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Prevalence of metabolic syndrome among female school children in Saudi Arabia: An anthropometric study

Adil O. Bahathiq, Bahaeldin E. Elawad*

Department of physiology. Faculty of medicine. University of Umm-AL Qura. Holy Makkah, Kingdom of Saudi Arabia, post code 21950
*Corresponding author: Bahaelawad@gmail.com, **Tel:** 00966535548549

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Metabolic syndrome is characterized by a group of metabolic risk factors which include abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, and insulin resistance or glucose intolerance. The aim of this study was to examine the prevalence of metabolic syndrome among female school children and adolescents. A cross-sectional study was conducted among 1356 female school children and adolescents between the age of 6 to 18 years. Body mass index, waist circumference, blood glucose level, lipid profile, and arterial blood pressure were determined. Criteria of ATP III were used to diagnose metabolic syndrome among participants. Among 1356 female school children and adolescents aged 6-18 years, 15.2% were overweight and 15.3% were obese. The prevalence of metabolic syndrome was 17.11% overall, 62.02% in obese and 50% in overweight participants. An enormous population of Saudi children and adolescents particularly females are potential to develop metabolic syndrome. We recommend a national obesity prevention program at community level to be implemented to promote leaner and consequently healthier community; Weight reduction program, lifestyle modification, and screening for risk factors of metabolic syndrome should be given rather special consideration.

Keywords: Metabolic syndrome, body mass index, waist circumference, anthropometric relationship.

INTRODUCTION

Metabolic syndrome is defined by a constellation of an interconnected physiological, biochemical, clinical and metabolic factors that directly increases the risk of atherosclerotic cardio vascular diseases, type 2 diabetes mellitus, and all cause mortality (1,2).

Metabolic syndrome is characterized by a group of metabolic risk factors which include abdominal obesity (excessive fat tissue in and around the abdomen); atherogenic dyslipidemia (blood fat disorders that is high triglycerides, low high density lipoprotein cholesterol, high low density lipoprotein cholesterol); elevated blood pressure; and insulin resistance or glucose intolerance (3). There have been several definitions of metabolic syndrome, but most commonly used criteria for definition at present are from the World Health Organization (WHO) (4), the European Group for the Study of Insulin Resistance (EGIR) (5), the National Cholesterol, Education, Program Adult treatment Panel III (NCEP ATP III) (6), American Association of Clinical Endocrinologists (AACE) (7), and the International Diabetes Federation (IDF) (8).

Metabolic syndrome is a state of chronic low grade inflammation as a consequence of complex interplay between genetic and environmental factors. Visceral

adiposity, insulin resistance, atherogenic dyslipidemia, endothelial dysfunction, genetic susciptibility, elevated blood pressure, hypercoagulable state, and chronic stress are the several factors which constitute the syndrome. Adipose tissue is a heterogenous mixture of adipocytes, stromal preadipocytes, immnne cells, and endothelium, and it can respond rapidly and dynamically to alterations in nutrient excess through adipocytes hypertrophy and hyperplasia (9). With obesity and progressive adipocyte enlargement, the blood supply to adipocytes may be reduced with consequent hypoxia (10). Hypoxia has been proposed to be an inciting etiology of necrosis and macrophage infiltration into adipose tissue that leads to an overproduction of biologically active metabolites known as adipocytokines which includes glycerol, free fatty acids, proinflammatory mediators (tumor necrosis factor alpha, interleukin-6, plasminogen activator inhibitor-1, and C-reactive protein (11). This results in a localized inflammation in adipose tissue that propagates an overall systemic inflammation associated with the development of obesity related comorbidities (12).

Adipocytokines integrate the endocrine, autocrine, and paracrine signals to mediate the multiple processes including insulin sensitivity (13), oxidant stress (14), energy metabolism, blood coagulation, and inflammatory responses (15) which are thought to accelerate atherosclerosis, plaque rupture, and thrombosis. This shows that the adipose tissue is not only specialized in the storage and mobilization of lipids but it is also a remarkable endocrine organ releasing the numerous cytokines. Characterestics of insulin-sensitive phenotype include a normal body weight (16), without abdominal or visceral obesity (17), benign moderate activity (18), and consuming a diet low in saturated fats (19). Alternatively, insulin-resistant individuals demonstrate an impaired glucose metabolism or tolerance by an abnormal response to a glucose challenge, an elevated fasting glucose levels and/or overt hyperglycemia, or a reduction in insulin action after intravenous administration of insulin (euglycemic clamp technique) with decreased insulin-mediated glucose clearance and/or reductions in the suppression of endogenous glucose production. It is defined as a pathological condition in which a normal insulin concentration does not adequately produce a normal insulin response in the peripheral target tissues such as adipose, muscle, and liver. Under this condition, pancreatic beta cells produce more insulin (i.e., hyperinsulinemia) to overcome the hyperglycemia among the insulin-resistant individuals. Although hyperinsulinemia may compensate for insulin resistance to some biological actions of insulin, that is, maintenance of normoglycemia, however, it may cause an over-expression of insulin activity in some normally sensitive tissues. This accentuation of some insulin actions coupled with a resistance to other actions of insulin results in the clinical manifestations of metabolic syndrome (20). An inability of the pancreatic beta cells overtime to produce a sufficient insulin to correct the worsening tissue insulin resistance leads to hyperglycemia and overt type 2 diabetes mellitus (21).

