The Relationship between Snacking and Risk of Individual Components of Metabolic Syndrome in Normal-weight Adults: A Cross-sectional Study

Ahmad Zare Javid; PhD[1], Nasim Niknejad; MSc[2*], Hajieh Shahbazian; MD[2],
Seyed Mahmoud Latifi; PhD[2], Bahar Niknejad; MD[3] & Razie Hormoznejad; MSc[4]

[1] Nutrition and Metabolic Diseases Research Center & Hyperlipidemia Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.
[2] Health Research Institute, Diabetes Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.
[3] Weill Cornell Medical College, New York, New York, USA.
[4] Student Research Committee, School of Paramedicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

ARTICLE INFO

ORIGINAL ARTICLE

ABSTRACT

Background: Developing obesity-related metabolic disturbances in spite of having normal weight is increasing in normal-weight people worldwide. This study aimed to evaluate the relationship between different types of snacking and risk of individual components of metabolic syndrome (MetS) in normal-weight adults. **Methods:** This cross-sectional study was carried out on a randomized sample of 328 normal-weight individuals ($18.5 \leq BMI \leq 24.9$ kg/m^2) older than 20 years in Ahvaz, Iran. Anthropometric indices, systolic and diastolic blood pressure, lipid profile and fasting blood glucose (FBG) were measured. MetS components were chosen based on the definition of international diabetes federation (IDF). Dietary intake was evaluated by a validated 50-item non-quantitative FFQ. Snacks were defined as energy-dense, nutrient-poor foods as well as low energy-dense and high-fiber foods. **Results:** Males had significantly higher rates of elevated FBG and triglyceride (TG), whereas higher rates of abdominal obesity and low HDL-c were observed in females. Older participants with lower education showed higher percentages in most of the MetS' components. The occurrence of abdominal obesity and hypertension increased in the third compared to the first tertile category of supermarket cakes and biscuits (OR = 1.23; 95% CI: 1.02 – 1.49) and chocolate (OR = 1.10; 95% CI: 1.03-1.18), respectively. However, other snacks showed no significant relationship. **Conclusions:** The consumption of unhealthy snacks with high fat, sugar, and refined carbohydrates in forms of supermarket cakes and biscuits as well as low-flavanol content chocolate products are the major dietary snacking habits contributing to abdominal obesity and hypertension in normal-weight adults in southwest of Iran.

Keywords: *Body mass index; Diet; Metabolic syndrome; Snacks*

Introduction

Metabolic syndrome (MetS) is associated with cardiovascular risk factors and type 2 diabetes mellitus which include abdominal obesity, elevated blood pressure (BP), fasting blood glucose (FBG), triglyceride (TG), and low level of high-density lipoprotein cholesterol (HDL-c).(Shahbazian *et al.*, 2013) Approximately 50% of patients with type 2 diabetes and severe coronary syndrome as well as 95% of patients with peripheral arterial disease suffer from MetS (Qadan *et al.*, 2008, Zaliūnas *et al.*, 2007).

Obesity is an important public health challenge worldwide and more than one billion people are predicted to be obese in 2030 (Esteghamati *et al.*, 2009, Kelly *et al.*, 2008). Obesity is associated with higher risk of several diseases such as non-alcoholic fatty liver disease, type 2 diabetes mellitus, MetS, and higher all-cause mortality (Kelishadi *et al.*, 2008, Kwon *et al.*, 2013, Phillips and Perry, 2015). Although the standard definition for obesity is to have high levels of body fat, epidemiologists believe that the definition of obesity is having a body mass index (BMI) $\geq$ 30 (Romero-Corral *et al.*, 2010).

Obesity is classified into three subtypes based on its metabolic risk which include: 1. Obese individuals with MetS, 2. Obese individuals without MetS, and 3. Normal-weight individuals with MetS (De Lorenzo *et al.*, 2006). The condition of having obesity-related metabolic disturbances in spite of normal weight (BMI < 30 kg/m^2) was first suggested by Ruderman in 1981 (Ruderman *et al.*, 1981). Metabolically obese, normal weight (MONW) is a subgroup of obesity that cannot be revealed by routine measurements such as body weight, BMI, skin fold thickness, and body fat mass (Ruderman *et al.*, 1981).

The prevalence of MetS in developed countries is reported as 25% (National Cholesterol Education Program Expert Panel on Detection and Treatment of High Blood Cholesterol in, 2002), while this rate has been estimated at 22.5-32.1% in different regions of Iran (Sarrafzadegan *et al.*, 2011, Shahbazian *et al.*, 2013, Sharifi *et al.*, 2009, Zabetian *et al.*, 2007). According to NHANES III, the prevalence rates of MONW condition among normal BMI men and women were 4.6% and 6.2%, respectively (Park *et al.*, 2003). The high occurrence rates of MetS in normal-weight Iranian men and women in Tehran were respectively 9.9 and 11.0% that is a great cause of concern (Hadaegh *et al.*, 2007b).

Evidences indicated that several risky characteristics reduce insulin sensitivity and increase visceral fat accumulation (Bednarek-Tupikowska *et al.*, 2012, Katsuki *et al.*, 2003, Ruderman *et al.*, 1998). These risky characteristics include: high carbohydrate diet and high sucrose consumption (Ruderman *et al.*, 1981), smoking and elevated hs-CRP (Kwon *et al.*, 2013), reduced basal metabolic rate (De Lorenzo *et al.*, 2006), low lean body mass (De Lorenzo *et al.*, 2006, Marques-Vidal *et al.*, 2010), low physical activity (Ruderman *et al.*, 1998, Ruderman *et al.*, 1981), and reduced VO$_2$max and aerobic fitness (Ruderman *et al.*, 1998). The specific ethnicities, gender, and age groups (Bednarek-Tupikowska *et al.*, 2012, Kelishadi *et al.*, 2008) also had a higher percentage in MONW individuals and ultimately lead to increased risk of CVD. In contrast, metabolically healthy obese (MHO) phenotype is a subgroup of obesity with normal metabolic profile despite having BMI $\geq$ 30 kg/m^2 (Primeau *et al.*, 2011). Evidences further showed that arterial stiffness, carotid atherosclerosis, and risk of fatal CVD events are higher in MONW than MHO participants within 10 years (Yoo *et al.*, 2014).

Diet, physical inactivity, and genetic susceptibility can be the causes of unhealthy metabolic profile among normal-weight adults (Hadaegh *et al.*, 2007b, Roche *et al.*, 2005). Sedentary lifestyle and transition of nutrition from healthy food choices to westernized diet, such as higher consumption of industrial and processed foods, sweetened artificial beverages, and junk foods are the most significant factors contributing to the alarming trend of MetS (Naeem, 2012, Popkin *et al.*, 2012).

