

Effect of Face to Face Nutrition Counseling on Metabolic Syndrome Parameters in Cigarette Quitters

Jalal Bohlooli^{1,2}, Azadeh Najarzadeh^{*2,3}, Hassan Mozaffari-Khosravi^{2,3,4}, Hossein Fallahzadeh⁵, Elham Tavakoli⁶

1. International Campus, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
2. Nutrition and Food Security Research Centre, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
3. Department of Nutrition, School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
4. Diabetes Research Center, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
5. Department of Epidemiology and Biostatistics, School of Public Health, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.
6. Department of English Language, Maybod Branch, Islamic Azad University, Maybod, Iran.

***Correspondence:**

Azadeh Najarzadeh, Nutrition and Food Security Research Centre, Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

Email: azadehnajarzadeh@gmail.com

Tel: (98) 912 202 2817

Received: 12 December 2016

Accepted: 01 September 2016

Published in January 2017

Clinical Trial Registration Number:
IRCT2014121610826N17

Abstract

Objective: Although smoking increase the risk of cardiovascular diseases, its quitting leads to weight gain due to increased appetite. The aim of this study was to determine the effect of face to face dietary counseling on metabolic syndrome (MetS) indices in cigarette quitter.

Materials and Methods: This 9-week parallel randomized clinical trial was conducted by participation of 50 patients with metabolic syndrome who have recently quit smoking. Participants were assigned randomly into two groups. Intervention group (IG) received weekly nutritional education and consultation, while control group (CG) did not receive any intervention. Before and after the trial, both groups were evaluated nutritionally using 24-hour dietary recall questionnaire, and metabolic syndrome parameters were measured. Paired T-test and ANCOVA were used for statistical analysis.

Results: After intervention, weight, body mass index (BMI), triglycerides and systolic blood pressure were significantly different between CG and IG, while indices of low-density lipoprotein (LDL-C), high-density lipoprotein (HDL-C) and diastolic blood pressure were not significantly different between two groups. Amount of energy, lipid, protein, and carbohydrate intake were significantly lower in IG compare to CG. After adjustment of age, duration of smoking and the initial concentration of biochemical and anthropometric factors, significant differences were found between IG and CG in the changes of LDL-C (*P*-value 0.001), BMI (*P*-value 0.001), weight (*P*-value 0.001), waist circumference (*P*-value 0.002), and triglyceride (*P*-value 0.001). While the difference in changes of HDL-C, FBS, systolic and diastolic blood pressure, were not significant between two groups.

Conclusion: Given the findings of this study, nutritional counseling could improve some indices of MetS in cigarette quitters.

Keywords: Metabolic syndrome, Ex-smoker, Nutritional counseling

Introduction

Metabolic syndrome (MetS) is a set of factors associated with abdominal obesity, hypertension, hypertriglyceridemia, reduced high-density

lipoprotein (HDL-C), and insulin resistance (1,2). Based on different criteria, the incidence of MetS in world is increasing, especially in older populations. Many factors, especially

obesity and age are involved in its increased incidence (3,4). According to some studies, approximately one-fifth of the population in all areas of the world are suffering from MetS (5,6).

MetS will impose a great burden on country in the future, since unusual weight gain, high blood lipid and sugar are seen in people with MetS (7,8). In the pathogenesis of this syndrome, various factors and process were considered. The most important of them is insulin resistance and role of inflammatory mediators (9,10).

Smoking is closely related to many diseases such as obesity, coronary heart disease, peripheral vascular and lung cancer. Some studies showed, about 15% of the 15-64 year-old population are smokers (11-13). Epidemiological evidences show that smoking is associated with the emergence of the MetS (14,15). Due to decreased sensitivity of insulin receptors, nicotine leads to insulin resistance and high blood sugar (BS) in smokers than non-smokers (16,17). Active smokers have 26% increased risk of MetS compared with non-smokers (18). Smoking causes abdominal obesity and increased level of triglyceride (TG) and low HDL cholesterol levels and as a result, it increases the risk of MetS (19).

