Impact of active smoking, passive smoking, and smoking cessation on overweight and obesity: First national cross-sectional study

Amani Haidar¹, Lama Jouni¹, Mohamad Koubar², Sarine El Daouk³, Maha Hoteit¹

¹Department of Nutrition and Dietetics, Faculty of Public Health, Section I, Lebanese University, Beirut, Lebanon, ²Department of Laboratory Science, Faculty of Public Health, Section I, Lebanese University, Lebanon, ³Department of Mothers and Children Care Center, University Medical Center, Faculty of Public Health, Section I, Lebanese University, Beirut, Lebanon

Correspondence to: Dr. Maha Hoteit, E-mail: m.hoteit@ul.edu.lb

Received: September 26, 2016; Accepted: November 12, 2016

ABSTRACT

Background: Smoking and obesity are two major modifiable leading causes of preventable mortality and morbidity worldwide. The relationship between smoking and body weight is still incompletely understood. Objective: To examine the impact of active smoking, passive smoking, and smoking cessation on overweight and obesity in the Lebanese population, aged 18 years and above in Beirut. Materials and Methods: In this cross-sectional study, 300 Lebanese adults aged 18 years and older were recruited. The Global Tobacco Surveillance System questionnaire was used to collect the data about smoking in the population studied. Anthropometric measurements (height, weight, and body mass index [BMI]) were also performed to test possible association between smoking status and body weight. Results: No significant association exists between current smoking and body weight (P = 0.166), but smokers may have 2.59 times more chance to be overweight, 2.37 times to be obese, and 2.68 times to have a morbid obesity. Former smokers have a 0.15 times higher risk of being overweight compared to nonsmokers (odds ratio = 0.15, 95% confidence interval: 0.036-0.70]. Passive smoking at home was associated with lower BMI compared to nonsmokers (P = 0.034). Conclusions: We conclude that smoking may be a crucial factor in overweight and obesity among men and women mainly who have stopped smoking. It may be expected that from a public health perspective the problems from weight gain do not outweigh the benefits from smoking cessation. There is a large unused potential to prevent weight gain among smokers before and after they quit. Overweight might probably be prevented by support of smokers who quit, for example, by the provision of information on weight control or by teaching coping skills. Furthermore, passive smokers should be provided with the information that the nicotine smoked prevents weight gain during the time of exposure and once quit weight gain will be unpreventable.

KEY WORDS: Body Mass Index; Overweight; Obesity; Smoking

INTRODUCTION

Smoking and obesity are two major modifiable leading causes of preventable mortality and morbidity worldwide.^[1,2]

Access this article online					
Website: http://www.ijmsph.com	Quick Response code				
DOI: 10.5455/ijmsph.2017.10956612112016					

Adult smoking prevalence in the Lebanese Population has been reported to be as high as 35% for females and 45% for males, which is considered to be higher than the regional average in North Africa region and the Middle East (7% and 38%, respectively).^[3]

On the other hand, overweight and/or obesity are also major risk factors for noncommunicable diseases. In Lebanon, the prevalence of overweight appeared stable between years 1997 and 2009 in adults aged 20 years and above (37.0% vs. 36.8%), whereas the prevalence of obesity increased significantly (17.4% vs. 28.2% in adults).^[4]

International Journal of Medical Science and Public Health Online 2016. © 2016 Amani Haidar et al. This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), allowing third parties to copy and redistribute the material in any medium or format and to remix, transform, and build upon the material for any purpose, even commercially, provided the original work is properly cited and states its license.

The relationship between smoking and body weight is still incompletely understood.

From one point of view, body weight, or body mass index (BMI) is lower in smokers compared with nonsmokers^[5-7] and this could be explained by the presence of nicotine in tobacco^[8] Moreover, there is a popular belief among both smokers and nonsmokers that smoking is an efficient and effective way to control body weight.^[9]

From another point of view, studies indicate that heavy smokers tend to have greater body weight than light smokers.^[10,11] In addition, there is also increasing evidence that smoking may have a role in body fat distribution which is in turn associated with central obesity and insulin resistance.^[12]

In a context of the worldwide obesity epidemic and a high prevalence of smoking, the relation between smoking and body weight has major public health relevance. Therefore, the aim of the study was to determine the relationship between smoking behavior and being obese or overweight, among Lebanese population, aged 18 years and above in Beirut.

