Research Paper Medical Science

# Systematic Review on Dietary Intake and Metabolic Syndrome in Children and Adolescents


| | |
|---|---|
| **Mojgan Nourian** | Ph.D student in community nutrition, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia (UPM), Malaysia & Department of Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, Iran |
| **Zaitun Yassin** | Department of Nutrition, School of Nutrition and Food Science, Isfahan University of Medical Sciences, Isfahan, Iran |
| **Mohd Nasir Mohd Taib** | Department of Nutrition and Dietetics, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Malaysia |
| **Fahimeh Haghighatdoost** | Food Security Research Center, Isfahan University of Medical Sciences, Isfahan, Iran |
| *** Roya Kelishadi** | Department of Pediatrics , Child Growth and Development Research Center and Faculty of Medicine, Isfahan University of Medical Sciences, Isfahan, Iran * Corresponding author |


**ABSTRACT**

Metabolic syndrome(MetS) has been broadly studied in adults. However,because of the lack of auniversal definition for MetS in pediatric populations, few studies have been conducted on this frequent disorder among children.Lifestyle factors play an important role in the pathogenesis of MetS. A cornerstone of the treatment in MetS patients is the use of a healthy diet to improve all abnormalities. Most of available studies focused on lifestyle changes for treatment of MetS in adolescents, and there are only few studies which assessed the specific role of diet therapy for MetS among children and adolescents. It is important since sometimes it is not applicable to encourage children to be more active. Hence, in the present article, we aimed to review the dietary intake and recommendations for treatment of pediatric MetS.

**KEYWORDS**

**Background**

In 1988, Reaven noticed that several risk factors commonly come together including obesity, high fasting triglycerides (TG), low levels of high-density lipoprotein cholesterol (HDL-C), high levels of low-density lipoprotein (LDL-C), increased fasting serum insulin, impaired glucose tolerance (IGT) and high blood pressure which seemed to be related to each other as metabolic syndrome (MetS) (1, 2). Several terms have been suggested to describe MetS such as "syndrome X" (3), "death quartet" (4) and "insulin resistance syndrome" (5). MetS is a constellation of interrelated cardiovascular disease (CVD) risks factors such as abdominal obesity, dyslipedemia, hyperglycemia, insulin resistance and hypertension (HTN)(6-8). Also, MetS is associated with higher risk for type II diabetes mellitus (T2DM) (9). Abdominal obesity and insulin resistance are the most important determinants of MetS (6, 10).

MetSis a common disorder among adults in both developed and developing countries in the last two decades (11-14). In adults, the MetS components have been developed by a number of expert groups which varies in the sense of their indicators and cut points(10, 15- 20).

MetS has been broadly studied in adults during the past two decades. However, because of some barriers,there is no universal definition for MetS in the pediatric populations and few studies have been conducted in this age group(21, 22). Some limitations to define pediatric-MetS are due to lack of information regarding the normal range of serum insulin level in children, the use of adults cut-point for children or not sorting cut-points by age groups for children, physiological insulin resistance in puberty, lack of clear cut points for central adiposity which could predict the risk of co-morbiditiesandMetS in youths and varied baseline levels for lipid profile in specific ethnicities.Given the current epidemic of obesity, it is not surprising that the so-called 'pediatric MetS' is still a comparatively little-heard-of term (23).

Therefore, because of the lack of a 'gold standard' diagnostic test, researchers have modified the adult criteria in different ways to create their own definition for children and adolescents (Table 1)(24- 30). The International Diabetes Federation(IDF) introduced a clear set of criteria to diagnose metabolic syndrome in children and adolescents (31). This single worldwide definition enables researchers to compare data from different studies, easily. The IDF criteria for MetS in children and adolescents have been shown in Table 2.

However, previous studies used different diagnosticcriteria for the MetS in youths and reported different prevalence rates of MetS in children by different definition in the same population. For example,using NCEP ATP III and IDF criteria, a cross-sectional study of 923 Italian obese children showed a 36.2% and 56.7% prevalence of MetS, respectively (32), whereas a study of Chinese children and adolescents found a higher prevalence of MetS by NCEP ATP III versus IDF (38.2% vs. 33.9%, respectively) (33). Another study among 99 Australian overweight pre-pubertal girls compared the prevalence of MetS using six different definitions. The findings of this study showed the prevalence rates of 0% by US NCEP to 60%

by Lambert criteria (34). Using two different definition, Saffari et al. determined the prevalence rates of 50% and 66.2% versus 25% and 42.5% among overweight and obese, respectively, in a single data set of Iranian children and adolescents (35). Overall, a recent systematic review on eighty-five study reported the prevalence of MetS in children. The median (and range) of MetS in whole populations, overweight and obese children were 3.3% (0-19.2%), 11.9% (2.8-29.3%) and 29.2 (10-66%), respectively (36).Friend et al. (36) found higher prevalence rate among boys rather than girls (5.1% vs. 3.0%) and also in older compared with younger children (5.6% vs. 2.9%). However, the prevalence of MetS varies widely around the world. A summary of the prevalence of MetSby different criteria in adolescents is shown in Table 3.

The underlying causes of the MetS remain to be determined, but obesity is regarded as an important underlying factor. Genetics, sedentary lifestyle, aging, pro-inflammatory states low birth weight (37) and hormone-related changes may also cause MetS(38, 39).

Obesity is linked to several metabolic abnormalities, specially insulin resistance andMetS. There is a close relation between the degree of obesity and MetS, while weiss et al (40) found that 38.7% and 49.7% of moderate and severe obese children were affected by MetS. The process of atherosclerosis,which begins at early age, is associated with obesity and other components of metabolic syndrome in children (41). These abnormalities are tracked from childhood to adulthood(42, 43).

Diabetes and heart disease are now increasing in children and the adolescent population. This is particularly of great concern because next generation will already have several health problems due to their childhood obesity. Therefore, obesity in these individuals means higher risk factor for their morbidity and mortality in their future life.

Abdominal fat measured by waist circumference is more symptomatic of MetSprofile than BMI (44). In recent years, abdominal obesity in both genders of adolescents had considerable increase at global level(45, 46). An excess of adipose tissues (especially visceral fat tissues of the abdomen) releases inflammatory cytokines which can contribute to a rise in insulin resistance (47). Moreover, central obesity is linked to a lower level of adiponectin, the adipose-specific, collagen-like molecule found to have antidiabetic, anti-atherosclerotic and anti-inflammatory properties (48).