Although quit of smoking can improve the health, due to improved appetite and high level of food intake, the risk of obesity and its complications may be greater than before. Approximately, 90% of smokers gain weight after quitting. Also, greater tobacco dependence make higher weight gain (20,21). Therefore, people should have a weight management program after quitting smoking to reduce the risk of obesity and MetS. Given the importance of quitting smoking and its possible role in the increased risk of MetS, the current study used an experimental model to study the effects of dietary counseling in people who had quit smoking and MetS. The aim of this study was to evaluate the effectiveness of nutrition education and counseling on MetS parameters in cigarette quitters.

Materials and Methods

Type of study and participants

This study was conducted as a parallel randomized clinical trial in nutrition and diet therapy clinic of Ghaem Hospital of Mashhad since June to November of 2015. By considering alpha 5% and study power 80% and according to standard deviation 4 for measurement of weight change and to achieve a significant difference of at least 3% in the mean of weight change between the EG and CG, 25 patients in each group were enrolled. Considering a 20% loss to follow up, 30 people per group were considered for this study. People who had smoking history for at least six months, 4 cigarettes .day and have passed at least one day and one month at maximum of their quitting were considered for this trial. Lack of specific diseases such as hypertension, hyperlipidemia or diabetes or use of drugs lead to changes in metabolic syndrome parameters were considered as additional inclusion criteria. Based on the ATP III criteria, 3 or more of the following are required to be present for the diagnosis of MetS: abdominal obesity (Waist circumference (WC) >102 cm in men and WC >88 cm in women), raised TG (≥ 150 mg/dL), low HDL-C levels (<40 mg/dL in men and <50 mg/dL in women), raised BP (systolic BP ≥ 130 mm Hg, diastolic BP ≥ 85 mm Hg), and raised Fasting Plasma Glucose (≥ 110 mg/dL). Re-smoking, no observing prescribed diet, using alcohol or drugs to help quitting, such as methadone, tramadol, or buprenorphine considered as exclusion criteria.

Sixty people included in study were divided into two groups in a 1:1 ratio using randomized numbers table. The aim of the study was explained to all participants and written informed consent was obtained from them. On the other hand, the proposal of this study was approved by Ethics Commission in Research and Technology of Shahid Sadoughi University of Medical Sciences, Yazd, and it was registered with code of IRCT2014121610826N17 on www.Irct.ir.

Measurements

Questionnaire collected and recorded general information, including age, number and duration of smoking, drugs and alcohol, and history of underlying disease using interview with subjects.

WC was measured between the last rib and three iliac blades by tape measure with accuracy of 0.5 cm. Height was measured by height gauge of Seca without shoes with accuracy of 0.5 cm. Weight was measured by Seca scale with light clothing and no shoes with an accuracy of 100 g. BMI was obtained by dividing weight in square of height. Blood pressure was measured using a mercury Erkameter 3000 sphygmomanometer.

Biochemical assessment

In this study, biochemical parameters of metabolic syndrome such as high blood cholesterol (HDL and LDL), TG and fasting blood sugar (FBS) were measured in the beginning and end of the study in participants. Two milliliter of fasting blood was taken of peripheral vein for biochemical tests. Serum concentration of TG, FBS, HDL, LDL (The LDL was calculated by the Fried-Wald formula) were measured in terms of mg/dL based on enzymatic and colorimetric method (Enzymatic-Colorimetric. CHOD-PAP) using the kit and autoanalyzer device (Machine Biosystem co, Spain).

Dietary intake assessment

It was conducted using 24-hour dietary recall questionnaire for three days (two week days and one weekend) at the beginning and end of study.

