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Adverse Childhood Experiences and BMI:

Lifecourse Associations in a Black-White U.S. Women Cohort

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ABSTRACT

Introduction: While adverse childhood experiences (ACEs) have been positively associated with adiposity, few studies have examined long-term race-specific ACE-body mass index (BMI) relations.

Methods: A Black and White all-women cohort (N=611; 48.6% Black) was followed between 1987-1997 from childhood (age 9/10) through adolescence (age 19/20) to midlife (age 36-43, between 2015-19). In these 2020-22 analyses, interaction between race and individual ACE exposures (physical abuse, sexual abuse, household substance abuse, multiple ACEs) on continuous BMI at age 19/20 and midlife were evaluated individually through multivariable linear regression models. Stratification by race followed as warranted at α=0.15.

Results: Race only modified ACE-BMI associations for sexual abuse. Among Black women, sexual abuse was significantly associated with BMI ($B_{adjusted}$ =3.24, 0.92, 5.57) at age 19/20 and marginally associated at midlife ($B_{adjusted}$=2.37, 95% CI: -0.62, 5.35); among White women, corresponding associations were null. Overall, having ≥2 ACEs was significantly associated with adolescent BMI ($B_{adjusted}$=1.47, 95% CI: 0.13, 2.80) and marginal at midlife ($B_{adjusted}$=1.45, 95% CI: -0.31, 3.22). This was similarly observed for physical abuse (for adolescent BMI: $B_{adjusted}$=1.23, 95% CI: -0.08, 2.54; midlife BMI: $B_{adjusted}$=1.03, 95% CI: -0.71, 2.78), but not for substance abuse.

Conclusions: Direct exposure to certain severe ACEs is associated with increased BMI among Black and White women. It is important to consider race, ACE type, and life stage to gain a more sophisticated understanding of ACE-BMI relations. This knowledge can help strengthen intervention, prevention, and policy efforts aiming to mitigate impacts of social adversities and trauma on persistent cardiometabolic health disparities over the lifecourse.

INTRODUCTION

Understanding racial/ethnic and gender patterning of relations between adverse childhood experiences (ACEs) and anthropometric measures including body mass index (BMI) may help advance health equity work. ACEs are experiences before age 18 that include childhood maltreatment (e.g., physical abuse, sexual abuse) and household hardships (e.g., having household member(s) that misuse/abuse alcohol/drugs),[1,2] and can adversely impact health.[3] With severe and/or chronic exposure, ACEs become biologically embedded in the body via changes in psychological/behavioral pathways, physiological/biochemical processes, and their interaction.[3–5]

That ACEs can increase weight is largely accepted in the literature.[3,4,6–10] Disruptions to nervous, endocrine (e.g., the hypothalamic-pituitary-adrenal [HPA] axis), and immune systems during childhood can lead to decreased executive function, emotional and self-regulation, and attention,[3] as well as higher basal cortisol levels,[11] which can increase appetite and promote central fat deposition,[5] among other physiological changes.[12] As greater health impacts are seen with maltreatment-type versus household dysfunction-type ACEs, research has urged study by adversity type.[13] A systematic review found associations between sexual abuse and obesity to be "robust" and, for physical abuse, "plausible [but] limited."[2] That review, like many studies in this field, based their analyses on increasingly controversial categorical weight classifications rather than continuous BMI.[2] However, reliance on such weight classifications (i.e., overweight: 25-29.99 kg/m^2; obesity: ≥ 30 kg/m^2) can muddy analyses when large sample proportions have higher weight or non-white racial/ethnic backgrounds.[14] Specific health risks[15,16] associated with continuous BMI may exist, but can be lost when otherwise dissimilar people are classed together.

Moreover, elevated disease risk corresponding to BMI classifications is debatable and varies by gender and race/ethnicity,[17] partly attributable to anthropometric and environmental differences.[18] Thus, using continuous BMI, which also confers statistical benefits,[19,20] is especially justified in heterogeneous populations.

