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Effect of low glycemic index and food exchange system diets on metabolic syndrome components in Saudi medical students.

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Abstract

Metabolic Syndrome (MetS), a condition that manifests as a set of metabolic risk factors, is considered a global epidemic. The prevalence of MetS in Saudi Arabia has grown rapidly over the past decade. The aim of the present study was to determine the effect of Low Glycemic Index (LGI) and Food Exchange System (FES) diets on MetS components in patients with MetS in the Saudi population. We selected 69 subjects and divided them into 3 groups: LGI diet, FES diet, and control. Each group consisted of 23 subjects (12 women). Both treatment groups followed diet and physical activity regimens for 5 days per week for 6 weeks. Serum samples were taken pre- and post-treatment in all 3 groups and used for biochemical analyses. The results of study found significant difference in post-treatment values of hypertension i.e. SBP (p=0.007), DBP (p=0.01), Fasting Blood Glucose (FBG) (p=0.005), Triglycerides (TG) (p=0.0006) and Total Cholesterol (TC) (p=0.0006). There were no significant differences between groups in waist circumference (p=0.23) and high density lipoprotein (p=0.12). When the data from men and women were examined separately, we found significant differences between groups in the pre-treatment values of TC (p=0.01) and the post-treatment values of FBG (p=0.65) and TG (p=0.99). We conclude that the LGI and FES diets are beneficial for patients with MetS, with a more potent impact produced by the LGI diet. We recommend implementing nutritional awareness and motivational counselling programs to help to improve the health of people with MetS in the Saudi population.

Keywords: Metabolic Syndrome (MetS), Low Glycemic Index (LGI), Food Exchange System (FES), Glycemic Index (GI), Diet.

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Introduction

Metabolic Syndrome (MetS) is a multifactor disorder characterized by three or more abnormal metabolic risk factor traits including high Triglycerides (TG), Hypertension (HTN), high Fasting Blood Glucose (FBG), high Waist Circumference (WC), and low High Density Lipoprotein Cholesterol (HDL-C) [1]. In Saudi Arabia and other countries, the prevalence of MetS has been estimated to be between 18% and 40%, depending on the specific population studied. Economic and social transformations have caused a shift to a sedentary lifestyle, resulting in an increasing prevalence of obesity, and an expected rise in the prevalence of MetS [2]. The National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) documented that about 50 to 75 million people in the USA have MetS. In Europe, the prevalence rates of MetS among men and women are 15.7% and 14.2%, respectively. In Saudi Arabia, 66% of adult men and 71% of adult women are either overweight or obese [3]. In the Gulf Cooperation Council (GCC) countries, Kuwait, Oman, Saudi Arabia, and the United Arab Emirates have the highest prevalence of diabetes [4]. Limited information is available on the prevalence of MetS in Saudi Arabia, and most surveys in the population involve standard research targets other than MetS.

Diet patterns play a crucial role in controlling MetS, which often requires weight reduction as most people with MetS are overweight, obese, or morbidly obese. Because abdominal obesity in particular is a predisposing factor for MetS, specific nutritional influences on body fat distribution are important, although mechanisms are not yet exhaustively defined [5,6]. Earlier diet concept studies have suggested that MetS can be effectively controlled by regulating insulin resistance through Glycemic Index (GI). This theory focuses on how carbohydrate-rich foods can influence the physiological positions of the human body. The GI theory compares the ability of carbohydrate-rich foods to elevate blood glucose levels with that of a portion of a reference food (usually glucose or white bread) during the first 2 hours after consumption [7]. This framework classifies carbohydrate-containing foods as having a high, medium, or low GI. For people with diabetes, LGI foods or carbohydrate-containing foods that result in a slight increase in blood glucose levels are considered more appropriate than High Glycemic Index (HGI) foods [8]. There have been no studies on the effect of diet therapy on MetS in Saudi Arabia. Therefore, the aim of the present study was to investigate the effects of LGI and FES diets on MetS components in patients in the Saudi population.

