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Original article

Abdominal obesity, metabolic dysfunction, and metabolic syndrome in U.S. adolescents: National Health and Nutrition Examination Survey 2011–2016

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A R T I C L E I N F O

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A B S T R A C T

Purpose: The objectives were to use National Health and Nutrition Examination Survey data to (1) estimate the prevalence of metabolic syndrome (MetS) risk factors (elevated blood pressure, triglycerides, blood glucose, and low HDL cholesterol); (2) estimate the prevalence of MetS using three common definitions; and (3) compare the odds of MetS risk factors/MetS when using different measures of abdominal obesity (sagittal abdominal diameter [SAD] versus waist circumference [WC]) among U.S. adolescents.
Methods: Analyses were performed on data collected from adolescents aged 12–19 years ($n = 1214$) participating in the 2011–2016 National Health and Nutrition Examination Survey. Prevalence of MetS risk factors and MetS were estimated. Unadjusted and adjusted binomial/multinomial logistic regressions were performed to test associations between WC and SAD z-scores and MetS risk factors/MetS. Analyses were performed for all participants and were stratified by sex as well as race/ethnicity.
Results: Males were more likely to have MetS risk factors. Depending on sex and the definition applied, the prevalence of MetS ranged from 2% to 11% and was lowest among females. Adjusted logistic regressions showed that one z-score increase in SAD and WC resulted in similar increased odds of MetS risk factors/MetS, but associations between abdominal obesity and MetS varied by the definition applied and race/ethnicity.
Conclusions: Metabolic dysfunction and MetS are prevalent among U.S. adolescents, and it is important to consider how MetS components and MetS are measured in population inference.

Introduction

The prevalence of adolescent (ages 12–19 years) obesity (body mass index [BMI] > 95th percentile for age and sex) almost doubled from approximately 11% in 1988–1989 to 21% in 2013–2014 and has remained stable in recent years (2007–2008 to 2015–2016) [1,2]. Obesity is hypothesized to be one of the predominant underlying causes of metabolic syndrome (MetS) [3]. Concomitant with the dramatic rise in obesity, children and adolescents are manifesting metabolic dysfunction at younger ages, making MetS a condition of concern for all life stages [4–10]. MetS is a cluster of risk factors including abdominal obesity, hypertension, dyslipidemia, and elevated glucose [7,11]. MetS often accompanies insulin resistance and is a strong predictor of both type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) [3,7,11,12]. Adolescents exhibiting MetS risk factors and MetS at younger ages could have decreased quality of life and premature development of T2DM and CVD as they transition to young and middle adulthood [13]. Therefore, it is of extreme importance to promote early interventions before chronic disease development. Recent studies suggest that there are several risk factors for MetS incidence including low socioeconomic status, parental history of CVD, health behaviors like short sleep duration and poor nutrition, and childhood obesity [10,14–16]. Obesity is a discernible MetS risk factor that could assist with identification of adolescents who may require further assessment for other MetS risk factors and MetS.

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Abdominal obesity or visceral fat storage is a strong yet modifiable risk factor for poor cardiometabolic health and an underlying cause of MetS [17]. Waist circumference (WC), although not the gold standard of assessing visceral fat, is conventionally used as the predominant measure of abdominal obesity across different definitions of MetS. However, with use of WC, there is an inability to distinguish between abdominal subcutaneous and visceral fats [18], with visceral fat being the detrimental fat surrounding the organs. Some research suggests that the less commonly collected sagittal abdominal diameter (SAD), the height of the abdomen when lying supine, overcomes the limitations of WC and may be a better predictor of T2DM and CVD by measuring visceral fat [19–21]. For example, in a nationally representative, longitudinal study of Finnish adults, researchers found that SAD was a stronger predictor of incident diabetes than WC and suggested that SAD/BMI measurements could be an improved predictor of T2DM incidence [20]. SAD could also be a stronger predictor for MetS; however, predictive ability may be hard to distinguish because MetS is measured in various ways resulting in different estimated prevalence of MetS [4,5,22].

To our knowledge, there is one cross-sectional study by Weber et al. that explored this association among adolescents [23]. They found that SAD was highly correlated with cardiometabolic risk factors, but SAD was not more strongly associated with MetS than other measures of abdominal obesity. However, this study was limited by a small sample size, nongeneralizability to the U.S. population, and use of only one definition of MetS despite the common use of different MetS definitions in the pediatric population.

The objectives of this cross-sectional analysis of National Health and Nutrition Examination Survey (NHANES) data were to (1) estimate the prevalence of individual risk factors for MetS; (2) estimate and compare the prevalence of MetS applying three commonly used definitions (Cook et al. [4], International Diabetes Federation [IDF] [22], and de Ferranti et al. [5]); and (3) assess whether the risk of MetS risk factors and MetS vary by measure of abdominal obesity (SAD vs. WC).

Methods

NHANES data

NHANES data for the years 2011–2016 were used for this analysis because measurement for SAD began in the 2011–2012 cycle. NHANES is an annually administered, cross-sectional survey that uses a complex, multistage probability sampling design and calculates complex weighting schemes to ensure representativeness of the resident, noninstitutionalized U.S. population [24]. A brief description of the NHANES survey protocol is described below and has previously been published [24]. NHANES protocol involved selection of individuals for home interviews and physical examinations in mobile examination centers. Individuals who participated in morning exams fasted before examination and biologic specimen collection [25]. Our analyses use data from individuals who fasted at least 8 hours before examination and had viable blood samples. The National Center for Health Statistics Research Ethics Review Board reviewed and approved NHANES protocols.