MATERIALS AND METHODS

Population and Study Design

A cross-sectional study was conducted in Beirut - Lebanon with a population sample of 300 Lebanese people (151 women and 149 men) with a mean age of 40.5 ± 17.5 years, chosen randomly from one primary health-care center.

Data Collection

Each subject was interviewed by trained health-care professional, using the validated modified Global Adult Tobacco Survey questionnaire translated to Arabic language. It includes information on respondents' background characteristics, tobacco use (smoking and smokeless), cessation, second-hand smoke, economics, media, and knowledge, attitudes, and perceptions toward tobacco use. Body weight and height were measured by health-care professionals at the set of data collection. Participants, wearing light indoor clothing and without shoes, had their weight measured to the nearest 0.1 kg using a balance scale, and height measured to the nearest 0.01 m using a stadiometer.

BMI was calculated by dividing weight (kg) by the square of height (m), and based on the BMI, study participants were classified into five categories; Underweight (BMI <18.5 kg/m²), normal body weight (BMI between 18.5 and 24.9 kg/m²), overweight (BMI between 24.9 and 29.9 kg/m²), Obese (BMI between 30 and 39.9 kg/m²), and morbid obesity (BMI \geq 40 kg/m²). Weight and height were measured twice and the means of the readings were used in our analysis.

Statistical Analysis

The association between body weight and each smoking status (smokers, former smokers, and nonsmokers) was examined, using logistic regression, Chi-square and *t*-test.

Further, the effect of public awareness on cigarette cessation was examined using Chi-square analysis. Data management and analysis was conducted using SPSS (Version 19.0).

RESULTS

Demographic Characteristics of the Study Participants

A total of 300 study subjects were randomly recruited, of which 50.3% were men. 58% of men and 42% of women were current smokers, 13.4% of men and 10.6% of women were former smokers, and 33.6% of men and 51% of women were nonsmokers (P = 0.009). Among the entire smoking population, 59% smokes cigarettes and at least 40% smokes water pipe. Few people combine both. 38% of the population was overweight, 22% were obese and <10% were underweight or morbidly obese. Remaining subjects had a normal body weight. The mean value of body weight, in kg, in nonsmokers was the lowest ($75.3 \pm 17.1 \text{ kg}$) compared to that of current smokers ($79.50 \pm 15.29 \text{ kg}$) and former smokers ($81.7 \pm 20.5 \text{ kg}$) (P = 0.048). All participants were matched in age and gender. The characteristics of the study participants are presented in Table 1.

Association between Body Weight Status (Overweight and Obesity) and Smoking Status

The relation between body weight and each of the following smoking status was examined: Current smoking, former smoking, and nonsmoking (Table 2). The majority (38%) of the current smokers and former smokers (41.6%) had overweight but not the nonsmokers where the majority had normal body weight to overweight (33.8%-37.7% respectively). There was no significant association between current smoking and the body weight of study subjects (P = 0.58), but as odds, we found that smokers may have 2.59 times more chance to be overweight, 2.37 times to be obese, and 2.68 times to have morbid obesity (data not shown). When stratifying this analysis by gender, no significant association was found (P > 0.05).

The mean of BMI in smokers was $27.25 \pm 4.85 \text{ kg/m}^2$ which was similar to that of nonsmoker's mean of BMI $(27.49 \pm 6.53 \text{ kg/m}^2)$ (P = 0.7). No difference was observed in women or men (P > 0.05). However, the mean of BMI was high in former smokers ($31.8 \pm 5.9 \text{ kg/m}^2$) compared to non-smokers ($26.5 \pm 6.16 \text{ kg/m}^2$) (P < 0.001). Stratifying this analysis by gender, results show that the mean of BMI in former smokers women ($33.1 \pm 7.4 \text{ k/m}^2$) was higher than nonsmokers women ($26.5 \pm 6.4 \text{ kg/m}^2$) (P = 0.007). Similarly,