Clarifying the role of race/ethnicity in the anthropometric burden of ACEs is needed.[2] However, it should be emphasized that differential risks associated with adiposity between groups are not thought to be the result of biology but societal forces. "Double jeopardy," weathering, social vulnerability, and cumulative risk models all postulate that minoritized groups including Black/African-Americans endure lived experiences rooted in oppressive systems and are at compounded vulnerability to stressful events/triggers such as ACEs.[21–24] Particularly in the U.S., race carries social, economic, and historical disadvantage via structural racism and individual-level discrimination.[25,26] Black communities often live with more chronic negative social, environmental, psychological stressors across the lifecourse[27] with demonstrated wear-and-tear on physiological systems.[24,26] Following the Environmental Affordances Model, Black individuals may manifest a stronger ACE-obesity link due to biopsychosocial processes and less healthy coping behaviors like using food to cope/stress-related eating,[27,28] common in more resource-poor and constrained settings.[27,29,30] Studies of ACE-excess weight in minoritized racial/ethnic groups, however, are inconclusive,[31,32] with few utilizing continuous BMI.[33]

In this study, ACE-BMI associations were examined by ACE type and race in a Black-White cohort of women across two life stages – late adolescence and midlife. The focus on women is valuable also because of growing interest in the intersectional influences aspects of persons'

identities (here, racism and sexism) may simultaneously have on health.[34] Hypotheses were: 1) associations between all ACE exposures and BMI will be stronger for Black women, and 2) overall, ACE-BMI associations will be larger with ACE severity (from "least" to "most" severe: household substance abuse, physical or sexual abuse, experiencing multiple ACEs) and recency (dissipating over time from late adolescence to midlife).

METHODS

Study Sample

This study utilized data from the original (1987-1999) National Heart Lung and Blood Institute's (NHLBI) Growth and Health Study (NGHS), which was conceived when the vast Black-White disparities in obesity rates among U.S. adolescent girls was gaining national public health attention,[35] and its follow-up (2015-2019). In the original three-site study, Black and White girls were followed annually from age 9/10 until age 19/20. The follow-up (ages 36-43) engaged the Richmond, CA site. The childhood eligibility criteria are outlined elsewhere.[36] For the follow-up, participants could not be pregnant, incarcerated, or recently given birth/miscarried. Of the original Richmond cohort (n=882), 624 (70.7%) participated though more Black women and those with lower household income and parental educational attainment were lost; baseline BMIs did not significantly differ. Individuals without at least one response for ACEs (n=12) and BMI (n=1) at age 19/20 or 36/43 were omitted (analytic sample *n*=611). As participants were recruited as girls at age 10, gender here is synonymous with biological sex.

Measures

Demographic information was reported by participants and/or their parent(s)/guardian(s) during the original study. At follow-up, online and paper (if preferred) surveys and new assessments were completed. Participants received remuneration and provided informed consent. Protocols were approved by the University of California, Berkeley Institutional Review Board.

For ACEs, at follow-up, experiences with physical, sexual, and household substance abuse (selected given their associations with obesity/adiposity in the literature)[4,31,32,37] before age 18 were retrospectively self-reported. Original questions[4] for these ACEs were adapted with minor modifications; answer choices were yes, no, and "prefer not to answer." To assess cumulative, co-occurring, and/or multiple ACEs exposure, exposures were summed to create a binary variable identifying women with "high ACEs exposure" ($\geq$ 2 ACEs).[38]

For BMI, at ages 9/10, 19/20, and for "local" ($\leq$60 miles) participants at follow-up, trained study staff measured height and weight three times each per National Health and Nutrition Examination Survey (NHANES) protocols.[39] For "distant" (>60 miles) participants at follow-up, staff mailed an NHANES-aligned set of tools to self-measure with staff guidance.[39] All participants also self-reported height and weight on study questionnaires; these values were used to calculate BMI when <3 measured heights and weights were available. No significant differences were observed between BMI calculated from measured vs. self-report values on sociodemographic and psychosocial attributes, but slight differential misclassification by weight was significant ($BMI_{self\text{-}report}$ = -0.04*$BMI_{measured}$, $p<0.001$; e.g., if $BMI_{measured}$ =30 kg/m^2, $BMI_{self\text{-}report}$ =28.8 kg/m^2).