Dyslipidemia is characterized by a spectrum of qualitative lipid abnormalities reflecting perturbations in the structure, metabolism, and biological activities of both atherogenic lipoproteins and antiatherogenic high density lipoprotein-C which includes an elevation of lipoproteins containing apolipoprotein B, elevated triglycerides, increased levels of small particles of low density lipoprotein, and low levels of high density lipoprotein-C. Insulin resistance leads to an atherogenic dyslipidemia in several ways. First, insulin normally suppresses lipolysis in adipoctyes, so an impaired insulin signaling increases lipolysis, resulting in increased free fatty acid levels. In the liver, free fatty acids serve as a substrate for synthesis of triglycerides. Free fatty acids also stabilize the production of apolipoprotein B, the major lipoprotein of very low density lipoprotein particles, resulting in a more very low density lipoprotein production. Second, insulin normally degrades apolipoprotein B through P13K-dependent pathways, so an insulin resistance directly increases very low density lipoprotein production. Third, insulin regulates the activity of lipoprotein lipase, the rate-limiting and major mediator of very low density lipoprotein clearance. Thus, hypertriglyceridemia in insulin resistance is the result of both an increase in very low density lipoprotein production and a decrease in very low density lipoprotein clearance. Very low density lipoprotein is metabolized to remnant lipoproteins and small dense low density lipoprotein, both of which can promote atheroma formation. The triglycerides in very low density lipoprotein are transferred to high density lipoprotein by cholesterol ester transport protein in exchange for cholesteryl esters resulting in the triglyceride-enriched high density lipoprotein and cholesteryl-enriched very low density lipoprotein particles. Further, the triglyceride-enriched high density lipoprotein is a better substrate for hepatic lipase, so it is cleared rapidly from the circulation, leaving a fewer high density lipoprotein particles to participate in a reverse cholesterol transport from the vasculature. Thus, the liver of insulin-resistant patients, free fatty acid flux is high, triglycerides synthesis and storage are increased, and excess triglycerides is secreted as very low density lipoprotein (22). For the most part, it is believed that the dyslipidemia associated with insulin resistance is a direct consequence of increased very low density lipoprotein secretion by the liver (23). Essential hypertension is frequently associated with the several metabolic abnormalities, of which obesity, glucose intolerance, and dyslipidemia are the most common (24). Studies suggest that both hyperglycemia and hyperinsulinemia activate the renin angiotensin aldosterone system by increasing the expression of angiotensinogen, angiotensi II, and angiotensin 1 receptor, which, in concert, may contribute to the development of hypertension in patients with insulin resistance (25).

There is also evidence that insulin resistance and hyperinsulinemia lead to sympathetic nervous system activation, and, as a result, the kidneys increase sodium reabsorption, the heart increases cardiac output, and arteries respond with vasoconstriction resulting in hypertension (26). It has been recently discovered that adipocytes also produce aldosterone in response to angiotensin II (27). In this regard, the adipocyte may be considered a miniature renin angiotensin aldosterone system.

Sedentary lifestyle is a medical term used to denote a type of lifestyle with no physical activity (28). It is commonly found on both developed and developing world and characterized by sitting, reading, watching television, and computer use for much of the day with little or no vigorous physical exercise (29). A sedentary lifestyle can contribute to many causes of death. People living in today's society are less physically active than they have probably ever been in history (30).

Insufficient sleep could greatly contribute to the occurrence of metabolic syndrome in obese children (31). The accepted wisdom has been that eating fats makes you fat, so it is easy to make the assumption that fast food is to blame for all society's obesity problems (32). Eating fewer calories while increasing activity is the best way to lose weight. Research on low-carbohydrate diets demonstrates that weight loss is more likely to result from eating fewer calories and staying with the diet for long time rather than cutting out the carbohydrate (33). Recent studies demonstrate that, although the prevalence of obesity is twice as high in families of obese individuals as in general population at large, this effect is dependent on the severity of obesity. It is also demonstrated that the risk of becoming obese is not entirely due to genetic factors as the risk is also elevated in cohabitating spouses of participants (34). There is a suggestion that a complex genotype-environmental interaction where the response of a phenotype to environmental changes such as diet or physical activity levels depend on the genotype of the individual (35).

The aim of this study was to examine the prevalence of metabolic syndrome among female school children and adolescents.