Materials and Methodology

Enrolment of subjects

This study was carried out in King Khalid University Hospitals (KKUH), King Saud University, Riyadh, Saudi Arabia. Obese and morbidly obese subjects (n=69) with Body Mass Indices (BMIs) between 28.6 and 58.1 were recruited from the obesity center clinic. To be included, subjects had to be within the age range of 19-65 years and exhibit MetS per the International Diabetes Foundation (IDF) criteria [9]. Subjects were excluded if they were following a weight reduction program or taking weight reduction supplements with or without an accompanying diet, affected by other metabolic disorders, had undergone bariatric surgery, were pregnant, or had adverse physical or psychological conditions. The selected 69 subjects were divided into 3 groups: LGI diet, FES diet, and control (Table 1). Each of the 3 groups consisted of 23 subjects (12 women). Selected subjects were examined in a designated room and anthropometric, clinical, demographic, and medical data were recorded and maintained confidentially. Ethical approval was obtained from KKUH and all the subjects signed the consent form prior to participating in the study.

Diet program and follow-up

Subjects followed prescribed diets and physical activity regimens for 6 weeks. During the study period, both treatment groups were also presented with a training program that focused on commitment to long-term lifestyle improvements. Control group subjects were not provided with any information about the program and were evaluated as regular clinic patients. Prior to the start of the treatment period, subjects were surveyed on their lifestyles and eating habits, including questions about beverages consumed, including soft drinks, vegetable and fruit juices. The baseline values for all subjects are tabulated in Table 2. After the completion of study period, all subjects were asked to attend the KKUH clinic with a 12 hour overnight fast before having post-treatment anthropometric, biochemical, and clinical measurements taken. The post-treatment measurements are tabulated in Table 3. The training program included lectures, workshops, and written materials. Ten- to fifteen-minute motivational counselling sessions were conducted by telephone. Dietary compliance was monitored through regular telephone interviews. In addition, regular contact was maintained through mobile messages or e-mails. The intervention protocol, risks of participation, and study applications were explained to both treatment groups through lectures Treatment group subjects were instructed on their respective diet plans, while control subjects were asked to maintain their routine diet activities during the intervention.

Anthropometric measurement and sample collection

Anthropometric measurements including HTN, BMI, and WC were measured. HTN was based on Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP), measured using an electrosphygmomanometer (Datascope Accutorr plus NIBP with Trend Screen and Datascope SpO_2 with the recorder module, Datascope Corporation, USA). Weight was measured without excess clothing, shoes, or socks, on electronic scales (InBody model 720, Germany). Height (cm) was measured using a freestanding stadiometer (Seca, Vogel and Halke, Hamburg, Germany). BMI was calculated as weight (kg) divided by height squared (m^2) [10,11]. WC (cm) was measured midway between the lowest rib and iliac crest [12]. Venous blood samples were collected after an overnight fast and 5 ml serum samples were separated for biochemical analyses. Serum was analysed for Fasting Blood Glucose (FBG) and lipid profile measurements, including TC, TG, HDL-C, and low Density Lipoprotein-Cholesterol (LDL-C) [13]. HDL-C levels were measured using enzymatic/ cholesterol oxidase and TG levels were measured using enzymatic/lipase/glycerol kinase [14].

Statistical analysis

Data were expressed as mean ± SEM values. Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) version 19 software (SPSS Inc., Chicago, USA). The significance level used for all the tests was set at P ≤ 0.05. A t-test or 1-way Analysis of Variance (ANOVA) was performed to compare changes between groups.

Table 1. *Clinical characteristic features of subjects involved in this study.*

S. no	Characteristic features	Total (n=69)	Range (Minimum-Maximum)
1	Age (Years)	43.5 ± 12.7	(19-65)
2	Gender (M: F)*	(47.8%: 52.2%)	(1-69)
3	BMI (kg/m^2)	41 ± 7.8	(28.6-58.1)
4	Weight (Kg)	112 ± 23.9	(69.5-167.3)
5	Height (cms)	164.4 ± 9.2	(128-193)
6	SBP (mmHg)	145 ± 16.9	(103-180)
7	DBP (mmHg)	88.8 ± 8.3	(69-110)
8	Waist (cms)	117.7 ± 12	(98-145)
9	FBG (mmol/l)	7.1 ± 2.5	(4.6-9.6)
10	TC (mmol/l)	5.0 ± 0.9	(3.5-7.9)
11	TG (mmol/l)	1.7 ± 0.8	(0.59-4.7)
12	HDL-C (mmol/l)	1.1 ± 0.3	(0.49-2.6)
13	LDL-C (mmol/l)	3.0 ± 0.8	(1.5-5.11)

*Male: Female

Table 2. Calculation of pre-values between the 3 subjects using ANOVA analysis.

