Health Outcomes of Youth in Clinical Pediatric Weight Management Programs in POWER

Seema Kumar, MD[1], Eileen C. King, PhD[2,3], Amy L. Christison, MD[4], Aaron S. Kelly, PhD[5], Adolfo J. Ariza, MD[6,7], Claudia Borzutzky, MD[8,9], Suzanne Cuda, MD[10], and Shelley Kirk, PhD, RD, LD[3,11], on behalf of the POWER Work Group*

Objective To describe treatment outcomes of children and adolescents enrolled in the Pediatric Obesity Weight Evaluation Registry, a consortium of multicomponent pediatric weight management programs in the US.

Study design This multicenter prospective observational cohort study, established in 2013, includes youth (2-18 years of age) with obesity enrolled from 31 Pediatric Obesity Weight Evaluation Registry (POWER) sites over a 2-year period and followed up to 12 months. Weight status was evaluated by the percentage of the 95th percentile for body mass index (%BMIp95). Associations of weight status outcomes with patient characteristics and program exposure were analyzed with multivariable mixed effects modeling.

Results We included 6454 children and adolescents (median age, 11 years; IQR, 9-14 years; 53% white, 32% Hispanic; 73% with severe obesity) who were enrolled in POWER. Median changes in %BMIp95 for this cohort were −1.88 (IQR, −5.8 to 1.4), −2.50 (IQR, −7.4 to 1.8), −2.86 (IQR, −8.7 to 1.9), at 4-6, 7-9, and 10-12 of months follow-up, respectively (all $P < .05$). Older age (≥12 years), greater severity of obesity, and Hispanic race/ethnicity were associated with better improvement in %BMIp95. A 5-percentage point decrease in %BMIp95 was associated with improvement in cardiometabolic risk factors.

Conclusions Overall, treatment in pediatric weight management programs is associated with a modest median decrease in BMI as measured by change in %BMIp95. Further studies are needed to confirm these findings, as well as to identify additional strategies to enhance the effectiveness of these multicomponent interventions for youth with severe obesity. *(J Pediatr 2019;208:57-65)*.

Trial registration ClinicalTrials.gov: NCT02121132.

The prevalence of childhood obesity from 2015 to 2016 in the US was 18.5% and 6.0% for severe obesity.[1,2] Childhood obesity is associated with adverse health consequences, including type 2 diabetes, dyslipidemia, and hypertension.[3-5] Although lifestyle modification is considered the cornerstone of pediatric obesity treatment, there is limited evidence from a small number of short-term studies showing its efficacy in reduction in body mass index (BMI) and cardiometabolic risk factor improvements.[6-8] Additional investigation is needed to better understand the effectiveness of pediatric obesity treatment provided in tertiary care settings, where program design, staffing, patient population served, and resources available to support program services are variable.[9]

In 2013, the Pediatric Obesity Weight Evaluation Registry (POWER) was established. POWER serves as a centralized data repository for the on-going collection and maintenance of demographic and clinical data from multicomponent pediatric weight management (PWM) programs across the nation.[10] Similar registries for pediatric obesity exist in Europe[11] and Canada.[12] In 2007, the Expert Committee established guidelines suggesting 4 stages of obesity care.[6] Stage 3

ALT	Alanine aminotransferase
BMI	Body mass index
%BMIp95	Percentage of the 95th percentile for BMI
ΔBMIp95	Difference in BMI units from the 95th percentile for BMI
BMIz	BMI z score
DSCF	Dwass, Steel, Critchlow-Figner
HbA1c	Hemoglobin A1c
HDL-C	High density lipoprotein cholesterol
POWER	Pediatric Obesity Weight Evaluation Registry
PWM	Pediatric weight management
TG	Triglycerides

From the [1]Division of Pediatric Endocrinology and Metabolism, Department of Pediatric and Adolescent Medicine, Mayo Clinic, Rochester, MN; [2]Division of Biostatistics and Epidemiology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH; [3]Department of Pediatrics, University of Cincinnati, College of Medicine, Cincinnati, OH; [4]Department of Pediatrics, University of Illinois College of Medicine at Peoria, Peoria, IL; [5]Department of Pediatrics, Center for Pediatric Obesity Medicine, University of Minnesota Medical School, Minneapolis, MN; [6]Department of Pediatrics, Ann & Robert H. Lurie Children's Hospital of Chicago; [7]Pediatrics, Feinberg School of Medicine, Northwestern University, Chicago, IL; [8]Department of Pediatrics, Keck School of Medicine of USC; [9]Diabetes and Obesity Program, Children's Hospital Los Angeles, Los Angeles, CA; [10]Department of Pediatrics, Pediatric Weight Management, Children's Hospital of San Antonio, Baylor College of Medicine, Houston, TX; and [11]The Heart Institute, Center for Better Health and Nutrition, Cincinnati Children's Hospital Medical Center, Cincinnati, OH

*List of additional members of the POWER Work Group is available at www.jpeds.com (**Appendix**).

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care suggests necessary program components of a comprehensive multidisciplinary intervention.

The identification of key program components is an important area for research. Previous studies have associated greater decreases in BMI z scores (BMIz) with increased number of contact hours.[8] Younger age and lower BMI at baseline have also been associated with improved BMI change.[13] However, these studies have examined impact of exposure to components/patient characteristics within a specific program but have not evaluated care between programs. Another factor that greatly impacts outcome evaluation is program attrition rate, which is generally high in obesity care programs.[14]

The outcomes of such programs could be measured across a wide array of metrics, including changes in particular health behaviors, changes in fitness, changes in body composition, and short- and long-term changes in BMI. However, interpreting change in BMI is complicated by characteristics of the age- and sex-dependent BMI reference standards. Therefore change in BMIz has been used as the outcome metric.[15,16] Recent reports suggest limitations in the commonly used metrics of BMIz and change in the BMIz, particularly for children <5 years of age and in children with severe obesity.[17-19] This limitation is explained by the flattening of BMIz (or SD) at values significantly above the 95th percentile, despite the wide variance of BMIs in youth with severe obesity, which therefore leads to an underestimation of clinically significant improvement in weight status. Thus, recent literature proposes that, rather than using BMIz as a metric, change in weight status for youth with severe obesity should be expressed relative to the BMI 95th percentile,[20] either as a difference in percentage units or a difference in BMI units.[18,21] The percentage of the 95th percentile for BMI (%BMIp95) is the ratio of the BMI relative to the BMI at the 95th percentile (based on sex and age) multiplied by 100. The difference in BMI units from the 95th percentile for BMI (ΔBMIp95) assesses the BMI unit distance from the BMI value at the 95th percentile.[18,21,22]

We describe treatment outcomes of youth with obesity enrolled in multicomponent PWM programs using the metrics of %BMIp95 and ΔBMIp95. The objective of the study was to describe changes in BMI in relation to patient characteristics, number of clinic visits, and changes in cardiometabolic risk factors as reflected by blood pressure measurements and laboratory measures.

