	17 (6.9)	4 (5.9)		19 (7.5)	2 (3.2)		18 (6 .9)	3 (5 .3)		17 (7 .1)	4 (5 .2)		1 (5 1)	1 0 (1 0 1)		1 0 (6 .2)	1 (7 .1)	
Dep ressi on																		
No	238 (96. 0)	66 (97. 1)	1.0 00	243 (95. 7)	61 (98.4)	0 4 7 2	24 9 (9 6. 1)	55 (9 6. 5)	1 0 0 0	23 1 (9 6. 7)	7 3 (9 4. 8)	0 4 9 5	2 0 7 (9 5 4)	9 7 9 8 0)	0 3 5 3	1 5 8 (9 8. 1)	1 4 6 (9 4. 2)	0 1 2 4
Yes	10 (4.0)	2 (2.9)		11 (4.3)	1 (1.6)		10 (3 .9)	2 (3 .5)		8 (3 .3)	4 (5 .2)		1 0 (4 6)	2 (2 0)		3 (1 .9)	9 (5 .8)	
Med icati on																		

for dep ressi on																		
No	234 (94. 4)	66 (97. 1)	0.5 37	239 (94. 1)	61 (98.4)	0 2 1 2	24 6 (9 5. 0)	54 (9 4. 7)	1 0 0	22 8 (9 5. 4)	7 2 (9 3. 5)	0 5 5 1	2 0 3 (9 3 5)	9 7 (9 8 0)	0 1 6 5	1 5 4 (9 5. 7)	1 4 6 (9 4. 2)	0 7 3 8
Yes	14 (5.6)	2 (2.9)		15 (5.9)	1 (1.6)		13 (5 .0)	3 (5 .3)		11 (4 .6)	5 (6 .5)		1 4 6 5)	2 (2 0)		7 (4 .3)	9 (5 .8)	
Stre ssful life even t																		
No	145 (58. 5)	41 (60. 3)	0.8 95	147 (57. 9)	39 (62.9)	0 5 6 4	15 6 (6 0. 2)	30 (5 2. 6)	0 3 6 4	14 6 (6 1. 1)	4 0 (5 1. 9)	0 1 9 9	1 2 8 (5 9 0)	5 8 (5 8 6)	1 0 0	9 6 (5 9. 6)	9 0 (5 8. 1)	0 8 6 7
Yes	103 (41. 5)	27 (39. 7)		107 (42. 1)	23 (37.1)		10 3 (3 9. 8)	27 (4 7. 4)		93 (3 8. 9)	3 7 (4 8. 1)		8 9 (4 1 0)	4 1 (4 1 4)		6 5 (4 0. 4)	6 5 (4 1. 9)	

Anx ious																		
No	164 (68. 4)	50 (73. 5)	0.3 32	169 (66. 8)	45 (72.6)	0 4 7 0	17 1 (6 6. 0)	43 (7 6. 8)	0 1 5 9	16 1 (6 7. 6)	5 3 (6 8. 8)	0 9 5 8	1 4 7 (6 8 1)	6 7 (6 7 7)	1 0 0	1 0 9 (6 7. 7)	1 0 5 (6 8. 2)	1 0 0
Yes	83 (33. 6)	18 (26. 5)		84 (33. 2)	17 (27.4)		88 (3 4. 0)	13 (2 3. 2)		77 (3 2. 4)	2 4 (3 1. 2)		6 9 (3 1 9)	3 2 (3 2 3)		5 2 (3 2. 3)	4 9 (3 1. 8)	
Cre atini ne level s (mg/ dL)	0.80 ±0.4 8	0.8 7±0 .36	0.0 01	0.81 ±0.5 1	0.83± 0.18	0 0 3 9	0. 79 ±0 .4 7	0. 91 ±0 .3 9	0 0 0 0	0. 82 ± 0. 49	$\begin{array}{c} 0. \\ 8 \\ 1 \\ \pm \\ 0. \\ 3 \\ 5 \end{array}$	0 7 6 2	0 7 9 ± 0 5 1	0 8 7 ± 0 3 1	0 0 0 0	0. 8 3 ± 0. 5 9	0. 7 9 ± 0. 2 7	0 5 2 6
Vita min D stat us																		
Opti mal	102 (41. 5)	20 (29. 9)	0.1 13	101 (40. 1)	21 (34.4)	0 5 0 5	10 2 (3 9. 7)	20 (3 5. 7)	0 6 8 8	99 (4 1. 8)	2 3 (3 0. 3)	0 0 9 8	9 0 (4 1 7)	3 2 (3 3 0)	0 1 8 3	7 1 (4 4. 1)	5 1 (3 3. 6)	0 0 7 2