S. no	Characteristic features	Controls (n=23)	FES (n=23)	LGI (n=23)	P-value
1	Age (Years)	43.5 ± 12.7	43.7 ± 13.7	42.1 ± 11.6	0.89
2	Gender (Male: Female)	(11:12)	(11:12)	(11:12)	1
3	BMI (kg/m^2)	40.9 ± 7.8	41.4 ± 8.6	41.1 ± 7.8	0.98
4	Weight (Kg)	112 ± 23.9	111 ± 23.9	113.3 ± 21.9	0.94
5	SBP (mmHg)	145 ± 16.9	144.5 ± 18.6	146.3 ± 15.3	0.93
6	DBP (mmHg)	88.8 ± 8.3	89.4 ± 9.8	87.6 ± 6.3	0.75
7	Waist (cms)	117.7 ± 12	115.1 ± 10.8	122.3 ± 14.3	0.41
8	FBG (mmol/l)	7.1 ± 2.5	6.9 ± 2.3	7.7 ± 2.3	0.49
9	TC (mmol/l)	4.8 ± 0.8	5.2 ± 1.0	4.9 ± 0.9	0.3
10	TG (mmol/l)	1.7 ± 0.8	1.8 ± 0.8	1.7 ± 0.9	0.89
11	HDL-C (mmol/l)	1.1 ± 0.3	1.1 ± 0.4	1.0 ± 0.2	0.45
12	LDL-C (mmol/l)	3.0 ± 0.8	3.1 ± 0.9	3.0 ± 0.8	0.89

Table 3. Calculation of post-values between the 3 subjects using ANOVA analysis.

S. no	Characteristic features	Controls (n=23)	FES (n=23)	LGI (n=23)	P-value
1	BMI (kg/m^2)	41.2 ± 7.5	39.1 ± 9.0	38 ± 7.8	0.4
2	Weight (Kg)	112.8 ± 28.1	105.4 ± 24.1	104.9 ± 22.1	0.48
3	SBP (mmHg)	141.9 ± 14.2	132.9 ± 14.3	123.3 ± 11.8	0.007
4	DBP (mmHg)	80.5 ± 9.8	81.2 ± 8.6	74.8 ± 5.8	0.01
5	Waist (cms)	117.2 ± 10.1	111.2 ± 11.8	115.3 ± 13.9	0.23
6	FBG (mmol/l)	7.5 ± 3.4	6.5 ± 1.9	6.2 ± 1.9	0.005
7	TC (mmol/l)	4.8 ± 0.6	5.0 ± 0.9	4.1 ± 0.8	0.0005
8	TG (mmol/l)	1.6 ± 0.7	2.1 ± 0.8	1.3 ± 0.5	0.0006
9	HDL-C (mmol/l)	1.0 ± 0.3	1.0 ± 0.3	1.0 ± 0.2	0.12
10	LDL-C (mmol/l)	3.0 ± 0.6	3.0 ± 0.9	2.4 ± 0.7	0.009

Results

General assessments

Sixty-nine subjects were enrolled in our study (Table 1). The mean age was 43.5 ± 12.7 with an age range of 19-65 years. Of all subjects, 47.8% were male and 52.2% were female. The mean BMI and weight were 41 ± 7.8 and 112 ± 23.9 kg, respectively. Mean SBP was 145 ± 16.9 and mean DBP was 88.8 ± 8.3. SBP and DBP were used as indicators of hypertension. Mean FBG was 7.1 ± 2.5. Mean values of lipid profile measurements were as follows: 5.0 ± 0.9 for TC, 1.7 ± 0.8 for TG, 1.1 ± 0.3 for HDL-C, and 3.0 ± 0.8 for LDL-C.

