

Assessing the Role of Health Behaviors, Socioeconomic Status, and Cumulative Stress for Racial/Ethnic Disparities in Obesity

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Objective: This study aimed to examine the explanatory role of health behaviors, socioeconomic position (SEP), and psychosocial stressors on racial/ethnic obesity disparities in a multiethnic and multiracial sample of adults.

Methods: Using data from the Chicago Community Adult Health Study (2001-2003), Oaxaca-Blinder decomposition analysis was conducted to quantify the extent to which health behaviors (fruit and vegetable consumption and physical activity), SEP, and cumulative stressors (e.g., perceived discrimination, financial strain) each explained differences in obesity prevalence in Black, US-born Hispanic, and non-US-born Hispanic compared with non-Hispanic White participants.

Results: SEP and health behaviors did not explain obesity differences between racial/ethnic minorities and White individuals. Having high levels of stress in four or more domains explained 4.46% of the differences between Black and White individuals, whereas having high levels of stress in three domains significantly explained 14.13% of differences between US-born Hispanic and White. Together, the predictors explained less than 20% of differences between any racial/ethnic minority group and White individuals.

Conclusions: Exposure to stressors may play a role in obesity disparities, particularly among Black and US-born Hispanic individuals. Other obesity-related risk factors need to be examined to understand the underlying mechanisms explaining obesity disparities.

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Introduction

The obesity epidemic is a public health issue in the US, disproportionately affecting certain racial/ethnic minority groups (1). Non-Hispanic Black and Hispanic/Latino individuals have a higher prevalence of obesity compared with non-Hispanic White individuals, and they also have higher levels of obesity-related diseases, such as hypertension, coronary heart disease, and stroke (2). As obesity prevalence continues to rise (1), it is likely that these groups will

continue to experience an excessive burden of obesity-related morbidity and mortality.

Obesity-related behaviors, such as diet and physical activity, have been a major focus of obesity prevention and interventions. Individuals who engage in physically active lifestyles and consume recommended amounts of fruits and vegetables display lower adiposity relative to individuals who do not engage in these health behaviors (3,4). Racial/ethnic minorities generally engage in less physical activity and consume fewer

Study Importance

What is already known?

- ▶ Obesity, defined using BMI, disproportionately affects certain racial/ethnic minority groups.
- ▶ Health behaviors (e.g., diet, physical activity) and socioeconomic position are frequently suggested as the reasons for these racial/ethnic disparities, though evidence suggests that other factors, such as psychological stress, may play a role.

What does this study add?

- ▶ Health behaviors and socioeconomic position do not fully explain racial/ethnic differences in adult obesity prevalence among adults in Chicago.
- ▶ After accounting for health behavior and socioeconomic differences, high levels of stress explain an additional 4.46% of differences between Black and White individuals and an additional 14.13% of differences between US-born Hispanic and White individuals.
- ▶ Together, health behaviors, socioeconomic position, and cumulative stress only partially explain racial/ethnic disparities in obesity.

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fruits and vegetables than White individuals, with differences being more pronounced in middle-aged adults compared with older-aged adults (5). Therefore, these lifestyle behaviors may contribute to racial/ethnic disparities in obesity (6). Researchers have suggested that racial/ethnic differences in health behaviors may largely be a function of underlying differences in socioeconomic position (SEP) (7).

Low SEP, commonly measured using resource-based measures of education and income, is an established risk factor for obesity (2). Analysis of data from 23,434 adults in the National Health Interview Survey shows that a large proportion of the association between SEP and obesity is mediated through health behaviors (e.g., diet, physical activity), such that those with higher SEP engage in more healthy behaviors (8,9). Given that racial/ethnic minorities are overrepresented in low-income and -education groups, racial/ethnic differences in obesity may be explained by SEP through differences in social environment and ability to acquire and maintain healthy dietary and exercise behaviors. However, researchers have found that non-Hispanic Black and Hispanic/Latino individuals are more sedentary during their leisure time than are non-Hispanic White individuals across different indicators of social class, such as education, family income, employment status, and marital status (10). While educational attainment and family income are known to influence dietary intake of fruits and vegetables, racial/ethnic differences in fruit and vegetable consumption persist after adjusting for education and income (11,12). Simple adjustment for income, education, and other SEP indicators artificially creates equality across dimensions of race/ethnicity and SEP, which can obscure mechanisms that drive racial/ethnic health inequities (13). Studies have found that racial differences in obesity vary significantly across gradients of socioeconomic indicators, with the greatest disparities at higher levels of SEP (14,15). For instance, in a study of 10,636 adults, researchers found that, in US households with incomes 130% of the federal poverty level or below, the obesity prevalence for non-Hispanic Black is approximately 47% compared with 36% for non-Hispanic White individuals. In contrast, in households with incomes 350% above the federal poverty level, the obesity prevalence for non-Hispanic Black individuals is approximately 49% compared with 31% for non-Hispanic White individuals (14). Bell and colleagues (16) found that racial disparities in obesity between African American ($n=3,950$) and non-Hispanic White ($n=8,777$) adults are largest in the highest income ($\geq \$100,000$) and education (college graduate or more) groups. The persistent racial/ethnic inequalities in obesity across every gradient of the SEP strata suggest that other factors contribute to the persistent racial/ethnic disparities in obesity in the US.