MATERIAL AND METHODS

A total of 1356 female school children and adolescents between the age of 6 to 18 years were recruited for this work. The study was conducted among six female schools at Makkah city In King Saudi Arabia. The permission was taken from the schools' authority. The University of Umm AL-Qura approved the study. Initially the aims of the study were explained to the participants. Written informed consent from parents and written assent from children (when possible) and adolescents were obtained. The informed consent was provided to the schools' administration. Those students who disagreed for participating in the research were not included. A questionnaire was developed, pretested, validated, and confidentiality was guaranteed. Girls were subjected to nonelectronic portable balance to monitor their weight and the scale was positioned at zero after each measurement in order to ensure accuracy. Girls were also asked to put off their shoes before measuring the weight. The height of participants was also measured in order to identify the body mass index. The participant was standing without footwear and the measurement was taken from the sole to the top of the head by the help of a fixed measuring tape. The heel and the subscapulae of the participants were kept aligned with the wall and the distance was recorded to be 1.0 cm. The body mass index of the girls was calculated as the body weight in kilograms divided by the height in square meters. According to body mass index, volunteers were classified into four groups: underweight (< 18.5 Kg/m^2; normal (18.5-24.9 Kg/m^2); overweight; (25-29.5 Kg/m^2); and obese (30 Kg/m^2 or above).Waist circumference was measured mid-way between the lower rib margin and the iliac crest upon exhalation with flexible anthropometric tape (myotape). The waist circumference was measured twice to the nearest 0.5 cm. If the variation between these two measurements was greater than 0.5 cm, a third measurement was taken and the mean was calculated by using the two closest measurements. Single cut-off points can't be used to define abnormalities in children. Percentiles, rather than absolute values of waist circumference have been used to compensate for variation in child development and ethnic origin. Several studies have used the 90^{th} percentile as a cut-off for waist circumference (36). Fasting blood glucose was measured using pen-like home blood glucose level testing kit. The result was obtained on a digital display screen (Accu-check Active). Blood pressure was measured while the volunteer seated calm for at least 10 minutes. Inflatable cuff width is at least 40% of the arm circumference and its length is at least 80% of the distance between the elbow and the acromion. (Digital automatic blood pressure MX2 basic, Omron company, Japan). Triglycerides and cholesterol were measured using automatic accoutered plus instrument (Lipid home test kit) (Cobas Roche company, Jermany). Participants of this study diagnosed as metabolic syndrome on the basis of presence of three or more of the following criteria: abdominal obesity (waist circumference), two dyslipidemia elements (elevated triglycerides and low high density lipoprotein cholesterol), elevated blood pressure, and a component representing glucose metabolism (impaired fasting glucose) (6, 37,38,39).

Body mass index has been shown to jointly predict risk factors clustering among children and adolescents. Combination of body mass index and waist circumference will be used in this work to evaluate health risk factors among children and adolescents (40)

The statistical package for social sciences (SPSS) software version 17 was used for the analysis of

descriptive results (SPSS Inc., Chicago, IL, USA). Mantel-Haenszel Chi-square test was used to evaluate trends in proportions across anthropometric measurements and risk factors for metabolic syndrome.

RSULTS AND DISCUSSION:

AS far as our knowledge is concerned, this is the first study to explore the prevalence of metabolic syndrome among children and adolescents in King Saudi Arabia. The present study demonstrates the prevalence of metabolic syndrome among 6-18-year-old 1356 female school children at Makkah city. The overall prevalence of metabolic syndrome is 17.11%. The prevalence of metabolic syndrome among overweight candidates is 50% and increases to 62.02 among obese participants (Table 1).

Table 1: Relationship between body mass index (BMI)/waist circumference (WC) classification and metabolic syndrome. MeS, metabolic syndrome; TG, Hypertriglyceredemia; CHO, high low density lipoprotein or low high density lipoprotein; DM, diabetes mellitus; BP, hypertension.

Volunteers Who have (3 or more) criteria of MeS	**Anthropometric Measurements (BMI / WC)**			**Total**
		Overweight (206)	Obese (208)	
TG and CHO	count	47	78	125
	% of total	22.80%	37.50%	56.81%
TG and DM	count	20	11	31
	% of total	9.70%	5.30%	14%
TG and BP	count	16	30	46
	% of total	7.80%	14.42%	20.90%
TG,CHO and DM	count	13	5	18
		6.31%	2.40%	8.18%
CHO and DM	count	7		7
	% of total	3.40%		3.40%
BP and DM	count		1	1
	% of total		0.50%	0.50%
BP , DM and TG	count		3	3
	% of total		1.40%	1.40%
BP , DM , TG and CHO	count		1	1
	% of total		0.50%	0.50%
Total	count	103	129	232
	% of total incidence of all population (1356)	50%	62.02%	17.11%

In a study conducted in eight European countries, including 18745 children, the 2.0 to 10.9 years metabolic syndrome prevalence was 5.5% (41). In Turkey the prevalence of metabolic syndrome among 7-15-year-old school children was found to be 6.3% (42). Prevalence of 3% of metabolic syndrome in Qatari school children was

reported (43). Zardast et al, reported prevalence of metabolic syndrome among Iranian school children aged 10-19 years to be 10.1% (44). The observed prevalence of the metabolic syndrome, below age of 20 years, in National Health and Nutritional Survey (NHANES) was 5% among the subjects of normal weight, 22% among the overweight, and 60% among the obese (45).

Metabolic syndrome consists of abdominal obesity, hypertension, glucose intolerance, and dyslipidemia (elevated triglycerides and decreased high-density lipoprotein cholesterol concentrations) (46).

Central obesity is excessive body fat in the abdomen, measured simply by waist circumference that is more indicative of metabolic syndrome profile than body mass index (47). Recent reports indicate that the prevalence of childhood metabolic syndrome has substantially increased during childhood and adolescents due to the increasing rate of childhood obesity on global scale (48).

In this current study, abdominal obesity indicated by large waist circumference was demonstrated in all participants of overweight and obese group. This current study showed that metabolic syndrome is preset in 50% and 62.02% of overweight and obese participants respectively. Thus, this study demonstrates that abdominal obesity is a major risk factor of metabolic syndrome among Saudi female school children. This is in accordance with Friek-Pedras et al. (49). This current work also revealed that children of the age group of 13-15 years were the most obese. This finding also supports the association of overweight and obesity with onset of puberty reported by Lingling et al (50). This is consistent with our clinical observation in this current work of appearance of some female secondary sexual characterestics, for example, thelarche (enlargement of the breast) and female body configuration among participants of this age group. There is a strong linear correlation between body mass index and waist circumference values. The presence of overweight in men and even normal body weight in women corresponds to an increased volume of visceral tissue in the abdomen (51). Overweight and obese participants at the age group of 13-15 years, represent 11.3% of the group whereas overweight and obese participants at the age group of 16-18 years, represent 10.8% of the group (Table 2).