Statistical analysis

Results are shown as mean \pm standard

deviation (SD). To analyze data, SPSS 21 software was used. To determine the distribution of quantitative data, Kolmogorov-smirnov test was used. Significance level was considered less than 0.05 (P -value <0.05). To compare quantitative variables between before and after the intervention in each group, Paired-Samples T-test was used. For between groups comparison analysis, analysis of covariance (ANCOVA) was used

Results

In this study, 60 people who quit smoking from one day to one month were included, 2 subjects of control group and 8 subjects of intervention group were excluded from study due to re-smoking, loss to follow up, and lack of cooperation and absence in nutrition counseling sessions during the study (Figure 1).

Table 1 shows the mean and SD of anthropometric and demographic variables of participants at the beginning of study. As this table shows, participants in this study were matched in terms of age, weight, BMI, duration of smoking at the beginning of study. Table 2 shows the mean and standard deviation of anthropometric, biochemical variables and blood pressure at the beginning and end of the study separately for two groups. As can be seen, weight, BMI, systolic blood pressure and TG were different between the two groups after the intervention.

However, comparing changes in indices in two groups showed no significant difference between intervention and control groups in terms of weight, BMI, waist circumference, triglycerides and LDL.

As shown in Table 3, after quitting smoking, macronutrients and therefore energy increased

Table 1. Anthropometric and demographic variables of participants at the baseline

Variables	control group (n=28)	intervention group (n=22)	P-value*
	Mean \pm SD	Mean \pm SD	
Age (year)	29.54 \pm 5.7	30.05 \pm 8.0	0.795
Body mass index (kg/m ²)	33.06 \pm 1.4	33.64 \pm 1.0	0.108
Weight(kg)	95.35 \pm 8.4	96.84 \pm 6.8	0.505
Duration of smoking (year)	8.54 \pm 5.5	7.96 \pm 4.8	0.701

