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Detailed Assessments of Childhood AdversityEnhance Prediction of Central Obesity Independent of Gender, Race, Adult Psychosocial Risk and Health Behaviors

Cynthia R. Davis[1], **Eric Dearing**[2], **Nicole Usher**[1], **Sarah Trifiletti**[1], **Lesya Zaichenko**[3], **Elizabeth Ollen**[1], **Mary T. Brinkoetter**[3,4], **Cindy Crowell-Doom**[1], **Kyoung Joung**[3,4], **Kyung Hee Park**[3,5], **Christos S. Mantzoros**[3,6], and **Judith A. Crowell**[1,7]

[1]Judge Baker Children's Center, Boston, MA

[2]Boston College, Boston, MA

[3]Division of Endocrinology, Diabetes & Metabolism, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA

[4]Division of Newborn Medicine, Boston Children's Hospital, Boston, MA

[5]Department of Family Medicine, Hallym University Sacred Heart Hospital, Hallym University, Gyeonggi-do, Korea

[6]Section ofEndocrinology, Boston VA Healthcare System, Harvard Medical School, Boston, MA

[7]Department of Psychiatry and Behavioral Science, Stony Brook University School of Medicine, Stony Brook, NY

Abstract

Objective—This study examined whether a novel indicator of overall childhood adversity, incorporating number of adversities, severity, and chronicity, predicted central obesity beyond contributions of "modifiable" risk factors including psychosocial characteristics and health behaviors in a diverse sample of midlife adults. The study also examined whether the overall adversity score (number of adversities X severity X chronicity) better predicted obesity compared to cumulative adversity (number of adversities), a more traditional assessment of childhood adversity.

Address correspondence and reprint requests: Cynthia R Davis, Ph.D. Judge Baker Children's Center, Boston, MA, 53 Parker Hill Avenue, Boston, MA 02120, USA. Phone: 617-278-4193; Fax: 617-232-8399; crdavis@jbcc.harvard.edu.

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Authors' Contributions

Cynthia R. Davis: Manuscript writing, data analysis, data interpretation, data collection, data coding Eric Dearing: Manuscript writing, data analysis, data interpretation Nicole Usher: data collection, data coding Sarah Trifiletti: data collection, data coding Lesya Zaichenko: data collection Elizabeth R. Weber: data collection, data coding Mary T. Brinkoetter: data collection Cindy Crowell-Doom: data coding Kyoung Joung: Manuscript writing, data interpretation, data coding Kyung Hee Park: Manuscript writing, data interpretation, data coding Christos S. Mantzoros: Manuscript writing, data interpretation, data collection Judith A. Crowell: Manuscript writing, data interpretation, data coding, data collection

Materials/Methods—210 Black/African Americans and White/European Americans, mean age = 45.8; ±3.3 years, were studied cross-sectionally. Regression analysis examined overall childhood adversity as a direct, non-modifiable risk factor for central obesity (waist-hip ratio) and body mass index (BMI), with and without adjustment for established adult psychosocial risk factors (education, employment, social functioning) and heath behavior risk factors (smoking, drinking, diet, exercise).

Results—Overall childhood adversity was an independent significant predictor of central obesity, and the relations between psychosocial and health risk factors and central obesity were not significant when overall adversity was in the model. Overall adversity was not a statistically significant predictor of BMI.

Conclusions—Overall childhood adversity, incorporating severity and chronicity and cumulative scores, predicts central obesity beyond more contemporaneous risk factors often considered modifiable. This is consistent with early dysregulation of metabolic functioning. Findings can inform practitioners interested in the impact of childhood adversity and personalizing treatment approaches of obesity within high-risk populations. Prevention/intervention research is necessary to discover and address the underlying causes and impact of childhood adversity on metabolic functioning.

