

# Adjusting for Pubertal Status Reduces Overweight and Obesity Prevalence in the United States

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Objective To compare pediatric overweight and obesity prevalence among non-Hispanic white, Mexican American, and non-Hispanic black US youths before and after adjusting body mass index (BMI) for pubertal status, as assessed by Tanner stage.

Study design We analyzed cross-sectional anthropometric and pubertal data from non-Hispanic white, Mexican American, and non-Hispanic black youths in the National Health and Nutrition Examination Survey (NHANES) III. We developed specialized Tanner stage and chronological age-adjusted models to establish Tanner-stage adjusted BMI z-scores, which were then used to determine adjusted overweight/obesity prevalence. We compared pediatric overweight/obesity prevalence before and after pubertal status adjustment.

Results Among 3206 youths aged 8-18 years (50% male; 26% non-Hispanic white, 35% Mexican American, 39% non-Hispanic black), adjusting BMI for Tanner stage significantly reduced overweight (males, from 29% to 21%; females, from 29% to 17%) and obesity (males, from 14% to 7%; females, from 11% to 5%) prevalence across all races/ethnicities. The obesity prevalence reduction was more pronounced in Mexican Americans (males, 11% reduction; females, 9% reduction) and non-Hispanic blacks (males and females, 10% reduction) compared with non-Hispanic whites (males, 6% reduction; females, 5% reduction). Similar patterns were seen in overweight

**Conclusions** Adjusting for pubertal status reduced the prevalence of overweight/obesity in non-Hispanic white, Mexican American, and non-Hispanic black youth. This suggests that adjusting for puberty incorporates changes otherwise not captured when only considering the age of a child. Adjusting BMI for pubertal status may be important when interpreting a youth's weight status and consideration for obesity management, as well as when interpreting pediatric overweight/obesity prevalence data. (J Pediatr 2021;231:200-6).

he Centers for Disease Control and Prevention (CDC) 2000 growth charts, based on cross-sectional national health examination surveys, are the main anthropometric assessment tool for US youths aged 2-20 years. A limitation of these growth charts is that they only account for chronological age and thus do not consider other factors that may affect normal growth timing and trajectory.

Evidence suggests that pubertal status may impact classification of anthropometric measures in youth, including height, weight, and body mass index (BMI).<sup>2-10</sup> For example, in a cross-sectional UK study, Gillison et al found that early-maturing youths were 5 times more likely to be misclassified as overweight compared with "on-time" maturers.<sup>2</sup> Furthermore, studies from the US, Germany, and Denmark suggest that youths who are tall and/or undergo early maturity are more likely to have higher BMI and/or be misclassified as obese.<sup>6-9</sup> During puberty, sexually dimorphic increases in bone mineral content, lean body mass, and adiposity occur due to increases in gonadal sex steroids. 11 "Early maturers" have increased lean mass and adiposity due to increased androgen and estrogen levels for age, respectively, which can increase BMI for age when compared with "on-time" maturers. 12-14 Therefore, those experiencing earlier puberty may be more likely misclassified as overweight/obese.

Given the importance of properly categorizing weight status, we sought to test our hypothesis that incorporating pubertal status into a commonly used

BMI Body mass index

CA-BMI Chronological age-adjusted body mass index CDC Centers for Disease Control and Prevention **NHANES** National Health and Nutrition Examination Survey TSA-BMI

Tanner stage-adjusted body mass index

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chronological age-only BMI metric decreases overweight/obesity prevalence among US youth. We first developed a BMI statistical model accounting for both chronological age (CA) and Tanner stage in US youths aged 6-18 years to develop a Tanner stage age-BMI (TSA-BMI) metric. We then examined overweight/obesity prevalence using TSA-BMI and compared overweight/obesity prevalence as determined by TSA-BMI with that determined by the commonly used CDC 2000 CA-only BMI metric (CA-BMI). As pubertal timing is not congruent among races/ethnicities,<sup>5</sup> we compared TSA-BMI with CA-BMI by race/ethnicity.

#### Methods

Our study population consisted of US children from the National Health and Nutritional Examinations Survey 1988-1994 (NHANES III), a complex cross-sectional survey of 39 695 individuals aged ≥2 months. <sup>15</sup> CDC/National Center for Health Statistics Institutional Review Board approval and documented consent was obtained from all participants. We used NHANES III because this was the last cycle to include pubertal assessment by Tanner staging. <sup>16</sup> We included only participants aged 8-18 years because this was the group in which Tanner staging was performed. We excluded prepubertal children (Tanner stage 1) given our interest in pubertal status, as well as those with missing data for any of the following: age, weight, height, sex, and Tanner stage (Figure 1; available at www.jpeds.com).