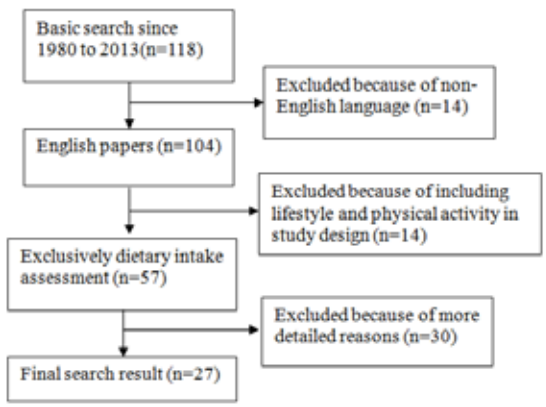
Insulin resistance is associated with wearing out of the beta cell of pancreas,hyperglycaemiaandtype 2 diabetes (49). Insulin resistance in adipose tissue leads to a flux of free fatty acids from the adipose tissues to liver and causes insulin resistance in it and some tissues. Fatty acids cause to stop glucose oxidation and glucose transport. They also cause atherogenic dyslipidemia by inducing very low-density lipoprotein production in the liver that leads to an increase in serum TG. A rise in TG, accompanied with high LDL-C levels, markedly increases the risk for CVDs (50).

In addition to metabolic abnormalities, lifestyle factors such as diet play an important role in the pathogenesis of MetS. Hence, a cornerstone of the treatment in MetS patients is the use of a healthy diet to improve all abnormalities including excess body weight, dyslipidemia, hyperglycemia, hyperinsulinemia and hypertension. In this article, we review some dietary recommendation for pediatric MetS.

**Methods**

In order to searchrelevant English and non-English published papers, we used online databases ofPubMed, ISI Web of Science, SCOPUS, Science Direct and EMBASE from January 1980 to February 2013. Following keywords were used in our search: Metabolic syndrome, syndrome X, insulin resistance syndrome, diet, adolescents, adolescence, childrenand their related Medical Subject Headings (MeSH terms). We found 103 English papers which published between 1980 and the February of 2013. Finally, we reached 57 papers which did not include lifestyle factors and physical activity in their study design. Around 30 papers of 57 papers were limited to the components of MetS, specially obesity and insulin resistance, or assessed the effect of adolescent dietary intake on MetS in early young, or studied the effect of maternal diet on MetS of children. Few studies (n=27) assessed the role of diet in MetS specifically. We included these papers as the main search results in the present paper.

**Figure 1:The schematic process of searching related articles.**




**Dietary treatment of Metabolic syndrome**

Diet as a modifiable risk factor plays an important role in the prevention of MetSconsequences (51) by affecting on MetS-components such as blood glucose and lipid profile. Because of the strong relationship between obesity and MetS, it is reasonable to suggest that primary aim in the treatment of MetS should focus on weight loss.The most prevalent form of the MetSin obese patients is excess visceral adiposity (52, 53). Dietary patterns by increasing dietary fiber consumption and decreasing fat consumption were associated with Insulin Resistant Syndrome (IRS) inversely among obese people and could reduce the risk of type 2 diabetes and CVD (54). Results of these studies implied that calorie content and diet composition are important factors in the prevention and treatment of metabolic syndrome in young people.

**Dietary pattern**

To resolve the concern regarding the interactions among nutrients or unknown components in foods and the colinearity among food and nutrient intakes, studying dietary patterns has been suggested instead of studying single nutrients or foods or food groups (55-58). In addition, dietary pattern could reflect individuals' dietary behaviors which may affect the etiology of chronic disease (56, 57).

Due to continued changes in adolescents' dietary patterns and their long term effects in adulthood, assessing dietary patterns among them is important. However, few studies were conducted to assess such association among adolescents and most of these studies were limited to adults or specific components of MetS. One study of 14-y old Australian adolescents found that western dietary patterns is positively associated with increased odds ratio for the "high risk metabolic cluster" and also higher mean values of total cholesterol, waist circumference and BMI among girls while healthy dietary pattern was negatively associated with serum glucose in both gender and low HDL-C in boys (59). A systematic review on 3,168 Korean adolescents (13-18 yr)between 1998-2009 showed a direct association between western dietary pattern and MetSas well as its components such as hypertriglyceridemia and obesity, whilst traditional dietary pattern had a negative association (60).Furthermore, the number of 16-18 year-old adolescents who had western dietary pattern

increased from1998 to 2009 but no changes observed in the dietary pattern of 13-15 year old ones (60). Other studies assessed the association between various dietary patterns and the risk factors of MetS. A cross-sectional study using 1995 Australian National Nutrition Survey of participants aged 12-18 y showed that after adjustment for confounding factors, a fruit, salad, cereals and fish dietary pattern is inversely associated with high diastolic blood pressure in 16-18 year-old adolescents (61). Positive association between energy-dense, high-fat, low-fiber dietary patterns and fat mass index was reported by Ambrosini et al. (62). However, several studies have linked western, sweetened cereal and corn dishes, whole milk and sweet patterns to general and abdominal obesity and insulin resistance in different populations (63-65). Briefly, it seems that dietary patterns which characterized by high content of sugary cereals, sweetened beverages, industrial snack, cakes, whole milk, sweets, fried foods, burgers, and pizza and low intake of fruits, vegetable, dairy, whole grains without added fats, mixed dishes and soupswere related to higher risk of different metabolic abnormalities (63, 66). Another noteworthy aspect of diet which is closely related to the whole diet is its quality. Although various diet quality indices such as healthy eating index (HEI) were suggested to reduce the burden of chronic diseases, few studies assessed the association between diet quality indices and MetS in adolescents. However, one study showed that higher HEI is inversely associated with the MetS prevalence among US adolescents (67).

**Calorie intake and obesity in youths**

Since obesity is the most important risk factors in the prevalence of metabolic syndrome, dietary interventions should be focused on weight reduction in obese people with metabolic syndrome. It is clear that obesity is the outcome of imbalance between energy intake and energy expenditure, therefore the first step in treating of obesity is by reducing the energy intake (67- 70). Growth and development are two parameters which should be considered in weight reduction in adolescents. A substantial slowing of weight gain may be achieved by relatively small but consistent changes in energy intake, expenditure, or both (200 to 500 kcal per day) (71, 72).

The first goal of weight control in obese children and adolescents is weight maintenance because the normal growth and development with concurrent reductions in weight is a challenge in the treatment of overweight children. Professional recommendations to help the obese children and adolescents to achieve the healthy weight are consuming diets that include primarily nutrient-dense foods and participating in regular moderate to vigorous physical activity most days of the week (73). If weight loss is desired, an appropriate starting goal is about 0.5 kg of weight loss per month (74). Satisfactory weight control could also determine by improving hyperlipidemia, hyperinsulinemia and hypertension (75).