ANOVA analysis of pre- and post-treatment values

ANOVA analysis was performed on the 3 study groups. There was no significant difference between groups with respect to age (p=0.89) or gender (p=1.0). We found no significant difference in SBP, DBP, FBG, TC, TG, HDL-C, or LDL-C between the 3 groups at baseline. However, we observed statistical differences between groups in the post-treatment measurements ($p<0.05$) of SBP (p=0.007), DBP (p=0.01), FBG (p=0.005), TC (p=0.0005), TG (p=0.0006), and LDL-C (p=0.009). We did not observe differences in BMI, weight, WC, or HDL-C ($p>0.05$) (Tables 2 and 3).

Gender association analysis of pre- and post-treatment values

We compared pre- and post-treatment values of anthropometric, biochemical, and clinical measurements by gender (Tables 4 and 5). The difference between mean ages of male (36.3 ± 12.1) and female (50.0 ± 9.3) subjects was not statistically significant ($p<0.05$). At baseline, there was a significant difference in TC (p=0.01) between the 3 groups and no significant difference in any of the other measures ($p>0.05$). Post treatment, FBG (p=0.01) and TC (p=0.04) were significantly different between the 3 groups ($p<0.05$). There were no significant differences in the other measures ($p>0.05$).

Table 4. Comparison of pre-values between males and female's subjects.

S. no	Characteristic features	Males	Females	P value
1	Age (Years)	36.3 ± 12.1	50.0 ± 9.3	0.13
2	BMI (kg/m^2)	40.6 ± 7.5	41.4 ± 8.1	0.66
3	Weight (Kg)	120.3 ± 23.2	104.5 ± 22.2	0.79
4	SBP (mmHg)	146.6 ± 18.3	143.4 ± 15.7	0.37
5	DBP (mmHg)	90.4 ± 8.4	87.4 ± 8.1	0.83
6	Waist (cms)	122.2 ± 11.1	113.5 ± 11.3	0.92
7	FBG (mmol/l)	6.7 ± 2.4	7.3 ± 2.6	0.65
8	TC (mmol/l)	5.0 ± 1.1	4.9 ± 0.7	0.01
9	TG (mmol/l)	1.9 ± 0.7	1.5 ± 0.7	0.99
10	HDL-C (mmol/l)	0.9 ± 0.3	1.2 ± 0.3	0.99
11	LDL-C (mmol/l)	3.1 ± 0.9	3.0 ± 0.8	0.49

Table 5. Comparison of post-values between males and females' subjects.

S. no	Characteristic features	Males	Females	P value

1	BMI (kg/m^2)	39.1 ± 7.1	39.8 ± 8.5	0.3
2	Weight (Kg)	115.7 ± 24.6	100.4 ± 22.8	0.65
3	SBP (mmHg)	131.1 ± 16.4	134.25 ± 14.3	0.42
4	DBP (mmHg)	79.4 ± 8.1	78.4 ± 9.1	0.5
5	Waist (cms)	118.6 ± 11.7	110.9 ± 11.5	0.91
6	FBG (mmol/l)	6.0 ± 1.9	7.4 ± 2.9	0.01
7	TC (mmol/l)	4.6 ± 1.0	4.7 ± 0.7	0.04
8	TG (mmol/l)	1.9 ± 0.8	1.4 ± 0.6	0.09
9	HDL-C (mmol/L)	0.9 ± 0.3	1.2 ± 0.3	0.99
10	LDL-C (mmol/l)	2.8 ± 0.9	2.8 ± 0.7	0.14

Discussion

The aim of the present study was to investigate the effects of LGI and FES diets on MetS components in Saudi patients. The post value results of our current study suggested the LGI and FES diets are recommendable for the MetS patients. GI is the impact of carbohydrate containing foods on blood glucose. According to the literature, the LGI diet is associated with a decreased risk of MetS, type 2 diabetes, cardiovascular disease, stroke, chronic kidney disease, uterine fibroids, and several cancers, including breast, colon, and pancreas, and prostate cancer. In this study, we found a substantial change in MetS measures in the LGI diet group from pre-treatment to post-treatment. The FES diet theory dictates that approximately equivalent calories, proteins, fats, and carbohydrates may be exchanged or substitute without significant alteration to nutritional balance. The changes in MetS measures in the FES group were moderate. We observed that the 3 groups had significantly different HTN, FBG, and TG values post-treatment based on ANOVA analysis. There were significant changes in the metabolic parameters HTN, TG, and FBG from pre- to post-treatment, whereas there were no significant changes in WC and HDL-C. There were substantial changes in subject BMIs in both the LGI and FES groups, but they were not statistically significant. This may have been due to the short program duration.

