Psychosocial Stress and Overweight and Obesity: Findings From the Chicago Community Adult Health Study

Adolfo G. Cuevas, PhD^{1,✉} • Ruijia Chen, MS² • Katherine A. Thurber, PhD³ • Natalie Slopen, ScD⁴ • David R. Williams, MPH, PhD^{2,5}

Published online: 27 March 2019

© Society of Behavioral Medicine 2019. All rights reserved. For permissions, please e-mail: journals.permissions@oup.com.

Abstract

Background Psychosocial stress has been implicated as a risk factor for overweight and obesity. However, research on psychosocial stressors and overweight and obesity has typically focused on single stressors in isolation, which may overestimate the impact of a specific stressor and fail to describe the role of cumulative stress on overweight and obesity risk.

Purpose This study explores the association between overweight/obesity and cumulative exposure to a wide range of psychosocial stressors, among a multiracial/ethnic sample of adults.

Methods Using secondary data from the Chicago Community Adult Health Study ($n = 2,983$), we conducted multinomial logistic regression analyses to quantify associations between eight psychosocial stressors, individually and in combination, and measured

overweight and obesity, adjusted for sociodemographic factors, alcohol use and smoking.

Results In separated covariate-adjusted models, childhood adversities (odds ratio [OR] = 1.16; confidence interval [CI] = [1.03, 1.30]), acute life events (OR = 1.18; CI = [1.04, 1.34]), financial strain (OR = 1.30; CI = [1.15, 1.47]), and relationship stressors (OR = 1.18; CI = [1.04, 1.35]) were associated with increased odds of obesity. In a model adjusted for all stressors simultaneously, financial strain was the only stressor independently associated with overweight (OR = 1.17; CI = [1.00, 1.36]) and obesity (OR = 1.21; CI = [1.05, 1.39]). Participants with stress exposure in the highest quintile across 2, 3, or ≥ 4 (compared to no) types of stressors had significantly higher odds of obesity.

Conclusions Multiple types of stressors may be risk factors for obesity, and cumulative exposure to these stressors may increase the odds of obesity. Reducing exposure to stressors at the population level may have the potential to contribute to reducing the burden of obesity.

✉ Adolfo G. Cuevas
adolfo.cuevas@tufts.edu

¹ Department of Community Health, Tufts University, 574 Boston Avenue, Suite 208, Medford, MA 02155

² Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Boston, MA

³ National Centre for Epidemiology and Population Health, Research School of Population Health, Australian National University, Canberra, ACT, Australia

⁴ Department of Epidemiology and Biostatistics, University of Maryland, College Park, MD

⁵ Department of African and African American Studies, Harvard University, Cambridge, MA

Keywords Obesity • Overweight • Stress • Psychosocial stressors

Background

Obesity is a persistent public health issue in the USA [1]. The prevalence of obesity has increased from 30.5 in 1999–2000 to 39.9 in 2015–2016 [2, 3], placing more than one-third of the population at an obesity-related increased risk for cardiovascular disease, prostate cancer, breast cancer, colorectal cancer, and premature

mortality [1, 4–6]. There has been a substantial increase in the prevalence of severe obesity (body mass index > 40.0) during this period as well, with 4.0% of the U.S. population having severe obesity in 1999–2000, rising to 7.7 in 2015–2016 [2, 3]. There is also evidence that individuals with BMI within the overweight range have an increased risk of cardiovascular disease risk [7] and are more likely to become obese over time [8]. Therefore, there is potential for substantial health gain through reducing overweight and obesity prevalence at the population level.

The etiology of obesity is multifactorial, reflecting a complex interaction of biological, behavioral, and socioenvironmental factors [7]. For instance, there is evidence that genetic markers independently predict obesity susceptibility [7]. Other factors, such as physical inactivity and poor dietary behaviors, interact together to increase risk of obesity. Mounting evidence suggests that stress increases the risk of overweight and obesity, and this may occur via psychological and/or behavioral pathways [9, 10]. For example, stressful life events are associated with increased depressive symptoms [11] and people with depression are at a greater risk of developing obesity compared to people without depression [12]. Exposure to stressors can also release appetite hormones that increase craving for foods high in fat and sugar [13] and weaken efforts to be physically active [14]. Similarly, Laugero et al. [15] found that participants who reported greater perceived stress were more likely to report lower fruit, vegetable, and protein intake, increased consumption of salty snacks and sweets, and lower participation in physical activities.

A meta-analysis of prospective studies revealed that psychosocial stressors including acute stressful events (e.g., being fired or losing a loved one) are risk factors for weight gain [16]. Other forms of psychosocial stressors, such as weight discrimination (i.e., unfair treatment toward individual based on their weight) [17], childhood trauma (e.g., physical abuse, verbal abuse, witnessed abuse, humiliation, neglect) [18, 19], financial strain (e.g., difficulty paying bills) [20, 21], and conflict within interpersonal relationships (e.g., adverse exchanges and conflict) [22] may also be risk factors for obesity in adulthood.

Stress is multidimensional and multiple psychosocial stressors often co-occur [16]. Thus, to comprehensively characterize the effect of stressors on well-being, it is necessary to capture different sources of psychosocial stressors. To date, the majority of research on psychosocial stressors and overweight and obesity has focused on single stressors in isolation [16], which is likely to overestimate the impact of that specific stressor and fail to characterize the role of stressors more generally in the development of overweight and obesity. Despite the

potential contribution of a broad range of stressors to overweight and obesity, to our knowledge, there has not been a comprehensive examination of the contribution of multiple psychosocial stressors, and their co-occurrence, to overweight and obesity risk. This study aims to quantify the association between a wide range of psychosocial stressors—individually and in combination—and overweight and obesity among non-Hispanic Black, native-born Hispanic, foreign-born Hispanic and non-Hispanic White adults in a representative sample of Chicago, IL residents. In addition, we quantified the association between cumulative high-stress exposure and overweight and obesity.