Race (Black, White) was explored as an effect modifier, sourced from baseline parent-reported data. These data were cross-checked with follow-up self-reported race data (100% agreement).

Possible confounders drawn from the literature[1] included household income, parental educational attainment, number of parents in household, and number of siblings (all parent-reported at baseline).

Statistical Analysis

Analyses took place in 2020-23. Interaction terms were constructed between each ACE exposure and race. Likelihood ratio tests assessed whether fully-adjusted models (controlling for all covariates) with each ACE-race interaction term individually provided better fits than nested models without. An expanded α-level (0.15) was used to assess modification.[40] When interaction was significant ($p<0.15$), all corresponding analyses at that and any subsequent time points (e.g., midlife if race*ACE interaction term for $BMI_{age\ 19/20}$ was significant) were stratified by race to calculate respective ACE-BMI associations. When interaction was not significant ($p \geq 0.15$), respective ACE-BMI associations for the overall sample were calculated in linear models, adjusting for race as warranted. Statistical significance was evaluated against $\alpha=0.05$ plus consideration of the clinical significance of point estimates and their 95% CIs. To evaluate hypotheses regarding ACE severity, qualitative comparisons of association magnitudes (i.e., point estimates, standardized betas) were made as more formal statistical testing was precluded. A complete case analysis approach was taken, and each model's analytical sample sizes are noted (see Tables 2-3). Analyses utilized Stata15 SE (College Station, Texas).

A substantial portion (13.4%, Table 1) at baseline met obesity criteria (BMI-for-age-and-sex $\geq 95^{th}$ percentile).[41] Early excess weight/weight gain is associated with childhood[42] and adulthood[43] excess weight status. To ensure ACE-BMI temporality and minimize influences of earlier childhood excess weight/weight gain, sensitivity analyses ran fully-adjusted models limited to women $<95^{th}$ BMI percentile at age 9/10.

RESULTS

Table 1 displays demographics of the analytical sample (M_{age} at follow-up: 39.5 years, 48.6% Black). At baseline, a higher percentage of Black vs. White women had lower household income, lower parental education, more siblings, and lived in single-parent households. While physical abuse, sexual abuse, and high (≥ 2) ACEs exposure was each endorsed by more than 20% of the women overall and household substance abuse by 37%, fewer Black than White women endorsed ACE exposures. More Black women also had BMI $>95^{th}$ percentile at age 9/10 and higher mean BMI at late adolescence and midlife.

Effect modification of ACE-BMI associations by race was only observed for sexual abuse and BMI at age 19/20 (Table 2). Among Black women, endorsing sexual abuse was associated with having 3.24 kg/m^2 higher BMI (95% CI: 0.92,5.57; p=0.02 for χ^2 interaction term) at late adolescence, even after covariate adjustment. By midlife, the adjusted association was still positive but became marginally significant ($p<0.10$)[40] and attenuated with wider confidence intervals reflecting greater imprecision ($B_{adjusted}$=2.37 kg/m^2; 95% CI: -0.62,5.35). Sensitivity

analyses mirrored the pattern of positive sexual abuse-BMI associations with significant associations at age 19/20 ($B_{adjusted}$=2.26 kg/m^2, 95% CI: 0.26,4.25) but only marginally significant associations at midlife ($B_{adjusted}$=2.65 kg/m^2; 95% CI: -0.16,5.46). Interestingly, with sensitivity analyses, the magnitudes of sexual abuse-BMI associations were decreased in late adolescence and slightly heightened at midlife. Among White women, patterns of these point estimates and their 95% CIs suggested very small (~0), if any, associations at late adolescence, midlife, and in sensitivity analyses.

For all other ACE exposures, race-related modification of ACE-BMI relations was not observed ($p>0.15$ for all χ^2 interaction terms). ACE-BMI associations (unadjusted, adjusted, and in sensitivity analyses) by ACE type for Black and White women combined are in Table 3 (including for sexual abuse for reference). Significant physical abuse-BMI associations were marginal at age 19/20 and suggested but more imprecise at midlife. After women with high baseline childhood obesity risk were removed though, a significant, positive association was observed for age 19/20 BMI ($B_{adjusted}$=1.45 kg/m^2, 95% CI: 0.42,2.47) that seemingly persisted at midlife ($B_{adjusted}$=1.42 kg/m^2, 95% CI: -0.18,3.02).