Study population

A total of 1848 adolescents participated in the morning fasting examination. NHANES weighting procedures include assigning an individual a weight of zero if necessary to maintain representativeness of the noninstitutionalized U.S. population [24]. Therefore, participants with a fasting examination weight value of zero were excluded because their data would not contribute to final models ($n = 324$). Among 1524 participants with nonzero weights, exclusion criteria included fasting less than 8 hours before examination ($n = 0$); previously diagnosed diabetes or current use of glucose regulating medication ($n = 8$) and missing values for exclusion criteria ($n = 4$), the MetS components ($n = 120$), or covariates included in final models ($n = 221$). Exclusion criteria were not mutually exclusive; therefore, some adolescents met more than one of the exclusion criteria. After exclusions, our analytic sample consisted of 1214 adolescents aged 12–19 years (615 males and 599 females). Except for higher prevalence of fasting blood glucose (FBG) ≥126 mg per dL among excluded participants compared to included participants (2.3% vs. 0.077%), included and excluded participants had similar characteristics (Supplemental Table 1).

Individual risk factors for MetS

Examination data

NHANES procedures to collect anthropometry and blood pressure (BP) are described elsewhere [26,27]. Anthropometry measures relevant to this analysis included WC (centimeters), SAD (centimeters), and weight (kilograms) and height (meters) that were used for the calculation of BMI. BMI categories were created based on age- and sex-specific percentiles provided by Centers for Disease Control and Prevention 2000 growth charts and included underweight/normal (BMI <85th percentile), overweight (85th ≤ BMI <95th percentile), and obesity (BMI ≥95th percentile) [28]. Between one and three BP measurements were taken [27]. We averaged BP measurements and classified adolescents' BP as normal (aged 12 years: ≤113/75 mm Hg [males], ≤114/75 mm Hg [females]; aged 13–19 years ≤ 120/80 mm Hg [all]) or elevated (aged 12 years: >113/75 mm Hg [males], >114/75 mm Hg [females]; aged 13–19 years > 120/80 mm Hg [all]) based on recent guidelines for BP screening among children and adolescents [29].

Laboratory data

NHANES laboratories measured fasting triglycerides, high-density lipoprotein (HDL) cholesterol, and FBG through assays of blood plasma [30]. Based on National Heart, Lung, and Blood Institute recommendations, we classified adolescents as having normal (<90 mg per dL), borderline (90–129 mg per dL), or high (≥130 mg per dL) triglycerides, and normal (>45 mg per dL), borderline (40–45 mg per dL), or low (<40 mg per dL) HDL cholesterol [31]. We classified FBG as normal (0–99 mg per dL), prediabetes (100–125 mg per dL), or diabetes (≥126 mg per dL) based on American Diabetes Association and American Academy of Pediatrics guidelines [32,33].

Common definitions of metabolic syndrome

We used three common definitions to define MetS—Cook et al. [4], de Ferranti et al. [5], and the IDF [22]. Cook et al. and de Ferranti et al. definitions are modified versions of the National Cholesterol Education Program Adult Treatment Panel III definition for adolescents [4,5,34]. The IDF definition is a simplified, unified global definition for MetS in children and adolescents [3,22]. The categorical thresholds for the individual risk factors (i.e., abdominal obesity, elevated BP, high triglycerides, low HDL, and high FBG) are listed in Supplemental Table 2. Each definition requires an individual to have at least three individual risk factors to meet the criteria for MetS. For the IDF definition, one of the three risk factors must be abdominal obesity (defined as WC ≥ 90th age- and sex-specific percentiles for adolescents aged 12–15 years and WC > 102 cm [males] or 88 cm [females] for adolescents aged 16–19 years).

Covariates

A priori covariates were chosen based on prior literature suggesting independent associations between each covariate and MetS

[10,14–16]. All covariates were self-reported or reported by a parent/caregiver. Covariates included race/ethnicity (Hispanic/Latino, non-Hispanic (NH) white, NH black, other), family income-to-poverty ratio which is used to determine family's eligibility for federal programs like the Supplemental Nutrition Assistance Program [35], age (years), average time spent in sedentary activity per day (hours), total caloric intake (kilocalories (kcal)), and physical activity (meets physical activity guidelines [≥60 minutes/day for adolescents aged 12–17 years and ≥150 minutes/week for adolescents aged 18–19 years] vs. does not meet [<60 minutes/day (12–17 years) and <150 minutes/week (18–19 years)]) [36]. Sedentary activity was calculated based on participant responses to average time spent sitting but not spent sleeping during a typical day. To calculate total caloric intake, we averaged the total caloric consumption from two 24-hour dietary recall interviews. Physical activity was calculated based on reports of time spent engaging in walking, bicycling, and recreational moderate or vigorous activity during a typical week.

Statistical analysis

To account for the complex sampling design of NHANES, estimates and standard errors (SEs) for all analyses were calculated by using appropriate fasting subsample weights. All analyses were completed for the total population and were stratified by sex. Frequencies, percentages, and means of demographic characteristics, SAD and WC, and covariates were calculated. We estimated the prevalence of each individual risk factor for MetS and of MetS using the categorizations and criteria identified previously and performed Rao-Scott χ^2 tests for sex differences. We calculated sex- and age (years)-specific z-scores of continuous WC and SAD to measure these parameters on the same scale. By using z-score standardization, a one-unit change in the z-score equally corresponds to the number of standard deviations (SDs) an individual's abdominal obesity measurement was from the mean for each parameter. To test the associations between WC and SAD z-scores and risk factors for MetS/MetS, we performed unadjusted binomial or multinomial logistic regressions models and subsequently adjusted for *a priori* covariates. We also sought to determine whether associations between abdominal obesity measures and risk factors for MetS/MetS varied by race/ethnicity. Therefore, we added abdominal obesity*race/ethnicity interaction terms to adjusted models and stratified the models by race/ethnicity if interaction terms were statistically significant. We limited the investigation of interaction by race/ethnicity to Hispanic/Latino, NH white, and NH black participants due to within-group racial/ethnic heterogeneity among participants of other races/ethnicities. We did not adjust for multiple comparisons due to the exploratory nature of this analysis. All analyses were conducted with SAS, Version 9.4 (SAS Institute, Cary, NC) using survey procedures (i.e., PROC SURVEYMEANS, PROC SURVEYFREQ, PROC SURVEYLOGISTIC). A two-sided *P*-value less than .05 was used for statistical significance.

