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Recommended Citation

Curtis, D., Fuller-Rowell, T. Doan, S.N. Zgierska, A. & Ryff, C. (2016). Racial and socioeconomic disparities in body mass index among college students: Understanding the role of early life adversity. *Journal of Behavioral Medicine*, 39, 866-875.

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Racial and socioeconomic disparities in body mass index among college students: understanding the role of early life adversity

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Received: December 23, 2015 / Accepted: June 4, 2016 / Published online: June 11, 2016
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Abstract The role of early life adversity (ELA) in the development of health disparities has not received adequate attention. The current study examined differential exposure and differential vulnerability to ELA as explanations for socioeconomic and racial disparities in body mass index (BMI). Data were derived from a sample of 150 college students ($M_{age} = 18.8$, $SD = 1.0$; 45 % African American; 55 % European American) who reported on parents' education and income as well as on exposure to 21 early adverse experiences. Body measurements were directly assessed to determine BMI. In adjusted models, African American students had higher BMI than European Americans. Similarly, background socioeconomic status was inversely associated with BMI. Significant mediation of group disparities through the pathway of ELA was detected, attenuating disparities by approximately 40 %. Furthermore, ELA was more strongly associated with BMI for African Americans than for European Americans. Efforts to achieve health equity may need to more fully consider early adversity.

Keywords Health disparities · Early life adversity · Body mass index · Differential exposure · Differential vulnerability · College students

Introduction

Racial and socioeconomic disparities in adolescent obesity are pronounced and appear to have long-term implications for health and well-being. In 2010, approximately 12 % of White/European American (EA) adolescents and 21 % of Black/African American (AA) adolescents were obese (Ogden et al., 2012). Similarly, 8 % of adolescents whose parents had at least a bachelor's degree were obese compared to nearly 25 % of adolescents whose parents had a high school diploma or less, a disparity which has grown in recent years (Frederick et al., 2014).

With established associations between adolescent BMI and subsequent morbidity and mortality (Juonala et al., 2011; Tirosch et al., 2011; Wong et al., 2002), disparities in BMI during adolescence are a likely contributing factor to racial and socioeconomic health disparities across the life span. Late adolescence/young adulthood is also a key period for understanding the emergence of weight problems and obesity because body weight is known to increase during this period, and health behavior patterns, which are likely to persist across the life span, are being formed (Laska et al., 2012; Nelson et al., 2008). Therefore, understanding early life risk factors for obesity may offer critically important insights into addressing health disparities.

Adverse experiences during childhood and adolescence represent one such risk factor as early adversity has been consistently associated with obesity risk and morbidity outcomes across the life span (Felitti et al., 1998; Shonkoff

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et al., 2012). However, few studies have examined the degree to which early life adversity (ELA) accounts for racial or socioeconomic disparities in obesity and other physical health outcomes (differential exposure hypothesis) (Fuller-Rowell et al., 2015; Lee & Hicken, 2013; Wells et al., 2010). Similarly, limited research has examined the extent to which individuals from marginalized racial/ethnic groups, or disadvantaged socioeconomic backgrounds, are more susceptible to the adverse effects of ELA (differential vulnerability hypothesis). Focusing on the association between ELA and body mass index (BMI) in a young adult sample of Black/African American (AA) and White/European American (EA) college students, the current study sought to address these gaps in current knowledge.

