

# BMI, Waist Circumference, and Selected Cardiovascular Disease Risk Factors Among Preschool-Age Children

Sarah E. Messiah<sup>1,2</sup>, Kristopher L. Arheart<sup>1,2</sup>, Ruby A. Natale<sup>3</sup>, WayWay M. Hlaing<sup>2</sup>, Steven E. Lipshultz<sup>1,2</sup> and Tracie L. Miller<sup>1,2</sup>

In adults, overweight is often associated with other cardiovascular disease (CVD) risk factors. We determined whether these associations were also present in young children. This study examined the relationships between elevated BMI ( $\geq 85$ th and  $\geq 95$ th percentiles for age and sex) and the highest quintile of waist circumference (WC) with CVD risk factors, including fasting triglyceride (TGL), high- and low-density lipoprotein (HDL and LDL), total cholesterol (TC), non-HDL cholesterol, and C-reactive protein (CRP) in 3,644 3- to 6-year-old children included in the 1999–2008 National Health and Nutrition Examination Surveys (NHANES). Results showed that 20% (highest quintile) of the sample had a TC  $>170$  mg/dl, LDL  $>109$  mg/dl, TGL  $>103$  mg/dl, non-HDL  $>128$  mg/dl, CRP  $>0.13$  mg/dl, WC  $>57.2$  cm, and HDL  $<42$  mg/dl. Increased BMI and WC were associated with increased CRP levels in non-Hispanic black boys and girls, Hispanic boys, and non-Hispanic white girls, whereas elevated TGL and non-HDL cholesterol and low HDL cholesterol were generally associated with elevated BMI and WC in Hispanic children. TC and LDL cholesterol were not significantly associated with elevated weight in 3- to 6-year-olds. BMI and WC were similar in predicting the same risk factors. In summary, this analysis shows that in preschool-age children, greater BMI and WC are associated with biomarkers that are related to CVD risk, but these associations vary by ethnicity. Child health providers should consider using both BMI and WC to identify young children who may be at risk for elevated CVD biomarkers.

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## INTRODUCTION

The *Healthy People 2010* report identified reducing childhood overweight and obesity as one of the 10 leading public-health priorities for the United States (1). However, the prevalence of childhood overweight and obesity continues to increase, even among preschool-age children and disproportionately affects ethnic minority children (2). One in four US children under age 5 is either overweight ( $\geq 85$ th to  $<95$ th percentile for age- and sex-adjusted percentiles for BMI) or obese ( $\geq 95$ th percentile for age- and sex-adjusted percentiles for BMI) and non-Hispanic blacks and Mexican Americans are affected more than their non-Hispanic white counterparts (2). Overweight preschool-age children are five times as likely to be overweight during adolescence and more than four times as likely to become obese as adults than are their normal-weight counterparts (3).

Childhood-onset obesity is a public health priority because it is a precursor to chronic conditions such as diabetes, cardiovascular disease (CVD), hypertension, stroke, osteoarthritis,

asthma, and certain cancers (4,5) and because it is becoming more prevalent at increasingly younger ages. However, the age in childhood at which increased weight begins to have health-related consequences is unknown. The concern is that childhood obesity will contribute to the earlier onset of overall morbidity and mortality in adulthood, making early intervention critically important (6).

BMI, expressed as weight/height<sup>2</sup> (kg/m<sup>2</sup>) is a common and easily obtained measure of adiposity that can stratify the risk for overweight and obesity among children and adults (2). A greater waist circumference (WC, measured in centimeters) is particularly linked with metabolic syndrome in adults (7,8) and in children and adolescents (9–11) and thus may be a better predictor of type 2 diabetes and CVD than BMI. However, the relationship between WC and BMI with other CVD risk factors among very young children and among various ethnic groups is unknown. Therefore, these relationships were examined in a population-based sample of 3- to 6-year-olds in various ethnic groups in the United States.

<sup>1</sup>Division of Pediatric Clinical Research, Department of Pediatrics, University of Miami Leonard M. Miller School of Medicine, Miami, Florida, USA;

<sup>2</sup>Department of Epidemiology and Public Health, University of Miami Leonard M. Miller School of Medicine, Miami, Florida, USA; <sup>3</sup>Division of Education,

Department of Pediatrics, University of Miami Leonard M. Miller School of Medicine, Miami, Florida, USA. Correspondence: Sarah E. Messiah (smessiah@med.miami.edu)

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## METHODS AND PROCEDURES

## Study population

The periodic National Health and Nutrition Examination Survey (NHANES) uses a stratified, multistage probability design to capture a representative sample of the civilian, noninstitutionalized US population (12). This design allows survey results from two or more periods to be combined to increase the sample size and analytic options. Each 2-year period, and any combination of 2-year periods, is a nationally representative sample. To produce estimates with greater statistical reliability for demographic subgroups and rare events, combining two or more 2-year periods of the survey results is strongly recommended. Therefore, for this study, NHANES data files for 1999–2000, 2001–2002, 2003–2004, 2005–2006, and 2007–2008 were combined to form a single analytic file.