Sensitivity analysis

Weight, stature, and metabolic risk factors are associated with puberty [37,38]; however, pubertal maturation data were not available for all participants. As a sensitivity analysis, we adjusted for self-reported age of menarche among girls in additional regression models.

Results

Study population characteristics

Approximately half of the sample were male and were between the ages of 12 and 15 years (Table 1). About 37% of adolescents were overweight or obese with average WC of 83 cm (SE 0.62) and SAD of 18 cm (SE 0.15). Overweight/obesity, WC, and SAD did not vary by sex ($P = .64$, $P = .32$, $P = .16$, respectively). Females were less likely to meet physical activity guidelines than males (52% vs. 66%, respectively; $P < .01$). Males consumed a greater number of calories per day compared to females (2.2 kcal vs. 1.7 kcal, respectively; $P < .01$).

Prevalence of individual risk factors for MetS and MetS

There were sex differences in the prevalence of MetS risk factors and MetS (Table 2). Males were more likely to have elevated BP, high triglycerides, FBG in the prediabetes/diabetes range, and low HDL cholesterol compared to females. Overall, borderline/low HDL cholesterol was the most prevalent MetS risk factor affecting approximately 36% of males and 25% of females. Although FBG suggestive of diabetes was rare, 31% of males and 13% of females had FBG in the prediabetes range. The prevalence of MetS varied by sex and MetS definition. Among all participants, the MetS prevalence ranged from 2% (Cook et al. among females) to 11% (de Ferranti et al. among males), and prevalence was lowest among females (range: 2%–9%).

Comparison of associations between measures of abdominal obesity and MetS risk factors/MetS

Unadjusted binomial and multinomial logistic regressions showed similar increases in the odds of MetS risk factors and MetS per one-SD increase in SAD and WC (Table 3). For BP and FBG, we created dichotomized categorizes due to low prevalence in the highest risk categories and tested associations between abdominal obesity and these outcomes with binomial logistic regression. Neither SAD nor WC was associated with high FBG among females before adjustment (odds ratios: 1.35, 95% confidence interval [CI]: [0.96, 1.88] and 1.29 [0.94, 1.77], respectively). Increases in SAD and WC z-scores were associated with similarly higher odds of MetS than the higher odds associated with individual risk factors alone.

After adjustment for confounders, results were comparable to results of the unadjusted models: each z-score increase in SAD and WC resulted in similar increased odds of MetS risk factors (Table 4). Associations for males and females were similar in magnitude. However, models for the Cook et al. definition of MetS were inestimable for females after adjustment. After adjustment for demographic characteristics, physical activity, sedentary behavior, and total caloric intake and compared to unadjusted results, the odds of borderline/high FBG associated with abdominal obesity remained approximately the same for males (adjusted odds ratio: SAD - 1.53 [1.23, 1.90] and WC - 1.65 [1.24, 1.97]) and remained unassociated with either SAD or WC for females Table 4.

Among all participants, higher z-scores of both measures of abdominal obesity were associated with similar higher odds of MetS across definitions. Although each measure of abdominal obesity was similarly associated with higher odds of MetS, a one SD increase in abdominal obesity was associated with approximately eight-fold higher odds of Cook et al.'s MetS and over ten-fold higher odds of IDF MetS compared to approximately five-fold higher odds of de Ferranti et al.'s MetS.

Associations between each abdominal obesity measure and IDF MetS varied by race/ethnicity among all participants (Supplemental Table 3). Positive associations between higher z-scores of abdominal obesity and IDF MetS were stronger among non-Hispanic white participants (SAD OR = 12.6 [5.89, 27.1], WC OR = 13.7 [5.22, 35.9] compared to their Hispanic/Latino (SAD OR = 4.84 [3.73, 6.29], WC OR = 4.46 [3.25, 6.13] and black counterparts (SAD OR = 5.31 [3.00, 9.38], WC OR = 4.56 [2.78, 7.4]) (Table 5). Results of the sensitivity analysis showed that associations among girls did not change with adjustment for age of first menses (Supplemental Table 4).

Table 1
Weighted population characteristics of U.S. adolescents aged 12–19 years (NHANES 2011–2016), $N = 1214$