The mediating role of ELA

Social conditions in the US are structured such that adverse experiences are unequally distributed among youth, with racial/ethnic minority and socioeconomically disadvantaged children being disproportionately exposed (Thoits, 2010; Wells et al., 2010), and thus at increased risk for the many adverse consequences (Felitti et al., 1998; Shonkoff et al., 2012). For example, AAs and individuals of low SES consistently score higher on measures of exposure to early life adverse events or high-risk contexts, such as living in a poor neighborhood, being exposed to family or neighborhood violence, having a parent with mental health problems, and experiencing childhood maltreatment (Chang, 2006; Felitti et al., 1998; Slopen et al., 2010; Thoits, 2010; Wells et al., 2010). In turn, such experiences and environments have been associated with obesity risk (Carroll-Scott et al., 2013; Danese & Tan, 2014; Garasky et al., 2009; Lohman et al., 2009; Nicholson & Browning, 2012). Despite existing evidence linking racial minority status and low SES to ELA, and ELA to poorer physical health, few studies have tested the mediating role of ELA in the development of group health disparities. Such tests are needed to demonstrate the degree to which early experiences may lead to health inequities. Notably, ELA has, in fact, been shown to mediate the effects of poverty on BMI trajectories (Wells et al., 2010) and Black-White differences in obesity among females (Lee & Hicken, 2013) in adolescent samples.

Higher education is often seen as a way to escape disadvantaged social circumstances and their negative sequelae. Students from less advantaged backgrounds demonstrate resiliency by seeking higher education degrees (Schoon et al., 2004). Interestingly, however, resiliency as indicated by academic success, may not extend to health status (Nelson et al., 2007). In fact, evidence suggests that achievement in the face of adversity may even take a biological toll, contributing to racial and socioeconomic

disparities (Chen et al., 2014). Thus, the presence of socioeconomic and racial disparities in obesity among college students, and the role of ELA in accounting for these disparities are understudied topics relevant to forming policy and intervention.

Differential vulnerability

In addition to differences in exposure, vulnerability to the sequelae of ELA may also vary according to one's racial/ethnic group membership or socioeconomic position. In particular, early adverse experiences and disadvantaged social status may have multiplicative health-related effects (Slopen et al., 2010). Consistent with this idea, and especially relevant to high-achieving students, the "weathering hypothesis" postulates that when faced with chronic stressors and social exclusion, high-effort, active coping may be detrimental to physical health (Geronimus, 2001; Levine & Crimmins, 2014). Accordingly, emerging evidence suggests that AA youth who attain high academic competency or matriculate into college are more vulnerable to the effects of early adversity on physiologic dysregulation than their lower achieving AA peers (Brody et al., 2013; Chen et al., 2014). These findings suggest that the stress associated with making it to college for those from high adversity circumstances may carry physiological costs despite other benefits. Whether youth from disadvantaged socioeconomic backgrounds are also more vulnerable to ELA has not previously been considered; however, as is the case with AAs, youth of low SES are at elevated risk of being exposed to ELA (Thoits, 2010) and of being socially excluded (Fuller-Rowell et al., 2012). Empirical investigation is therefore needed to consider whether background SES moderates the association between ELA and health. Additionally, although some evidence supports AAs' increased vulnerability to ELA (Slopen et al., 2010), to our knowledge, no studies have considered this hypothesis for BMI. Because of established group disparities in obesity as well as known associations between ELA and obesity, BMI is an important extension to prior research.

Hypotheses

The current study adds to existing literature by testing ELA as a mediator of racial and socioeconomic disparities in BMI, and by examining the moderating role of race and background SES on the association between ELA and BMI. We hypothesize that expected racial and socioeconomic differences in BMI among first and second year college students will be mediated by their past exposure to ELA. Further, we predict that ELA will be more strongly