Psychosocial stress is increasingly being recognized as a risk factor for obesity (17). Psychosocial stress may increase the risk of obesity through biological and/or behavioral pathways (2). Psychosocial stress can induce chronic inflammation (18,19) that can alter insulin sensitivity. The repeated activation of inflammation substantially increases secretion of insulin and decreases the release of growth hormone, all of which can lead to accumulation of visceral fat (20). People experiencing psychological distress can experience weight loss because of a loss of appetite (21,22) but can also gain weight through increased food consumption (2,23). Differences in stress response may depend upon a variety of factors, including the type, duration, and severity of stressors, as well as coping behaviors. Nevertheless, psychosocial stress is generally thought to weaken efforts to be physically active and induce the release of appetite hormones to increase food consumption (23). For instance, individuals with high levels of stress tend to consume foods high in fats and sugar as a way to activate brain reward systems and reduce stress responses (24).

Evidence from the US indicates that a range of psychosocial stressors relate to obesity risk across racial/ethnic groups (25). Racial/ethnic minorities report experiencing greater exposure to common stressors (e.g., financial strain, employment stress) concurrent with greater exposure to race-related stressors (e.g., racial discrimination) than their White counterparts (26); this may contribute to their increased obesity risk. However, given that levels of psychosocial stress, on average, are higher among those with lower SEP (27), the association between psychosocial stress and obesity risk may be confounded by SEP. While stress exposure has been found to contribute to racial/ethnic disparities in some health outcomes (such as self-rated health and chronic illness) independently of SEP (26,28), there is insufficient evidence on the extent to which exposure to psychosocial stressors explains racial/ethnic obesity disparities.

The Oaxaca-Blinder decomposition approach allows us to detect how much differences in an outcome would be reduced if one group had the same mean levels of the measured attributes compared with another group (29). The Oaxaca-Blinder decomposition approach can be used to quantify the individual and joint contribution of potentially correlated exposures to health outcomes as well. This method, from economics, is now being applied in health research (30) and used to assess racial/ethnic differences in BMI (31). For instance, in a cross-sectional study of 16,741 men and women, behavioral (e.g., fruit and vegetable consumption) and socioeconomic factors accounted for 10% of racial disparities in adult BMI (31). However, studies to date have not assessed the contribution of psychosocial stressors to racial/ethnic differences, and they have assessed differences only between Black and White, discounting the impact that social determinants may have in explaining differences between Hispanic/Latino and White.

In the present study, we applied the Oaxaca-Blinder decomposition method to examine the relative contribution of health behaviors, SEP, and psychosocial stressors in explaining obesity disparities among Black and Hispanic/Latino versus non-Hispanic White adults in a probability sample of adults in Chicago, Illinois. These findings can inform future interventions as they identify key areas that can be targeted to reduce disparities in obesity.

Methods

We analyzed data from the Chicago Community Adult Health Study (2001-2003), a cross-sectional study of 3,105 adults, aged 18 and above, who lived in 343 neighborhood clusters within the city of Chicago. Face-to-face interviews with one individual per selected household were conducted between May 2001 and March 2003. Data were weighted to match the demographics of the city, including age, race/ethnicity, and sex distribution based on 2000 census estimates. A more detailed description of the study design is found elsewhere (32). In the analysis, we excluded a total of 122 (3.94%) respondents who had missing information on stressor variables required for our study. There were no significant differences in race/ethnicity, sex, education, obesity, or stress exposure between included and excluded individuals.

Dependent variable

Trained interviewers administered survey-based measures and measured the respondent's height and weight. BMI was calculated based

on measured height and weight, and individuals were categorized as without obesity ($<30 \text{ kg/m}^2$) or with obesity ($\geq 30 \text{ kg/m}^2$).

Independent variable

The primary predictor was race/ethnicity, which comprised three racial/ethnic groups, Black, Hispanic, and White. We separated non-US-born and US-born Hispanic in all analyses, as stress exposure and obesity is patterned by nativity among Hispanic in the US (33). We measured nativity with a dichotomous indicator of whether the person was born in any of the 50 states or outside of the US. Although Puerto Rico is a US territory, Puerto Ricans born in the island do not consider themselves to be “US-born” and generally have a different health profile than those born in the mainland US (34,35). Therefore, we considered Puerto Ricans born outside the US as “non-US-born.” A total of 78 individuals identified as “other” race/ethnicity. Given that small number, we could not examine this group as a separate category. These individuals were most similar to White participants in terms of sociodemographic characteristics; therefore, we followed a previous study using Chicago Community Adult Health Study data (26) and combined the “other” category with White to enhance available data.

Sociodemographics

We included the following sociodemographic variables in the models: age (by year), sex (male or female), marital/partner status (yes or no), and parental status (i.e., having any children, yes or no).

Health behaviors

Both diet and physical activity have been associated with stress and with obesity (23,36). Diet was assessed by asking participants how many servings of fruits or vegetables (combined) they usually eat in a day. A serving was defined as a cup of fruit or vegetable juice or a half cup of raw or cooked vegetables or fruits. This included juices and all types of raw or cooked fruits and vegetables. Responses ranged from 0 to 20 and were dichotomized into two categories, meeting or not meeting the recommended fruit and vegetable intake (i.e., five or more vs. less than five servings of combined fruits and vegetables) (37). Physical activity was assessed using six items from the National Health Interview Survey (38), which captures frequency, intensity, and duration of activities. Responses were categorized as inactive (i.e., no physical activity), insufficiently active (i.e., some physical activity but not enough to meet guidelines), and active (i.e., more than four times per week engaging in light/moderate to vigorous leisure activities).