Having specific dietary intake and eating behavior patterns are significant factors in

initiation and development of insulin resistance and MONW (Choi *et al.*, 2012). A few studies have examined the relationship between dietary patterns and MONW (Choi *et al.*, 2012, O'Connor *et al.*, 2015, Suliga *et al.*, 2015). Suliga et al. reported that dietary patterns involving high consumption of fish and whole grains as well as reduced intake of sugar, sweets, refined grains, and cold cured meat are associated with lower risk of MONW (Suliga *et al.*, 2015).

In recent years, increased frequency of both healthy and unhealthy snacking, portion sizes, trans and saturated fats, total fats, and added sugar content of snacks consumed by the public have been a major warning towards the ascending trend of obesity and metabolic disorders (Mirmiran *et al.*, 2014, O'Connor *et al.*, 2015). Snacking habit among USA adults increased from 71% to 97% and the contribution of snacking to the total energy intake increased from 18% to 24% from 1997 to 2006 (Piernas and Popkin, 2010). Diets with high-energy and low-nutrient density are also the major factor in rising trend of non-communicable diseases among Iranian population (Ghassemi *et al.*, 2002). A recent study in Iranian population showed that high intake of energy-dense snacks, especially salty types is significantly associated with high rate of MetS after a 3-year follow-up (Mirmiran *et al.*, 2014). Though there are various definitions for a 'snack', according to O'Conner et al., snacks can be defined as "energy-dense and nutrient-poor foods, commonly referred to as snack foods, and also low energy-density and high-fiber foods such as fruits" (O'Connor *et al.*, 2015).

To the best of our knowledge, limited facts have been found concerning the relationship between snacking and MONW condition or each MetS component in normal-weight Iranian population. Therefore, the present study targeted at investigating the relationship between snacking and each MetS component in normal-weight adults in southwest of Iran.

Materials and Methods

Study design and population: This study was performed with random cluster sampling in Ahvaz, southwest of Iran in 2015. Six health centers were randomly chosen from 25 health centers, 55 households were then randomly selected from each health center and invited to take part in the examinations. A total of 780 individuals participated in the study. Male or female participants older than 20 years with normal BMI ($18.5 \leq BMI \leq 24.9$ kg/m^2) were included for further investigation. Individuals with incomplete questionnaires or missing anthropometric, BP, or biochemical measurements were excluded. Finally, data collected from 328 normal-weight individuals were suitable for statistical analysis and written consent was obtained from all participants.

Other variables such as age, gender, ethnicity (Persian, Arabian, others), marital status, education, regular physical activity (defined as doing at least 30 minutes of brisk walking or high level activity three times or more per week) (Fan *et al.*, 2016), and smoking status (never, former, and current) were obtained by a trained interviewer.

Measurements: Anthropometric indices including weight, height, and waist circumference (WC) were measured by trained technicians. Weight was measured by an analog scale (Seca, Hamburg, Germany) to the nearest 0.1 kg with light clothing. Height was then measured with barefoot by a wall-mounted stadiometer (Seca model 220, Hamburg, Germany) to the nearest 0.1 cm while head was in Frankfort horizontal plane and 4 sites touching the wall (head, shoulder blades, buttocks, and heels). BMI was calculated by dividing weight (kg) by the square of height (m^2). WC was measured just above the right iliac crest at the end of a normal expiration to the nearest 0.1 cm using a non-elastic tape measure. All anthropometric indices were measured on the basis of world health organization standards (WHO, 1987).

The BP of each participant was measured by a standard mercury column sphygmomanometer on the right arm at sitting position after a minimum of 5 minutes of rest. Systolic and diastolic BP (SBP and DBP respectively) were measured twice to the nearest 2 mmHg, and mean of BP was taken as

participant's arterial BP. The initial Korotkoff sound phase was accepted as SBP and disappearance of sound (the fifth phase) was recorded as DBP.

Blood samples were collected, centrifuged, stored in refrigerator, and then sent to Diabetes Research Center laboratory for biochemical analysis after 10 to 12 h of fasting. Serums were analyzed for TG, FBG, and HDL-c levels through enzymatic calorimetric method by an auto-analyzer with Pars Azmoon kits (Pars Azmon Inc., Tehran, Iran).

The information on dietary consumption was obtained using a validated 50-item non-quantitative food frequency questionnaire (FFQ). The FFQ used in this research was a short form of a 168-item semi-quantitative FFQ formerly used by Mirmiran et al. in Tehran Lipid and Glucose Study (Mirmiran *et al.*, 2010). Reliability of the 50-item FFQ is optimal, with Cronbach's alpha and Split-half reliability estimates of 0.825 and 0.732, respectively. Validity of FFQ for measuring intake of 50 food items was confirmed by 5 nutrition professionals. These results indicate that the 50-item FFQ is a valid and reliable tool for dietary assessment among people residing in southwest of Iran.

The FFQ considered dietary intakes as the frequency of consumption of a medium-size serving of each food item during the previous year on daily, weekly, monthly, or yearly bases. Afterwards, all frequencies were converted to daily intake frequency.

"Snack" was defined by O'Conner et al. as "energy-dense and nutrient-poor foods and also low energy-dense and high-fiber foods such as fruits" (O'Connor *et al.*, 2015). Investigating each individual type of energy-dense snack compared to total amount of snacks consumed will give more distinct and accurate results concerning its subsequent cardio-metabolic risks (Mirmiran *et al.*, 2014).

MetS components: According to international diabetes federation (IDF) consensus worldwide, the definition of MetS components included: 1. Central obesity determined by WC ≥ 94 cm in males and ≥ 80 cm in females (Alberti *et al.*, 2009), 2. Elevated TG level ≥ 150 mg/dL, 3. Reduced HDL-c level <4 0 mg/dL in males and < 50 mg/dL in females, 4. Elevated FBG level ≥ 100 mg/dL or drug treatment for elevated FBG, 5. Elevated BP, SBP ≥ 130 mmHg or DBP ≥ 85 mmHg or antihypertensive drug treatment.

As suggested by Hadaegh et al. WHO's WC cut-off points are not suitable for Iranian population (Hadaegh *et al.*, 2007a). Hence, for abdominal obesity, the Middle East specific WC that is a compulsory component of MetS definition proposed by IDF was applied (Alberti *et al.*, 2009).