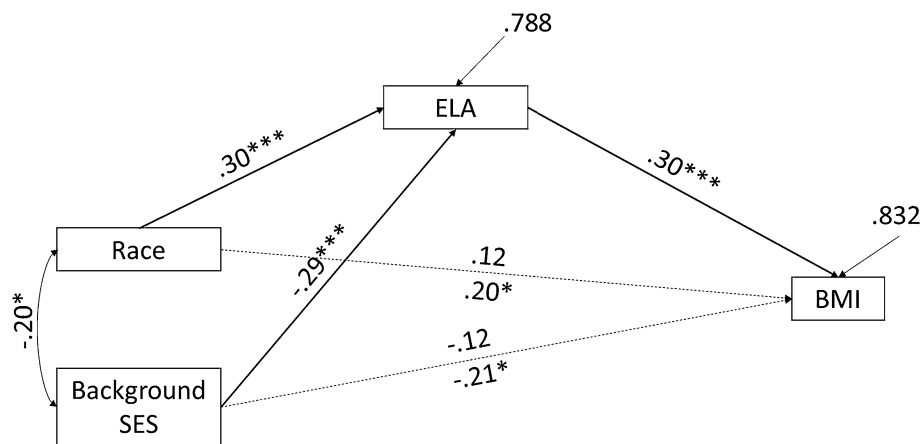


Fig. 1 Fitted mediation model ($N = 150$). *Note.* Solid lines indicate significant paths. ELA and BMI are abbreviations for early life adversity and body mass index. Race is coded as European American = 0, African American = 1. Standardized coefficients

and residual variances are presented. In order to demonstrate mediation, parameter estimates without ELA as a covariate are included below arrows. Controls include sex, age, heavy alcohol use, and current cigarette smoker. * $p \leq .05$; *** $p \leq .001$

associated with BMI for AAs and for those from less advantaged SES backgrounds.

Method

Data and sample

The data were derived from the College Student Health Study, an ongoing study of college students at a predominantly white university (3 % African American, 4 % Asian, 4 % Hispanic/Latino, 1 % American Indian) in the Midwestern United States. In 2012, 235 AA students and an equally sized stratified random sample of EA students were invited to participate. First-generation EA college students (neither parent graduated from college) were over-sampled to ensure that race and parent education would not be confounded. A total of 150 first and second year college students (mean age = 18.8, SD = 1.0; 45 % AA, 57 % female) participated in the study. The protocol was approved by the University's institutional review board and informed consent was obtained from all participants.

Measures

Demographic and control variables

Race was collected from university records and confirmed via self-report. Students also reported their sex, age, whether they currently smoke cigarettes (1 = yes, 0 = no), and the number of alcoholic drinks they consume "on a typical day when they are drinking". A dichotomous variable indicating heavy alcohol use was included in analyses (≥ 5

alcoholic drinks = 1, <5 drinks = 0). Participants reported each parent's level of education on a seven-point scale ranging from 8th grade or less (coded as 1) to graduate degree (coded as 7), and parent education was coded as the mean education level for students from two parent families or the education level of a single parent. Income-to-needs ratio was calculated from youth reports of their parents' combined household income from the year prior to the baseline assessment, measured on a scale with 28 possible categories (1 = <\$5000 to 28 = >\$200,000), and adjusted for household size, using 2011 US Federal poverty guidelines. A composite representing background socioeconomic status was scored as the mean of standardized parent education and household income-to-needs variables.

Early life adversity

The number of early life adversities experienced was measured using a 21-item inventory that was adapted for the current study from previous research (Felitti et al., 1998; Finkelhor et al., 2013). Twelve of the items were based on the Adverse Childhood Experiences Questionnaire (Felitti et al., 1998). Additional childhood risk factors with known influences on health were measured, including crime and violence exposure, family environment dynamics, and illness in the family (Finkelhor et al., 2013; Garasky et al., 2009). The ELA inventory is not an exhaustive list of potential types of adversity, rather it is meant to index broadly one's exposure to early stressful events. Items measuring parental educational attainment and employment status were not included to avoid conceptual overlap with background socioeconomic status. Items were coded to indicate the occurrence of each risk factor

Table 1 Descriptives for African American (AA; $n = 68$) and European American (EA; $n = 82$) first and second year college students

Variables	AA	EA	Race difference <i>p</i>
Sex (female)	41 (60.3 %)	43 (52.4 %)	.34
Age (years)	18.90 ± 1.01	18.74 ± .91	.33
Current cigarette smoker	5 (7.4 %)	5 (6.1 %)	.76
Heavy drinker (≥5 drinks)	17 (25.0 %)	41 (50.0 %)	.002
Socioeconomic status	−.18 ± .86	.14 ± .81	.022
Early life adversity	4.49 ± 3.66	2.09 ± 2.62	<.001
Body mass index (kg/m ²)	25.31 ± 5.36	23.54 ± 3.00	.017
Body adiposity index ^a	23.88 ± 7.19	21.44 ± 4.37	.016