## Eligibility criteria

All Mexican American, other Hispanic (combined to form the Hispanic group), non-Hispanic white, and non-Hispanic black boys and girls aged 3–6 years were included. The following measurements were available for analysis in this age group: age, sex, ethnicity, height and weight (for BMI), WC, total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein cholesterol (LDL), triglyceride (TGL), and C-reactive protein (CRP). LDL cholesterol and TGL were available in the morning-only fasting subsample; we limited the data to those children that had fasted 4–14 h.

## Measures and data collection

Persons selected to participate in the NHANES were invited to be interviewed in their homes. Household interview data were collected with computer-assisted personal interviewing procedures and included demographic, socioeconomic, dietary, and health-related information. After the interview, participants were asked to undergo a physical exam at a Medical Examination Center.

Laboratory methods used at the Medical Examination Centers are described in *The NHANES Laboratory/Medical Technologists Procedures Manual* (13). Briefly, anthropometric measures taken during the standardized examination consisted of barefoot standing height (with a stadiometer), weight with minimal clothing (on a digital, electronic scale), (13) and WC (in the horizontal plane at a point marked just above the right ileum on the midaxillary line, at minimal respiration) (13,14).

LDL cholesterol and TGL were measured on a subsample of all children 3- to 11-years-old who were examined in the morning session

only. LDL cholesterol was calculated using the Friedewald computation ( $LDL = TC - HDL \text{ cholesterol} - (\text{triglyceride}/5)$ ) (15). HDL cholesterol was measured in supernatants after precipitation of apolipoprotein B (apo B)-containing lipoproteins with heparin-manganese chloride and removal of excess manganese by precipitation with sodium bicarbonate. TGLs were analyzed enzymatically with the use of commercial reagents in serum or plasma using a series of coupled reactions in which TGLs are hydrolyzed to produce glycerol. Glycerol is then oxidized using glycerol oxidase, and  $H_2O_2$ , one of the reaction products, is measured. HDL cholesterol was measured by a direct immunoassay technique. The average coefficient of variation for TC for all survey years 1999–2008 was 1.64 (range 1.3–2.2), 2.0 for HDL cholesterol (range 1.8–2.3), and 2.0 for TGL (range 1.8–2.3).

All serum blood samples were collected, processed, stored at  $-20^\circ\text{C}$  and shipped to the Lipid Laboratory, Johns Hopkins University, Baltimore, MD (lipids) for the 1999–2006 surveys and to the University of Minnesota, Minneapolis, MN for the 2007–2008 survey for analysis (16).

CRP was quantified by latex-enhanced nephelometry. Particle-enhanced assays were based on the reaction between a soluble analyte and the corresponding antigen or antibody bound to polystyrene particles. A dilute solution of test sample was mixed with latex particles coated with mouse monoclonal anti-CRP antibodies. CRP present in the test sample forms an antigen-antibody complex with the latex particles. Serum blood specimens are processed, stored, and shipped to University of Washington, Seattle, WA (13). The average coefficient of variation for CRP for all survey years 1999–2008 was 5.61 (range 3.2–9.9).

## Statistical methods

The Centers for Disease Control and Prevention standardized BMI percentiles ( $\geq 85$ th percentile for age and sex to define those who are overweight and  $\geq 95$ th percentile for age and sex to determine those who are obese) were used as cut-points for BMI, and the highest quintile or the 80th percentile for WC (for this population). These applied cut-points correspond to those of older overweight and obese children (1). All ages were collapsed into one category after finding no consistent or significant trends or differences between the specific ages (3-, 4-, 5-, and 6-year-olds).

The highest quintile for CRP, total, LDL, and non-HDL (TC-HDL cholesterol) cholesterol, and TGL and the lowest quintile for HDL cholesterol for the entire study population were used as cutoff points for analysis. These cutoffs are consistent with the percentages used for abnormal values in studies of older children and were used because no standardized cutoffs currently exist for this age group (17,18).

**Table 1** Estimates of cardiovascular disease risk factors among 3- to 6-year-old children, 1999–2008 National Health and Nutrition Examination Surveys

Characteristics	Sample, N	Weighted population, N	National estimate, mean	95% CI for the national estimate
Age	3,644	14,535,068	4.52	4.48–4.57
<i>Anthropometrics</i>				
BMI ( $\text{kg}/\text{m}^2$ )	3,555	14,165,622	16.3	16.20–16.39
BMI percentile	3,555	14,165,622	58.90	57.49–60.32
Waist circumference (cm)	3,456	13,795,833	53.86	53.56–54.17
<i>CVD risk factor</i>				
C-reactive protein (mg/dl)	2,521	9,679,388	0.16	0.13–0.19
HDL cholesterol (mg/dl)	1,752	6,885,787	51.95	51.18–52.72
LDL cholesterol (mg/dl)	625	5,807,247	92.75	90.40–95.11
Total cholesterol (mg/dl)	1,754	6,895,867	161.9	160.07–163.68
Triglyceride (mg/dl)	626	5,811,466	84.87	77.82–91.91
Non-HDL cholesterol (mg/dl)	1,751	6,884,098	109.91	108.15–111.67