Methods

All participating POWER sites provide multicomponent PWM services that include medical, nutrition, physical activity, and behavioral assessments and counseling. Other than the requirement for sites to offer a multicomponent intervention for youth with obesity, there are no other specifications required in regard to program duration, clinical staffing, or credentials/training of personnel. However, among POWER sites that completed a program profile survey (n = 30), common features reported included multidisciplinary clinic visits (93%), offer on-going treatment (83%), and have variable visit frequency to help tailor treatment to best fit patient needs (80%). Although all PWM program patients are required to have a medical evaluation at baseline, there are no minimum requirements for numbers of provider contact hours with specific interventionists such as registered dietitians or psychologists, frequency of follow-up visits, or duration/intensity of exercise needed during the program.

Sites obtain institutional review board approval for the POWER study protocol including informed consent/assent from patient families, and collect a required set of patient demographic and clinical information.[10] This information is then submitted to the POWER Data Coordinating Center at Cincinnati Children's Hospital Medical Center.[10] To participate in POWER, sites paid a $5000 enrollment fee to cover administrative costs and regulatory oversight of the registry, in addition to data management and analysis for each 2-year cycle. This prospective observational cohort study includes patient information from all POWER sites that submitted data to the Data Coordinating Center between May 2014 and December 2016. The POWER study is registered with Clinical.Trials.gov (NCT02121132).

Subjects included youth 2-18 years of age with obesity (age- and sex-specific BMI ≥95th percentile), ability to consent/assent in English or Spanish, and new or returning patients who had not received care at that program in the prior 3 years. Patients who had undergone bariatric surgery were excluded. Demographics, anthropometric measurements, and laboratory data from the initial medical visit and all subsequent follow-up visits were submitted as a limited dataset in accordance with the Health Insurance Portability and Accountability Act regulations. POWER did not impose any standardized protocol for the frequency of clinical encounters.

The primary study outcome was change from baseline in the %BMIp95 at 3 follow-up time periods: 4-6 months, 7-9 months, and 10-12 months. Additional weight status outcomes included ΔBMIp95 and percent change from baseline for BMI (for youth older >12 years of age). These 3 outcome measures were selected to gain perspective on changes in weight status using BMI reference curves-related metrics (%BMIp95 and ΔBMIp95), in addition to relative changes in actual BMI (percent change BMI).[17-19,21,22] Obesity class I was defined as BMI between 100% and 119% of the 95th percentile. Severe obesity was defined as a BMI between 120% and 139% of the 95th percentile or BMI between 35 and 39 kg/m^2, whichever was lower (class 2), or patients with a BMI of ≥140% of the 95th percentile or BMI of ≥40 kg/m^2, whichever was lower (class 3).[3] Level of program exposure was based on the number of follow-up visits that included a height and weight measurement during each time period.

Secondary outcomes were limited to youth age 6-18 years and included change in blood pressure percentiles and cardiometabolic laboratory measures, including hemoglobin A1c (HbA1c), triglycerides (TG), alanine aminotransferase

(ALT), high-density lipoprotein cholesterol (HDL-C), non-HDL-C, and fasting glucose. Blood pressure measurements were based on percentiles of systolic and diastolic readings for age, sex, and height.[23] Cutoff values for laboratory measures were based on expert recommendations: fasting blood glucose, normal <100 mg/dL; prediabetes, 100-125 mg/dL, and diabetes, ≥126 mg/dL.[24] For HbA1c (%) cutoffs were: prediabetes, 5.7%-6.4% and diabetes, ≥6.5%.[24] For TG, cutoffs for those ages 0-9 years were: borderline high, 75-99 mg/dL and high, ≥100 mg/dL; for those ages 10-19 years: borderline high, 90-129 mg/dL and high, ≥130 mg/dL; for non-HDL-C, cutoffs were: borderline high, 120-144 mg/dL and high, ≥145 mg/dL[25]; and for ALT (U/L), the cutoff was: high, ≥40 U/L (boys and girls). Laboratory measures were further categorized into normal or abnormal status. Prediabetes and diabetes were classified as abnormal for HbA1c and fasting glucose; borderline high or high non-HDL-C or TG as abnormal; and high ALT was classified as abnormal.

Statistical Analyses

Patient demographics, height, weight, and blood pressure were obtained at the baseline medical visit. Baseline laboratory measures were defined as those obtained within one year prior to the baseline medical visit and ≤4 weeks after the baseline medical visit. The measurement closest to the baseline visit was selected for analysis. Demographics, baseline weight status, laboratory, and blood pressure measurements were summarized using the median and IQR for continuous variables and frequency and percent for categorical variables. Patients were categorized into 4 age groupings for summarizing weight status outcomes: 2-5, 6-11, 12-14, and 15-18 years. The Fisher exact test for categorical data and Kruskal-Wallis tests for continuous measures were used to compare demographic and baseline weight status, blood pressure and laboratory measures. The Dwass, Steel, Critchlow-Figner (DSCF) nonparametric method was used for pairwise testing to adjust for multiple comparisons.

Weight status measures include %BMIp95, ΔBMIp95, and BMI. Changes from baseline in %BMIp95 and ΔBMIp95 and percent change from baseline in BMI were summarized using median and IQR. Wilcoxon signed rank tests were used to evaluate change in weight status outcomes from baseline within age groups and follow-up intervals. The Kruskal-Wallis and DSCF tests were used to compare changes in the weight status outcomes among the age groups 6-11, 12-14, and 15-18 years. The 2-5 year age group was not included in these comparisons, but changes in weight status outcomes for this age group are presented.

Patients were categorized into 2 age groupings (6-11 and 12-18 years) for summarizing laboratory and blood pressure outcomes. The recommended duration of the program for patients varied among sites. Because all sites recommended ≥6 months of participation, the 6-month time point was selected to evaluate the associations between changes in laboratory and blood pressure outcomes vs changes in weight status.[26] The Wilcoxon signed rank test was used to evaluate change from baseline measures and the Kruskal-Wallis test and DSCF multiple comparison tests were performed to compare changes from baseline between age groups.