Values are expressed as mean ± SD or number of participants (%)

^a Body adiposity index is an estimate of percent body fat, calculated as hip circumference (cm)/height (m)^{1.5} − 18

(1 = yes, 0 = no) and a total scale score, which could range from 0 to 21, was calculated by summing all items ($\alpha = .81$).

Body size

Anthropometric measurements of weight, height, and hip circumference were collected by trained nursing staff. Body mass index (BMI) was calculated as weight (kg) divided by height (m)². Racial differences in BMI are limited because AAs, on average, have less abdominal visceral fat than EAs, which is not captured by the BMI (Carroll et al., 2008). To enhance our confidence in the findings, the body adiposity index (BAI), a direct estimate of percentage of body fat and an alternative method for between-race comparisons (Barreira et al., 2011; Bergman et al., 2011), was also considered as a secondary outcome. BAI was calculated as hip circumference (cm)/height (m)^{1.5} − 18.

Statistical analyses

Univariate descriptive statistics and bivariate correlations were examined to consider unadjusted racial and socioeconomic disparities in key variables. A series of regression analyses were estimated using Mplus 7.11 (Muthén & Muthén, 2013) to test mediation and moderation hypotheses. Missing data were handled using Full Information Maximum Likelihood (FIML) estimation, which utilizes all available data to estimate model parameters and ensures a consistent sample size across models. Of the 150 individuals, 6 had missing data on background SES, 2 had missing data on ELA, 14 had missing data on heavy alcohol use, and all other variables had no missing data.

Initially, racial and socioeconomic differences in ELA and BMI were estimated in a multivariate regression model (Model 1). In addition to race and SES, BMI was adjusted for sex, age, current smoker status, and heavy alcohol use,

and ELA was adjusted for sex. The full mediation model (Model 2) was then tested using path analysis, in which ELA was added as a predictor of BMI. Mediation was tested using the product of coefficients methods, and bias-corrected confidence intervals were estimated using a bootstrapping procedure (Hayes & Scharkow, 2013; MacKinnon et al., 2007). Lastly, to examine whether the association between ELA and BMI is conditioned by sociodemographic factors, ELA was interacted with race and SES and interaction terms were tested in a multiple regression model.

Results

Descriptive statistics for AAs and EAs are shown in Table 1. AA students had lower background SES [$M_{\text{difference}} = .32$, 95 % CI (.05, .60), $p = .023$] and reported having experienced greater ELA [$M_{\text{difference}} = 2.41$ events, 95 % CI (1.35, 3.46), $p < .001$] than EAs. AAs also had higher BMI than did EAs [$M_{\text{difference}} = 1.77$ units, 95 % CI (.32, 3.21), $p = .017$]. Bivariate correlations are presented in Table 2 and were in line with study hypotheses. In particular, background SES was inversely correlated with ELA ($p < .001$) and with BMI ($p = .029$). ELA was also correlated with BMI ($p < .001$). In addition, body adiposity index was strongly correlated with BMI and showed comparable associations to BMI with race, background SES, and ELA. Racial and socioeconomic differences in the likelihood of experiencing each adverse event were also tested. Odds ratios from logistic regression models and bivariate correlations between adverse events and BMI are presented in Table 3. Large racial differences in adverse events were detected for 8 of the 21 items with AAs being more than 3 times as likely to experience each event, after controlling for SES. Moreover, differences by SES were detected for 6 of the 21 items, after controlling for race.

