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The association between allostatic load and anthropometric measurements among a multiethnic cohort of children

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Summary

Objective: This study investigated if levels of allostatic load (ALoad) differed according to race/ethnicity in children and if ALoad was associated with obesity-related measures.

Methods: A multiethnic sample of 307 children aged seven to 12 was evaluated, composed of 39% European American (EA), 35% African American (AA), and 26% Hispanic American (HA) youth. Anthropometric measurements were evaluated by dual-energy X-ray absorptiometry, and other measurements included body mass index (BMI) and waist circumference (WC). Allostatic load scores were estimated based on two different calculations, including seven and eight biomarkers (ALoad1 and ALoad2), respectively. Analyses of variance, independence tests, and multiple regression models were performed.

Results: From the total sample, 22.80% ($n = 70$) of children were characterized as “no load,” 46.58% ($n = 143$) “low load,” and 30.62% ($n = 94$) “high load.” Hispanic American children showed the highest ALoad scores (2.07 ± 1.54 ; 95% CI, 1.73–2.41) compared with AA children (1.71 ± 1.43 ; 95% CI, 1.43–1.99) and EA children (1.56 ± 1.34 ; 95% CI, 1.32–1.80) ($P < 0.05$). Higher scores of ALoad (using both ALoad1 and ALoad2 calculations) were associated with higher BMI, total body fat mass, body percent fat, and WC ($P < 0.05$).

Conclusion: Significant differences in ALoad were observed in children according to race/ethnicity. Increased exposure to stressors captured by ALoad may result in increased risk for excessive adiposity and potential health risk in children. Further, ALoad may serve as a preventive marker for conditions known to continue throughout adulthood.

Keywords

body composition; children; multiethnic; stress

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CONFLICTS OF INTEREST

No conflict of interest was declared.

1 | INTRODUCTION

Disparities in health in the United States have been well documented in the scientific literature and are greatly influenced by the unequal burden of obesity among individuals of diverse populations. One-third of children aged 2–19 years in the United States are considered to be overweight or obese¹ However, Hispanic (25.8%) and non-Hispanic black (22%) children show higher obesity rates compared with non-Hispanic whites (14.1%).² The differences in obesity prevalence among communities have been associated with multiple factors including socioeconomic status, parental feeding practices, maternal BMI, country of birth, and acculturation status.^{3,4}

Exposure to stressful events or circumstances have the potential to contribute to psychosocial and physiological responses that may impair maintaining individual's normal physiological systems through a process known as allostasis.⁵ Through this process, physiological respondents of stress systematically accumulate which damage tissues and organs, and dysregulate normal function of the human body.⁶ Allostatic load (ALoad), a measure of cumulative stress in humans, has been associated with increased risk of physical deterioration, mental health diagnoses, and mortality.⁶

Since its initial operationalization and study in the late 1990's, ALoad has been measured using markers reflecting the hypothalamic–pituitary–adrenal (HPA) axis, sympathetic-adrenal-medullary system, metabolic response to energy expenditure, immune/inflammatory system, and cardiovascular system that are active after exposure to a stressful environment.⁶ These biomarkers used to calculate the ALoad index reflect different stages of stress-related disease processes, and are captured through diverse scoring systems that differ in the inclusion of biomarkers. For example, around 18 different ALoad calculations and 26 different biomarkers have been used in various studies using data from the National Health and Nutrition Examination Survey (NHANES).⁷ However, research on ALoad index calculations in children is limited, and prior studies have utilized different combinations of bio-markers.^{8,9} While there is no consensus on how to measure ALoad, the use of a previously published scoring system or developing a new definition tailored to a specific question is preferred.⁷ Despite the variability in ALoad indexes, its use in predicting health risk associated with stress has been well documented, and it has been shown that the ALoad index improves the prediction of mortality and health outcomes compared with the use of metabolic syndrome components alone.⁶

Although the perception of stress and activation of the allostatic mechanism are influenced by individual differences,¹⁰ the long-term effects of ALoad from adverse circumstances such as social and economic stressors have been observed in different races/ethnicities, countries, and ages.¹¹ Given the increasing evidence linking stress exposure and childhood obesity,¹² ALoad has been suggested as a potentially useful approach to understand the aetiology of racial/ethnic disparities in childhood obesity.¹³ Therefore, the aim of this study was to investigate if levels of ALoad differed by race/ethnicity and the potential relationship between ALoad and obesity-related measures in children. First, we examined differences in ALoad among a multiethnic sample of children. Second, we examined the relationship

between ALoad and obesity-related measures while controlling for race/ethnicity, biological, and parental socioeconomic factors.