Population characteristics	All ($N = 1214$)		Males ($n = 615$)		Females ($n = 599$)		P
	N	% or mean (SE)	n	% or mean (SE)	n	% or mean (SE)	
Age (years)							.15
12–15	619	50.3 (1.90)	304	47.7 (2.86)	315	53.2 (2.51)	
16–19	595	49.7 (1.90)	311	52.3 (2.869)	284	46.8 (2.51)	
Race/ethnicity							.91
Hispanic (of any race)	382	21.6 (2.28)	189	21.8 (2.45)	193	21.5 (2.57)	
Non-Hispanic white	329	55.9 (3.04)	169	56.4 (3.37)	160	54.2 (4.42)	
Non-Hispanic black	316	14.3 (1.71)	174	13.9 (1.94)	142	14.2 (1.96)	
Other (including multiracial)	187	8.46 (1.13)	83	7.92 (1.29)	74	9.05 (1.58)	
Annual family income							.72
$0 to $24,999	385	23.3 (2.29)	201	23.6 (2.86)	184	22.9 (2.55)	
$25,000-$44,999	309	20.3 (1.47)	155	19.3 (2.28)	154	21.4 (2.46)	
$45,000-$74,999	203	20.3 (1.96)	90	18.9 (2.31)	113	21.8 (2.78)	
$75,000-$99,999	123	14.1 (1.82)	65	13.9 (2.38)	58	14.2 (2.704)	
$100,000 and Over	194	22.2 (2.43)	104	24.3 (3.01)	90	19.8 (3.02)	
Body mass index (BMI), kg/m^2*							.64
Under/normal weight (BMI <85th percentile)	744	62.5 (1.95)	379	63.7 (2.64)	365	61.1 (2.62)	
Overweight (85th $\leq$ BMI <95th percentile)	196	15.8 (1.45)	92	14.7 (1.93)	104	17.1 (1.91)	
Obese (BMI $\geq$95th percentile)	273	21.7 (1.60)	144	21.6 (2.28)	129	21.8 (2.11)	
Meets physical activity guidelines†							<.01
Yes	691	59.2 (1.51)	399	65.8 (1.95)	292	52.0 (2.43)	
No	523	40.8 (1.51)	216	34.2 (1.95)	307	48.0 (2.43)	
Experienced menarche (among females, yes)					571	94.6 (1.19)	
Age (years), mean (SE)					570	12.1 (0.0728)	
	Mean (SE)		Mean (SE)		Mean (SE)		
Family income-to-poverty ratio	2.41 (0.100)		2.44 (0.129)		2.37 (0.118)		.60
Waist circumference, cm	82.6 (0.617)		83.1 0.915		82.0 (0.689)		.32
Sagittal abdominal diameter, cm	18.3 (0.148)		18.5 0.213		18.2 (0.154)		.16
Total caloric intake, kcal/day	1.96 (0.0369)		2.20 0.0543		1.69 (0.0307)		<.01
Average time sedentary per day, hours	8.11 (0.134)		7.91 0.163		8.33 (0.182)		.064

P-value corresponds to Rao-Scott χ^2 test or t test for differences between males and females.
* Percentiles are age- and sex-specific. Less than 1% missing for BMI and age of menarche.
† Meets physical activity guidelines—For adolescents aged 12–17 years, meeting guidelines corresponds to 60 minutes of moderate or vigorous physical activity per day. For adolescents aged 18–19 years, meeting guidelines corresponds to at least 150 minutes of moderate or 75 minutes of vigorous physical activity per week.

Discussion

Using NHANES data, we estimated the prevalence of MetS risk factors, compared three commonly used MetS definitions, and assessed whether the odds of MetS and MetS individual risk factors varied by measure of abdominal obesity (SAD vs. WC) among adolescents. We found that MetS risk factors, including elevated FBG were prevalent, especially among adolescent males, and that the estimated MetS prevalence had a wide range that depended on sex and the definition applied. Use of the Cook et al.'s and the IDF

Table 2
Weighted prevalence of risk factors for metabolic syndrome among U.S. adolescents aged 12–19 years (NHANES 2011–2016), $N = 1214$

Risk factors for metabolic syndrome and definitions of metabolic syndrome	All, $n = 1214$	Males, $n = 615$	Females, $n = 599$	P
	% (SE)	% (SE)	% (SE)	
Blood pressure *				<.01
Normal	84.9 (1.61)	78.2 (3.21)	92.3 (1.21)	
Elevated	15.1 (1.61)	21.8 (3.21)	7.75 (1.21)	
Triglycerides				.03
Normal (<90 mg per dL)	73.9 (1.86)	70.8 (2.84)	77.3 (2.04)	
Borderline (90–129 mg per dL)	16.0 (1.58)	16.6 (2.44)	15.4 (1.50)	
High ($\geq$130 mg per dL)	10.1 (1.18)	12.7 (2.00)	7.22 (1.22)	
High-density lipoprotein cholesterol				<.01
Normal (>45 mg per dL)	69.4 (1.44)	64.0 (1.90)	75.3 (2.38)	
Borderline (40–45 mg per dL)	17.0 (1.19)	17.5 (1.80)	16.4 (1.96)	
Low (<40 mg per dL)	13.6 (1.35)	18.5 (2.01)	8.23 (1.37)	
Fasting blood glucose				<.01
Normal (0–99 mg per dL)	77.0 (1.99)	68.4 (2.79)	86.5 (1.80)	
Prediabetes (100–125 mg per dL)	22.9 (1.99)	31.4 (2.82)	13.4 (1.74)	
Diabetes ($\geq$126 mg per dL)	0.163 (0.0921)	0.217 (0.153)	0.103 (0.102)	
Metabolic syndrome				
Cook et al. [4]	3.70 (0.668)	5.09 (1.13)	2.17 (0.762)	.04
de Ferranti et al. [5]	10.1 (1.24)	11.4 (1.73)	8.63 (1.53)	.19
International Diabetes Federation [22]	4.24 (0.892)	6.04 (1.59)	2.28 (0.611)	<.01

P-value corresponds to the Rao Scott χ^2 test for differences between males and females.
* Blood pressure defined as normal ($\leq$113/75 mm Hg [males aged 12 years], $\leq$114/75 mm Hg [females aged 12 years], and $\leq$120/80 mm Hg [all aged $\geq$13 years]), and elevated (>113/75 mm Hg [males aged 12 years], >114/75 mm Hg [females aged 12 years], and >120/80 mm Hg [all aged $\geq$13 years].