Height and weight in NHANES III, used to determine BMI, were measured following standardized protocols. <sup>15</sup> For race/ethnicity, participants were categorized as non-Hispanic white, Mexican American, or non-Hispanic black based on self-report according to NHANES III groupings. Overall health was assessed by the question: "Would you say [your child's] health is excellent, very good, good, fair, or poor?"

Pubertal status was determined by Marshall-Tanner criteria and evaluated by physicians who received standardized training. 15,17,18 Tanner staging was based on inspection and comparison with standardized photos of the breast and pubic hair in girls and of the genitalia and pubic hair in boys. For our analyses, we used breast assessment for girls and genitalia assessment for boys, because these are better markers of pubertal staging than pubic hair, which could falsely elevate pubertal status in children with premature adrenarche. We defined boys and girls as "early maturers" if their CA was less than US published national timing estimates for their sex-race/ethnicity population's median age at entry into Tanner stage 2. 20

We developed a specialized TSA-BMI metric incorporating both chronological age and pubertal stage using an extended function of the semiparametric lambda-mu-sigma (LMS) approach. We used the LMS method in a generalized additive model for location, scale, and shape technique of growth modeling to develop specialized age-conditioned growth functions within each Tanner stage. 21-23 This technique

ensures that each age and Tanner stage is incorporated into estimations of maturation-adjusted anthropometric normalized *z*-scores and is similar to the approach used to develop the CDC and World Health Organization growth charts. Model diagnostics were followed to ascertain the adequacy of fit according to standard protocols. With each fitted function, TSA-BMI *z*-scores, analogous to US CDC 2000 CA *z*-scores, were calculated, as were corresponding TSA-BMI percentile scores. These *z*-scores were then used to derive indicators of weight status (overweight/obesity/severe obesity), to calculate the prevalence within each category.

Overweight/obesity status for each participant was defined by a BMI-adjusted *z*-score  $\geq$ +1.036 SD for overweight (equal to BMI  $\geq$ 85th percentile; age and sex-adjusted),  $\geq$ +1.645 SD for obesity (equal to BMI  $\geq$ 95th percentile), and  $\geq$ +1.975 SD for severe obesity (equal to BMI  $\geq$ 1.2 times the 95th percentile<sup>24</sup>). We compared the overweight/obesity/severe obesity prevalence obtained via CA-BMI with that obtained via TSA-BMI across race/ ethnicity using the theorem of Fieller.<sup>25</sup>

Descriptive statistics are presented as mean and percentage with standard errors (SE). To control for the 3 race/ethnicity groups, multiple comparisons of weight status indicators (overweight/obesity/severe obesity) were conducted at an  $\alpha$  value of 0.0167 (alpha/3). CIs were set a priori at 98.33% around each point estimate and derived from 5000 resample bootstrap replications. For all other analyses, statistical significance was set at P < .05, with complex survey design effects and weighting adjustments as appropriate. All analyses were conducted in R version 3.6.0 (R Foundation for Statistical Computing) and SAS version 9.4 (SAS Institute).

# **Results**

Our analysis included 3206 participants aged 8-18 years, at Tanner stage 2-5, with complete anthropometric data. Primary descriptive characteristics of the study population are summarized in **Table I**. The mean age was 14.3 years, and mean BMI was 21.3 kg/m<sup>2</sup>. Mexican American youths had a higher BMI compared with non-Hispanic white and non-Hispanic black youths; however, there were no overall mean race/ethnicity-based differences (boys, P = .97; girls, P = .08). Between 4% and 11% of participants were "early maturers", with a higher prevalence in non-Hispanic blacks compared to non-Hispanic whites and Mexican Americans. The sample was largely in good health (<1% reported "poor health").

As shown in **Figure 2**, chronological age- and sex-adjusted (based on the CDC 2000 growth curves according to standard conventions<sup>26,27</sup>) overweight/obesity prevalence varied greatly across pubertal stage, race/ethnicity, and sex before Tanner stage-age adjustments. For example, non-Hispanic white and Mexican American girls were more likely to be classified as overweight at early puberty (Tanner 2: 34.9% and 32.8%, respectively) compared with non-Hispanic black girls (24.3%), and pubertal (Tanner 5) non-Hispanic