For high ACEs exposure, significant, positive adjusted associations were observed for BMI at age 19/20 ($B_{adjusted}$=1.47 kg/m^2, 95% CI: 0.13,2.80) and suggested at midlife ($B_{adjusted}$=1.52 kg/m^2, 95% CI: -0.25,3.29) with CI's reflecting a range from of slight increases (or small decreases) in BMI at adolescence to larger increases in BMI at midlife. In sensitivity analyses, these associations were significant and heightened (age 19/20: $B_{adjusted}$=1.66 kg/m^2, 95% CI: 0.61,2.71; midlife: $B_{adjusted}$=1.76 kg/m^2, 95% CI: 0.13,3.40). While adolescent high ACEs associations

appeared to be driven by sexual abuse, they appeared to arise from more combinatory influences at midlife. Household substance abuse was not associated with BMI in any model.

Qualitative comparisons of the magnitude and/or statistical significance of ACE-BMI point estimates and standardized betas found them to be greater with ACE severity/saliency and generally diminish from late adolescence to midlife (given overlapping 95% CIs), save for household substance abuse that appeared to change direction. Association estimates were null/smallest for household substance abuse ("least" severe) and greatest for high ACEs exposure ("most" severe).

DISCUSSION

This study examined BMI from age 19/20 to midlife in a Black-White cohort by ACE type to advance understanding of racial/ethnic differences in associations of trauma/severe stress on BMI across the lifecourse. Among Black women, sexual abuse was associated with greater BMI at late adolescence and marginally associated at midlife. Among White women, sexual abuse-BMI associations were not observed at either time point. For all women, high (≥ 2) ACEs exposure and physical abuse were positively associated with BMI. Associations of larger magnitudes were observed with more severe and proximal exposures. While these findings (particularly regarding sexual and physical abuse) were perhaps unexpected given prior systematic reviews,[2] the contrast underscores the value of using continuous BMI and including a more diverse sample.

Still, these findings in women coincided with those of other studies (including those of both men and women as well as those of only women) reporting positive influences of ACEs on BMI at late adolescence/early adulthood[44,45] and midlife[16,32,37,46] in majority White samples, and with others reporting more pronounced ACE-BMI associations in contexts of "more" severe ACEs—e.g., physical abuse[9,16,37,44,46,47] and high ACEs exposures.[9,10,45] Additionally, the notion of greater potency[8,10,31] with particularly severe ACE exposure was supported, given that: 1) ACE-BMI associations appeared to diminish over the lifecourse (as more time elapsed from the stressor), echoing other studies,[10,33] 2) for women endorsing high ACEs exposure, the associations did not appear to emerge beyond the effects of sexual abuse until midlife, and 3) the associations between high ACEs and physical abuse and BMI were heightened in sensitivity analyses.

Effect modification by race was only observed for sexual abuse and BMI; otherwise, associations for the other ACE types analyzed were no different between Black and White women. In other studies investigating race-specific ACE associations, Black/White status did not modify associations between physical abuse and household alcohol abuse with early or mid-adulthood obesity.[32] However, in two race-stratified studies examining childhood physical and/or sexual abuse in early-mid childhood with BMI or incident "class III obesity" at midlife, while race differences were observed, their nature—significant positive associations were observed for physical abuse/physical+sexual abuse combined, only among Whites[31,46]—contrasted the findings here. In another race-stratified analysis, significant associations were not observed between having ≥ 1 ACE and midlife BMI for non-Hispanic White or Black adults.[33] On a macrolevel, reasons for these differences may arise from the fact that these aforementioned studies included both men and women, methodology (e.g., when/how ACEs and anthropometrics were assessed

and/or operationalized, follow-up length, use of continuous/dichotomous models as interactions are scale-dependent, etc.), and other sample characteristics (e.g., proportion non-White, high obesity risk samples).