Table 2: Relationship between body mass index (BMI)/waist circumference (WC) classification and age groups.

Age					
Anthropometric Measurements (BMI/WC)		**6-12 y**	**13-15y**	**16-18y**	**Total**
Underweight	count	58	31	58	147
	% of Total	4.30%	2.30%	4.30%	10.80%
Normal	count	195	235	365	795
	% of Total	14.40%	17.30%	26.90%	58.70%
Overweight	count	65	83	58	206
	% of Total	4.80%	6.10%	4.30%	15.20%
Obese	count	50	70	88	208
	% of Total	3.70%	5.20%	6.50%	15.30%
Total	count	368	419	569	1356
	% of Total	27.10%	30.90%	42.00%	100.00%

There is a positive correlation between age and anthropometric measurement at least among obese participants (Chi-square = 0.05).

Abdominal obesity is one of the main causes that leads to increased insulin resistance which can lead to type 2 diabetes mellitus (52).

This current study demonstrates no correlation between anthropometric measurement and diabetes (Chi-square = 0.442) (Table 3).

Incidence of diabetes mellitus is almost the same among overweight and obese children. Such discrepancy can be explained as follows: The body fat distribution has a critical role in the determination of whole-body insulin sensitivity and its consequences. The relation of obesity and peripheral insulin resistance depends more on the lipid distribution (lipid partitioning) in specific fat depots rather than on absolute amount of fat per se. Importantly, the distinctions are not reflected in body mass index assessments. Different lipid depots have distinct metabolic characteristics that are reflected by their adipocytokines and cytokine secretion profile, sensitivity to hormones typically affecting adipose tissue (such as norepinephrine or insulin), and anatomical blood supply drainage (portal versus systemic) (53). The secretory role of visceral fat-derived cytokines and adipocytokines {such as adiponectin (54) and leptin)} appears to be directly associated with obesity and insulin resistance. Indeed increased visceral fat

Table 3: Relationship between body mass index (BMI)/waist circumference (WC) classification and diabetes mellitus.

Diabetes					
Anthropometric Measurements (BMI/WC)		**Low**	**Normal 90-140mg/dl**	**Hi >140mg/dl**	**Total**
Underweight	Count	11	127	9	147
	% of Total	0.80%	9.40%	0.70%	10.80%
Normal	Count	59	658	78	795
	% of Total	4.40%	48.50%	5.80%	58.60%
Overweight	Count	16	167	23	206
	% of Total	1.20%	12.30%	1.70%	15.20%
Obese	Count	10	172	26	208
	% of Total	0.70%	12.70%	1.90%	15.30%
Total	Count	96	1124	136	1356
	% of Total	7.10%	82.90%	10.00%	100.00%

accumulation in obese children has been associated with increased insulin resistance and cardiovascular risk factor clustering as well as with worsening of each factor individually (55). Some obese children tend to demonstrate a lipid partitioning characterized by a large visceral fat depot along with a relatively smaller subcutaneous fat depot. This lipid partitioning profile is associated with an adverse metabolic profile in comparison with individuals with larger subcutaneous fat depots, even when the latter have greater body mass index and percent body fat and may thus be seemingly more obese (56).Waist circumference has been demonstrated to be an independent predictor of insulin resistance and intra-abdominal fat independent of body mass index in obese adolescents (57). Moreover, waist circumference has been shown to be tightly linked to systolic and diastolic blood pressure and to triglycerides and high density lipoprotein cholesterol concentrations in this age group(58). For these reasons, the international diabetes federation task force chose waist circumference, the best anthropometric correlate of intra-abdominal fat as the obesity factor of pediatric metabolic syndrome definition (59).

In this current work, hypertriglyceridemia has been encountered in 547 (40.30%) of all participants, 11.40% of them were obese and 9.1% were overweight. (Table 4).

Table 4: Relationship between body mass index (BMI)/waist circumference (WC) classification and hypertriglyceredemia.

Triglyceride				
Anthropometric Measurements (BMI/WC)		**Normal**	**Hypertriglyceridemia >150**	**Total**
Underweight	Count	112	35	147
	% of Total	8.30%	2.60%	10.80%
Normal	Count	562	233	795
	% of Total	41.40%	17.20%	58.60%
Overweight	Count	82	124	206
	% of Total	6.00%	9.10%	15.20%
Obese	Count	53	155	208
	% of Total	3.90%	11.40%	15.30%
Total	Count	809	547	1356
	% of Total	59.70%	40.30%	100.00%

There is a positive correlation between anthropometric measurement and hypertriglyceridemia (chi-square 0.002). Intramyocellular lipid deposition is inversely correlated with peripheral insulin sensitivity and has been demonstrated to be increased in obese children with impaired glucose tolerance (60). Hepatic fat accumulation is strongly associated with obesity and with hepatic resistance to the action of insulin in the context of pathways related to glucose metabolism and is also associated with an adverse cardiovascular risk profile in children (61).