	Boys (N = 1606)					Girls (N = 1600)					
Variables	All boys	Non-Hispanic white (n = 402)	Non-Hispanic black (n = 638)	Mexican American (n = 566)	P value*	All girls	Non-Hispanic white (n = 429)	Non-Hispanic black (n = 605)	Mexican American (n = 566)	<i>P</i> value*	
Age, y, mean (SE)	14.3 (0.1)	14.3 (0.2)	14.1 (0.1)	14.4 (0.1)	.47	14.4 (0.1)	14.5 (0.2)	14.0 (0.1)	14.0 (0.2)	.04	
Height, cm, mean (SE)	163.8 (0.7)	164.6 (0.9)	162.2 (0.7)	160.3 (0.6)	.009	158.2 (0.5)	158.8 (0.6)	158.1 (0.5)	154.2 (0.6)	.13	
Weight, kg, mean (SE)	58.3 (1.0)	58.8 (1.4)	56.8 (1.0)	57.7 (0.8)	.19	54.0 (0.7)	53.9 (1.0)	55.2 (0.7)	52.9 (1.1)	.65	
BMI, kg/m <sup>2</sup> , mean (SE)	21.2 (0.3)	21.1 (0.3)	21.0 (0.20)	22.0 (0.2)	.97	21.4 (0.2)	21.1 (0.3)	21.8 (0.2)	22.0 (0.3)	.08	
Health rating, % (SE) <sup>†</sup>											
Excellent	43.8 (2.4)	48.8 (2.9)	34.2 (2.7)	23.0 (2.5)	<.001	46.6 (2.4)	52.0 (3.5)	34.1 (3.0)	32.2 (2.8)	<.001	
Very good	30.3 (2.1)	31.6 (2.5)	27.6 (2.7)	24.7 (2.0)		27.2 (2.3)	27.1 (3.1)	31.3 (2.6)	20.2 (2.5)		
Good	22.4 (2.2)	18.2 (2.6)	30.9 (3.1)	39.1 (2.4)		21.1 (2.1)	18.1 (3.2)	27.3 (2.8)	30.4 (2.3)		
Fair	3.1 (0.6)	1.1 (0.5)	6.6 (1.2)	11.9 (2.8)		4.5 (0.8)	2.3 (0.9)	6.8 (1.7)	15.0 (1.6)		
Poor	0.5 (0.3)	0.3 (0.3)	0.6 (0.4)	1.3 (0.6)		0.7 (0.4)	0.5 (0.5)	0.6 (0.4)	2.2 (1.1)		
"Early maturers", %	8.6 (1.2)	8.1 (1.5)	10.8 (1.3)	7.7 (1.5)	.22	5.2 (0.7)	4.0 (0.9)	9.7 (1.3)	5.0 (1.4)	<.001	

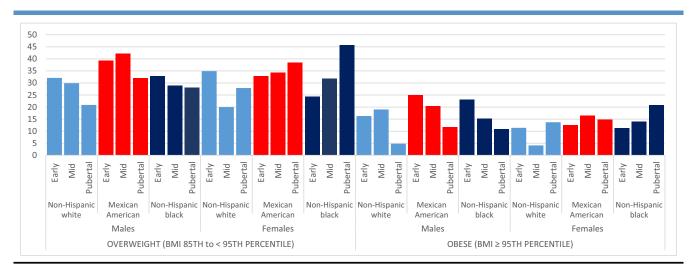
<sup>\*</sup>P value set at .05 and accounted for complex survey design effects and race/ethnicity differences within sex.

black girls had the highest overweight prevalence (45.7%). Mexican American boys had higher overweight/obesity prevalence in early to mid-puberty (Tanner 2-4: 39.4%-42.2% overweight, 20.4%-25.0% obesity) compared with non-Hispanic white and non-Hispanic black boys.

Table II summarizes overweight/obesity prevalence by race/ethnicity, comparing CA-BMI with TSA-BMI. Overall, using TSA-BMI significantly decreased overweight and obesity prevalence across all races/ethnicities for both sexes; for example, overweight prevalence decreased from 37.5% in Mexican American boys and 35.8% in Mexican American girls to 20.8% and 18.5%, respectively. Similarly, obesity prevalence decreased from 15.2% in non-Hispanic black boys and 17.3% in non-Hispanic black girls to 5.4%

and 7.2%, respectively. There was no significant difference in severe obesity prevalence by race/ethnicity between CA-BMI and TSA-BMI; however, sample sizes were small (0-10 participants in each group).

To quantify the magnitude of overweight/obesity misclassification by race/ethnicity, we calculated a percent prevalence difference of overweight/obesity by subtracting the prevalence obtained by CA-BMI from that obtained by TSA-BMI (**Figure 3**). Overall, the decrease in prevalence of overweight/obesity comparing CA-BMI with TSA-BMI ranged from 5.1% (for non-Hispanic white boys with obesity) to 22.5% (for non-Hispanic black girls with overweight). The differences in overweight/obesity prevalence between CA-BMI and TSA-BMI were more



**Figure 2.** Percent prevalence in each category. The prevalence of overweight and obesity shown in this figure is chronological age- and sex-adjusted per the CDC 2000 growth charts according to standard conventions. \*Pubertal status categorized as early (Tanner stage 2), mid (Tanner stage 3-4), and pubertal (Tanner stage 5).

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<sup>†</sup>Determined from self/family-reported health rating question (NHANES III).