Characteristics	Overall N=300 (%)	Women <i>N</i> =151 (%)	Men <i>N</i> =149 (%)	P value*
Age				
18-39 years	154 (51.3)	76 (50.3)	73 (49)	0.05
(73) 40+	146 (48.7)	75 (49.7)	76 (51)	
Anthropometric measurements				
Height (cm)	168.6±10	162.1±6.9	175.3±8.29	< 0.001
Weight (kg)	77.9±16.9	71.8±15.7	84.2±15.7	< 0.001
BMI (kg/m ²)	27.3±5.7	27.2±6.3	27.4±5.03	0.079
BMI categories				
<18.5 kg/m ² (underweight)	7 (2.3)	6 (4)	1 (0.7)	0.3
18.5-24.9 kg/m ² (normal body weight)	99 (33)	48 (32)	48 (32.2)	
25-29.9 kg/m ² (overweight)	113 (37.7)	54 (36)	61 (40.9)	
30-39.9 kg/m ² (obese)	68 (22.7)	35 (23.3)	33 (22.1)	
\geq 40 kg/m ² (morbid obesity)	13 (4.3)	7 (4.7)	6 (4)	
Smoking status				
Smoker	137 (45.7)	58 (38.4)	79 (53)	0.009
Former-smoker	36 (12)	16 (10.6)	20 (13.4)	
Non-smoker	127 (42.3)	77 (51)	50 (33.6)	
Type of smoking [#]				
Cigarette	88 (59)	31 (35.2)	57 (64.8)	0.03
Water pipe	67 (45)#	33 (49.3)	34 (50.7)	0.1
Passive smoking				
At home				
Yes	163 (54.3)	91 (60.3)	72 (48.3)	0.038
No	137 (45.7)	60 (39.7)	77 (51.7)	
At work				
Yes	57 (19)	17 (11.3)	40 (26.8)	0.24
No	113 (37.7)	44 (29.1)	69 (46.3)	
Unemployed	130 (43)	90 (59.6)	40 (26.8)	

Table 1: Demographic and anthropometric characteristics of the population

BMI: Body mass index, p = 0.05

Table 2: The relation between body weight and smoking status

Characteristics	Smokers	Non-smokers	Former smokers	<i>P</i> value [†]
Age	42.1±16.6	37.4±17.5	36±45.7	0.01
Gender (%)				
Men	79 (57.7)	50 (39.4)	20 (55.6)	0.009
Women	58 (42.3)	77 (60.6)	16 (44.4)	
Anthropometrics				
Height	169.7±9.4	167.3±10.6	169.4±10	0.14
Weight	79.5±15.2	75.3±17.1	81.7±20.5	0.04
BMI	27.6±4.88	26.8±6.3	28.3±6.2	0.32
BMI categories (%)				
<18.5 kg/m ² (underweight)	1 (0.7)	6 (4.7)	0 (0)	0.009
18.5-24.9 kg/m ² (normal body weight)	45 (32.8)	44 (34.6)	10 (27.8)	
25-29.9 kg/m ² (overweight)	51 (37.2)	47 (37)	15 (41.7)	
30-39.9 kg/m ² (obese)	36 (26.3)	24 (18.9)	8 (22.2)	
\geq 40 kg/m ² (morbid obesity)	4 (2.9)	6 (4.7)	3 (8.3)	

Continuous variables are summarized as mean \pm standard deviation with statistical comparison using *t*-test. Categorical variables are summarized as count (%) with statistical comparison using Chi-square. [†]*P* value for difference between genders. BMI: Body mass index

in men where BMI was $30.9 \pm 4.9 \text{ kg/m}^2$ in former smokers and $26.7 \pm 4.8 \text{ kg/m}^2$ in nonsmokers (P = 0.007). Former smokers have a 0.15 times higher risk of being overweight compared to nonsmokers (odds ratio [OR] = 0.15, 95% confidence interval [CI]: 0.036-0.70). When adjusted the backward binary logistic regression test by age, since gender was already adjusted, results show that former smokers and age were independent risk factor for this association (OR = 1.04 and 95% CI: 1.02-1.06).