Keywords

Central obesity; childhood adversity; psychosocial risk factors; modifiable risk factors

Introduction

Obesity, especially central adiposity, and metabolic syndrome (MetS) place adults at high risk for other physical health problems, especially diabetes mellitus (DM), cardiovascular disease (CVD), and hypertension [1–9]. Central obesity has been associated with early stressful environments and events [10–13], including intra-uterine stresses and early illnesses [14, 15], poverty [16], and specific and cumulative stresses such as physical and sexual abuse in childhood or death of a close family member [17–20], in both animal models and human studies [14, 21–23]. Psychosocial factors including socioeconomic status (SES), education, and functional status (adjustment or functioning in the domains of mental health, work, leisure/interests, and close relationships) provide a mediated link between early life stressors and later health [12, 16, 24–27]. Impaired psychosocial functioning is associated with health risk factors [28, 29], such as smoking, drinking, poor diet, and sedentary lifestyle that set the stage for poor health outcomes in general.

Many psychosocial factors and health risk factors are considered modifiable, with the potential to decrease obesity rates and costs [30], and are the focus of many prevention/intervention programs. However, it is rare for such programs to assess childhood adversity and its potential direct, non-mediated impact on metabolic functioning, central obesity versus overall obesity, and outcomes. [12, 23, 31, 32].

Examining childhood adversity

The growing literature examining associations between early adversity and adult physical health typically uses cumulative adversity scores to assess the number of adversities an individual has experienced [10–12, 19]. This work addresses the concepts of severity and chronicity of stress [11, 12], but severity and chronicity of experiences are often inferred from the nature of the childhood adversity (e.g., maltreatment is considered to be severe and low SES chronic), rather than assessed and incorporated into measurements of adversity. The large sample sizes of many investigations preclude more in-depth assessments of these dimensions. Nevertheless, specific information on severity and chronicity could address issues of resilience and also allow for more personalized treatment plans and outcome expectations [33–35]. Unlike a large scale study, samples in which detailed, interview-based, childhood adversity histories are obtained allow for assessments of severity and chronicity. They also provide an opportunity to compare the predictive power of a cumulative score with a potentially more clinically relevant adversity score that incorporates number of adversities with severity and chronicity information.

The current study examines the impact of childhood adversity on midlife obesity in a racially and socioeconomically diverse, moderate-risk, but non-clinical sample. We explore a novel assessment of childhood adversity (number of adversities X severity X chronicity) as a direct predictor of central obesity compared with overall obesity. Additionally, we examine the contributions of current psychosocial (education, employment, social functioning) and health risk factors (smoking, drinking, diet and exercise). We hypothesize that the enhanced, interview-based overall childhood adversity score is a better predictor of central obesity than the cumulative adversity score, and will contribute to the prediction of central obesity beyond the more proximal midlife psychosocial and health risk factors.

Methods

Sample

Participants were 210 adults (mean age = 45.8; ±3.3; range 35–55 years), of diverse SES backgrounds who were part of a study examining psychosocial influences on physical and mental health in midlife. The sample was generally representative of the population of Boston, MA with regard to proportion of men and women, European Americans, and those with a Bachelor’s degree or higher, although it included a greater proportion of Black/African Americans [36]. The sample had an approximately equal distribution of men and women, and Black/African Americans and White/European Americans. Recruitment aimed at balancing first employment status and then educational level within groups divided by race and gender. Institutional Review Boards of all participating institutions approved the study. Participants gave written informed consent.

Forty-seven participants of predominantly European American-descent (96.5%) were recruited from a 30+ year longitudinal study that originally assessed a range of psychosocial functioning in adolescents of middle-to-high SES (mean age = 14.6; range 13–18 years). This cohort is described elsewhere [37, 38]. An additional 163 participants of similar age and socioeconomic status were recruited over 20 months through advertising (radio,

newspapers, flyers, health fairs, academic conferences) in the Boston area. Eligibility criteria included being between 40–50 years old and identification of a stable residence. Among the eligible population, those with serious medical illness, e.g., heart disease, cancer, diabetes were excluded from the study. Of 963 individuals who inquired about participation, 247 did not return calls or never showed up for their visit, 44 were not interested after learning more, 501 were ineligible or their inclusion would skew the balance among groups with respect to employment and education, and 171 came for assessment. Nine potential participants were excluded from overall study participation because of electrocardiogram (ECG) findings indicative of past myocardial infarction or fasting finger-stick blood glucose level indicative of diabetes during the physical health evaluation.