Reduced calorie diet such as very low calorie diet (VLCD) used in weight management in adults could not be applied in children and adolescents because the normal growth and development in these age groups are important and reduced calorie diets may threaten the normal growth. Furthermore, the low-carbohydrate diet was associated with a greater improvement in some risk factors for CHD but the adherence is poor and longer and larger studies are required to determine the long-term safety and efficacy of low-carbohydrate, high-protein, high-fat diets in obese people especially in obese children (76).

On the other hand, general dietary recommendations of the AHA for children stress a diet that primarily relies on fruits and vegetables, whole grains, low-fat and nonfat dairy products, beans, fish, and lean meat (77). Finally, applying a maintenance diet for obese children and adolescents to achieve the normal growth is more important.

**Carbohydrate**

Carbohydrates as the main part of the diet play an important role to produce of daily energy requirement. In severely obese subjects with a high prevalence of diabetes and the metabolic syndrome, carbohydrate-restricted diet induces more weight loss in comparison with fat- and calorie-restricted diet (78). Carbohydrate composition of diets, including the glycemic index (GI) is another factor which affects calorie intake due to satiety responses. In a randomized crossover study in 16 obese adolescents (8 female and 8 male) to investigate the effect of a low-GI and high-GI meal on metabolic, hormonal, and satiety responses in overweight adolescents the results showed that prolonged satiety associated with low GI foods may prove an effective method for reducing caloric intake and achieving long-term weight control in obese adolescents (79). An ad libitum low glycemic load (GL) diet comparing with a low fat diet led to greater reduction in BMI and fat mass and a small but significant increment in insulin resistance after 12 months. In both treatment groups, the dietary GL was the principle predictor of treatment response while dietary fat was not (80).

Furthermore, the low-carbohydrate diet is associated with a greater improvement in some risk factors for CHD, insulin resistance and obesity (76, 81, 82). However, there are some controversial findings. Demol et al (81) compared the impact of three isoenergetic diets containing low-carbohydrate and different fat content (low or high) to high-carbohydrate low-fat diet on weight and metabolic markers. Findings showed that all three diets lead to a significant weight loss and metabolic markers improvement. However, only both low carbohydrate diets were associated with lower insulin level and homeostasis model assessment (HOMA). A recent well-designed study showed that a low carbohydrate diet (42% energy from CHO) vs. a standard diet (55% of energy from CHO) could not cause a greater decrement in body weight but favorably affect the homeostasis of glucose and insulin up to 3 hours postingestionand decreased significantly the serum TG concentration among African-American obese girls(82). However, based on some findings longer and larger studies are required to determine the long-term safety and efficacy of low-carbohydrate diet in weight management specially in obese children because there are some evidences which show that consuming a low-carbohydrate high-protein diet over six weeks could increase the calcium loss (83).In contrast, among obese patients, weight loss was associated with longer diet duration, restriction of calorie intake, but not with reduced carbohydrate content. Low-carbohydrate diets had no significant adverse effect on serum lipid, fasting serum glucose, and fasting serum insulin levels, or blood pressure (84).

Among the dietary carbohydrates, consumption of simple carbohydrate especially sugar and sugar-sweetened beverages have important role in the prevalence of the obesity in adolescents. There are some evidences about increasing consumption of sugar-sweetened beverages in children and adolescents. They consume 10% to 15% of total calories from sugar-sweetened beverages (SSB). Fruit juice drinkers consumed, on average, 148 (ages 2–5), 136 (ages 6–11), and 184 (ages 12–19) kcal/day (85). SSB consumption is independently and positively associated with HOMA-IR, waist circumference, systolic blood pressure and BMI, and inversely with HDL-C concentrations (86).

**Dietary Fat**

Fat is the most energy-dense macronutrient and is a target for reducing the energy content. A diet high in fat and fast food is often associated with obesity and metabolic syndrome. However, traditional foods which are high in fiber and low in fat (e.g. beans and squash) as a part of healthy eating pattern plus physical activity could prevent obesity and its consequences such as CVD and diabetes (87). Reinehr et al (88) showed that a low-fat high-carbohydrate diet decreases significantly body weight and serum insulin and leptin levels, and increases adiponectin. However, another study showed that dietary composition is more important rather than physical activity (89). This study reported that while higher carbohydrate intake is associated with higher WC, TG and serum glucose, a diet with high-

er intake of fat is associated with smaller WC and lower TG and glucose (89). Overall, daily fat intake of 25-35% of total calories is advocated in management and control of metabolic syndrome components (69).

The majority of studies were aimed at reducing cardiovascular risk-factors in the form of a decreased consumption of saturated fat, total fat, cholesterol, sodium, and sugar and an increased consumption of complex carbohydrates. Most of the studies concerned both behavioral changes and changes in attitudes and knowledge (90- 92).

Because the major sources of saturated fat and cholesterol in children's diets are full-fat milk and cheese and fatty meats, use of low-fat dairy products and lean cuts of meat in appropriate portion sizes will be critical in meeting dietary needs and nutrient requirements (73). In other words, not only the amount of fat but also the type of dietary fat intake play significant role in achieving weight loss and health promotion.

In sum, the primary goal of childhood obesity interventions is regulation of body weight and fat with adequate nutrition for growth and development. Ideally, these interventions are should modify eating and exercise behaviors such that new, healthier behaviors develop and replace unhealthy behaviors, thereby allowing healthier behaviors to persist throughout development and into adulthood (68). Recent public health dietary guidelines emphasizes in low intakes of saturated and trans fat, cholesterol, and added sugar and salt; appropriate calorie intake and physical activity for achieving the normal weight for height; and adequate intake of micronutrients (93).

**Dietary recommendations**

Gouverie al. (94) indicated that following the Mediterranean diet can reduce the prevalence of metabolic syndrome and, consequently, prevent diabetes mellitus and CVDs.On the other hand, some other researches have demonstrated that an empty calorie eating pattern, which includes high percentages of total fat and sweetened beverages intake, or patterns predominated by refined cereals or sweets and cakes, are closely linked with the elevated risk of the metabolic syndrome (95).

Links between carbohydrate quality and other components of metabolic syndrome in overweight adolescents have been shown in a number of studies. The findings showed that the total amount of sugar and sweetened beverages were associated with poor beta-cell function and beta-cell compensatory reaction deteriorates in people with increased sugar intakes (96).

Obesity prevention and treatment have a profound impact on reducing MetSrisk in youth. The number of studies on dietary interventions aimed at reducing the incidence of metabolic syndrome in overweight youths is very limited. Most of the interventional studies included physical activity components beside dietary changes. However, because of the important role of obesity in MetS, diet and exercise are two key components of the treatment of obesity and consequently the metabolic abnormalities and MetS.