Multivariable mixed effects modeling with site as a random factor was used to identify patient and program characteristics that were associated with changes in % BMIp95 at 4-6, 7-9, and 10-12 months separately. Race and ethnicity were combined into 1 variable (race/ethnicity) for this model: Hispanic, white/non-Hispanic, black/non-Hispanic, other non-Hispanic, and other/missing. Patient characteristics in the model included sex, age group (6-11, 12-14, 15-18 years), race/ethnicity, health insurance (public, private, self-pay, unknown), baseline obesity status (obesity class 1, severe obesity class 2, severe obesity class 3), and number of visits in follow-up interval (2-3, 4-6, ≥7). The Tukey-Kramer multiple comparison adjustment for pairwise comparisons was used for variables with >2 levels.

To evaluate the association between changes in laboratory measures and changes in weight status, medical visit dates and laboratory measure dates were matched such that, at most, 45 days elapsed between the 2 dates. Only laboratory measures done ≥4 months after baseline medical visit were used. For patients with multiple laboratory measures that met these criteria, the test closest to the 6-month postbaseline time point was selected. Changes in laboratory measures were calculated as absolute change and percent change from baseline. Spearman correlation analysis was used to assess associations of absolute change and percent change from baseline in laboratory measures vs change in %BMIp95.

In addition, laboratory measures were classified as normal or abnormal at baseline and follow-up based on predetermined criteria (discussed in Methods). The Kruskal-Wallis and DSCF tests were used for comparing changes in % BMIp95 among the categorized changes in laboratory values. To estimate the magnitude of the changes in %BMIp95 that are associated with the categorized changes in laboratory values, medians and IQRs are presented.

Statistical tests were performed using 2-sided tests at the 5% level of significance. SAS version 12.1 (Cary, North Carolina) statistical software was used for all analyses and SigmaPlot version 13.0 (Systat, San Jose, California) was used for graphical displays.

Results

A total of 6454 patients, ages 2-18 years, were enrolled into the registry in 31 POWER sites from May 2014 to December 2016. Baseline participant demographics are detailed by age group in **Table I**. Combining across all age groups, 73% had severe obesity (classes 2 and 3), with a median % BMIp95 of 132.2% (IQR, 119-138).

Baseline blood pressure and laboratory measurements were available on 95% and 58% of patients, respectively. Among those with laboratory measures, TG were abnormal in 67%, non-HDL-C in 58%, fasting glucose in 10%, HbA1c in 25%, and ALT in 23%. **Table II** (available at www.jpeds.com) shows the outcomes of these measures by

Table I. Demographics and baseline characteristics of study participants

Characteristics	2-5 years	6-11 years	12-14 years	15-18 years	
No. of patients (%)	354 (5)	3101 (48)	1728 (27)	1271 (20)	*P* Value
Sex					<.001
Male	40	47	48	41	
Female	60	53	52	59	
Ethnicity					<.001
Hispanic	44	35	29	26	
Non-Hispanic	51	56	62	63	
Unknown/not reported	6	9	9	11	
Race					<.001
White	49	50	56	56	
Black/African American	13	19	19	19	
Other/mixed	15	15	14	12	
Unknown	23	16	11	12	
Health insurance (%)					.0014
Public/Medicaid	67	64	63	58	
Private	28	29	29	34	
Self-pay/other/none	2	1	1	1	
Unknown/not reported	3	6	7	8	
Weight status					
Obesity category (%)					<.0001
Class 1	21	30	26	23	
Class 2	32	37	36	32	
Class 3	48	33	38	45	
%BMIp95, median (IQR)	138.2 (123-159) n = 351	130.6 (118-146) n = 3094	132.6 (119-150) n = 1720	134.7 (120-154) n = 1266	<.0001*,†,‡,§
ΔBMIp95, median (IQR)	7.0 (4-11) n = 351	6.6 (4-10) n = 3094	8.5 (5-13) n = 1720	9.8 (6-15) n = 1266	<.0001†,‡,§,¶,‖
BMI, median (IQR)	N/A	N/A	34.4 (31-39) n = 1726	38.5 (34-44) n = 1271	N/A

N/A, not applicable because absolute BMI does not reflect the physiological variability within these age groups.
P value from Kruskal-Wallis nonparametric test.
Nonparametric pairwise comparisons between age groups – DSCF method.
*2-5 significantly different than 6-11.
†2-5 significantly different than 12-14.
‡6-11 significantly different than 15-18.
§12-14 significantly different than 15-18.
¶2-5 significantly different than 15-18.
‖6-11 significantly different than 12-14.

age group. The median changes of %BMIp95 from baseline for the entire cohort at selected follow-up periods were: at 4-6 months (n = 2133), −1.88 (IQR, −5.8 to 1.4); at 7-9 months (n = 1253), −2.50 (IQR, −7.4 to 1.8); and at 10-12 months (n = 782), −2.86 (IQR, −8.7 to 1.9). All age groups showed an improvement in %BMIp95 at all follow-up periods (**Table III**). At the 7- to 9-month follow-up period, improvement in %BMIp95 was better in the 15- to 18-year-old age group than the 6- to 11-year-old age group (−3.4 vs −2.0, respectively, adjusted P = .006). At 10-12 months, the 12- to 14-year-old age group had a greater improvement in %BMIp95 than the 6- to 11-year-old group (−3.7 vs −2.2, respectively; adjusted P = .02). There were no differences in improvement in %BMIp95 among age groups at 4-6 months (**Table III**).

A greater improvement in %BMIp95 occurred in class 3 severe obesity in comparison with class 1 obesity at the 4-6 months (−2.7 vs −0.6, adjusted P = .0001) and at 7-9 months (−3.2 vs −1.5; adjusted P = .028; **Figure 1**). At the 10- to 12-month interval, Hispanic youth had a greater reduction in %BMIp95 than black/non-Hispanic youth (−2.5 vs 1.5; adjusted P = .036). There was a trend toward a larger reduction in %BMIp95 at the 10- to 12-month interval for patients with ≥7 visits than for those that only had 2-3 visits (−2.4 vs 1.3; adjusted P = .055). No other significant associations were detected (**Figure 1**).

