International Journal of Home Science 2021; 7(3): 186-191

International Journal of Home Science

ISSN: 2395-7476
IJHS 2021; 7(3): 186-191

www.homesciencejournal.com
Received: 26-07-2021
Accepted: 29-08-2021

T Vasanthi
Ph.D. Scholar,
Department of Home Science,
Sri Padmavati Mahila Visvavidyalayam, Tirupati,
Andhra Pradesh, India

Dr. M Aruna
Professor, Head of the,
Department of Home Science,
Sri Padmavati Mahila Visvavidyalayam, Tirupati,
Andhra Pradesh, India

Corresponding Author:
T Vasanthi
Ph.D. Scholar,
Department of Home Science,
Sri Padmavati Mahila Visvavidyalayam, Tirupati,
Andhra Pradesh, India

Changes in central obesity/abdominal obesity in women with metabolic syndrome risk factors by changing their regular diet pattern

T Vasanthi and Dr. M Aruna

Abstract
In the context of globalization, westernized dietary pattern i.e. using more processed foods, ready to eat convenient foods (changed) has increased. At the same time Metabolic syndrome has become a major health issue. This study investigated the association between consumption of these processed foods/convenient foods on metabolic syndrome risk factors in women population. In this study, health survey was conducted among 300 women aged 20-70 yrs, who were visiting for periodic check up in a diagnostic centre. Dietary pattern, family history of disease, socio economic status were derived by validated self-administered questionnaire. From this, 160 interested participants were selected for 6 weeks nutrition intervention program after signing concern form. IDF definition of MS was used for screening of the subjects. Anthropometric measurements such as weight, height, BMI, waist circumference, Tummy measurements and visceral fat analyzed at base line and after 6 weeks of nutritional intervention and results were analyzed using appropriate statistical measures. Participants' dietary pattern was analyzed for processed foods/Ready to eat and convenient foods. We observed a significant weighty loss (74.668±10.086) after 6 week intervention compared with base line values (76.786±10.194) (t=12.602,P <0.05) in experimental groups than control gps (68.867±7.928@ baseline; 68.582±7.897 @6wk intervention). A significant reduction in BMI, V. Fat, WC, Tummy measurement & Hip measurement were also observed. Central obesity in MS is directly associated with increased intake of processed food/Ready to eat and convenient foods. Further studies with larger sample sizes and of longer duration are needed to examine the role of these foods in the prevention and management metabolic syndrome.

Keywords: Metabolic syndrome, central obesity, BMI, waist circumference, processed food

Introduction
The metabolic syndrome (MS) is a complex disorder. According the recent research studies [1-8]. It is identified with major component of central obesity, hypertriglyceridemia and low high density lipoprotein cholesterol concentration, elevated blood pressure and elevated plasma glucose levels. They are associated with insulin resistance, pro-inflammatory state & decreased level of cardio-respectively fitness. Sedentary life style and obesity are (strongly) major contributing factors for developing metabolic syndrome. This represents a growing public health problem [9]. General Raven described this as syndrome X [10]. And proposed that because of insulin resistance, glucose imbalance and several metabolic abnormalities happening. This concept was named as Metabolic Syndrome by World Health Organization (WHO). In 1998, WHO defined Metabolic syndrome has person having diabetes, impaired glucose tolerance, impaired fasting glucose or Insulin resistance, plus 2 or more of the following abnormalities.
Blood pressure ≥160/90mm of Hg, Triglycerides ≥150mgldl HDL cholesterol ≤ 35 mg/dl (Male) ≤39 mg/dl in female. Waist and Hip ratio ≥0.90 male, ≥ 0.85 female & BMI ≥30 kg/m², urinary albumin excretion rate ≥20µg/min or Alumina/Creatinine ratio ≥ 20. There is no clinically applicable test for insulin resistance. Hyper insulinemic clamp test for insulin resistance is complex and expensive. Considering this, National Cholesterol Education program expert panel (III) (in 2001) and International Diabetic Federation (2005) proposed new definition for metabolic syndrome based on anthropometric measurements. International Diabetic Federation suggested a sample screening tool for metabolic syndrome, that is a