CI, confidence interval; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Relationships between these anthropometric measurements and CVD risk factors for each age–sex–ethnic group were analyzed with separate logistic regression analyses which included survey year in order to control for possible variations in the data that resulted from combining data from multiple surveys. Finally, logistic regression analysis was performed to predict elevated levels of all CVD risk factors using both BMI and WC as continuous measures. There was no adjustment for potential confounding factors such as dietary patterns or physical activity level because the children in this analysis were so young and their eating and exercise patterns tend to be inconsistent as a result; however, survey year was included in the logistic regression models to control for the effects of combining data over survey years. Separate models were run for gender by race/ethnicity categories. Multiple comparisons were not controlled for because we considered these comparisons to address a priori hypotheses.

$\alpha$  was set at 0.05 and all tests were two-tailed. Analyses were performed with SAS SURVEY procedures (SAS version 9.2, SAS Institute, Cary, NC) to accommodate the weighting and design effects of the complex survey data.

## RESULTS

Data from 3,644 (weighted sample size, 14,535,068) children were analyzed (Table 1). Age was evenly distributed (25% for 3-, 4-, 5-, and 6-year-olds), as was gender (51% boys, 49% girls) and there was 62% non-Hispanic white, 23% Hispanic, and 15% non-Hispanic black. Overall, 13.8% of children had a BMI greater than the 95<sup>th</sup> percentile and 26.4% were above the 85<sup>th</sup> percentile for age and sex (Table 2).

Among non-Hispanic black and white girls and non-Hispanic black boys, higher levels of CRP were significantly more likely to occur among those who had a BMI above the 85<sup>th</sup> and 95<sup>th</sup> percentile when compared to those who were below these percentiles (Figures 1 and 2). HDL cholesterol levels were significantly more likely to be lower in both Hispanic girls and boys above the 85<sup>th</sup> and 95<sup>th</sup> percentiles of BMI than were their peers in the same sex and ethnic group who were below these percentiles. This finding was consistent for non-HDL cholesterol in Hispanic boys above the 85<sup>th</sup> and 95<sup>th</sup> percentiles of BMI vs. those below these percentiles. TGL levels were significantly more likely to be higher for Hispanic boys above the 85<sup>th</sup> percentile of BMI than they were for their counterparts whose levels were below these percentiles.

Similarly, non-Hispanic black and white girls and non-Hispanic black and Hispanic boys at the highest quintile for WC were significantly more likely to have higher CRP levels than their counterparts whose values were below these WC percentiles (Figure 3). HDL cholesterol levels were significantly more likely to be lower in Hispanic girls and boys with a WC in the highest quintile vs. those with a smaller WC. TGL levels in the highest quintile were more frequently found among Hispanic boys who had a WC in the highest quintile vs. those below.

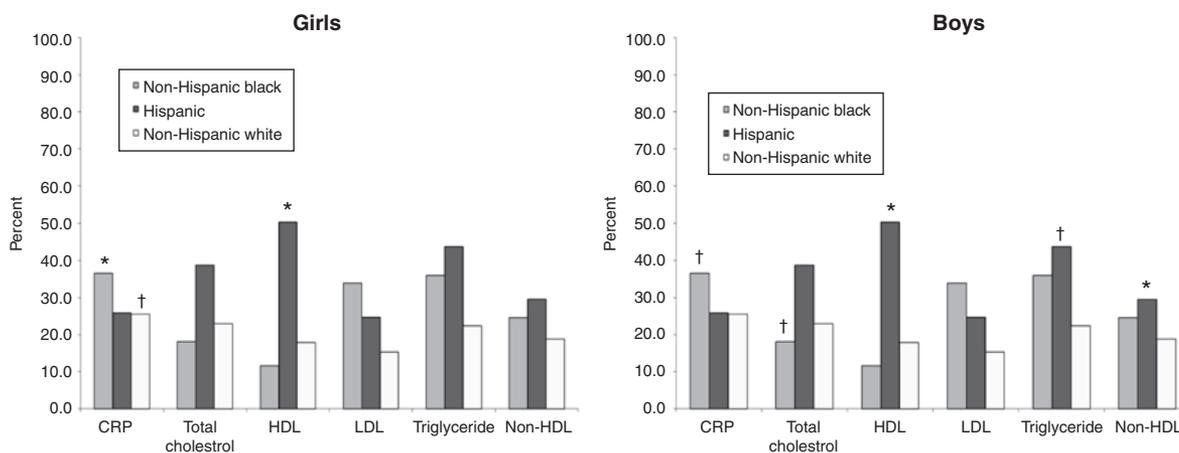
Consistent with the  $\chi^2$  analysis, logistic regression analysis showed that the highest BMIs and WCs were significantly associated with the highest levels of CRP among non-Hispanic black and white girls ( $P \leq 0.001$  for all) and among non-Hispanic black and Hispanic boys. BMI and WC were associated with the lowest quintile of HDL cholesterol, and the highest quintile of non-HDL cholesterol and TGL in Hispanic boys (Table 3).