The combined moderate-risk sample was 23% (n=49) White/European American men; 19% (n=39) White/European American women; 24% (n=51) Black/African American men; 34% (n=71) Black/African American women. Thirty-four percent of participants had a Bachelor's degree or higher, and 31% were unemployed.

Procedure

Participants arrived at 8:00 AM at the Clinical Research Center of Beth Israel Deaconess Medical Center after an overnight fast. Nurses conducted screening ECGs and measured seated blood pressure twice with a 5-minute interval, and once standing. A physician conducted a physical examination and standardized medical history. A dietician measured height, weight, smallest waist and iliac hip circumferences. Participants then went to Judge Baker Children's Center for psychosocial assessments.

Measures

Obesity—Obesity indicators were obtained by a registered dietician. Body mass index (BMI) was calculated as (weight kg)/(height m^2) and is used as a measure of overall obesity. A ratio score for the waist-hip ratio (WHR) measurement was created to compensate for differing cut-off scores for men versus women. Scores above 1.00 indicated a non-optimal waist-hip ratio indicative of central obesity.

Overall Childhood Adversity—Cumulative adversity [10] occurring before age 18 was assessed using a) the Evaluation of Lifetime Stressors interview assessing trauma exposure [39], b) the Structured Clinical Interview for Diagnoses Diagnostic and Statistical Manual (DSM) IV-R Non-Patient Version Axis 1 including the Post-Traumatic Stress Disorder module [40], and c) the Adult Attachment Interview yielding narrative descriptions of childhood adversities [41].

All participants were assessed during the current phase. Two coders reviewed each interview. An adversity was tallied if the participant presented an unambiguous description, regardless of meaning attributed to the experience. The most prevalent adversities were parental divorce (42%), physical abuse (41%), prolonged separation from parent (34%), sexual abuse (30%), domestic violence (29%), emotional abuse (23%), parental substance abuse (21%), and death of a first-degree family member (20%). A cumulative adversity sum score was obtained (range 0–13).

Severity of adversity was assessed using a modified version of DSM-III-R Axis IV scale for children and adolescents. Childhood and adolescence was broken into 4 blocks (0–5 years, 6–10 years, 11–14 years, and 15–18 years). Adversities for each block of years were rated using a 4-point scale (0 = none, 1= mild, 2 =moderate, 3 =severe, 4 =extreme), e.g., a time block that included abuse could be rated as moderate, severe, or extreme depending on such factors as who the perpetrator was, reactions of caregivers, use of weapons, and other life contexts that may not typically be assessed with rating scales, e.g., bullying, racial discrimination, witnessing a sibling being murdered. A summary score covering ages 0–18 years was generated from the mean of the four block ratings and rounded to the nearest half number (M = 2.35; ±2.35). Although the DSM Axis IV no longer uses this scale because agreement among clinicians was not optimal [42], coding agreement between two blind coders on 24% of the sample using the expanded version of the scale was good, r = .85.

Chronicity of adversity was assessed on a 3-point scale. As for severity, chronicity was rated for the 4 time blocks of childhood and adolescence. A score of 1 was given for single episodes or acute events, e.g., a hospitalization of the child for an acute illness or death of a grandparent, 2 = a chronic situation such as an alcoholic caregiver or poverty that lasted throughout the time block, and 3 = mixed, i.e., acute stresses occurring in the context of ongoing, chronic adversity. A summary score for covering ages 0–18 years was generated from the four block ratings and rounded to the nearest whole number (M = 1.91; ± .90). Inter-rater agreement was very high, r = .96.