SEP

We included the following variables as indicators of SEP: education (less than high school, high school, some college, or college degree or higher), employment status (yes or no), and household income ($< \$10,000$, $\$10,000$ – $\$29,999$, $\$30,000$ – $\$49,999$, $\$50,000$ or more, or missing income). Individuals with missing income data were included in the sample as a separate category to avoid losing participants.

Psychosocial stressors

The measures of psychosocial stress used in this study have been previously used to assess the association between psychosocial stress and obesity and other health outcomes (e.g., depressive symptoms,

chronic illnesses, physical limitations) (26). A more detailed description of each stressor domain can be found in a published article by Sternthal and colleagues (26). Briefly, “childhood adversity” was assessed with eight Likert-style items that asked participants about their childhood experiences, with questions ranging from how often their parents made them feel loved to how well off their family was when they were growing up. “Acute life events” was assessed with two life-event inventories that assessed acute life events over the life span (4 items) and acute life events in the past 5 years (11 items). “Financial strain” was assessed with two measures that evaluated self-reported financial strain (two items) and a financial event inventory that appraised serious economic problems (seven items). “Neighborhood stressors” contained three measures that assessed community violence in the past 6 months (five items), personal victimization in the community (four items), and community disorder (five items). “Employment stressors” included six measures that assessed job dissatisfaction (one item), job autonomy (three items), job security (two items), work demand (three items), work–life conflicts (two items), and job hazards (three items). “Job discrimination” contained two measures that assessed job harassment (two items) and unfair treatment in the workplace (three items). “Relationship stressors” comprised five measures that assessed marital problems (eight items), child-related problems (nine items), and friendship issues (two items). “Lifetime discrimination” measured racial and nonracial discrimination using questions from an inventory of major discriminatory events (four items) and a shortened version of the Everyday Discrimination Scale (five items). For domains that included multiple measures, we transformed each measure into a z score and summed them together. We then re-standardized the sum score into a z score to allow for comparisons across domains (26). Following other research (26), each stressor domain was dichotomized to contrast the top versus other quintiles. A cumulative high-stress score was created to identify individuals experiencing high levels of stress across multiple domains. The cumulative stress score reflects the number of domains in which the individual was in the top quintile of stress exposure. The score ranged from 0 to 8, used as a categorical variable.

Statistical analysis

We used ANOVA and χ^2 tests to examine distributions of stressors, SEP, and health behaviors in the whole study sample and by race/ethnicity. We then used the Oaxaca-Blinder decomposition to assess the explanatory effects of the independent variables (sociodemographic factors [i.e., age, sex, marital status, and parental status], health behavior [i.e., fruit and vegetable consumption and physical activity], SEP [income, education, and employment status], and cumulative high-stress) on obesity disparities between racial/ethnic groups.

The Oaxaca-Blinder decomposition quantifies the proportion of racial/ethnic differences in obesity prevalence with the independent variable, which is referred to as the “explained” portion. It also produces the proportion “unexplained,” which is the differences in obesity prevalence that would remain even if the disadvantaged minority groups had the same mean levels on all the independent variable as White participants. A more detailed description of the approach is found elsewhere (29).

Three separate analyses were conducted to estimate the associations between the independent variables and obesity status (without obesity vs. with obesity). The first analysis assessed obesity differences

between Black and White, the second analysis assessed differences between US-born Hispanic and White, and the third analysis assessed differences between non-US-born Hispanic and White. Using the “oaxaca” command and “logit” option in Stata version 14 to analyze absolute differences in obesity status (without obesity vs. with obesity), we included five categories of variables in the models: (1) age and sex, (2) marital status and parental status, (3) health behavior, (4) SEP, and (5) cumulative stressors. While the Oaxaca-Blinder approach is sensitive to which category is chosen as the reference group, we have included the “pooled” option to overcome this issue (39). All analyses adjusted for sample weights and neighborhood cluster to account for the complex survey design.

Exploratory analyses

The prevalence of different types of psychosocial stressors vary by race/ethnicity (26). Therefore, we explored whether the different types of psychosocial stressors contribute to racial/ethnic differences in obesity. In addition, studies find racial/ethnic differences in obesity to be more pronounced among women (1); therefore, we conducted stratified analyses using the decomposition approach.

Results

Of the 2,983 participants in the final sample, 1,802 (60.4%) were female. The mean age was 42.3 years, 34.4% were White, 39.7% were Black, 11.8% were US-born Hispanic, and 14.1% were non-US-born Hispanic. Among all the participants, 1,030 had obesity, more than half of whom (50.2%) were Black. White individuals had significantly higher levels of household income and education than other racial/ethnic groups. Across domains, the mean stress exposure was generally higher among Black and US-born Hispanic individuals than White and non-US-born Hispanic individuals. The two exceptions were childhood adversities and employment stressors, for which non-US-born Hispanic individuals reported the highest levels. Black and US-born Hispanic also had higher prevalence of high exposure to cumulative stressors than White or non-US-born Hispanic. The distribution of sociodemographic variables, psychosocial stressors, health behaviors, and obesity for the overall sample and by race/ethnicity status are shown in Table 1.