**Table 2 Cutoff values defining cardiovascular disease risk factors for 3- to 6-year-old children by sex and ethnicity**

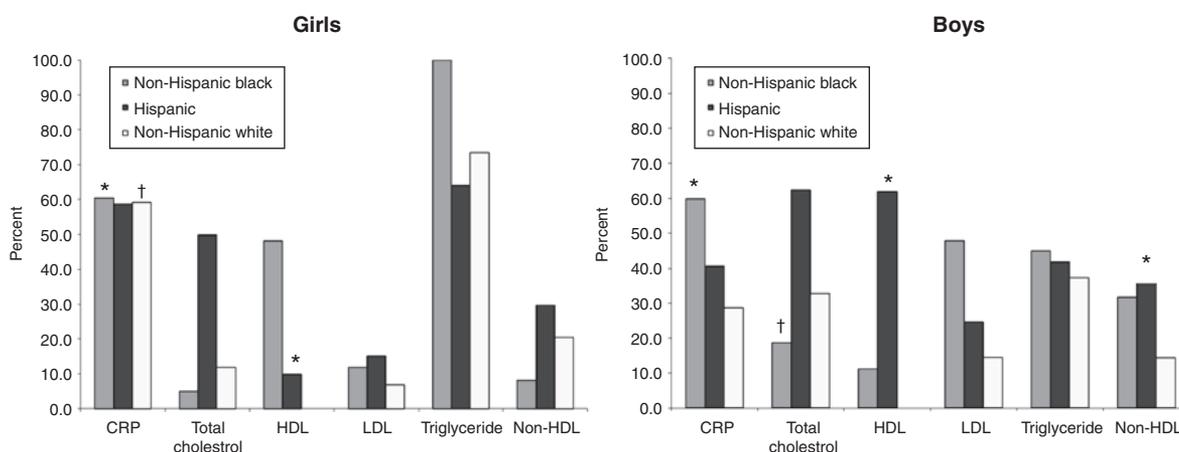
Group	n	BMI <sup>a</sup> , kg/m <sup>2</sup>		Waist Circumference <sup>a</sup> , cm		C-reactive protein, mg/dl		Total cholesterol, mg/dl		HDL cholesterol, mg/dl		LDL cholesterol, mg/dl		Triglyceride, mg/dl		Non-HDL cholesterol, mg/dl	
		85 <sup>th</sup> percentile <sup>b</sup>	95 <sup>th</sup> percentile <sup>c</sup>	n	Highest quintile	n	Highest quintile	n	Highest quintile	n	Lowest quintile	n	Highest quintile	n	Highest quintile	n	Highest quintile
Overall	3,555	26.5	13.8	3,456	57.2	2,521	0.13	1,754	182	1,752	42	625	109	626	103	1,751	128
Girls																	
Overall	1,740	25.3	12.9	1,689	57.1	1,208	0.14	852	183	850	42	160	109	160	98	850	129
Non-Hispanic black	516	26.2	13.0	506	55.6	365	0.10	268	182	268	44	100	112	100	87	268	127
Hispanic	694	27.8	14.6	670	57.9	502	0.20	346	181	344	41	113	102	113	110	344	127
Non-Hispanic white	530	21.3	10.8	513	57.0	341	0.11	238	188	238	40	90	109	90	105	238	135
Boys																	
Overall	1,815	27.6	14.7	1,767	57.2	1,313	0.12	902	181	902	43	158	111	158	107	901	126
Non-Hispanic black	555	23.4	12.4	538	55.5	391	0.08	272	183	272	46	120	108	120	85	272	123
Hispanic	684	33.9	19.0	660	59.0	516	0.16	368	179	369	42	119	105	120	112	368	124
Non-Hispanic white	576	24.0	11.8	569	57.2	406	0.10	262	181	261	41	83	112	83	118	261	130

HDL, high-density lipoprotein; LDL, low-density lipoprotein.

<sup>a</sup>Centers for Disease Control and Prevention standardized cutoff values. <sup>b</sup>Cutoff point for defining overweight. <sup>c</sup>Cutoff point for defining obesity.



**Figure 1** Overweight 3- to 6-year-olds, as defined by BMI  $\geq$ 85th percentile for age and sex, and the prevalence of abnormal cardiovascular disease risk factors represented in the 1999–2008 NHANES data, by sex and ethnicity. \* $P < 0.01$ ; † $P < 0.05$ , when comparing children within the specific sex-ethnic group who had an abnormal cardiovascular risk factor value (highest quintile for CRP, total and LDL cholesterol, and triglyceride and the lowest quintile for HDL cholesterol) and a normal BMI value ( $<$ 85th percentile for age and sex) with those with an abnormal cardiovascular risk factor value and elevated BMI ( $\geq$ 85th percentile for age and sex). CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NHANES, National Health and Nutrition Examination Survey.