<sup>‡</sup>Chronological age less than US published national timing estimates for sex-race/ethnic population median age at entry into Tanner stage 2.20

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Table II. Overweight/obesity prevaler race/ethnicity	nce before an	nd after adjustment for	pube	tal status (Ta	nnner stage) by sex and	1
	Boys			Girls		
	CA-BMI.	TSA-BMI.		CA-BMI.	TSA-BMI.	

			Boys		Girls			
Overweight/obesity category	Race/ethnicity	CA-BMI, prevalence (98.3% CI)	TSA-BMI, prevalence (98.3% CI)	<i>P</i> value	CA-BMI, prevalence (98.3% CI)	TSA-BMI, prevalence (98.3% CI)	<i>P</i> value	
Overweight	Non-Hispanic white	27.3 (22.0-32.8)	22.0 (17.2-27.2)	<.001	25.7 (21.0-30.9)	17.0 (12.9-21.3)	<.001	
(BMI 85th to <95th	Mexican American	37.5 (32.0-42.9)	20.8 (16.3-25.6)	<.001	35.8 (30.5-41.3)	18.5 (14.2-23.2)	<.001	
percentile)	Non-Hispanic black	29.5 (25.2-34.0)	16.3 (13.1-19.7)	<.001	38.1 (33.1-43.1)	15.6 (12.2-19.5)	<.001	
	Overall	28.6 (24.8-32.6)	20.8 (17.4-24.7)	<.001	29.0 (25.4-32.8)	16.9 (13.9-20.1)	<.001	
Obese (BMI ≥95th	Non-Hispanic white	13.4 (9.5-17.7)	7.0 (4.0-10.6)	<.001	9.2 (6.2-12.8)	4.1 (2.4-6.2)	<.001	
percentile)	Mexican American	18.2 (14.3-22.6)	7.2 (4.6-10.2)	<.001	15.0 (11.3-19.3)	5.9 (3.3-9.2)	<.001	
	Non-Hispanic black	15.2 (12.1-18.5)	5.4 (3.6-7.4)	<.001	17.3 (13.5-21.2)	7.2 (4.6-10.1)	<.001	
	Overall	14.1 (11.3-17.3)	6.7 (4.5-9.4)	<.001	11.3 (9.0-14.0)	4.9 (3.3-6.5)	<.001	

pronounced in non-Hispanic black and Mexican American youths compared with non-Hispanic white youths; for example, obesity prevalence decreased by 5.1% in non-Hispanic white girls, compared with 10.1% in non-Hispanic black girls and 9.1% in Mexican American girls.

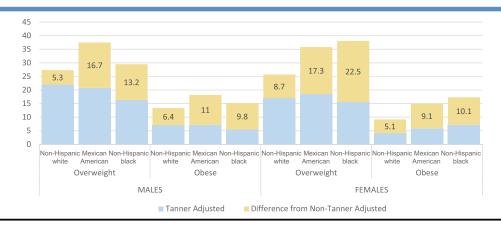
We found that before Tanner adjustment, the BMI curves were disparate across the race/ethnicity categories, with overall higher BMI z-scores among non-Hispanic black and Mexican American youths compared with non-Hispanic white youths at most ages. However, after Tanner adjustment, the BMI curves condensed into similar curves overall (Figure 4; available at www.jpeds.com). This demonstrates that our adjustment corrects for differences in the effect of pubertal status on BMI by race/ethnicity, indicating that the model performs as intended and that within our cohort, much of the variability in CA-BMI reference data may be related to race/ethnicity-based differences in maturation progression.

To demonstrate the clinical utility of our model, **Figure 5** shows sample TSA-BMI curves for Tanner stage 2 females

superimposed on the CDC 2000 curves. This example demonstrates how the use of our model may avoid misclassifying an "earlier maturing" female as overweight or a "late maturing" female as underweight (BMI <5th percentile<sup>28</sup>), when both should have been classified as normal weight based on their BMI after considering pubertal status.

### **Discussion**

In a multiethnic cross-sectional population of US youths, adjusting BMI for Tanner stage relative to chronological age resulted in reductions in pediatric overweight/obesity prevalence. Adjusting BMI for Tanner stage decreased overweight prevalence by 5.3%-22.5% and decreased obesity prevalence by 5.1%-11.0%. The reductions in overweight/obesity prevalence were more pronounced in Mexican American and non-Hispanic black youths compared with non-Hispanic white youths. Although adjusting for Tanner



**Figure 3.** Numbers correspond to the percent prevalence difference, calculated by substracting the prevalence obtained from CA-BMI from that obtained by TSA-BMI in each category.