Because both tissues develop insulin resistance in association with increased lipid deposition, the normal adaptation response consists of insulin secretion along

with reduced insulin clearance, leading to increased circulating insulin levels (hyperinsulinemia). Importantly, other metabolic pathways within the liver that are not involved in glucose metabolism or other insulin-sensitive tissues that do not share the pattern of increased lipid deposition within them, such as the kidney or the ovary, maintain their baseline insulin sensitivity levels yet are now exposed to hyperinsulinemia. This situation may result in a normal response of these tissues to the elevated insulin levels and manifest as sodium retention and reduced uric acid clearance by the kidney (62) (potentially elevating systemic blood pressure) and by increased androgen production by the theca cells of the ovary manifesting as polycystic ovary syndrome (63). Other metabolic pathways within the liver, especially those related to lipoprotein metabolism, maintain their baseline insulin sensitivity and respond to the elevated insulin levels in a pattern that creates the typical dyslipidemia characteristic of insulin resistance individuals. This result is manifested as elevated concentration of large very low density lipoprotein cholesterol, low high density lipoprotein cholesterol, and elevated small low density lipoprotein cholesterol (64).

This current study demonstrates a positive correlation between anthropometric measurement and hypercholesterolemia (Chi-square 0.004). (Table 5).

The current study indicated that 118 (8.7%) participants suffered from hypertension, 2.7% of them are obese and 1.2% are overweight. There is a positive correlation between anthropometric measurement and hypertension (Chi-square=0.003) (Table 6).

Table 5: Relationship between body mass index (BMI)/waist circumference (WC) classification and hyoercholesterolemia.

Cholesterol				
Anthropometric Measurements (BMI/WC)		**Normal**	**Hypercholesterolemia >200mg/dl**	**Total**
Underweight	Count	142	5	147
	% of Total	10.50%	0.40%	10.80%
Normal	Count	744	51	795
	% of Total	54.90%	3.80%	58.60%
Overweight	Count	144	62	206
	% of Total	10.60%	4.60%	15.20%
Obese	Count	117	91	208
	% of Total	8.60%	6.70%	15.30%
Total	Count	1147	209	1356
	% of Total	84.60%	15.40%	100.00%

Table 6: Relationship between body mass index (BMI)/waist circumference (WC) classification and hypertension.

Blood Pressure				
Anthropometric Measurements (BMI/WC)		**Normal**	**Hypertension 85-140mmHg**	**Total**
Underweight	Count	140	7	147
	% of Total	10.30%	0.50%	10.80%
Normal	Count	736	59	795
	% of Total	54.30%	4.40%	58.60%
Overweight	Count	190	16	206
	% of Total	14.00%	1.20%	15.20%
Obese	Count	172	36	208
	% of Total	12.70%	2.70%	15.30%
Total	Count	1238	118	1356
	% of Total	91.30%	8.70%	100.00%

Although blood pressure has been found to be elevated in association with increased body mass index in childhood (65), Sun et al found that childhood body mass index values did not contribute significantly to adult hypertension or the metabolic syndrome independent of childhood blood pressure. Therefore, the effects of childhood body mass index on adulthood hypertension and the metabolic syndrome seem to be mediated by childhood blood pressure (66).

There is a clear correlation between anthropometric measurement and physical activity (Chi-square=0.000) (Table 7). This is consistent with Alberit et al (67).

Table 7: Relationship between body mass index (BMI)/waist circumference (WC) classification and performance of physical activity.

Anthropometric Measurements (BMI/WC)	Physical Activity			
		Not performed	Performed	Total
Underweight	Count	92	55	147
	% of Total	6.80%	4.10%	10.80%
Normal	Count	463	332	795
	% of Total	34.10%	24.50%	58.60%
Overweight	Count	127	79	206
	% of Total	9.40%	5.80%	15.20%
Obese	Count	160	48	208
	% of Total	11.80%	3.50%	15.30%
Total	Count	842	514	1356
	% of Total	62.10%	37.90%	100.00%

Table 8 demonstrates a clear correlation between anthropometric measurement and sleeping hours (Chi-square 0.041). It is reported that decrease in sleeping hours will increase the risk of obesity (68).

A positive correlation is demonstrated between anthropometric measurement and type of food (Chi-square 0.04) (Table 9). This could be attributed to higher consumption of less expensive and high-calorie dense foods (69).

Table 8: Relationship between body mass index (BMI)/waist circumference (WC) classification and sleeping hours.

Sleeping Hours				
Anthropometric Measurements (BMI/WC)		9-8 hr	12-10 hr	Total
Underweight	Count	104	43	147
	% of Total	7.70%	3.20%	10.80%
Normal	Count	527	268	795
	% of Total	38.90%	19.80%	58.60%
Overweight	Count	131	75	206
	% of Total	9.70%	5.50%	15.20%
Obese	Count	151	57	208
	% of Total	11.10%	4.20%	15.30%
Total	Count	913	443	1356
	% of Total	67.30%	32.70%	100.00%

Table 9: Relationship between body mass index (BMI)/waist circumference (WC) classification and type of food.

Type of Food				
Anthropometric Measurements (BMI/WC)		**Unhealthy**	**Healthy**	**Total**
Underweight	Count	78	69	147
	% of Total	5.80%	5.10%	10.80%
Normal	Count	437	358	795
	% of Total	32.20%	26.40%	58.60%
Overweight	Count	99	107	206
	% of Total	7.30%	7.90%	15.20%
Obese	Count	113	95	208
	% of Total	8.30%	7.00%	15.30%
Total	Count	727	629	1356
	% of Total	53.60%	46.40%	100.00%

There is a clear positive correlation between anthropometric measurement and number of meals (Chi-square 0.01) (Table 10). There is a significant reduction of obesity with increasing number of daily meals. Given the consistent association of skipping meals with an increased obesity risk in children, it appears prudent to promote a regular meal pattern with five meals per day with adequate composition to children and their families (70).