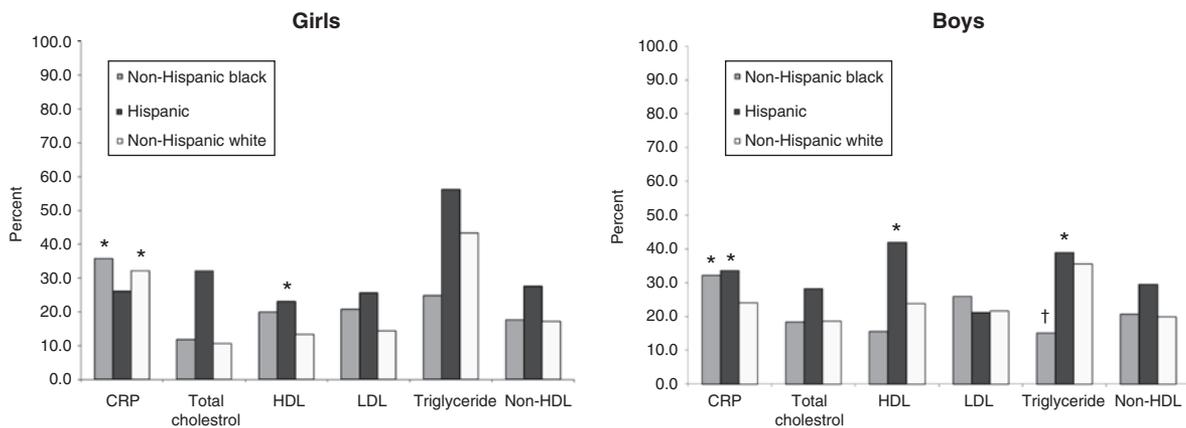
**Figure 2** Obese 3- to 6-year-olds, as defined by BMI  $\geq$ 95th percentile for age and sex and the prevalence of abnormal cardiovascular disease risk factors represented in the 1999–2008 NHANES data, by sex and ethnicity. \* $P < 0.01$ ; † $P < 0.05$ , when comparing children within the specific sex-ethnic group who had an abnormal cardiovascular risk factor value (highest quintile for CRP, total and LDL cholesterol, and triglyceride and the lowest quintile for HDL cholesterol) and a normal BMI value ( $<$ 95th percentile for age and sex) with those with an abnormal cardiovascular risk factor value and elevated BMI ( $\geq$ 95th percentile for age and sex). CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NHANES, National Health and Nutrition Examination Survey.

## DISCUSSION

The results reported here indicate that the cardiovascular health risks of being overweight begin early in life but vary by ethnic group. Specifically, we found that both increased BMI and WC were associated with biomarkers associated with inflammation (as indicated by CRP levels) in non-Hispanic black boys and girls, Hispanic boys, and non-Hispanic white girls, whereas elevated TGL, non-HDL cholesterol, and low HDL cholesterol were associated with elevated BMI and WC in Hispanic children in general. TC and LDL cholesterol were not significantly associated with elevated weight in 3- to 6-year-olds in general. These findings indicate that the current obesity epidemic is probably associated with risk factors for CVD but varies by ethnic group, even in preschool children, suggesting a potential threat to cardiometabolic health.

Because our findings are among very young children, they may have implications throughout childhood. Other studies have noted that several CVD risk factors persist strongly and consistently through childhood into adulthood. For example, our findings are consistent with those of Nader *et al.* (3), who reported that preschool children who were above the 50th percentile of BMI for age and sex at any time during their preschool years had a sixfold increased likelihood of being overweight in later childhood, and of Valerio *et al.* (19), who reported that BMI z-scores and overweight–obese status remained more or less constant well into later childhood.

Other longitudinal studies have shown that cardiometabolic disease risk factors present in childhood predict adult disease. The Princeton Lipid Research Clinics Follow-up Study showed



**Figure 3** Prevalence of abnormal cardiovascular disease risk factors among 3- to 6-year-olds, as defined by highest quintile of waist circumference, represented in the 1999–2008 NHANES data, by sex and ethnicity. \* $P < 0.01$ ; † $P < 0.05$ , when comparing children within the specific sex-ethnic group who had an abnormal cardiovascular risk factor value (highest quintile CRP, total and LDL cholesterol, and triglyceride and the lowest quintile for HDL cholesterol) and a normal waist circumference (< highest quintile) vs. children within the specific sex-ethnic group who had an abnormal cardiovascular risk factor value (highest quintile for CRP, total and LDL cholesterol, and triglyceride and the lowest quintile for HDL cholesterol) and an abnormal waist circumference (highest quintile). CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NHANES, National Health and Nutrition Examination Survey.

that over 30 years, the risk for CVD was nine times as high, and that for type 2 diabetes mellitus was four times as high, in children with metabolic syndrome than in children without the syndrome, after adjusting for age, sex, ethnicity, and family history (20). This same study reported that the first appearance of differences between adults with and without metabolic syndrome occurred at ages 8 and 13 for BMI and at ages 6 and 13 for WC in boys and girls, respectively (21). The authors concluded that children with a BMI and WC at or above the 90th percentile are at increased risk for the adult metabolic syndrome.