Data Analysis: SPSS software version 22 was used to conduct the statistical analysis. Chi-square test and *t-test* were applied to compare the categorical and continuous baseline characteristics, respectively. Bonferroni post-hoc test was performed for further comparisons between different age groups and different education levels. The relationships between MetS components and the type of consumed snacks were evaluated using multiple binary logistic regression model adjusted for potential confounders including age, gender, ethnicity, marital status, education, smoking, physical activity, and BMI. In multiple regression model, different types of snacks were considered as continuous variables so they were entered in unique blocks. Afterwards, the intake frequency of snack types with significant association with MetS components was divided into tertiles based on their 33th and 66th percentile values. Then, the relationships between MetS components and each tertile category of snacks were evaluated using multiple binary logistic regression model adjusted for potential confounders. The level of statistical significance was set at P-value < 0.05 and 95% confidence interval (CI) was used.

Ethical consideration: The research was approved by the research ethics committee of Ahvaz Jundishapur University of Medical Sciences (ethical approval reference number 193043).

Results

A total population of 328 normal-weight adults including 157 (47.9%) males and 171 (52.1%)

females were investigated in this study. The prevalence of MetS for total, male, and female participants were 12.5%, 6.4%, and 18.1%, respectively. Mean ± SD of age was 39.2 ± 16.8 for all participants, 42.4 ± 18.0 in males, and 36.0 ± 15.0 in females ($P = 0.001$). Regarding the metabolic health, male participants had higher TG, SBP, DBP, WC, and lower HDL-c level when compared with female participants ($P < 0.05$). However, there was no significant difference in FBG and BMI between the two groups ($P > 0.05$). A greater percentage of smoking was observed in males than females ($P = 0.0001$), while no significant difference was observed between proportion of different education levels and regular physical activity between the two genders ($P > 0.05$). Furthermore, 89.6% of participants were educated, 50% of whom had university degree. Regarding the smoking status, 16.6% of males were current or past smokers while none of the female participants has ever been smokers. Regular physical activity was achieved only in 6.7% of participants (**Table 1**).

According to **Table 2**, 26.2% of participants were hyperglycemic. The proportion of hyperglycemia was significantly different based on gender, age groups, and educational levels ($P < 0.05$). The 20-34 years old participants with high FBG level were significantly lower in proportion than the other 3 age groups ($P = 0.0001$). The proportion of 35-49 years participants with high FBG level was also lower than ≥ 65 years participants ($P = 0.008$, chi-square test with Bonferroni correction). The proportion of hyperglycemic participants was significantly different among all 3 education levels ($P = 0.0001$). So, 20.1% of the study population had hypertriglyceridemia The proportion of participants with hypertriglyceridemia was significantly different between different genders and different age groups ($P < 0.05$); it was specifically lower in 20-34 years age group than the 50-64 and ≥ 65 years age groups ($P = 0.002$). Low HDL-c level was observed in 48.2% of individuals. The occurrence of low HDL-c was only different between males and females ($P =$

0.003). Hypertension was observed in 19.8% of participants. The proportion of hypertensive participants were significantly different between different age groups and different education levels ($P = 0.0001$). The proportion of hypertensive participants of 20-34 and 35-49 years age groups were significantly lower than that of both 50-64 and ≥ 65 years age groups ($P = 0.0001$). The proportion of hypertensive illiterate participants was significantly higher than that of 2 other education levels ($P = 0.0001$). Abdominal obesity was seen in 27.4% of participants. The proportion of abdominal obesity was significantly different between different genders, age groups, education levels, and smoking categories ($P < 0.05$). The proportion of abdominal obesity in 20-34 years age group was significantly lower than other 3 age groups ($P < 0.05$). The proportion of abdominal obesity was significantly different among all 3 education levels ($P < 0.05$). No other significant difference was observed for the proportion of MetS components among categories of demographic variables.

According to **Table 3**, there was a significant increasing trend for the occurrence of abdominal obesity and hypertension across increasing tertile of supermarket cakes and biscuits (OR = 1.23; 95% CI: 1.02–1.49) as well as chocolate (OR = 1.10; 95% CI: 1.03-1.18), respectively. Other snack types had no significant relationship with MetS components.

Discussion

In this study carried out on normal-weight adults, high intake of supermarket cakes and biscuits, as an important source of refined grains, is associated with higher risk of abdominal obesity. In addition, high intake of commercially available chocolate products is associated with high BP measurements.

It is noteworthy that although no significant relationship is observed between regular physical activity and MetS components, low percentage of physical activity (6.7%) in the studied population, compared to the global rates is a cause for concern.

Commercially available cakes and biscuits consumed by the general population are usually

a source of refined grains. A number of observational and interventional studies validated the dependencies between whole grains consumption and reduction in WC measurement when compared with refined grains (Jonnalagadda *et al.*, 2011, Koh-Banerjee *et al.*, 2004, Kristensen *et al.*, 2012, Landberg *et al.*, 2010, Shimizu *et al.*, 2008). In a meta-analysis performed by Harland et al. consumers with higher intake of whole grains had lower WC and waist to hip ratio compared with consumers with lower amounts of intake (Harland and Garton, 2008). On the other hand, Halkjaer et al. found no significant relationship between the consumption of whole grains and forthcoming WC change, though the consumption of refined grains revealed a positive relationship with WC change in women (Halkjær *et al.*, 2006). A 12-week whole grains dietary intervention conducted on 40-65 years old adults with MetS resulted in no change in WC measurements when compared to the refined grains group (Giacco *et al.*, 2013).

The results of present study revealed a positive relationship between consumption of cakes and biscuits and WC in normal-weight adults. O'Neil et al. reported that the relationship between the consumption of whole grains and decreased WC reduced to nil after adjustment for cereal fiber (O'Neil *et al.*, 2010). The probable cause for the increase in WC is low fiber and high content of refined grains in common cakes and biscuits obtainable in stores. Furthermore, supermarket cakes and biscuits may have high amounts of added sugar, fat, and sodium (Williams, 2012). Dietary guidelines usually differentiate cereal products with much added fat and sugar content (e.g., cakes, biscuits, pizza and pastries) from plain cereal products (Williams, 2012). One of the fundamental mechanisms for reducing the percentage of abdominal fat by the consumption of whole grains is a reduction in insulin and glucose responses and the subsequent enhancement in lipid oxidation and lipolysis (Pauline and Rimm, 2003, Pol *et al.*, 2013).

