American Journal of
Preventive Medicine

RESEARCH ARTICLE

Adverse Childhood Experiences and BMI: Lifecourse Associations in a Black—White U.S. Women Cohort

Dorothy T. Chiu, PhD,[1,2] Erika M. Brown, PhD,[1,3] A. Janet Tomiyama, PhD,[4] Kristy E. Brownell, BA,[1,5] Barbara Abrams, DrPH,[6] Mahasin S. Mujahid, PhD,[6] Elissa S. Epel, PhD,[7,8] Barbara A. Laraia, PhD[1]

Introduction: Although adverse childhood experiences (ACEs) have been positively associated with adiposity, few studies have examined long-term race-specific ACE—BMI relationships.

Methods: A Black and White all-women cohort (N=611; 48.6% Black) was followed between 1987 and 1997 from childhood (ages 9—10 years) through adolescence (ages 19—20 years) to midlife (ages 36—43 years, between 2015 and 2019). In these 2020—2022 analyses, the interaction between race and individual ACE exposures (physical abuse, sexual abuse, household substance abuse, multiple ACEs) on continuous BMI at ages 19—20 years and midlife was 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, 95% CI=0.92, 5.57) at ages 19—20 years 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 was marginally associated at midlife ($B_{adjusted}$=1.45, 95% CI= −0.31, 3.22). This was similarly observed for physical abuse (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 relationships. This knowledge can help strengthen intervention, prevention, and policy efforts aiming to mitigate the impacts of social adversities and trauma on persistent cardiometabolic health disparities over the lifecourse.

Am J Prev Med 2024;66(1):73—82.

INTRODUCTION

Understanding racial/ethnic and gender patterns of the relationships between adverse childhood experiences (ACEs) and anthropometric measures, including BMI, may help advance health equity work. ACEs are experiences before the age of 18 years that include childhood maltreatment (e.g., physical abuse, sexual abuse) and household hardships

From the [1]Community Health Sciences Division, Berkeley School of Public Health, University of California, Berkeley, California; [2]Osher Center for Integrative Health, University of California San Francisco, San Francisco, California; [3]California Policy Lab, Berkeley, California; [4]Department of Psychology, College of Life Sciences, University of California, Los Angeles, Los Angeles, California; [5]Kaiser Permanente Division of Research, Oakland, California; [6]Epidemiology Division, Berkeley School of Public Health, University of California, Berkeley, California; [7]Weill Institute of Neurosciences, Department of Psychiatry, University of California San Francisco, San Francisco, California; and [8]The Center for Health and Community, University of California San Francisco, San Francisco, California

Address correspondence to: Dorothy T. Chiu, PhD, Osher Center for Integrative Health, University of California San Francisco, # 301D, 1545 Divisadero Street, San Francisco CA 94115. E-mail: dorothy.chiu@ucsf.edu.

0749-3797/$36.00
https://doi.org/10.1016/j.amepre.2023.09.004

(e.g., having household member(s) who 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 through changes in psychological/behavioral pathways, physiologic/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 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 physiologic changes.[12] Because 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 the associations between sexual abuse and obesity to be "robust" and, for physical abuse, "plausible [but] limited."[2] That review, similar to 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 disadvantages through structural racism and individual-level discrimination.[25,26] Black communities often live with more chronic negative social, environmental, and psychological stressors across the lifecourse[27] with demonstrated wear and tear on physiologic systems.[24,26] Following the Environmental Affordances Model, Black individuals may manifest a stronger ACE−obesity link owing to biopsychosocial processes and less healthy coping behaviors such as using food to cope/stress-related eating,[27,28] common in more resource-poor and constrained settings.[27,29,30] However, studies of ACE-associated excess weight in minoritized racial/ethnic groups 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 the growing interest in intersectional influences that aspects of persons' identities (referred to as racism and sexism in this study) may simultaneously have on health.[34] Hypotheses were that (1) associations between all ACE exposures and BMI will be stronger for Black women and that (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 Growth and Health Study, which was conceived when the vast Black−White disparities in obesity rates among U.S. adolescent girls were gaining national public health attention,[35] and its follow-up (2015−2019). In the original 3-site study, Black and White girls were followed annually from ages 9−10 years until ages 19−20 years. The follow-up (ages 36−43 years) engaged the Richmond, CA site. The childhood eligibility criteria are outlined elsewhere.[36] For the follow-up, participants could not be pregnant, be incarcerated, or have recently given birth/miscarried. Of the original Richmond cohort (n=882), 624 (70.7%) participated, although 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 ages 19−20 years or 36−43 years were omitted (analytic sample N=611). Because participants were recruited as girls at age of 10 years, gender in this study 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 IRB.