Identifying young children with lipid elevations is critical to tracking their CVD risk profile through childhood and has been shown to track strongly into adulthood (22). In addition to elevated BMI and WC, abnormal lipid levels that are left undiagnosed and/or untreated in childhood are linked to increased risk for CVD in adulthood (22). Our results here indicate that overweight Hispanic children in particular may need more intensive lifestyle modification recommendations from their primary care physician or pediatrician, due to their elevated TGLs and low HDL cholesterol. These findings are similar to previous NHANES III analysis that reported Hispanic children have lower HDL and higher TGL concentrations vs. non-Hispanic black and white children (23). Furthermore, the Bogalusa Heart Study reported that ~70% of the children with elevated cholesterol levels continued to have cholesterol elevations in young adulthood (24).

Although CRP is a well-documented biomarker of inflammation, elevated CRP levels in this age group may not indicate the explicit inflammation attributed to CVD risk. Our findings do suggest that CRP is more likely to be higher in young children who are overweight than in those who are not, especially in certain ethnic groups. In addition, less than 1.3% of our sample had CRP values above 2.0, indicating that few of the children in this study had CRP values that might be associated

with acute infections caused by the common cold, or influenza for example (typically associated with CRP levels >10). These findings certainly warrant more investigation into the specific cause of increased levels of CRP among young overweight children.

Because these CVD risk factors were associated with higher anthropometric outcomes in these preschool-age children, further longitudinal evaluations will be necessary to determine whether they persist and track with clinical disease (25). It will also be necessary to examine if ethnic group differences in CVD persist throughout later childhood into adulthood. Long-term follow-up of well-characterized pediatric cohorts into adulthood are needed to define appropriate age-specific, sex-specific, and race- or ethnicity-specific CVD risks in childhood. Our findings here suggest that race- or ethnicity-specific CVD risks begin early in life and longitudinal studies would allow earlier and more aggressive lifestyle interventions as well as more appropriate pharmacologic treatment of these children, perhaps designated by their race/ethnic background.

Longitudinal studies have reported that elevated CRP was a predictor of adult metabolic syndrome and could help identify children and adolescents at greater risk of the syndrome later in life (6). Indeed, some researchers have suggested that adding a measure of CRP to adult standard cholesterol evaluation protocols would improve the ability to predict CVD risk (25). Our current analysis suggests there might be great utility in adding a lipid profile to identify the risk of chronic disease, not only in adults but in young children as well, especially for those with elevated BMI and higher WC.

Clinically, BMI and WC screening may detect children in this age group who are at higher risk for subsequent CVD (26,27). We have reported elsewhere that age-, sex-, and ethnicity- or race-specific threshold values for BMI and WC may have great clinical utility in identifying older children at risk for CVD (27). Both measures are potentially valuable in detecting the risk of chronic

**Table 3 Odds ratios and 95% confidence intervals for predicting elevated levels of cardiovascular disease risk factors by BMI and waist circumference for 3- to 6-year-olds by sex and ethnicity**

	C-reactive protein		HDL cholesterol		LDL cholesterol		Total cholesterol		Triglyceride		Non-HDL cholesterol	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
<i>BMI</i>												
<i>Girls</i>												
Non-Hispanic black	1.25 (1.13–1.38)	<0.001	1.00 (0.90–1.12)	0.98	1.13 (0.84–1.52)	0.41	1.01 (0.91–1.12)	0.86	1.11 (0.87–1.41)	0.39	0.96 (0.85–1.10)	0.59
Hispanic	1.10 (0.97–1.25)	0.12	1.28 (1.13–1.46)	<0.001	0.97 (0.75–1.25)	0.80	1.03 (0.91–1.16)	0.65	1.23 (0.92–1.64)	0.17	1.07 (0.95–1.21)	0.25
Non-Hispanic white	1.26 (1.11–1.44)	<0.001	0.93 (0.78–1.12)	0.47	0.73 (0.47–1.11)	0.14	0.85 (0.72–1.02)	0.07	1.02 (0.62–1.66)	0.94	0.95 (0.80–1.12)	0.54
<i>Boys</i>												
Non-Hispanic black	1.22 (1.11–1.34)	<0.001	0.99 (0.86–1.13)	0.83	0.75 (0.56–1.01)	0.06	1.12 (0.99–1.28)	0.07	0.87 (0.66–1.14)	0.30	1.07 (0.95–1.21)	0.27
Hispanic	1.09 (1.00–1.18)	0.034	1.30 (1.16–1.45)	<0.001	0.96 (0.78–1.19)	0.74	1.03 (0.91–1.16)	0.64	1.31 (1.10–1.56)	0.002	1.19 (1.07–1.32)	0.002
Non-Hispanic white	1.06 (0.90–1.26)	0.49	1.23 (1.09–1.39)	0.001	1.18 (0.84–1.65)	0.33	0.91 (0.82–1.02)	0.10	1.01 (0.73–1.41)	0.93	0.97 (0.86–1.09)	0.60
<i>Waist circumference</i>												
<i>Girls</i>												
Non-Hispanic black	1.08 (1.04–1.13)	0.001	0.97 (0.92–1.02)	0.23	1.06 (0.95–1.18)	0.30	1.00 (0.96–1.04)	0.85	1.00 (0.90–1.12)	0.97	0.98 (0.93–1.02)	0.32
Hispanic	1.03 (0.99–1.08)	0.18	1.09 (1.03–1.15)	0.002	1.01 (0.94–1.09)	0.72	1.03 (0.99–1.07)	0.11	1.08 (0.96–1.22)	0.20	1.05 (1.01–1.09)	0.026
Non-Hispanic white	1.09 (1.05–1.13)	<0.001	0.96 (0.90–1.02)	0.17	0.94 (0.85–1.05)	0.28	0.96 (0.91–1.02)	0.21	1.08 (0.94–1.25)	0.27	0.97 (0.92–1.04)	0.42
<i>Boys</i>												
Non-Hispanic black	1.07 (1.03–1.12)	<0.001	0.99 (0.94–1.04)	0.62	0.95 (0.86–1.04)	0.25	1.03 (0.98–1.08)	0.20	0.92 (0.84–1.00)	0.047	1.02 (0.98–1.07)	0.28
Hispanic	1.05 (1.02–1.08)	0.001	1.06 (1.02–1.10)	0.001	1.00 (0.94–1.07)	0.99	1.02 (0.98–1.06)	0.41	1.13 (1.05–1.21)	0.001	1.05 (1.02–1.09)	0.003
Non-Hispanic white	1.03 (0.98–1.08)	0.21	1.06 (1.02–1.11)	0.006	1.05 (0.93–1.18)	0.46	0.97 (0.93–1.01)	0.09	0.98 (0.82–1.18)	0.86	0.97 (0.91–1.02)	0.24