Decomposition of Black–White difference

The “explained” portions of the Oaxaca-Blinder models are presented in Table 2. There was no evidence that indicators of SEP and health behaviors explained racial differences in obesity. Notably, having high stress in four or more domains explained 4.46% of differences between Black and White. The combined predictors explained 15.16% of the differences between Black and White.

Decomposition of US-born Hispanic–White difference

None of the SEP and health behavior indicators significantly explained racial/ethnic differences in obesity. Having high stress in three domains significantly explained 14.13% of differences between US-born Hispanic and White. While the predictors together explained 19.53% of the differences between US-born Hispanic and White, there was no evidence that they explained difference at $P < 0.05$.

Decomposition of non-US-born Hispanic–White difference

The indicators for SEP, cumulative stress, and health behaviors did not explain racial/ethnic differences in obesity between US-born Hispanic and White. Overall the predictors explained 14.30%, albeit not significantly at $P < 0.05$.

Exploratory analyses

We examined the effects of the individual stressors in explaining racial/ethnic obesity differences. Financial strain significantly explained 3.65% of differences in obesity between Black and White individuals (Supporting Information Table S1). Neighborhood stress significantly explained 6.48% of differences between US-born Hispanic and White individuals. No other individual stressors explained obesity differences between Black and White and US-born Hispanic and White. None of the individual stressors explained differences between non-US-born Hispanic and White individuals.

Given the documented racial differences in obesity by sex, we conducted sex-stratified analyses. Having a college degree or more explained 24.45% of the differences between Black and White women (Supporting Information Table S2). No other predictor explained racial differences between these groups. Having a college degree or more explained 47.26% of the differences between US-born Hispanic women and White women. Having three stressors and four or more stressors explained 23.35% and 18.72%, respectively, of the differences between US-born Hispanic women and White women. Between non-US-born Hispanic women and White women, partner status explained 18.44% of obesity differences. None of the predictors significantly explained differences in obesity between racial/ethnic minority men and White men.

Discussion

To the best of our knowledge, this is the first study to use the Oaxaca-Blinder decomposition method to examine the explanatory effects of health behaviors, SEP, and cumulative stress on racial/ethnic obesity disparities among a multiracial/multiethnic probability sample of adults. Health behaviors (i.e., fruit and vegetable consumption and physical activity) and SEP did not explain racial/ethnic differences between racial/ethnic minorities and White individuals. In other words, if racial/ethnic minorities and White individuals consumed equal amount of fruits and vegetables, engaged in the same level of physical activity, and were equivalent in SEP, the differences in obesity would remain relatively unchanged. However, having high stress in four or more domains explained approximately 4% of differences between Black and White. Black participants report greater exposure to a wide range of psychosocial stressors compared with their White counterparts (26), placing them at an increased risk for obesity.

Our findings suggest that, if Black and White individuals were equally exposed to four or more stressors, the racial difference in obesity prevalence would be reduced by 4%. While this magnitude is not substantial, it may be that other factors mitigate the obesogenic effects of psychosocial stressors. For instance, Black individuals may engage in effective coping behaviors to buffer the effects of stress on obesity (e.g., religiosity). Future research using the decomposition

TABLE 1 Sample characteristics

	Full sample (N = 2,983)	Stratified by race/ethnicity status											
		Non-Hispanic White (n = 1,027)			Non-Hispanic Black (n = 1,184)			US-born Hispanic (n = 353)			Non-US-born Hispanic (n = 419)		
		Mean	SE	n	Mean	SE	n	Mean	SE	n	Mean	SE	n
Age (y)	42.28	0.66	43.65	0.64	44.02	0.64	35.89	1.15	39.54	0.89		<0.001	
Stress exposure													
Childhood adversities	-0.02	0.03	-0.12	0.03	-0.08	0.03	0.1	0.07	0.3	0.06		<0.001	
Acute life events	-0.07	0.03	-0.22	0.03	0.22	0.04	0.08	0.07	-0.44	0.04		<0.001	
Financial	-0.11	0.03	-0.28	0.03	0.1	0.04	-0.02	0.06	-0.15	0.04		<0.001	
Life discrimination	-0.02	0.03	-0.21	0.03	0.36	0.04	0.23	0.07	-0.55	0.03		<0.001	
Neighborhood	-0.12	0.03	-0.36	0.03	0.22	0.03	0.03	0.07	-0.3	0.05		<0.001	
Job discrimination	0	0.04	-0.02	0.04	0.05	0.04	0.2	0.08	-0.2	0.05		<0.001	
Employment	0.02	0.04	0.07	0.04	-0.09	0.04	-0.01	0.07	0.18	0.06		<0.001	
Relationship	-0.01	0.03	-0.27	0.03	0.22	0.04	0.11	0.06	0.14	0.06		<0.001	
Diet													
Servings of fruits and vegetables	2.3	0.05	2.6	0.05	2.09	0.05	2.1	0.08	2.07	0.07		<0.001	
Gender	N		n		n		n		n				
Male	1,181	44.01	452	33.61	398	33.61	142	40.23	189	45.11			
Female	1,802	55.99	575	66.39	786	66.39	211	59.77	230	54.89		0.05	
Education													
Less than high school	753	11.68	120	23.82	282	23.82	115	32.58	236	56.32			
High school	729	19.96	205	28.63	339	28.63	100	28.33	85	20.29			
Some college	790	24.15	248	32.43	384	32.43	94	26.63	64	15.27			
College degree and above	711	44.21	454	15.12	179	15.12	44	12.46	34	8.11		<0.001	
Income													
Less than \$10,000	345	5.74	59	17.65	209	17.65	47	13.31	30	7.16			
\$10,000-\$29,999	843	19.57	201	32.26	382	32.26	110	31.16	150	35.80			
\$30,000-\$49,999	563	18.31	188	18.07	214	18.07	69	19.55	92	21.96			
\$50,000 or more	689	35.05	360	16.05	190	16.05	73	20.68	66	15.75			
Missing	543	21.32	219	15.96	189	15.96	54	15.30	81	19.33		<0.001	
Employment status													
Has a job	1,789	63.19	649	54.81	649	54.81	207	58.64	284	67.78			
Does not have a job	1,194	36.81	378	45.19	535	45.19	146	41.36	135	32.22		<0.01	
Marital status													
Single/never married	1,129	39.53	406	39.95	473	39.95	160	45.33	90	21.48			
Currently married	1,050	38.56	396	23.56	279	23.56	111	31.44	264	63.01			
Formerly married	804	21.91	225	36.49	432	36.49	82	23.23	65	15.51		<0.001	