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 years 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 (≥2 ACEs).[38]

For BMI, at ages 9−10 and 19−20 years and for local (≤60 miles) participants at follow-up, trained study staff measured height and weight 3 times each per National Health and Nutrition Examination Survey protocols.[39] For distant (>60 miles) participants at follow-up, staff mailed a National Health and Nutrition Examination Survey−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 and self-report values on sociodemographic and psychosocial attributes, but slight differential misclassification by weight was significant ($BMI_{self\text{-}report} = -0.04 \times BMI_{measured}$, $p<0.001$; 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 the household, and number of siblings (all parent-reported at baseline).

Statistical Analysis

Analyses took place in 2020−2023. 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 *X* ACE interaction term for $BMI_{age\ 19-20\ years}$ was significant) were stratified by race to calculate respective ACE −BMI associations. When the interaction was not significant ($p\geq0.15$), respective ACE−BMI associations for the overall sample were calculated in linear models, adjusting for race as warranted. Statistical significance was evaluated against α=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 because more formal statistical testing was precluded. Analyses utilized Stata15 SE (College Station, TX).

A substantial portion (13.4%) (Table 1) at baseline met obesity criteria (BMI for age and sex ≥95th 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 <95th BMI percentile at ages 9−10 years. A complete case-analysis approach was taken, and each model's analytical sample sizes are noted (Tables 2 and 3).

RESULTS

Table 1 displays the demographics of the analytical sample (mean age at follow-up=39.5 years, 48.6% Black). At baseline, a higher percentage of Black than White women had lower household income, had lower parental education, had more siblings, and lived in single-parent households. Although physical abuse, sexual abuse, and high (≥2) ACE exposure were each endorsed by >20% of the women overall and household substance abuse by 37%, fewer Black than White women endorsed ACE exposures. More Black women also had a BMI >95th percentile at ages 9−10 years 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 ages 19 −20 years (Table 2). Among Black women, endorsing sexual abuse was associated with having a 3.24 kg/m^2 higher BMI (95% CI=0.92, 5.57; p=0.02 for chi-square 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 CIs 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 ages 19−20 years ($B_{adjusted}$=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

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]
ACEs (aged <18 years), *n* (%) unless otherwise noted				
Number of ACEs[b], mean (SD) *n* (%) missing[c]	0.82 (0.95)	0.98 (1.00)	0.65 (0.87)	**<0.0001**
Physical abuse *n* (%) missing[c]	143 (23.4%) 8 (1.3%)	91 (29.0%) 2 (0.6%)	52 (17.5%) 6 (2.0%)	**0.002**
Sexual abuse *n* (%) missing[c]	131 (21.4%) 28 (4.6%)	77 (24.5%) 11 (3.5%)	54 (18.2%) 17 (5.7%)	0.09
Substance abuse *n* (%) missing[c]	227 (37.2%) 9 (1.5%)	140 (44.6%)	87 (29.3%) 9 (3.0%)	**<0.001**
≥2 ACEs *n* (%) missing[c]	141 (23.1%) 18 (3.0%)	90 (28.7%) 8 (2.6%)	51 (17.2%) 10 (3.4%)	**0.003**
BMI, *n* (%)				
BMI >95th percentile at ages 9–10 years *n* (%) missing[c]	82 (13.4%) 6 (1.0%)	31 (9.9%) 2 (0.6%)	51 (17.2%) 4 (1.4%)	**0.02**
BMI at ages 19–20 years, mean(SD) *n* (%) missing[c]	25.4 (6.6) 26 (4.3%)	24.2 (5.6) 10 (3.2%)	26.6 (7.4) 16 (5.4%)	**<0.0001**
BMI at ages 36–43 years, mean(SD) *n* (%) missing[c]	31.7 (9.1) 2 (0.3%)	29.3 (8.0) 1 (0.3%)	34.3 (9.6) 1 (0.3%)	**<0.0001**
Baseline sociodemographic characteristics				
Race: Black, *n* (%) *n* (%) missing[c]	297 (48.6%)	—	—	
Current age (years), mean (SD) *n* (%) missing[c]	39.5 (1.3)	39.5 (1.3)	39.5 (1.2)	0.88
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[c]	27 (4.4%)	10 (3.2%)	17 (5.7%)	
Highest parental educational attainment, *n* (%)				**<0.001**
High school or lower	131 (21.4%)	61 (19.4%)	70 (23.6%)	
Some college	281 (46.0%)	116 (36.9%)	165 (55.6%)	
College graduate and higher	198 (32.4%)	137 (43.6%)	61 (20.5%)	
n (%) missing[c]	1 (0.2%)		1 (0.3%)	
Single parent household *n* (%) *n* (%) missing[c]	196 (32.1%)	62 (19.8%)	134 (45.1%)	**<0.001**
Number of siblings, mean (SD) *n* (%) missing[c]	1.31 (1.12)	1.14 (0.97)	1.48 (1.24)	**0.0001**