2 | METHODOLOGY

2.1 | Participants

A total of 307 children aged seven to 12 (47.23% girls and 52.77% boys) were recruited as part of a cross-sectional study, which aimed to identify the effect of genetic and environmental parameters on racial/ethnic differences in aspects of insulin secretion among healthy children. Participants were self-identified as European American (EA, 39%), African American (AA, 35%), and Hispanic American (HA, 26%). Participants were recruited from the Birmingham, Alabama area at churches, schools, and health fairs, and through newspapers, parent magazines, radio, and participant referrals. Exclusion criteria included a current diagnosis of type 1 or type 2 diabetes, or a medical diagnosis that affects body composition, metabolism, or fat distribution, and/or use of medication known to affect body composition.

2.2 | Protocol

Data were collected during two visits. At the first visit, anthropometric measurements (weight in kilograms [kg] and height in meters [m]), pubertal status, and body composition by dual-energy X-ray absorptiometry (DXA) were measured. In addition, a questionnaire that included open and closed format questions was administered by a registered dietitian in the presence of a parent or guardian. Within 30 days the children and their parents returned for the second visit, where the blood samples were collected. The measurements were completed at the General Clinical Research Center (GCRC) and the Department of Nutrition Science at the University of Alabama at Birmingham (UAB). The Institutional Review Board (IRB) at UAB approved this study, and the children and parents provided written informed consent.

2.3 | Pubertal status and sex

Pubertal stage and biological sex play a role in adiposity distribution and accumulation¹⁴ and were considered as a covariate in statistical models. A paediatrician assessed the pubertal stage of participants according to the system developed by Marshall and Tanner, basing on the Tanner stage according to both breast and pubic hair development in girls and genitalia and pubic hair in boys.^{15,16} One composite number was assigned for Tanner staging, representing the higher of the two values defined by breast/genitalia and pubic hair development. Children of pubertal stage less than or equal to 3 were included in this study.

2.4 | Socioeconomic status

The Hollingshead four-factor index that includes educational level, occupational prestige, marital status, and retired/employed status were used to determine the social status of each parent/guardian.¹⁷ Socioeconomic status (SES) has been associated with body composition in children⁴; therefore, it was used as a covariate in the statistical models. Educational level was scored on a seven-point scale (1 = less than seventh grade completed; 7 = graduate degree). Occupational prestige was scored on a nine-point scale (1 = farm labourers/service

workers; 9 = higher executives). Occupation was weighted for a factor of five, and education was weighted for a factor of three. After a total SES was computed, family income was added (1 = 0–9999; 9 = 80 000+). The resultant scores range from eight to 66 with higher scores indicating higher socioeconomic status.

2.5 | Anthropometric measurements

Body composition (total body fat mass and non-bone lean tissue mass) was measured by dual-energy X-ray absorptiometry (DXA) using a GE-Lunar Prodigy densitometer (GE LUNAR Radiation Corp, Madison, WI). Participants were scanned in light clothing while lying flat on their backs with arms at their sides. Dual-energy X-ray scans were performed and analysed with paediatric software encore 2002 (version 6.10.029). For this study, total body fat mass and DXA weight were used to calculate the children body percentage of fat (BPF). Waist circumference (WC) in centimetres was measured at the narrowest part of the torso or the area between the ribs and iliac crest. Waist circumference measures were obtained using a flexible tape measure (Gulick II, Country Technology Inc, Gays Mills, WI) and were recorded to the nearest 0.1 cm. Participants were weighed (Scale-tronix 6702 W, Scale-tronix, Carol Stream, IL) to the nearest 0.1 kg (in minimal clothing without shoes). The measure of height was recorded without shoes using a digital stadiometer (Heightronic 235, Measurement Concepts, Snoqualmie, WA). Body mass index (BMI) was classified using CDC-defined BMI percentile cut points (85th to less than 95th percentile, overweight and greater than or equal to 95th percentile, obesity).