Changes in blood pressures and laboratory values were evaluated over 6 months (±1.5 months) for patients grouped by age (6-11 years and 12-18 years) and obesity status. Statistically significant improvements in diastolic blood pressure were noted in 6- to 11-year-olds with class 2 obesity, and in 12- to 18-year-olds with class 3 obesity. Of the 3579 patients aged 6-18 years that had baseline laboratory measures available, only 421 (12%) had follow-up laboratory tests within the time frame of 6 months (±1.5 months). Significant improvements were noted in TG, non-HDL-C, fasting glucose, ALT, and diastolic blood pressure primarily in youth with class 2 and 3 obesity (**Table IV**; available at www.jpeds.com).

Changes in all laboratory measures were positively associated with changes in %BMIp95 (**Figure 2**; available at www.jpeds.com). Normalization of ALT and HbA1c were associated with improvement in %BMIp95 (**Figure 3**).

Table III. Change in weight status from baseline, stratified by age group and follow-up period

Age groups (y)	ΔBMIp95, median (IQR)						Percent change BMI, median (IQR)		
	4-6 mo	7-9 mo	10-12 mo	4-6 mo	7-9 mo	10-12 mo	4-6 mo	7-9 mo	10-12 mo
2-5	−0.6 (−5 to 4) n = 113	−1.7 (−6 to 4) n = 69	−1.9 (−8 to 3) n = 45	−0.09 (−0.94 to 0.79) n = 113	−0.22 (−1.1 to 0.88) n = 69	−0.37 (−1.4 to 1.0) n = 45	N/A	N/A	N/A
6-11	−1.8* (−6 to 1) n = 1035	−2.0* (−7 to 2) n = 635	−2.2* (−7 to 2) n = 403	−0.29* (−1.10 to 0.44) n = 1035	−0.26* (−1.30 to 0.64) n = 635	−0.16* (−1.40 to 0.87) n = 403	N/A	N/A	N/A
12-14	−2.0* (−6 to 1) n = 534	−2.9* (−8 to 1) n = 319	−3.7* (−11 to 1) n = 207	−0.43* (−1.40 to 0.41) n = 534	−0.62* (−1.90 to 0.52) n = 319	−0.74* (−2.50 to 0.57) n = 207	−0.32 (−3.2 to 2.2) n = 534	−0.21 (−4.2 to 3.1) n = 319	0.02 (−5.0 to 4.0) n = 207
15-18	−2.1* (−7 to 1) n = 451	−3.4* (−9 to 1) n = 230	−3.7* (−11 to 2) n = 127	−0.52* (−1.90 to 0.35) n = 451	−0.85* (−2.6 to 0.46) n = 230	−0.83* (−2.90 to 0.84) n = 127	−0.77 (−4.1 to 1.6) n = 451	−1.03 (−5.4 to 2.4) n = 230	−0.66 (−5.6 to 3.8) n = 127
P value: age group comparisons (6-18 years only)	NS	†	‡	†	‡,†	‡,†	§	§	NS

N/A, not applicable because absolute BMI does not reflect the physiological variability within these age groups; *NS*, No significant differences among age groups.
Nonparametric pairwise comparisons between age groups: DSCF method.
*$P < .05$ based on signed-rank test for change from baseline.
†6-11 years significantly different than 15-18 years.
‡6-11 years significantly different than 12-14 years.
§12-14 years significantly different than 15-18 years.

Patients with normalization of an initially abnormal ALT at 6 months had significantly greater improvement in % BMIp95 than those with persistent abnormality in the ALT (median, −5.2 vs −1.35; adjusted $P = .002$). Youth with an increase in ALT from a normal value at baseline to an abnormal value at 6 months had a significant worsening in %BMIp95, whereas those who had persistently normal ALT showed improvement in %BMIp95 (median, 0.48 vs −2.5; adjusted $P = .006$). Similarly, patients with normalization of HbA1c at 6 months from baseline abnormal status and those with normal HbA1c at both baseline and at 6 months had significantly greater improvements in %BMIp95 than those who went from normal HbA1c to abnormal status at 6 months (median, −4.6 vs 1.2 [adjusted $P = .005$], −2.5 vs 1.2 adjusted $P = .02$, respectively). Thus, for subjects with initially abnormal ALT and HbA1c, an approximate 5 percentage point reduction in %BMIp95 (shown by the dashed line in **Figure 3**) was associated with improvements in these cardiometabolic measures. However, there were no significant differences among the 4 categories for TG nor non-HDL-C owing to all 4 categories showing some level of improvement in %BMI95.

Discussion

In the US, patients in this nationally-representative PWM registry demonstrate a modest reduction in BMI over 12 months as measured by change in %BMIp95. Our study is unique in its utilization of %BMIp95 as the primary metric of response and in our focus on reporting the association of cardiometabolic risk factors in relation to reduction in %BMIp95.

In contrast with previous studies,[27-30] we observed more improvement in BMI for adolescent patients (12-14 years old and 15-18 years old) at follow-up, for both the 7- to 9- and 10- to 12-month intervals, than for younger patients (6-11 years old). Previous studies have demonstrated a greater improvement in response to weight management interventions in younger patients compared with older; these studies report change in terms of BMIz.(27-30). The contrast in results between our study (using change in %BMIp95), and those of prior studies may be explained by limitations of using BMIz as an outcome measure for patients at the extreme ends of BMI, such as youth with severe obesity.[22] We chose to follow progress with change in %BMIp95 and ΔBMIp95 to address this inherent problem. These contrasting results may also be explained in part owing to differences in study populations with POWER, including a greater number of older adolescent patients, more diversity in ethnicity, and the use of varying clinical protocols.