Method

Sample

We conducted secondary analysis of data from the Chicago Community Adult Health Study (CCAHS), a cross-sectional multistage, probability sample of 3,105 adults (aged 18 and older) living in 343 neighborhood areas within the city of Chicago. One adult was selected per household to participate in a face-to-face interview. Data collection occurred between May 2001 and March 2003. Data were weighted to match the city's age, race/ethnicity, and sex distribution based on 2000 census estimates. A more detailed description of the study design is available elsewhere [23].

In the analysis, we excluded a total of 122 (3.94%) respondents who had missing information on study variables. There were no differences in race/ethnicity, sex, education, obesity, or stress exposure between included and excluded individuals at p value $< .05$.

Dependent Variables

Trained interviewers administered survey-based measures and measured the respondent's height and weight. The primary outcomes were overweight and obesity based on body mass index (BMI). BMI was calculated based on measured height and weight, and values were categorized as normal or underweight (< 25 kg/m²), overweight (≥ 25 and < 30 kg/m²), or obese (≥ 30 kg/m²). We combined participants who were underweight and normal weight due to the small number of participants who were underweight ($n = 52$).

Psychosocial Stressors

We examined eight domains of psychosocial stressors (childhood adversity, acute life events, financial

strain, neighborhood stressors, employment stressors, job discrimination, relationship stressors, and life discrimination), and a measure of cumulative exposure across these stress domains. Previous publications provide a more detailed description of each stressor domain [24, 25]. Each domain included one or more measures, as described below. 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 [24, 25]. These measures have been previously used to assess the association between psychosocial stress and a variety of mental and physical health outcomes (e.g., depressive symptoms, chronic illnesses, physical limitations) [24].

Childhood adversity included eight Likert-style items that asked participants how often their parents: (i) made them feel loved, (ii) physically held and comforted them, (iii) physically threatened or abused them, (iv) verbally threatened or abused them, (v) participated in activities in their school, and (vi) read to them; (vii) how often they went to bed at night feeling hungry; and (viii) how well off their family was when they were growing up. *Acute life events* included two life event inventories: (i) acute life events over the life span (four items); and (ii) acute life events in the past 5 years (11 items). *Financial strain* included two measures: self-reported financial strain (two items) and a financial event inventory reflecting serious economic problems (seven items). *Neighborhood stressors* contained three measures: (i) community violence in the past 6 months (five items); (ii) personal victimization in community (four items); and (iii) community disorder (five items). *Employment stressors* included six measures reflecting different domains of work-place stress: (i) job dissatisfaction (one item); (ii) job autonomy (three items); (iii) job security (two items); (iv) work demand (three items); (v) work-life conflicts (two items); and (vi) job hazards (three items). *Job discrimination* contained two measures: (i) job harassment (two items); (ii) treated unfairly in job (three items). *Relationship stressors* were comprised of five measures: (i) marital stress (four items); (ii) marital abuse (four items); (iii) child-related stress (three items); (iv) total problems of children (six items); and (v) friend criticism (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).

A *cumulative stress score* was created to identify individuals experiencing high levels of stress across multiple domains. Consistent with previous research [24–26], each stressor domain was dichotomized to contrast the top quintile versus others. The cumulative

stress score reflects the number of domains in which the individual was in the top quintile of stress exposure. The score ranges from 0 to 8, and was categorized as 0, 1, 2, 3, or 4 or more.

Covariates: Sociodemographics and Health Behaviors

Consistent with previous literature [24, 25], we included the following sociodemographic covariates in the adjusted regression models: age (in years), sex (male or female), race/ethnicity (Black, native-born Hispanic, foreign-born Hispanic, or White/other), education (less than high school, high school, some college, or college degree or higher), employment status (yes or no), marital/partner status (yes or no), having any children (yes or no), and household income (<\$10,000, \$10,000–\$29,999, \$30,000–\$49,999, \$50,000 or more, or missing income). A total of 78 individuals identified as “other” race/ethnicity. Given that the small number, we could not examine this group as a separate category. These individuals were most similar to Whites in terms of sociodemographic characteristics. Consistent with prior CCAHS studies [24, 25, 27], we combined the “other” category with whites to enhance available data. Although participants with missing data on the outcome, stressors, or other covariates were excluded, individuals with missing income data were included in the sample as a separate category to minimize any potential bias resulting from differences between those who chose to report versus not to report their income. We included smoking and alcohol consumption as covariates as they are potential confounders of the relationship between stress and obesity (i.e., smoking and alcohol consumption could be associated with both stressors and BMI) [28–33]. Alcohol intake and tobacco use were coded as current, former, or never, consistent with prior studies using CCAHS [34, 35].

Statistical Analysis

Descriptive analyses were performed to characterize the sample's sociodemographic characteristics, health behaviors, and stress exposure, stratified by BMI category. Multinomial logistic regression models were used to quantify associations between the eight stressors and overweight and obesity, individually and then together. A separate model was used to quantify the association between cumulative high-stress and overweight and obesity. Sociodemographic factors and health behaviors were adjusted in all models. All analyses were conducted using SAS 9.4 and included sample weights and accounted for neighborhood clustering.

Exploratory Analyses

Previous analysis of CCAHS data has shown a different effect of everyday discrimination on waist circumference across racial groups [34]; therefore, we tested for effect modification by race/ethnicity for all stressors using interaction terms. In addition, because prior research has shown that women and men differ in their exposure and reactions to stressors [36–38], we tested sex as an effect modifier using the same approach. We performed stratified analyses if significant interactions were detected.