Note: Boldface indicates statistical significance ($p<0.05$).
[a]Percentages and *p*-values were calculated including missing.
[b]Calculated from nonmissing ACE data; some participants did not provide a response (*yes*/*no*) to all ACE items.
[c]Left blank if no missing data for this variable in the analytical sample.
ACE, adverse childhood experience; K, thousand.

their 95% CIs suggested very small (~0), if any, associations at late adolescence and midlife and in sensitivity analyses.

For all other ACE exposures, race-related modification of ACE–BMI relationships was not observed ($p>0.15$ for all chi-square 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 ages 19–20 years and suggested but more imprecise at midlife. Although after women with high baseline childhood obesity risk were removed, a significant, positive association was observed for BMI at ages 19–20 years ($B_{adjusted}$=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 ACE exposure, significant, positive adjusted associations were observed for BMI at ages 19–20 years ($B_{adjusted}$=1.47 kg/m^2, 95% CI=0.13, 2.80) and were

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

Life stage	Unadjusted	Adjusted[a]	Sensitivity analysis[a,b]
Late adolescence (ages 19–20 years)[c]	(*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 (Ages 36–43 years)[d]	(*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)

Note: Boldface indicates statistical significance ($p<0.05$).
[a]Adjusted for household income, highest parent education, number of parents in the household, and number of siblings at baseline.
[b]Sensitivity analyses: restricted analyses to girls with BMI for age below the 95th percentile at ages 9–10 years.
[c]At ages 19–20 years, race *X* sexual abuse chi-square term $p=0.02$ in fully adjusted models.
[d]At ages 36–43 years, race *X* sexual abuse chi-square term $p=0.25$ in fully adjusted models.

suggested at midlife ($B_{adjusted}$=1.52 kg/m^2, 95% CI= −0.25, 3.29) with CIs 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 (ages 19–20 years: $B_{adjusted}$=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). Although adolescent high ACE 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 to generally diminish from late adolescence to midlife (given overlapping 95% CIs), except for household substance abuse that appeared to change direction. Association estimates were null/

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

Life stage	Unadjusted	Adjusted[a]	Sensitivity analysis[a,b]
Late adolescence (ages 19–20 years)			
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)
Sexual abuse[c]	**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 (ages 36–43 years)			
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)
Sexual abuse[c]	0.73 (−1.05, 2.51) (*n*=582)	1.04 (−0.73, 2.81) (*n*=555)	1.25 (−0.40, 2.90) (*n*=480)

Note: Boldface indicates statistical significance ($p<0.05$).
[a]Adjusted for race, household income, highest parent education, number of parents in the household, and number of siblings at baseline.
[b]Sensitivity analyses: restricted analyses to girls with BMI for age below the 95th percentile at ages 9–10 years.
[c]Please note that associations for sexual abuse and BMI for the overall cohort are presented here as reference only and are not fully accurate given the existence of statistically significant differences between sexual abuse and BMI associations of Black versus White women precluding their actual combination.
ACE, adverse childhood experience.

smallest for household substance abuse (least severe) and greatest for high ACE exposure (most severe).