Table 1. General characteristics of normal-weight participants by gender

Variables	Male (n=157)	Female (n=171)	P-value[a]	Total (n=328)
Education (year)				
0	14 (8.9)[b]	20 (11.7)	0.653	34 (10.4)
1-11	65 (41.4)	65 (38.0)		130 (39.6)
≥12	78 (49.7)	86 (50.3)		164 (50)
Smoking	26 (16.6)	0 (0)	0.0001	26 (7.9)
Regular physical activity	13 (8.3)	9 (5.3)	0.275	22 (6.7)
Metabolic syndrome	10 (6.4)	31 (18.1)	0.001	41 (12.5)
Age (year)	42.4 ± 18.0[c]	36.4 ± 15.0	0.001	39.2 ± 16.8
Body mass index (kg/m^2)	22.5 ± 1.9	22.2 ± 2.4	0.265	22.3 ± 2.2
Waist circumference (cm)	84.0 ± 8.9	77.3 ± 8.3	0.0001	80.5 ± 9.2
Fasting blood glucose (mg/dL)	102.2 ± 35.6	95.4 ± 31.0	0.069	98.7 ± 33.4
Triglyceride (mg/dL)	131.8 ± 100.9	101.1 ± 65.0	0.001	115.8 ± 85.4
Systolic blood pressure (mmHg)	112.8 ± 12.7	108.8 ± 12.6	0.005	110.8 ± 12.8
Diastolic blood pressure (mmHg)	71.7 ± 12.5	66.8 ± 10.6	0.0001	69.2 ± 11.8
High density lipoprotein (mg/dL)	42.9 ± 8.3	49.8 ± 11.2	0.0001	46.5 ± 10.5

[a] : χ^2 test for categorical and Student *t-test* for continuous variables, [b]: number (%), [c]: mean±SD

The current research established a significant positive relationship between the consumption of chocolate and BP measurement in the study population. Observational and epidemiological studies reveal BP controlling and lowering

properties of dark chocolate in humans (Desch *et al.*, 2010, Hooper *et al.*, 2008). The Zutphen Elderly study revealed that males with highest tertile of cocoa intake had lower SBP and DBP than men with lowest tertile of cocoa intake

(Buijsse *et al.*, 2006). A meta-analysis by Taubert et al. revealed that consumption of cocoa and flavanol-rich dark chocolates has antihypertensive properties when compared to chocolates containing no or trifle flavanols (Taubert *et al.*, 2007). A number of dietary intervention studies established potential antihypertensive and cardio-protective effects of flavanol-rich products as well (Faridi *et al.*, 2008, Heiss *et al.*, 2007, Rostami *et al.*, 2015, Schroeter *et al.*, 2006, Taubert *et al.*, 2007).

A few mechanisms have been suggested that contribute to antihypertensive effect of cocoa-rich products. Most of the studies provide support for the involvement of flavanol content of cocoa in nitric oxide (NO) formation and its subsequent mediated effects on platelets inhibition, vascular tone regulation, and arterial vasodilation (Fitzpatrick *et al.*, 2000, Karim *et al.*, 2000, Kelishadi, 2010, Taubert *et al.*, 2007, Zhu *et al.*, 2002). Another possible pathway is cocoa stearic acid, magnesium, and the theobromine content (Muniyappa *et al.*, 2008). A DBP lowering effect for stearic acid content of cocoa products was observed by Simon et al. (Simon *et al.*, 1996), although no significant relationship was found in other experiments (Ding *et al.*, 2006, Storm *et al.*, 1997). On the other hand, no significant relationship was found between the consumption of cocoa products and BP levels in some other studies (Balzer *et al.*, 2008, Davison *et al.*, 2008, Muniyappa *et al.*, 2008, Njike *et al.*, 2011, Wang-Polagruto *et al.*, 2006).

Table 2. Comparison of proportion of high components of metabolic syndrome among demographic variables

Variables	High FBG[a]	High TG[a]	Low HDL-c[a]	High BP[a]	High WC[a]
Gender					
Male	50 (31.8)[b]	41 (26.1)	62 (39.5)	35 (22.3)	19 (12.1)
Female	36 (21.1)	25 (14.7)	96 (56.1)	30 (17.5)	71 (41.5)
P-value	0.026	0.009	0.003	0.281	0.0001
Total	86 (26.2)	66 (20.1)	158 (48.2)	65 (19.8)	90 (27.4)
Age (years)					
20-34	17 (10.4)	20 (12.2)	86 (52.4)	10 (6.1)	24 (14.6)
35-49	20 (29.9)	16 (23.9)	32 (47.8)	8 (11.9)	23 (34.3)
50-64	30 (46.9)	19 (29.7)	28 (43.8)	28 (43.8)	31 (48.4)
≥65	19 (57.6)	11 (33.3)	12 (36.4)	19 (57.6)	12 (36.4)
P-value[c]	0.0001*	0.003*	0.315	0.0001*	0.0001*
Education (years)					
0	18 (52.9)	8 (23.5)	16 (47.1)	20 (58.8)	22 (64.7)
1-11	40 (30.8)	30 (23.1)	66 (50.8)	26 (20.0)	41 (31.5)
≥12	28 (17.1)	28 (17.1)	76 (46.3)	19 (11.6)	27 (16.5)
P-value	0.0001	0.387	0.745	0.0001	0.0001
Smoking[d]					
Yes	9 (34.6)	8 (30.8)	10 (38.5)	6 (23.1)	1 (3.8)
No	77 (25.5)	58 (19.2)	148 (49.0)	59 (19.5)	89 (29.5)
P-value	0.310	0.158	0.302	0.664	0.005
Regular physical activity					
Yes	6 (27.3)	6 (27.3)	7 (31.8)	6 (27.3)	6 (27.3)
No	80 (26.1)	60 (19.6)	151 (49.3)	59 (19.3)	84 (27.5)
P-value	0.907	0.386	0.112	0.364	0.986

[a]: fasting blood glucose ≥100 mg/dL or drug treatment for elevated it; triglyceride ≥ 150 mg/dL; High density lipoprotein < 40 mg/dL in male and < 50 mg/dL in female; Bood pressure: Systolic ≥ 130 mmHg or Diastolic ≥ 85 mmHg or antihypertensive drug treatment; Waist circumference ≥ 94 cm in male and ≥ 80 cm in female [b] : All values are frequency (%), [c]: χ^2 test., Significant differences in multiple comparisons between different age groups (P-value $<\frac{0.05}{6}$), and education levels (P-value $<\frac{0.05}{3}$) obtained by post-hoc Bonferroni test, [d]: Smoking: Yes, current or past smoker; No, never smoker.