Changing dietary habit represent a major challenge for many people [15]. It has been showed that modifying dietary and physical activity habits through lifestyle interventions is an effective method of improving MetS components [16]. One potential yet controversial mechanism for the effect of GI on obesity is that HGI foods induce the secretion of insulin and suppression of glucagon release [17]. Insulin inhibits lipolysis and gluconeogenesis and stimulates lipogenesis and glycogenesis [18]. The consequent rapid decrease in blood glucose stimulates hunger [19], which may result in greater energy intake and ultimately, obesity. Moreover, the long-term effect of insulin on the suppression of fatty acid oxidation may induce insulin resistance [20], which may be a risk factor for obesity. However, HGI foods may simultaneously induce insulin secretion and the release of leptin, a food intake suppressor. Leptin function depends on the presence of insulin [21]. Therefore, there are two opposing theories about the role of the GI of foods in obesity. The strong relationship between MetS and dietary pattern, tobacco use, and physical inactivity has also been reported in the literature [22]. In our study, we did not collect information on tobacco use, as this was not the focus of our study.

MetS is a constellation of risk factors associated with increased risk of diabetes and cardiovascular disease. It is a growing health problem in developed and developing countries. In GCC states, the current prevalence is estimated to be 20.7-37.2% [ATPIII] or 29.6-36.2% [IDF] for men, and 32.1-42.7% [ATPIII] or 36.1-45.9% [IDF] for women. However, the prevalence rates for women in GCC countries are almost 10-15% higher than in most developed countries. The cause is not clear, but socio-economic status may influence MetS and its components by affecting environmental and social factors [3,23]. Several studies on MetS have been carried out in Saudi Arabia. The overarching conclusion is that weight reduction owing to diet and physical exercise is significantly associated with an increase in HDL-C and a decrease in TG [3,24-31]. In this study, we did observe a significant change in TG values but not HDL-C values, which may be due to the low sample size of our study. Similar intervention program studies have been carried out in different parts of the globe in MetS subjects [32-36]. Fappa et al. concluded from his studies that telephonic counselling was an effective way to implement behavioural counselling to improve lifestyle habits in patients with MetS [16]. Based on our study observations, we agree with the effectiveness of this strategy. The results of the Scicchitano et al. studies suggested the dyslipidaemia accelerates the atherosclerotic process and its morbid consequences; statins represent the evidence based treatment of choice for reducing low density lipoprotein cholesterol levels and decreasing cardiovascular events [37]. The mechanisms underlying such actions are not fully understood but may be related to reducing 7a-hydroxylase, increasing faecal excretion of cholesterol, decreasing 3-hydroxy-3-methylglutaryl-CoA reductase mRNA levels or reducing the secretion of very low-density lipoprotein. This contribution provides an overview of the mechanism of action of nutraceuticals and functional food ingredients on lipids and their role in the management of lipid disorders Ciccone et al. studies conclude the healthy diet can improve cardiovascular risk profile of individuals [38].

The strength of our current study was designing the study with two different diet pattern groups along and control subjects with equal number of subjects in all the 3 groups. The current limitations of our study was (i) low sample size (ii) short intervention programme (6 weeks only) (iii) excluded diet pattern details. Based on the results of our current study, we conclude that LGI and FES diet patterns are recommendable for MetS patients. However, future studies are recommended for implementing the nutritional awareness motivational counselling programs in MetS in our population.