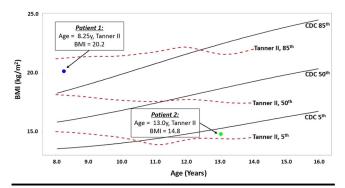


Figure 5. A, As an example of the clinical utility of our model, this figure depicts the TSA-BMI curves for Tanner stage 2 females (red dashed lines) superimposed on the CDC 2000 curves (black lines). Adjusting BMI by pubertal stage may allow a provider to avoid misclassifying an "early maturing" child as having a BMI in the overweight/obese category, or a "late maturing" child as having a BMI in the underweight category, when both actually have BMIs in the normal weight category after pubertal status is considered. B, Patient 1 (blue dot) is an 8.25-year-old "earlier maturing" Tanner stage 2 female. She would be considered overweight according to the CDC 2000 BMI-for-age charts with a sex- and age-adjusted BMI ≥85th percentile. However, after adjusting for pubertal stage (TSA-BMI), her BMI is in the normal range. C, Patient 2 (green dot) is a 13.0-year-old "late maturing" Tanner stage 2 female. She would be considered underweight according to the CDC 2000 BMI-for-age charts with a sex- and ageadjusted BMI <5th percentile. However, after adjusting for pubertal stage (TSA-BMI), her BMI is in the normal range.

stage did not reduce severe obesity prevalence, our analysis was limited by small sample sizes in this category.

Our findings are consistent with previous studies and further elucidate the importance of considering puberty when determining weight status. Indeed, studies have shown that standard CA-BMI z-scores may overestimate weight status prevalence if maturation is unaccounted for.<sup>5-7</sup> For example, Sorensen and Juul, in a cross-sectional study of Danish Caucasian youth, found a higher overweight/obesity prevalence in early maturers compared with late maturers despite similar body fat percentages. Gillison et al, in a study of 9- to 11-year-old UK children, found that adjusting weight for maturational status resulted in 32% of girls and 15% of boys with overweight being reclassified as normal weight and 11% of boys and 8% of girls with obesity reclassified as overweight.<sup>2</sup> However, in this study, maturational status was determined via the Khamis-Roche method, which calculates predicted adult height from a combination of a child's height and weight with midparental height, 29 and race/ ethnicity was not considered owing to underrepresentation.<sup>2</sup>

There are several biological factors that may lead to higher BMI in children with earlier puberty. Increased androgen production, which occurs at pubertal onset compared with prepuberty, is associated with lower leptin levels and greater lean body mass. <sup>30,31</sup> This may increase weight and

subsequently BMI in early maturers compared with ontime maturers. Moreover, increased estrogen, both directly and through aromatization of androgens during puberty, is associated with increased adiposity, which could also increase BMI in early maturers. Finally, studies have shown a higher overweight/obesity prevalence in those who are relatively taller for age compared with those of average height or shorter. This may be due to relatively greater adiposity (if caloric intake is more than sufficient to achieve rapid linear growth, excess could be stored as subcutaneous fat) or lean mass in taller children. Stored as subcutaneous fat) or lean mass in taller children. Such a scenario would occur in children experiencing earlier pubertal growth spurts compared with their peers, leading to a higher likelihood of overweight/obesity misclassification.

It is notable that overweight/obesity prevalence reductions after pubertal status adjustment were more pronounced in non-Hispanic black youths compared with non-Hispanic white youths. We hypothesize that this may stem from the higher prevalence of early puberty in non-Hispanic black youths. Indeed, in our study, the prevalence of early maturers was higher in non-Hispanic black youths compared with non-Hispanic white youths, a finding supported by the previous literature. For example, among US girls, we previously showed that pubertal onset (defined by increases in luteinizing hormone and inhibin B) was delayed by 0.5 year in non-Hispanic black girls versus Mexican American and non-Hispanic white girls.<sup>35</sup> Herman-Giddens et al found that US non-Hispanic black boys reached Tanner stage 2-4 genital volume and pubic hair significantly earlier compared with non-Hispanic white boys.

We also found that reductions in overweight/obesity prevalence were more pronounced in Mexican American youths compared with non-Hispanic white youths, despite the similar prevalence of early pubertal onset in the 2 groups. This may be because Mexican American youths are more likely to be misclassified as short and tend to be shorter and heavier for their height.<sup>2,37</sup> In our previous study evaluating the effect of Tanner stage adjustment on short/tall stature prevalence, among "early maturers", Mexican American youths were 45%-60% more likely to be classified as short compared with non-Hispanic white and non-Hispanic black youths; however, after pubertal adjustment, there were no significant differences in short stature prevalence among the 3 groups. The apparent more pronounced misclassification of short stature in Mexican American youths compared with non-Hispanic black and non-Hispanic white youths, in conjunction with how BMI is calculated (kg/m<sup>2</sup>), suggests that pubertal status adjustments could have a greater impact on overweight/obesity prevalence in Mexican American youths compared with their counterparts.