These findings underscore the importance of context to better address racial disparities in cardiometabolic health. For example, disparities could arise through coping differences forged by the necessity of navigating more constrained environments.[27,30] Racism is pervasive, making the Black lived experience fraught with multiple levels of disadvantage.[25,26] In the context of the everyday, this manifests as increased stress[27] in an already psychologically and physiologically weathered group.[21–24,26] For Black women, moreover, who sit at a specific intersection of gendered racism, sociohistorical contexts of oppression combined with cultural obligations to protect their communities (e.g., Black men) from dominant systems[48] conspire with aforementioned drivers towards a "Superwoman Schema" or "Strong Black Woman" ideology. This representation is depicted as a prosocial, caretaking role adopted at the expense of one's own needs.[49,50] With unequal social contexts for race and gender dictating coping mechanisms for dealing with stress and trauma,[51] avoidant/numbing behaviors are often chosen[49] and using food for relief (particularly those high in fat/sugar) is prevalent.[28,50,52] Moreover, considering environmental factors like easier access to fast food and convenience stores[27], heightened activation of the HPA axis,[5] and greater weight stigma observed in Black women[53] and with sexual abuse[54] (with its own links to food addiction[55]), the obesogenic risks posed by the lot is formidable.

Lastly, greater prevalence of baseline obesity was present among Black vs. White girls, as were higher mean BMIs among Black vs. White women at late adolescence and midlife. Sensitivity analyses proved important as ~13% of the analytical sample met CDC criteria of high obesity risk at baseline. Interestingly, the direction of how estimates changed differed by ACE. For sexual abuse, magnitudes of ACE-BMI associations were 1) attenuated among Black women at late adolescence but 2) heightened at midlife, as were associations for both physical abuse and high ACE exposure at late adolescence and midlife. Attenuation may partly reflect excluding a greater percentage of Black girls in sensitivity analyses.[42] "Reverse causation" could result as girls with early excess weight would be at increased risk for earlier pubertal onset,[56] which could then increase risk for adversities[57,58] with further weight implications. However, most of the sample was prepubertal at baseline.[59] Unexpectedly, heightened associations were also observed, perhaps reflecting greater analytical sensitivity after removing women with baseline obesity plus large sample variation in BMI. Nonetheless, BMI, which reflects significantly ranging adiposity even at the same values,[18] is not reliably associated with individual cardiometabolic risk and risk misclassification is common when solely based on this measure.[14,17]

Limitations

ACEs were retrospectively reported, and self-reported height/weight were used to calculate midlife BMIs when measured values were unavailable. However, retrospectively and prospectively assessed ACEs tend to be in moderate agreement and, while associations based on retrospective ACEs have been found to be underestimated for objectively measured outcomes and overestimated for subjectively reported outcomes, self-reported BMI was in the minority (<20%) and significantly more reported in White vs. Black women. Combined with selection

bias of a more White sample with lower socioeconomic status for these analyses compared to the population, these ACE-BMI estimates could be more conservative than reality.[60] Another factor potentially biasing estimates to the null is that Black women may have underreported ACEs, though this is consistent with the literature and the groups' sociohistorical/cultural contexts.[48,61] Other ACEs, including other household-type ACEs and those with documented ties to excess weight (e.g., emotional abuse),[62] were also not collected in this study, preventing more thorough examination of Black-White modification of ACE-BMI relations. Residual confounding is also possible as similar measures of socioeconomic status may not foster equivalent health contexts and confer similar degrees of health protection for Black and White populations.[35] Additionally, only a single interaction was significant at the α=0.05 level (p=0.02), suggesting the utility of more studies replicating these findings. Lastly, other ACE-BMI-related work has begun to examine the role of moderators like income[63] and mediators, including mental health[6,8,33,47], psychological attributes,[37] identity,[49–51] and unique co-stressors (e.g., perceived racial discrimination).[33] Future work should further consider racial/ethnic and gendered nuances with moderation/mediation analyses and also utilize additional anthropometric and metabolic health indicators.

This study had notable strengths. The 30-year cohort included near-equal numbers of Black and White women facilitating examination of racial differences with sufficient sample size and sensitivity to childhood obesity risk. The original sample encompassed great income diversity, minimizing confounding, and ACEs could be discretely examined and their BMI associations assessed at key life stages.