DISCUSSION

This study examined BMI from ages 19−20 years to midlife in a Black−White cohort by ACE type to advance the 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) ACE exposure and physical abuse were positively associated with BMI. Associations of larger magnitudes were observed with more severe and proximal exposures. Although 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 the majority White samples and with others reporting more pronounced ACE−BMI associations in contexts of more severe ACEs—for example, physical abuse[9,16,37,44,46,47] and high ACEs exposures.[9,10,45] In addition, the notion of greater potency,[8,10,31] 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 childhood and mid-childhood with BMI or incident Class III obesity at midlife, although race differences were observed, their nature—significant positive associations were observed for physical abuse/physical plus sexual abuse combined, only among Whites[31,46]—contrasted the findings in this study. 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 studies mentioned earlier 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 because interactions are scale dependent), 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] Every day, this manifests as increased stress[27] in an already psychologically and physiologically weathered group.[21−24,26] Moreover, for Black women, 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 the drivers mentioned earlier toward 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 such as easier access to fast food and convenience stores,[27] heightened activation of the hypothalamic−pituitary−adrenal 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.

Finally, a greater prevalence of baseline obesity was present among Black girls than among White girls, as were higher mean BMIs among Black than among White women at late adolescence and midlife. Sensitivity analyses proved important because ∼13% of the analytical sample met the Centers for Disease Control and Prevention 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 because girls with early excess weight would be at increased risk for earlier pubertal onset,[56] which could then increase the 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 although 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 than in Black women. Combined with selection bias of a more White sample with lower SES than the general population for these analyses, 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, although 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 a more thorough examination of Black–White modification of ACE–BMI relationships. Residual confounding is also possible because similar measures of SES may not foster equivalent health contexts and confer similar degrees of health protection for Black and White populations.[35] In addition, only a single interaction was significant at the α=0.05 level (p=0.02), suggesting the utility of more studies replicating these findings. Finally, other ACE–BMI-related work has begun to examine the role of moderators such as income[63] and mediators, including mental health,[6,8,33,47] psychological attributes,[37] identity,[49–51] and unique costressors (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 the 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 combating 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 multifacetedness of ACE–BMI relations.

ACKNOWLEDGMENTS

We recognize the National Heart, Lung, and Blood Institute's Growth and Health Study staff, past and present, for their talents and dedication, without which the study and these analyses would not be possible. We also thank the Nutrition Policy Institute, which provided consultation and support with historical study data. In addition, we express immense gratitude to Wendy Hartogensis, for her generous statistical expertise and consult. Most of all, we thank the incredible NGHS participants for their time and efforts over the years.

Research reported in this publication was supported by the Eunice Kennedy Shriver National Institute of Child Health and Human Development grant "Race, stress and dysregulated eating: Maternal to child transmission of obesity" ([R01HD073568]; BAL and ESE, principal investigators [PIs]); the National Heart, Lung, and Blood Institute grant "Neighborhood Environments and Intergenerational Transmission of Cardiovascular Health" ([R56HL141878]; MSM, PI); and the National Institute on Aging grants "Early Life Adversity, Cumulative Life Stress, Race, and Cellular Ageing in Midlife Women and Offspring" ([R56AG059677 and R01AG059677]; both ESE and BAL, PIs). The participation of DTC was supported by the University of California San Francisco Osher Center research training fellowship program (National Center for Complementary and Integrative Health [T32AT003997]; F.M. Hecht and S. R. Adler, PIs).

No financial disclosures were reported by the authors of this paper.

CREDIT AUTHOR STATEMENT

Dorothy T. Chiu: Conceptualization, Methodology, Formal analysis, Data curation, Writing — original draft, Writing — review & editing. Erika M. Brown: Conceptualization, Methodology, Data curation, Writing — review & editing. A. Janet Tomiyama: Supervision, Writing — review & editing. Kristy E. Brownell: Project administration, Resources, Writing — review & editing. Barbara Abrams: Supervision, Writing — review & editing. Mahasin S. Mujahid: Supervision, Writing — review & editing, Funding acquisition. Elissa S. Epel: Supervision, Writing — review & editing, Funding acquisition. Barbara A. Laraia: Conceptualization, Supervision, Writing — review & editing, Funding acquisition, Resources.