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References

1. Kim YR, Hong SH. Association between the polymorphisms of the vascular endothelial growth factor gene and metabolic syndrome. Biomed Rep 2015; 3: 319-326.
2. Aljohani NJ. Metabolic syndrome: Risk factors among adults in Kingdom of Saudi Arabia. J Family Community Med 2014; 21: 170-175.
3. Al-Daghri NM, Alkharfy KM, Al-Attas OS, Khan N, Alfawaz HA, Alghanim SA, Al-Yousef MA, Al-Ajlan AS, Alokail MS. Gender dependent associations between socioeconomic status and metabolic syndrome: A cross-sectional study in the adult Saudi population. BMC Cardiovasc Disord 2014; 14: 51.
4. Mabry RM, Reeves MM, Eakin EG, Owen N. Gender differences in prevalence of the metabolic syndrome in Gulf Cooperation Council Countries: A systematic review. Diabet Med 2010; 27: 593-597.
5. Panagiotakos DB, Pitsavos C, Skoumas Y, Stefanadis C. The association between food patterns and the metabolic syndrome using principal components analysis: The ATTICA Study. J Am Diet Assoc 2007; 107: 979-987.
6. Riccardi G, Rivellese AA. Dietary treatment of the metabolic syndrome-the optimal diet. Br J Nutr 2000; 83: S143-S148.
7. Fletcher JA, Perfield II, JW TJ, Rector RS. The second meal effect and its influence on glycemia. J Nutr Disorders Ther 2012; 2: 2161-0509.
8. Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H. Glycemic index of foods: A physiological basis for carbohydrate exchange. Am J Clin Nutr 1981; 34: 362-366.
9. International Diabetes Federation. The IDF consensus worldwide definition of the metabolic syndrome. IDF Commun 2006.
10. Khan IA, Movva S, Shaik NA, Chava S, Jahan P. Investigation of Calpain 10 (rs2975760) gene polymorphism in Asian Indians with gestational diabetes mellitus. Meta Gene 2014; 2: 299-306.
11. Khan IA, Vattam KK, Jahan P, Mukkavali KK, Hasan Q, Rao P. Correlation between KCNQ1 and KCNJ11 gene polymorphisms and type 2 and post-transplant diabetes mellitus in the Asian Indian population. Genes Dis 2015; 2: 276-282.
12. Alharbi KK, Ali Khan I, Syed R, Alharbi FK, Mohammed AK. Association of JAZF1 and TSPAN8/LGR5 variants in relation to type 2 diabetes mellitus in a Saudi population. Diabetol Metab Syndr 2015; 7: 92.
13. Alharbi KK, Khan IA, Munshi A, Alharbi FK, Al-Sheikh Y, Alnbaheen MS. Association of the genetic variants of insulin receptor substrate 1 (IRS-1) with type 2 diabetes mellitus in a Saudi population. Endocrine 2014; 47: 472-477.
14. Alharbi KK, Khan IA, Al-Daghri NM, Munshi A, Sharma V. ABCA1 C69T gene polymorphism and risk of type 2 diabetes mellitus in a Saudi population. J Biosci 2013; 38: 893-897.
15. Leblanc V, Begin C, Hudon AM, Royer MM, Corneau L, Dodin S, Lemieux S. Gender differences in the long-term effects of a nutritional intervention program promoting the Mediterranean diet: changes in dietary intakes, eating behaviours, anthropometric and metabolic variables. Nutr J 2014; 13: 107.
16. Fappa E, Yannakoulia M, Ioannidou M, Skoumas Y, Pitsavos C, Stefanadis C. Telephone counselling intervention improves dietary habits and metabolic parameters of patients with the metabolic syndrome: A randomized controlled trial. Rev Diabet Stud 2012; 9: 36-45.
17. Wilcox G. Insulin and insulin resistance. Clin Biochem Rev 2005; 26: 19-39.
18. Barba G, Sieri S, Russo MD, Donatiello E, Formisano A. Glycaemic index and body fat distribution in children: The results of the ARCA project. Nutr Metab Cardiovasc Dis 2012; 22: 28-34.
19. Parillo M, Licenziati MR, Vacca M, De Marco D, Iannuzzi A. Metabolic changes after a hypocaloric, low-glycemic-index diet in obese children. J Endocrinol Invest 2012; 35: 629-633.
20. Buyken AE, Cheng G, Gunther AL, Liese AD, Remer T. Relation of dietary glycemic index, glycemic load, added sugar intake, or fiber intake to the development of body composition between ages 2 and 7 y. Am J Clin Nutr 2008; 88: 755-762.
21. Buyken AE, Trauner K, Gunther AL, Kroke A, Remer T. Breakfast glycemic index affects subsequent daily energy intake in free-living healthy children. Am J Clin Nutr 2007; 86: 980-987.
22. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome: The atherosclerosis risk in communities study. Circulation 2008; 117: 754-761.
23. Rajabi S, Mazloom Z, Zamani A, Tabatabaee HR. Effect of low glycemic index diet versus metformin on metabolic syndrome. Int J Endocrinol Metab 2015; 13: e23091.
24. Bahijri SM, Al Raddadi RM. The importance of local criteria in the diagnosis of metabolic syndrome in Saudi Arabia. Ther Adv Endocrinol Metab 2013; 4: 51-59.
25. Al-Zahrani MS, Alghamdi HS. Effect of periodontal treatment on serum C-reactive protein level in obese and normal weight women affected with chronic periodontitis. Saudi Med J 2012; 33: 309-314.
26. Al-Daghri NM. Extremely high prevalence of metabolic syndrome manifestations among Arab youth: A call for early intervention. Eur J Clin Invest 2010; 40: 1063-1066.
27. Barrimah IE, Mohaimeed AR, Midhat F, Al-Shobili HA. Prevalence of metabolic syndrome among qassim