It is also possible that Mexican American youths had a greater reduction in overweight/obesity prevalence compared with non-Hispanic white youths despite similar timing of pubertal onset because of differences in the pattern of developing overweight/obesity by age between these populations. For example, in a study by Ogden et al examining obesity prevalence in US youths, Mexican American youths

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had higher obesity prevalence in the 6- to 11-year-old age group (Mexican American, 25.0%; non-Hispanic white, 13.6%),<sup>38</sup> during which time most youths are prepubertal or peripubertal. However, in the 12- to 19-year-old age group, in which most youths are pubertal or postpubertal, obesity prevalence was similar in the 2 populations (Mexican American, 22.8%; non-Hispanic white, 19.6%).<sup>38</sup> The earlier development of obesity in Mexican American youths during prepuberty and peripuberty increases the obesity prevalence in these categories, in conjunction with earlier pubertal progression, may partially explain why, after adjusting for pubertal status, Mexican American youths had more pronounced reductions in obesity prevalence compared with non-Hispanic white youths.

The diagnosis of pediatric obesity accompanies both medical and psychological sequelae and is associated with increased healthcare utilization. According to the Endocrine Society, youths diagnosed with obesity should be prescribed intensive lifestyle interventions, including diet modifications and increased physical activity.<sup>39</sup> The American Diabetes Association recommends that type 2 diabetes mellitus testing be considered in youths with overweight/obesity and 1 or more risk factors, including certain races/ethnicities (including Mexican American and non-Hispanic black). 40 Further, medical providers often screen for additional obesityrelated complications and comorbidities in youths diagnosed with obesity, including nonalcoholic fatty liver disease, renal disease, and obstructive sleep apnea. These recommendations and practices all come at an increased cost, both financially 41,42 and in time. Further, a diagnosis of obesity may be associated with weight stigma and discrimination. 43 On the other hand, if a child or adolescent is not diagnosed with obesity when they indeed have obesity, opportunities for earlier intervention and prevention of complications may be missed. Therefore, accurate diagnosis of overweight/ obesity is imperative.

Although our results suggest that after adjusting for pubertal status, overweight/obesity prevalence decreases, they do not suggest that US overweight/obesity prevalence is decreasing or otherwise is not alarming. Previous investigations of US overweight/obesity prevalence did not apply our adjustment, and thus proper comparisons cannot be made. Furthermore, we do not know whether or, if so, how adjusting for Tanner stage affects the prevalence of other aspects of the metabolic syndrome, including hyperglycemia, hyperlipidemia, and hypertension. Indeed, the type 2 diabetes mellitus prevalence among youths continues to increase. 44

Our study has some limitations. First, our results are based on NHANES III data, which occurred between 1988 and 1994. This was toward the beginning of the obesity epidemic, and largely predated the significant rise in pediatric severe obesity. Therefore, we could not adequately assess whether pubertal status reduces pediatric severe obesity prevalence owing to sample size limitations. We used NHANES III because this was the last cycle to incorporate Tanner staging, and more contemporaneous NHANES samples were not available.

It is unclear how our results apply to other races/ethnicities. Moreover, because these results are based on cross-sectional data, we could not determine the temporality of relationships within individuals. Analyses of longitudinal data incorporating age, anthropometric variables, and pubertal status would allow for improved modeling than can be ascertained through cross-sectional studies. Future NHANES cycles and/or large-scale multiethnic longitudinal studies should include Tanner stage assessments. This is even more important in light of more recent NHANES cycles including body composition measurements via such techniques as dual X-ray absorptiometry and bioelectrical impedance, 16 which may differentially reflect metabolic status and cardiometabolic risk compared with BMI measures, and may become more useful in the clinical setting when such techniques become more widespread in clinical practice. 45,46 Along these lines, including pubertal assessments into more primary care and weight management provider visits, when such body composition assessments may be adopted more readily, could lead to the development of more robust longitudinal data registries to explore this.

Finally, it is important to note that creation of the CDC 2000 growth charts excluded weight from NHANES III participants aged ≥6 years to avoid an upward shift in weight- and BMI-for-age curves owing to rising overweight prevalence, thereby underclassifying overweight/obesity status. <sup>47</sup> Because of this, our curves using NHANES III participants may not align completely with CDC 2000 BMI curves and may be biased toward higher weight categories.

Adjusting for pubertal status appears to have a more profound impact on decreasing overweight/obesity prevalence among non-Hispanic black and Mexican American youths compared with non-Hispanic whites, likely due to differences in timing of pubertal onset and patterns of weight gain between these racial/ethnic groups. Pubertal adjustments may be important when interpreting overweight/obesity prevalence data. When considering an adolescent's weight status in the clinical setting, it is also be important to account for pubertal status.