Table 3. The associations among metabolic syndrome components and snack types in normal-weight adults

Variables	High FBG[a]	High TG[a]	Low HDL-c[a]	High BP[a]	High WC[a]
Fruits	0.99 (NS)[b]	1.00 (NS)	1.00 (NS)	1.00 (NS)	0.99 (NS)
Cooked vegetables	1.00 (NS)	0.98 (NS)	1.00 (NS)	0.99 (NS)	1.00 (NS)
Raw vegetables	1.00 (NS)	0.99 (NS)	0.99 (NS)	0.99 (NS)	0.99 (NS)
Corn	1.01 (NS)	1.01 (NS)	0.97 (NS)	1.06 (NS)	1.02 (NS)
Corn puffs	1.00 (NS)	0.97 (NS)	1.00 (NS)	0.99 (NS)	0.99 (NS)
Seed kernel	1.02 (NS)	0.98 (NS)	1.00 (NS)	1.01 (NS)	0.98 (NS)
Biscuits or akes					
T1[c]	1.00 (--)	1.00 (--)	1.00 (--)	1.00 (--)	1.00 (--)
T2	1.01 (NS)	1.08 (NS)	0.91 (NS)	0.82 (NS)	1.14 (1.05–1.25)[d]
T3	1.37 (NS)	1.25 (NS)	1.18 (NS)	0.96 (NS)	1.23 (1.02–1.49)[d]
Chocolate					
T1	1.00 (--)	1.00 (--)	1.00 (--)	1.00 (--)	1.00 (--)
T2	1.06 (NS)	0.82 (NS)	0.99 (NS)	1.03 (1.01-1.05)[d]	1.11 (NS)
T3	1.25(NS)	1.08 (NS)	1.32 (NS)	1.10 (1.03-1.18)[d]	1.34 (NS)
Sweets	0.99 (NS)	1.01 (NS)	0.99 (NS)	1.01 (NS)	1.00 (NS)
Candy	0.99 (NS)	1.06 (NS)	0.96 (NS)	0.98 (NS)	0.93 (NS)
Carbonated beverages	0.99 (NS)	1.00 (NS)	1.00 (NS)	1.00 (NS)	1.00 (NS)
Fast foods	1.01 (NS)	0.99 (NS)	0.99 (NS)	1.01 (NS)	1.00 (NS)
Falafel or Samboose	1.00 (NS)	0.99 (NS)	0.99 (NS)	0.99 (NS)	0.99 (NS)

[a]: fasting blood glucose ≥100 mg/dL or drug treatment for elevated it; triglyceride ≥ 150 mg/dL; High density lipoprotein < 40 mg/dL in male and < 50 mg/dL in female; Bood pressure: Systolic ≥ 130 mmHg or Diastolic ≥ 85 mmHg or antihypertensive drug treatment; Waist circumference ≥ 94 cm in male and ≥ 80 cm in female, [b]: OR (95% CI) are obtained by multiple binary logistic regression adjusted for age, gender, ethnicity, marital status, education, smoking, physical activity, and BMI, [c] : ORs in T2 and T3 rows indicate the risk of incident metabolic syndrome component among those in the 2nd and 3rd tertile of the independent variable, respectively, compared with those in the 1st tertile. T1: 1st tertile; T2: 2nd tertile; T3: 3rd tertile. [d] : Significant correlation with the MetS component, NS: Non-significant

In a group of university graduates no protection of chocolate consumption for new-onset hypertension was observed (Alonso *et al.*, 2005). Nearly little or no antihypertensive effects were observed in four studies with long-term consumption of high doses of separated cocoa flavanols, despite endothelial function improvements (Balzer *et al.*, 2008, Davison *et al.*, 2008, Muniyappa *et al.*, 2008, Wang-Polagruto *et al.*, 2006). This series of studies concluded that BP changes cannot be determined only by the dose of cocoa flavanols (Davison *et al.*, 2010), synergistic action of chocolate matrix with flavanols is crucial for antihypertensive effects of chocolate (van den Bogaard *et al.*, 2010).

Variations between the findings of current research and studies in favor of antihypertensive effects of high-cocoa chocolate products can be partly explained by the fact that the pure dark chocolate samples, rich in flavanols, prepared for interventional studies differ from readily commercial products in stores (Alonso *et al.*, 2005). Moreover, all sorts of products containing any amount of cocoa are known as chocolate by the public. The flavanol content of commercially available chocolates varies widely (Bordeaux *et al.*, 2007, Grassi *et al.*, 2010, Kelishadi, 2010) and largely depends on the cultivar type, post-harvest handling, processing, preparation, and percentage of flavanol-rich cocoa used (Bordeaux *et al.*, 2007,

Grassi *et al.*, 2010). Therefore, consumption of cocoa and flavanol-poor chocolate products by the general population (Alonso *et al.*, 2005) is one of the factors contributing to these inconsistencies.

High content of sugar and fat in common chocolate products consumed by public (Forslund *et al.*, 2005) is another factor responsible for this inconsistency (Davison *et al.*, 2010). Commercially available chocolate products are a combination of natural cocoa solids, sugar, and/or other additional ingredients (Grassi *et al.*, 2010). Large amounts of fat and sugar in usual varieties of chocolate products, such as milk chocolate and chocolate candy inhibit the beneficial effects of chocolate and convert it to a high-calorie (500 kcal/100g) and low nutrient density food product (Drewnowski, 1987, Jean, 1994, Khanafari and Porgham, 2012, Maddah *et al.*, 2009). So, excessive consumption of high energy-density chocolates will result in obesity and subsequently hypertension, diabetes, dyslipidemia, and other cardiovascular risk factors (Buitrago-Lopez *et al.*, 2011, Corti *et al.*, 2009).

A positive role was suggested for fructose-containing sugars in development of hypertension, obesity, diabetes, MetS, and kidney disease (Johnson *et al.*, 2007). A data analysis by Jalal et al. on NHANES data showed that high consumption of foods such as chocolates and candies with high quantities of fructose-containing sweeteners (e.g. sucrose and high-fructose corn syrup) was related to elevated BP among US adults without a history of hypertension (Jalal *et al.*, 2010). It is notable that fructose is the only sugar that can induce elevated uric acid levels (Stirpe *et al.*, 1970), which is a byproduct of fructose metabolism process (Perheentupa and Raivio, 1967) and an independent predictor of hypertension in humans (Forman *et al.*, 2007, Krishnan *et al.*, 2007, Shankar *et al.*, 2006). According to Johnson et al., the fructose consumption and its uric acid-mediated mechanism are considerably important in initial development of MetS and they have a less significant role when obesity, hypertension, and renal disease are established (Johnson *et al.*, 2007).

The current study probably had some strengths and limitations. The study population consisted of a random selection of normal-weight adults. To the best of our knowledge, this was the first study investigating the association between snacking and MetS components in normal-weight adults in southwest of Iran. Further, the newly developed 50-item FFQ questionnaire can be a valuable tool for investigating dietary intake in both clinical settings and large population studies with time constraints.