Table 3
Unadjusted odds ratios of metabolic syndrome risk factors and MetS among included adolescents, NHANES (2011–2016), $N = 1214$

Measures of abdominal obesity	OR (95% CI)								
	Blood pressure*,†	Triglycerides‡ (reference: <90 mg per dL)		High-density lipoprotein Cholesterol‡ (reference: >45 mg per dL)		Fasting blood glucose* (reference: 0–99 mg per dL)	MetS*		
	Elevated	Borderline (90–129 mg per dL)	High (≥130 mg per dL)	Borderline (40–45 mg per dL)	Low (<40 mg per dL)	Borderline/high (≥100 mg/dL)	Cook et al. [4]	de Ferranti et al. [5]	IDF [22]
All									
SAD z-score	**1.70 (1.44,2.01)**	**1.70 (1.32,2.17)**	**2.45 (1.95,3.07)**	**1.98 (1.60,2.47)**	**2.74 (2.11,3.55)**	**1.46 (1.22,1.75)**	**6.77 (4.72,9.73)**	**4.02 (3.23,5.01)**	**5.61 (4.16,7.80)**
WC z-score	**1.64 (1.40,1.92)**	**1.73 (1.35,2.21)**	**2.47 (2.01,3.03)**	**2.09 (1.67,2.63)**	**2.82 (2.16,3.69)**	**1.46 (1.22,1.77)**	**6.41 (4.30,9.55)**	**4.16 (3.25,5.32)**	**5.53 (4.08,7.51)**
Males, $n = 615$									
SAD z-score	**1.66 (1.34,2.04)**	**1.91 (1.34, 2.71)**	**2.58 (1.95,3.41)**	**1.96 (1.52,2.53)**	**3.1 (2.26,4.34)**	**1.53 (1.24,1.88)**	**7.26 (4.19,12.58)**	**4.26 (3.28,5.52)**	**6.79 (3.96,11.66)**
WC z-score	**1.65 (1.36, 1.99)**	**1.93 (1.37,2.74)**	**2.67 (2.01,3.53)**	**1.99 (1.50,2.64)**	**3.13 (2.23,4.39)**	**1.57 (1.26,1.97)**	**7.03 (4.18,11.81)**	**4.52 (3.50,5.85)**	**7.96 (4.65,13.64)**
Females, $n = 599$									
SAD z-score	**1.80 (1.34, 2.42)**	**1.48 (1.09,2.01)**	**2.32 (1.61,3.34)**	**2.03 (1.53,2.70)**	**2.12 (1.36,3.30)**	1.35 (0.96,1.88)	**5.99 (3.88, 9.27)**	**3.72 (2.68,5.15)**	**4.20 (2.75,6.41)**
WC z-score	**1.62 (1.22,2.15)**	**1.54 (1.10,2.14)**	**2.24 (1.64,3.07)**	**2.21 (1.68,2.91)**	**2.76 2.31 (1.49,3.578)**	1.29 (0.94,1.77)	**5.50 (3.17,9.56)**	**3.72 (2.60,5.32)**	**3.40 (2.20,5.25)**

The reference category for each regression is normal or no (MetS).
Bolded values indicate statistical significance at $P < .05$.
* ORs estimated from binomial logistic regression.
† Blood pressure defined as elevated (>113/75 mm Hg [males aged 12 years], >114/75 mm Hg [females aged 12 years], >120/80 mm Hg [all aged ≥13 years] vs. normal (reference, ≤113/75 mm Hg [males aged 12 years], ≤114/75 mm Hg [females aged 12 years], and ≤120/80 mm Hg [all aged ≥13 years]).
‡ ORs estimated from multinomial logistic regression.

definitions resulted in similar, lower estimated prevalence of MetS than the de Ferranti et al.'s definition. The de Ferranti et al.'s estimated prevalence of MetS was two and a half to three times that of the other two definitions. We also found that increases in SAD and WC were associated with approximately equal increased odds of MetS risk factors and MetS. However, associations between abdominal obesity and MetS may be stronger for the Cook et al. and IDF definitions compared to the de Ferranti et al.'s definition. Furthermore, associations between abdominal obesity and IDF-defined MetS were stronger among white compared to Hispanic/Latino and black participants.

Our results, in agreement with previous studies, suggest that many adolescents, in addition to being overweight or obese, are showing other signs of metabolic dysfunction including elevated blood glucose [3–5]. Furthermore, in prior NHANES survey years, researchers have estimated that the prevalence of MetS in U.S. adolescents aged 12–19 years ranged between 4.2% and 9.2% depending on which MetS definition was used [4,5,39,40]. Our results similarly suggest that MetS prevalence ranges between 3.3% and 8.8% among U.S. adolescents for the years 2011–2016.

Although previous longitudinal studies of adults suggest that SAD may be a better predictor of T2DM and CVD than WC [20], our cross-sectional results do not support SAD as a better indicator of possible metabolic dysfunction and MetS among adolescents. There were strong associations between these measures of abdominal obesity and MetS risk factors, further supporting abdominal obesity as a strong risk factor for metabolic dysfunction. Therefore, it is probable that use of either SAD or WC in a clinical setting will equally identify adolescents who may need further laboratory testing for other MetS risk factors. However, more research is needed to examine the predictive power of SAD while longitudinally following children and adolescents into adulthood.