REFERENCES

1. Risk and protective factors. CDC National Center for Injury Prevention and Control - Division of Violence Prevention. https://www.cdc.gov/violenceprevention/aces/riskprotectivefactors.html. Published 2021. Accessed May 21, 2021.
2. Sethi D, Bellis M, Hughes K, Gilbert R, Mitis F, Galea G. *European Report on Preventing Child Maltreatment*. Geneva, Switzerland: World Health Organization; Published 2013. https://apps.who.int/iris/handle/10665/326375.
3. Hughes K, Bellis MA, Hardcastle KA, et al. The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *Lancet Public Heal.* 2017;2(8):e356–e366. https://doi.org/10.1016/S2468-2667(17)30118-4.
4. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The adverse childhood experiences (ACE) study. *Am J Prev Med.* 1998;14(4):245–258. https://doi.org/10.1016/s0749-3797(98)00017-8.
5. Tomiyama AJ. Stress and obesity. *Annu Rev Psychol.* 2019;70:703–718. https://doi.org/10.1146/annurev-psych-010418-102936.
6. Wiss DA, Brewerton TD. Adverse childhood experiences and adult obesity: a systematic review of plausible mechanisms and meta-analysis of cross-sectional studies. *Physiol Behav.* 2020;223:112964. https://doi.org/10.1016/j.physbeh.2020.112964.
7. Hemmingsson E, Johansson K, Reynisdottir S. Effects of childhood abuse on adult obesity: a systematic review and meta-analysis. *Obes Rev.* 2014;15(11):882–893. https://doi.org/10.1111/obr.12216.
8. Boynton-Jarrett R, Rosenberg L, Palmer JR, Boggs DA, Wise LA. Child and adolescent abuse in relation to obesity in adulthood: the black women's health study. *Pediatrics.* 2012;130(2):245–253. https://doi.org/10.1542/peds.2011-1554.
9. Li L, Pinto Pereira SM, Power C. Childhood maltreatment and biomarkers for cardiometabolic disease in mid-adulthood in a prospective British birth cohort: associations and potential explanations [published correction appears in *BMJ Open*. 2019;9:e024079corr1]. *BMJ Open.* 2019;9(3):e024079. https://doi.org/10.1136/bmjopen-2018-024079.
10. Schulze LN, Van der Auwera S, Janowitz D, et al. The impact of childhood trauma and depressive symptoms on body mass index. *Glob Psychiatry.* 2019;2(1):97–105. https://doi.org/10.2478/gp-2019-0008.
11. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav.* 2012;106(1):29–39. https://doi.org/10.1016/j.physbeh.2011.08.019.
12. Ortiz R, Kluwe B, Lazarus S, Teruel MN, Joseph JJ. Cortisol and cardiometabolic disease: a target for advancing health equity. *Trends Endocrinol Metab.* 2022;33(11):786–797. https://doi.org/10.1016/j.tem.2022.08.002.
13. Negriff S. ACEs are not equal: examining the relative impact of household dysfunction versus childhood maltreatment on mental health in adolescence. *Soc Sci Med.* 2020;245:112696. https://doi.org/10.1016/j.socscimed.2019.112696.
14. Stanford FC, Lee M, Hur C. Race, Ethnicity, sex, and obesity: is it time to personalize the scale? *Mayo Clin Proc.* 2019;94(2):362–363. https://doi.org/10.1016/j.mayocp.2018.10.014.
15. Papalia T, Greco R, Lofaro D, Maestripieri S, Mancuso D, Bonofiglio R. Impact of continuous value of body mass index on graft loss in overweight patients. *Transplant Proc.* 2010;42(4):1074–1076. https://doi.org/10.1016/j.transproceed.2010.03.049.