Table 2 Bivariate correlations

	1	2	3	4	5	6	7	8
1. Race	–							
2. Sex	.08	–						
3. Age	.08	–.22**	–					
4. Cigarette smoker	.03	–.03	.22**	–				
5. Heavy drinker	–.22**	–.22*	.10	.23**	–			
6. Socioeconomic status	–.19*	.01	–.20*	.04	.16*	–		
7. Early life adversity	.36***	.08	.16	.21**	–.10	–.32***	–	
8. Body mass index	.21*	.06	.02	–.05	–.06	–.18*	.34***	–
9. Body adiposity index	.21*	.43***	–.01	–.07	–.19*	–.18*	.36***	.62***

Race is coded as European American = 0, African American = 1. Sex is coded as Male = 0, Female = 1

* $p < .05$; ** $p < .01$; *** $p < .001$

Table 3 Frequency of event occurrence for Early Life Adversity items for African American (AA) and European American (EA) college students, as well as racial and socioeconomic (SES) differences in likelihood of experiencing and predictive validity of adverse events

Items	AA (%)	EA (%)	Race estimate ^a OR	SES estimate ^b OR	Predictive validity ^c r with BMI
Lived with someone who was alcoholic	14.9	9.9	.85	.32**	.15
Live with someone who used street drugs	20.9	8.6	2.54	.88	–.03
Lived with someone who was depressed	19.4	18.5	.92	.80	.16
Lived with someone who attempted suicide	1.5	6.2	.25	.88	–.10
Parents argued a lot	34.3	22.2	1.45	.50**	.20*
Physical altercations between parents	10.4	4.9	1.90	.67	.02
Reason to worry about parent's safety	25.4	6.2	3.63*	.29**	.18*
You were sexually abused	9.0	3.7	1.40	.39	.31***
Parent verbally abused you	26.9	29.6	.68	.62*	.10
Parent physically threatened you	16.4	7.4	2.88	1.08	.08
Parent physically abused you	31.3	12.3	3.09*	.80	.11
Parent injured you	10.4	6.3	1.77	1.22	.10
Parent spanked you with object	62.7	21.0	6.04***	.44**	.16
Required to take care of younger family member	22.4	16.0	.99	.57	.24**
Immediate family member had illness	37.3	13.6	3.93**	.98	.28**
Immediate family member died	23.9	3.7	7.09**	.66	.21**
Immediate family member was shot, stabbed, or raped	6.0	.0	N/A	.62	.19*
Immediate family member was attacked or robbed	14.9	3.7	4.65*	.67	.20*
Immediate family member went to prison	22.7	3.7	5.06*	.54	.25**
Lived in a high crime neighborhood	23.9	4.9	4.75*	.37**	.22**
House was broken into	14.9	6.2	1.98	.57	.27**

* $p < .05$; ** $p < .01$; *** $p < .001$

^{a,b} Odds ratios are derived from logistic regression models using individual adverse events as outcomes and both race and SES as predictors

^c Predictive validity of early life adversity items demonstrated using bivariate correlations with body mass index (BMI)

Results for racial and socioeconomic disparities in health and mediation hypotheses

First, we considered group differences in ELA and BMI controlling for sex, age, heavy alcohol use, and smoking status. AAs reported experiencing .60 *SD* units greater ELA [$B = 2.01$, $p < .001$, 95 % CI (1.02, 2.99)] than did

EAs. After adjustment for background SES and other covariates, AAs also had .41 *SD* units higher BMI [$B = 1.76$, $p = .014$, 95 % CI (.36, 3.16)] than EAs. Due to the racial disparity in BMI often being found only between females, a race by sex interaction term was also considered but it was not significant ($p = .58$) and was dropped from subsequent models. Adjusting for race and