TABLE 1. (continued).

	Full sample (N = 2,983)	Stratified by race/ethnicity status							
		Non-Hispanic White (n = 1,027)	Non-Hispanic Black (n = 1,184)	US-born Hispanic (n = 353)	Non-US-born Hispanic (n = 419)				
Parental status									
Has child(ren)	2,027	510	49.66	928	78.38	238	67.42	351	83.77
Does not have children	956	517	50.34	256	21.62	115	32.58	68	16.23
Physical activity									
Inactive	611	193	18.79	263	22.21	55	15.58	100	23.87
Insufficiently active	1,905	688	66.99	735	62.08	235	66.57	247	58.95
Meets guideline	467	146	14.22	186	15.71	63	17.85	72	17.18
Number of domains of high stress exposure (cumulative stress)									
0	851	383	37.29	267	22.55	76	21.53	125	29.83
1	847	316	30.77	301	25.42	91	25.78	139	33.17
2	542	236	22.98	157	13.26	67	18.98	82	19.57
3	375	82	7.98	185	15.63	65	18.41	43	10.26
4+	368	89	8.67	195	16.47	54	15.30	30	7.16
Obesity									
With obesity	1,030	225	21.91	517	43.67	139	39.38	149	35.56
Without obesity	1,953	802	78.09	667	56.33	214	60.62	270	64.44

TABLE 2 Proportion of racial differences in obesity attributable to age and sex, marital status/parental status, health behaviors, socioeconomic status, and cumulative stressors

Predictor	Black vs. White, coeff (95% CI)	Percent explained	US-born Hispanic vs. White, coeff (95% CI)	Percent explained	Non-US-born Hispanic vs. White, coeff (95% CI)	Percent explained
Age	0.001 (-0.005 to 0.008)	0.59%	-0.03 (-0.05 to -0.005)*	-13.37%	-0.01 (-0.02 to -0.003)*	-7.76%
Sex						
Male (reference)						
Female	0.006 (0.00002 to 0.01)*	2.73%	-0.0001 (-0.003 to 0.002)	-0.06%	0.00005 (-0.002 to 0.002)	0.03%
Marital status						
Not married (reference)						
Married	-0.006 (-0.01 to 0.001)	-3.01%	-0.002 (-0.01 to 0.007)	-1.16%	0.02 (-0.003 to 0.04)	9.81%
Parental status						
Does not have children (reference)						
Has child(ren)	0.01 (-0.005 to 0.03)	6.21%	0.009 (-0.005 to 0.02)	4.39%	0.01 (-0.01 to 0.04)	8.13%
Physical activity						
Inactive (reference)						
Insufficiently active	-0.002 (-0.006 to 0.002)	-0.94%	-0.00002 (-0.0007 to 0.0007)	-0.01%	0.0005 (-0.005 to 0.006)	0.27%
Meets guideline	0.0002 (-0.001 to 0.002)	0.11%	-0.002 (-0.006 to 0.003)	-0.80%	-0.00008 (-0.0009 to 0.0008)	-0.05%
Diet						
Does not meet recommended fruit and vegetable intake (reference)						
Meets recommended fruit and vegetable intake	-0.002 (-0.007 to 0.003)	-1.03%	-0.006 (-0.01 to 0.001)	-2.82%	-0.006 (-0.01 to 0.002)	-3.66%
Education						
Less than high school (reference)						
High school	0.002 (-0.005 to 0.009)	0.93%	0.001 (-0.01 to 0.01)	0.62%	0.0006 (-0.003 to 0.004)	0.37%
Some college	-0.002 (-0.008 to 0.004)	-0.83%	-0.0008 (-0.006 to 0.005)	-0.39%	-0.002 (-0.008 to 0.005)	-0.88%
College degree and above	0.01 (-0.01 to 0.04)	6.48%	0.01 (-0.03 to 0.05)	5.67%	0.005 (-0.03 to 0.04)	2.67%
Income						
Less than \$10,000 (reference)						
\$10,000-\$29,999	-0.001 (-0.01 to 0.01)	-0.54%	0.01 (-0.002 to 0.02)	4.75%	0.002 (-0.003 to 0.007)	1.21%
\$30,000-\$49,999	-0.002 (-0.01 to 0.01)	-0.83%	0.01 (-0.004 to 0.03)	5.69%	-0.001 (-0.01 to 0.01)	-0.80%
\$50,000 or more	-0.0005 (-0.003 to 0.002)	-0.22%	0.0006 (-0.004 to 0.005)	0.31%	-0.001 (-0.006 to 0.004)	-0.55%
Missing	0.002 (-0.01 to 0.02)	0.86%	-0.002 (-0.02 to 0.01)	-0.80%	0.005 (-0.01 to 0.02)	3.04%
Employment status						
Does not have a job (reference)						
Has a job	-0.004 (-0.01 to 0.003)	-1.69%	-0.001 (-0.005 to 0.003)	-0.48%	-0.0005 (-0.004 to 0.003)	-0.32%
Number of domains of high stress exposure (cumulative stress)						
0 (reference)						