16. Francis MM, Nikulina V, Widom CS. A prospective examination of the mechanisms linking childhood physical abuse to body mass index in adulthood. *Child Maltreat.* 2015;20(3):203–213. https://doi.org/10.1177/1077559514568892.
17. Tomiyama AJ, Hunger JM, Nguyen-Cuu J, Wells C. Misclassification of cardiometabolic health when using body mass index categories in NHANES 2005–2012. *Int J Obes (Lond).* 2016;40(5):883–886. https://doi.org/10.1038/ijo.2016.17.
18. Heymsfield SB, Peterson CM, Thomas DM, Heo M, Schuna JM Jr.. Why are there race/ethnic differences in adult body mass index-adiposity relationships? A quantitative critical review. *Obes Rev.* 2016;17(3):262–275. https://doi.org/10.1111/obr.12358.
19. Jolliffe D. Continuous and robust measures of the overweight epidemic: 1971–2000. *Demography.* 2004;41(2):303–314. https://doi.org/10.1353/dem.2004.0015.
20. Lovasi GS, Underhill LJ, Jack D, Richards C, Weiss C, Rundle A. At odds: concerns raised by using odds ratios for continuous or common dichotomous outcomes in research on physical activity and obesity. *Open Epidemiol J.* 2012;5:13–17. https://doi.org/10.2174/1874297101205010013.
21. Farmer MM, Ferraro KF. Are racial disparities in health conditional on socioeconomic status? *Soc Sci Med.* 2005;60(1):191–204. https://doi.org/10.1016/j.socscimed.2004.04.026.
22. Krueger PM, Chang VW. Being poor and coping with stress: health behaviors and the risk of death. *Am J Public Health.* 2008;98(5):889–896. https://doi.org/10.2105/AJPH.2007.114454.
23. Morales JR, Guerra NG. Effects of multiple context and cumulative stress on urban children's adjustment in elementary school. *Child Dev.* 2006;77(4):907–923. https://doi.org/10.1111/j.1467-8624.2006.00910.x.
24. Geronimus AT, Pearson JA, Linnenbringer E, et al. Weathering in Detroit: place, race, ethnicity, and poverty as conceptually fluctuating social constructs shaping variation in allostatic load. *Milbank Q.* 2020;98(4):1171–1218. https://doi.org/10.1111/1468-0009.12484.
25. Williams DR. Race and health: basic questions, emerging directions. *Ann Epidemiol.* 1997;7(5):322–333. https://doi.org/10.1016/s1047-2797(97)00051-3.
26. Williams DR, Lawrence JA, Davis BA. Racism and health: evidence and needed research. *Annu Rev Public Health.* 2019;40:105–125. https://doi.org/10.1146/annurev-publhealth-040218-043750.
27. Jackson JS, Knight KM, Rafferty JA. Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. *Am J Public Health.* 2010;100(5):933–939. https://doi.org/10.2105/AJPH.2008.143446.
28. Woods-Giscombe CL, Lobel M, Zimmer C, et al. Use of food to cope with culturally relevant stressful life events is associated with body mass index in African American women. *Nurs Res.* 2021;70(5S Suppl 1):S53–S62. https://doi.org/10.1097/NNR.0000000000000532.
29. Miller AL, Gearhardt AN, Retzloff L, Sturza J, Kaciroti N, Lumeng JC. Early childhood stress and child age predict longitudinal increases in obesogenic eating among low-income children. *Acad Pediatr.* 2018;18(6):685–691. https://doi.org/10.1016/j.acap.2018.01.007.
30. Mezuk B, Abdou CM, Hudson D, et al. "White Box" epidemiology and the social neuroscience of health behaviors: the environmental affordances model. *Soc Ment Health.* 2013;3(2):10.1177/2156869313480892. https://doi.org/10.1177/2156869313480892.