2.6 | Allostatic load measurement

The use of ALoad as measure of chronic stress and its association with health outcomes has been highly described in the literature.^{6,8–10,18,19} While there are variations in how ALoad is calculated, it has been suggested that ALoad calculations in adults should include biomarkers from three systems (cardiovascular, metabolic, and immune/inflammatory).⁷ In children, to measure the association with paediatric obesity, ALoad index typically includes markers such as homeostasis model assessment-insulin resistance (HOMA-IR), total cholesterol, triglycerides, blood pressure (systolic and diastolic), C-reactive protein (CRP), and others.¹³ For the purpose of this research and based on the available data, seven and eight biomarkers (markers of metabolism, cardiovascular, and inflammatory system) were used to create two ALoad calculations. The first ALoad (ALoad1) calculation included seven biomarkers: total cholesterol, triglycerides, low-density lipoprotein (LDL)-cholesterol, high-density lipoprotein (HDL)-cholesterol, HOMA-IR (markers of metabolism), and both systolic blood pressure (SBP) and diastolic blood pressure (DBP) (markers of the cardiovascular system). The second ALoad (ALoad2) calculation were estimated in a subsample (EA and AA) (n = 56) with eight biomarkers that added CRP (marker of inflammation). In both calculations, the biomarkers used relate to metabolic and cardiovascular systems and were guided by previous ALoad studies that used these parameters as a proxy of stress based on data availability.^{18,20}

Although the biomarkers used for the calculation of ALoad are similar to those used to define the criteria for the metabolic syndrome (MetS), the cut-off values for the biomarkers used for MetS are different than those considered for ALoad. Consequently, despite of the

similarity of biomarkers, both MetS and ALoad represent a unique construct; the former a measure of metabolic dysfunction and the latter a measure of physiological stress. Each of these measures serves as a different risk factor for health. The rationale for using ALoad as a measure of stress is that when individuals are exposed to chronic stress, systems such as cardiovascular, immune, and nervous respond by increasing the “wear and tear” of the body. This “wear and tear” alters the normal state of cardio-metabolic parameters that are involved in the preservation of physiological stability to environmental demands that characterize the normal allostasis process.^{6,21}

Lipids were measured in pooled serum from fasting blood samples that were collected at UAB GCRC. Lipids (total cholesterol, HDL cholesterol, and triglycerides) were measured by using a Stanbio SIRRUS analyser, and LDL cholesterol was calculated by using the method of Friedewald.²² Concentration of all serum-derived analyses was measured at the UAB in the Metabolism Core Laboratory that services the GCR and Nutrition Obesity Research Center. Glucose was measured in 10 µL serum with a SIRRUS analyser (Stanbio Laboratory, Boerne, TX), and insulin was assessed by using a double-antibody radioimmunoassay with 100-µL serum aliquots in duplicate (Linco Research Inc, St. Charles, MO). Homeostasis model assessment was used to measure insulin resistance. The HOMA-IR was calculated using the equation: $HOMA-IR = \text{fasting insulin } (\mu\text{U/mL}) \times \text{fasting glucose } (\text{mg/dL}) / 405$, assuming that normal young subjects have an insulin resistance of 1.²³ Blood pressure was measured four times during an overnight visit at the UAB GCRC. An automated paediatric blood pressure cuff (Dinamap Pro 200: GE Medical Systems) was used, and appropriate child-sized cuffs were used based on participation arm size. Participants were seated at rest, with feet flat on the floor for greater than or equal to 10 minutes before the measurement was taken. The measurements were separated by 5 minutes seated rest and were not significantly different in the evening and morning. For this study, the measurements of blood pressure were averaged. High-sensitivity enzyme-linked immunosorbent assays were used to determine C-reactive protein (CRP) (ALPCO Diagnostics, Windham, HN). The cut-off values were set in the top quartile for total cholesterol, triglycerides, LDL-cholesterol, homeostasis model assessment-insulin resistance (HOMA-IR), C-reactive protein, and both systolic and diastolic pressure, and lower quartile for HDL-cholesterol, sample-based. For each biomarker, a dichotomous variable was created in which “1” reflected values in the high-risk and “0” reflected values in the lowest-risk range. Total ALoad scores (0–7, 0–8) reflect the number of these singular indicators on which each child scored in the upper/lower quartile of risk; higher scores indicated higher ALoad. For this study, ALoad was evaluated in three groups by a number of high-risk indicators (1) zero biomarkers “no load,” (2) one to two biomarkers “low load,” and (3) greater than or equal to three biomarkers “high load.”