In many developing countries which are in nutritional transition most of adolescents consume a high-carbohydrate and saturated fat diet with sugar-rich beverages (97, 98). Therefore, dietary strategies should include an emphasis on regular meals and increased fiber intake.

In some studies, high-fat diets in comparison with high-carbohydrate diets had deteriorate insulin action in the body. Although several studies have demonstrated the relationship between fat-containing foods and insulin resistance, other research implied the role of sub-types of fat in the diet and the importance of this effect needs to be investigated (99). Dietary intake of saturated fatty acids (SFA) was associated with insulin resistance while, polyunsaturated fatty acids (PUFA) intake had a protective effects against the obesity processes and insulin resistance. Adding PUFA or monounsaturated fatty acids (MUFA) to SFA-rich diets can reduce the total cholesterol and LDL-C (100). However, an observational study of 2128 adolescents (12-19 yr old) showed that dietary fiber intake and nutrient density are inversely associated with MetS in US adolescents while, neither SFA nor cholesterol indices were associated with MetS (101). Another long-term interventional study did not show any significant difference between a low-GI diet and low-fat diet on MetS, BMI and insulin resistance among adolescents after two years (102).

Some evidences implied that only dietary soluble fiber was associated with components of metabolic syndrome. According to Ventura et al. (96) people with no features of metabolic syndrome consumed more soluble fiber than people who had three features of metabolic syndrome. So, increasing daily consumption of soluble fiber through using one extra serving of fruit, vegetables, or legumes in the diet could reduce the risk of metabolic syndrome and metabolic health in obese children. Interestingly, the effect of dietary intake modification including sugar and fiber intake with diabetes and adiposity have shown that only increasing in the total dietary fiber and insoluble ones after two years is associated with lower visceral adipose tissue among the overweight children and adolescents (87). Therefore, small to moderate decrease in dietary

Whole grains have been known as good fiber sources with some benefits in weight management and prevention of metabolic syndrome. The Framingham Offspring Cohort study indicated that higher consumption of cereal fiber and wholegrain was associated with lower prevalence of metabolic syndrome (103). These findings were supported by another study (104).

Soft drinks contain relatively higher calories due to their sugar contents and are linked with obesity (105). Relationship between metabolic syndrome and soft drink consumption is reported by some studies. The findings indicated that daily soft drink consumption was associated with high prevalence of metabolic syndrome, increased waist circumference, impaired fasting glucose, higher blood pressure, hypertriglyceridemia and high LDL-C levels (106). It was postulated that limiting the consumption of soft drinks would reduce the prevalence of multiple CVD risk factors.

Dietary pattern also have an important role in weight control and could normalize the lipid profile in obese children. Healthful eating patterns among this group of population should be encouraged in order to improve their lipid profile. Parents and caregivers should be educated to substitute the consumption of fruits, vegetables, and whole grains with unhealthy snacks such as soft drinks, cookies, and crackers (107).

Even short-duration and strict diet and exercise program can help the youths to improve the metabolic syndrome components (108). Such studies suggest that lifestyle modifications improve the insulin resistance and body weight in young people with metabolic syndrome.

The biggest benefit for individuals with the metabolic syndrome can be derived from effective lifestyle interventions. Major lifestyle intervention programs involve weightloss in overweight or obese individuals, enhanced physical activity,and the alteration of diet composition which contribute to dyslipidemia and impaired glucose tolerance (109). Because childhood metabolic syndrome is likely to continue into adulthood, early diagnosis may help target interventions to control future CVD risk (21). Sweetened beverages, high saturated and trans fat-containing foods should be restricted and empty calorie snacks between meals should be discouraged (95). Having diets which are rich in

fruits, vegetables, and low-fat dairy products and low salty foods and processed food products are most suitable for children and adolescents to prevent chronic non-communicable diseases notably diabetes mellitus and CVDs in their future life.

**Table 1- various definitions of metabolic syndrome in children and adolescents**

| Definition | Obesity | Dyslipidemia | HDL-cholesterol | blood pressure | fasting blood glucose | More explanation |
|---|---|---|---|---|---|---|
| Weiss et al (40) | WC ≥ 75th percentile for age and sex | Fasting TGs ≥ fifth percentile for age and sex | HDL< fifth percentile for age and sex | SBP/DBP > 95th percentile for age and sex | FBS≥100mg/dL | 3 or more of the mentioned abnormalities |
| Cook et al (24) | WC ≥ 90th percentile for age and sex | Fasting TGs ≥ 110 mg/dL | HDL-C≤40 mg/dL for all ages and sexs | ≥90% for age, sex, and height | FBS≥110mg/dL (ADA) | 3 or more of the mentioned abnormalities |
| de Ferranti et al (23) | WC ≥ 75th percentile for age and sex | Fasting TGs ≥ 1.1 mmol/L (or ≥100 mg/dL) | HDL-C<1.3 mmol/L (<50mg/dL)‡ | SBP/DBP > 90th percentile for age and sex | FBS ≥6.1 mmol/l (or ≥110mg/dL) | 3 or more of the mentioned abnormalities |
| Cruz (25) | WC ≥ 90th percentile for age, sex and race specific | Fasting TGs ≥ 90th percentile for age and sex | HDL< tenth percentile for age and sex | ≥90% for age, sex, and height | FBS≥110mg/dL (ADA) | 3 or more of the mentioned abnormalities |
| Lambert et al (26) | BMI ≥ 85th percentile for age and sex | Fasting TGs ≥ 75th percentile for age and sex | HDL< 25th percentile for age and sex | SBP >95th or 75thpercentile for age and sex | FBS≥110mg/dL (ADA) ≥75th fasting insulin for age and sex | 3 or more of the mentioned abnormalities |
| Rodríguez-Morán et al (110) | BMI ≥ 90th percentile for age and sex | Fasting TGs ≥ 90th percentile for age and sex | - | SBP/DBP ≥ 90th percentile for age and sex | FBS ≥6.1 mmol/l | This definition includes a screening stage which subjects should have ≥2 of following risks: -having one or more family phenotype of HTN, obesity and T2DM, - having low or high birth weight - being obese or HTN |
| Seoet al (111) | WC ≥ 90th percentile for age and sex | Fasting TGs ≥ 110 mg/dL | HDL-C≤40 mg/dL | systolic or diastolic blood pressure≥90th percentile | FBS≥110mg/dL | 3 or more of the mentioned abnormalities |