Student T-test*

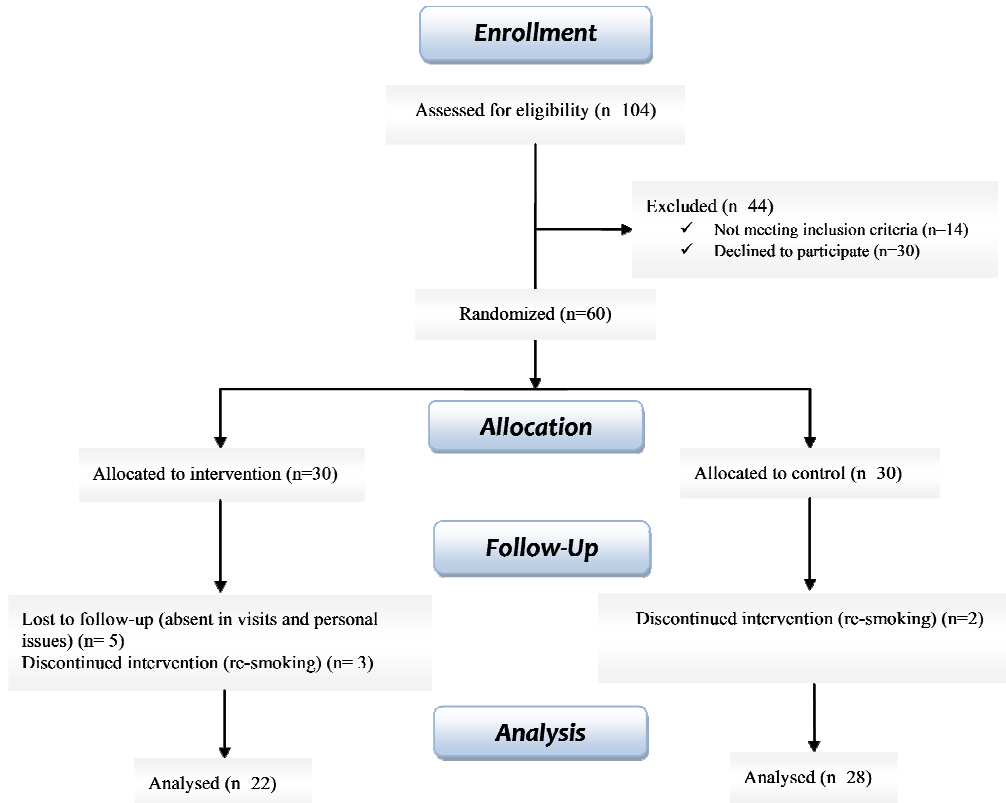


Figure 1. Flowchart of CONSORT

in the control group, while energy intake of intervention group who were under dietary counseling program decreased significantly compared to the beginning of the study.

Discussion

The results of this study showed that weight of people in the control group increased significantly after study compared to beginning of study. However, nutrition counseling in the intervention group resulted in significant reduction in the weight of smoking quitters. Weight gain observed in the control group reflects the impact of smoking quitting in increased appetite and food intake. Comparing changes between two groups showed significant difference. Providing diet counseling and applying the principles of proper nutrition can help people to quit smoking and minimize the weight gain. Additionally, some findings showed that the improvement of diet compounds can improve

the health of these people, even in those who were not able to quit smoking (18).

Kmetova's study conducted in 2014 showed that women gain higher weight compared to men after quitting smoking (20). In addition, this weight gain after quitting smoking was higher among those who have spent many more years on smoking (20). It was proven that these people have greater need to use a weight management program as part of their smoking quitting program (20). The prevalence of metabolic syndrome in smokers is very high (15,36). Smoking is a very important factor in the development of this disease, and the risk increases as the number of cigarettes smoked per day increases (22). A study conducted by Bush showed that weight gain is a significant factor after quitting (37). Concern about weight gain especially in those who have previously attempted to quit smoking and have returned again to re-use it, is more and it has a negative impact on their

self-efficacy and successful quitting (23,37). ANCOVA analysis showed significant differences between two groups in terms of BMI change. In 2011, in a study conducted by Kamaura on 946 patients, it was showed that increased body mass index had lower impacts on these metabolic disorders than smoking (39). BMI value was increased by 36.2% in people quit smoking during the first two years and 46% in the third year, and 15% in the fourth year. They demonstrated that the risk of metabolic disorders due to increased body mass index could be decreased by nutrition education and counseling.

The data of the present study revealed that nutrition counseling can prevent the increase in abdominal obesity. In one of the studies conducted by Wang-Youn Won in 2014 in

order to find the causes of increased energy intake and weight gain after quitting smoking, it was found that smoking quitting significantly increased the level of plasma adiponectin in such a way that it increased within four weekly significantly ($P<0.016$) (22). This increased plasma adiponectin in the complex process leads to increased appetite and fat distribution in the body, especially in the abdominal area (22). This increase in appetite was evident in this study, although we did not measure it.

After intervention in terms of LDL concentrations, no significant difference was shown between two groups, but the analysis of covariance showed that changes in this variable between the two groups are different. In other words, reduction of LDL in

Table 2. Comparison of mean of quantitative variables in two groups before and after intervention