2.7 | Statistical analyses

Descriptive statistics (mean, standard deviation, and frequencies) were calculated to summarize sex, pubertal stage (Tanner), race/ethnicity, age, SES, and anthropometric measurements (weight, BMI [kg/m²], BMI percentile, lean tissue mass, total body fat mass [TBFM], BPF, and WC). The cut-off points used to calculate ALoad scores, and to group the children by a number of high-risk indicators were total cholesterol, 173 mg/dL;

triglycerides, 76 mg/dL; LDL-cholesterol, 104.6 mg/dL; SBP, 110.5 mmHg and DBP, 64.5 mmHg; HOMA-IR, 3.66; HDL-cholesterol, 41 mg/dL; and CRP, 0.46 mg/L. Mean differences in anthropometric measurements among groups, and mean differences among race/ethnicity groups were analysed using *t*-test, and analysis of variance (ANOVA) with Tukey's post hoc test. Chi-squared analyses were used to assess differences in sex and pubertal stage among groups.

Multiple regression analyses were performed with covariates to evaluate the main associations of interest. The dependent variables included BMI (kg/m^2), BMI percentile, TBFM (kg), BPF (%), and WC (cm). Independent variables included ALoad1 and ALoad2 calculations (0–7 and 0–8, respectively). Covariates included age, sex, pubertal stage (Tanner stage), race/ethnicity, SES, and lean tissue mass (used only in the TBFM model). In the sample where ALoad was analysed using calculation two, power analysis were performed in order to ensure that sample size was appropriate to detect differences at a significance level of $\alpha = 0.05$ with 0.80 statistical power. Categorical variables were dummy-coded. All residuals were tested for normality, BPF and WC were log-transformed after visual evaluation of residuals from the regression models. The significance level was considered $\alpha = 0.05$ for all statistical analyses. All analyses were performed with SAS statistical software (version 9.4, 2002–2012 by SAS Institute Inc., Cary, NC).

3 | RESULTS

3.1 | Descriptive statistics

Table 1 summarizes the demographic characteristics for the total sample and by ALoad groups based on ALoad1 (seven biomarkers included). From the total sample, 22.80% ($n = 70$) of children showed “no high-risk indicators,” 46.58% ($n = 143$) exhibited “low load,” and 30.62% ($n = 94$) showed “high load.” Significant differences were found in pubertal stage X^2 ($df = 4$, $n = 300$) = 10.21, $P = 0.0370$, and race/ethnicity X^2 ($df = 4$, $n = 307$) = 12.02, $P = 0.0172$. HA children and those children with pubertal stage Tanner II were more likely to be in the group of “high load.” No significant differences were found in age, SES, sex, for pairwise comparison between ALoad groups.

3.2 | Anthropometric measurements

Anthropometric measurements means and standard deviations for the total sample by ALoad groups are reported in Table 1. The ANOVA *F*tests grouping ALoad, yielded statistical significance in the majority of the anthropometric measurements ($P < 0.05$), except LTM. The mean differences in weight (kg) ($F_{2,297} = 15.23$, $P < 0.0001$), BMI (kg/m^2) ($F_{2,306} = 19.54$, $P < 0.0001$), BMI percentile ($F_{2,306} = 7.78$, $P < 0.0005$), TBFM (kg) ($F_{2,297} = 25.74$, $P < 0.0001$), BPF (%) ($F_{2,297} = 22.23$, $P < 0.0001$), and WC (cm) ($F_{2,300} = 24.83$, $P < 0.0001$) were significant among ALoad groups. Tukey post hoc analyses that were used for pairwise comparisons showed that the mean of the six anthropometric measurements was higher in the “high load” group ($P < 0.005$) in comparison with “low load” and “no load” groups.