Health and mortality risks are significantly greater for those with higher classes of obesity, particularly those with Class III obesity [39, 40]. Individuals with Class III obesity have higher rates of depression compared with individuals with Class I and Class II obesity [41], which may be indicative of greater exposure to psychosocial stressors. Therefore, psychosocial stressors might be more strongly associated with Class III obesity than to obesity overall. Therefore, we used multinomial logistic regression models to explore the association between the stressors and the three classes of obesity, Class I obesity (i.e., BMI = 30.0–34.9), Class II obesity (BMI = 35.0–39.9) and Class III obesity (BMI $\geq$ 40.0).

Sensitivity Analyses

We conducted two sensitivity analyses to assess the robustness of our results. First, we conducted a sensitivity analysis excluding respondents who were underweight from the analyses ($n = 52$). Second, as described above, the primary analysis combined the “other” category with Whites to enhance available data [24, 25, 27]. To test if including this group within the White group influenced results, we replicated the analyses excluding these 78 participants.

Results

A total of 2,983 participants were included in the main analyses, of whom 1,030 (34.53%) were obese, 965 (32.35%) were overweight, and 988 (33.12%) were normal or underweight ($n = 52$). The average age of the sample was 42.28 ($SD = 0.42$) years old. Less than half of the participants were male (39.6%). Table 1 presents the distribution of sociodemographic characteristics and health behaviors in the full sample overall and by BMI category. Mean stressor z -scores differed across categories of BMI, with individuals who were obese having the highest exposure to childhood adversities, acute life events, financial strain, and relationship stressors, compared with other BMI categories (Table 2).

Individuals who were obese also were disproportionately likely to have high exposure to four or more stressors. Correlations between the stressors range from -0.07 to 0.54 .

Financial strain was the only stressor that was associated with overweight at p -value $< .05$, both when examined individually (odds ratio [OR] = 1.16, confidence interval [CI] = [1.02, 1.33]; Model 1A, Table 3) or when including all stressors together (OR = 1.17, CI = [1.00, 1.36]; Model 2A, Table 3).

When the stressors were examined individually in relation to obesity, increased exposure to early life adversities (OR = 1.16, 95% CI = [1.03, 1.30]), acute life events (OR = 1.18, CI = [1.04, 1.34]), financial strain (OR = 1.30, CI = [1.15, 1.47]), and relationship stressors (OR = 1.18, CI = [1.04, 1.35]), were associated with increased odds of obesity (Model 2A, Table 3). In contrast, lifetime discrimination, job discrimination, neighborhood stress, and employment stress did not display associations with obesity at p value $< .05$.

In the model mutually adjusted for all stressors, only financial strain was independently associated with obesity (OR = 1.21, CI = [1.05, 1.39]; Model 2B). Participants who were in the top quintile of exposure in two domains (OR = 1.43, CI = [1.02, 2.00]), three domains (OR = 1.73, CI = [1.16, 2.59]), or four or more domains (OR = 1.57, CI = [1.05, 2.39]) domains had increased odds of obesity compared with participants who were not in the top quintile of any domain (Table 4; Fig. 1).

Exploratory Analyses

We examined race/ethnicity as a possible effect modifier for each stress–weight association in individual models, adjusting for the same covariates as described above. We did not observe any significant interactions.

In the respective individual models, interaction terms between sex and neighborhood stressors, relationship stressors, and financial strain were significant, for both overweight and obesity. We conducted sex-stratified analyses for these three individual stressors; high neighborhood stress (OR = 1.17, CI = [1.01, 1.35]), financial strain (OR = 1.48, CI = [1.26, 1.72]), and relationship stress (OR = 1.28, CI = [1.09, 1.52]) were associated with elevated odds obesity for women, but not for men (neighborhood stress: OR = 1.01, CI = [0.83, 1.23]; financial strain: OR = 1.04, CI = [0.83, 1.32]; relationship stress: OR = 0.99, CI = [0.77, 1.28]). Neighborhood stress was associated with lower odds of overweight for men (OR = 0.83, CI = [0.69, 0.99]), but not for women (OR = 1.10, CI = [0.93, 1.35]).

We explored the association between psychosocial stressors and the three classes of obesity: Class I ($n = 580$), Class II ($n = 230$), Class III ($n = 220$);

Table 1 Sample Characteristics of 2,983 Adults from the Chicago Community Adult Health Study, 2002–2003