Correlation between anthropometric measurement and parents' obesity shows a clear positivity (Chi-square=0.000) (Table11). Like many other medical conditions, obesity is the result of interplay between environment and genetic factors (71).

Table 10: Relationship between body mass index (BMI)/waist circumference (WC) classification and number of meals.

Number of Meals				
Anthropometric Measurements (BMI/WC)		Less than 3 meals	More than 3 meals	Total
Underweight	Count	105	42	147
	% of Total	7.70%	3.10%	10.80%
Normal	Count	632	163	795
	% of Total	46.60%	12.00%	58.60%
Overweight	Count	165	41	206
	% of Total	12.20%	3.00%	15.20%
Obese	Count	169	39	208
	% of Total	12.50%	2.90%	15.30%
Total	Count	1071	285	1356
	% of Total	79.00%	21.00%	100.00%

Table 11: Relationship between body mass index (BMI)/waist circumference (WC) classification and parents' obesity.

Parents Obesity				
Anthropometric Measurements (BMI/WC)		**Obese**	**Not obese**	**Total**
Underweight	Count	67	80	147
	% of Total	4.9%	5.9%	10.8%
Normal	Count	273	522	795
	% of Total	20.1%	38.5%	58.6%
Overweight	Count	102	104	206
	% of Total	7.5%	7.7%	15.2%
Obese	Count	114	94	208
	% of Total	8.4%	6.9%	15.3%
Total	Count	556	800	1356
	% of Total	41.0%	59.0%	100.0%

In conclusion, the current obesity pandemic is expected to result in considerable morbidity, mortality, and incremental costs to health care system around the world. The current prospective work found that all conditions studied interact with each other in a cascade of events which merge into metabolic syndrome. The latter can lead to serious long-term potentially fatal complications including heart attack and cerebrovascular accident. Therefore, if the growing prevalence of overweight and obesity continues at this pace, an enormous population of Saudi children and adolescents particularly females will be affected by metabolic syndrome. This might lead to high mortality rates in young adults. Reduction in weight to eliminate obesity is considered an important issue to public health. We recommend a national obesity prevention program at community level to be implemented to promote leaner and consequently healthier community. Change of lifestyle and regular follow up for earlier detection and screening of risk factors for metabolic syndrome should be given rather special consideration.