Variables	Before	After	Changes	P-value*
Weight(kg)				
Control (n=28)	95.35±8.4	97.48±8.7	2.14±2.8	
Intervention (n=22)	96.84±6.8	94.64±6.7	-2.02±3.9	<0.001
P-value**	0.505	<0.001	<0.001	0.016
Body mass index (kg.m²)				
Control (n=28)	33.60±1.4	33.83±1.9	0.77±0.94	
Intervention (n=22)	33.64±1.0	33.00±1.3	-0.64±1.46	<0.001
P-value**	0.108	<0.001	<0.001	0.053
Waist circumference (cm)				
Control (n=28)	104.25±6.5	105.07±6.9	0.82±2.3	
Intervention (n=22)	107.41±6.6	105.68±6.4	-1.73±3.0	0.069
P-value**	0.096	0.75	0.002	0.014
Systolic blood pressure(CmHg)				
Control (n=28)	13.45±0.4	12.98±0.5	-0.47±0.6	
Intervention (n=22)	13.34±0.6	12.64±0.4	-0.70±0.4	0.001
P-value**	0.458	0.018	0.133	<0.001
Diastolic blood pressure (CmHg)				
Control (n=28)	9.02±0.8	9.17±1.0	-.047±0.6	
Intervention (n=22)	8.83±0.7	8.70±0.4	-0.7±0.4	0.553
P-value**	0.219	0.005	0.220	0.462
TG (mg/dl)				
Control (n=28)	170.36±17.9	170.25±31.2	-0.11±27.2	
Intervention (n=22)	174.86±17.8	137.59±21.7	-37.27±20.9	0.984
P-value**	0.379	<0.001	<0.001	<0.001
FBS (mg/dl)				
Control (n=28)	106.25±20.2	94.18±8.7	-047±0.6	
Intervention (n=22)	109.91±6.6	94.55±7.0	-0.70±0.4	0.002
P-value**	0.419	0.825	0.367	<0.001
HDL(mg/dl)				
Control (n=28)	35.36±3.8	41.29±4.4	5.93±3.7	
Intervention (n=22)	35.14±3.1	42.59±5.2	7.45±5.4	0.001
P-value**	0.827	0.180	0.162	<0.001
LDL(mg/dl)				
Control (n=28)	94.18±17.8	90.82±19.1	-3.36±21.3	
Intervention (n=22)	113.55±15.0	90.14±14.2	-23.41±16.5	0.411
P-value**	<0.001	0.179	0.001	<0.001

Student T-test*, ANCOVA test ** using age, duration of smoking, and basal values of each variable as covariates

experimental group was higher than control group. Regarding this index, few studies have been done in people quit smoking, making it difficult to compare findings.

In the present study, HDL cholesterol significantly increased in both groups and there was no significant difference between two groups. That means that quitting smoking with or without nutrition counseling increases the concentration of this index. Triglyceride (TG) concentration in our participants reduced significantly in intervention group compared to beginning of study. The changes rate of concentration reduction of this index in the intervention group who were under nutritional counseling was higher than the control group based on ANCOVA by adjusting for age, duration of smoking, and basal concentration on TG. Compared with a study conducted by Calo et al, it was showed that concentrations of HDL increased significantly in people who quit smoking ($P<0.05$) and triglycerides decreased significantly (41).

In this study, the mean of fasting blood glucose and systolic blood pressure significantly decreased in both groups compared to beginning of study. However, the reduction of these variables were not different between two groups.

Diastolic blood pressure significantly decreased in both groups, although comparison of changes in this index showed

no difference between the two groups. Therefore, it can be said that quitting smoking alone can reduce the diastolic blood pressure. However, if much time passes of quitting and the person gains weight due to increased appetite, high blood pressure was not unexpected. In a study conducted in 2013 by Calo, it was found that those who have quit smoking, these two indices of blood pressure decreased (41).

Energy intake in both groups was significantly different compared to beginning of study, and the mean of change was different between two groups. The results showed smokers receive increased energy after quitting so and weight BMI increase in these people. This study showed that increased energy intake in smokers with metabolic syndrome leads to risk after quitting, since nutrition counseling during the period of quitting smoking is preventive factor that improve the metabolic syndrome. So the role of the dietitian in the control of food intake was evident. Lerman et al study in 2010 showed that most people may eat proper food, but most of them, especially the smokers, have weak food selection (43), this issue was clear in comparing it with present study. In this investigation, more than 90% of smokers who quit smoking had bad nutritional habits, including excessive use of carbohydrates such as bread and cereals, fats especially saturated ones containing much

Table 3. Mean of energy and macronutrient intakes in control and intervention groups before and after study