	Full sample	Stratified by BMI category						<i>p</i> Value
	<i>N</i> = 2,983	Obesity (<i>N</i> = 1,030)		Overweight (<i>N</i> = 965)		Normal or underweight (<i>N</i> = 988)		
	Mean	Mean	<i>SE</i>	Mean	<i>SE</i>	Mean	<i>SE</i>	
Age (years)	42.28	44.81	0.66	44.31	0.73	38.04	0.71	<.001
	<i>n</i>	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	
Education								
Less than high school	753	302	40.11	273	36.25	178	23.64	
High school	729	265	36.35	230	31.55	234	32.10	
Some college	790	283	35.82	240	30.38	267	33.80	
College degree and above	711	180	25.32	222	31.22	309	43.46	<.001
Gender								
Male	1181	347	29.38	465	39.37	369	31.24	
Female	1802	683	37.90	500	27.75	619	34.35	<.0001
Race								
Black	1,184	517	43.67	339	28.63	328	27.70	
Native-born Hispanic	353	139	39.38	117	33.14	97	27.48	
Foreign-born Hispanic	419	149	35.56	186	44.39	84	20.05	
White	1,027	225	21.91	323	31.45	479	46.64	<.001
Income								
less than \$10,000	345	128	37.10	94	27.25	123	35.65	
\$10,000 through \$29,999	843	312	37.01	278	32.98	253	30.01	
\$30,000 through \$49,999	563	180	31.97	190	33.75	193	34.28	
\$50,000 or more	689	228	33.09	230	33.38	231	33.53	
Missing income	543	182	33.52	173	31.86	188	34.62	.07
Partner								
Yes	1,288	463	35.95	465	36.10	360	27.95	
No	1,695	567	33.45	500	29.50	628	37.05	<.001
	Full sample (<i>N</i> = 2,983)	Obesity (<i>N</i> = 1,030)		Overweight (<i>N</i> = 965)		Normal or underweight (<i>N</i> = 988)		<i>p</i> Value
	<i>n</i>	<i>n</i>	%	<i>N</i>	%	<i>n</i>	%	
Children								
Yes	2,027	792	39.07	690	34.04	545	26.89	
No	956	238	24.90	275	28.77	443	46.34	<.001
Working status								
Yes	1,789	607	33.93	586	32.76	596	33.31	
No	1,194	423	35.43	379	31.74	392	32.83	.13
Smoking								
Current smoker	787	232	29.48	235	29.86	320	40.66	
Former smoker	586	223	38.05	202	34.47	161	27.47	
Never smoker	1,610	575	35.71	528	32.80	507	31.49	<.001
Alcohol consumption								
Current drinker	1,791	548	30.60	599	33.45	644	35.96	
Former drinker	617	259	41.98	187	30.31	171	27.71	
Never drinker	575	223	38.78	179	31.13	173	30.09	<.001

Differences by BMI categories were calculated using chi-squared analysis (categorical variables) and analysis of variance (continuous variables). *SE* standard error.

Table 2 Distribution of Each Stress Domain for the Full Sample and by BMI Categories, Chicago Community Health Study ($N = 2,983$, 2002–2003)

	Full sample (<i>N</i> = 2,983)		Obesity (<i>N</i> = 1,030)		Overweight (<i>N</i> = 965)		Normal or underweight (<i>N</i> = 988)		<i>p</i> Value
	Mean	<i>SE</i>	Mean	<i>SE</i>	Mean	<i>SE</i>	Mean	<i>SE</i>	
Stressors									
Childhood adversities	−0.02	0.02	0.10	0.04	0.01	0.04	−0.17	0.04	<.001
Acute life events	−0.08	0.02	0.08	0.04	−0.14	0.04	−0.15	0.04	<.001
Financial	−0.11	0.02	0.01	0.04	−0.15	0.04	−0.18	0.04	<.001
Relationship	−0.01	0.03	0.21	0.04	−0.02	0.04	−0.20	0.04	<.001
Life discrimination	−0.02	0.03	0.02	0.04	−0.06	0.04	−0.02	0.04	.46
Job discrimination	0.00	0.02	0.00	0.04	−0.02	0.04	0.02	0.04	.77
Neighborhood	−0.12	0.04	−0.02	0.05	−0.20	0.05	−0.13	0.05	.04
Employment	0.02	0.02	−0.03	0.04	0.04	0.04	0.05	0.04	.14
Number of stressors	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	<i>N</i>	%	
0	851	31.76	247	29.02	284	33.37	320	37.60	
1	847	27.91	277	32.70	281	33.18	289	34.12	
2	542	17.65	190	35.06	179	33.03	173	31.92	
3	375	11.84	163	43.47	112	29.87	100	26.67	
4+	368	10.84	153	41.58	109	29.62	106	28.80	.002

Continuous stress *z* scores were presented. Data were weighted to reflect sample population. The cumulative stress score reflects the number of domains in which the individual was in the top quintile of stress exposure. The score ranges from 0 to 8, and was categorized as 0, 1, 2, 3, or 4 or more.

Supplementary Tables 1 and 2). Greater childhood adversity was associated with higher odds of having Class II obesity (OR = 1.30, CI = [1.07, 1.57]). Greater acute life events were associated with higher odds of having Class I (OR = 1.17, CI = [1.01, 1.35]) and Class III obesity (OR = 1.30, CI = [1.08, 1.56]). Greater financial strain was associated with higher odds of having Class I (OR = 1.20, CI = [1.03, 1.38]), Class II (OR = 1.38, CI = [1.14, 1.67]), and Class III obesity (OR = 1.53, CI = [1.30, 1.82]). Greater lifetime discrimination was associated with higher odds of having Class II obesity (OR = 1.21, CI = [1.00, 1.48]). Greater neighborhood stress was associated with higher odds of having Class III obesity (OR = 1.25, CI = [1.04, 1.52]). Greater relationship stress was associated with higher odds of having Class III obesity (OR = 1.34, CI = [1.09, 1.66]). We did not observe a significant associations between job discrimination or employment stressors and any of the different classes of obesity. Participants who were in the top quintile of exposure in one domain (OR = 1.90, CI = [1.15, 3.14]), two domains (OR = 2.52, CI = [1.42, 4.48]), three domains (OR = 3.08, CI = [1.68, 5.66]), or four domains (OR = 2.00, CI = [0.99, 4.02]) had increased odds of having Class III obesity compared with participants who were not in the top quintile of any domain. In addition, participants with high-stress in

four or more domains had roughly double the odds of Class II obesity relative to individuals with zero stressors in the top quintile.

Results of Sensitivity Analyses

There were no material changes to results after excluding individuals who were underweight, and after excluding individuals who reported “other” race/ethnicity. This suggests that inclusion of these groups within the normal weight and White category, respectively, did not influence the results.