To investigate the association between obesity and smoking in current active smokers, passive smokers, and former smokers, χ^2 test and binary logistic regression analyses were performed, but no significant results were obtained (P > 0.05) (Data not shown).

Concerning the public awareness related to smoking cessation, although all smokers had noticed the health warning label on cigarettes packages, the majority of the smokers did not think about quitting (P = 0.04).

Second Hand Smoking and Overweight and Obesity

Passive smoking was also evaluated at home and work. Testing the association between passive smoking at home and BMI, results show that the mean values of BMI were $28.2 \pm 5.8 \text{ kg/m}^2$ for subjects with no exposure to passive smoking at home compared to $26.7 \pm 5.2 \text{ kg/m}^2$ for subjects exposed to second hand smoking in their homes (P = 0.034). When splitting the analysis by gender, the results show that there is no effect of passive smoking on body weight in men (P=0.2). However, women exposed to passive smoking had a low BMI ($26.6 \pm 5.8 \text{ kg/m}^2$) compared to non-exposed women $(28.5 \pm 6.2 \text{ kg/m}^2)$ (P = 0.06). Binary logistic regressions show an odd of 1.6 higher risk of overweight with 95 % CI: 1.0-2.7 in nonpassive smokers compared to passive smokers. No association was found between passive smoking at work and body weight (P = 0.38). Furthermore, no association was found between passive smoking and obesity (P > 0.05).

DISCUSSION

Our study results show that, compared to nonsmokers, there was no observed association between current smoking and body weight in Lebanese adult's population. Although the relation studied between current smoking and body weight, did not appear to be significant, 38% of the current smokers and 41.6% of former smokers were overweight, not the nonsmokers where the majority had a mean of body weight ranging between normal to overweight (33.8-37.7%, respectively). Furthermore, despite being not significant, smoking increased the likelihood of being overweight by 2.59 times, being obese by 2.59 times and to have morbid obesity by 2.68 times (data not shown). Our findings are in accordance with John et al. who reported that the number of cigarette per day was not associated with overweight or

obesity in current smokers in a sample of 7124 subjects.^[13] However, our study results contradict many previous studies that showed that smokers tend to have a lower BMI compared to nonsmokers.^[7] One major study is the NHANES II study (1976-1980), which showed that smokers weighed less than nonsmokers and body leanness increased with the duration of smoking, rather with the intensity of smoking.^[14]

Concerning the passive smoking, it appeared, through our study, that the subjects who are exposed to passive smoking at home have a lower BMI compared to those not exposed to such type of smoking mainly the women. Our finding contradicts the only relation revealed by Bernstein et al. in which, women passive smokers were heavier than never exposed participants, explained by a hypothesis that passive smokers were also exposed to unhealthy dietary pattern.^[15] Our finding concerning passive smoking is explained by the presence of nicotine which in turn reduces body weight by raising the resting metabolic rate while weakening the expected increase in food intake in response to the increase in metabolic rate. Nicotine could have an acute anorexic effect; hunger and food consumption were negatively related whereas fullness and satiety were positively associated with the increasing doses of nicotine.^[16] Nicotine does not affect the hunger sensations but it results in smaller caloric intake during a meal, in both smokers and non-active smokers.^[17] It has many potential effects on central nervous system regulation of eating and energy expenditure (EE), due to the fact that the regulation of eating behavior and metabolic rate by the brain occurs in the hypothalamus. Nicotine metabolic effect is represented by increasing the EE through which smoking a single cigarette induced a 3% rise in EE within 30 min^[18] and smoking four cigarettes each containing 0.8 mg nicotine increased resting EE by 3.3% for 3 h.^[19] Another hypothesis is that the low intake of food is physiologically and mainly related to leptin which is released from adipose tissue in proportion to the amount of adipose tissues and acts on the central nervous system to redact the food intake and increase metabolic rate. Studies comparing leptin levels in smokers with those in nonsmokers show different confusing results. However, it has been shown that nicotine may enhance the effects of leptin in the brain through strengthening the binding of the leptin or increasing the sensitivity of downstream transduction cascades.^[20] Furthermore, the release of hormones such as, dopamine, norepinephrine serotonin, and y-aminobutyric acid by the central nervous system affects the brain chemicals that will in-turn decreases eating and increase metabolic rate (such as pro-opiomelanocortin and cocaine-amphetamineregulated transcript) as well as those that suppress eating and decrease metabolic rate (such as neuropeptide Y, Agouti-related peptide, melanin-concentrating hormone, and orexin).^[21] Another explanation could be elucidated by an opposite alliance revealed previously between current smokers and body weight and most particularly central obesity, explaining our study results, where it was shown the nicotine is related to insulin resistance, which may alter the body