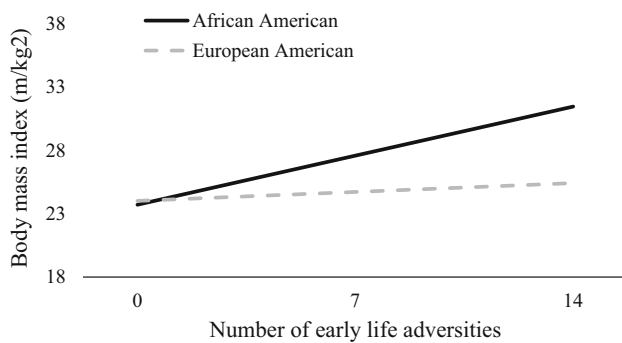


Fig. 2 Estimated body mass index as a function of early life adversity for African American ($n = 68$) and European American ($n = 82$) college students

other covariates, background SES was significantly associated with ELA [$B = -1.15$, $p < .001$, 95 % CI (-1.74 , $-.55$)] and BMI [$B = -1.08$, $p = .018$, 95 % CI (-1.98 , $-.18$)]. The magnitude of this effect suggests that one *SD* unit increase in background SES was associated with a .29 *SD* unit decrease in ELA and a .21 *SD* unit decrease in BMI. Because SES is often differentially associated with health outcomes between AAs and EAs (Fuller-Rowell et al., 2015), a race by background SES interaction was considered but was not found to be significant ($p = .79$).

Next, we tested the extent to which ELA explained (mediated) group differences in BMI, adjusting for all covariates in previous model (see Fig. 1 for fitted path analysis). ELA was significantly associated with BMI [$B = .38$, $p < .001$, 95 % CI (.16, .61)], accounting for 7 % of the variance in BMI above all other covariates. Each one *SD* unit increase in ELA (3.3 events) was associated with a .30 *SD* unit increase in BMI. Estimated racial and socioeconomic differences in BMI were attenuated by 42 and 43 % when accounting for ELA, respectively, and neither race ($p = .16$) nor income-to-needs ($p = .18$) remained significantly associated with BMI. Furthermore, significant indirect effects of race on BMI were detected via the pathway of ELA [$B = .77$, 95 % CI (.20, 1.97)]. Background SES was also associated with BMI through the pathway of ELA [$B = -.44$, 95 % CI (-1.08 , $-.10$)]. In sum, results of the mediational analyses suggest that differential exposure to ELA is one pathway through which group disparities in BMI among college students develop.

Results for moderation hypotheses

We also tested whether the strength of the associations between ELA and BMI varied as a function of race or background SES. Results indicated that race significantly moderated the relation between ELA and BMI [$B = .45$, $p = .033$, 95 % CI (.04, .87)], accounting for an additional

3 % of the variance in BMI. Figure 2 demonstrates that ELA was more strongly associated with BMI for AAs compared to EAs. Specifically, no race difference was found for those who experienced low levels of ELA, yet a large racial gap existed for those coming from backgrounds high in adversity. Background SES was not a statistically significant moderator of the association between ELA and BMI ($p = .71$).

Supplementary analyses

Additional path models were fit using BAI (body adiposity index) rather than BMI to enhance our confidence in the results. Findings from these analyses were generally consistent with those presented for BMI. Significant indirect effects were detected for both race [$B = 1.09$, 95 % CI (.35, 2.52)] and background SES [$B = -.61$, 95 % CI (-1.45 , $-.22$)] as predictors of BAI through the pathway of ELA. Moderation findings were also consistent, such that the association between ELA and BAI was larger for AAs as compared to EAs ($p = .029$), and SES was not a significant moderator ($p = .37$).

Discussion

There is growing evidence for the long-term health consequences of ELA, with recent reports recommending action by Congress, pediatricians, and other health professionals aimed at reducing exposure to or the consequences of such adversity (Block et al., 2013; Garner et al., 2012; Shonkoff et al., 2012). Accordingly, the social patterning of ELA is a public health concern as well as a social justice issue. We demonstrated that even among college students, there are large racial and socioeconomic differences in exposure to early adverse events. Moreover, differences in early adversity account for a substantial portion of racial and socioeconomic disparities in BMI (and BAI). This finding is consistent with existing literature linking various early life adverse contexts to elevated BMI (Carroll-Scott et al., 2013; Suglia et al., 2012) and studies that have documented ELA as an explanation for group disparities in BMI (Lee & Hicken, 2013; Wells et al., 2010). These results extend this work to a college student population, showing that the sequelae of ELA are present among students despite the demonstrated resiliency of college enrollment.