Discussion

This study exams a variety of psychosocial stressors, individually and together, in relation to overweight and obesity for non-Hispanic Black, Hispanics/Latino and non-Hispanic White adults, using data from a large representative sample of Chicago, IL residents. When considered in separate models, greater exposure to childhood adversity, acute life event, financial strain, and relationship stress was associated with increased odds of obesity. The odds ratios for these stressors ranged

Table 3 Relationship Between Stressor and Overweight/Obesity of Adults

Stressors	Overweight vs. normal/under weight				Obesity vs. normal/under weight				
	Model 1A (separate multinomial model for each stressor)				Model 2A (single multinomial model with all stressors included)				
	OR	95% CI	p Value	OR	95% CI	p Value	OR	95% CI	p Value
Childhood adversities	1.04	0.92–1.17	.52	1.01	0.90–1.15	.99	1.16	1.03–1.30	.02
	0.99	0.87–1.13	.91	0.95	0.82–1.10	.48	1.18	1.04–1.34	.01
Acute life events	1.16	1.02–1.33	.03	1.17	1.00–1.35	.046	1.30	1.15–1.48	<.0001
Financial	1.00	0.86–1.16	.99	0.99	0.84–1.15	.85	1.18	1.04–1.35	.01
Relationship ^a	1.02	0.90–1.16	.79	1.01	0.87–1.18	.88	1.12	0.98–1.27	.09
Life discrimination	1.00	0.86–1.15	.91	0.97	0.83–1.15	.88	1.07	0.94–1.22	.32
Job discrimination ^b	0.95	0.85–1.05	.42	0.94	0.83–1.07	.37	1.10	0.98–1.23	.11
Neighborhood	1.05	0.87–1.27	.61	1.03	0.84–1.26	.80	1.05	0.87–1.27	.59
Employment ^b									

All models controlled for age, education, gender, income, race, smoking, and drinking; Model 1: separate models for each stressor, Model 2: single models including all stressor domains simultaneously. Data were weighted and models accounted for sample cluster.

^aAdjusted for age, education, gender, income, race, smoking, drinking, employment status, spouse/partner, and whether the respondent has one or more children.

^bAdjusted for age, education, gender, income, race, smoking, drinking, and employment status. Data are weighted and models account for sample cluster.

between 1.15 and 1.30, suggesting similar magnitudes of associations. We also found evidence that cumulative exposure to high levels of stress across multiple domains was associated with increased odds of obesity, with odds of obesity more than 60% higher for those experiencing high levels of stress in four or more domains compared to none. With the exception of financial strain, we did not observe associations between stressors and overweight, for individual stressors, or combined.

Our findings are consistent with previous studies which have found that childhood maltreatment [42], adverse life events [43], financial strain [20, 21], and poor social relationships were associated with weight gain over time [22, 44]. For example, Kershaw et al. [22] found that chronically high negative ties with family and friends, and increases in negativity, were associated with increased waist circumference over a 10-year period among Whites and Blacks aged 33–45 years. Contrary to previous studies, we did not find an association between lifetime discrimination and obesity. Longitudinal studies have found that individuals who had experienced weight discrimination were more likely to become obese or remain obese over time [17, 45]. Another study found that general everyday unfair treatment predicted increases in central adiposity (measured by waist circumference) over time, even after accounting for the other stressors (e.g., neighborhood disorder and family stress). In our study, lifetime discrimination captured both major acute types of discrimination and chronic everyday discrimination. While this construct attempted to comprehensively assess multiple forms of discrimination, future studies should replicate these findings by assessing the discrimination measures individually, as well as examine whether the associations with overweight/obesity are contingent upon attributions (e.g., weight discrimination vs. racial/ethnic discrimination).

We examined the influence of each stressor, while controlling for other sources of stress. Financial strain remained significantly associated with the risk of overweight and obesity, even after adjusting for co-occurring stressors. Those who are financially strained are less likely to have healthy food options or engage in health promoting behaviors (e.g., physical activity) [21, 46, 47]. People experiencing financial strain report greater depressive and anxiety disorder, which can increase their risk of obesity. Greater financial strain is also associated with decreased self-reported sleep quality [48, 49]. There is compelling evidence that reduced sleep and poor sleep quality predict the development of obesity [50–52]. Future research should examine potential psychological and behavioral pathways that may link financial strain to obesity.

It is important not to discount the potential adverse effects of the other stressors not found in our study

Table 4 Relationship Between Cumulative Stressors and Obesity

Number of stressors	Overweight vs. normal/under weight			Obesity vs. normal/under weight		
	OR	95% CI	<i>p</i> Value	OR	95% CI	<i>p</i> Value
0 (ref)						
1	1.05	0.79–1.39	.74	1.16	0.86–1.56	.32
2	1.11	0.80–1.55	.52	1.43	1.02–2.00	.04
3	1.03	0.68–1.58	.89	1.73	1.16–2.59	.01
4+	1.02	0.67–1.55	.95	1.59	1.05–2.39	.03

Models adjusted for adjusted for age, education, gender, income, race, physical activity, smoking, drinking, diet, employment status, spouse/partner, and whether the respondent has one or more children. Data were weighted and models accounted for sample cluster. The cumulative stress score reflects the number of domains in which the individual was in the top quintile of stress exposure. The score ranges from 0 to 8, and was categorized as 0, 1, 2, 3, or 4 or more.

Fig. 1. Forest plots of the association between cumulative stress and overweight/obesity.

to be independently associated with obesity (i.e., job discrimination, neighborhood stress, and employment stress). One psychosocial stressor can lead to stress proliferation, that is, stress exposure in one domain increases the risk of facing additional adversities [53]. For example, being fired from one's job can lead to financial strain and relationship stress. The accumulation of these stressors across the lifespan significantly increases the risk for disease [54]. It may be that financial strain mediates the relationship between the other stressors and overweight/obesity; this could be explored in depth in

future research. Using the cumulative stress score allowed us to assess the obesogenic influence of multiple stressor exposure. We found that individuals with 2, 3, and 4 or more high stressors had increased odds of overweight and obesity compared with individuals who reported no high exposure to the stressor domains. These findings suggest that cumulative exposure to stressors may place individuals at increased risk for overweight and obesity. Future research should examine the potential biobehavioral and psychological pathways by which the individual stressors as well as cumulative stress operate

to influence the onset and progression of overweight and obesity over time.