Variables	Baseline	After	P-value*
Carbohydrate (g/d)			
Control (n=28)	264.88±33.2	307.33±38.7	<0.001
Intervention (n=22)	254.95±36.7	255.48±19.8	
P-value**	0.322	<0.001	
Protein (g/d)			
Control (n=28)	97.07±16.7	112.62±16.4	0.001
Intervention (n=22)	98.44±16.2	89.76±7.3	
P-value**	0.772	<0.001	
Lipid(g/d)			
Control (n=28)	65.76±8.1	75.02±8.6	<0.001
Intervention (n=22)	76.17±9.8	58.18±3.5	
P-value**	<0.001	<0.001	
Energy (kcal/d)			
Control (n=28)	2039.67±242.1	2355.02±268.3	<0.001
Intervention (n=22)	2099.08±274.3	1904.61±132.6	
P-value**	0.421	<0.001	

*Paired sample T-test ** Student T-test

cholesterol, and generally abundant consumption of foods with low nutritional value such as fast foods and snacks such as crisps. The base of education and diets given in this study in the intervention group involved low calorie, since BMI of all of these people was in the obesity range. Conducted educations were successful and high percentage of people carried out almost 60% of these educations in this study. Carbohydrates, protein and fat intake reduced and highly use of vegetables and fruits, and dairy were included in dietary program of them. Khan Sun study in 2012 showed that quitting smoking is effective treatment in patients with metabolic disorders and cardiovascular diseases reducing the risk of metabolic syndrome (16). They also concluded that quitting smoking reduced the prevalence of metabolic syndrome (16). This study showed that nutrition counseling along with quitting smoking have more impact in patients with metabolic syndrome.

In a study conducted by Leeman, it was revealed that people trying to quit smoking and controlling food intake (intake restriction) lead to failure quitting smoking programs (43). In the present study, according to educations and training provided in diet and emphasis on observing them by researchers, dietary intake was restricted for participants. In fact, to do good research on people who have tried to quit smoking and reduction in the number of people who turn to smoking again, we need an

intensive consultation program. In this study, to reduce the possibility of people returning to smoke and their exclusion of this study it was tried that observing the nutritional recommendations to be increased by enhancing the motivation of the participants. In this regard, we avoided using negative and forbidding words, instead we emphasized to use high nutritional value foods rather than low-value and high-energy foods.

This study has some limitations such as short duration, male participants and use of dietary recall that may be an issue for under and over estimation of dietary intakes.

But, to our knowledge, it was the first clinical trial investigating the effect of dietary counseling on decreasing parameters of metabolic syndrome among smoking quitters.

Conclusion

We conclude that face to face nutrition education and dietary counseling may have beneficial effects on weight, BMI, waist circumference, triglyceride and LDL-cholesterol in smoking quitters, and it has no additive effect on fasting blood sugar, HDL, systolic and diastolic blood pressure.

Acknowledgements

This paper was extracted from a thesis approved by Shahid Sadoughi University of Medical Sciences, Yazd, Iran

References

1. Grundy SM, Brewer HB, Cleeman JI. Definition of Metabolic Syndrome report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition, Circulation, 2004;109(3):433-8.
2. Eckel RH, Grundy SM, Zimmet P. The metabolic syndrome. *The Lancet*, 2005;365(9468):1415-28.
3. DeFronzo RA, Ferrannini E. Insulin resistance: A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care*. 1991;14(3):173-94.
4. Biolo G, Toigo G, Guarnieri G. Slower activation of insulin action in upper body obesity. *Metabolism*. 2001;50(1):19-23.
5. Heidari Z, Hosseiniapanah F, Mehrabi Y, Azizi F. Evaluation of power of components of metabolic syndrome for prediction of its development: a 6.5 year longitudinal study in Tehran Lipid and Glucose Study (TLGS). *Iranian Journal of Endocrinology and Metabolism*. 2009;11(5):530-42.
6. Tremblay M, Gaudet D, Brisson D. Metabolic syndrome and oral markers of cardiometabolic risk. *J Can Dent Assoc*. 2011;77:125.