3.3 | Allostatic load by race/ethnicity

Means and standard deviations of ALoad scores and ALoad biomarkers from both calculations ALoad1 and ALoad2 (seven and eight biomarkers) by race/ethnicity are reported in Table 2. In ALoad1, the ANOVA *F* tests grouping race/ethnicity yielded statistical significance in ALoad scores and in five of seven ALoad biomarkers ($P < 0.05$). The mean differences in triglycerides (mg/dL) ($F_{2,292} = 15.47$, $P < 0.0001$), HDL-cholesterol (mg/dL) ($F_{2,292} = 15.04$, $P < 0.001$), HOMA-IR ($F_{2,273} = 12.94$, $P < 0.0001$), and blood pressure in both components; SBP (mmHg) ($F_{2,301} = 4.91$, $P = 0.0080$), DBP (mmHg) ($F_{2,301} = 10.61$, $P < 0.0001$) were significant among race/ethnicity group. In ALoad2, mean differences in triglycerides (mg/dL) and HDL-Cholesterol (mg/dL) were significant ($P < 0.05$) among EA and AA children.

In ALoad1, Tukey post hoc analyses that were used for pairwise comparisons showed that the mean of triglycerides and HOMA-IR were higher in HA children ($p < 0.05$) in comparison with EA and AA children. In addition, HDL-cholesterol and blood pressure (both components SBP and DBP) were lower in HA children ($P < 0.001$) in comparison with EA and AA children. HA children showed the highest ALoad scores (2.07 ± 1.54 ; 95% CI, 1.73–2.41) compared with AA (1.71 ± 1.43 ; 95% CI, 1.43–1.99) and EA children (1.56 ± 1.34 ; 95% CI, 1.32–1.80) ($P < 0.05$).

3.4 | ALoad and anthropometric measurements

Table 3 showed results for the multiple regression models assessing the contribution of ALoad1 to anthropometric measurements while adjusting for sex, pubertal stage (Tanner), SES, and race/ethnicity. Model for total body fat mass was also adjusted for lean tissue mass. Body mass index (kg/m^2) ($P < 0.0001$), BMI percentile ($P < 0.0001$), TBFM (kg) ($P < 0.0001$), BPF (%) ($P < 0.0001$), and WC (cm) ($P < 0.0001$) were associated with higher ALoad scores. Across all models, HA children were positively associated with higher BMI, TBFM, BPF, and WC ($P < 0.05$). In the ALoad2, all anthropometric measurements were also associated with higher ALoad scores ($P < 0.005$).

4 | DISCUSSION

This study investigated racial/ethnic differences in ALoad among children and its relationship with obesity-related measures. Our results showed that ALoad differs in children according to race/ethnicity, with HA children showing higher scores compared with EA and AA children, and that higher ALoad scores were associated with higher anthropometric measurements.

The testing of our hypotheses was based on the identification of the most parsimonious statistical models balancing statistical and physiological criteria. As such, our preliminary analyses evaluated the potential contributions of energy intake (kcal) and physical activity to our main outcome in response to previously published data supporting associations of these parameters with body composition and with stress response.^{3,9} However, when energy-intake variables and physical activity variables (obtained from 24-h dietary recalls and questionnaires, respectively, as described elsewhere²⁴) were added as covariates in the

models no statistical contributions were observed from these measures (data not shown). Consequently, these variables were not included in our statistical models.