In our exploratory analyses, we did not find evidence of variation in the association between psychosocial stress and overweight and obesity by racial/ethnic groups. This suggests that while the extent of exposure to these stressors may vary between groups, the obesogenic influence of these stressors may be similar. It is unknown whether the pathways between stressors and obesity are similar across groups. For example, racial/ethnic groups may differ in the way they cope with stress. Approaches to coping with stress may reduce or exacerbate disparities attributable to differences in health behaviors. Therefore, it is important to assess cultural and contextual determinants of stress coping behaviors across racial/ethnic groups. We found some evidence of sex differences in the relationship between stressors and obesity. However, confidence intervals around the point estimates for males and females overlapped, so caution should be taken in interpreting the interactions. Further research is needed to examine the interplay between stressors, gender, and obesity.

We explored the association between the stressors and the three classes of obesity. There were more individual stressors associated with Class III obesity compared with the other classes of obesity. Moreover, individuals who were in the top quintiles across 1, 2, 3, or ≥ 4 had increased odds of having Class III obesity. These findings highlight the potential value of segmenting obesity into classes as assessing them as a whole may obscure important relationships. Future research should replicate these findings with adequate sample sizes to better understand the associations between the stressors and the three classes of obesity. Although this study provides evidence of association between stressors and obesity, the cross-sectional nature of the data precludes us from establishing a causal link between stressors and obesity, or examining potential pathways (e.g., diet or physical activity). It may be that individuals who are obese are exposed to more psychosocial stressors than individuals who are normal weight. For example, individuals who are obese experience weight-related discrimination in the workplace and other social contexts [55]. Future research should seek to replicate these patterns with longitudinal data and examine the extent to which stress exposure contributes to the onset and progression of obesity. If a causal link between stress and obesity is indicated, future research should investigate coping mechanisms, such as eliciting social support or engaging in emotion-focused coping, that may buffer the obesogenic effects of stress among those burdened by high stress exposure [56].

This study used a single measure of adiposity (i.e., BMI) to assess overweight and obesity. Although BMI

does not capture total body fat or fat distribution, BMI is a reliable and valid measure that is commonly used for identifying adults at increased risk of overweight- and obesity-related morbidity and mortality [57–59]. Nevertheless, further research using additional measures, such as tomography scans (to measure visceral and subcutaneous fat), may reveal additional insight into patterns in the stress–obesity relationship.

The data from this cross-sectional study were collected between 2001 and 2003. The prevalence of obesity nationally has increased since the conclusion of data collection. Based on the National Health and Nutrition Examination Survey (NHANES), the prevalence of obesity in 2003–2004 was 32.2, with 4.8 of individuals having severe obesity [2]. The most recent study using NHANES reveal that during 2015–2016, the prevalence of obesity rose to 39.6, with 7.7 of individuals having severe obesity [3]. Therefore, it is ever more important to continue assessing the contemporary impact of psychosocial stressors on the onset and progression of obesity. To the best of our knowledge, there have not been major initiatives on stress management at a national or state level during this period. However, there have been policies that target certain psychosocial stressors and may be promising areas for potential intervention at those levels. For instance, financial strain can potentially be reduced by increasing wages for workers. Increasing wages can allow individuals and their families to afford healthy foods and medical care, pay bills, and live in better quality neighborhoods, and, therefore, reduce financial burden. Some research finds that higher wages correlate with lower obesity prevalence [60, 61]. In fact, one study using the Behavioral Risk Factor Surveillance System (1984–2006) found that for one dollar increase in the minimum wage was associated with a 0.06 decrease in mean BMI for men and women [61]. While wage increase can be a national level policy that can be implemented to assist those most vulnerable to financial strain, other national or state-level programs that target other stressors (e.g., discrimination, childhood adversity) should be considered within the context of obesity prevention. Programs that facilitate social capital (e.g., neighborhood cohesion and social support), for example, may help buffer the obesogenic effects of multiple stressors given that they provide the necessary means to goods and resources [62]. This study is strengthened by incorporation of a wide range of psychosocial stressors, assessed individually and together in relation to overweight and obesity. Within most stress domains, multiple measures were included, which helped capture different dimensions of the respective stressor. For example, the relationship stressor domain contained five measures that assessed the quality of different social relationships, ranging from marital stress

to child-related stress. The large representative sample of Chicago residents allowed us to test for differences across racial/ethnic groups. One strength of the CCAHS is the roughly equal number of foreign- and native-born Hispanics, which we categorized separately in our race/ethnicity variable.

Conclusion

Our study extends the existing evidence on the role psychosocial stress may play in obesity risk. Although we did not observe racial/ethnic differences in the relationship between psychosocial stress and overweight and obesity, psychosocial stress may play an important role in racial/ethnic obesity disparities, given higher exposure to stressors among minorities compared to Whites [24, 63]. Some evidence of sex differences in the relationship of psychosocial stressors to obesity was found, and could be explored in further research. Prospective studies are needed to understand the direction of these associations and to identify the pathways by which these stressors contribute to the development of obesity. If replicated, these findings would call for rigorous analyses of the extent to which reducing exposure to multiple forms of stress could contribute to population-level improvements in BMI.

Supplementary Material

Supplementary data are available at *Annals of Behavioral Medicine* online.