Another notable observation was the greater magnitude of improvement in BMI in those with greater severity of obesity at baseline. Previous studies report mixed outcomes, with some showing better outcomes for those with less severe obesity,[31] and others showing better outcomes for those with severe obesity.[29] Our findings may be accounted for

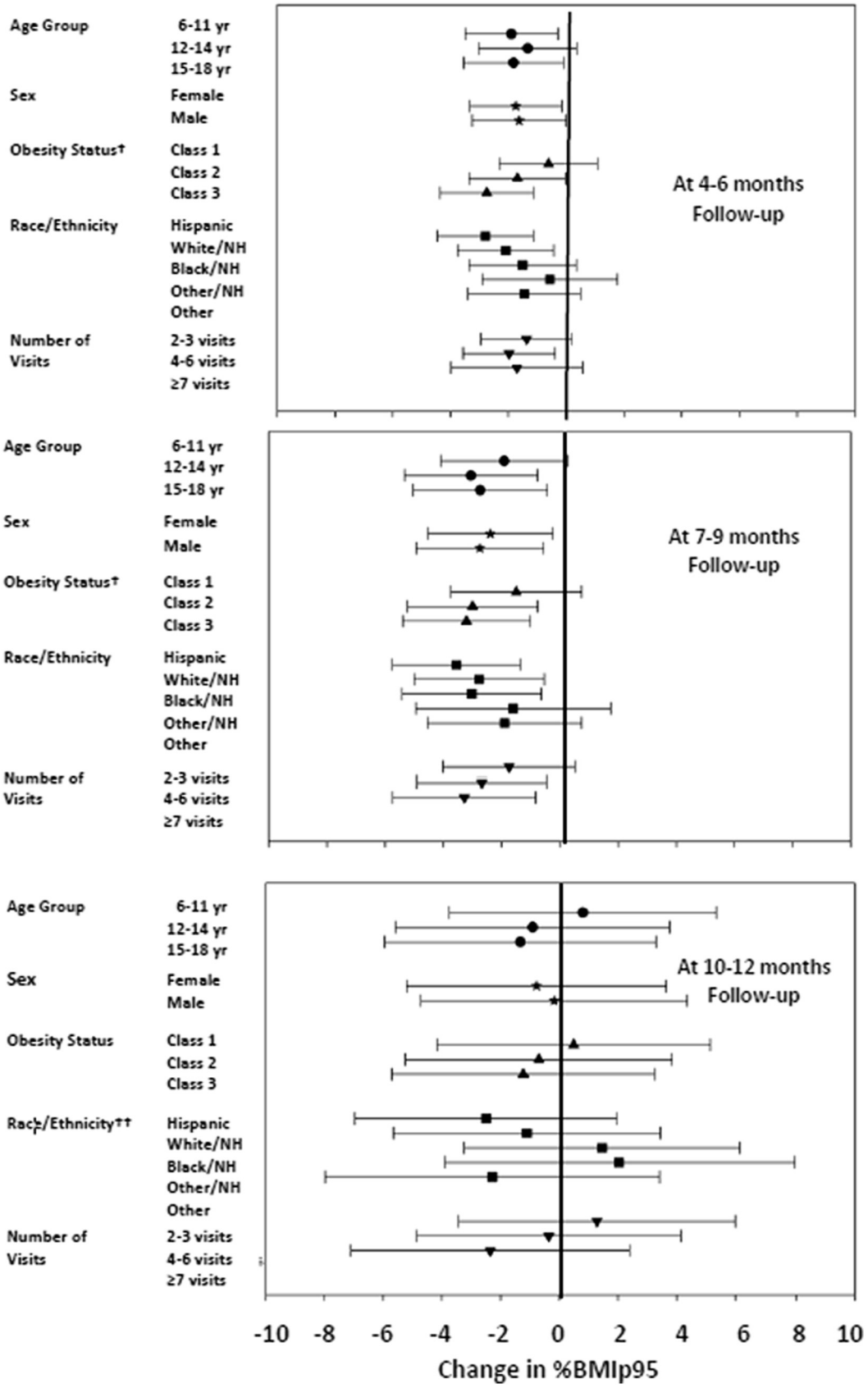

Figure 1. Change in weight status by patient characteristics and follow-up period. Significant associations with change in weight status: *Obesity status (class 3 > class 1): At 4-6 months of follow-up, $P = .0001$; at 7-9 months follow-up, $P = .03$. †Race/ethnicity (Hispanic > black/non-Hispanic [NH]), at 10-12 months of follow-up: $P = .036$.