Prior longitudinal and cross-sectional studies have associated ALoad in adults with mortality, and physical, psychological, and cognitive health.¹⁹ Physical aspects related to growth in children, however, tend to limit the accuracy of certain measures defining ALoad, and consequently may provide a limited understanding of the impact of ALoad in paediatric health, when compared with adults.¹³ Despite this limitation, research has demonstrated associations between ALoad with both physiological and neurological outcomes after stressful childhood experiences,²⁵ and with stress from the cumulative risk of social and physical environmental aspects such as crowding, noise, and housing problems.^{18,26} It has been documented that childhood stressors have a long-lasting effect into adulthood that contributes to elevated age-related-disease risks, including depression, high inflammation levels, and metabolic risk markers.²⁷ Furthermore, recent evidence has suggested that high ALoad increased likelihood of some specific health outcomes such as asthma in adolescent boys.⁸

While ALoad has served as a basis to measure cumulative physical and social demands (stressors) throughout life, some calculations defining ALoad used similar biomarkers as those considered for the definition of MetS. As previously indicated, the cut-off definition of each biomarker defining ALoad and MetS is different, resulting in two unique and independent constructs. Hence, it is not surprising that participants with high levels of ALoad in our sample were not necessarily classified as having MetS (data not shown). Nonetheless, our results support the notion that this index in early ages could be a possible risk factor for obesity and obesity comorbidities among children of different race/ethnicity.

Racial/ethnic differences of ALoad in adults have been studied more extensively, with non-Hispanic blacks showing higher ALoad scores compared with non-Hispanic whites and Hispanics.²⁸ These results differ from our data, where Hispanic children showed the highest level of ALoad. It is relevant to consider that in our sample, Hispanics were the most recent migrant group in the area of data collection, probably exposed to greater levels of stress than others.

Additional factors that could influence ALoad in Hispanic children's scores may be related to their acculturation process and length of residence in the United States. Although the mechanisms for different levels of ALoad according to race/ethnicity are beyond the scope of this study, it might be that this index serves as a marker of other complex psychosocial processes, including perceived racial discrimination during childhood, as previously documented in our data.²⁹ Racial and ethnic minorities seem to have worse overall health and higher obesity prevalence than non-Hispanic whites, an observation considered to be explained in part by social and economic factors.³⁰ However, our data showed no significant contributions of ALoad to body composition according to parental SES, which supports previous reports in NHANES adult data where there was no association between SES and obesity in non-Hispanic blacks and Mexican-Americans.³¹ Whitaker et al³² showed similar results from a cross-sectional study where high prevalence of obesity among Hispanic

children relative to non-Hispanic whites or non-Hispanic blacks was not explained by racial differences in household income.

There are some limitations in the present study. First, our cross-sectional design did not allow for examination of developmental trajectories of ALoad and anthropometric measurements. Second, the sample size in ALoad2 calculation were smaller compared with ALoad1 calculation; however, power analyses were performed to correct the statistical analyses. Finally, we did not show differences of ALoad among Hispanics by place of birth (US born vs Foreign-born) and length of residence, which has been suggested to influence biological risk profiles and ALoad scores.²⁸ Despite these limitations, our results support that ALoad may play a role as a factor on influencing adiposity markers in children, and that ALoad differs based on race/ethnicity. ALoad may serve as a predictor of excessive adiposity and health risk in children and potentially as a preventive marker for conditions known to continue throughout adulthood. Further investigation is required to determine if ALoad is associated with other obesity risk factors, comorbidities, and behavioural practices in children, as well as to understand the mechanisms underlying the association among stress, ALoad, and health during early stages of the lifespan.

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TABLE 1
Demographic characteristics of children, and anthropometric measurements based on ALoad group