7. Pietropaoli D, Monaco A, Del Pinto R, Cifone M, Marzo G, Giannoni M. Advanced glycation end products: possible link between metabolic syndrome and periodontal diseases. *International journal of immunopathology and pharmacology*. 2012;25(1):9-17.
8. DeFronzo RA. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidaemia and atherosclerosis. *Neth J Med*. 1997;50(5):191-7.
9. Grundy SM, Brewer HB, Cleeman JI, Smith SC, Lenfant C. Definition of metabolic syndrome report of the National Heart, Lung, and Blood Institute. American Heart Association Conference on scientific issues related to definition. *Circulation*. 2004;109(3):433-8.
10. American Heart A, National Heart L, Blood I, Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome. An American Heart Association. National Heart, Lung, and Blood Institute Scientific Statement. Executive summary. *Cardiol Rev*. 2005;13(6):322-7.
11. Shahbazi H, Baghianimoghadam MH, Zinalabediny M, Amoie A, Zolghadr R. View of the people on the impact of visual media on the prevention and incidence of smoking among adolescents and young adults. *Iranian Journal of Health Education and Health Promotion*. 2013;2013;1(3):33-46.
12. World Health Organization: process for a global strategy on diet, physical activity and health. 2003.
13. Mehrabi S, Delavari A, Moradi G, Esmailnasab EN, Pooladi A, Alikhani S, et al. Smoking among 15-to 64-year-old Iranian people in 2005. *iranian Journal of epidemiology*. 2007;3(1):1-9.
14. Donny EC, Caggiula AR, Weaver MT, Levin ME, Sved AF. The reinforcement-enhancing effects of nicotine: implications for the relationship between smoking, eating and weight. *Physiol Behav*. 2011;104(1):143-8.
15. Wada T, Urashima M, Fukumoto T. Risk of metabolic syndrome persists twenty years after the cessation of smoking. *Intern Med*. 2007;46(14):1079-82.
16. Sun K, Liu J, Ning G. Active smoking and risk of metabolic syndrome: a meta-analysis of prospective studies. *PLoS One*. 2012;7(10):47791.
17. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold ACM, et al. Combined effects of smoking and alcohol on metabolic syndrome: the LifeLines cohort study. *PLoS one*. 2014;9(4): 96406.
18. Leslie WS, Koshy PR, Mackenzie M, Murray HM, Boyle S, Lean ME, et al. Changes in body weight and food choice in those attempting smoking cessation: a cluster randomised controlled trial. *BMC public health*. 2012;12(1):1.
19. Levine MD, Bush T, Magnusson B, Cheng Y, Chen X. Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine & tobacco research*. 2013;15(6):1136-40.
20. Kmetova A, Kralikova E, Stepankova L, Zvolaska K, Blaha M, Sticha M, et al. Factors associated with weight changes in successful quitters participating in a smoking cessation program. *Addict Behav* 2014;239-45.
21. Aveyard P, Lycett D, Farley A. Managing smoking cessation-related weight gain. *Polskie Archiwum Medycyny Wewnętrznej*. 2012;122(10):494.
22. Won WY, Lee CU, Chae JH, Kim JJ, Lee C, Kim DJ. Changes of plasma adiponectin levels after smoking cessation. *Psychiatry Investig*. 2014;11(2):173-8.
23. Cena H, Tesone A, Niniano R, Cerveri I, Roggi C, Turconi G. Prevalence rate of metabolic syndrome in a group of light and heavy smokers. *Diabetology & metabolic syndrome*. 2013;5(1):1.
24. Haug S, Meyer C, Dymalski A, Lippke S, John U. Efficacy of a text messaging (SMS) based smoking cessation intervention for adolescents and young adults: study protocol of a cluster randomised controlled trial. *BMC Public Health*. 2012;12(1):1.
25. Leslie WS, Koshy PR, Mackenzie M, Murray HM, Boyle S, Lean ME, et al. Changes in body weight and food choice in those attempting smoking cessation: a cluster randomised controlled trial. *BMC public health*. 2012;12(1):389.
26. Slagter SN, van Vliet-Ostaptchouk JV, Vonk JM, Boezen HM, Dullaart RP, Kobold AC, et al. Combined effects of smoking and alcohol on metabolic syndrome: the LifeLines cohort study. *PLoS One*. 2014;9(4):96406.
27. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association. National Heart, Lung, and Blood Institute Scientific Statement. *Circulation*. 2005;112(17):2735-52.
28. American Heart Association Nutrition C, Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation*. 2006;114(1):82-96.
29. Bush T, Levine MD, Deprey M, Cerutti B, Zbikowski SM, McAfee T, et al. Prevalence of Weight Concerns and Obesity Among Smokers Calling a Quitline. *J Smok Cessat*. 2008;4(5):74-8.
30. Berrington de Gonzalez A, Hartge P, Cerhan JR, Flint AJ, Hannan L, MacInnis RJ, et al. Body-mass index and mortality among 1.46 million white adults. *New England Journal of Medicine*. 2010;363(23):2211-9.