Our results showed that U.S. adolescent males and females had similar prevalence of overweight and obesity but differ in behaviors as well as in associations between abdominal obesity and metabolic dysfunction. Females were less likely to meet physical activity guidelines, and males were more likely to consume a greater number of calories. Sex-specific targeted interventions toward these health behaviors may be necessary. Despite the association between abdominal obesity and elevated glucose among males, higher abdominal obesity was not associated with elevated glucose in females. In addition, there were racial/ethnic differences in associations between abdominal obesity and IDF-defined MetS. Research has posited that insulin resistance may vary by sex and race/ethnicity at birth, in childhood, and in adolescence [41–43]. These studies suggest that there may be genetic and pathophysiologic differences affecting the progression to MetS and T2DM among adolescent subpopulations. Beyond abdominal obesity, insulin resistance throughout the life course should be further explored. Sex and racial/ethnic differences in lifestyle factors related to obesity and insulin resistance should also be explored in these studies. It is also possible that some environmental contaminants, to which females may be more highly exposed, may be associated with insulin resistance [44]. Future studies are necessary to explore environmental factors, health behaviors, and biologic mechanisms that may contribute to our observations.

There were limitations that require careful interpretation of these results. The cross-sectional study design restricts our ability to make causal interpretations of the data. Longitudinal studies are necessary to strengthen the current results. Although we estimated the prevalence of several signs of metabolic dysfunction, these are not indicative of actual diagnoses. For males, we were unable to adjust for pubertal status in models estimating associations with abdominal obesity. In our sensitivity analysis, pubertal status did not significantly affect associations between abdominal obesity and MetS risk

Table 4
Adjusted odds ratios of metabolic syndrome risk factors and MetS among included adolescents, NHANES (2011–2016), $N = 1214$

Measures of abdominal obesity	OR (95% CI)								
	Blood Pressure†,‡	Triglycerides§ (reference: <90 mg per dL)		High-density lipoprotein cholesterol§ (reference: >45 mg per dL)		Fasting blood glucose† (reference: 0–99 mg per dL)	MetS†		
	Elevated	Borderline (90–129 mg per dL)	High (≥130 mg per dL)	Borderline (40–45 mg per dL)	Low (<40 mg per dL)	Borderline/high (≥100 mg/dL)	Cook et al. [4]	de Ferranti et al. [5]	IDF [22]
All*									
SAD z-score	**1.66 (1.42,1.95)**	**1.75 (1.37,2.22)**	**2.63 (2.10,3.31)**	**2.04 (1.65,2.54)**	**2.91 (2.23,3.80)**	**1.46 (1.21,1.77)**	**7.13 (4.19,10.36)**	**4.32 (3.35,5.56)**	**6.82 (4.94,9.42)**
WC z-score	**1.63 (1.40,1.90)**	**1.75 (1.38,2.21)**	**2.58 (2.09,3.18)**	**2.13 (1.69,2.68)**	**2.92 (2.21,3.86)**	**1.45 (1.20,1.77)**	**6.61 (4.41,9.91)**	**4.32 (3.32,5.62)**	**6.32 (4.58,8.72)**
Males $n = 615$									
SAD z-score	**1.64 (1.33,2.02)**	**2.01 (1.4,2.88)**	**2.83 (2.05,3.90)**	**2.00 (1.58,2.54)**	**3.38 (2.35,4.87)**	**1.53 (1.23,1.90)**	**7.79 (4.72,12.83)**	**4.53 (3.72,6.27)**	**10.17 (5.42,91.09)**
WC z-score	**1.64 (1.35,2.00)**	**1.99 (1.38,2.86)**	**2.89 (2.06,4.05)**	**2.03 (1.53,2.70)**	**3.27 (2.27,4.73)**	**1.65 (1.24,1.97)**	**7.74 (4.74,12.64)**	**4.74 (3.35,6.70)**	**11.94 (6.26,22.78)**
Females $n = 599$									
SAD z-score	**1.71 (1.27,2.30)**	**1.47 (1.09,2.00)**	**2.49 (1.74,3.55)**	**2.19 (1.67,2.88)**	**2.29 (1.46,3.61)**	1.37 (0.98,1.90)	NE	**4.36 (2.74,6.92)**	**4.79 (3.07,7.47)**
WC z-score	**1.56 (1.19,2.05)**	**1.50 (1.10,2.05)**	**2.32 (1.73,3.11)**	**2.32 (1.79,3.00)**	**2.38 (1.55,3.67)**	1.29 (0.95,1.77)	NE	**4.24 (3.10,5.79)**	**3.93 (2.67,5.80)**

The reference category for each regression is normal or no (MetS).
Bolded values indicate statistical significance at $P < .05$.
Models are adjusted for physical activity (meets physical activity guidelines for adolescents vs. does not meet), sedentary behavior (average time [hours] per day), total caloric intake (kcals/day), race (Hispanic, non-Hispanic white, non-Hispanic black, other), age (years), and family income-to-poverty ratio.
* Adjusted for gender in addition to previously listed covariates.
† Adjusted ORs estimated from binomial logistic regression.
‡ Blood pressure defined as elevated (>113/75 mm Hg [males aged 12 years], >114/75 mm Hg [females aged 12 years], and >120/80 mm Hg [all aged ≥13 years] vs. normal (reference, ≤113/75 mm Hg [males aged 12 years], ≤114/75 mm Hg [females aged 12 years], and ≤120/80 mm Hg [all aged ≥13 years]).
§ Adjusted ORs estimated from multinomial logistic regression.