Acknowledgements This project was partially supported by the National Institute of Health (3R25CA057711). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH. The development of the article was partially supported by Cancer Disparities Research Network/Geographic Management Program (GMAP) Region 4 funded by 3 P30 CA006927-52S2 and CTSI Mentored Career Development Award (KL2 TR002545).

Compliance with Ethical Standards

Authors' Statement of Conflict of Interest and Adherence to Ethical Standards The authors declare that they have no conflict of interest.

Authors' Contributions All authors contributed to the conception and study design. R.C. conducted the analysis of data, with all authors providing interpretation of the results. A.G.C. wrote the original draft. All other authors revised and edited the article and approved the final version.

Ethical Approval Ethical approval was obtained from Tufts University's Institutional Review Board.

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. Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA*. 2006;295:1549–1555.
3. Hales CM, Fryar CD, Carroll MD, Freedman DS, Ogden CL. Trends in obesity and severe obesity prevalence in US youth and adults by sex and age, 2007–2008 to 2015–2016. *JAMA*. 2018;319:1723–1725.
4. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: A systematic review and meta-analysis. *BMC Public Health*. 2009;9:88.
5. Calle EE, Kaaks R. Overweight, obesity and cancer: Epidemiological evidence and proposed mechanisms. *Nat Rev Cancer*. 2004;4:579–591.
6. Vucenik I, Stains JP. Obesity and cancer risk: Evidence, mechanisms, and recommendations. *Ann N Y Acad Sci*. 2012;1271:37–43.
7. Hruby A, Hu FB. The epidemiology of obesity: A big picture. *Pharmacoecon*. 2015;33:673–689.
8. Zilanawala A, Davis-Kean P, Nazroo J, Sacker A, Simonton S, Kelly Y. Race/ethnic disparities in early childhood BMI, obesity and overweight in the United Kingdom and United States. *Int J Obes (Lond)*. 2015;39:520–529.
9. Tomiyama AJ. Stress and obesity. *Annu Rev Psychol*. 2019;70:703–718.
10. Ouakinin SRS, Barreira DP, Gois CJ. Depression and obesity: Integrating the role of stress, neuroendocrine dysfunction and inflammatory pathways. *Front Endocrinol (Lausanne)*. 2018;9:431.
11. Hammen C. Stress and depression. *Annu Rev Clin Psychol*. 2005;1:293–319.
12. Luppino FS, de Wit LM, Bouvy PF, et al. Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Arch Gen Psychiatry*. 2010;67:220–229.
13. Scott KA, Melhorn SJ, Sakai RR. Effects of chronic social stress on obesity. *Curr Obes Rep*. 2012;1:16–25.
14. Stults-Kolehmainen MA, Sinha R. The effects of stress on physical activity and exercise. *Sports Med*. 2014;44:81–121.
15. Laugero KD, Falcon LM, Tucker KL. Relationship between perceived stress and dietary and activity patterns in older adults participating in the Boston Puerto Rican Health Study. *Appetite*. 2011;56:194–204.
16. Wardle J, Chida Y, Gibson EL, Whitaker KL, Steptoe A. Stress and adiposity: A meta-analysis of longitudinal studies. *Obesity (Silver Spring)*. 2011;19:771–778.
17. Sutin AR, Terracciano A. Perceived weight discrimination and obesity. *PLoS One*. 2013;8:e70048.
18. Thomas C, Hyppönen E, Power C. Obesity and type 2 diabetes risk in midadult life: The role of childhood adversity. *Pediatrics*. 2008;121:e1240–e1249.
19. 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;17:1900–1905.
20. Siahpush M, Huang TT, Sikora A, Tibbits M, Shaikh RA, Singh GK. Prolonged financial stress predicts subsequent obesity: Results from a prospective study of an Australian national sample. *Obesity (Silver Spring)*. 2014;22:616–621.
21. Conklin AI, Forouhi NG, Brunner EJ, Monsivais P. Persistent financial hardship, 11-year weight gain, and health behaviors in the Whitehall II study. *Obesity (Silver Spring)*. 2014;22:2606–2612.