31. Rang HP, Dale MM. Rang and Dale's Pharmacology: Churchill Livingstone 2007.
32. Angelini C. Disorders of lipid metabolism. *Handb Clin Neurol*. 2007;86:183-91.
33. Adibhatla RM, Hatcher JF. Altered lipid metabolism in brain injury and disorders. *Subcell Biochem*. 2008;49:241-68.
34. Bakhtari Z, Sayehmiri K, Abdi J. Prevalence of The Hypertriglyceridemia in Iran: A Meta-Analysis and Systematic Review Study. *sjimu*. 2013;21(2):103-111
35. Azizi. The agreed definition of the Metabolic Syndrome ATP by insulin resistance in study Tehran Lipid and Glucose IDF and Modified III. 2005.
36. Abdolahi H, Iraj B, Mirpourian M, Shariatifar B. Association of neck circumference as an indicator of upper body obesity with cardio-metabolic risk factors among first degree relatives of diabetes patients. *Adv Biomed Res*. 2014;3:237.
37. Bush T, Levine MD, Deprey M, Cerutti B, Zbikowski SM, McAfee T, et al. Prevalence of weight concerns and obesity among smokers calling a quitline. *Journal of smoking cessation*. 2009;4(02):74-8.
38. Levine MD, Bush T, Magnusson B, Cheng Y, Chen X. Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine Tob Res*. 2013;15(6):1136-40.
39. Kamaura M1 FH MS, Tochikubo O. Weight gain and risk of impaired fasting glucose after smoking cessation. *Epidemiol* 2011;21(6):431-9.
40. Stein JH, Asthana A, Smith SS, Piper ME, Loh WY, Fiore MC, et al. Smoking cessation and the risk of diabetes mellitus and impaired fasting glucose: three-year outcomes after a quit attempt. *PLoS One*. 2014;9(6):98278.
41. Calo WA, Ortiz AP, Suárez E, Guzmán M, Pérez CM, Pérez CM. Association of cigarette smoking and metabolic syndrome in a Puerto Rican adult population. *Journal of Immigrant and Minority Health*. 2013;15(4):810-6.
42. Nakanishi N, Takatorige T, Suzuki K. Cigarette smoking and the risk of the metabolic syndrome in middle-aged Japanese male office workers. *Industrial health*. 2005;43(2):295-301.
43. Leeman RF, O'Malley SS, White MA, McKee SA. Nicotine and food deprivation decrease the ability to resist smoking. *Psychopharmacology (Berl)*. 2010;212(1):25-32.