CI, confidence interval; OR, odds ratio.

disease at the very earliest stages of life, are minimally invasive, and can be acquired with relative ease in virtually any setting. Children as young as 8-years-old can have metabolic syndrome (28) so it is critical to identify those at risk as early as possible (29). Then, modifiable risk factors, such as nutrition (30), and physical activity (31), can be discussed with the family during well-child visits (32). Our analysis supports starting these discussions as early in the child's life as possible, as well as expanding counseling to families about CVD and risk prevention, particularly among those families with overweight children.

#### Limitations of the study

In a cross-sectional study, causality cannot be inferred. Blood pressure, insulin, and glucose measures are not collected in this age group and thus were not available for analysis, yet they are important components of metabolic syndrome and risk factors for adult-onset CVD and diabetes. Because LDL and TGLs were measured on a subsample of the surveyed children, analyses for these variables may not have sufficient statistical power to detect significant differences. Finally, dietary and physical activity level data were not included because the children in this analysis were so young and their eating and exercise patterns tend to be inconsistent as a result. While these limitations may not produce a comprehensive analysis of the consequential health effects of the current obesity epidemic in this age group, they nevertheless do provide important and useful information that has not been previously reported.

#### Conclusion

Our large, population-based analysis shows that easily obtained anthropometric measures in very young children, such as BMI and WC, are associated with biomarkers of CVD risk. This age group is important to monitor for the onset of future chronic disease risk due to the current obesity epidemic (33). Pediatricians should consider both BMI and WC as potential clinical tools to detect preschool-age children who may be at higher risk for CVD.

#### DISCLOSURE

The authors declared no conflict of interest.