Table 5
Adjusted odds ratios of metabolic syndrome among included adolescents, stratified by race/ethnicity, NHANES (2011–2016), $N = 1027$

Measures of abdominal obesity	MetS† (reference: No)
	IDF [22]
	OR (95% CI)
All*	
SAD z-score	
Hispanic	**4.84 (3.73,6.29)**
NH white	**12.6 (5.89,27.1)**
NH black	**5.31 (3.00,9.38)**
WC z-score	
Hispanic	**4.46 (3.25,6.13)**
NH white	**13.7 (5.22,35.9)**
NH black	**4.56 (2.78,7.48)**
Males $n = 532$	
SAD z-score	
Hispanic	**5.39 (3.27,8.87)**
NH white	**21.4 (7.40,61.8)**
NH black	**19.2 (4.07–90.2)**
WC z-score	
Hispanic	**6.96 (3.91,12.4)**
NH white	**29.5 (3.59,242)**
NH black	**13.0 (3.91,43.3)**
Females $n = 495$	
SAD z-score	
Hispanic	**5.21 (2.58,10.5)**
NH white	**10.3 (1.44,73.4)**
NH black	**4.52 (1.54,13.3)**
WC z-score	
Hispanic	**3.73 (2.21,6.30)**
NH white	**7.77 (1.62,37.4)**
NH black	**3.95 (1.73,9.01)**

Bolded values indicate statistical significance at $P < .05$.
Adjusting for physical activity (meets physical activity guidelines for adolescents vs. does not meet), sedentary behavior (average time [hours] per day), total caloric intake (kcals/day), race (Hispanic, non-Hispanic white, non-Hispanic black, other), age (years), and family income-to-poverty ratio.
* Adjusted for gender in addition to previously listed covariates.
† Adjusted ORs estimated from binomial logistic regression.

factors/MetS among females. If the association is similar among males, it is not likely that lack of adjustment for pubertal status among males would greatly affect the current results. There is also the possibility for residual confounding in final models assessing associations between WC, SAD, and MetS/MetS risk factors.

Despite these limitations, there were several strengths. Due to the large sample size, there was sufficient power to detect associations between abdominal obesity and MetS risk factors/MetS. We also used exclusion criteria to make inferences about adolescents currently without diagnosed diabetes. In addition, NHANES is a robust data source that allows generalizability to U.S. adolescents. NHANES also follows a stringent protocol and completes quality assurance for the laboratory assays, anthropometrics, and physical examination data, thus reducing the likelihood of measurement bias. Despite the limitations and given the strengths of this study, we interpret these results with caution, suggest public health implications, and offer future directions for research.

Conclusions

It is evident that U.S. adolescents are experiencing metabolic dysfunction, and the percentage with metabolic syndrome varies by sex and the definition applied. It is important to consider which definition of MetS is being used when making population inferences and planning public health interventions. Regardless of which MetS definition is used, abdominal obesity, whether measured by SAD or WC in adolescents, remains almost equally associated with MetS risk factors, except for elevated blood glucose in females. Other factors, such as increased insulin resistance, may be driving elevated blood glucose in females. Identifying and considering noninvasive measures beyond SAD or WC may be necessary to identify adolescents at risk of metabolic dysfunction and MetS.