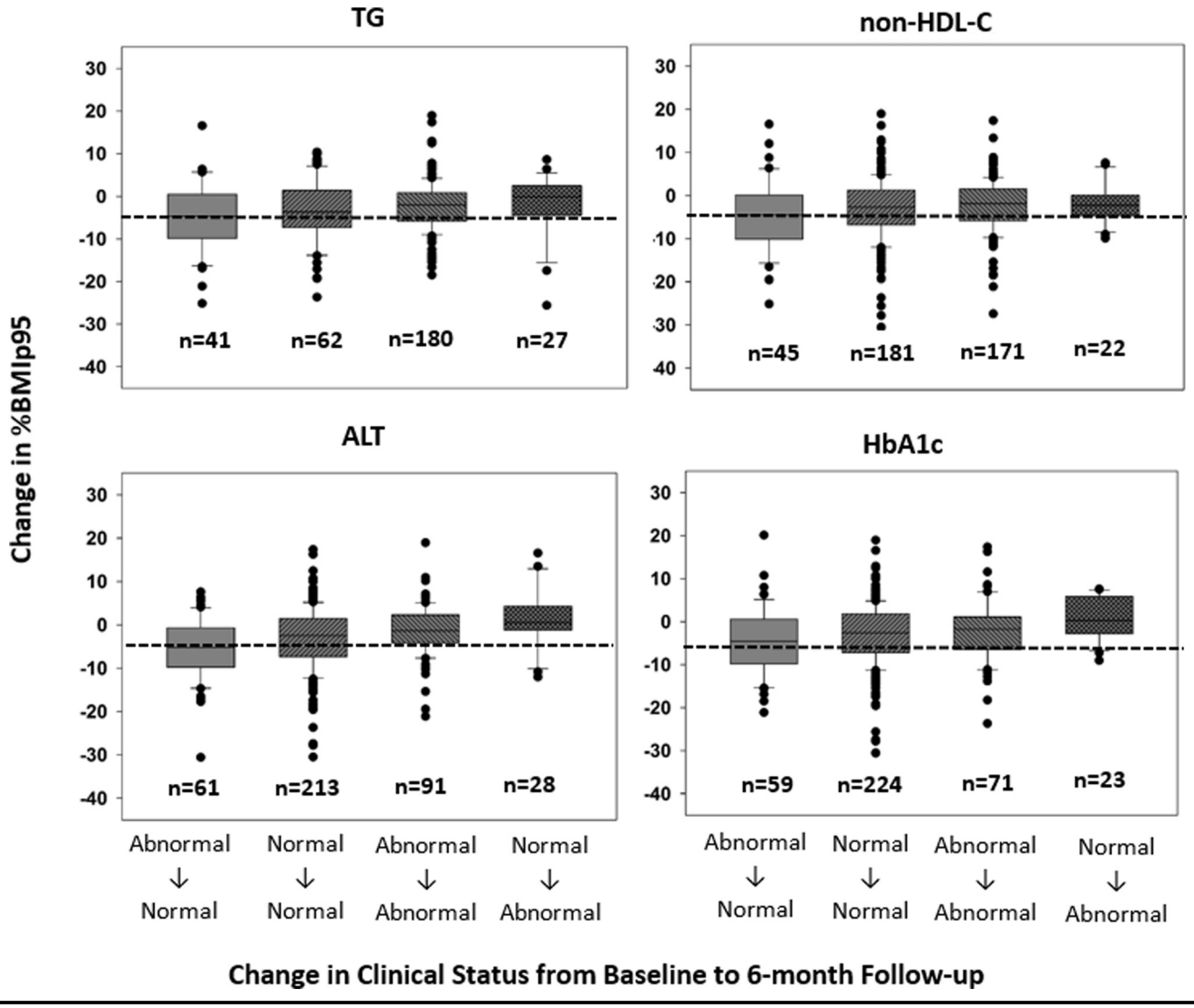

Figure 3. Association between change in laboratory measure classification group from baseline to 6-month follow-up in relationship to change in %BMIp95. Reference line (—) drawn at a 5 percentage-point decrease in the %BMIp95.

by the plausible explanation that greater BMI reduction may be due to the greater excess weight in youth with class 3 relative to those with class 1 obesity. Additionally, for youth ages 12-18 years, we report significant reductions in %BMIp95 and ΔBMIp95, but not significant changes in percent change in BMI. These contrasting findings may be attributed to the widening of the reference curve as children age. Thus, measures in relation to the reference curve will be decreasing while absolute BMI may remain unchanged. Last, Hispanic youth had greater improvement at 10-12 months than non-Hispanic youth; previous studies of similarly diverse US populations in PWM have not shown any associations between ethnic or racial background and greater clinical outcomes.[32,33] More research is needed to investigate whether biologic/phenotypic, socioeconomic, and/or cultural characteristics have predictive value in PWM and how to tailor programs to be simultaneously customized to, yet equally effective for, different patient populations.

Similar to previous reports of youth with severe obesity, a significant proportion of youth in our study had comorbidities, including dyslipidemia, prediabetes,[30,31] and elevated liver enzymes.[3,4,26-29] A greater decrease in %BMIp95 was associated with a higher likelihood of improvement in TG, non-HDL cholesterol, and HbA1c. Indeed, a 5-percentage point decrease in %BMIp95 was identified as an approximate threshold at which cardiometabolic risk factors seemed to improve. Our findings of greater improvement in cardiometabolic risk in youth with severe obesity (class 2 and 3) relative to those with nonsevere obesity (class 1) are in contrast with other studies.[13,33] The variation in these findings may be due to racial/ethnic and socioeconomic differences in participants and in type and intensity of interventions. Furthermore, the worsening of metabolic markers (normal to abnormal classification) was observed even with no change in %BMIp95 during follow-up. Such changes may be explained by persistent or worsening lifestyle habits that may

negatively impact laboratory measures more than an increase in BMI or may reflect physiological decompensation resulting from persistent obesity.

In line with a recent meta-analysis of PWM programs,[34] we report a trend indicating association between increased frequency of clinic visits during a 10- to 12-month program and greater improvements in patient %BMIp95. This finding may have been stronger if we had accounted for program contact hours, such as group nutrition or exercise sessions where height and weight measurements were not obtained. The only data available for analyses were number of clinic visits that included height and weight measurements. Owing to diverse program offerings across sites, 1 clinic visit could represent anywhere from 1 to 4 contact hours in some POWER sites and cumulative contact hours for patients at some sites may have reached the recommended ≥26 hours over a period of 2-12 months despite a lower visit frequency.[26]

The current study has multiple strengths. First, clinical registries such as this have the advantage of reporting on real-world treatment outcomes, which have a high degree of relevance to clinical care and generalizability, unlike a randomized clinical trial. Second, the large sample size allowed for the evaluation of several patient-level variables and program characteristics in relation to BMI reduction and other health outcomes. Finally, the generalizability of the results is supported by the relatively diverse cohort of participants and POWER sites that use an array of treatment approaches and represent varied geographical regions of the US.

Conversely, our study had several limitations that should be acknowledged. Owing to the nature of the study design, there was likely variation in the intensity and frequency of intervention offered at each program owing to inherent differences in the skills and qualifications of the program personnel, program duration and frequency, and intensity of intervention. Patients were not required to necessarily demonstrate adherence to the program by engaging in healthy behavior such as amount of physical activity; therefore, the results demonstrate the aggregate data from treatment adherent as well as nonadherent subjects. We did not capture clinical visits or group visits that did not have weight or height measurements. Additionally, weight-influencing medications in use at the time of the initial visit were not routinely reported by all sites; medications prescribed at initial and follow-up visits were not collected uniformly. Other limitations include potential bias owing to the requirement of an enrollment fee for sites to participate, the lack of opportunity for all enrolled subjects to reach 1 year of follow-up, and by virtue of its registry-type design, our study did not include a control group. Therefore, the results should not be interpreted as efficacy estimates, but rather as uncontrolled effectiveness outcomes for patients who continued program participation at the selected follow-up time intervals and had been enrolled early enough in the data period to complete the desired follow-up. Inferences can only be made about the population of patients that remain in the program for those time periods. Moreover, it is possible that the BMI outcomes were influenced by the regression to the mean phenomenon, particularly in the subanalyses by baseline BMI classification. Owing to the clinical and observational nature of the study, not all variables were systematically measured and reported including missing data from participant attrition.

Limitations related to the laboratory results reported on cardiometabolic risk are that repeat laboratory measures may have been obtained more often in patients with abnormal levels at baseline, in which case bias would have been introduced as well as the potential for regression to the mean. Decreases in ALT may or may not reflect histologic improvement of liver disease,[35] and abnormal HbA1c values were not validated in pediatric populations[36] nor were they adjusted for interlaboratory assay variability.[37] Furthermore, pubertal staging was not a required data element in the registry and so an important confounder influencing insulin resistance was not considered in the analysis. Finally, because we report laboratory results at the 4- to 6-month time point, which is when weight status improvement may be at its maximum, it is possible that the improvements in some of these laboratory measures were overestimated.