person must have central obesity (Waist Circumference) ≥ 94 cm for Europid men & ≥80 cm for Europid women (with ethnicity specific values for other groups). Plus any two of the following four factors:
Triglycerides ≥ 150mg/dl or under treatment for lipid abnormality. HDL cholesterol < 40mg/dl in males and <50mg/dl in females. Under treatment for lipid abnormality. Blood pressure systolic ≥ 130mmof Hg or Diastolic ≥ 85mmof Hg or treatment for hypertension, Fasting plasma glucose ≥100mg/dl or diagnosed with Type 2 diabetes. For Indians IDF proposed central obesity to be measured as waist circumference ≥ 90cm for men, ≥ 80cm for women. According to several research reports [14-17] for any given BMI Indians tend to have increased waist circumference and also have excess body fat and abdominal/central obesity. WHO has revised the BMI cut off for Asian Indians to be 25 kg/m² for obese instead of 30kg/m² for recommended [18].
Metabolic syndrome is a multi-factorial disorder associated with genetic factor and dietary factors. Research evidence it is clear that dietary pattern characterized by high intakes of processed food, meat, refined grains and fried foods were associated with increased incidence of Metabolic Syndrome risk factors.

Research methodology
A total of 300 women aged between 20-70 years were randomly interviewed through a questionnaire (Prepared for dietary pattern analysis after pilot study) depending on IDF definition for Mets i.e., abdominal obesity or central obesity as a tool for this study who were attending to diagnostic centre for health checkup. Out of this 160 women who were willing to participate in 6 week nutrition intervention program were enrolled after giving their written concern to participate in the study. These women were divided in to 4 groups according to their medical history. One of the experimental group consists of subjects who were already diagnosed has diabetic or hypertensive or Hypothyroid or on medication for health issues. The other experimental group consists of subjects, who were not using any kind of medication or not diagnosed with any health issues. And respective control groups (E-No Medication Group, E-2 Medication Group).

Study design
All the participants were recruited to attend the program for 6 weeks. Anthropometric data and dietary pattern were assessed at base line and after 6 weeks.
Dietary counseling was given to all the Experimental participants at base line to discuss about the dietary intervention for both the experimental groups. The dietary intervention consisted of discussion about Metabolic Syndrome, Health hazards of processed/ready to eat foods and health benefits of whole food groups. Participants were visiting twice a week for diet counseling and their diet recalls/Diet diaries were analyzed. Participants were encouraged to increase the whole food groups instead of processed or convenient foods in take.

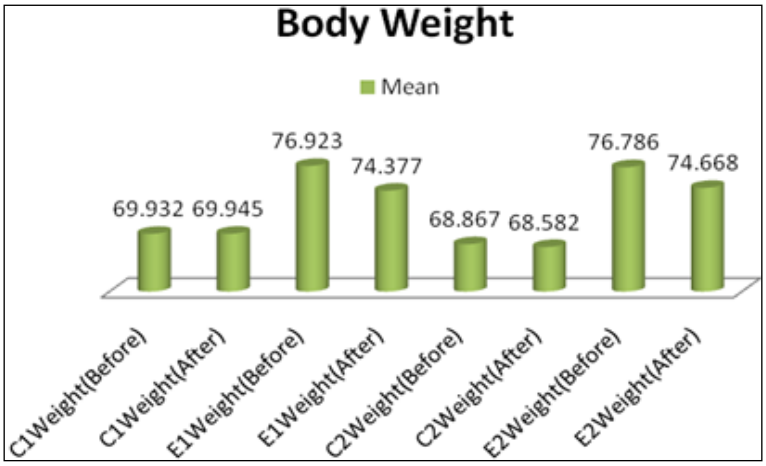
Anthropometric measures
Weight was measured in light clothes and not wearing shoes to the nearest of 100g. Height was measured by using tape while standing not wearing shoes and had the shoulders in normal position. Body mass Index (BMI) was calculated as weight (in kg) divided by height (in Mt²). Waist circumference (WC) was measured at the iliac crest. Hip circumference was measured at the maximum level, by using an un stretchable tape. Tummy measurements were measured at novel point. Visceral fat was assessed by a bio electrical impedance device (OMRON).