CONCLUSIONS

Certain severe ACEs are associated with increased BMI among Black and White women. To supplant federally-identified ACE prevention strategies (e.g., supporting families, changing community norms),[64] other initiatives combatting the cardiometabolic toll of social adversity can prioritize efforts to promote stress management, reduce/improve comfort eating, cope with race-related stress, and mobilize systemic medical, scientific, structural, and institutional change against discrimination to right social determinants of health.[28,65] Given considerable prevalences of ACEs and high BMI in Black and White populations and women especially, it remains critical to recognize the multifacetness of ACE-BMI relations.

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TABLES

Table 1. Analytical Sample Participant Characteristics (Mean[SD] and n[%]) Overall and by Race (n=611)

Characteristics	Total[a]	White[a] (n=314)	Black[a] (n=297)	p-value[a,b]
Adverse Childhood Experiences (ACEs, <18 years old), n(%) unless otherwise noted				
Number of ACEs[c], mean(sd)	0.82 (0.95)	0.98 (1.00)	0.65 (0.87)	**<0.0001**
N (%) Missing[d]				
Physical Abuse	143 (23.4%)	91 (29.0%)	52 (17.5%)	**0.002**
N (%) Missing[d]	8 (1.3%)	2 (0.6%)	6 (2.0%)	
Sexual Abuse	131 (21.4%)	77 (24.5%)	54 (18.2%)	0.09
N (%) Missing[d]	28 (4.6%)	11 (3.5%)	17 (5.7%)	
Substance Abuse	227 (37.2%)	140 (44.6%)	87 (29.3%)	**<0.001**
N (%) Missing[d]	9 (1.5%)		9 (3.0%)	
Two or more (2+) ACEs	141 (23.1%)	90 (28.7%)	51 (17.2%)	**0.003**
N (%) Missing[d]	18 (3.0%)	8 (2.6%)	10 (3.4%)	
Body Mass Index (BMI), n(%)				
BMI >95th %-ile at age 9/10	82 (13.4%)	31 (9.9%)	51 (17.2%)	**0.02**
N (%) Missing[d]	6 (1.0%)	2 (0.6%)	4 (1.4%)	
BMI at age 19/20, mean(sd)	25.4 (6.6)	24.2 (5.6)	26.6 (7.4)	**<0.0001**
N (%) Missing[d]	26 (4.3%)	10 (3.2%)	16 (5.4%)	
BMI at age 36/43, mean(sd)	31.7 (9.1)	29.3 (8.0)	34.3 (9.6)	**<0.0001**
N (%) Missing[d]	2 (0.3%)	1 (0.3%)	1 (0.3%)	
Baseline Sociodemographic Characteristics				
Race – Black, n(%)	297 (48.6%)	--	--	
N (%) Missing[d]				
Current Age (years), mean(sd)	39.5 (1.3)	39.5 (1.3)	39.5 (1.2)	0.88
N (%) Missing[d]				
Household Income, n(%)				**<0.001**
<$10K	107 (17.5%)	20 (6.4%)	87 (29.3%)	
$10K-$19,999	106 (17.4%)	39 (12.4%)	67 (22.6%)	
$20K-$39,999	169 (27.7%)	97 (30.9%)	72 (24.2%)	
$40K+	202 (33.1%)	148 (47.1%)	54 (18.2%)	
N (%) Missing[d]	27 (4.4%)	10 (3.2%)	17 (5.7%)	
Highest Parental Educational Attainment, n(%)				**<0.001**
≤ High School	131 (21.4%)	61 (19.4%)	70 (23.6%)	
Some College	281 (46.0%)	116 (36.9%)	165 (55.6%)	
College Graduate+	198 (32.4%)	137 (43.6%)	61 (20.5%)	
N (%) Missing[d]	1 (0.2%)		1 (0.3%)	
Single Parent Household n(%)	196 (32.1%)	62 (19.8%)	134 (45.1%)	**<0.001**
N (%) Missing[d]				
Number of Siblings, mean(sd)	1.31 (1.12)	1.14 (0.97)	1.48 (1.24)	**0.0001**
N (%) Missing[d]				

[a] Percentages and p-values were calculated including missings.