Pediatric patients with obesity receiving care in multicomponent weight management programs across the US demonstrated a modest decrease in %BMIp95 over 12 months. Future studies should be directed towards confirming these findings, as well as identifying additional strategies to enhance the effectiveness of these multicomponent interventions for youth with obesity. ■

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Reprint requests: Shelley Kirk, PhD, RD, LD, Associate Professor, University of Cincinnati College of Medicine, Department of Pediatrics, Director, HealthWorks!, Cincinnati Children's Hospital Medical Center, The Heart Institute – Center for Better Health and Nutrition, 3333 Burnet Avenue, MLC 5016, Cincinnati, OH 45229-3039. E-mail: Shelley.Kirk@cchmc.org

Data Statement

Data sharing statement available at www.jpeds.com.

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Appendix

POWER Work Group Site Leads/Co-leads and Affiliations: Abraham-Pratt I, Florida Hospital for Children, Orlando, FL; Ali L, OU Physicians – Early Lifestyle Interventions Clinic, Tulsa, OK; Armstrong S, Duke Healthy Lifestyles, Durham, NC; Binns H, Ann & Robert H. Lurie Children's Hospital, Chicago, IL; Brubaker J, Cleveland Clinic Children's, Avon, OH; Cristison A, Universtiy of Illinois College of Medicine at Peoria, Healthy Kids U, Peoria, IL; Fox C, University of Minnesota Masonic Children's Hospital, Minneapolis, MN; Gordon C, Barbara Bush Children's Hospital at Maine Medical Center, Portland, ME; Hendrix S, Arkansas Children's Hospital, Little Rock, AR; Hes D, Gramercy Pediatrics, New york, NY; Jenkins L, Dell Children's Hospital Medical Center, Austin, TX; Joseph M, UF Health Pediatric Weight Management Center – Wolfson Children's Hospital, Jacksonville, FL; Heyrman M, Mayo Clinic, Rochester, MN; Liu L, Seattle Children's Hospital, Seattle, WA; McClure A and Hofley M, Children's Hospital at Dartmouth Pediatric Lipid & Weight Management Center, Lebanon, NH; Negrete S, University of New Mexico Children's Hospital, Albuquerque, NM; Novick M, Penn State Hershey Children's Hospital, Hershey, PA; O'Hara V, Eastern Maine Medical Center, Bangor, ME; Rodrue J, Arnold Palmer Hospital Center for Digestive Health & Nutrition, Orlando, FL; Santos M, Connecticut Children's Medical Center, Hartford, CT; Stoll J, St. Louis Children's Hospital, St. Louis, MO; Stratbucker W, Helen DeVos Children's Hospital-Spectrum Health, Grand Rapids, MI; Sweeney B, Children's Mercy Hospital, Kansas City, MO; Tester J, UCSF Benioff Children's Hospital Oakland, Oakland, CA; Walka S and deHeer H, Fit Kids of Arizona at Northern Arizona Healthcare, Flagstaff, AZ; Wallace S, UAB Pediatrics/Children's of Alabama, Birmingham, AL; Walsh S, Children's Healthcare of Atlanta (Strong4Life Clinic), Atlanta, GA; Wittcopp C, Baystate Children's Hospital, Springfield, MA; Weedn A, University of Oklahoma Health Sciences Center, Oklahoma City, OK; and Yee J and Grace B, Harbor-UCLA Medical Center, Torrance, CA.

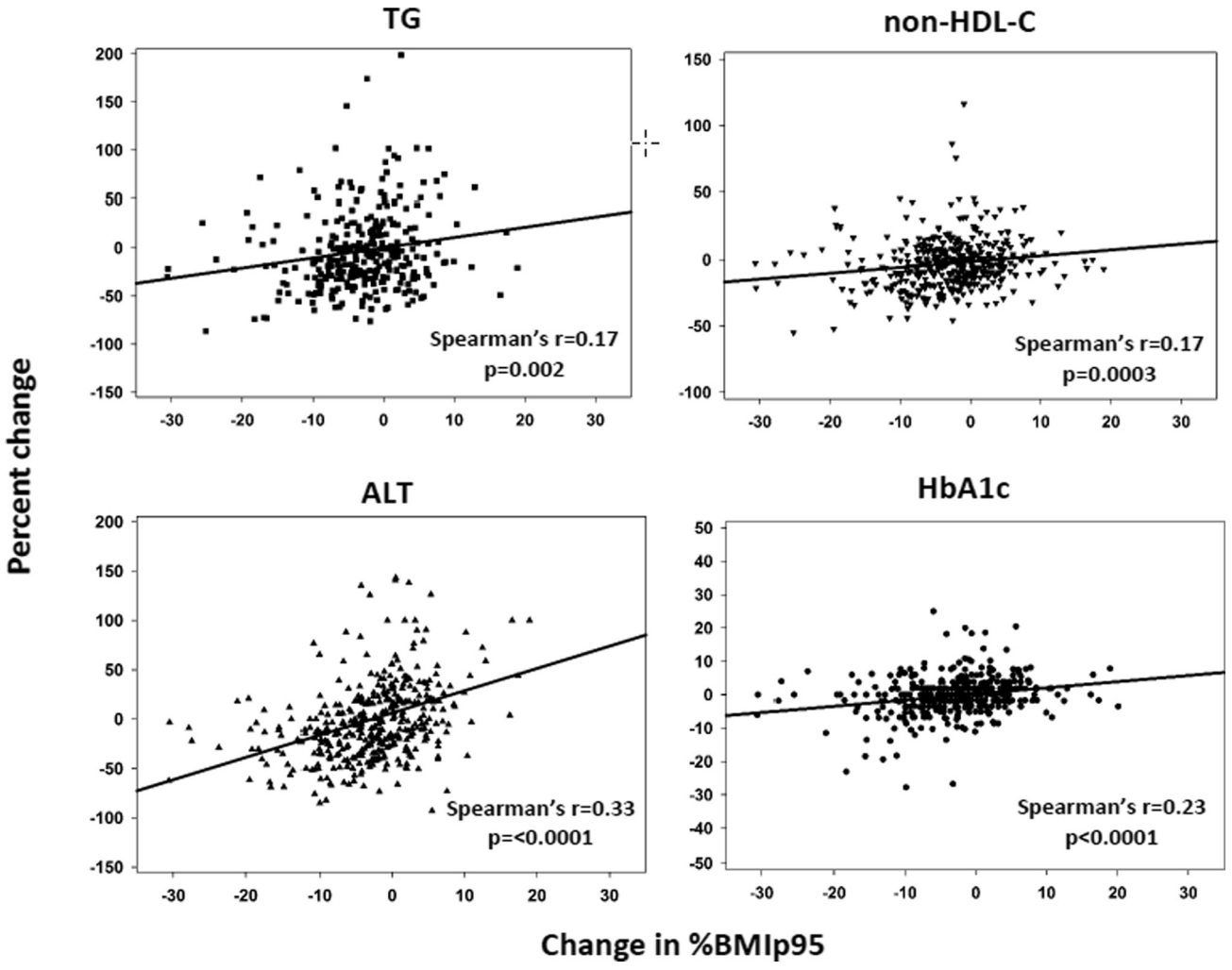

Figure 2. Correlations between percent change in laboratory measures and change in percent of the 95th percentile for BMI.