Cumulative adversity, severity of adversity, and chronicity of adversity were strongly correlated with each other (range r = .50 – .76). An overall adversity score was created by multiplying the number of childhood adversities X the overall severity of childhood adversity X the overall chronicity of childhood adversity. Scores for overall adversity ranged from 0 to 156. This variable was positively skewed. Therefore a log transformation was performed, which improved the normality of the distribution.

Social adjustment/mental health—The Social Adjustment Scale is a semi-structured interview assessing functioning in the preceding 2 months in domains of work (including employment functioning, homemaking and other household functions, and/or student/educational functioning), friendships/leisure, and relationships with extended family [43]. If applicable, relationships with immediate family members (spouse/partner and/or children) are also assessed. The SAS is closely linked to mental health and can be used as a tool for assessing treatment response to psychotropc medications or therapies. Positive adjustment is the ability to carry out each activity/role effectively, deriving satisfaction/support from that domain, whereas poor adjustment reflects maladaptation, dissatisfaction, disengagement, and/or discord. Scores range from 1 (excellent adjustment) to 7 (very poor adjustment). Coding was completed during an audio-recorded interview; 12% were coded for agreement (91%).

Education and Employment—Self-reported years of formal education and employment status (employed/unemployed) were obtained with a structured demographic interview.

Psychosocial Risk Factor Score—An index score of psychosocial risk factors was created. Education less than a Bachelor's degree, unemployment, and a social adjustment scale score indicative of non-optimal functiong (≥ 3) were considered risk factors, coded as "1", and then tallied. The range of scores for psychosocial risk factors in the current sample was between 0 and 3 psychosocial risk factors.

Smoking and Alcohol Use—A standardized medical history form was used by a study physician to record medical history. Health behaviors reported by the participant on the date of their study visit were also recorded, including smoking status (packs per day and for how many years) and alcohol consumption (drinks per day and drinks per week).

Diet—The Block Food Frequency Questionnaire (FFQ) [44] was used to assess participants' dietary intake. A nutrient intake score was calculated by multiplying the consumption frequency for each food item by its pre-specified common serving size, and then summing according to the Alternative Healthy Eating Index food groups (AHEI) [45] that are associated with disease and mortality risk [46]. The AHEI score was calculated from the FFQ based on these components: vegetables, fruit, cereal fiber, nuts and soy, ratio of white to red meat, trans fat, ratio of polyunsaturated to saturated fatty acids, multivitamin use, and alcohol consumption.

Exercise—Exercise variables were recorded in a self-reported physical health questionnaire. Participants reported the type and typical duration of their aerobic and anaerobic regular exercise, including the type of sport, hours per week, months per year, and total years of practice as well as daily activities such as walking, resting, dancing, or gardening. The energy expenditure of the reported regular exercise was estimated using metabolic equivalent hours per week [47].

Health Behavior Risk Factor Score—An index score of health behavior risk factors was created. Any amount of smoking, non-optimal drinking (≥ 7 drinks per week for women; ≥ 14 drinks per week for men), a score in the bottom tertile of the AHEI, and minimal exercise (< 6 metabolic hours per week) were considered risk factors, coded as "1", and then tallied. The range of scores for health risk factors in the current sample was between 0 and 4 health risk factors.

Statistical Analysis

Study data were collected and managed using REDCap electronic data capture tools hosted at Beth Israel Deaconess Medical Center [48]. REDCap (Research Electronic Data Capture) is a secure, web-based application designed to support data capture for research studies, providing: 1) an intuitive interface for validated data entry; 2) audit trails for tracking data manipulation and export procedures; 3) automated export procedures for seamless data downloads to common statistical packages; and 4) procedures for importing data from external sources.

Descriptive analyses were performed and Pearson correlations examined bivariate relations among study variables to check for multicolinearity; they were not hypothesis-generating. Analyses were performed using a series of linear regression models. Analyses were

performed using SPSS v20 and STATA. Across all participants and variables, 86% of the data was complete. Reasons for missingness varied (e.g., unable to draw blood, recording equipment failure) and were unrelated to key variables. To adjust for potential bias caused by missing data, we used full information maximum likelihood estimation [49, 50].