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REFERENCES

1. Grundy SM, Cleeman JI, Daniels SR, et al. (2005) "Diagnosis and management of metabolic syndrome: an American Health Association/National Heart, Lung and Blood Institute Scientific statement." Circulation;112(17):2735-2752
2. Wilson PWF, D'Agostino RB, Parise H et al. (2005) "Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus." Circulation;112(20):3066-3072
3. Despres J-P, Lemieux I, Bergeron J, et al. (2008) "Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk." Arterioscler Thromb Vasc Biol;28(6):1039-1049
4. Alberti KG and Zimmet PZ. (1998) "Definition, diagnosis, and classification of diabetes mellitus and its complications. Part I: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation." Diabetic Medicine.;15(7):539-553
5. Balkau B and Charles MA. (1999) "Comment on the provisional report from the WHO consultation: European Group for the Study of Insulin Resistance (EGIR)." Diabetes Medicine;16(5):442-443
6. Cleeman JI. (2001) "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)." Journal of the American Medical Association;285(19):2486-2497
7. Einhom D, Reaven GM, Cobin RH, et al. (2003) "American College of Endocrinology position statement on the insulin resistance syndrome." Endocrine Practice;9(3):237-252
8. International Diabetes Federation: "The IDF consensus worldwide definition of the metabolic syndrome." http://www.idf.org/metabolic-syndrome
9. Halberg N, Wernstedt-Astherholm, Scherer PE. (2008) "The adipocyte as an endocrine cell." Endocrinology and metabolism Clinics of North America;37(3):753-768
10. Cinti S, Mitchell G, Barbatelli G, et al. (2005) "Adipocyte death defines macrophage localization and function of adipose tissue in obese mice and humans." Journal of Lipid Research;46(11):2347-2355
11. Lau DCW, Dhillon B, Yan H, et al.(2005) "Adipokines: molecular links between obesity and atherosclerosis." The American Journal of Physiology-Heart and Circulatory Physiology;288(5):H2031-H2041
12. Trayhurn P and Wood SI. (2004) "Adipokines: inflammation and pleiotropic role of white adipose tissue." British Journal of Nutrition;92(3):347-355
13. Saleem U, Khaleghi M, Morgenthaler NG, et al. (2009) "Plasma carboxy-terminal provasopressin (copeptin): a novel marker of insulin resistance and metabolic syndrome." Journal of Clinical Endocrinology and Metabolism;94(7):25-58-25-64
14. Tsimikas S, Willeit J, Knoflach M, et al. (2009) "Lipoprotein-associated phospholipase A2 activity, ferritin levels, metabolic syndrome, and 10-year cardiovascular and non-cardiovascular mortality: results from the Bruneck study." European Heart Journal;30(1):107-115
15. Jacobs M, Van Grrevenbroek MMJ, Van Derr Kallen CJH, et al. (2009) " Low-grade inflammation can partly explain the association between the metabolic syndrome and either coronary artery disease or severity of peripheral arterial disease: the CODAM Study." European Journal of Clinical investigation;39(6):437-444
16. Bravata DM, Wells CK, Concato J, et al. (2004) "Two measures of insulin sensitivity provided similar information in a U.S. population." Journal of Clinical Epidemiology;57(11):1214-1217
17. Carey DG, Jenkins AB, Campbell LV, et al. (1996) "Abdominal fat and insulin resistance in normal and overweight women: direct measurements revealed a strong relationship in subjects at both low and high risk of NIDDM." Diabetes;45(5):633-638
18. Mayer-Davis EJ, D'Agostino R, Karter AG, et al. (1998) "Intensity and amount of physical activity in relation to insulin sensitivity the Insulin Resistance Atherosclerosis Study." Journal of the American Medical Association;279(9):669-674
19. Vessby B, Uusitupa M, Hermansen K, et al. (2002) "Substituting dietary saturated for monosaturated fat impairs insulin sensitivity in healthy men and women: the KANWU Study." Diabetologia;44(3)312-319
20. Gill H, Mugo M, Whaley-Connell C, et al. (2005) "The key role of insulin resistance in the cardiometabolic syndrome." The American Journal of the Medical sciences;330(6):290-294
21. Petersen KF and Shulman GI. (2006) "Etiology of insulin resistance." The American Journal of Medicine;119(5):S10-S16
22. Lewis GF and Steiner G. (1996) "Acute effects of insulin in the control of very low density lipoprotein production in humans: implications for the insulin-resistant state." Diabetes Care;19(4):390-393
23. Ginsberg HN, Zhang Y-L, Hernandez Ono A. (2005) "Regulation of plasma triglycerides in insulin resistance and diabetes." Archives of Medical Research;36(3):232-240
24. Ferrannini E, and Natali A. (1991) "Essential hypertension, metabolic disorders, and insulin resistance." The American Heart Journal;121(4):1274-1282
25. Malhotra A, Kang BPS, Cheung S. (2001) "Angiotensin II promotes glucose-induced activation of cardiac protein kinase C isoenzyme and phosphorylation of troponin I." Diabetes;50(8):1918-1926
26. Morse SA, Zhang R, Thakur V. (2005) "Hypertension and metabolic syndrome." The American Journal of the Medical Sciences;330(6):303-310
27. Briones AM, Cat AND, Callera GE, et al. (2012) "Adipocytes produce aldosterone through calcineurin-dependent signaling pathway: implications in diabetes mellitus-associated obesity and vascular dysfunction." Hypertension;59(5):1069-1078
28. Hallal P, Bertoldi D, Goncalves H, et al. (2006) "Prevalence of sedentary lifestyle and associated factors in adolescents 10 to 12 years of age." Cad Saudi Publica;22(6):1277-1287
29. Brownson RC, Boehmer TK, Luke DA (2005) "Declining rates of physical activity in the United states: What are the contributor?" Annu Rev Public Health;26:421-443
30. Borodulin K, Laatikainen T,Juolevi A, et al. (2008) "Thirty-year trends of physical activity in relation to age calendar time and birth cohort in Finnish adults." Eur J Public Health;18(3):339-344
31. Fang Zhou, Junfen Fu, Chun-lin wang, et al. (2010) "Relationship between sleep duration and metabolic syndrome in obese children." Zhonghua Liu Xing Bing Xue Za Zhi;31(4):455-458
32. Rosenheck R. (2008) "Fast food consumption and increased caloric intake: a systemic review of a trajectory towards weight gain and obesity risk." Obes Rev;9:535-547
33. Sacks FM, Bray GA, Garey VJ et al. (2009) "Composition of weight-loss diets with different composition of fat, protein , and carbohydrate." N Eng1 J Med;390(9):859-873
34. Munsch S and Beglinger C (2005) "Obesity and Binge Eating Disorders." Bip1 Psychiatr;171:62-73
35. Farooqi S and O'Rahilly S. (2006) "Genetics of obesity in humans." Endocr Fev;27(7):710-718
36. Singh R, Shaw J, Zimmet P. (2004) "Epidemiology of childhood type 2 diabetes in the developing world." Pediatric Diabetes;5:154-168
37. de Ferranti SD, Gauvereau K, Ludwig DS, et al. (2004) "Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutritional Examination Survey. Circulation;110:2494-2497
38. Weiss R, Dziura J, Burgert TS, et al. (2004) "Obesity and the metabolic syndrome in children and adolescents." N Engl J Med;350:2362-2374
39. Zimmet P, Alberti G, Kaufman F, et al. (2007) "The metabolic syndrome in children and adolescents." Lancet;369:2059-2061
40. Janseen I, Katzmarzyk PT, Srinivasan SR, et al. (2005) "Combined influence of body mass index and waist circumference on coronary heart disease risk factors among children and adolescents." Pediatrics;115(6):1623-1630
41. Ahrens W, Moreno LA, Marid S, et al. (2014) "Metabolic syndrome in young children: definitions and results of the IDEEICS Study." Int J Obes (Lond);38(Suppl 2):S4-s14
42. Bataloglu Inanc B. (2014) "Metabolic syndrome in school children in Mardin, South-Eastern of Turkey." Eurasian J Med.;46(3):156-163
43. Rizk N, Amin M, Yousef M. et al. (2011) "A pilot Study on metabolic syndrome and its associated features among Qatari school children." Int J Med.;4:521-525