**‡ except in boys aged 15 to 19 years, in whom the cut-point was 45 mg/dl**

**Table 2: IDF metabolic syndrome criteria for children and adolescents by age groups.**

| Age groups (yr) | Obesity (waist circumference) | TG (mmol/l) | HDL-C (mmol/l) | B.P* (mmHg) | F.B.S* (mmol/l) |
|---|---|---|---|---|---|
| 6 to <10 | ≥90th percentile | Metabolic syndrome cannot be diagnosed, but further measurements should be made if there is a family history of metabolic syndrome, T2DM, dyslipidemia, CVD, hypertension, and/or obesity | | | |
| 10 to <16 | ≥90th percentile or adult cutoff if lower | ≥ 1.7 | < 1.03 | Systolic ≥130 or diastolic ≥85 | ≥5.6 or known T2DM |
| 16+ | Use existing IDF criteria for adults:<br>Central obesity plus any two of the following four factors:<br>• Raised triglycerides: ≥1.7 mmol/l<br>• Reduced HDL-C < 1.03 mml/l in males and < 1.29 mmol/l in females or specific treatment for these lipid abnormalities<br>Raised blood pressure: systolic blood pressure ≥130 mmHg or diastolic blood pressure ≥85<br>• mmHg or treatment of previously diagnosed hypertension<br>*IFG: fasting serum glucose ≥ 5.6 mmol/liter or previously diagnosed T2DM | | | | |

***B.P: Blood pressure; F.B.S: Fasting blood sugar; IFG: Impaired fasting glucose.**

**Table 3: Summary of the prevalence of the Mets in children and adolescents by different criteria**

| Study | Country/population | Criteria | Prevalence of MetS |
|---|---|---|---|
| Csábiet al. (2000) | Hungary/ 8–18-y-old obese girls and boys | Presence of hyperinsulinemia, HTN, IGTT and dyslipidemia (high cholesterol and/or high TG and/or low HDL cholesterol) | 8.9% in obese children. three risk factors occurred in 0.4% of controls |
| Cook et al. (24) | USA/12–19-y-old female and male | Modified-ATP III | 4.2% (6.1% in males, 2.1% in females). 28.7% in overweight adolescents |
| Cruz et al. ( 25 ) | USA/ 8–13-y-old obese children | Presence of at least three of the following: abdominal obesity, low HDL cholesterol, hypertriglyceridemia, hypertension and/or impaired glucose tolerance | 30% in obese children |
| Esmailzadehet al. (112) | Iran /10-19-y-old overweight and at risk of overweight | Modified ATPIII | 10.1% |
| Kelishadiet al. (113) | Iran /6-18-y-boy and girl | Modified ATPIII | 14.1% with no difference between boys and girls where, was higher in boys |

