Original Article

The Prevalence of Metabolic Syndrome and Its Components among Overweight and Obese Nigerian Adolescents and Young Adults

CP Onyenekwu[1], AO Dada[1], OT Babatunde[2]

[1]Department of Chemical Pathology, Benjamin S. Carson (Snr) School of Medicine, Babcock University and Babcock University Teaching Hospital, Ilishan-Remo, Ogun State, [2]Department of Paediatrics, Benjamin S. Carson (Snr) School of Medicine, Babcock University and Babcock University Teaching Hospital, Ilishan-Remo, Ogun State, Nigeria

Abstract

Background: The incidence of metabolic syndrome (MetS), is rapidly increasing in developing countries. However, the epidemiology of MetS is not well reported in the pediatric and young adult population. We determined the prevalence of MetS and its components among overweight and obese Nigerian adolescents and young adults presenting for university admission. **Materials and Methods:** A cross-sectional study of overweight and obese adolescents and young adults was performed. Blood pressure readings were taken while participants were seated. Anthropometric measures of waist circumference, weight and height were also taken using standard protocols and the body mass index was computed thereafter. Venous blood for fasting plasma glucose, triglycerides and high density lipoprotein cholesterol was collected and assayed using standard laboratory methods. Metabolic syndrome was defined by the International Diabetes Federation criteria. Statistical significance was set at 0.05. **Results:** 91 individuals (18 males) aged 18.1 ± 4.85 years were studied. 13 (14.3%) of them had MetS and 11 (84.6%) of these were adolescents. Abdominal obesity was prevalent in 89 (97.8%) participants, hypertension was prevalent in 39 (42.9%) participants and hyperglycaemia was prevalent in 5 (5.5%) participants. Hypertriglyceridaemia was least prevalent in one (1.1%) participant who did not have MetS. All the participants who had hyperglycaemia (5.5%) had MetS. **Conclusions:** There is a high prevalence of MetS in obese and overweight Nigerian adolescents and young adults with the clustering of two components in half of the population. These findings have profound implications hence there is an urgent need to institute primary and secondary interventions in this population.

Keywords: *Adolescents, Metabolic Syndrome, Obese, Overweight, Young Adults*

Date of Acceptance:
30-Sep-2016

Introduction

There is a gradual rise in obesity amongst young people in developing countries.[1] Obesity plays a pivotal role in metabolic syndrome (MetS), a constellation of clinico-biochemical parameters that places an individual at increased risk for cardiovascular disease. The syndrome is characterized by the presence of any three or more of obesity, hyperglycaemia, hypertension, and atherogenic dyslipidaemia (hypertriglyceridaemia and/or hypoalphalipoproteinaemia).[2] It was first described nearly three decades ago to result from insulin resistance in adults,[3] but later reports suggested an occasional onset in utero.[4] A survey among adolescents in the United States reported a prevalence of MetS in more than one quarter of the obese population and nearly seven percent of the overweight population.[5] Previous literature has also highlighted the paucity of knowledge with regards to the prevalence of paediatric MetS, its criteria and clinical implications. The few reported studies utilized varying definitions and different age groups, thus making comparisons of study findings challenging.[6,7] To comprehend and better characterize the epidemiologic

Access this article online

Quick Response Code:

Website: www.njcponline.com

DOI: 10.4103/1119-3077.196085

Address for correspondence: Dr. CP Onyenekwu, Department of Chemical Pathology, Benjamin S. Carson (Snr) School of Medicine, Babcock University and Babcock University Teaching Hospital, Ilishan-Remo, Ogun State, Nigeria.
E-mail: chineloonyenekwu@gmail.com

How to cite this article: Onyenekwu CP, Dada AO, Babatunde OT. The prevalence of metabolic syndrome and its components among overweight and obese Nigerian adolescents and young adults. Niger J Clin Pract 2017;20:670-6.

trend in obesity, this study was performed to evaluate the prevalence of MetS as well as the prevalence of the individual components of the syndrome, in overweight and obese adolescents and young adults presenting for university degree admissions at a private tertiary institution in Nigeria.

Materials and Methods

Ethical considerations

The study was approved by the Health Research Ethics Committee of the university. An informed consent was obtained from each participant and/or the participant's parent or guardian for participants who were aged less than 18 years. The study was performed according to the Declarations of Helsinki (2013).