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Supplementary data

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References

[1] Ogden CL, Carroll MD, Lawman HG, Fryar CD, Kruszon-Moran D, Kit BK, et al. Trends in obesity prevalence among children and adolescents in the united states, 1988-1994 through 2013-2014. JAMA 2016;315(21):2292–9.
[2] Hales CM, Fryar CD, Carroll MD, Freedman DS, Ogden CL. Trends in obesity and severe obesity prevalence in us youth and adults by sex and age, 2007-2008 to 2015-2016. JAMA 2018;319(16):1723–5.
[3] Grundy SM. Metabolic Syndrome: Connecting and Reconciling Cardiovascular and Diabetes Worlds. J Am Coll Cardiol 2006;47(6):1093–100.
[4] 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(8):821–7.
[5] 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(16):2494–7.
[6] Dhuper S, Cohen HW, Daniel J, Gumidyala P, Agarwalla V, St Victor R, et al. Utility of the modified ATP III defined metabolic syndrome and severe obesity as predictors of insulin resistance in overweight children and adolescents: a cross-sectional study. Cardiovasc Diabetol 2007;6:4.
[7] Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005;365(9468):1415–28.
[8] Goodman E, Daniels SR, Morrison JA, Huang B, Dolan LM. Contrasting prevalence of and demographic disparities in the World Health Organization and National Cholesterol Education Program Adult Treatment Panel III definitions of metabolic syndrome among adolescents. J Pediatr 2004;145(4):445–51.
[9] Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. Diabetes 2002;51(1):204–9.
[10] Weiss R, Bremer AA, Lustig RH. What is metabolic syndrome, and why are children getting it? Ann N Y Acad Sci 2013;1281:123–40.
[11] Grundy SM, Brewer Jr HB, Cleeman JI, Smith Jr SC, Lenfant C, American Heart A, et al. Definition of Metabolic Syndrome. Report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on Scientific Issues Relat Definition. Circulation 2004;109(3):433–8.
[12] National Center for Health Statistics. Health, United States, 2015: With Special Feature on Racial and Ethnic Health Disparities. 2016. Hyattsville, MD.
[13] Al-Hamad D, Raman V. Metabolic syndrome in children and adolescents. Transl Pediatr 2017;6(4):397–407.
[14] Jääskeläinen P, Magnussen CG, Pahkala K, Mikkila V, Kahonen M, Sabin MA, et al. Childhood nutrition in predicting metabolic syndrome in adults. Cardiovasc Risk Young Finns Study 2012;35(9):1937–43.
[15] Seo Y-G, Choi MK, Kang JH, Lee HJ, Jang HB, Park SI, et al. Cardiovascular disease risk factor clustering in children and adolescents: a prospective cohort study. Arch Dis Child 2018;103:968–73.
[16] Lichtveld K, Thomas K, Tulve NS. Chemical and non-chemical stressors affecting childhood obesity: a systematic scoping review. J Expo Sci Environ Epidemiol 2017;28:1.
[17] Després J-P. Body fat distribution and risk of cardiovascular disease. An Update 2012;126(10):1301–13.
[18] Pou KM, Massaro JM, Hoffmann U, Lieb K, Vasan RS, O'Donnell CJ, et al. Patterns of abdominal fat distribution: the Framingham Heart Study. Diabetes Care 2009;32(3):481–5.
[19] Iribarren C, Darbinian JA, Lo JC, Fireman BH, Go AS. Value of the sagittal abdominal diameter in coronary heart disease risk assessment: cohort study in a large, multiethnic population. Am J Epidemiol 2006;164(12):1150–9.
[20] Pajunen P, Rissanen H, Laaksonen MA, Heliovaara M, Reunanen A, Knekt P. Sagittal abdominal diameter as a new predictor for incident diabetes. Diabetes Care 2013;36(2):283–8.
[21] Radholm K, Tengblad A, Dahlen E, Lanne T, Engvall J, Nystrom FH, et al. The impact of using sagittal abdominal diameter to predict major cardiovascular events in European patients with type 2 diabetes. Nutr Metab Cardiovasc Dis 2017;27(5):418–22.
[22] Zimmet P, Alberti KG, Kaufman F, Tajima N, Silink M, Arslanian S, et al. The metabolic syndrome in children and adolescents - an IDF consensus report. Pediatr Diabetes 2007;8(5):299–306.
[23] Weber DR, Levitt Katz LE, Zemel BS, Gallagher PR, Murphy KM, Dumser SM, et al. Anthropometric measures of abdominal adiposity for the identification of cardiometabolic risk factors in adolescents. Diabetes Res Clin Pract 2014;103(3):e14–7.
[24] Johnson C, Dohmann S, Burt V, Modhadjer L. In: Statistics National Center for Health Statistics, editor. National Health and Nutrition Examination Survey: Sample design, 2011-2014; 2014. Hyattsville, MD.
[25] NHANES 2013-2014 Lab Data Overview. https://www.cdc.gov/nchs/nhanes/nhanes2013-2014/labdoc_h.htm. [Accessed 16 November 2016].
[26] Anthropometry Procedures Manual. National Health and Nutrition Examination Survey (NHANES). https://wwwn.cdc.gov/nchs/data/nhanes/2011-2012/manuals/Anthropometry_Procedures_Manual.pdf. [Accessed 5 May 2017].
[27] Physical Examination Procedures Manual. National Health and Nutrition Examination Survey (NHANES). https://wwwn.cdc.gov/nchs/data/nhanes/2011-2012/manuals/Physician_Exam_Manual.pdf. [Accessed 5 May 2017].
[28] CDC. CDC Growth Charts. Hyattsville, MD: National Center for Health Statistics; 2000.
[29] Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. Pediatrics 2017;140(3):e20171904.
[30] NCHS. Laboratory Procedures Manual. National Health and Nutrition Examination Survey (NHANES). https://wwwn.cdc.gov/nchs/data/nhanes/2011-2012/manuals/2011-12_Laboratory_Procedures_Manual.pdf. [Accessed 5 May 2017].
[31] Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. Pediatrics 2011;128(Suppl 5):S213–56.
[32] Standards of Medical Care in Diabetes—2014. Diabetes Care 2014;37(Suppl 1):S14–80.
[33] Copeland KC, Silverstein J, Moore KR, Prazar GE, Raymer T, Shiffman RN, et al. Management of newly diagnosed type 2 diabetes mellitus (T2DM) in children and adolescents. Pediatrics 2013;131:364–82.
[34] NCEP. 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) final report. Circulation 2002;106(25):3143–421.
[35] ASPE.U.S.. Federal Poverty Guidelines Used to Determine Financial Eligibility for Certain Federal Programs. https://aspe.hhs.gov/poverty-guidelines. [Accessed 29 June 2017].
[36] U.D.o.H.a.H. Services, editor. 2008 Physical Activity Guidelines for Americans; 2008. Washington, DC.
[37] Day FR, Elks CE, Murray A, Ong KK, Perry JR. Puberty timing associated with diabetes, cardiovascular disease and also diverse health outcomes in men and women: the UK Biobank study. Sci Rep 2015;5:11208.
[38] Ahmed ML, Ong KK, Dunger DB. Childhood obesity and the timing of puberty. Trends Endocrinol Metab 2009;20(5):237–42.
[39] Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among u.s. Adolescents, 1999-2000. Diabetes Care 2004;27(10):2438–43.
[40] Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH. Prevalence of the metabolic syndrome among U.S. adolescents using the definition from the International Diabetes Federation. Diabetes Care 2008;31(3):587–9.
[41] Kuchay MS, Kudyar RP, Gupta A, Pandita KK, Ganie MA. Gender differences in insulin and C-peptide concentrations at birth using cord blood collection. Arch Endocrinol Metab 2016;60(3):264–6.
[42] Arslanian SA. Type 2 diabetes mellitus in children: pathophysiology and risk factors. J Pediatr Endocrinol Metab 2000;13(Suppl 6):1385–94.
[43] Reinehr T. Type 2 diabetes mellitus in children and adolescents. World J Diabetes 2013;4(6):270–81.
[44] Hectors TL, Vanparys C, Van Gaal LF, Jorens PG, Covaci A, Blust R. Insulin resistance and environmental pollutants: experimental evidence and future perspectives. Environ Health Perspect 2013;121(11-12):1273–81.