Results

Table 1 shows descriptive information for the entire sample. Anthropometric assessments are consistent with those described in the Third National Health and Nutrition Examination Survey [51].

Zero-order correlations are presented in Table 2. The psychosocial risk factor score was significantly associated with both WHR and BMI, but the health risk factor score was associated only with BMI.

Table 3 shows the results of two linear regression analyses predicting WHR, first with the overall adversity score (cumulative X severity X chronicity), then with the cumulative adversity score alone. Two parallel analyses predict BMI.

We combined the two samples to enhance statistical power. Yet, to ensure that the two samples were, in fact, comparable, we included a dummy variable indicating the sample group and interactions between sample group and each of the predictors of interest. We then included these covariates in the regression models presented in Table 3, first with just the dummy and second with the dummy and interactions. There were no significant differences in the outcomes associated with the sample group, and the patterns of association between predictors and outcomes did not statistically vary across groups. Moreover, the pattern of results presented in Table 3 remained evident with or without the sample group covariates.

Predicting WHR

Model 1a shows that overall childhood adversity is a significant predictor of WHR, and remains a significant predictor of WHR, even when current psychosocial risk factor scores and health risk factor scores are entered into the model (2a). In contrast, Model 3a shows that cumulative adversity predicts WHR, but when health risk factors and psychosocial risk factors are added to the model, psychosocial risk factors is the only predictor of WHR (Model 4a).

Predicting BMI

The series of regressions starting with Model 1b shows that overall adversity and gender predict BMI (Table 3). However, Model 2b shows that once psychosocial and health risk factor scores were entered into the model, neither overall adversity nor the two risk factors account for enough unique variance to be statistically significant. Model 3b, which examines cumulative adversity as a predictor of BMI, shows that only gender and race are significant predictors.

Discussion

Results indicate that overall childhood adversity is an important predictor of central obesity, over and above the more proximal contributions of adult psychosocial and health risk factors. This underscores the impact of childhood adversity, taking into account its severity and chronicity, on metabolic functioning well into adulthood. We do not see the same effect of overall adversity in models predicting BMI, suggesting that overall childhood adversity is more specific to central, rather than overall, obesity. Findings for cumulative adversity indicate that it is a less predictive than overall childhood adversity of both central (WHR) and overall obesity (BMI) in this moderate-risk population. While the findings are consistent with other studies that demonstrate childhood adversity is predictive of adult obesity [6, 13, 23, 52–57], it is unique in its comparison of central versus overall obesity, incorporation of psychosocial and health risk factor pathways, and its effort to quantify stress more specifically and in a way that is compatible with clinical care [13].

Childhood adversity impacts two general paths leading to central adiposity, a physiological route whereby repetitive early stress alters inflammatory and metabolic functioning, and a psychosocial route whereby risk and protective factors accumulate over time impacting on adult functioning and health behaviors [3, 12, 58]. The path from adversity to altered metabolic functioning involves hypothalamic-pituitary-adrenal (HPA) axis and inflammatory responses to high levels of stress and to subsequent disturbances in this system with accumulating allostatic load [11, 12]. Children who have experienced chronic or repetitive stress such as maltreatment [11] are at risk for obesity, likely due, in part, to increased appetite and preference for high fat, high energy foods [23, 53]. Glucocorticoids (GCs) also impact on free fatty acids, both their preferential uptake into central adipose tissue and their release in association with stress and emotions of fear and anger [23, 31, 58]. The development of insulin and leptin resistance under conditions of stress further leads to alterations of appetite and metabolism [13]. The findings lend evidence to a pathways hypothesis that these metabolic alterations are carried into adulthood, even accounting for more proximal psychosocial risk and health risk factors [13].