[b] Boldface indicates statistical significance (p<0.05).
[c] Calculated from non-missing ACE data; some participants did not provide a response (yes/no) to all ACE items.
[d] Left blank if no missing data for this variable in the analytical sample.

Table 2. Associations (Bs, 95% CIs) Between Sexual Abuse and Late Adolescent/Midlife BMI by Race[a]

Life Stage	Unadjusted	Adjusted[d]	Sensitivity Analysis[d,e]
Late Adolescence (Age 19/20) [b]	(N=558)	(N=531)	(N=460)
White	0.41 (-1.06, 1.88) (n=293)	-0.01 (-1.51, 1.48) (n=284)	0.61 (-0.55, 1.78) (n=256)
Black	**3.13 (0.90, 5.36)** (n=265)	**3.24 (0.92, 5.57)** (n=247)	**2.26 (0.26, 4.25)** (n=204)
Midlife (Age 36/43)[c]	(N=582)	(N=555)	(N=480)
White	0.39 (-1.70, 2.49) (n=303)	-0.10 (-2.25, 2.06) (n=294)	0.39 (-1.69, 2.48) (n=265)
Black	2.15 (-0.70, 5.00) (n=279)	2.37 (-0.62, 5.35) (n=261)	2.65 (-0.16, 5.46) (n=215)

[a] Boldface indicates statistical significance (p<0.05).
[b] At age 19/20 Race x Sexual Abuse χ^2 term p=0.02 in fully-adjusted models.
[c] At age 36/43 Race x Sexual Abuse χ^2 term p=0.25 in fully-adjusted models.
[d] Adjusted for household income, highest parent education, # of parents in the household, and number of siblings at baseline.
[e] Sensitivity analyses – restricted analyses to girls with BMI-for-age below the 95th percentile at age 9/10.

Table 3. Overall Associations (Bs, 95% CIs) Between ACE exposure(s) and Late Adolescent/Midlife BMI[a]

Life Stage	Unadjusted	Adjusted[b]	Sensitivity Analysis[b,c]
Late Adolescence (Age 19/20)			
Physical Abuse	1.04 (-0.23, 2.30) (N=577)	1.23 (-0.08, 2.54) (N=549)	**1.45 (0.42, 2.47)** (N=472)
Substance Abuse	-0.43 (-1.55, 0.69) (N=576)	-0.52 (-1.71, 0.66) (N=548)	-0.01 (-0.94, 0.93) (N=470)
High (≥ 2) ACEs	**1.41 (0.13, 2.70)** (N=567)	**1.47 (0.13, 2.80)** (N=540)	**1.66 (0.61, 2.71)** (N=466)
[d] (Sexual Abuse)	***1.38 (0.07, 2.69)*** *(N=558)*	***1.47 (0.16, 2.79)*** *(N=531)*	***1.66 (0.61, 2.71)*** *(N=460)*
Midlife (Age 36/43)			
Physical Abuse	0.42 (-1.29, 2.13) (N=601)	1.03 (-0.71, 2.78) (N=573)	1.42 (-0.18, 3.02) (N=493)
Substance Abuse	0.48 (-1.04, 1.99) (N=600)	0.60 (-0.99, 2.18) (N=572)	0.58 (-0.88, 2.04) (N=491)
High (≥ 2) ACEs	1.14 (-0.58, 2.87) (N=592)	1.52 (-0.25, 3.29) (N=565)	**1.76 (0.13, 3.40)** (N=487)
[d] (Sexual Abuse)	*0.73 (-1.05, 2.51) (N=582)*	*1.04 (-0.73, 2.81) (N=555)*	*1.25 (-0.40, 2.90) (N=480)*

[a] Boldface indicates statistical significance ($p<0.05$).

[b] Adjusted for race, household income, highest parent education, # of parents in the household, and number of siblings at baseline.

[c] Sensitivity analyses – restricted analyses to girls with BMI-for-age below the 95th percentile at age 9/10.

[d] Associations for sexual abuse and BMI for the overall cohort are presented here as reference, but are not fully accurate given the existence of statistically significant differences between sexual abuse and BMI associations of Black vs. White women precluding their combination, hence italics.