Some limitations of this study should be considered in interpretation of findings. The most important limitation of this study was its cross-sectional nature that prevents making causal inferences. Although some relevant confounding factors were adjusted, other possible factors such as unhealthy lifestyle behaviors associated with metabolic disorders can be more precisely determined and ruled out in future studies. Though the short FFQ is a valuable tool for rapid assessment of dietary intake, it has the potential bias for dietary recall. The relatively small odds ratios for the positive effect of chocolates, cakes, and biscuits on MetS components could be inferred as a low clinical significance. Thus, large prospective cohort studies with prospective quantitative dietary assessment methods (e.g., dietary records) are required to investigate the causal relationship between consumption of high-carbohydrate snacks and metabolic disorders in normal-weight adults in southwest of Iran.

Conclusions

Intake of snacks containing high fat, sugar, and refined carbohydrate in forms of supermarket cakes and biscuits as well as low-flavanol content chocolate products is one of the dietary habits contributing to abdominal obesity and hypertension in normal-weight adults in southwest of Iran. High occurrence of hypertension and abdominal obesity among older participants with lower education shows the immediate need to improve public knowledge about healthy snacking. Educational courses should be conducted to prevent the development

of metabolic disorders in normal-weight participants, control the ascending trend of MetS, and enhance public health status.

Authors' contribution

The contribution of all authors to this work is as follows: Niknejad N, Zare Javid A and Shahbazian H designed research; Niknejad N, Latifi SM and Hormoznejad R conducted research; Niknejad N, Latifi SM and Niknejad B performed statistical analysis; Niknejad N and Niknejad B wrote the paper; Niknejad N and Zare Javid A had primary responsibility for final content. All authors have read and approved the final manuscript.

Conflict of interest

None declared.

References

Alberti K, et al. 2009. Harmonizing the Metabolic Syndrome A Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation.* **120 (16)**: 1640-1645.

Alonso A, de la Fuente C, Beunza JJ, Sánchez-Villegas A & Martínez-González MÁ 2005. Chocolate consumption and incidence of hypertension. *Hypertension.* **46 (6)**: e21-e22.

Balzer J, et al. 2008. Sustained benefits in vascular function through flavanol-containing cocoa in medicated diabetic patients: a double-masked, randomized, controlled trial. *Journal of the American college of cardiology.* **51 (22)**: 2141-2149.

Bednarek-Tupikowska G, et al. 2012. Evaluation of the prevalence of metabolic obesity and normal weight among the Polish population. *Endokrynologica polska.* **63 (6)**: 447-455.

Bordeaux B, et al. 2007. Casual chocolate consumption and inhibition of platelet function. *Preventive cardiology.* **10 (4)**: 175-180.

Buijsse B, Feskens EJ, Kok FJ & Kromhout D 2006. Cocoa intake, blood pressure, and cardiovascular mortality: the Zutphen Elderly Study. *Archives of internal medicine.* **166 (4)**: 411-417.

Buitrago-Lopez A, et al. 2011. Chocolate consumption and cardiometabolic disorders: systematic review and meta-analysis. *British medical journal.* **343**: d4488.

Choi J, et al. 2012. Characteristics of diet patterns in metabolically obese, normal weight adults (Korean National Health and Nutrition Examination Survey III, 2005). *Nutrition, metabolism & cardiovascular diseases.* **22 (7)**: 567-574.

Corti R, Flammer AJ, Hollenberg NK & Lüscher TF 2009. Cocoa and cardiovascular health. *Circulation.* **119 (10)**: 1433-1441.

Davison K, et al. 2010. Dose-related effects of flavanol-rich cocoa on blood pressure. *Journal of human hypertension.* **24 (9)**: 568-576.

Davison K, Coates A, Buckley J & Howe P 2008. Effect of cocoa flavanols and exercise on cardiometabolic risk factors in overweight and obese subjects. *International journal of obesity.* **32 (8)**: 1289-1296.

De Lorenzo A, Martinoli R, Vaia F & Di Renzo L 2006. Normal weight obese (NWO) women: an evaluation of a candidate new syndrome. *Nutrition, metabolism & cardiovascular diseases.* **16 (8)**: 513-523.

Desch S, et al. 2010. Effect of cocoa products on blood pressure: systematic review and meta-analysis. *American journal of hypertension.* **23 (1)**: 97-103.

Ding EL, Hutfless SM, Ding X & Girotra S 2006. Chocolate and prevention of cardiovascular disease: a systematic review. *Nutrition & metabolism.* **3 (1)**: 1-12.

Drewnowski A 1987. Changes in mood after carbohydrate consumption. *The American journal of clinical nutrition.* **46 (4)**: 703-705.

Esteghamati A, et al. 2009. Third national Surveillance of Risk Factors of Non-Communicable Diseases (SuRFNCD-2007) in Iran: methods and results on prevalence of diabetes, hypertension, obesity, central obesity, and dyslipidemia. *BMC Public Health.* **9 (1)**: 167.

Fan Y, et al. 2016. Plasma endothelin-1 level as a predictor for poor collaterals in patients with≥ 95% coronary chronic occlusion. *Thrombosis research.* **142**: 21-25.

Faridi Z, Njike VY, Dutta S, Ali A & Katz DL 2008. Acute dark chocolate and cocoa ingestion and endothelial function: a randomized controlled crossover trial. *The American journal of clinical nutrition.* **88 (1)**: 58-63.

Fitzpatrick DF, Fleming RC, Bing B, Maggi DA & O'Malley RM 2000. Isolation and characterization of endothelium-dependent vasorelaxing compounds from grape seeds. *Journal of agricultural and food chemistry.* **48 (12)**: 6384-6390.

Forman JP, Choi H & Curhan GC 2007. Plasma uric acid level and risk for incident hypertension among men. *Journal of the American society of nephrology.* **18 (1)**: 287-292.

Forslund HB, Torgerson JS, Sjöström L & Lindroos A-K 2005. Snacking frequency in relation to energy intake and food choices in obese men and women compared to a reference population. *International journal of obesity.* **29 (6)**: 711-719.

Ghassemi H, Harrison G & Mohammad K 2002. An accelerated nutrition transition in Iran. *Public health nutrition.* **5 (1a)**: 149-155.

Giacco R, et al. 2013. Effects of rye and whole wheat versus refined cereal foods on metabolic risk factors: a randomised controlled two-centre intervention study. *Clinical nutrition.* **32 (6)**: 941-949.

Grassi D, Desideri G & Ferri C 2010. Blood pressure and cardiovascular risk: what about cocoa and chocolate? *Archives of biochemistry and biophysics.* **501 (1)**: 112-115.