TABLE 2. (continued).

Predictor	Black vs. White, coeff (95% CI)	Percent explained	US-born Hispanic vs. White, coeff (95% CI)	Percent explained	Non-US-born Hispanic vs. White, coeff (95% CI)	Percent explained
1	-0.002 (-0.007 to 0.002)	-1.01%	-0.005 (-0.01 to 0.002)	-2.27%	0.001 (-0.002 to 0.005)	0.80%
2	0.002 (-0.002 to 0.006)	1.02%	0.005 (-0.003 to 0.01)	2.71%	0.0007 (-0.002 to 0.004)	0.43%
3	0.004 (-0.005 to 0.01)	1.88%	0.03 (0.008 to 0.05)*	14.13%	0.003 (-0.002 to 0.008)	1.80%
4+	0.01 (0.001 to 0.02)*	4.46%	0.007 (-0.002 to 0.02)	3.42%	-0.0004 (-0.004 to 0.004)	-0.21%
Explained total	0.03 (0.001 to 0.06)*	15.16%	0.04 (-0.01 to 0.09)	19.53%	0.02 (-0.03 to 0.08)	14.30%
Unexplained	0.18 (0.12 to 0.24)**	84.84%	0.16 (0.08 to 0.24)**	80.47%	0.15 (0.07 to 0.22)**	85.70%
Total predicted gap	0.21 (0.17 to 0.26)**		0.20 (0.13 to 0.27)**		0.17 (0.11 to 0.23)**	

All results are weighted. Coeff, coefficients. *P<0.05. **P<0.01.

method should consider other relevant psychosocial factors that may explain racial differences in obesity. Having high stress in three domains explained approximately 14% of differences between US-born Hispanic and White participants. That is to say, if US-born Hispanic and White individuals were equally exposed to three stressors (regardless of the type of stressors), racial/ethnic differences in obesity prevalence would be reduced by 14%. Yet we did not find evidence that psychosocial stressors play a significant role in explaining differences between non-US-born Hispanic and White individuals. Previous research has found that US-born Hispanic individuals have similar prevalence rates of stressors compared with Black individuals, whereas non-US-born Hispanic individuals have stress profiles similar to White individuals (26). According to Tillman and Weiss (40), non-US-born Hispanic individuals may appraise stressors differently compared with US-born Hispanic. While the level of exposure may be similar between US-born Hispanic and non-US-born Hispanic, the effects on health may vary (40). Study findings need to be replicated to examine the role that stressors and stress appraisal may play in the relationship between race/ethnicity and obesity.

In our exploratory analyses to consider the explanatory effects of individual stressors, financial strain was the only stressor that significantly explained differences in obesity between Black and White individuals, explaining 4.15% of obesity differences. Neighborhood stress explained 7.08% of differences between US-born Hispanic and White. While these stressors warrant further investigation, it is important not to discount the potential adverse obesogenic effects of the other stressors as they can co-occur and accumulate to increase obesity risk (26). Future research should examine how stressors individually and together are pathways for existing racial/ethnic disparities in obesity.

The predictors together explained less than 20% for any of the racial/ethnic differences in obesity. While health behaviors and SEP did not significantly explain racial/ethnic differences in obesity, they remain risk factors for obesity and mechanisms of persistent health inequities. For instance, education is thought to increase the risk of obesity (41). Racial/ethnic gaps in college attendance remains large despite increases in Black and Hispanic/Latino enrollment and graduation over the last 3 decades (42). In fact, when we stratified analyses by sex, we found that having a college degree or more explained a substantial proportion of obesity differences between Black and White women and US-born Hispanic women and White women. Our findings suggest that other unexamined social and psychological factors may further explain racial/ethnic differences in obesity. Our findings need to be replicated and include other obesity-related risk factors that disproportionately affect racial/ethnic minorities. We also did not examine environmental influences of obesity. For instance, residential segregation is a known social determinant of health (43). Even after adjusting for SEP, segregation is a strong predictor of poor physical and mental health (43). Given that Black and Hispanic are more residentially segregated than White, they are excessively exposed to limited access to health-promoting resources (27). Our operationalization of stressors does not fully capture individuals' socioenvironmental context, particularly racial segregation. Therefore, in future studies, it will be important to examine aspects of the social environment (e.g., racial density) in relation to the onset of obesity.