Statistical analysis: Descriptive statistics were performed for the study ad continuous variables are presented as means ± standard deviation. Variables were compared by T-Test (Paired). The results were discussed based on a significance level of 5%.

Results & Discussion
Participants in the both the experimental groups had significant reduction in body weight, BMI, visceral fat, waist circumference (WC), Tummy measurements & Hip measurements at 5% level. Over the 6 week of intervention period.

Table 1: Mean Comparison of Body Weight of the Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

S. No.	Weight(Wt)	Group	Mean	Std.	Std. err	Mean difference	T-Values	Sig (2 tailed)
1.	Weight (Before)	Control Gp1	69.932	13.273	2.384	0.0129±0.1231	-0.583	0.564
2.	Weight (After)	N= 31	69.945	13.253	2.38			
3.	Weight (Before)	Experimental Gp1	76.923	10.371	1.753	2.5457±0.736	20.469	0.000
4.	Weight (After)	N=35	74.377	10.209	1.726			
5.	Weight (Before)	Control Gp2	68.867	7.928	1.38	0.285±0.381	4.296	0.000
6.	Weight (After)	N=33	68.582	7.897	1.375			
7.	Weight (Before)	Experimental Gp2	76.786	10.194	1.676	2.1189±1.023	12.602	0.000
8.	Weight (After)	N=37	74.668	10.086	1.658			

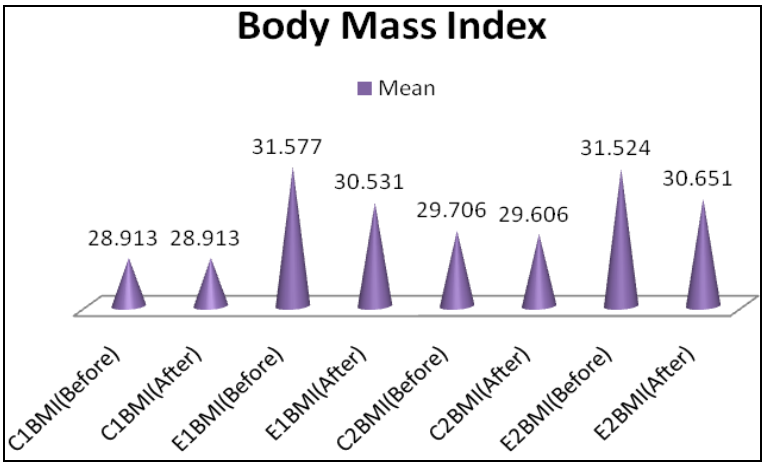


Graph 1: Mean Comparison of Body Weight of Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

From the table: 1 and graph:1, Experimental group 1 significantly reduced more weight than experimental group 2, at 5% level while no significant change was seen in control groups. (E 1-2.5457±0.784, E2-2.1189±1.023).

Table 2 Mean Comparison of Body Mass Index of the Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

S. No.	Body Mass Index(BMI)	Group	Mean	Std.	Std. err	Mean Difference	T-Values	Sig (2 tailed)
1.	BMI (Before)	Control Gp1	28.913	4.545	0.816	NA	NA	NA
2.	BMI (After)	N= 31	28.913	4.545	0.816			
3.	BMI (Before)	Experimental Gp1	31.577	2.9185	0.4933	1.0457±0.3311	18.686	0.000
4.	BMI (After)	N=35	30.531	2.9112	0.4921			
5.	BMI (Before)	Control Gp2	29.706	3.082	0.536	0.1±0.1837	3.127	0.004
6.	BMI (After)	N=33	29.606	3.0364	0.529			
7.	BMI (Before)	Experimental Gp2	31.524	3.2262	0.5304	0.873±0.4426	11.997	0.000
8.	BMI (After)	N=37	30.651	3.2085	0.5275			