## REFERENCES

1- Reaven GM.. Role of insulin resistance in human disease. Diabetes Care.1998; 37:1595-1607. | | 2- Misra A. Insulin resistance syndrome: current perspective and its relevance in Indians. Indian Heart Journal. 1998; 50:385–395. | | 3- Reaven GM. Role of insulin resistance in human disease (syndrome X): an expanded definition. Annu Rev Med. 1993;44:121–131. | | 4- Kaplan NM. The deadly quartet: upper-body obesity, glucose intolerance, hypertriglyceridemia, and hypertension. Arch Intern Med. 1989; 149:1514 –1520. | | 5- DeFronzo RA, Ferrannini E. Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care. 1991;14:173–194. | | 6- Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. The Lancet. 2005; 365:1415-1428. | | 7- Lemieux I, Pascot A, Couillard C. Hypertriglyceridemic waist: a marker of the atherogenic metabolic triad (hyperinsulinemia; hyperapolipoprotein B; small, dense LDL) in men? Circulation. 2000; 102: 179-184. | | 8- Kraja AT, Hunt SC, Pankow JS, Myers RH, Heiss G, Lewis CE, Rao DC, Province MA. An evaluation of the metabolic syndrome in the HyperGEN study. Nutrition Metabolism. 2005; 2: 2. | | 9- STERN MP, WILLIAMS K, GONZALEZ-VILLALPANDO C, HUNT KJ, HAFFNER SM. Does the metabolic syndrome improve identification of individuals at risk of type 2 diabetes and/or cardiovascular disease? Diabetes Care 2004: 27: 2676–2681. | | 10- Grundy SM, Brewer HB, Cleeman JI, Smith SC, Lenfant C. Definition of Metabolic Syndrome Report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on Scientific Issues Related to Definition. Circulation. 2004; 109: 433-438. | | 11- Amemiya S, Dobashi K, Urakami T, Sugihara S, Ohzeki T, Tajima N. Metabolic syndrome in youths. Pediatric Diabetes. 2007; 9: 48-54. | | 12- Brietzke SA. Controversy in diagnosis and management of the metabolic syndrome. The Medical Clinics of North America. 2007;91:1041-1061. | | 13- Groop L. Genetics of the metabolic syndrome. British Journal of Nutrition. 2000; 83(1): 39-48. | | 14- Ornstein RM & Jacobson MS. Supersize teens: The metabolic syndrome. Adolescent Medicine Clinics. 2006; 17:565-587. | | 15- World Health Organization (WHO). Definition, diagnosis and classification of diabetes mellitus and its complications : report of a WHO consultation. 1999. Retrieved from: http://www.who.int/hq/1999/who_ncd_ncs_99.2. | | 16- National Cholesterol Education Program (NCEP). Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Journal of American Medical Association. 2001; 285:2486-2497. | | 17- Balkau B & Charles MA. Comment on the provisional report from the WHO consultation. Diabetic Medicine. 1999; 16:442-443. | | 18- Alberti KG, Zimmet P, Shaw J. IDF Epidemiology Task Force Consensus Group. The metabolic syndrome–a new worldwide definition. Lancet. 2005; 366:1059-1062. | | 19- Bloomgarden ZT. consensus conference on the insulin resistance syndrome. Diabetes Care. 2003; 26(3): 933-939. | | 20- Ferrannini E, Natali A, Bell P, Cavallo-Perin P, Lalic N, Mingrone G. Insulin resistance and hypersecretion in obesity. European Group for the Study of Insulin Resistance (EGIR). Journal of Clinical Investigation. 1997; 100(5): 1166-1173. | | 21- Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among U.S. Adolescents, 1999–2000. Diabetes Care. 2004; 27: 2438-2443. | | 22- Misra A & Vikram NK. Metabolic syndrome in children and adolescents:Problems in definition, and ethnicity-related Determinants Diabetes and Metabolic Syndrome. Clinical Research & Reviews. 2007; 1:121-126. | | 23- de-Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW, Rifai N. Prevalence of themetabolic syndrome in American adolescents: findings from theThird National Health and Nutrition Examination Survey. Circulation. 2004; 110: 2494 -2497. | | 24- Cook S, Weitzman M, Auinger P, Nguyen M, Dietz WH. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the Third National Health and Nutrition Examination Survey, 1988-1994. Archives of Pediatrics & Adolescent Medicine. 2003; 157:821-827. | | 25- Cruz ML, Weigensberg MJ, Huang TT, Ball G, ShaibiGQ, Goran MI. The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. Journal of Clinical Endocrinology and Metabolism. 2004; 89: 108-113. | | 26- Lambert M, Paradis g, O'Loughlin J. Insulin resistancesyndrome in a representative sample of children and adolescentsfrom Quebec, Canada. International journal of obesity and related metabolic disorders. 2004; 28:833-841. | | 27- Moran M, Salazar-Vazquez B, Violante R, Guerrero-Romero F. Metabolic syndrome among children and adolescents aged 10–18 years. Diabetes Care. 2004; 27:2516-2517. | | 28- Weiss R, Dufour S, Taksali SE, Tamborlane WV, Petersen KF, Bonadonna RC, Boselli L, Barbetta G, Allen K, Rife F, Savoye M, Dziura J, Sherwin R, Shulman GI, Caprio S. Prediabetes in obese youth: a syndrome of impaired glucose tolerance, severe insulin resistance, and altered myocellular and abdominal fat partitioning. Lancet. 2003; 362: 951-957. | | 29- Kelishadi R. Childhood overweight, obesity, and the metabolic syndrome in developing countries. Epidemiologic Reviews. 2007; 29: 62–76. | | 30- Kurçer MA & Ozbay A. Anadolu Kardiyol Derg.Effects of patient education and counseling about lifestyle on quality of life in patients with coronary artery disease. Anatolian Journal of Cardiology. 2011; 11(2):107-113. | | 31- Zimmet P, Alberti G, Kaufman F, Tajima N, Silink M, Arslanian S. The metabolic syndrome in children and adolescents. Lancet. 2007; 369: 2059–2061. | | 32- Prodam F, Ricotti R, Genoni G, Parlamento S, Petri A, Balossini C, Savastio S, Bona G, Bellone S. Comparison of two classifications of metabolic syndrome in the pediatric population and the impact of cholesterol. J Endocrinol Invest. 2012 Nov 27. [Epub ahead of print] | | 33- Wan NJ, Mi J, Wang TY, Duan JL, Li M, Gong CX, Du JB, Zhao XY, Cheng H, Hou DQ, Wang L. [Metabolic syndrome in overweight and obese schoolchildren in Beijing]. ZhonghuaErKeZaZhi. 2007;45(6):417-21. | | 34- Golley RK, Magarey AM, Steinbeck KS, Baur LA, Daniels LA. Comparison of metabolic syndromeprevalence using six different definitions in overweight pre-pubertal children enrolled in a weight management study. Int J Obes (Lond). 2006;30(5):853-60. | | 35- Saffari F, Jalilolghadr S, Esmailzadehha N, Azinfar P. Metabolic syndrome in a sample of the 6- to 16-year-old overweight or obese pediatric population: a comparison of two definitions. TherClin Risk Manag. 2012;8:55-63. | | 36- Friend A, Craig L, Turner S. The Prevalence of Metabolic Syndrome in Children: A Systematic Review of the Literature. MetabSyndrRelatDisord. 