The study has limitations. First, the data are cross-sectional, which prevents us from prospectively examining whether health behaviors,

SEP, and stressors across the life course explain racial/ethnic weight differences over time. The operationalization of SEP and health behaviors influenced our conclusions. We used three traditional indicators of SEP (income, employment status, and education), which could underestimate the contribution of SEP to observed racial and ethnic disparities in obesity as they do not comprehensively capture the SEP of racial/ethnic groups. For instance, Black and Hispanic individuals have less wealth, receive less income, and have less purchasing power compared with White at the same education levels (43). Relatedly, diet and physical activity are multidimensional health behaviors. We did not measure intake of fat and sugar for dietary behaviors, nor did we measure work-related physical activity. Including more comprehensive measures of SEP and health behaviors can help elucidate the mechanisms underlying obesity disparities. The sample sizes for US-born and non-US-born Hispanic were small, therefore the analyses may have been underpowered to adequately address the research questions. Moreover, we only examined a subset of potential life stressors. Acculturation stress is commonly experienced by immigrants and may influence weight gain more than other stressors for non-US-born Hispanic. Future studies should consider a wider range of stressors in relation to obesity risk. Moreover, while non-US-born immigrants tend to have lower obesity rates compared with their US-born counterparts, this advantage tends to diminish over time (44). This may be due to cumulative stress exposure the longer individuals live in the US. We were not able to take length of residence in the US into account in our study. Future research should replicate these findings and examine the role of length of residence within the context of stress and obesity. Finally, this study was of Chicago residents, and results may not generalize to the wider population.

Conclusion

Our study examined the role of health behaviors, SEP, and cumulative stress exposure in explaining racial/ethnic obesity disparities. Using Oaxaca-Blinder decomposition, we found that having high levels of stress in four or more domains explained approximately 4% of differences between Black and White individuals, and having high levels of stress in three domains explained around 14% of differences between US-born Hispanic and White individuals. There was no evidence that SEP and health behaviors explained racial/ethnic differences in obesity prevalence. In order to advance our understanding of the social determinants affecting obesity disparities at the population level, future research is needed to examine socioenvironmental-level and individual-level factors that are associated with an elevated risk for obesity, such as racial segregation (27). **O**