Study setting and population

The University is located in the South-Western Region of Nigeria. Each academic year, the institution admits individuals from different parts of the country, into various pre-degree, undergraduate and post-graduate programs. As part of the admission process, these individuals undergo a clinical and laboratory screening exercise at the university teaching hospital. The findings from the exercise are incorporated into each person's case file as his or her baseline medical records. The individuals presenting for the routine pre-admission screening for the 2015/2016 academic year were recruited at the family medicine outpatient department over a four-week period from August to September 2015.

Eligibility criteria included age less than 35 years; the presence of generalized obesity or overweight using the World Health Organization (WHO) body mass index (BMI) classification[8] and the updated International Obesity Task Force (IOTF) criteria for children and adolescents,[9] or the presence of abdominal obesity identified by waist circumference (WC) cut points using the International Diabetes Federation (IDF) criteria.[10] Individuals who were known diabetics, hypertensives or who had any known chronic medical conditions were excluded. Pregnant females were also excluded. Identified eligible individuals were consecutively recruited for participation and informed consent sought from same.

Data collection

Data on current medications and previous medical history were collected using an interviewer-administered questionnaire. The participants were then requested to undergo a 10-12 hour overnight fast for specimen collection at the laboratory, the following day.

Blood pressure (BP) readings were taken from the participant's left arm using an automated sphygmomanometer (Omron Medical, United Kingdom), after being seated for ten minutes. The reading was taken to the nearest mmHg.

The participants' weights and heights were measured using Gulfex scales (Gulfex Medical and Scientific, England). Each measurement was taken after nulling the scale to zero reading. The measurements were taken with participants standing barefooted, in light clothing with pockets emptied, headgears and excessive hair ornaments taken off, and their arms hanging naturally by the sides, while looking forwards. The weight reading was taken to the nearest 0.1 kilogram, while the height was taken to the nearest centimeter, with the buttocks, upper back or head touching the measuring surface of the rule.[11] As a measure of generalized obesity, each adult participant's BMI was computed by dividing the weight in kilograms, by the square of the height in meters (kg/m^2), while adolescents BMI were matched on the IOTF chart. To determine abdominal obesity, measurement of the WC was taken using a stretch-resistant tape (HTS, China). Each participant's WC measurement was taken from the zero mark on the tape rule. The tape rule was wrapped snugly, but not constrictingly, around the participant at a level parallel to the floor, midpoint between the top of the iliac crest and the lower margin of the last palpable rib in the mid axillary line. The reading was taken to the nearest centimeter at the end of expiration, after a few consecutive natural breaths.[12]

Specimen collection

Venous blood was collected at the hospital laboratory, following a 10-12 hour overnight fast. Using a multi-sample needle, blood was collected from the cubital fossa after antiseptic preparation of the venepuncture site. The blood for fasting plasma glucose (FPG) assay was collected into fluoride oxalate vacutainers while that for serum triglycerides (TG) and high density lipoprotein cholesterol (HDL-c) was collected into plain vacutainers and allowed to clot. Specimens were centrifuged at 4000 rpm for five minutes and the supernatant collected and stored at -20°C for a maximum of three months, prior to laboratory analyses.

Laboratory analyses

Plasma glucose was determined by the glucose oxidase method. Serum TG assay was performed using standard enzymatic methods. The HDL-c was determined by a two-step method using a precipitant to isolate the non-HDL-c components in the serum in the first step, and subsequent quantitative determination of the HDL-c by standard enzymatic methods for cholesterol determination. All three assays for FPG, TG and HDL-c were performed on the Sinnowa® Chemistry Analyzer

using reagents from Biolabo, France and quality control sera from Randox Laboratories, UK.