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## References

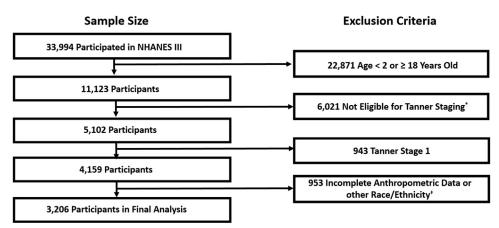
- Grummer-Strawn LM, Garza C, Johnson CL. Childhood growth charts. Pediatrics 2002;109:141-2.
- Gillison F, Cumming S, Standage M, Barnaby C, Katzmarzyk P. Assessing the impact of adjusting for maturity in weight status classification in a cross-sectional sample of UK children. BMJ Open 2017;7: e015769.
- 3. Addo OY, Sarafoglou K, Miller BS. Effect of adjusting for Tanner stage age on prevalence of short and tall stature of youths in the United States. J Pediatr 2018;201:93-9.e4.

- 4. Ramnitz MS, Lodish MB. Racial disparities in pubertal development. Semin Reprod Med 2013;31:333-9.
- Wang Y, Adair L. How does maturity adjustment influence the estimates
  of overweight prevalence in adolescents from different countries using
  an international reference? Int J Obes Relat Metab Disord 2001;25:
  550-8
- Himes JH. Maturation-related deviations and misclassification of stature and weight in adolescence. Am J Hum Biol 1999;11:499-504.
- 7. Sørensen K, Juul A. BMI percentile-for-age overestimates adiposity in early compared with late maturing pubertal children. Eur J Endocrinol 2015;173:227-35.
- 8. Bonthuis M, Jager KJ, Abu-Hanna A, Verrina E, Schaefer F, van Stralen KJ. Application of body mass index according to height-age in short and tall children. PLoS One 2013;8:e72068.
- Stovitz SD, Demerath EW, Hannan PJ, Lytle LA, Himes JH. Growing into obesity: patterns of height growth in those who become normal weight, overweight, or obese as young adults. Am J Hum Biol 2011;23: 635-41.
- Miller BS, Sarafoglou K, Addo OY. Development of Tanner stage ageadjusted CDC height curves for research and clinical applications. J Endocr Soc 2020;4:bvaa098.
- Soriano-Guillén L, Sarafoglou K, Argente J. Precocious puberty. In: Sarafoglou K, Hoffman GF, Roth KS, eds. Pediatric endocrinology and inborn errors of metabolism. 2nd ed. New York: McGraw-Hill; 2017. p. 643-62.
- 12. Sarafoglou K, Forlenza GP, Addo OY, Kyllo J, Lteif A, Hindmarsh PC, et al. Obesity in children with congenital adrenal hyperplasia in the Minnesota cohort: importance of adjusting body mass index for height-age. Clin Endocrinol (Oxf) 2017;86:708-16.
- Grantham JP, Henneberg M. The estrogen hypothesis of obesity. PLOS One 2014:9:e99776.
- 14. Lizcano F, Guzmán G. Estrogen deficiency and the origin of obesity during menopause. Biomed Res Int 2014;2014;757461.
- Centers for Disease Control and Prevention. National Center for Health Statistics. NHANES III (1988-1994), updated February 21, 2020. https://www.n.cdc.gov/nchs/nhanes/%20nhanes3/Default.aspx. Accessed July 27, 2020.
- NHANES. Historical summary of component content over time [updated May 7, 2009]. https://wwwn.cdc.gov/nchs/data/nhanes/%20Historical\_NHANES\_component\_matrix.pdf. Accessed July 27, 2020.
- 17. Marshall WA, Tanner JM. Variations in the pattern of pubertal changes in boys. Arch Dis Child 1970;45:13-23.
- Marshall WA, Tanner JM. Variations in pattern of pubertal changes in girls. Arch Dis Child 1969;44:291-303.
- Dunkel L, Sarafoglou K, Rey R, Lee PA. Variants of pubertal progression.
   In: Sarafoglou K, Hoffman GF, Roth KS, eds. Pediatric endocrinology and inborn errors of metabolism. 2nd ed. New York: McGraw Hill; 2017. p. 663-80.
- Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, et al. National estimates of the timing of sexual maturation and racial differences among US children. Pediatrics 2002;110:911-9.
- Cole TJ, Green PJ. Smoothing reference centile curves: the LMS method and penalized likelihood. Stat Med 1992;11:1305-19.
- 22. Rigby RA, Stasinopoulos DM. Generalized additive models for location, scale and shape. J R Stat Soc C Appl 2005;54:507-54.
- 23. Cole TJ. The development of growth reference and growth charts. Ann Hum Biol 2012;39:382-94.
- 24. Kelly AS, Barlow SE, Rao G, Inge TH, Hayman LL, Steinberger J, et al. Severe obesity in children and adolescents: identification, associated health risks, and treatment approaches: a scientific statement from the American Heart Association. Circulation 2013;128:1689-712.
- **25.** Fieller EC. Some problems in interval estimation. J Royal Stat Soc Series B Methodol 1954;16:175-85.
- Han JC, Lawler DA, Kimm SYS. Childhood obesity. Lancet 2010;375: 1737-48.