44. Zaradast M, Mamakin K, Chahkandi T, et al. (2015) “Prevalence of metabolic syndrome in elementary school children in East Iran.” J Cardiovasc Thorac Res;7(4):158-163
45. Park Y-W, Zhu S, Palaniappan L, et al. (2003) “The metabolic syndrome: Prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988-1994. Archives of Internal Medicine;163(4):427-436
46. Eckel RH, Grundy SM, Zimmet PZ. (2005) “The metabolic syndrome.” Lancet;365:1415-1428
47. Rexrode KM, Garey VJ, Hennekens CH, et al. (1998) “Abdominal adiposity and coronary heart disease in women.” JAMA;208:1843-8
48. Taheri F, Namakin K, Zardast M, et al. (2012) “Cardiovascular Risk Factors: a Study on the Prevalence of Metabolic Syndrome among 11-18 years old school children in East of Iran.” Nutr Food Sci Res;2(1):27-34
49. Firek-Pedras M, Malecka-Tendra E, Klimek K, et al. (2006) “Influence of fat tissue distribution on metabolic complications on children and adolescents with simple obesity.” Endokrinol Diabetol Chor Przemiany Materii Wieku Rozw;12(1):19-24
50. Lingling Zhai, Jinhong lui, Jian Zhao et al. (2015) “Association of obesity with the onset of puberty and sex hormones in Chinese girls.” PLoS On;10(8)e0134656
51. Marcin Gierach, Joanna Gierach, Marlena Ewertowska, et al. (2014) “Correlation between body mass index and waist circumference in patients with metabolic syndrome” ISRN Endocrinology;2014:514589-514595
52. Marcovecchio ML, Mohn Angelika, Chiarelli Francesco. (2010) “Obesity and insulin resistance in children.” J Pediatr Gastroenterol Nutr;51(suppl 3):S149-S150
53. Wajchenberg BL. (2002) Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. “Endocr Rev;21:697-738
54. Shaibi GQ, Cruz ML, Weigensberg MJ, et al. (2007) “Adiponectin independently predicts metabolic syndrome in overweight latino youth.” J clin Endocrinol Metabo;92:1809-1813
55. Bacha F, Saad R, Gungor N et al. (2003) “Obesity regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabetogenic and atherogenic risk factors.” J Clin Endocinol Metabo;88:2534-2540
56. Taksali SE, Caprio S, Dziura J, et al. (2008) “High visceral and low abdominal subcutaneous fat stores in the obese adolescent: a determinant of an adverse metabolic phenotype.” Diabetes;57:367-371
57. Lee S, Bacha F, Gungor N, et al. (2006) “Waist circumference is an independent predictor of insulin resistance in black and white youth.” J Pediatr;148:188-194
58. Lee S, Bacha F, Arslanian SA. (2006) “Waist circumference, blood pressure, and lipid components of the metabolic syndrome.” J Pediatr;149:809-816
59. Zimmet P, Alberti G, Kaufman F, et al. (2007) “The metabolic syndrome in children and adolescents.” Lancet;369:2059-2061
60. Weiss R, Dufour S, Taksali SE, et al. (2003) “Prediabetes in obese youth: a syndrome of impaired glucose tolerance, severe insulin resistance, and altered myocellular and abdominal fat partitioning.” Lancet;362:951-957
61. Cali AM, De Oliveira AM, Kim H et al. (2009) “Glucose dysregulation and hepatic steatosis in obese adolescents: Is there any link?” Hepatology;49:1896-1903
62. Fecchini F, Chen YD, Holenbeck CB et al. (1991) “Relationship between resistance to insulin-mediated glucose uptake, urinary uric acid clearance, and plasma uric acid concentration.” JAMA1991;266:3008-3011
63. Dunaif A. (1997) “Insulin resistance and poly cystic ovary syndrome: mechanism and implications for pathogenesis.” Endocr Rev;18:774-800
64. Krauss RM and Siri PW. (2004) “Metabolic abnormalities: Triglyceride and low-density lipoprotein.” Endocrinol Metab Clin North Am;33:405-415
65. Guo SS, Wisemandle WA, Chumlea WC et al. (1998) “Serial changes in blood pressure from childhood into young adulthood for females in relation to body mass index and maturational age.” Am J Hum Biol.;10:589-598
66. Sun SS, Grave GD, Siervogel RM et al. (2007) “Systolic blood pressure in childhood predicts hypertension and metabolic syndrome later in life.” Pediatrics;119(2):237-246
67. Alberit G, Zimmet P, shaw J, et al. (2004) “Type 2 diabetes in the young: the evolving epidemic: the international Diabetes Federation Consensus work shop.” Diabetes Care;27:1798-1811
68. Bawazeer NM, Aldghri NM, Valsamakis G, et al. (2009) “Sleep duration and quality associated with obesity among Arab children.” Obesity;17(12):2251-2253
69. Sobal J. (1990) “Obesity and socioeconomic status: a framework for examining relationship between physical and social variables.” Med Anthropol;13(3):231-247
(70. Koletzko B. and Toschke AM. (2010) “Meals patterns and frequencies: do they affect body body weight in children and adolescents?” Crit Rev Food Sci Nutr; 50(2):100-105
71. Albuquerque D, Nobrega Clevio, Manco Licinio, et al. (2017) “The contribution of genetics and environment to obesity.” British Medical Bulletin; 123(1): 159-173