Definition of terms

Generalized Obesity

This was defined as a BMI ≥ 30 kg/m^{2}in participants ≥ 18 years older BMI exceeding cut points for age and gender using the international obesity task force (IOTF) criteria for overweight and obesity in children.[9]

Overweight

This was defined as a BMI 25.00-29.99 kg/m^2 in participants ≥ 18 years old[9] or BMI meeting criteria for overweight using the international obesity task force (IOTF) cut points for overweight and obesity in children and adolescents.[9]

Abdominal obesity

This was defined as a WC ≥ 80cm in females or ≥94 cm in male participants above 16 years of age or WC > 90th percentile for participants aged less than 16 years.[10]

Ideal BMI

This was defined as a BMI of 18.50 -24.99 kg/m^2 in participants aged ≥ 18 years or BMI within normal for age and gender using the IOTF criteria for overweight and obesity in children and adolescents.[9]

Metabolic syndrome

This was defined as the presence of at least three components of hyperglycaemia, abdominal obesity, elevated blood pressure, hypertriglyceridaemia and hypo alphalipoproteinaemia (low HDL-c) as defined by the IDF criteria for MetS [Table 1].[10]

Statistical analysis

Data were analyzed using IBM SPSS Statistics for Windows version 23.0. Armonk, NY: IBM Corp. Measures of location and variability were computed for continuous variables and reported as Mean ± standard deviation (SD). The prevalence of MetS was determined as the number of participants with three or more of the components of the IDF criteria for MetS divided by the total number of participants. The participants were further grouped into three classes based on their BMI - ideal BMI, overweight, and obese. One-way analysis of variance (ANOVA) was used to assess differences in age and MetS components across the three groups of participants with ideal BMI, those who were overweight and the obese participants. A Tukey post-hoc test was conducted for the MetS components to assess significant differences among the groups on ANOVA. For all the participants, the likelihood ratio for Metabolic Syndrome in the presence of each MetS component was also determined. Statistical significance level was set at p<0.05.

Results

A total of 1382 individuals presented for pre-admission screening during the four-week study period, 192 (13.9%) adolescents and young adults were identified to be overweight or have abdominal or generalized obesity. Following exclusion of 101 individuals as a result of incomplete data in 62 individuals who did not present subsequently for a fasting venous blood collection and 39 individuals who did not consent to blood collection for the purpose of the study, data from 91 individuals was analyzed. 76 (83.5%) participants were adolescents aged 14 -19 years old, 15 participants were young adults, aged 20-34 years old. 18 (19.8%) participants were males. 54 (59.3%) participants were obese, 26 (28.6%) were overweight, while 11 (12.1%) had an ideal BMI. All the participants with ideal BMI had abdominal obesity and were females. [Table 2] shows the age and clinico-biochemical characteristics of study participants in the obese, overweight and ideal BMI groups.

wOne-way ANOVA of age and various clinico-biochemical parameters of obese, overweight and ideal BMI participants showed statistically significant differences in the WC (p=0.000), SBP (p=0.003) and DBP (p=0.007) among the three groups. There were no significant differences in the age (p=0.311), HDL-c (p=0.781), TG (p=0.537) and FPG (p=0.494) distribution among these three groups [Table 2]. A Tukey post-hoc analysis demonstrated that the SBP of the obese participants was significantly higher than the overweight participants (p=0.019) and the participants with ideal BMI (p=0.017). Though the SBP of the overweight participants was higher than that of the participants with ideal BMI, the difference was not statistically significant (p=0.735). The DBP of the obese participants was statistically significantly higher than

Table 1: The International Diabetes Federation Consensus Definition for Metabolic Syndrome. Adapted from Zimmet *et al*.

Components/criteria	Age 10-16 years	Age > 16 years
Abdominal Obesity	WC ≥ 90th percentile or adult cut-off if lower	WC ≥ 94cm in males WC ≥ 80cm in females
Hypertension	SBP ≥ 130mmHg or DBP ≥ 85mmHg	SBP ≥ 130mmHg or DBP ≥ 85mmHg
Hyperglycaemia	FPG ≥ 5.6mmo/L	FPG ≥ 5.6 mmol/L
Dyslipidaemia	TG ≥ 1.7mmol/L and/or HDL-c <1.03mmol/L	TG ≥ 1.7mmol/L and/or HDL-c <1.03mmol/L in males or HDL-c <1.29 in females

WC=Waist circumference; SBP=Systolic blood pressure; DBP=Diastolic blood pressure; FPG=Fasting plasma glucose; TG=Triglycerides; HDL-c=High density lipoprotein cholesterol

Table 2: Clinico-biochemical Characteristics of Study Participants according to the Various Classes of Body Mass Index.