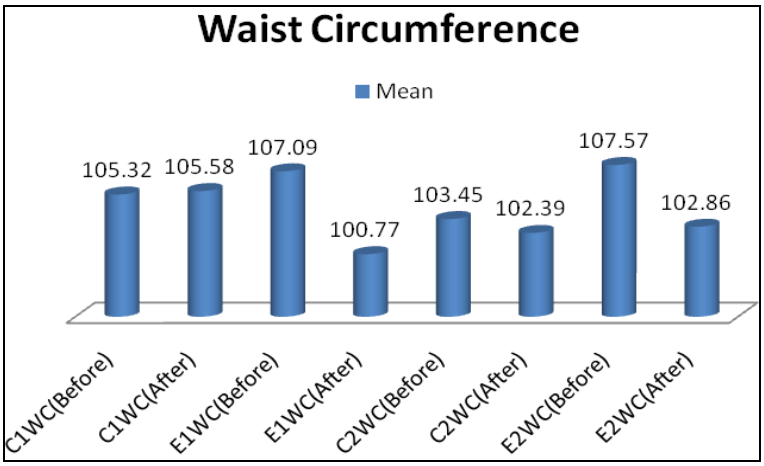


Graph 2: Mean Comparison of Body Mass Index of Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

The reduction in BMI (Table: 2) change in the experimental groups was observed from baseline to post-intervention. The experimental group1 showed more reduction in BMI (1.0457±0.331) than the experimental group 2 (0.873±0.441) while no significant change was seen in control groups.

Table 3: Mean Comparison of Waist Circumference of the Sample according to Paired T-Test After 6weeks of Nutritional Intervention

S. No.	Waist Circumference(WC)	Group	Mean	Std.	Std. Err	Mean difference	T-Values	Sig(2 tailed)
1.	WC (Before)	Control Gp1	105.32	8.871	1.593	0.258±0.815	-1.763	0.088
2.	WC (After)	N= 31	105.58	8.902	1.599			
3.	WC (Before)	Experimental Gp1	107.09	6.926	1.171	6.314±1.795	20.812	0.000
4.	WC (After)	N=35	100.77	6.933	1.172			
5.	WC (Before)	Control Gp2	103.45	6.746	1.174	1.061±1.619	3.763	0.001
6.	WC (After)	N=33	102.39	6.046	1.053			
7.	WC (Before)	Experimental Gp2	107.57	6.449	1.06	4.703±2.197	13.022	0.000
8.	WC (After)	N=37	102.86	6.378	1.048			

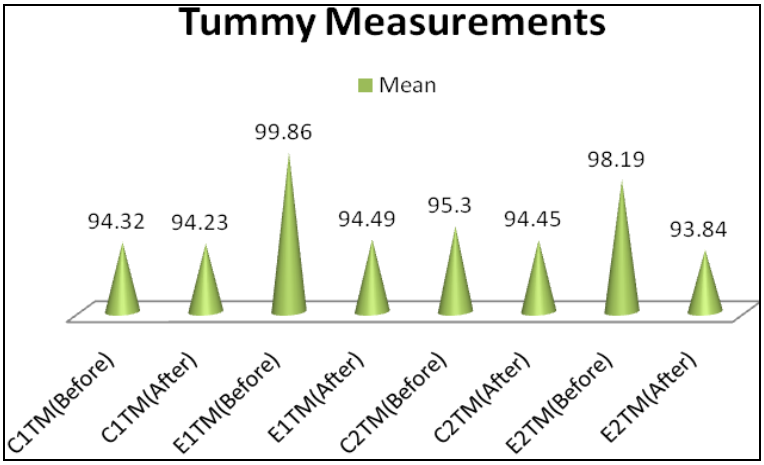


Graph 3: Mean Comparison of Waist Circumference (Wc) of Sample according to Paired T-Test After 6weeks of Nutritional Intervention

The reduction in waist circumference (Table 3 and graph: 3) was observed in the groups from baseline to post intervention period. The experimental group 1 showed more significant reduction in waist circumference (6.314±1.795) than the experimental group 2 (4.703±2.197). While the change in control groups was not significant at 5% level.