Hadaegh F, Esmaillzadeh A & Azizi F 2007a. Metabolic risks in individuals with normal body mass index and normal waist circumference. *European journal of cardiovascular prevention and rehabilitation : official journal of the European Society of Cardiology, Working Groups on Epidemiology & Prevention and Cardiac Rehabilitation and Exercise Phys.* **14 (2)**: 200-207.

Hadaegh F, Zabetian A, Harati H & Azizi F 2007b. Metabolic syndrome in normal-weight Iranian adults. *Annals of Saudi medicine.* **27 (1)**: 18-24.

Halkjær J, Tjønneland A, Thomsen BL, Overvad K & Sørensen TI 2006. Intake of macronutrients as predictors of 5-y changes in waist circumference. *The American journal of clinical nutrition.* **84 (4)**: 789-797.

Harland JI & Garton LE 2008. Whole-grain intake as a marker of healthy body weight and adiposity. *Public health nutrition.* **11 (06)**: 554-563.

Heiss C, et al. 2007. Sustained increase in flow-mediated dilation after daily intake of high-flavanol cocoa drink over 1 week. *Journal of cardiovascular pharmacology.* **49 (2)**: 74-80.

Hooper L, et al. 2008. Flavonoids, flavonoid-rich foods, and cardiovascular risk: a meta-analysis of randomized controlled trials. *The American journal of clinical nutrition.* **88 (1)**: 38-50.

Jalal DI, Smits G, Johnson RJ & Chonchol M 2010. Increased fructose associates with elevated blood pressure. *Journal of the American society of nephrology.* **21 (9)**: 1543-1549.

Jean AP 1994. Bowes & Church's Food Values of Portions Commonly Used: Spiral (Bowes and Church's Food Values of Portions Commonly Used).

Johnson RJ, et al. 2007. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *The American journal of clinical nutrition.* **86 (4)**: 899-906.

Jonnalagadda SS, et al. 2011. Putting the whole grain puzzle together: health benefits associated with whole grains—summary of American Society for Nutrition 2010 Satellite Symposium. *The journal of nutrition.* **141 (5)**: 1011S-1022S.

Karim M, McCormick K & Kappagoda CT 2000. Effects of cocoa extracts on endothelium-dependent relaxation. *The journal of nutrition.* **130 (8)**: 2105S-2108S.

Katsuki A, et al. 2003. Increased visceral fat and serum levels of triglyceride are associated with insulin resistance in Japanese metabolically obese, normal weight subjects with normal glucose tolerance. *Diabetes care.* **26 (8)**: 2341-2344.

Kelishadi R 2010. Cacao to cocoa to chocolate: healthy food? *ARYA atherosclerosis journal.***88 (6)** 1685-1696.

Kelishadi R, et al. 2008. Metabolically obese normal weight and phenotypically obese metabolically normal youths: the CASPIAN Study. *Journal of Amerrican diet association.* **108 (1)**: 82-90.

Kelly T, Yang W, Chen C-S, Reynolds K & He J 2008. Global burden of obesity in 2005 and projections to 2030. *International journal of obesity.* **32 (9)**: 1431-1437.

Khanafari A & Porgham SH 2012. Investigation of probiotic chocolate effect on Streptococcus mutans growth inhibition. *Jundishapur journal of microbiology.* **5 (4)**: 590-597.

Koh-Banerjee P, et al. 2004. Changes in whole-grain, bran, and cereal fiber consumption in relation to 8-y weight gain among men. *The American journal of clinical nutrition.* **80 (5)**: 1237-1245.

Krishnan E, Kwoh CK, Schumacher HR & Kuller L 2007. Hyperuricemia and incidence of hypertension among men without metabolic syndrome. *Hypertension.* **49 (2)**: 298-303.

Kristensen M, et al. 2012. Whole grain compared with refined wheat decreases the percentage of body fat following a 12-week, energy-restricted dietary intervention in postmenopausal women. *The journal of nutrition.* **142 (4)**: 710-716.

Kwon BJ, et al. 2013. Metabolically obese status with normal weight is associated with both the prevalence and severity of angiographic coronary artery disease. *Metabolism: clinical and experimental.* **62 (7)**: 952-960.

Landberg R, et al. 2010. Rye whole grain and bran intake compared with refined wheat decreases urinary C-peptide, plasma insulin, and prostate specific antigen in men with prostate cancer. *The journal of nutrition.* **140 (12)**: 2180-2186.

Maddah M, Rashidi A, Mohammadpour B, Vafa R & Karandish M 2009. In-school snacking, breakfast consumption, and sleeping patterns of normal and overweight Iranian high school girls: a study in urban and rural areas in Guilan, Iran. *Journal of nutrition education and behavior.* **41 (1)**: 27-31.

Marques-Vidal P, et al. 2010. Normal weight obesity: relationship with lipids, glycaemic status, liver enzymes and inflammation. *Nutrition, metabolism and cardiovascular diseases.* **20 (9)**: 669-675.

Mirmiran P, Bahadoran Z, Delshad H & Azizi F 2014. Effects of energy-dense nutrient-poor snacks on the incidence of metabolic syndrome: a prospective approach in Tehran Lipid and Glucose Study. *Nutrition.* **30 (5)**: 538-543.

Mirmiran P, Esfahani FH, Mehrabi Y, Hedayati M & Azizi F 2010. Reliability and relative validity of an FFQ for nutrients in the Tehran lipid and glucose study. *Public health nutrition.* **13 (5)**: 654-662.

Muniyappa R, et al. 2008. Cocoa consumption for 2 wk enhances insulin-mediated vasodilatation without improving blood pressure or insulin resistance in essential hypertension. *The American journal of clinical nutrition.* **88 (6)**: 1685-1696.

Naeem Z 2012. Increasing trend of Junk food use in Saudi Arabia and health implications.

International journal of health sciences. **6 (1)**: V-VI.

National Cholesterol Education Program Expert Panel on Detection E & Treatment of High Blood Cholesterol in A 2002. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation.* **106 (25)**: 3143-3421.

Njike VY, et al. 2011. Effects of sugar-sweetened and sugar-free cocoa on endothelial function in overweight adults. *International journal of cardiology.* **149 (1)**: 83-88.

O'Connor L, Brage S, Griffin SJ, Wareham NJ & Forouhi NG 2015. The cross-sectional association between snacking behaviour and measures of adiposity: the Fenland Study, UK. *British journal of nutrition.* **114 (8)**: 1286-1293.

O'Neil CE, Zanovec M, Cho SS & Nicklas TA 2010. Whole grain and fiber consumption are associated with lower body weight measures in US adults: National Health and Nutrition Examination Survey 1999-2004. *Nutrition research.* **30 (12)**: 815-822.