Variable	Ideal BMI	Overweight	Obese	*P* value
Age (years)	16.4 ± 0.81	17.6 ± 4.67	18.7 ± 5.34	0.311
WC (cm)	84.8 ± 3.28	90.3 ± 6.52	98.6 ± 9.98	0.000
SBP (mmHg)	117.2 ± 11.11	120.2 ± 11.07	127.4 ± 11.01	0.003
DBP (mmHg)	66.7 ± 10.63	69.1 ± 6.84	74.4 ± 9.42	0.007
FPG (mmol/L)	3.9 ± 0.55	4.2 ± 0.67	4.1 ± 0.82	0.494
HDL-c (mmol/L)	1.42 ± 0.31	1.37 ± 0.48	1.35 ± 0.33	0.781
TG (mmol/L)	0.56 ± 0.25	0.54 ± 0.19	0.61 ± 0.25	0.537

WC=Waist circumference; SBP=Systolic blood pressure; DBP=Diastolic blood pressure; FPG=Fasting plasma glucose; TG=Triglycerides; HDL-c=High density lipoprotein cholesterol; BMI: Body mass index

Table 3: Likelihood Ratios for Metabolic Syndrome in the Presence of Each Metabolic Syndrome Component in an Adolescent or Young Adult with Abdominal or General Obesity or an Overweight Body Mass Index.

IDF Metabolic Syndrome Component	Likelihood Ratio	Significance
Hypertension	11.29	0.001
Hyperglycaemia	11.99	0.001
Hypertriglyceridaemia	0.310	0.578
Hypoalphalipoproteinaemia	25.01	0.000

IDF=International Diabetes Federation

Table 4: Clinico-biochemical Characteristics of Study Participants with Metabolic Syndrome and those without Metabolic Syndrome.

Variable	Metabolic syndrome	No metabolic syndrome	p value
Age (yrs)	18.6 ± 5.94	18.0 ± 4.69	0.722
WC (cm)	101.6 ± 8.42	93.4 ± 9.68	0.005
BMI (kg/m^2)	34.11 ± 5.23	30.56 ± 5.33	0.038
SBP (mmHg)	131.8 ± 5.09	122.8 ± 11.97	0.000
DBP (mmHg)	75.7 ± 14.02	71.3 ± 8.27	0.294
FPG (mmol/l)	4.8 ± 1.33	4.0 ± 0.53	0.045
HDL-c (mmol/l)	1.00 ± 0.20	1.43 ± 0.36	0.000
TG (mmol/l)	0.66 ± 0.24	0.57 ± 0.23	0.238

WC=Waist circumference; SBP=Systolic blood pressure; DBP=Diastolic blood pressure; FPG=Fasting plasma glucose; HDL-c=High density lipoprotein cholesterol; TG=Triglycerides

that of the overweight participants (p=0.038) and the participants with ideal BMI (p=0.029). The DBP of the overweight participants was also higher than that of the participants with ideal BMI, however this difference was not statistically significant (p=0.745). Likewise, the WC of the obese participants was significantly higher than that of the overweight participants (p=0.000), and the participants with ideal BMI (p=0.000). Despite the fact that the WC of the overweight participants was higher than that of participants with ideal BMI, the observed difference was not statistically significant (p=0.181).

In the whole group, participants with low HDL-c levels were more likely to have MetS. The same associations were observed with hyperglycaemia and low HDL-c levels although there was no association between hypertriglyceridaemia and MetS. [Table 3] shows the likelihood ratios for MetS in the presence of each MetS component excluding abdominal obesity. The clinico-biochemical characteristics of study participants with MetS and those without MetS are depicted in [Table 4].

Discussion

Obesity is an alarming public health challenge of the 21st century.[13] The co-morbidities associated with it are rife and their consequences dire. Metabolic syndrome is one of the associated co-morbidities of obesity. Previous research works suggest that the occurrence of MetS in pre-adult life persists into adulthood and the existence of obesity in childhood predisposes an individual to developing MetS in adult life.[14,15] Adolescents and young adults constitute a major part of tomorrow's workforce, hence their health status is of paramount importance in an economy that is resource-limited, with low average life expectancy. We assessed overweight and obese adolescents and young adults of Nigerian origin, using the IDF criteria for MetS. We observed that one in seven overweight and obese adolescent and young adult had MetS. The most prevalent MetS component was abdominal obesity in 97.8% of the participants, followed by hypertension in over 40% of these individuals, low HDL-c levels in one third of the population and hyperglycaemia in about six percent of the population. Of noteworthy is the low prevalence of hypertriglyceridaemia, which was present in only one participant, this participant did not have MetS. Obese/overweight adolescents and young adults with low HDL-c were 25 times more likely to have MetS than those with desirable HDL-c levels. The participants with hyperglycaemia were 12 times more likely to have MetS than those without, while participants with hypertension were 11 times more likely to have MetS than those with normal blood pressure.