Because weight gain is common during college, students are an important target demographic for obesity research (Vella-Zarb & Elgar, 2009, p. 5). Furthermore, university policies and regulations have influence on—and are able to change—the availability of healthy food choices and

exercise opportunities as well as other health promoting resources (Horacek et al., 2013; Levitsky et al., 2004). College populations are also relatively accessible for psychosocial and behavioral interventions due to shared spaces and course requirements (LaRose et al., 2011). Educational contexts, and colleges in particular, are therefore critical settings in which to consider weight-related interventions (Nelson et al., 2008; Office of the Surgeon General, 2001).

Additionally, because many resources (e.g., campus recreation programs) are widely available to the college students of a particular campus, colleges have the potential to play an important role in achieving health equity by increasing access to health promoting resources. However, psychological stress—often a continuation or even proliferation of ELA (Nurius et al., 2015)—has also been shown to have an important influence on weight change and weight-related behaviors during this time (Freeman & Gil, 2004; Serlachius et al., 2007), such that access to healthy environmental influences may not confer the same benefit to members of social groups experiencing disproportionate psychological stress. Accordingly, in order to reduce group disparities in obesity, effective weight-related interventions and policies, such as university courses on obesity and related health behaviors (Laska et al., 2012), may also benefit from incorporating or being supplemented by psychosocial or economic supports for disadvantaged students (e.g., mentoring programs or mindfulness-based interventions; O'Reilly et al., 2014; Quintiliani & Whiteley, 2016). Research addressing the effectiveness of such multidimensional weight-related interventions is needed.

Our findings also support previous recommendations regarding effective screening of early adversity within the health care system in order to ameliorate its long-term consequences (Brcic et al., 2011; Garner et al., 2012). For example, for patients who have experienced significant adversity, pediatricians and general practitioners may spend additional time probing about support networks and can make recommendations for counseling or other types of supportive interventions when warranted. Physicians at University medical clinics are especially well-positioned to be aware of university resources and programs.

The specific mechanisms linking ELA to obesity are beyond the scope of this paper but are an important topic of research. Current evidence suggests that the pathways of influence appear to be varied, including physiologic and behavioral processes. For example, ELA may alter epigenetic processes that govern inflammatory and stress physiology, leading to further physiologic dysregulation (Miller et al., 2009). In addition, the development of higher level cognitive structures and processes (i.e., executive functioning and self-regulatory skills) may be affected by ELA, which may influence health cumulatively across the life span through changes in health behaviors (Evans et al.,

2012). Moreover, in circumstances of high ELA, opportunities to develop and engage in healthy behaviors may be constrained. For example, high-crime, low-resource neighborhoods appear to be obesogenic due to poor access to healthy food, and fewer opportunities for physical recreation (Carroll-Scott et al., 2013). Indeed, differences in physical activity and television viewing have been shown to account for a portion of estimated black-white disparities in obesity (Nelson et al., 2007). Further research is needed to consider the primary mechanisms through which ELA affects obesity risk among college students, and in turn, whether intervening on these pathways leads to greater health equity.