Variables	Total Sample (n = 307) Percentage (n)	No Load (n = 70)	Low Load (n = 143)	High Load (n = 94)
Sex				
Boys	52.77% (n = 162)	24.07% (n = 39)	45.06% (n = 73)	30.86% (n = 50)
Girls	47.23% (n = 145)	21.38% (n = 31)	48.28% (n = 70)	30.34% (n = 44)
Tanner stages				
Stage I	64.00% (n = 192)	27.08% (n = 52)	45.83% (n = 88)	27.08% (n = 52)
Stage II	23.00% (n = 69)	17.39% (n = 12)	39.13% (n = 27)	43.48% (n = 30)
Stage III	13.00% (n = 39)	15.38% (n = 6)	58.97% (n = 23)	25.64% (n = 10)
Race/ethnicity				
European American	39.41% (n = 121)	24.79% (n = 30)	52.07% (n = 63)	23.14% (n = 28)
African American	34.53% (n = 106)	21.70% (n = 23)	50.00% (n = 53)	28.30% (n = 30)
Hispanic American	26.06% (n = 80)	21.25% (n = 17)	33.75% (n = 27)	45.00% (n = 36)
Mean $\pm$ Standard deviation				
Age	9.54 $\pm$ 1.57	9.26 $\pm$ 1.55	9.53 $\pm$ 1.57	9.78 $\pm$ 1.55
SES	38.89 $\pm$ 14.36	39.59 $\pm$ 14.23	40.21 $\pm$ 13.75	36.40 $\pm$ 15.16
Anthropometric measurements				
Weight (kg)	35.86 $\pm$ 9.53	32.97 $\pm$ 7.10	34.48 $\pm$ 7.62	40.15 $\pm$ 12.04 ^c
BMI (kg/m ²)	18.61 $\pm$ 3.01	17.67 $\pm$ 2.14	18.07 $\pm$ 2.40	20.12 $\pm$ 3.78 ^c
BMI percentile	66.44 $\pm$ 26.16	62.60 $\pm$ 25.84	62.62 $\pm$ 26.65	75.12 $\pm$ 23.72 ^b
LTM (kg)	25.61 $\pm$ 5.19	24.77 $\pm$ 4.58	25.44 $\pm$ 5.10	26.51 $\pm$ 5.65
TBFM (kg)	8.96 $\pm$ 5.76	6.97 $\pm$ 3.65	7.78 $\pm$ 4.28	12.27 $\pm$ 7.44 ^c
BPF (%)	23.53 $\pm$ 9.36	20.41 $\pm$ 7.15	21.78 $\pm$ 8.67	28.57 $\pm$ 9.94 ^c
WC (cm)	64.43 $\pm$ 8.96	61.60 $\pm$ 6.77	62.48 $\pm$ 6.40	69.47 $\pm$ 11.35 ^c

Abbreviations: BMI percentile, body mass index for sex and age percentile; BPF, body percent fat; LTM, lean tissue mass; SES, socioeconomic status; TBFM, total body fat mass; WC, waist circumference. One-way ANOVA tests were performed to assess mean differences in age, SES, and anthropometric measures by ALoad group (eg, no high-risk indicators versus low load versus high load).

^a $p < 0.05$.

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$P_{c} > 0.001$
 $P_{q} > 0.01$

TABLE 2

Differences of ALoad scores and ALoad biomarkers based on race/ethnicity

Variables	Total Sample (n = 307) Mean ± Standard deviation	European American (n = 121)	African American (n = 106)	Hispanic American (n = 80)
ALoad1	1.74 ± 1.43	1.56 ± 1.34	1.71 ± 1.43	2.07 ± 1.54 ^a
TC (mg/dL)	153.76 ± 27.68	153.14 ± 24.77	153.81 ± 28.03	154.62 ± 31.42
TG (mg/dL)	66.68 ± 36.25	66.07 ± 30.86	54.26 ± 26.70	83.35 ± 46.61 ^c
LDL-C (mg/dL)	90.30 ± 26.51	91.56 ± 23.36	87.56 ± 27.40	91.90 ± 29.66
HDL-C (mg/dL)	50.15 ± 12.78	48.37 ± 11.23	55.47 ± 13.43	46.05 ± 11.97 ^c
HOMA-IR	3.10 ± 1.60	2.64 ± 1.09	3.03 ± 1.32	3.80 ± 2.15 ^c
SBP (mmHg)	103.20 ± 10.60	102.39 ± 10.48	105.75 ± 11.40	101.16 ± 9.10 ^b
DBP (mmHg)	60.05 ± 6.53	59.03 ± 6.21	62.39 ± 6.88	58.60 ± 5.75 ^b
ALoad2	2.05 ± 1.51	2.05 ± 1.23	2.06 ± 1.66	...
TC (mg/dL)	152.32 ± 25.18	149.20 ± 26.05	153.82 ± 25.18	...
TG (mg/dL)	61.05 ± 37.06	72.05 ± 49.69	52.35 ± 22.35 ^a	...
LDL-C (mg/dL)	86.55 ± 25.21	88.34 ± 22.32	84.91 ± 27.13	...
HDL-C (mg/dL)	53.56 ± 14.06	46.45 ± 9.00	58.44 ± 14.31 ^b	...
HOMA-IR	3.20 ± 1.09	2.96 ± 0.916	3.30 ± 1.18	...
SBP (mmHg)	106.68 ± 11.86	109.11 ± 9.78	104.71 ± 12.64	...
DBP (mmHg)	63.02 ± 6.90	63.42 ± 4.18	62.33 ± 7.80	...
CRP (mg/L)	0.58 ± 1.02	0.70 ± 1.46	0.48 ± 0.66	...