22. Kershaw KN, Hankinson AL, Liu K, et al. Social relationships and longitudinal changes in body mass index and waist circumference: The coronary artery risk development in young adults study. *Am J Epidemiol*. 2014;179:567–575.
23. 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.
24. Sternthal MJ, Slopen N, Williams DR. Racial disparities in health: The impact of multi-dimensional measures of race/ethnicity on the self-reported health status of Latinos. *Bois Rev Soc Sci Res Race*. 2011;8:95–113.
25. Slopen N, Williams DR. Discrimination, other psychosocial stressors, and self-reported sleep duration and difficulties. *Sleep*. 2014;37:147–156.
26. Katz MJ, Derby CA, Wang C, et al. Influence of perceived stress on incident amnesic mild cognitive impairment: Results from the Einstein Aging Study. *Alzheimer Dis Assoc Disord*. 2016;30:93–98.
27. Slopen N, Non A, Williams DR, Roberts AL, Albert MA. Childhood adversity, adult neighborhood context, and cumulative biological risk for chronic diseases in adulthood. *Psychosom Med*. 2014;76:481–489.
28. Lawless MH, Harrison KA, Grandits GA, Eberly LE, Allen SS. Perceived stress and smoking-related behaviors and symptomatology in male and female smokers. *Addict Behav*. 2015;51:80–83.
29. Sacco P, Bucholz KK, Harrington D. Gender differences in stressful life events, social support, perceived stress, and alcohol use among older adults: Results from a National Survey. *Subst Use Misuse*. 2014;49:456–465.
30. Kaufman A, Augustson EM, Patrick H. Unraveling the relationship between smoking and weight: The role of sedentary behavior. *J Obes*. 2012;2012:735465.
31. Traversy G, Chaput JP. Alcohol consumption and obesity: An update. *Curr Obes Rep*. 2015;4:122–130.
32. Kassel JD, Stroud LR, Paronis CA. Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychol Bull*. 2003;129:270–304.
33. Parrott AC. Does cigarette smoking cause stress? *Am Psychol*. 1999;54:817–820.
34. Hunte HE, Williams DR. The association between perceived discrimination and obesity in a population-based multiracial and multiethnic adult sample. *Am J Public Health*. 2009;99:1285–1292.
35. Hicken MT, Lee H, Morenoff J, House JS, Williams DR. Racial/ethnic disparities in hypertension prevalence: Reconsidering the role of chronic stress. *Am J Public Health*. 2014;104:117–123.
36. Mayor E. Gender roles and traits in stress and health. *Front Psychol*. 2015;6:779.
37. Matud MP. Gender differences in stress and coping styles. *Personal Individ Differ*. 2004;37(7):1401–1415.
38. Taylor SE, Klein LC, Lewis BP, Gruenewald TL, Gurung RA, Updegraff JA. Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychol Rev*. 2000;107:411–429.
39. Lavie CJ, McAuley PA, Church TS, Milani RV, Blair SN. Obesity and cardiovascular diseases: Implications regarding fitness, fatness, and severity in the obesity paradox. *J Am Coll Cardiol*. 2014;63:1345–1354.
40. Kitahara CM, Flint AJ, Berrington de Gonzalez A, et al. Association between class III obesity (BMI of 40–59 kg/m²) and mortality: A pooled analysis of 20 prospective studies. *PLoS Med*. 2014;11:e1001673.
41. Preiss K, Brennan L, Clarke D. A systematic review of variables associated with the relationship between obesity and depression. *Obes Rev*. 2013;14:906–918.
42. Danese A, Tan M. Childhood maltreatment and obesity: Systematic review and meta-analysis. *Mol Psychiatry*. 2014;19:544–554.
43. Palmisano GL, Innamorati M, Vanderlinden J. Life adverse experiences in relation with obesity and binge eating disorder: A systematic review. *J Behav Addict*. 2016;5:11–31.
44. Birditt KS, Newton NJ, Cranford JA, Webster NJ. Chronic stress and negative marital quality among older couples: Associations with waist circumference. *J Gerontol B Psychol Sci Soc Sci*. 2016;5:318–328.
45. Tomiyama AJ, Carr D, Granberg EM, et al. How and why weight stigma drives the obesity ‘epidemic’ and harms health. *BMC Med*. 2018;16:123.
46. Conklin AI, Forouhi NG, Suhrcke M, Surtees P, Wareham NJ, Monsivais P. Variety more than quantity of fruit and vegetable intake varies by socioeconomic status and financial hardship. Findings from older adults in the EPIC cohort. *Appetite*. 2014;83:248–255.
47. Carvalho LS, Meier S, Wang SW. Poverty and economic decision-making: Evidence from changes in financial resources at payday. *Am Econ Rev*. 2016;106:260–284.
48. Hall M, Buysse DJ, Nofzinger EA, et al. Financial strain is a significant correlate of sleep continuity disturbances in late-life. *Biol Psychol*. 2008;77:217–222.
49. Hall MH, Matthews KA, Kravitz HM, et al. Race and financial strain are independent correlates of sleep in midlife women: The SWAN sleep study. *Sleep*. 2009;32:73–82.
50. Bayon V, Leger D, Gomez-Merino D, Vecchierini MF, Chennaoui M. Sleep debt and obesity. *Ann Med*. 2014;46:264–272.
51. Zimberg IZ, Dâmaso A, Del Re M, et al. Short sleep duration and obesity: Mechanisms and future perspectives. *Cell Biochem Funct*. 2012;30:524–529.
52. Beccuti G, Pannain S. Sleep and obesity. *Curr Opin Clin Nutr Metab Care*. 2011;14:402–412.
53. Williams DR, Mohammed SA. Racism and health I: Pathways and scientific evidence. *Am Behav Sci*. 2013;57(8).
54. Pearlín LI. The life course and the stress process: Some conceptual comparisons. *J Gerontol B Psychol Sci Soc Sci*. 2010;65B:207–215.
55. Puhl RM, Heuer CA. The stigma of obesity: A review and update. *Obesity (Silver Spring)*. 2009;17:941–964.
56. Folkman S. Stress: Appraisal and coping. In: Gellman MD, Turner JR, eds. *Encyclopedia of Behavioral Medicine*. New York: Springer; 2013:1913–1915.
57. Stein CJ, Colditz GA. The epidemic of obesity. *J Clin Endocrinol Metab*. 2004;89:2522–2525.
58. Rothman KJ. BMI-related errors in the measurement of obesity. *Int J Obes (Lond)*. 2008;32(suppl 3):S56–S59.
59. US Preventive Services Task Force. Screening for obesity in adults: Recommendations and rationale. *Ann Intern Med*. 2003;139(11):930.
60. Kim D, Leigh JP. Estimating the effects of wages on obesity. *J Occup Environ Med*. 2010;52:495–500.
61. Meltzer DO, Chen Z. The impact of minimum wages on body weight in the United States. In: *Economic Aspects of Obesity*. Chicago, IL: University of Chicago Press; 2011:17–34.
62. Carrillo-Álvarez E, Kawachi I, Riera-Romani J. Neighbourhood social capital and obesity: A systematic review of the literature. *Obes Rev*. 2019;20:119–141.
63. American Psychological Association. *Stress in America 2017 Snapshot: Coping with Change*. Washington, DC: American Psychological Association; 2017.