Of note, findings fit with important goals of personalized medicine, that is, identifying individual characteristics that shape treatment selection and outcomes [34]. In this case, specific history gathering regarding the severity and duration of adversities could be of value in understanding risk and prognosis for individual patients and the degree to which they might respond to interventions. The results are informative regarding health disparities, given the association between overall adversity and race in the current sample, suggesting that a contributing factor to observed disparities in health is the greater level of childhood adversity experienced by Black/African American individuals. Indeed, if we are to understand both risk and protective factors related to resilience, fine-grained assessments of experience may offer important insights. Lastly, the overall adversity score is beneficial from a statistical standpoint, as it captures more variance in childhood adversity.

Limitations

The relatively small sample is a potential limitation but the findings are robust and significant. Indeed, careful assessment of severity and chronicity variables is easier to achieve in a small sample that utilizes in-depth assessments.

Bias in retrospective reporting of childhood adversities may represent another limitation. However, several important studies support the validity of such retrospective reports, finding negligible differences between prospective and retrospective reports [12, 59, 60].

Conclusion

A number of large epidemiological investigations indicate that childhood adversity presents a considerable public health issue in midlife. Many studies and social policies highlight modifiable factors such as current health behaviors and social support as important contributors to physical health [61]. However, clinicians, policy makers, and researchers focusing on intervention should also take into consideration those in moderate to high-risk populations who are likely to have past experiences of childhood adversities. The processes by which childhood adversity exerts its impact need further delineation, including enhanced assessments of risk and protective factors. More research is needed to better understand whether and how long-term disruption of metabolism can be reversed through intervention and prevention programs aimed at lifestyle changes, and whether the addition of novel approaches to established interventions may minimize the impact of childhood adversity across the life span.

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Abbreviations

MetS	Metabolic Syndrome
HPA	hypothalamic-pituitary-adrenal
GC	Glucocorticoids
SES	Socioeconomic Status
ECG	electrocardiogram
BMI	body mass index
WHR	waist-hip ratio
DSM	diagnostic and statistical manual
FFQ	food frequency questionnaire

AHEI alternative healthy eating index

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Table 1

Means (standard deviations) for study variables

	Mean/Frequency	Standard Deviation	Range
Age (years)	45.79	3.30	35–55
Race (Black/African American)	58.1%		
Gender (Female)	52.4%		
Cumulative Adversity	3.24	2.73	0–13
Overall Childhood Adversity	22.41	29.98	0–156
Bachelors Degree	36.9%		
Employed	68.8%		
Social Adjustment	2.47	1.21	1–7
Smoking (pack per year)	70.60	134.90	0–912.50
Alcohol use (servings per week)	3.03	7.30	0–70
Diet (AHEI score)	44.81	12.22	18.17–76.06
Exercise (metabolic hours/week)	16.84	17.54	0–67.50
Psychosocial Risk Factors	1.03	.88	0–3
Health Risk Factors	1.33	1.06	0–3
Waist-Hip Ratio (ratio by gender)	1.06	.09	.86–1.27
BMI (kg/m^2)	30.41	7.22	18.20–51.46

Cumulative adversity = sum of unique adverse experiences; overall adversity = cumulative adversity X adversity severity X adversity chronicity. Psychosocial risk = sum of psychosocial risk factors (less than a Bachelors degree + unemployed + non-optimal social functioning); health risk = sum of health risk factors (any amount of smoking + non optimal drinking + poor diet + minimal exercise).