Abbreviations: DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides. One-way ANOVA tests were performed to assess mean differences in ALoad1 calculation by race/ethnicity group (eg, European American versus African American versus Hispanic American). *t*-test were performed to assess mean differences in ALoad2 calculation.

^a $P < 0.05$.

^b $P < 0.01$.

^c $P < 0.001$.

Multiple regression analysis exploring the relation between ALoad, and anthropometric measurements in a multiethnic sample of children

Independent variables	BMI		BMI Percentile		Log Total Body Fat Mass		Body Percent Fat		Log Waist Circumference	
	β	P	β	P	β	P	β	P	β	P
BMI $F_{7,295} = 13.04; P < 0.0001; R^2 = 0.2465$										
Age	-0.05944	0.560	-8.11212	<0.0001	-0.01813	0.4475	0.13517	0.6951	0.01785	0.0002
Sex	-0.34374	0.2126	-7.72838	0.005	0.33588	< 0.0001	4.40479	< 0.0001	-0.02178	0.0899
Tanner	1.19598	< 0.0001	13.78366	< 0.0001	0.02286	0.6707	1.22412	0.1234	0.04214	0.0001
SES	-0.01135	0.3467	-0.12052	0.3122	-0.00422	0.0784	-0.06286	0.1225	-0.0008736	0.1166
Race, AA	-0.3013	0.3891	-3.98149	0.2507	-0.27914	< 0.0001	-4.27341	0.0003	-0.03504	0.0314
Ethnicity, HA	1.15722	0.0082	11.50297	0.0079	0.20867	0.0152	3.78046	0.0096	0.05669	0.0055
LTM	...	...	...	...	0.0000526	< 0.0001	...	...	...	...
ALoad1	0.43761	< 0.0001	4.22034	< 0.0001	0.11372	< 0.0001	2.07235	< 0.0001	0.02811	< 0.0001
BMI $F_{5,45} = 4.32; P = 0.0027; R^2 = 0.3243$										
BMI Percentile $F_{5,45} = 5.30; P = 0.0007; R^2 = 0.3705$										
Log total body fat mass $F_{6,43} = 5.07; P = 0.0005; R^2 = 0.4143$										
Body percent fat $F_{5,44} = 4.11; P = 0.0038; R^2 = 0.3185$										
Log waist circumference $F_{5,44} = 5.21; P = 0.0008; R^2 = 0.3720$										
Age	0.00091348	0.3166	-11.94891	0.0001	-0.10429	0.1776	-1.66946	0.1755	0.00102	0.9421
Sex	0.00007818	0.9740	1.12941	0.8822	0.22739	0.1927	3.18020	0.2784	-0.02390	0.5239
Tanner	-0.00540	0.0152	18.68888	0.0085	0.31258	0.1364	5.75345	0.0565	0.08003	0.0198
Race, AA	0.00147	0.5702	-1.48059	0.8568	-0.45311	0.0179	-7.16196	0.0261	-0.07088	0.0875
LTM	...	...	...	...	0.00003726	0.1277	...	...	...	...
ALoad2	-0.00236	0.0035	5.01814	0.0450	0.12226	0.0364	2.27431	0.0211	0.04090	0.0011

Abbreviations: AA, African American; BMI percentile, body mass index for sex and age percentile; HA, Hispanic American; LTM, lean tissue mass; SES, socioeconomic status. All models adjusted for age, pubertal stage, and socioeconomic status. Model for log fat mass were also adjusted for lean mass. ALoad1 (seven biomarkers) and ALoad2 (eight biomarkers) models used European American children as a reference group.