Park Y-W, et al. 2003. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. *Archives of internal medicine.* **163 (4)**: 427-436.

Pauline K-B & Rimm EB 2003. Whole grain consumption and weight gain: a review of the epidemiological evidence, potential mechanisms and opportunities for future research. *Proceedings of the nutrition society.* **62 (01)**: 25-29.

Perheentupa J & Raivio K 1967. Fructose-induced hyperuricaemia. *The lancet.* **290 (7515)**: 528-531.

Phillips CM & Perry IJ 2015. Lipoprotein particle subclass profiles among metabolically healthy and unhealthy obese and non-obese adults: Does size matter? *Atherosclerosis.* **242 (2)**: 399-406.

Piernas C & Popkin BM 2010. Snacking increased among US adults between 1977 and 2006. *The journal of nutrition.* **140 (2)**: 325-332.

Pol K, et al. 2013. Whole grain and body weight changes in apparently healthy adults: a systematic review and meta-analysis of randomized controlled studies. *The American journal of clinical nutrition.* **98 (4)**: 872-884.

Popkin BM, Adair LS & Ng SW 2012. Global nutrition transition and the pandemic of obesity in developing countries. *Nutrition reviews.* **70 (1)**: 3-21.

Primeau V, et al. 2011. Characterizing the profile of obese patients who are metabolically healthy. *International journal of obesity.* **35 (7)**: 971-981.

Qadan LR, Ahmed AA, Safar HA, Al-Bader MA & Ali AA 2008. Prevalence of metabolic syndrome in patients with clinically advanced peripheral vascular disease. *Angiology.***29:** 198-202

Roche HM, Phillips C & Gibney MJ 2005. The metabolic syndrome: the crossroads of diet and genetics. *Proceedings of the nutrition society* **64 (3)**: 371-377.

Romero-Corral A, et al. 2010. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. *European heart journal.* **31 (6)**: 737-746.

Rostami A, et al. 2015. High-cocoa polyphenol-rich chocolate improves blood pressure in patients with diabetes and hypertension. *ARYA atherosclerosis.* **11 (1)**: 21.

Ruderman N, Chisholm D, Pi-Sunyer X & Schneider S 1998. The metabolically obese, normal-weight individual revisited. *Diabetes.* **47 (5)**: 699-713.

Ruderman NB, Schneider SH & Berchtold P 1981. The "metabolically-obese," normal-weight individual. *The American journal clinical nutrition.* **34 (8)**: 1617-1621.

Sarrafzadegan N, et al. 2011. Metabolic syndrome and health-related quality of life in Iranian population. *Journal of research in medical sciences.* **16 (3)**: 254-261.

Schroeter H, et al. 2006. (–)-Epicatechin mediates beneficial effects of flavanol-rich cocoa on

vascular function in humans. *Proceedings of the national academy of sciences of the united states of America.* **103 (4)**: 1024-1029.

Shahbazian H, et al. 2013. Metabolic syndrome and its correlated factors in an urban population in South West of Iran. *Journal of diabetes and metabolic disorders.* **12 (1)**: 1-11.

Shankar A, Klein R, Klein B & Nieto F 2006. The association between serum uric acid level and long-term incidence of hypertension: population-based cohort study. *Journal of human hypertension.* **20 (12)**: 937-945.

Sharifi F, Mousavinasab S, Saeini M & Dinmohammadi M 2009. Prevalence of metabolic syndrome in an adult urban population of the west of Iran. *Experimental diabetes research.* **2009**.

Shimizu C, et al. 2008. Effect of high β-glucan barley on serum cholesterol concentrations and visceral fat area in Japanese men—a randomized, double-blinded, placebo-controlled trial. *Plant foods for human nutrition.* **63 (1)**: 21-25.

Simon JA, Fong J & Bernert JT 1996. Serum fatty acids and blood pressure. *Hypertension.* **27 (2)**: 303-307.

Stirpe F, et al. 1970. Fructose-induced hyperuricaemia. *The lancet.* **296 (7686)**: 1310-1311.

Storm H, et al. 1997. Comparison of a carbohydrate-rich diet and diets rich in stearic or palmitic acid in NIDDM patients: effects on lipids, glycemic control, and diurnal blood pressure. *Diabetes care.* **20 (12)**: 1807-1813.

Suliga E, Kozieł D, Cieśla E & Głuszek S 2015. Association between dietary patterns and metabolic syndrome in individuals with normal weight: a cross-sectional study. *Nutrition journal.* **14 (1)**: 141-155.

Taubert D, Roesen R, Lehmann C, Jung N & Schömig E 2007. Effects of low habitual cocoa intake on blood pressure and bioactive nitric oxide: a randomized controlled trial. *The journal of American medical association.* **298 (1)**: 49-60.

van den Bogaard B, et al. 2010. Effects on peripheral and central blood pressure of cocoa with natural or high-dose theobromine a randomized, double-blind crossover trial. *Hypertension.* **56 (5)**: 839-846.

Wang-Polagruto JF, et al. 2006. Chronic consumption of flavanol-rich cocoa improves endothelial function and decreases vascular cell adhesion molecule in hypercholesterolemic postmenopausal women. *Journal of cardiovascular pharmacology.* **47**: S177-S186.

WHO 1987. Measuring obesity—classification and description of anthropometric data. Report on a WHO consultation of the epidemiology of obesity. Warsaw 21-23 October 1987. Copenhagen: WHO, 1989.

Williams PG 2012. Evaluation of the evidence between consumption of refined grains and health outcomes. *Nutrition reviews.* **70 (2)**: 80-99.

Yoo HJ, et al. 2014. Association of metabolically abnormal but normal weight (MANW) and metabolically healthy but obese (MHO) individuals with arterial stiffness and carotid atherosclerosis. *Atherosclerosis.* **234 (1)**: 218-223.

Zabetian A, Hadaegh F & Azizi F 2007. Prevalence of metabolic syndrome in Iranian adult population, concordance between the IDF with the ATPIII and the WHO definitions. *Diabetes research and clinical practice.* **77 (2)**: 251-257.

Zaliūnas R, et al. 2007. [Prevalence of metabolic syndrome components in patients with acute coronary syndromes]. *Medicina (Kaunas, Lithuania).* **44 (3)**: 182-188.

Zhu QY, Holt RR, Lazarus SA, Orozco TJ & Keen CL 2002. Inhibitory effects of cocoa flavanols and procyanidin oligomers on free radical-induced erythrocyte hemolysis. *Experimental biology and medicine.* **227 (5)**: 321-329.