This is one of the first studies to report on differential vulnerability to early life adversity as a function of race and SES. In particular, we show that the number of reported adverse events were more strongly related to the BMI of AA college students, as compared to EAs. Our results are congruent with past research showing increased stress vulnerability and accelerated aging among socially disadvantaged groups (Geronimus, 2001; Slopen et al., 2010; Tomiyama et al., 2013). For example, ELA has been shown to be associated with the proinflammatory physiology of AA adults, but not for EAs (Slopen et al., 2010). Differential vulnerability to the effects of *psychological stress* on BMI trajectories among AA and EA female adolescents has also been documented (Tomiyama et al., 2013). One potential explanation for AA adolescents' greater vulnerability to ELA may be that fewer resources exist during their youth, including social support systems and positive role models, likely due in part to racial biases as well as higher residence in at-risk communities (Noguera, 2003). As a result, achieving upward mobility in such contexts often requires overcoming several barriers (Hardaway & McLoyd, 2009). AA youth from high adversity backgrounds may therefore be required to engage in particularly high-effort, active coping during their youth in order to meet the demands associated with gaining entrance to a 4-year university. Active coping (e.g., John Henryism) appears to be well suited for attaining educational success, but comes with biological costs when also confronting early adversity (Brody et al., 2013; Chen et al., 2014). This research suggests that, in order to be optimally beneficial to physical health, academic achievement and upward mobility among AAs will require additional support during childhood and adolescence to offset exposure to ELA.

Contrary to our hypothesis, background SES did not moderate the association between ELA and BMI. One explanation for this null finding may be that the sample came from a “highly competitive plus” public university (~50 % acceptance rate), according to Barron's Profiles of American Colleges (2009), with relatively few students from families below the federal poverty line (7 %). The

stigma associated with disadvantaged social status may primarily be experienced by those from very poor households rather than on a SES gradient (Fuller-Rowell et al., 2012), thereby reducing our ability to detect the multiplicative effects of SES and ELA. Alternatively, the lack of moderation findings may be due to unmeasured adversity that is patterned by race to a greater degree than SES. For example, AA's experience discrimination and live in areas of concentrated poverty at particularly high rates as compared to socioeconomically disadvantaged EAs (Chang, 2006; Fuller-Rowell et al., 2011; Kessler et al., 1999; Lichter et al., 2012). Additional research is needed to consider reasons for AA's increased vulnerability to ELA, and whether very poor EA youth also show greater susceptibility.

Several limitations of the current study should be noted. First, although college students are an important and understudied demographic in disparities research, the findings on this particular population are limited by their generalizability to other groups. One important question for future research is whether non-college-bound AA youth are also vulnerable to the weight-related sequelae of ELA. Second, although the sample size was adequate based on the expected magnitude of associations, the number of participants and their recruitment from a single college campus are additional limitations. Larger and more representative samples of college students will increase the generalizability of findings and add additional possibilities to consider important moderators of risk (e.g., resilience factors). Third, because the study utilizes cross-sectional data, causality cannot be inferred and future research should utilize longitudinal data. However, while youth who are obese may experience greater adversity as a result of their weight, the majority of early adverse experiences assessed would likely not be affected by children's body size, including each of the items for which a bivariate association with BMI was detected (see Table 3). A fourth limitation is that retrospective reports of ELA were used, leading to potential recall bias. However, adverse experiences measured are primarily objective events or major stressors that are less susceptible to recall bias (Hardt & Rutter, 2004), and research has shown retrospective and prospective reports of ELA to be comparably predictive of health outcomes (Patten et al., 2014).

In sum, the findings of this study demonstrate that racial and socioeconomic disparities in body size among college students are partly accounted for by differential exposure to ELA, and that, as compared to EAs, AAs are more vulnerable to ELA. These results support recent recommendations to incorporate assessment of early adverse experiences into pediatrician visits and public health initiatives. Both reducing exposure to ELA as well as providing additional social and community supports to

mitigate the deleterious consequences of ELA will likely be important steps to achieving health equity.

Acknowledgments Support for this project was provided by the Robert Wood Johnson Health and Society Scholars Program at the University of Wisconsin—Madison, and by the Clinical and Translational Science Award (CTSA) program, through the NIH National Center for Advancing Translational Sciences (NCATS), Grant UL1TR000427.

Compliance with ethical standards

Conflict of interest David S. Curtis, Thomas E. Fuller-Rowell, Stacey N. Doan, Aleksandra E. Zgierska, and Carol D. Ryff declare that they do not have any conflict of interest.

Human and animal rights and Informed consent All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

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