The prevalence of MetS in the present study is similar to the 14.4% reported in obese and overweight Japanese children.[16] The Japanese report utilized a criterion for MetS established for Japanese children. Our prevalence rate for MetS was lower than the rate of one in three observed in obese and overweight adolescents in the National Health and Nutrition Examination Survey (NHANES) in the US.[17] The differences observed in the prevalence of MetS in the US and the present study may be due to two reasons; first, the use of different criteria

for the definition of MetS. The NHANES study defined MetS using criteria analogous to the Adult Treatment Panel III, which had stricter dyslipidaemia, abdominal obesity and hypertension cut points.[17] A second reason may be due to environmental factors. Obesity and its co-morbidities were initially problems of industrialized nations but the incidence in developing countries have been noted to be on the increase.[13] Though the observed prevalence rates were different, a similar observation in the NHANES study and the present study is that none of the participants in the NHANES study had the five components of MetS. In another NHANES report in adults, 30% of overweight males and 65% of obese males had MetS while 33% of overweight females and 56% of obese females had MetS. The prevalence of MetS in young adult females aged 20-39 years was 16% and in males of same age group, 20%.[18]

Emerging data on MetS in Africa has been mainly from studies in older adults, individuals with type 2 diabetes mellitus or hypertension. Few studies have been conducted in persons aged less than 20 years.[19-21] There is no clear pattern in the frequency of the different components of MetS from previous studies in African adults.[20] In obese and overweight Lebanese children, using the ATP III criteria, hypertriglyceridaemia was observed in 91.7% and low HDL-c in 66.7% of them.[22] Among Indian children and adolescents, employing the ATP III age-modified criteria, the commonest components observed were abdominal obesity and hypertriglyceridaemia, while none of the participants had hypertension.[23] Whereas in the present study, only one participant had hypertriglyceridaemia and this participant did not have MetS. Furthermore, hypertension was the most common component of MetS in the present study, after abdominal obesity. The nearly 100% prevalence of abdominal obesity in the present study was expected as the study population was obese and overweight individuals and abdominal obesity was one of the criteria for eligibility for inclusion in the study. The variations in the frequency of the different components of MetS may be due to racial and environmental differences in the studied population. African origin is a known non-modifiable risk factor for hypertension,[24] while obesity also predisposes to hypertension. Thus, the common occurrence of hypertension in this population of overweight and obese individuals of African descent may be as a result of the simultaneous existence of these two predisposing factors in them. Hyperglycaemia was only prevalent in 5.5% of our study population but it is noteworthy that all the participants with hyperglycaemia had MetS. This is not surprising as one of the proposed mechanism underlying the pathophysiology of MetS is majorly centred on insulin resistance.[25]

Metabolic syndrome, which confers a high risk for cardiovascular disease is thought to be as a result of dysfunctional adipose tissue in a background of insulin resistance.[25] The syndrome causes target organ damage through its various components; hypertension leads to left ventricular hypertrophy, renal dysfunction and peripheral vascular disease; microvascular dysfunction further worsens insulin resistance and in turn, the hypertension that is already existent. It increases the risk for cardiovascular disease through increased oxidative stress, endothelial dysfunction, arterial wall stiffness and release of pro atherogenic cytokines.[26,27] Numerous complications are associated with MetS and some of them include coronary heart disease, ischaemic stroke, atrial fibrillation, non-alcoholic fatty liver disease and obstructive sleep apnoea.[28-30] Metabolic syndrome has also been implicated in various cancers.[31] The environmental and genetic variations, which affect the components of MetS are poorly-defined though the risks attributable to MetS are well understood. There is still a lack of understanding regarding the highly variable nature of MetS manifestations by age and ethnicity.[32] One of the challenges in epidemiology of MetS is the numerous criteria available for its definition. This challenge is more pronounced in the paediatric population where there are no standardized criteria or universal consensus for the definition of paediatric MetS,[33] making case finding and comparisons of epidemiological data very difficult.