Table 4: Mean Comparison of Tummy Measurements of the Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

S. No.	Tummy Measurement	Group	Mean	Std.	Std. err	Mean difference	T-Values	Sig (2 tailed)
1.	TM (Before)	Control Gp1	94.32	8.324	1.495	0.097±0.651	0.828	0.414
2.	TM (After)	N= 31	94.23	8.139	1.462			
3.	TM (Before)	Experimental Gp1	99.86	6.643	1.123	5.371±1.911	16.628	0.000
4.	TM (After)	N=35	94.49	6.577	1.112			
5.	TM (Before)	Control Gp2	95.3	6.237	1.086	0.848±1.395	3.495	0.001
6.	TM (After)	N=33	94.45	5.663	0.986			
7.	TM (Before)	Experimental Gp2	98.19	6.48	1.065	4.351±2.898	9.132	0.000
8.	TM (After)	N=37	93.84	7.143	1.174			

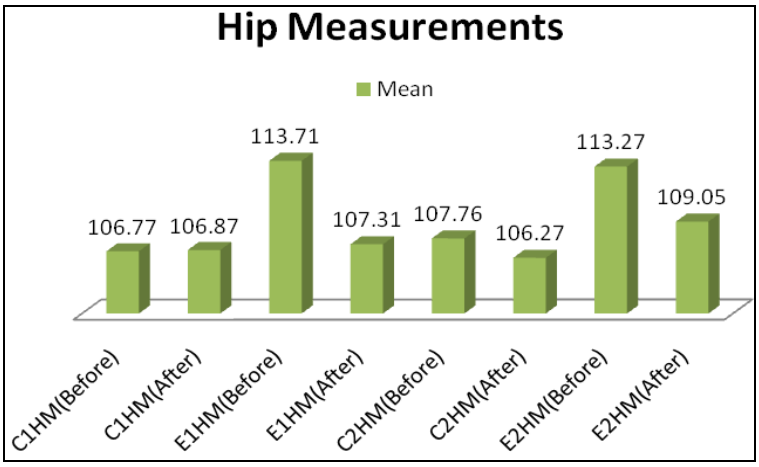


Graph 4: Mean Comparison of Tummy Measurements of Sample according to Paired T-Test Before and after 6 weeks of Nutritional Intervention

The reduction in Tummy measurements (Table 4 and graph: 4) was observed in all the groups from baseline to post intervention period. The experimental group 1 showed more significant reduction in tummy measurements (5.371±1.91) than the experimental group 2 (4.351±2.898). There was no significant change in both control groups.

Table 5: Mean Comparison of Hip Measurements of the Sample according to Paired T-Test Before and after 6 weeks of Nutritional Intervention

S. No.	Hip Measurement	Group	Mean	Std.	Std. Err	Mean difference	T-Values	Sig(2 tailed)
1	HM(Before)	Control Gp1	106.77	8.667	1.557	0.097±0.908	-0.594	0.557
2	HM(After)	N= 31	106.87	8.535	1.533			
3	HM(Before)	Experimental Gp1	113.71	7.148	1.208	6.400±2.061	18.373	0.000
4	HM(After)	N=35	107.31	6.574	1.111			
5	HM(Before)	Control Gp2	107.76	6.759	1.177	1.485±1.787	4.772	0.000
6	HM(After)	N=33	106.27	6.54	1.138			
7	HM(Before)	Experimental Gp2	113.27	7.026	1.155	4.216±2.175	11.792	0.000
8	HM(After)	N=37	109.05	7.416	1.219			



Graph 5: Mean Comparison of Hip Measurements of Sample according to Paired T-Test Before and after 6weeks of Nutritional Intervention

The reduction in HIP measurements (Table: 5) was observed in both the experimental groups from baseline to post intervention program. The experimental group 1 showed more significant change in HIP measurements (6.400±2.061) than the experimental group 2 (4.216±2.175). There was no significant change in control groups.

Overall, the experimental group 1 showed significant reduction in body weight, BMI, Visceral fat, waist circumference, Tummy measurements and hip measurements than the experimental group 2. From baseline to post-intervention.

This study demonstrated improvements in body composition and metabolic indicators following a 6 week nutrition intervention program. This study aimed to provide all the participants information and guide lines to understand the link between diet pattern metabolic syndrome risk factors.