composition, mainly increasing the visceral fat mass.^[22] This is in accordance with Khaw et al. who reported that's smokers tends to have a higher percentage of visceral fat compared to total fat in the body and thus a higher waist-to-hip ratio than nonsmokers^[23] which was mainly due the nicotinic stimulated release of cortisol and altered balance of sex hormones, in both males and females. Through which, women who smokes, had no changes in estrogen concentrations, higher androgen concentrations^[23] and a lower bioavailability of estrogens^[24] compared to nonsmoker women, which is in turn associated with visceral fat accumulation.^[25] And, in men, smoking may decrease testosterone concentrations, causing weight gain.^[15]

Another association with body weight was observed in former smokers who have a higher BMI, and a higher risk of being overweight compared to nonsmokers. Our results are in accordance with the findings of John et al., who found that men who formerly had smoked more than 30 cigarettes a day had 5 times higher risk of being obese compared to men who had never smoked (OR = 5 and 95% CI: 2.5-10).^[13] In addition, Scherr et al. showed that marked weight gain following smoking cessation was characterized by a wide margin of body weight changes.^[26] The results are consistent with the evidence that revealed increased EE from current smoking and from the nicotine supply.^[19,27-29] Our results suggest that increased BMI among former smokers might be due to the evidence that a high amount of nicotine consumed is related to low food intake, change of the metabolic rate or decreased body weight set point.^[30-33] With the smoking cessation and nicotine abstinence, all the above mechanism will be inverted and one possible explanation is that the anorexic effects of nicotine on the brain are reversed, thus resulting in increased hunger.^[34] Research also suggests that the absence of nicotine increases the rewarding value of food,^[34] in other nicotine withdrawal produces a less pleasure derived from typical reinforcers,^[35] thus a greater amounts of rewarding foods rich in sugar and fats may help achieve a pleasure similar to that derived from smoking,^[36] eventually leading to weight gain. Moreover, the amount of body weight gained after smoking cessation is considered to be highly variable, with an average of 4.5 kg, within 6-12 months following smoking cessation, and some smokers (~13%) may gain much more weight that could reach 10 kg.^[5]

In Lebanon the current law (Law No. 394/95), to encourage quitting smoking, requires one text warning ("The ministry of health warns you that tobacco use leads to dangerous and deadly diseases") on both the front and back surfaces of tobacco product packages, but this law did not affect the smoking population.^[37] Thus, more health campaigns could be further applied to reduce smoking as much as possible.

Strengths and Limitation

To our knowledge, this is the first national and regional study on the passive smoking, active smoking, and smoking cessation and its impact on weight in adults. Limitation of our approach includes, first, that the study is cross-sectional. Second, there was missing information about the dietary intake, physical activity, socioeconomic status, and education that were not taken into consideration which may either have a direct or indirect effect on body weight. Third, the study sample was only obtained from one province of Lebanon (Beirut), thus the findings of this study may not be representative. In addition, the sample size was small because we had a small budget and limited time. Fourth, the data were collected regardless of participant's medical history which may have a direct relation with being overweight or obese.