2012 Dec 18. [Epub ahead of print] | | 37- Nobili V, Alisi A, Panera N, Agostoni C. Low birth weight and catch-up-growth associated with metabolic syndrome: a ten year systematic review.PediatrEndocrinol Rev. 2008;6(2):241-7. | | 38- Weiss R. Metabolic syndrome in childhood - causes and effects.Endocrine development. 2010; 19: 62-72. | | 39- Varda NM & Gregoric A. Metabolic syndrome in the pediatric population: a short overview. Pediatric Reports. 2009;1(1):1. | | 40- Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoye M, Morrison J, Sherwin RS, Caprio S. Obesity and the metabolic syndrome in children and adolescents. New England Journal of Medicine. 2004; 350: 2362-2374. | | 41- Berenson GS, Srinivasan SR, Bao W, Newman WP, Tracy RE, Wattigney WA. Association between Multiple Cardiovascular Risk Factors and Atherosclerosis in Children and Young Adults. The new England journal of medicine. 1998; 338:1650-1656. | | 42- Katzmarzyk PT, Perusse L, Malina RM, Bergeron J, Despres JP, Bouchard C. Stability of indicators of the metabolic syndrome from childhood and adolescence to young adulthood: the Quebec Family Study. Journal of Clinical Epidemiolgy. 2001; 54:190–195. | | 43- Torok K, Szelenyi Z, JP. Low physical performance in obese adolescent boys with metabolic syndrome. International journal of obesity and related metabolic disorders. 2001; 25:966-970. | | 44- Phillips LK & Prins JB. The link between abdominal obesity and the metabolic syndrome.Current Hypertension Reports. 2008; 10(2):156-164. | | 45- James PT. Obesity: The worldwide epidemic. Clinics in Dermatology. 2004; 22: 276-280. | | 46- Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. Pediatrics. 1998; 101( 2): 518-525. | | 47- Bertrand OF & Poirier P. Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk.Arteriosclerosis, Thrombosis, and Vascular Biology. 2008; 28(6): 1039-1049. | | 48- Després JP. Abdominal obesity: the most prevalent cause of the metabolic syndrome and related cardiometabolic risk. Eur Heart J. 2006; 8(suppl B): B4-B12. | | 49- Nelson RA & Bremer AA. Insulin resistance and metabolic syndrome in the pediatric population. Metabolic Syndrome Related Disorders. 2010; 8(1):1-14. | | 50- Lann D, Gallagher E, Leroith D. Insulin resistance and the metabolic syndrome. Minerva Medica Journal. 2008; 99(3): 253-262. | | 51- Knoops KT, de Groot LC, Kromhout D, Staveren WA. Mediterranean Diet, Lifestyle Factors, and Mortality. JAMA. 2005; 293(6): 674-675. | | 52- Lemieux I, Pascot A, Prud'homme D, Alme´ras N, Bogaty P, Nadeau A, Bergeron J, Després JP. Elevated C-reactive protein: another component of the atherothrombotic profile of abdominal obesity. Arterioscler Thromb Vasc Biol. 2001;21:961–967. | | 53- Després JP, Lemieux I, Bergeron J, Pibarot P, Mathieu P, Larose E, Rodés-Cabau J, Bertrand OF, Poirier P. Abdominal Obesity and the Metabolic Syndrome: Contribution to Global Cardiometabolic Risk.Arteriosclerosis, Thrombosis, and Vascular Biology. 2008; 28:1039-1049. | | 54- McCarty MF, Merzer G, Pereira D, LudwigS. Dairy Products and Insulin Resistance. Journal of the American Medical Association. 2002;288(6):693-694. | | 55- Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. CurrOpinLipidol. 2002;13:3–9. | | 56- Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. Nutr Rev. 2004;62:177–203. | | 57- Kant AK. Dietary patterns and health outcomes. J Am Diet Assoc. 2004;104:615–35. | | 58- Jacobs DR Jr, Steffen LM. Nutrients, foods, and dietary patterns as exposures in research: a framework for food synergy. Am J ClinNutr. 2003;78:S508–13. | | 59- Ambrosini GL, Huang RC, Mori TA, Hands BP, O'Sullivan TA, de Klerk NH, Beilin LJ, Oddy WH. Dietary patterns and markers for the metabolic syndrome in Australian adolescents. NutrMetabCardiovasc Dis. 2010;20(4):274-83. | | 60- Joung H, Hong S, Song Y, Ahn BC, Park MJ. Dietary patterns and metabolic syndrome risk factors among adolescents. Korean J Pediatr. 2012;55(4):128-35. | | 61- McNaughton SA, Ball K, Mishra GD, Crawford DA. Dietary patterns of adolescents and risk of obesity and hypertension. J Nutr. 2008;138(2):364-70. | | 62- Ambrosini GL, Emmett PM, Northstone K, Howe LD, Tilling K, Jebb SA. Identification of a dietary pattern prospectively associated with increased adiposity during childhood and adolescence. Int J Obes (Lond). 2012;36(10):1299-305. | | 63- Rodríguez-Ramírez S, Mundo-Rosas V, García-Guerra A, Shamah-Levy T. Dietary patterns are associated with overweight and obesity in Mexican school-age children. Arch LatinoamNutr. 2011;61(3):270-8. | | 64- Shang X, Li Y, Liu A, Zhang Q, Hu X, Du S, Ma J, Xu G, Li Y, Guo H, Du L, Ma G. Dietary pattern and its association with the prevalence of obesity and related cardiometabolic risk factors among Chinese children. PLoS One. 2012;7(8):e43183. | | 65- Romero-Polvo A, Denova-Gutiérrez E, Rivera-Paredez B, Castañón S, Gallegos-Carrillo K, Halley-Castillo E, Borges G, Flores M, Salmerón J. Association between dietary patterns and insulin resistance in Mexican children and adolescents. Ann NutrMetab. 2012;61(2):142-50. | | 66- Ritchie LD, Spector P, Stevens MJ, Schmidt MM, Schreiber GB, Striegel-Moore RH, Wang MC, Crawford PB. Dietary patterns in adolescence are related to adiposity in young adulthood in black and white females. J Nutr. 2007;137(2):399-406. | | 67- Pan Y, Pratt CA. Metabolicsyndrome and its association with diet and physical activity in US adolescents. J Am Diet Assoc. 2008;108(2):276-86. | | 68- Epstein LH, Myers MD, Raynor HA, Saelens BE. Treatment of Pediatric Obesity. Pediatrics. 1998; 101: 554-570. | | 69- Grundy SM, Cleeman JI , Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith SC, Spertus JA, Costa F. Diagnosis and Management of the Metabolic Syndrome .Circulation. 2005; 112:2735–3752. | | 70- Seagle HM, Strain GW, Makris A, Reeves RS; AmericanDieteticAssociation. Position of the American Dietetic Association: Weight Management. J Am Diet Assoc. 2009;109(2):330-46. | | 71- Hill GO, Wyatt HR, Reed GW, Peters JC. Obesity and the Environment: Where Do We Go from Here?. Science. 2003; 299: 853-855. | | 72- Butte NF & Ellis KJ. Comment on "Obesity and the envirement : Where do we go from here?. Science. 2003; 301: 598b. | | 73- Gidding SS, Dennison BA, Birch LL, Daniels SR, Gilman MW, Lichtenstein AH, Rattay KT, Steinberger J, Stettler N, Van Horn L. Dietary Recommendations for Children and Adolescents : A Guide for Practitioners. Circulation. 2005; 112:2061-2075. | |