university personnel in Saudi Arabia. Int J Health Sci 2009; 3: 133-142.

28. Al-Qahtani DA, Imtiaz ML, Saad OS, Hussein NM. A comparison of the prevalence of metabolic syndrome in saudi adult females using two definitions. Metab Syndr Relat Disord 2006; 4: 204-214.
29. Al-Nozha M, Al-Khadra A, Arafah MR, Al-Maatouq MA, Khalil MZ. Metabolic syndrome in Saudi Arabia. Saudi Med J 2005; 26: 1918-1925.
30. Al-Qahtani DA, Imtiaz ML. Prevalence of metabolic syndrome in Saudi adult soldiers. Saudi Med J 2005; 26: 1360-1366.
31. Akbar DH, Siddique AM, Ahmed MM. Prevalence of type-2 diabetes in patients with hepatitis C and B virus infection in Jeddah, Saudi Arabia. Med Princ Pract 2002; 11: 82-85.
32. Beckie TM, Beckstead JW, Groer MW. The influence of cardiac rehabilitation on inflammation and metabolic syndrome in women with coronary heart disease. J Cardiovasc Nurs 2010; 25: 52-60.
33. Miller ST, Marolen KN, Beech BM. Perceptions of physical activity and motivational interviewing among rural African-American women with type 2 diabetes. Womens Health Issues 2009; 20: 43-49.
34. Barakatun Nisak MY, Ruzita AT, Norimah AK, Gilbertson H, Nor Azmi K. Improvement of dietary quality with the aid of a low glycemic index diet in Asian patients with type 2 diabetes mellitus. J Am Coll Nutr 2010; 29: 161-170.
35. Davis JN, Kelly LA, Lane CJ, Ventura EE, Byrd-Williams CE, Alexandar KA, Azen SP, Chou CP, Spruijt-Metz D, Weigensberg MJ, Berhane K, Goran MI. Randomized control trial to improve adiposity and insulin resistance in overweight Latino adolescents. Obesity 2009; 17: 1542-1548.
36. Frost G, Wilding J, Beecham J. Dietary advice based on the glycaemic index improves dietary profile and metabolic control in type 2 diabetic patients. Diabet Med 1994; 11: 397-401.
37. Scicchitano P, Cameli M, Maiello M. Nutraceuticals and dyslipidaemia: Beyond the common therapeutics. J Function Foods 2014; 6: 11-32.
38. Ciccone MM, Scicchitano P, Cortese F, Gesualdo M. Endothelial function in obese and overweight patients: The role of olive oil, fish and nuts. Int J Diab Clin Res 2014; 1: 1.

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