31. Richardson AS, Dietz WH, Gordon-Larsen P. The association between childhood sexual and physical abuse with incident adult severe obesity across 13 years of the National Longitudinal Study of Adolescent Health. *Pediatr Obes.* 2014;9(5):351–361. https://doi.org/10.1111/j.2047-6310.2013.00196.x.
32. Rehkopf DH, Headen I, Hubbard A, et al. Adverse childhood experiences and later life adult obesity and smoking in the United States. *Ann Epidemiol.* 2016;26(7):488–492.e5. https://doi.org/10.1016/j.annepidem.2016.06.003.
33. Vásquez E, Udo T, Corsino L, Shaw BA. Racial and ethnic disparities in the association between adverse childhood experience, perceived discrimination and body mass index in a national sample of U.S. Older adults. *J Nutr Gerontol Geriatr.* 2019;38(1):6–17. https://doi.org/10.1080/21551197.2019.1572569.
34. Lewis JA, Williams MG, Peppers EJ, Gadson CA. Applying intersectionality to explore the relations between gendered racism and health among black women. *J Couns Psychol.* 2017;64(5):475–486. https://doi.org/10.1037/cou0000231.
35. Tomiyama AJ, Puterman E, Epel ES, Rehkopf DH, Laraia BA. Chronic psychological stress and racial disparities in body mass index change between Black and White girls aged 10−19. *Ann Behav Med.* 2013;45(1):3–12. https://doi.org/10.1007/s12160-012-9398-x.
36. The NHLBI Growth and Health Study Research Group. Obesity and cardiovascular disease risk factors in Black and White girls: the NHLBI Growth and Health Study. *Am J Public Health.* 1992;82(12):1613–1620. https://doi.org/10.2105/AJPH.82.12.1613.
37. Midei AJ, Matthews KA, Bromberger JT. Childhood abuse is associated with adiposity in midlife women: possible pathways through trait anger and reproductive hormones. *Psychosom Med.* 2010;72(2):215–223. https://doi.org/10.1097/PSY.0b013e3181cb5c24.
38. Purswani P, Marsicek SM, Amankwah EK. Association between cumulative exposure to adverse childhood experiences and childhood obesity. *PLoS One.* 2020;15(9):e0239940. https://doi.org/10.1371/journal.pone.0239940.
39. National Health and Nutrition Examination Survey (NHANES) anthropometry procedures manual. Atlanta, Georgia: Centers for Disease Control and Prevention; Published 2007. https://www.cdc.gov/nchs/data/nhanes/nhanes_07_08/manual_an.pdf.
40. Thiese MS, Ronna B, Ott U. P value interpretations and considerations. *J Thorac Dis.* 2016;8(9):E928–E931. https://doi.org/10.21037/jtd.2016.08.16.
41. 2−20 years: girls body mass index-for-age percentiles. Atlanta, Georgia: Centers for Disease Control and Prevention; Published 2000. http://www.cdc.gov/growthcharts/data/set1clinical/cj41l024.pdf.
42. Gardner DSL, Hosking J, Metcalf BS, Jeffery AN, Voss LD, Wilkin TJ. Contribution of early weight gain to childhood overweight and metabolic health: a longitudinal study (EarlyBird 36). *Pediatrics.* 2009;123(1):e67–e73. https://doi.org/10.1542/peds.2008-1292.
43. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. *Obes Rev.* 2016;17(2):95–107. https://doi.org/10.1111/obr.12334.
44. Gooding HC, Milliren C, Austin SB, Sheridan MA, McLaughlin KA. Exposure to violence in childhood is associated with higher body mass index in adolescence. *Child Abuse Negl.* 2015;50:151–158. https://doi.org/10.1016/j.chiabu.2015.08.005.
45. Pando C, Santaularia NJ, Erickson D, Lust K, Mason SM. Classes of lifetime adversities among emerging adult women by race/ethnicity and their associations with weight status in the United States. *Prev Med.* 2022;154:106880. https://doi.org/10.1016/j.ypmed.2021.106880.
46. Bentley T, Widom CS. A 30-year follow-up of the effects of child abuse and neglect on obesity in adulthood. *Obesity (Silver Spring).* 2009;17(10):1900–1905. https://doi.org/10.1038/oby.2009.160.
47. Sacks RM, Takemoto E, Andrea S, Dieckmann NF, Bauer KW, Boone-Heinonen J. Childhood maltreatment and BMI trajectory: the mediating role of depression. *Am J Prev Med.* 2017;53(5):625–633. https://doi.org/10.1016/j.amepre.2017.07.007.