CONCLUSION

We conclude that smoking may be a crucial factor in overweight and obesity among men and women mainly who have stopped smoking. It may be expected that from a public health perspective the problems from weight gain do not outweigh the benefits from smoking cessation. There is a large unused potential to prevent weight gain among smokers before and after they quit. Overweight might probably be prevented by support of smokers who quit, for example, by the provision of information on weight control or by teaching coping skills. Furthermore, passive smokers should be provided with the information that the nicotine smoked prevents weight gain during the time of exposure and once quitted weight gain will be unpreventable.

ACKNOWLEDGMENT

The research received no specific grant from any funding agency in the public, commercial or not-for-profit sector.

REFERENCES

- 1. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. JAMA. 2004;291(10):1238-45.
- 2. Haslam DW, James WP. Obesity. Lancet. 2005;366(9492):1197-209.
- National Tobacco Control Provisions; 2002. Available from: http://www.who.int/tobacco/media/en/Lebanon.pdf. [Last assessed on 2016 Jun 7].
- 4. Nasreddine L, Naja F, Chamieh MC, Adra N, Sibai AM, Hwalla N. Trends in overweight and obesity in Lebanon: Evidence from two national cross-sectional surveys (1997 and 2009). BMC Public Health 2012;12:798.
- 5. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. N Engl J Med. 1991;324(11):739-45.
- Shimokata H, Muller DC, Andres R. Studies in the distribution of body fat. III. Effects of cigarette smoking. JAMA. 1989;261(8):1169-73.
- Huot I, Paradis G, Ledoux M; Quebec Heart Health Demonstration Project research group. Factors associated with overweight and obesity in Quebec adults. Int J Obes Relat Metab Disord. 2004;28(6):766-74.

- Hofstetter A, Schutz Y, Jéquier E, Wahren J. Increased 24-hour energy expenditure in cigarette smokers. N Engl J Med. 1986;314(2):79-82.
- Potter BK, Pederson LL, Chan SS, Aubut JA, Koval JJ. Does a relationship exist between body weight, concerns about weight, and smoking among adolescents? An integration of the literature with an emphasis on gender. Nicotine Tob Res. 2004;6(3):397-425.
- Bamia C, Trichopoulou A, Lenas D, Trichopoulos D. Tobacco smoking in relation to body fat mass and distribution in a general population sample. Int J Obes Relat Metab Disord. 2004;28(8):1091-6.
- Eliasson B. Cigarette smoking and diabetes. Prog Cardiovasc Dis. 2003;45(5):405-13.
- Houston TK, Person SD, Pletcher MJ, Liu K, Iribarren C, Kiefe CI. Active and passive smoking and development of glucose intolerance among young adults in a prospective cohort: CARDIA study. BMJ. 2006;332(7549):1064-9.
- John U, Meyer C, Rumpf HJ, Schumann A, Dilling H, Hapke U. No considerable long-term weight gain after smoking cessation: Evidence from a prospective study. Eur J Cancer Prev. 2005;14(3):289-95.
- 14. Andersson K, Arner P. Systemic nicotine stimulates human adipose tissue lipolysis through local cholinergic and catecholaminergic receptors. Int J Obes Relat Metab Disord. 2001;25(8):1225-32.
- 15. Meikle AW, Liu XH, Taylor GN, Stringham JD. Nicotine and cotinine effects on 3 alpha hydroxysteroid dehydrogenase in canine prostate. Life Sci. 1988;43(23):1845-50.
- Jessen A, Buemann B, Toubro S, Skovgaard IM, Astrup A. The appetite-suppressant effect of nicotine is enhanced by caffeine. Diabetes Obes Metab. 2005;7(4):327-33.
- Perkins KA, Epstein LH, Stiller RL, Fernstrom MH, Sexton JE, Jacob RG, et al. Acute effects of nicotine on hunger and caloric intake in smokers and nonsmokers. Psychopharmacology (Berl). 1991;103(1):103-9.
- Dallosso HM, James WP. The role of smoking in the regulation of energy balance. Int J Obes. 1984;8(4):365-75.
- Collins LC, Cornelius MF, Vogel RL, Walker JF, Stamford BA. Effect of caffeine and/or cigarette smoking on resting energy expenditure. Int J Obes Relat Metab Disord. 1994;18(8):551-6.
- Jo YH, Talmage DA, Role LW. Nicotinic receptormediated effects on appetite and food intake. J Neurobiol. 2002;53(4):618-32.
- Valassi E, Scacchi M, Cavagnini F. Neuroendocrine control of food intake. Nutr Metab Cardiovasc Dis. 2008;18(2):158-68.
- White MA, Peters EN, Toll BA. Effect of binge eating on treatment outcomes for smoking cessation. Nicotine Tob Res. 2010;12(11):1172-5.
- Khaw KT, Tazuke S, Barrett-Connor E. Cigarette smoking and level of adrenal androgens in postmenopausal women. N Engl J Med. 1988;318(26):1705-9.
- 24. Michnovicz JJ, Hershcopf RJ, Naganuma H, Bradlow HL, Fishman J. Increased 2-hydroxylation of estradiol as a possible mechanism for the anti-estrogenic effect of cigarette smoking.