Table II. Baseline blood pressure and laboratory measures of study participants

Characteristics	2-5 years	6-11 years	12-14 years	15-18 years	
No. of patients (%)	354 (5)	3101 (48)	1728 (27)	1271 (20)	*P* Value
Blood pressure					
SBP percentile, median (IQR)	77.2 (55-92) n = 310	77.3 (55-91) n = 2984	77.3 (55-92) n = 1657	76.2 (51-93) n = 1207	.84
DBP percentile, median (IQR)	74.5 (56-90) n = 311	60.1 (41-79) n = 2986	62.1 (38-79) n = 1655	65.3 (42-84) n = 1208	<.0001*,†,‡,§,¶
Laboratory measures					
TG (mg/dL), median (IQR)	86.5 (59-127) n = 104	106.0 (74-154) n = 1224	115.0 (82-169) n = 672	103.0 (75-146) n = 495	<.0001*,†,‡,¶,‖
HDL-C (mg/dL), median (IQR)	44.0 (38-51) n = 167	43.0 (37-51) n = 1816	41.0 (35-48) n = 1045	41.0 (35-47) n = 732	<.0001†,‡,¶,‖
Non-HDL-C (mg/dL), median (IQR)	111.0 (9-128) n = 167	114.0 (95-135) n = 1812	113.0 (94-136) n = 1038	115.0 (95-139) n = 729	.12
Fasting glucose (mg/dL), median (IQR)	85.0 (80-90) n = 108	88.0 (82-93) n = 1213	89.0 (84-95) n = 680	89.0 (82-95) n = 511	<.0001*,†,‡,§,‖
ALT (U/L), median (IQR)	25.0 (18-34) n = 165	27.0 (20-37) n = 1765	26.0 (18-39) n = 1756	26.0 (19-39) n = 725	.047**
HbA1c (%), median (IQR)	5.30 (5.1-5.5) n = 121	5.40 (5.2-5.6) n = 1537	5.40 (5.2-5.7) n = 980	5.40 (5.1-5.7) n = 703	.0001†,‖

DBP, diastolic blood pressure; *SBP*, systolic blood pressure.
P value from Kruskal-Wallis nonparametric test.
Nonparametric pairwise comparisons between age groups: DSCF method.
*2-5 significantly different than 6-11.
†2-5 significantly different than 12-14.
‡2-5 significantly different than 15-18.
‖6-11 significantly different than 12-14.
§6-11 significantly different than 15-18.
¶12-14 significantly different than 15-18.
**No pairwise differences after multiple comparison adjustment.

Table IV. Changes in laboratory measures and blood pressure at 6 months from baseline

	Stratified by weight status and age group					
	Class 1		Class 2		Class 3	
	6-11 years	12-18 years	6-11 years	12-18 years	6-11 years	12-18 years
Blood pressure						
SBP percentile, median (IQR)	3.57 (−14.1 to 20.6) n = 132	−2.73 (−17.2 to 10.2) n = 111	−0.81 (−17.5 to 15.7) n = 177	−3.30 (−22.0 to 21.2) n = 128	0.05 (−12.5 to 13.4) n = 120	−1.67 (−18.7 to 12.4) n = 140
DBP percentile, median (IQR)	−0.33 (−20.3 to 20.5) n = 132	−5.63 (−26.5 to 17.9) n = 111	−5.27* (−24.0 to 14.2) n = 176	−2.90 (−19.1 to 16.0) n = 128	−3.19 (−24.6 to 14.4) n = 120	−6.21† (−23.2 to 10.2) n = 140
Laboratory measures						
TG (mg/dL), median (IQR)	−1.5 (−33 to 20) n = 58	−13.0* (−57 to 15) n = 43	−20.0† (−56 to 18) n = 68	−15.0‡ (−69 to −1) n = 57	−3.5 (−46 to 17) n = 34	−18.0† (−53 to 11) n = 53
HDL-C (mg/dL), median (IQR)	1.0 (−3 to 6) n = 78	1.0 (−4 to 5) n = 61	0.0 (−4 to 4) n = 89	1.0 (−5 to 4) n = 83	−3.0 (−5 to 2) n = 44	−1.0 (−4 to 3) n = 70
Non-HDL-C (mg/dL), median (IQR)	−1.0 (−13 to 8) n = 77	−6.0* (−20 to 7) n = 60	−3.5* (−24 to 10) n = 88	−9.0‡ (−27 to 3) n = 82	−7.5† (−19 to 7) n = 44	−2.0 (−12 to 7) n = 70
Fasting glucose (mg/dL), median (IQR)	−0.5 (−6 to 5) n = 44	1.5 (−8 to 9) n = 38	−4.0† (−8 to 3) n = 66	−1.0 (−8 to 6) n = 46	2.0 (−3 to 5) n = 37	−3.0* (−9 to 5) n = 55
ALT (U/L), median (IQR)	−2.0 (−12 to 6) n = 63	−2.5 (−9 to 4) n = 60	−3.5† (−14 to 4) n = 92	−3.0* (−12 to 4) n = 61	−1.0 (−9 to 7) n = 50	−3.0 (−11 to 6) n = 69
HbA1c (%), median (IQR)	0.00 (−0.1 to 0.1) n = 53	0.00 (−0.2 to 0.1) n = 63	0.00 (−0.1 to 0.2) n = 70	0.00 (−0.2 to 0.1) n = 69	−0.10 (−0.2 to 0.1) n = 45	0.00 (−0.2 to 0.2) n = 78

Significance tests of changes from baseline for blood pressure and laboratory measures: *$P \leq .05$; †$P \leq .01$; ‡$P \leq .001$.