Eliminating all the processed/Convenient foods refined foods and eating whole grain food for 6 weeks, resulted in decreasing body weight, BMI, VF, WC & HIP measurements. High values of waist line and body mass index are main parameters and the most important indicators for screening & diagnosing the Mets [24]. A number of studies in recent years have found that dietary pattern is associated with insulin resistance, which is an important risk factor Mets [25]. More ever most of the whole grain foods dietary pattern have a low glycemic load, which will reduce the risk of insulin resistance [26]. The western dietary pattern (Process foods, fast foods, refined flours, snacks and drinks) have a high glycaemic index, which has been associated with an increased risk of Mets [27-30]. Based on this research studies, we found similar results in the present study that the healthy dietary pattern (whole grain) is associated with reduced risk factors than western diet pattern or processed/convenient food pattern [31-34].

Conclusion

The current findings indicate that Changes in dietary pattern associated with reduction in central obesity and overall weight in women with Metabolic Syndrome risk factors. Nevertheless, additional prospective studies are needed to confirm the associations between dietary pattern and Metabolic Syndrome.

Limitations

There are clear limitations in this study. The main limitation is small sample and small period of time and the other most important limitation is Diet Diaries, over weight individuals are more likely under report their energy intake. Because of this current estimates of dietary components could be under estimated and they play a major role in metabolic risk factors. Further research is needed, has this study was done as a part of Ph.D. study with limited funds and period.

References

1. Reaven G. Characteristic of Metabolic Syndrome Endocrinol Metab 1995;2(B):37-42.
2. National Cholesterol Education program: Third report of the National Cholesterol Education program (NCEP) expert panel on detection, Evaluation and Treatment of high blood cholesterol in adults (Adults treatment panel III) final report circulation 2002;106:3143-3421.
3. Devraj S, Roseeson Rs. Jilal1: Metabolic Syndrome an appraisal of the pro inflammatory and pro coagulant status. Endocrinal Metab Clin. North Am 2004;33:431-453.
4. Stewart KJ, Bacher AC, Turner K, Lim JG, Hees PS, Shapiro EP, Tay back M, Onyang P, Exercise & Risk factors associated with metabolic syndrome in older adults. Am J prev. Med 2005, 28: 9-18.
5. Hansel B,Giral P, Nobecount E, Chant pie S, Bruckert E, Chapman MJ, Kontush A: Metabolic syndrome is associated with elevated oxidative stress and dysfunctional dense high density lipo protein particles displaying impaired antioxidative activity. J clin endocrinal Metab 2004;89:4963-71.
6. Saltin B, Helge JW. Metabolic capacity of skeletal muscles and health. Ugeskar larger 2000;162:2159-64.
7. Mother DE, Kanfman KD. Meatbolic syndrome: A clinical and molecular prospective. Annu. Rev Med 2005;56:45-62.
8. Findey CE, La Monte MJ, Waslien El, Barlow CE, Blair SN, Nichaman MZ. Cardio respiratory fitness Macro nutrient intake, and the Metabolic Syndrome: The Aerobics centre longitudinal study. J Am med Assoc 2006; 106:673-97.
9. Grundey SM, Cleeman J1, 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;112:2735-52.
10. Reaven GM. Role of Indian Resistance in Human disease diabetes 1988:37:1595-1609.
11. World Health Organization definition, diagnosis and classification of Diabetes mellitus Geneva: WHO department of non-communicable Disease surveillance 1999.