Sub opti mal WC	144 (58. 5)	47 (70. 1)		151 (59. 9)	40 (65.6)		15 5 (6 0. 3)	36 (6 4. 3)		13 8 (5 8. 2)	5 3 (6 9. 7)		1 2 6 (5 8 3)	6 5 (6 7 0)		9 0 (5 5. 9)	1 0 1 (6 6. 4)	
(cm) Nor mal	156 (62. 9)	5 (7.4)	0.0 00	142 (55. 9)	19 (30.6)	0 0 0 1	14 9 (5 4. 5)	12 (2 1. 1)	0 0 0	13 2 (5 5. 2)	2 9 (3 9. 7)	0 0 1 1	1 3 1 (6 0 4)	3 0 (3 0 3)	0 0 0			
Abd omi nal obes ity	92 (37. 1)	63 (92. 6)		112 (44. 1)	43 (69.4)		11 0 (4 2. 5)	45 (7 8. 9)		10 7 (4 4. 8)	4 8 (6 2. 3)		8 6 (3 9 6)	6 9 (6 9 7)				
Bod y com posi tion (tot al bod y fat %)	29.6 7±7. 84	34. 81± 7.1 6	0.0 00	30.2 6±7. 81	32.88 ±8.33	0 0 2 1	30 .1 9± 7. 66	33 .4 1± 81 .8 4	0 0 6	30 .3 7± 8. 05	3 2. 0 6 ± 7. 6 5	0 1 0 9	2 9 8 8 ± 7 9 7	3 2 7 6 ± 7 6 6	0.003	$2 \\ 6. \\ 5 \\ 1 \\ \pm \\ 6. \\ 0 \\ 6$	$\begin{array}{c} 3 \\ 5. \\ 2 \\ \pm \\ 7. \\ 2 \\ 8 \end{array}$	0.0000000000000000000000000000000000000
BM I																		

(kg/ m ²)																		
Und erwe ight	2 (0.8)	0 (0.0)	0.0 00	2 (0.8)	0 (0.0)	0 0 0 0	2 (0 .8)	0 (0 .0)	0 0 0 0	2 (0 .8)	0 (0 .0)	0 0 1 9	2 (0 9)	0 (0 0)	0 0 0 0	2 (1 .2)	0 (0 .0)	0 0 0 0
Nor mal weig ht	116 (46. 8)	0 (0.0)		114 (44. 9)	2 (3.2)		11 1 (4 2. 9)	5 (8 .8)		96 (4 0. 2)	2 0 (2 6. 0)		1 0 6 (4 8 8)	1 0 (1 0 1)		9 7 (6 0. 2)	1 9 (1 2. 3)	
Ove rwei ght	96 (38. 7)	25 (36. 8)		92 (36. 2)	29 (46.8)		97 (3 5. 5)	24 (4 2. 1)		92 (3 8. 5)	2 9 (3 7. 7)		7 6 (3 5 0)	4 5 (4 5 5)		5 9 (3 6. 6)	6 2 (4 0. 0)	
Obe se	34 (13. 7)	43 (63. 2)		46 (18. 1)	31 (50.0)		49 (1 8. 9)	29 (4 9. 1)		49 (2 0. 5)	2 8 (3 6. 4)		3 3 (1 5 2)	4 4 4 4 4)		3 (1 .9)	7 4 (4 7. 7)	
CR P ³ (mg/ L)																		
Mod erate	119 (48. 0)	11 (16. 2)	0.0 00	111 (43. 7)	19 (30.6)	0 0 8 4	11 5 (4 4. 4)	15 (2 6. 3)	0 0 1 8	11 3 (4 7. 3)	1 7 (2 2. 1)	0 0 0 0	1 1 (5 1	1 9 (1 9	0 0 0 0	8 3 (5 1. 6)	4 7 (3 0. 3)	0 0 0 0

Hig h	129 (52. 0)	57 (83. 8)	143 (56. 3)	43 (69.4)	14 4 (5 5. 6)	42 (7 3. 3)	12 6 (5 2. 7)	6 0 (7 7. 9)	2) 1 0 6 (4 8	2) 8 0 (8 0	7 8 (4 8. 4)	1 0 8 (6 9. 7)	
									8 8)	8)		7)	

<u>Table 2:</u> Association between metabolic syndrome and its components and sociodemographic characteristics and biochemical measurements among study participants