The present study is limited by its cross-sectional nature, which prevents the determination of causality in the observations made. Another study limitation is the high rate of drop-outs, which occurred largely due to non-sampling of eligible individuals who were not fasting at the point of identification. Many of these individuals subsequently failed to present for a fasting venous sample collection for the laboratory analysis of glucose and triglycerides. Nevertheless, the distribution of the biophysical parameters (age, BMI, WC and BP) of the drop-outs are comparable to those of the study participants, hence we expect that the findings from the study are representative of the originally identified eligible sample population. Finally, the adolescent population studied was restricted to those aged 14 years and above, this was as a result of the age group of individuals presenting for university admission. Notwithstanding this limitation, the study utilized a population that is representative of the Nigerian population in the age group evaluated. It also employed international criteria for the assessment of obesity and overweight in these individuals.

In conclusion, our study showed a high prevalence of MetS in obese and overweight adolescents and young adults, with half of them having at least two components of MetS. Hypertension was the most prevalent component of MetS after abdominal obesity while hypertriglyceridaemia was very rare and not associated with MetS. The immediate and remote implications of these findings are staggering. There may be a need to ensure that BP measurements are performed routinely as part of the physical examination of the pediatric patient during clinic visits for early identification of hypertension in them. Pediatricians, family physicians, and general practitioners who constitute the first point of care for these individuals, need to be aware of the clustering of the components of MetS in obese and overweight persons. These care givers should endeavour to screen such persons on encounter, and institute urgent preventive and risk-reduction strategies.