48. Tillman S, Bryant-davis T, Smith K, Marks A. Shattering silence: exploring barriers to disclosure for African American sexual assault survivors. *Trauma Violence Abuse.* 2010;11(2):59–70. https://doi.org/10.1177/1524838010363717.
49. Bey GS, Ulbricht CM, Person SD. Theories for race and gender differences in management of social identity-related stressors: a systematic review. *J Racial Ethn Health Disparities.* 2019;6(1):117–132. https://doi.org/10.1007/s40615-018-0507-9.
50. Harrington EF, Crowther JH, Shipherd JC. Trauma, binge eating, and the "strong black woman.". *J Consult Clin Psychol.* 2010;78(4):469–479. https://doi.org/10.1037/a0019174.
51. Bey GS, Waring ME, Jesdale BM, Person SD. Gendered race modification of the association between chronic stress and depression among black and white U.S. adults. *Am J Orthopsychiatry.* 2018;88(2):151–160. https://doi.org/10.1037/ort0000301.
52. Hayman LW, McIntyre RB, Abbey A. The bad taste of social ostracism: the effects of exclusion on the eating behaviors of African-American women. *Psychol Health.* 2015;30(5):518–533. https://doi.org/10.1080/08870446.2014.983923.
53. Ciciurkaite G, Perry BL. Body weight, perceived weight stigma and mental health among women at the intersection of race/ethnicity and socioeconomic status: insights from the modified labelling approach. *Sociol Health Illn.* 2018;40(1):18–37. https://doi.org/10.1111/1467-9566.12619.
54. Keirns NG, Tsotsoros CE, Addante S, et al. Adverse childhood experiences associated with greater internalization of weight stigma in women with excess weight. *Obesities.* 2021;1(1):49–57. https://doi.org/10.3390/obesities1010005.
55. Mason SM, Flint AJ, Field AE, Austin SB, Rich-Edwards JW. Abuse victimization in childhood or adolescence and risk of food addiction in adult women. *Obesity (Silver Spring).* 2013;21(12):E775–E781. https://doi.org/10.1002/oby.20500.
56. Li W, Liu Q, Deng X, Chen Y, Liu S, Story M. Association between obesity and puberty timing: a systematic review and meta-analysis. *Int J Environ Res Public Health.* 2017;14(10):1266. https://doi.org/10.3390/ijerph14101266.
57. Chen FR, Rothman EF, Jaffee SR. Early puberty, friendship group characteristics, and dating abuse in US girls. *Pediatrics.* 2017;139(6):e20162847. https://doi.org/10.1542/peds.2016-2847.
58. Haynie DL, Piquero AR. Pubertal development and physical victimization in adolescence. *J Res Crime Delinq.* 2006;43(1):3–35. https://doi.org/10.1177/00224278052800.
59. Hamlat EJ, Laraia B, Bleil ME, et al. Effects of early life adversity on pubertal timing and tempo in black and white girls: the national growth and health study. *Psychosom Med.* 2022;84(3):297–305. https://doi.org/10.1097/PSY.0000000000001048.
60. Reuben A, Moffitt TE, Caspi A, et al. Lest we forget: comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. *J Child Psychol Psychiatry.* 2016;57(10):1103–1112. https://doi.org/10.1111/jcpp.12621.
61. Black women and sexual assault. Washington, DC: The National Center on Violence Against Women in the Black Community; Published 2018. https://ujimacommunity.org/wp-content/uploads/2018-12/Ujima-Womens-Violence-Stats-v7.4-1.pdf.
62. Mason SM, Maclehose RF, Katz-Wise SL, et al. Childhood abuse victimization, stress-related eating, and weight status in young women. *Ann Epidemiol.* 2015;25(10):760–766.e2. https://doi.org/10.1016/j.annepidem.2015.06.081.
63. Islam SJ, Kim JH, Joseph E, et al. Association between early trauma and ideal cardiovascular health among black Americans. *Circ Cardiovasc Qual Outcomes.* 2021;14:955–963. https://doi.org/10.1161/CIRCOUTCOMES.121.007904.

64. Ottley PG, Barranco LS, Freire KE, et al. Preventing childhood adversity through economic support and social norm strategies. *Am J Prev Med.* 2022;62(6 Suppl 1):S16–S23. https://doi.org/10.1016/j.amepre.2021.11.016.

65. Golden SH, Joseph JJ, Hill-Briggs F. Casting a health equity lens on endocrinology and diabetes. *J Clin Endocrinol Metab.* 2021;106(4):e1909–e1916. https://doi.org/10.1210/clinem/dgaa938.