BP, blood pressure; high blood pressure as defined by systolic/diastolic BP $\ge 130/85$ mmHg IFG, impaired fasting glucose; IFG as defined by FG ≥ 100 mg/dl

TG, triglyceride; high TG as defined by TG≥150 mg/dl

HDLc, high density lipoprotein cholesterol; low HDLc as defined by: <40 mg/dl in men and <50 mg/dl in women

WC, waist circumference; high WC as defined by ≥ 102 cm in men and ≥ 88 cm in women LDLc, low density lipoprotein cholesterol; high LDLc as defined by LDL ≥ 100 mg/dl

BMI, body mass index; underweight as defined BMI <18.5 kg/m2, normal weight as defined by BMI between 18.5 and 24.9 kg/m2, overweight as defined by BMI between 25 and 29.9 kg/m2, obese as defined by BMI >30 kg/m2

CRP, c-reactive protein; low = as defined by CRP <1 mg/l, moderate as defined by CRP between 1 and 3 mg/l, high as defined by CRP >3 mg/l

	β	S.E	p-value	OR	95% C.I. for EXP (B)		R-square
					Lower	Upper	
Metabolic syndrome						L	
Occasional smokers*	1.434	0.517	0.006	4.197	1.523	11.564	0.322- 0.947

Female*	-2.339	0.533	0.000	0.096	0.034	0.274	
High school degree*	-1.891	0.745	0.011	0.151	0.035	0.649	
University bachelor's degree*	-1.992	0.753	0.008	0.136	0.031	0.597	
University graduate's degree*	-1.713	0.767	0.025	0.180	0.040	0.811	
Body composition (fat %)	0.163	0.035	0.000	1.177	1.098	1.261	
Metabolic syndrome components							
Raised blood pressure							
Age	0.045	0.018	0.012	1.046	1.010	1.083	0.201- 0.319
Female*	-1.111	0.376	0.003	0.329	0.158	0.688	0.517
Clinical diagnosis of hypertension*	1.239	0.548	0.024	3.452	1.180	10.103	
Waist circumference risky*	0.785	0.384	0.041	2.193	1.033	4.653	
Impaired							
fasting glucose							
Age	0.042	0.019	0.031	1.043	1.004	1.083	0.269- 0.440
Female*	-1.360	0.433	0.002	0.257	0.110	0.600	
Clinical diagnosis of diabetes*	2.670	0.818	0.001	14.439	2.905	71.778	

Waist circumference risky*	1.489	0.449	0.001	4.432	1.839	10.682	
Decreased HDL cholesterol							
Daily smokers*	0.951	0.355	0.007	2.588	1.290	5.191	0.147- 0.219
Former smokers*	1.735	0.852	0.042	5.668	1.067	20.121	0.217
Clinical diagnosis of hypertension^	-1.187	0.558	0.033	0.305	0.102	0.911	
Family history of diabetes*	1.290	0.610	0.035	3.632	1.099	12.006	
High CRP level*	0.982	0.342	0.004	2.670	1.365	5.222	
High triglyceride level							
Former smokers*	2.755	1.372	0.045	15.720	1.069	231.187	0.342- 0.481
Age	0.046	0.017	0.006	1.047	1.014	1.082	
Female*	-1.652	0.359	0.000	0.192	0.095	0.388	
High school degree*	-2.162	0.779	0.006	0.115	0.025	0.530	
University bachelor's degree*	-1.712	0.759	0.024	0.181	0.041	0.799	
Family history of obesity*	1.944	0.719	0.007	6.983	1.706	28.586	
Waist circumference risky*	0.972	0.356	0.006	2.645	1.315	5.317	

High CRP levels*	1.076	0.361	0.003	2.934	1.447	5.952	
Waist circumference risky							
Occasional smokers*	0.967	0.422	0.022	2.629	1.150	6.012	0.424- 0.566
Female*	-1.229	0.418	0.003	0.293	0.129	0.663	
University graduate's degree*	-1.935	0.973	0.047	0.144	0.021	0.972	
Clinical diagnosis of hypertension*	1.638	0.692	0.018	5.144	1.326	19.962	
Body composition (Fat %)	0.261	0.035	0.000	1.298	1.212	1.390	

<u>Table 3:</u> Association between smoking status and metabolic syndrome and its components, as assessed by logistic regression

*Reference group: never smokers/ males/ less than high school degree/ no clinical diagnosis of hypertension/ normal waist circumference/ no clinical diagnosis of diabetes/ moderate CRP levels/ no clinical diagnosis of obesity