Acknowledgements

The authors are grateful to the doctors and nursing staff of the Department of Family Medicine for their assistance with the identification of eligible study participants. We are also grateful to Dr. Gbadebo AA and Dr. Oshunbade AA for their help with data acquisition.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Poobalan A, Aucott L. Obesity among young adults in developing countries. A systematic overview. Curr Obes Rep 2016;5:2-3.
2. Sinaiko AR, Jacobs DR, JrSteinberger J, Moran A, Leupker R, Rocchini AP, *et al.* Insulin resistance syndrome in childhood: associations of the euglycemic insulin clamp and fasting insulin with fatness and other risk factors. J Pediatr 2001;139:700-7.
3. Reaven GM. Banting Lecture 1988: role of insulin resistance in human disease. Diabetes 1988;37:1595-607.
4. Ozanne SE, Hales CN. Early programming of glucose-insulin metabolism. Trends Endocrinol Metab 2002;13:368-73.
5. Levitt NS, Lambert EV. The foetal origins of the metabolic syndrome - a South African perspective. Cardiovasc J S Africa 2002;13:179-80.
6. 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. Arch Pediatr Adolesc Med 2003;157:821-27.
7. Lee S, Bacha F, Gungor N, Arslanian S. Comparison of different definitions of pediatric metabolic syndrome: relation to abdominal adiposity, insulin resistance, adiponectin, and inflammatorybiomarkers. J Pediatr 2008;152:177-84.
8. World Health Organization Obesity and overweight fact sheet. Available from http://www.who.int/mediacentre/factsheets/fs311/en/. [Last accessed on 2015 June 20].
9. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes 2012;7:284-94.
10. Zimmet P, Alberti G, Kaufman F, Tajima N, Arslanian S, Wong G, *et al.* International Diabetes Federation Task Force on Epidemiology and Prevention of Diabetes. The metabolic syndrome in children and adolescents. Lancet 2007;369:2059-61.
11. World Health Organization. WHO child growth standards: training course on child growth assessment. Geneva. World Health Organization 2008.
12. World Health Organization. Waist circumference and waist-hip ratio: Report of a WHO expert consultation. Geneva, 8-11 Dec 2008; 20-21. Geneva. World Health Organization 2011.
13. Popkin BM, Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. Int J Obes Relat Metab Disord 2004;28: [Suppl 3] S2-9.
14. Vanhala MJ, Vanhala PT, Keinänen-Kiukaanniemi SM, Kumpusalo EA, Takala JK. Relative weight gain and obesity as a child predict metabolic syndrome as an adult. Int J Obes Relat Metab Disord 1999;23:656-9.
15. Katzmarzyk PT, Pérusse L, Malina RM, Bergeron J, Després JP, Bouchard C. Stability of indicators of the metabolic syndrome from childhood and adolescence to young adulthood: the Québec Family Study. J Clin Epidemiol 2001;54:190-5.
16. Yoshinaga M, Tanaka S, Shimago A, Sameshima K, Nishi J, Nomura Y, *et al.* Metabolic syndrome in overweight and obese Japanese children. Obes Res 2005;13:1135-40.
17. de Ferranti SD, Gauvreau K, Ludwig DS, Neufeld EJ, Newburger JW, Rifai N. Prevalence of the metabolic syndrome in American adolescents. Findings from the third National Health and Nutrition Examination Survey. Circulation 2004;110:2494-7.
18. Ervin RB. Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003-2006. Natl Health Stat Report 2009:1-7.
19. Okafor CI. The metabolic syndrome in Africa: current trends. Indian J Endocrinol Metab 2012;16:56-66.
20. Udenze IC, Azinge EC, Arikawe AP, Egbuagha EU, Onyenekwu C, Ayodele O. The prevalence of metabolic syndrome in persons with type 2 diabetes at the Lagos University Teaching Hospital, Lagos, Nigeria. West Afr J Med 2013;32:126-32.
21. Oguoma VM, Nwose EU, Ulasi II, Akintunde AA, Chukwukelu EE, Araoye MA, *et al.* Maximum accuracy obesity indices for screening metabolic syndrome in Nigeria: A consolidated analysis of four cross-sectional studies. Diabetes Metab Syndr 2016;10:121-7.
22. Nasreddine L, Ouaijan K, Mansour M, Adra N, Sinno D, Hwalla N. Metabolic syndrome and insulin resistance in obese prepubertal children in Lebanon: A primary health concern. Ann Nutr Metab 2010;57:135-42.
23. Andrabi SMS, Bhat MH, Andrabi SRS, Kamili MMA, Imran A, Nisar I, *et al.* Prevalence of metabolic syndrome in 8-18-year-old school-going children of Srinagar city of Kashmir India. Indian J Endocrinol Metab 2013;17:95-100.
24. Wang X, Poole JC, Treiber FA, Harshfield GA, Hanevold CD, Snieder H. Ethnic and gender differences in ambulatory blood pressure trajectories. Results from a 15-year longitudinal study in youth and young adults. Circulation 2006;114:2780-87.
25. Goossens GH. The role of adipose tissue dysfunction in the pathogenesis of obesity-related insulin resistance. Physiol Behav 2008;94:206-18.
26. Alessi MC, Juhan-Vague I. Metabolic syndrome, haemostasis and thrombosis. Thromb Haemost 2008;99:995-1000.
27. Elnakish MT, Hassanain HH, Janssen PM, Angelos MG, Khan M. Emerging role of oxidative stress in metabolic syndrome and cardiovascular diseases: important role of Rac/NADPH oxidase. J Pathol 2013;231:290-300.

28. Watanabe H, Tanabe N, Watanabe T, Darbar D, Roden DM, Sasaki S. Metabolic syndrome and risk of development of atrial fibrillation: the Niigata preventive medicine study. Circulation 2008;117:1255-60.
29. Tarantino G, Finelli C. What about non-alcoholic fatty liver disease as a new criterion to define metabolic syndrome?. World J Gastroenterol 2013;19:3375-84.
30. Tasali E, Ip MSM. Obstructive sleep apnea and metabolic syndrome. Proc Am Thorac Soc 2008;5:207-17.
31. Esposito K, Chiodini P, Colao A, Lenzi A, Giugliano D. Metabolic syndrome and risk for cancer: a systematic review and meta-analysis. Diabetes Care 2012;35:2402-11.
32. Steinberger J, Daniels SR, Eckel RH, Hayman L, Lustig RH, McCrindle B, *et al*. Progress and challenges in metabolic syndrome in children and adolescents: a scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee of the Council on Cardiovascular Disease in the Young; Council on Cardiovascular Nursing; and Council on Nutrition, Physical Activity, and Metabolism. Circulation 2009;119:628-47.
33. Weiss R. Childhood metabolic syndrome. Must we define it to deal with it?. Diabetes Care 2011;34:[Suppl 2]S171-6.