## REFERENCES

74- Barlow SE & Dietz WH. Management of child and adolescent obesity: summary and recommendations based on reports from pediatricians, pediatric nurse practitioners, and registered dietitians. Pediatrics. 2002; 110:236–238. | | 75- Dietz WH. Overweight in Childhood Adolescence. New England Journal of Medicine. 2004; 350: 855-858. | | 76- Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Selma-Mohammed B, Szapary PO, Rader DJ, Edman JS, Klein S. A Randomized Trial of a Low-Carbohydrate Diet for Obesity. The New England Journal of Medicin. 2003; 348:2082-90. | | 77- Kavey RW, Daniels SR, Lauer RM, Atkins DL, Hayman LL, Taubert K. American Heart Association Guidelines for Primary Prevention of Atherosclerotic Cardiovascular Disease Beginning in Childhood. Circulation. 2003; 107: 1562-1566. | | 78- Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams T, Williams M, Gracely EJ, Stern L. A low-carbohydrate as compared with a low-fat diet in severe obesity. New England Journal of Medicine. 2003; 348(21): 2074-81. | | 79- Ball SD, Keller KR, Moyer-Mileur LJ, Ding YW, Donaldson D, Jackson WD. Prolongation of satiety after low versus moderately high glycemic index meals in obese adolescents. Pediatrics. 2003; 111(3):488-94. | | 80- Ebbeling CB, Leidig MM, Sinclair KB, Hangen JP, Ludwig DS. A reduced-glycemic load diet in the treatment of adolescent obesity. Arch PediatrAdolesc Med. 2003;157(8):773-9. | | 81- Demol S, Yackobovitch-Gavan M, Shalitin S, Nagelberg N, Gillon-Keren M, Phillip M. Low-carbohydrate (low & high-fat) versus high-carbohydrate low-fat diets in the treatment of obesity in adolescents. ActaPaediatr. 2009;98(2):346-51. | | 82- Casazza K, Cardel M, Dulin-Keita A, Hanks LJ, Gower BA, Newton AL, Wallace S. Reduced carbohydrate diet to improve metabolic outcomes and decrease adiposity in obese peripubertal African American girls. J PediatrGastroenterolNutr. 2012;54(3):336-42. | | 83- Reddy ST, Wang CY, Sakhaee K, Brinkley L, Pak CY. Effect of low-carbohydrate high-protein diets on acid-base balance, stone-forming propensity, and calcium metabolism. American Journal of Kidney Diseases. 2002; 40 (2): 265-274 | | 84- Bravata DM, Sanders L, Huang J, Krumholz HM, Olkin I, Gardner CD.Efficacy and safety of low-carbohydrate diets: a systematic review. Journal of the American Medical Association. 2003; 9:289(14), 1837-50. | | 85- Wang YC, Bleich SN, Gortmaker SL. Increasing caloric contribution from sugar-sweetened beverages and 100% fruit juices among US children and adolescents, 1988-2004. Pediatrics. 2008; 121(6): 1604-14 | | 86- Bremer AA, Auinger P, Byrd RS. Sugar-Sweetened Beverage Intake Trends in US Adolescents and Their Association with Insulin Resistance-Related Parameters. J NutrMetab. 2010;2010. pii: 196476. | | 87- Davis B & Carpenter C. Proximity of fast-food restaurants to schools and adolescent obesity. American Journal of Public Health. 2009;99:505-510. | | 88- Reinehr T, Roth CL, Alexy U, Kersting M, Kiess W, Andler W. Ghrelin levels before and after reduction of overweight due to a low-fat high-carbohydrate diet in obese children and adolescents. Int J Obes (Lond). 2005;29(4):362-8. | | 89- Casazza K, Dulin-Keita A, Gower BA, Fernandez JR. Differential influence of diet and physical activity on components of metabolic syndrome in a multiethnic sample of children. J Am Diet Assoc. 2009;109(2):236-44. | | 90- Hursti UK & Sjoden PO. Changing food habits in children and adolescents experiences from intervention studies. Scandinavian Journal of Nutrition. 1997; 41: 102-110. | | 91- White AA& Skinner JD. Can goal setting as a component of nutrition education effect behavior change among adolescents? Journal of Nutrition Education and Behavior. 1998; 20: 327-335. | | 92- Contento IR, Manning AD, Shannon B. Research perspective on school-based nutrition education. Journal of Nutrition Education. 1992; 24:247-60. | | 93- Gidding SS, Dennison BA, Birch LL, Daniels SR, Gillman MW, Lichtenstein AH, Rattay KT, Steinberger J, Stettler N, Van Horn L; American Heart Association. Dietaryrecommendations for children and adolescents: a guide for practitioners. Pediatrics. 2006;117(2):544-59. | | 94- Gouveri ET, Tzavara C, Drakopanagiotakis F, Tsaoussoglou M, Marakomichelakis GE, Tountas Y, Diamantopoulos EJ. Mediterranean Diet and Metabolic Syndrome in an Urban Population: The Athens Study. NutrClinPract. 2011; 26(5): 598-606. | | 95- Bremer AA, Auinger P, Byrd RS. Sugar-Sweetened Beverage Intake Trends in US Adolescents and Their Association with Insulin Resistance-Related Parameters. Journal of Nutrition and Metabolism. 2010; 1964-1976. | | 96- Ventura ee, Davis jn, Alexander ke, Shaibi gq, Lee w, Byrd-williams CE, Toledo-Corral CM, Lane CJ, Kelly LA, Weigensberg MJ, Goran MI. Dietary intake and the metabolic syndrome in overweight latino children. Journal of american dietetics association. 2008; 108: 1355-1359. | | 97- Wasir JS & Misra A. The metabolic syndrome in Asian Indians: the impact of nutritional and socio-economic transition in India,. Metabolic Syndrome Related Disorders. 2004; 2: 14-23. | | 98- Delavar MA, Lye MS, Khor GL, Hassan ST, Hanachi P. Dietary patterns and the metabolic syndrome in middle aged women, Babol, Iran. Asia Pacific Journal Clinical Nutrition. 2009; 18(2): 285-292. | | 99- Storlien L, Kraegen E, Chisholm D, Ford G, Bruce D, Pascoe W. Fish oil prevents insulin resistance induced by high- fat feeding in rats. Science. 1987; 237: 885-888. | | 100- Wardlaw GM & Snook TJ. Effect of diets high in butter, corn oil or high oleic acid sunflower oil on serum lipids and apolipoproteins in men. American Journal of Clinical Nutrition. 1990; 51:815-821. | | 101- Carlson JJ, Eisenmann JC, Norman GJ, Ortiz KA, Young PC. Dietary fiber and nutrient density are inversely associated with the metabolic syndrome in US adolescents. J Am Diet Assoc. 2011;111(11):1688-95. | | 102- Mirza NM, Palmer MG, Sinclair KB, McCarter R, He J, Ebbeling CB, Ludwig DS, Yanovski JA. Effects of a low glycemic load or a low-fat dietary intervention on body weight in obese Hispanic American children and adolescents: a randomized controlled trial. Am J ClinNutr. 2013;97(2):276-85. | | 103- McKeown NM, Meigs JB, Liu S, Saltzman E, Wilson PW, Jacques PF. Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham Offspring Cohort. Diabetes Care. 2004; 27(2): 538-546. | | 104- Howarth NC, Huang TT, Roberts SB, McCrory MA. Dietary fiber and fat are associated with excess weight in young and middle-aged US adults. Journal of American Dietetics Association. 2005; 105(9): 1365-1372. | | 105- Malik VS, Popkin BM, Hill NC, Bray GA, Després JP, Hu FB. Sugar sweetened beverages, obesity, type 2 diabetes and cardiovascular disease risk. Circulation. 2010; 121(11): 1356–1364. | | 106- Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM,Vasan RS. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 2007; 116(5): 480-488. | | 107- Williams CL, Hayman LL, Daniels SR, Robinson TN, Steinberger J, Paridon S, Bazzarre T. Cardiovascular Health in Childhood : A Statement for Health Professionals From the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of theCouncil on Cardiovascular Disease in the Young, American Heart Association. Circulation. 2002;106:143-160. | | 108- Chen Ak, Roberts CK, Barnard RJ. Effect of a short-term diet and exercise intervention on metabolic syndrome in overweight children. Metabolism Clinical and Experimental. 2006; 55:871-878. | | 109- Magkos F, Yannakoulia M, Chan JL, Mantzoros CS. Management of the metabolic syndrome and type 2 diabetes through lifestyle modification. Annual Review of Nutrition. 2009; 29:223-256. | | | | 110- Rodríguez-Morán M, Salazar-Vázquez B, Violante R, Guerrero-Romero F. Metabolicsyndrome among children and adolescentsaged10-18years. Diabetes Care. 2004;27(10):2516-7. | | 111- Seo SL, Lee HY, Lee SW. The Prevalence of the Metabolic Syndrome in Korean Children and Adolescents: Comparisons of the Criteria of Cook et al., Cruz and Goran, and Ferranti et al. Yonsei Medical Journal. 2008; 49(4): 563–572. | | 112- Esmaillzadeh A, Mirmiran P, Azadbakht L, Etemadi A, Azizi F. High Prevalence of the Metabolic Syndrome in Iranian Adolescents. Obesity.2006; 14: 377-382. | | 113- Kelishadi R, Ardalan G, Gheiratmand R, Adeli K, Delavari A, Majdzade R. Paediatric metabolic syndrome and associated anthropometric indices: The CASPIAN Study. Acta Paediatr.2006; 95: 1625–1634. |