Table 2

Pearson correlation coefficients for study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Race	--													
2. Gender	.15*	--												
3. Cumulative Adversity	.37***	.09	--											
4. Overall Adversity (Log)	.45***	.13	.89***	--										
5. Bachelors Degree	−.33***	.14	−.28***	−.36***	--									
6. Employed	−.04	.02	−.22**	−.32***	.28***	--								
7. Social Adjustment	.14	−.08	.30***	.42***	−.29***	−.37***	--							
8. Smoking	−.07	−.05	.18*	.25*	−.22**	−.25**	.24**	--						
9. Alcohol use	−.04	−.05	−.15	−.26**	−.03	−.09	−.10	.04	--					
10. Diet	−.12	.12	−.13	−.23*	.29***	.11	−.42***	−.15	.08	--				
11. Exercise	−.14	−.09	−.08	−.09	.10	−.03	−.31***	−.02	.14	.28***	--			
12. Psychosocial Risk Factors	.22**	−.07	.34***	.47***	−.70***	−.75***	.68***	.27***	.02	−.31***	−.13	--		
13. Health Risk Factors	.22**	−.03	.14	.19	−.41***	−.29***	.45***	.42**	.09	−.62***	−.46***	.49***	--	
14. Waist-Hip Ratio	.01	−.08	.17*	.29**	−.23**	−.13	.27***	.15	−.13	−.26**	−.19*	.27**	.19*	--
15. BMI	.24**	.19*	.17*	.31**	−.22**	−.13	−.17*	−.12	−.13	−.27***	−.20*	.23**	.14	.45***

*** $p < .001$;

** $p < .01$;

* $p < .05$.

Note: Race: 1 = Black/African American; 0 = White/European American. Gender: 1 = Female; 0 = Male. Cumulative adversity = sum of unique adverse experiences; overall adversity = cumulative adversity X adversity severity X adversity chronicity. Psychosocial risk = sum of psychosocial risk factors (less than a Bachelors degree + unemployed + non-optimal social functioning); health risk = sum of health risk factors (any amount of smoking + non optimal drinking + poor diet + minimal exercise).

Table 3

Multivariate linear regression analyses predicting waist-hip ratio and body mass index in relation to study variables (N = 210).

Outcome	Model	Predictor	*b*	Beta	Standard Error
Waist-Hip Ratio	M1a	Overall Adversity	.04**	.27**	.01
		Race	−.01	−.05	.01
		Gender	−.02	−.12	.01
	M2a	Overall Adversity	.03*	.20*	.01
		Race	−.01	−.07	.01
		Gender	−.02	−.09	.01
		Psychosocial Risk	.01	.17	.01
		Health Risk	.01	.06	.01
	M3a	Cumulative Adversity	.01*	.17*	.00
		Race	−.00	−.02	.01
		Gender	−.02	−.12	.01
	M4a	Cumulative Adversity	.00	.11	.00
		Race	−.01	−.05	.01
		Gender	−.01	−.08	.01
		Psychosocial Risk	.02*	.20*	.01
		Health Risk	.01	.06	.01
Body Mass Index	M1b	Overall Adversity	2.08*	.19*	.92
		Race	1.94	.13	1.07
		Gender	2.30*	.16*	1.04
	M2b	Overall Adversity	1.39	.13	.97
		Race	1.67	.11	1.07
		Gender	2.68*	.18*	1.06
		Psychosocial Risk	1.15	.16	.68
		Health Risk	.25	.03	.73
	M3b	Cumulative Adversity	.26	.10	.20
		Race	2.32*	.16*	1.08
		Gender	2.36*	.16*	1.06

Outcome	Model	Predictor	*b*	Beta	Standard Error
	M4b	Cumulative Adversity	.10	.04	.21
		Race	1.94	.13	1.08
		Gender	2.82 *	.19 *	1.08 *
		Psychosocial Risk	1.37	.19	.70
		Health Risk	.28	.04	.73

*** $p < .001$;

** $p < .01$;

* $p < .05$.

Note: Cumulative adversity = sum of unique adverse experiences; overall adversity = cumulative adversity X adversity severity X adversity chronicity. Race: 1 = Black/African American; 0 = White/European American. Gender: 1 = Female; 0 = Male. Psychosocial risk = sum of psychosocial risk factors (less than a Bachelors degree + unemployed + non-optimal social functioning); health risk = sum of health risk factors (any amount of smoking + non optimal drinking + poor diet + minimal exercise).