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#### REFERENCES

1. U.S. Department of Health and Human Services. *Healthy People 2010: Understanding and Improving Health*, 2nd edn. U.S. Government Printing Office: Washington, DC, 2000. <<http://www.healthypeople.gov>>. Accessed 15 July 2010.
2. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. *JAMA* 2010;303:242-249.
3. Nader PR, O'Brien M, Houts R *et al.*; National Institute of Child Health and Human Development Early Child Care Research Network. Identifying risk for obesity in early childhood. *Pediatrics* 2006;118:e594-e601.
4. Centers for Disease Control and Prevention, 2007. Obesity among adults in the United States—no statistically significant change since 2003-2004. <<http://www.cdc.gov/nchs/pressroom/07newsreleases/obesity.htm>>. Accessed 6 April 2010.
5. The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity. Public Health Service, Office of the Surgeon General: Rockville, MD, 2001.
6. Steinberger J, Daniels SR. Obesity, insulin resistance, diabetes, and cardiovascular risk in children: an American Heart Association scientific statement from the Atherosclerosis, Hypertension, and Obesity in the Young Committee (Council on Cardiovascular Disease in the Young) and the Diabetes Committee (Council on Nutrition, Physical Activity, and Metabolism). *Circulation* 2003;107:1448-1453.
7. Morrison JA, Friedman LA, Gray-McGuire C. Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: the Princeton Lipid Research Clinics Follow-up Study. *Pediatrics* 2007;120:340-345.
8. Shen W, Punyanitya M, Chen J *et al.* Waist circumference correlates with metabolic syndrome indicators better than percentage fat. *Obesity (Silver Spring)* 2006;14:727-736.
9. Li C, Ford ES, Mokdad AH, Cook S. Recent trends in waist circumference and waist-height ratio among US children and adolescents. *Pediatrics* 2006;118:e1390-e1398.
10. Esmailzadeh A, Mirmiran P, Azizi F. Clustering of metabolic abnormalities in adolescents with the hypertriglyceridemic waist phenotype. *Am J Clin Nutr* 2006;83:36-46.
11. Maffei C, Pietrobelli A, Grezzani A, Provera S, Tatò L. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res* 2001;9:179-187.
12. Centers for Disease Control and Prevention. National Center for Health Statistics. National Health and Nutrition Examination Survey Data. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention: Hyattsville, MD, 2009. (<[http://www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004/questexam03\\_04.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004/questexam03_04.htm)>).
13. The Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey: Laboratory Procedures Manual; 2004. <<http://www.cdc.gov/nchs/data/nhanes/lab1-6.pdf>>. Accessed 11 January 2011.
14. Chumlea NC, Kuczmarski RJ. Using a bony landmark to measure waist circumference. *J Am Diet Assoc* 1995;95:12.
15. Bachorik PS, Walker RE, Virgil DG. High-density-lipoprotein cholesterol in heparin-MnCl<sub>2</sub> supernates determined with the Dow enzymic method after precipitation of Mn<sup>2+</sup> with HCO<sub>3</sub><sup>-</sup>. *Clin Chem* 1984;30:839-842.
16. The Centers for Disease Control and Prevention. NHANES 1999-2000 Public Release Dataset—September 2003: Laboratory 10AM—Glucose, Insulin, and C-peptide; 2004. <<http://www.cdc.gov/nchs/data/nhanes/frequency/10amdoc.pdf>>. Accessed 11 January 2011.
17. 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;157:821-827.
18. de Ferranti SD, Gauvreau K, Ludwig DS, Newburger JW, Rifai N. Inflammation and changes in metabolic syndrome abnormalities in US adolescents: findings from the 1988-1994 and 1999-2000 National Health and Nutrition Examination Surveys. *Clin Chem* 2006;52:1325-1330.
19. Valerio G, D'Amico O, Adinolfi M *et al.* Determinants of weight gain in children from 7 to 10 years. *Nutr Metab Cardiovasc Dis* 2006;16:272-278.
20. Huang TT, Nansel TR, Belsheim AR, Morrison JA. Sensitivity, specificity, and predictive values of pediatric metabolic syndrome components in relation to adult metabolic syndrome: the Princeton LRC follow-up study. *J Pediatr* 2008;152:185-190.
21. Morrison JA, Friedman LA, Gray-McGuire C. Metabolic syndrome in childhood predicts adult cardiovascular disease 25 years later: the Princeton Lipid Research Clinics Follow-up Study. *Pediatrics* 2007;120:340-345.
22. Daniels SR, Greer FR. Lipid screening and cardiovascular health in childhood. *Pediatrics* 2008;122:198-208.
23. Hickman TB, Briefel RR, Carroll MD *et al.* Distributions and trends of serum lipid levels among United States children and adolescents ages 4-19 years: data from the Third National Health and Nutrition Examination Survey. *Prev Med* 1998;27:879-890.
24. Webber LS, Srinivasan SR, Wattigney WA, Berenson GS. Tracking of serum lipids and lipoproteins from childhood to adulthood. The Bogalusa Heart Study. *Am J Epidemiol* 1991;133:884-899.
25. Pearson TA, Mensah GA, Alexander RW *et al.*; Centers for Disease Control and Prevention; American Heart Association. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation* 2003;107:499-511.

26. de Ferranti SD, Gauvreau K, Ludwig DS *et al*. Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. *Circulation* 2004;110:2494–2497.
27. Messiah SE, Arheart KL, Lipshultz SE, Miller TL. Body mass index, waist circumference, and cardiovascular risk factors in adolescents. *J Pediatr* 2008;153:845–850.
28. Messiah SE, Arheart KL, Luke B, Lipshultz SE, Miller TL. Relationship between body mass index and metabolic syndrome risk factors among US 8- to 14-year-olds, 1999 to 2002. *J Pediatr* 2008;153:215–221.
29. Lipshultz SE, Wilkinson JD, Messiah SE, Miller TL. Clinical research directions in pediatric cardiology. *Curr Opin Pediatr* 2009;21:585–593.
30. Lee Y, Mitchell DC, Smiciklas-Wright H, Birch LL. Diet quality, nutrient intake, weight status, and feeding environments of girls meeting or exceeding recommendations for total dietary fat of the American Academy of Pediatrics. *Pediatrics* 2001;107:E95.
31. Berkey CS, Rockett HR, Gillman MW, Colditz GA. One-year changes in activity and in inactivity among 10- to 15-year-old boys and girls: relationship to change in body mass index. *Pediatrics* 2003;111:836–843.
32. Strauss RS, Knight J. Influence of the home environment on the development of obesity in children. *Pediatrics* 1999;103:e85.
33. Yanovski SZ, Yanovski JA. Obesity prevalence in the United States—up, down, or sideways? *N Engl J Med* 2011;364:987–989.