N Engl J Med. 1986;315(21):1305-9.

- Björntorp P. Abdominal obesity and the development of noninsulin-dependent diabetes mellitus. Diabetes Metab Rev. 1988;4(6):615-22.
- 26. Scherr A, Seifert B, Kuster M, Meyer A, Fagerstroem KO, Tamm M, et al. Predictors of marked weight gain in a population of health care and industrial workers following smoking cessation. BMC Public Health. 2015;15:520.
- 27. Walker JF, Collins LC, Rowell PP, Goldsmith LJ, Moffatt RJ, Stamford BA. The effect of smoking on energy expenditure and plasma catecholamine and nicotine levels during light physical activity. Nicotine Tob Res. 1999;1(4):365-70.
- Warwick PM, Busby R. Prediction of twenty-four-hour energy expenditure in a respiration chamber in smokers and nonsmokers. Eur J Clin Nutr. 1993;47(8):600-3.
- 29. Audrain JE, Klesges RC, DePue K, Klesges LM. The individual and combined effects of cigarette smoking and food on resting energy expenditure. Int J Obes. 1991;15(12):813-21.
- Perkins KA. Effects of tobacco smoking on caloric intake. Br J Addict. 1992;87(2):193-205.
- Hughes JR, Hatsukami DK. Effects of three doses of transdermal nicotine on post-cessation eating, hunger and weight. J Subst Abuse. 1997;9:151-9.
- Miyata G, Meguid MM, Varma M, Fetissov SO, Kim HJ. Nicotine alters the usual reciprocity between meal size and meal number in female rat. Physiol Behav. 2001;74(1-2):169-76.
- Li MD, Kane JK, Konu O. Nicotine, body weight and potential implications in the treatment of obesity. Curr Top Med Chem. 2003;3(8):899-919.
- Spring B, Pagoto S, McChargue D, Hedeker D, Werth J. Altered reward value of carbohydrate snacks for female smokers withdrawn from nicotine. Pharmacol Biochem Behav. 2003;76(2):351-60.
- 35. Kenny PJ, Markou A. Nicotine self-administration acutely activates brain reward systems and induces a long-lasting increase in reward sensitivity. Neuropsychopharmacology. 2006;31(6):1203-11.
- 36. Cryan JF, Bruijnzeel AW, Skjei KL, Markou A. Bupropion enhances brain reward function and reverses the affective and somatic aspects of nicotine withdrawal in the rat. Psychopharmacology (Berl). 2003;168(3):347-58.
- Country Details for Lebanon; 2015. http://www. tobaccocontrollaws.org/legislation/country/lebanon/pl-healthwarnings. [Last assessed on 2016 Jun 7].

How to cite this article: Haidar A, Jouni L, Koubar M, El Daouk S, Hoteit M. Impact of active smoking, passive smoking, and smoking cessation on overweight and obesity: First national cross-sectional study. Int J Med Sci Public Health 2017;6(3):577-582.

Source of Support: Nil, Conflict of Interest: None declared.