- **27**. 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;319:1723-5.
- 28. Maring B, Greenspan LC, Chandra M, Daniels SR, Sinaiko A, Prineas RJ, et al. Comparing US pediatric and adult weight classification at the transition from late teenage to young adulthood. Pediatr Obes 2015;10: 371-9.
- **29.** Khamis HJ, Roche AF. Predicting adult stature without using skeletal age: the Khamis-Roche method. Pediatrics 1994;94(4 Pt 1):504-7.
- Mason KA, Schoelwer MJ, Rogol AD. Androgens during infancy, child-hood, and adolescence: physiology and use in clinical practice. Endocr Rev 2020;41:bnaa003.
- Luukkaa V, Pesonen U, Huhtaniemi I, Lehtonen A, Tilvis R, Tuomilehto J, et al. Inverse correlation between serum testosterone and leptin in men. J Clin Endocrinol Metab 1998;83:3243-6.
- Biro FM, Pinney SM, Huang B, Baker ER, Chandler DW, Dorn LD. Hormone changes in peripubertal girls. J Clin Endocrinol Metab 2014;99: 3829-35.
- **33.** Freedman DS, Thornton JC, Mei Z, Wang J, Dietz WH, Pierson RN Jr, et al. Height and adiposity among children. Obes Res 2004;12:846-53.
- **34.** Forbes GB. Relation of lean body mass to height in children and adolescents. Pediatr Res 1972;6:32-7.
- Addo OY, Miller BS, Lee PA, Hediger ML, Himes JH. Age at hormonal onset of puberty based on leutinizing hormone, inhibin B, and body composition in preadolescent US girls. Pediatr Res 2014;76:564-70.
- **36.** Herman-Giddens ME, Steffes J, Harris D, Slora E, Hussey M, Dowshen SA, et al. Secondary sexual characteristics in boys: data from the Pediatric Research in Office Settings Network. Pediatrics 2012;130: e1058-68.
- Martorell R, Mendoza FS, Castillo RO, Pawson IG, Budge CC. Short and plump physique of Mexican-American children. Am J Phys Anthropol 1987;73:475-87.
- **38.** 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;315:2292-9.
- **39.** Styne DM, Arslanian SA, Connor EL, Farooqi IS, Murad MH, Silverstein JH, et al. Pediatric obesity: assessment, treatment, and prevention: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab 2017;102:709-57.
- 40. Arslanian S, Bacha F, Grey M, Marcus MD, White NH, Zeitler P. Evaluation and management of youth-onset type 2 diabetes: a position statement by the American Diabetes Association. Diabetes Care 2018;41: 2648-68.
- Tremmel M, Gerdtham UG, Nilsson PM, Saha S. Economic burden of obesity: a systematic literature review. Int J Environ Res Public Health 2017;14:435.
- 42. Tsai AG, Williamson DF, Glick HA. Direct medical cost of overweight and obesity in the USA: a quantative systematic review. Obes Rev 2011;12:50-61.
- **43.** Pont SJ, Puhl R, Cook SR, Slusser W. Stigma experienced by children and adolescents with obesity. Pediatrics 2017;140:e20173034.
- **44.** Mayer-Davies EJ, Lawrence JM, Dabelea D, Divers J, Isom S, Dolan L, et al. Incidence trends of type 1 and type 2 diabetes among youths, 2002-2012. N Engl J Med 2017;376:1419-29.
- Vanderwall C, Eickhoff J, Clark RR, Carrel AL. BMI z-score in obese children is a poor predictor of adiposity changes over time. BMC Pediatr 2018;18:187.
- **46.** Kelly AS, Daniels SR. Rethinking the use of body mass index *z*-score in children and adolescents with severe obesity: time to kick it to the curb? J Pediatr 2017;188:7-8.
- **47.** Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC growth charts for the United States: methods and development. Vital Health Stat 11 2002;246:1-190.

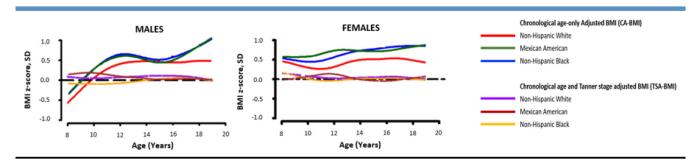
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<sup>\*</sup> Tanner stage only performed in participants ages 2 through 18 years old

Figure 1. Study cohort flow diagram.



**Figure 4.** This figure illustrates that after adjusting for Tanner stage, BMI growth curves condense into similar curves. This suggests that adjusting BMI for Tanner stage corrects for the racial/ethnic differences in the effect of pubertal status on BMI, and that much of the variability in current BMI-for-age reference data may be due to race/ethnicity-based differences in maturational progression.

<sup>&</sup>lt;sup>†</sup> Analysis limited to non-Hispanic White, Mexican American, and non-Hispanic black due to sample size limitations