12. Executive summary of the 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) J Am. Med. Assoc 2001;285:2486-2497.
13. International Diabetes Federation New IDF worldwide definition of the Metabolic Syndrome. Press conference 1st International congress on "Pre-Diabetes" and the Metabolic Syndrome. Berlin, Germany 2005. (www.idf.org).
14. Mckeigue PM, Shah B, Marmott MG. Relationship of contra Obesity and insulin resistance with high diabetes prevalence and cardio vascular risk in south Asians. Lancet 1991;337:382-386.
15. Chandalia M, Abate N, Garg A, Stray Gundersen J, Gundy SM. Relationship between generalized and upper body obesity to insulin resistance in Asian Indian Men. J Clin Endocrinal Metab 1999;84:2329-2335.
16. Enas EA, Yusuf S, Mehta JL. Prevalence of artery disease in Asian Indians. Am J Cardiol 1992;70:945-949.
17. Banerji MA, Faridi N, Atluri R, Chaiken RL, Lebovitz HE. Body composition, visceral fat, Leption and Insulin resistance in Asian Indian Men. J Clin Endocrinal Metab 1999;84:137-144.
18. World Health Organization WHO recommendations Obesity: Preventing and meaning the Global Epidemic. Geneva, World Health org 2000.
19. Ambrosini GL, Huang RC, Mori TA *et al*. Dietary Patterns and markers for the Metabolic Syndrome in Australian adolescents Nutr Metab cardio Vasc Dis 2010;20:274-283.
20. He Y, Li Y, Lai J *et al*. Dietary patterns as compared with physical activity in relation to Metabolic Syndrome among Chinese adults Nutr Metab Cardio Vasc Dis 2013;23:920-928.
21. Esmaillzadh A *et al*. Dietary patterns insulin resistance and prevalence of the Metabolic Syndrome in women. Am J Clin Nutn 2007;85(3):910-918.
22. Denora-Gutierrez E, Castanon S *et al*. dietary patterns are associated with metabolic syndrome in an urban Mexican population. J Nutr 2010;140(10):1855-1863.
23. Lutsey PL, Steffen LM, Stevens J. Dietary intake and the development of the metabolic syndrome; The Atherosclerosis Risk in communities study. Circulation 2008;117(6):754-761.
24. Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS *et al*. Prevalence of Obesity, diabetes and obesity related health risk factors, JAMA 2001, 2003;289(1):76-79.
25. Musso G, Gambino R, De Michichi F *et al*. Dietary habits and their relation to insulin resistance and post prandial lipemia in non-alcoholic steato hepatitis. Hepatology 2003; 3:909-916.
26. McKeown NM, Meigs JB, Lin S *et al*. Carbohydrates nutrition, insulin resistance and the prevalence of the metabolic syndrome in the Framingham off spring cohort. Diabetes care 2004;27:538-546.
27. Hosseini-Esfahani F, Mirmiran P, Danesh Power MS *et al*. Western dietary pattern interaction with APOC3 polymorphism in the risk of Metabolic Syndrome Tehran Lipid and Glucose study. J Nutrient Nutrigenomics 2014;7:105-117.
28. Junola-Falgarona M, Salas-salvado J, Bnil-Cosiales P *et al*. Dietary glycemic index and glycemic load are positively associated with risk of developing metabolic syndrome in middle-aged and elderly adults. J Am Geriatr Soc 2015;63:1991-2000.
29. Fung TT, Rimm EB, Spiegel D *et al*. Association between dietary patterns and plasma bio markers of obesity and cardio vascular disease risk. Am J Clin Nutr 2001;73:61-7.
30. Villegas R, Salin A, Ilynn A, Perry IJ. Prudent diet and the risk of insulin resistance Nutr. Metab. Cardio Vasc. Dis 2004;14:334-43.
31. Esmaillazadeh A, Mirmiran P, Azizi F. Whole grain consumption and the metabolic syndrome: A favorable association in Tehranian adults. Eur J clin Nutr 2005;59:353-362.
32. Azadbakht L, Mirimram P *et al*. Beneficial effects of a dietary approaches to stop hypertension eating plan on features of the metabolic syndrome. Diabetes care 2005;28:2823-31.
33. Frerie RD, Cardoso MA, Ginemo SG *et al*. Japanes-Brazilian diabetes study group. Dietary fat is associated with metabolic syndrome in Japanese Brazilians. Diabetes care 2005, 2008, 1779-85.
34. Esmaillzadeh A, Mirmiram P, Azizi F. Comparative evaluation of anthropometric measures to predict cardio vascular risk factors in Tehranian adults women. Public Health Nutr 2006;9:61-69.