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References

1. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in obesity among adults in the United States, 2005 to 2014. *JAMA* 2016;315:2284-2291.
2. Hruby A, Hu FB. The epidemiology of obesity: a big picture. *Pharmacoeconomics* 2015;33:673-689.
3. Ledoux TA, Hingle MD, Baranowski T. Relationship of fruit and vegetable intake with adiposity: a systematic review. *Obes Rev* 2011;12:e143-e150.
4. Wiklund P. The role of physical activity and exercise in obesity and weight management: time for critical appraisal. *J Sport Health Sci* 2016;5:151-154.
5. August KJ, Sorkin DH. Racial/ethnic disparities in exercise and dietary behaviors of middle-aged and older adults. *J Gen Intern Med* 2011;26:245-250.
6. Winkleby WA, Cubbin C. Racial/ethnic disparities in health behaviors: a challenge to current assumptions. In: Anderson NB, Bulatao RA, Cohen B, National Research Council (US) Panel on Race, Ethnicity, and Health in Later Life, eds. *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*. Washington, DC: National Academies Press; 2004:450-491.
7. Krueger PM, Reither EN. Mind the gap: race/ethnic and socioeconomic disparities in obesity. *Curr Diab Rep* 2015;15:95. doi:10.1007/s11892-015-0666-6
8. Shaikh RA, Siahpush M, Singh GK, Tibbits M. Socioeconomic status, smoking, alcohol use, physical activity, and dietary behavior as determinants of obesity and body mass index in the United States: findings from the National Health Interview Survey. *Int J MCH AIDS* 2015;4:22-34.
9. Pampel FC, Krueger PM, Denney JT. Socioeconomic disparities in health behaviors. *Annu Rev Sociol* 2010;36:349-370.
10. Marshall SJ, Jones DA, Ainsworth BE, Reis JP, Levy SS, Macera CA. Race/ethnicity, social class, and leisure-time physical inactivity. *Med Sci Sports Exerc* 2007;39:44-51.
11. Dubowitz T, Heron M, Bird CE, et al. Neighborhood socioeconomic status and fruit and vegetable intake among whites, blacks, and Mexican Americans in the United States. *Am J Clin Nutr* 2008;87:1883-1891.
12. Dubowitz T, Heron M, Basurto-Davila R, Bird CE, Lurie N, Escarce JJ. Racial/ethnic differences in U.S. health behaviors: a decomposition analysis. *Am J Health Behav* 2011;35:290-304.
13. Nuru-Jeter AM, Michaels EK, Thomas MD, Reeves AN, Thorpe RJ Jr, LaVeist TA. Relative roles of race versus socioeconomic position in studies of health inequalities: a matter of interpretation. *Annu Rev Public Health* 2018;39:169-188.
14. Ogden CL, Fakhouri TH, Carroll MD, et al. Prevalence of obesity among adults, by household income and education—United States, 2011-2014. *MMWR Morb Mortal Wkly Rep* 2017;66:1369-1373.
15. Sánchez-Vaznaugh EV, Kawachi I, Subramanian SV, Sánchez BN, Acevedo-Garcia D. Do socioeconomic gradients in body mass index vary by race/ethnicity, gender, and birthplace? *Am J Epidemiol* 2009;169:1102-1112.
16. Bell CN, Thorpe RJ, Bowie JV, LaVeist TA. Race disparities in cardiovascular disease risk factors within socioeconomic status strata. *Ann Epidemiol* 2018;28:147-152.
17. Block JP, He Y, Zaslavsky AM, Ding L, Ayanian JZ. psychosocial stress and change in weight among US adults. *Am J Epidemiol* 2009;170:181-192.
18. Deng T, Lyon CJ, Bergin S, Caligiuri MA, Hsueh WA. Obesity, inflammation, and cancer. *Annu Rev Pathol* 2016;11:421-449.
19. Ramos-Nino ME. The role of chronic inflammation in obesity-associated cancers. *ISRN Oncol* 2013;2013:697521. doi:10.1155/2013/697521
20. Lumeng CN, Saltiel AR. Inflammatory links between obesity and metabolic disease. *J Clin Invest* 2011;121:2111-2117.
21. Maxwell MA, Cole DA. Weight change and appetite disturbance as symptoms of adolescent depression: toward an integrative biopsychosocial model. *Clin Psychol Rev* 2009;29:260-273.
22. Iversen LB, Strandberg-Larsen K, Prescott E, Schnohr P, Rod NH. Psychosocial risk factors, weight changes and risk of obesity: the Copenhagen City Heart Study. *Eur J Epidemiol* 2012;27:119-130.
23. Stults-Kolehmainen MA, Sinha R. The effects of stress on physical activity and exercise. *Sports Med* 2014;44:81-121.
24. Scott KA, Melhorn SJ, Sakai RR. Effects of chronic social stress on obesity. *Curr Obes Rep* 2012;1:16-25.
25. Kwarteng JL, Schulz AJ, Mentz GB, Israel BA, Perkins DW. Independent effects of neighborhood poverty and psychosocial stress on obesity over time. *J Urban Health* 2017;94:791-802.
26. Sternthal MJ, Slopen N, Williams DR. Racial disparities in health: how much does stress really matter? *Du Bois Rev* 2011;8:95-113.
27. Williams DR, Priest N, Anderson N. Understanding associations between race, socioeconomic status and health: patterns and prospects. *Health Psychol* 2016;35:407-411.
28. Dolezsar CM, McGrath JJ, Herzig AJM, Miller SB. Perceived racial discrimination and hypertension: a comprehensive systematic review. *Health Psychol* 2014;33:20-34.
29. Elder TE, Goddeeris JH, Haider SJ. Unexplained gaps and Oaxaca-Blinder decompositions. *Labour Econ* 2010;17:284-290.
30. O'Donnell O, Van Doorslaer E, Wagstaff A. Decomposition of inequalities in health and health care. In: Jones AM, ed. *The Elgar Companion to Health Economics*. Cheltenham: Edward Elgar Publishing Limited; 2006. doi:10.4337/9781845428914.00027
31. Sen B. Using the Oaxaca-Blinder decomposition as an empirical tool to analyze racial disparities in obesity. *Obesity (Silver Spring)* 2014;22:1750-1755.
32. Morenoff JD, House JS, Hansen BB, Williams DR, Kaplan GA, Hunte HE. Understanding social disparities in hypertension prevalence, awareness, treatment, and control: the role of neighborhood context. *Soc Sci Med* 2007;65:1853-1866.
33. Wen M, Kowaleski-Jones L, Fan JX. Ethnic-immigrant disparities in total and abdominal obesity in the US. *Am J Health Behav* 2013;37:807-818.
34. Landale NS, Oropesa RS. White, Black or Puerto Rican? Racial self-identification among mainland and island Puerto Ricans. *Soc Forces* 2002;81:231-254.

35. Rodríguez F, Hastings KG, Hu J, et al. Nativity status and cardiovascular disease mortality among Hispanic adults. *J Am Heart Assoc* 2017;6. doi:10.1161/JAHA.117.007207
36. Yau YHC, Potenza MN. Stress and eating behaviors. *Minerva Endocrinol* 2013;38: 255-267.
37. Krauss RM, Eckel RH, Howard B, et al. AHA Dietary Guidelines: revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association. *Circulation* 2000;102:2284-2299.
38. Botman SL, Moore TF, Moriarity CL, Parsons VL. Design and estimation for the National Health Interview Survey, 1995-2004. *Vital and Health Statistics*, series 2, no. 130. Hyattsville, MD: National Center for Health Statistics; 2000.
39. Taber DR, Robinson WR, Bleich SN, Wang YC. Oaxaca-Blinder decomposition of disparities in adolescent obesity: deconstructing both race and gender differences. *Obesity (Silver Spring)* 2016;24:719-726.
40. Tillman KH, Weiss UK. Nativity status and depressive symptoms among Hispanic young adults: the role of stress exposure. *Soc Sci Q* 2009;90:1228-1250.
41. Cohen AK, Rai M, Rehkopf DH, Abrams B. Educational attainment and obesity: a systematic review. *Obes Rev* 2013;14:989-1005.
42. Musu-Gillette L, de Brey C, McFarland J, Hussar W, Sonnenberg W, Wilkinson-Flicker S. *Status and Trends in the Education of Racial and Ethnic Groups 2017. NCES 2017-051*. Washington, DC: National Center for Education Statistics, US Department of Education; 2017.
43. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep* 2001;116:404-416.
44. Isasi CR, Ayala GX, Sotres-Alvarez D, et al. Is acculturation related to obesity in Hispanic/Latino adults? Results from the Hispanic community health study/study of Latinos. *J Obes* 2015;2015:186276. doi:10.1155/2015/186276