J Urban Health (2017) 94:791–802
DOI 10.1007/s11524-017-0193-7

Independent Effects of Neighborhood Poverty and Psychosocial Stress on Obesity Over Time

Jamila L. Kwarteng · Amy J. Schulz · Graciela B. Mentz · Barbara A. Israel · Denise White Perkins

Published online: 11 September 2017

Abstract The objective of the study was to examine the independent effects of neighborhood poverty and psychosocial stress on increases in central adiposity over time. Data are from a community sample of 157 Non-Hispanic Black, Non-Hispanic White, and Hispanic adults collected in 2002–2003 and 2007–2008, and from the 2000 Decennial Census. The dependent variable was waist circumference. Independent variables included neighborhood poverty, perceived neighborhood physical environment, family stress, safety stress, everyday unfair treatment, and a cumulative stress index. Weighted 3-level hierarchical linear regression models for a continuous outcome were used to assess the effects of neighborhood poverty and psychosocial stress on central adiposity over time. We also assessed whether psychosocial stress mediated the association between neighborhood poverty and central adiposity. Neighborhood poverty and everyday unfair treatment at baseline were independently associated with increases in central adiposity over time, accounting for the other indicators of stress. Perceptions of the neighborhood physical environment and cumulative stress mediated associations between neighborhood poverty and central adiposity. Results suggest that residing in neighborhoods with higher concentrations of poverty and exposure to everyday unfair treatment independently heighten risk of increased central adiposity over time. Associations between neighborhood poverty and central adiposity were mediated by perceptions of the neighborhood physical environment and by the cumulative stress index. Public health strategies to reduce obesity should consider neighborhood poverty and exposure to multiple sources of psychosocial stress, including everyday unfair treatment.

Keywords Central adiposity · Psychosocial stress · Everyday unfair treatment · Perceived discrimination · Neighborhood poverty

J. L. Kwarteng (✉)
Medical College of Wisconsin, Milwaukee, WI, USA
e-mail: jkwarteng@mcw.edu

A. J. Schulz · G. B. Mentz · B. A. Israel
University of Michigan, School of Public Health, Ann Arbor, MI, USA

D. W. Perkins
Henry Ford Health System, Institute on Multicultural Health, Detroit, MI, USA

Introduction

Non-Hispanic Blacks (NHBs), Hispanics, and low-income non-Hispanic Whites (NHWs) in the USA are disproportionately at risk of cardiovascular disease (CVD), diabetes, and some cancers [1]. Research suggests that this disproportionate risk is driven, at least in part, by higher rates of obesity among these racial and income groups [1]. Obesity rates often vary by neighborhood characteristics, with, for example, neighborhood concentrations of poverty associated with heightened risk of obesity [2–5]. Counterintuitively, one recent cross-sectional study found a reduced risk of obesity for African Americans living in poverty, and increased risk among their Whites counterparts [6]. In contrast, a number of studies have reported findings suggesting that neighborhood poverty in urban environments

may be associated with increased risk of obesity through its effects on access to healthy foods [7–9] and by shaping characteristics of the physical environment associated with physical activity [10–13]. These results suggest a need for research that helps us understand more clearly the potential pathways linking neighborhood poverty with increased rates of obesity.

An under-examined pathway between neighborhood poverty with obesity is the role of psychosocial stress [14, 15]. Substantial evidence links neighborhood poverty to greater exposure to psychosocial stress [16–18], and these exposures may mediate the relationship between neighborhood poverty and increased obesity risk. Poverty creates stressful environments where residents have poor access to a number of material resources such as employment opportunities, quality education, and municipal services (e.g., police). This environment is stressful and fosters financial strain, lower socioeconomic status, and higher crime rates [19, 20]. For example, residents of high poverty neighborhoods may experience heightened psychosocial stress from a variety of sources including the physical and social environments they encounter [21–24], family relationships [25, 26], concerns about safety [26], and experiences of discrimination or unfair treatment [27–29].

Psychosocial stress [30–32] is often conceptualized as experiences that are perceived as harmful, threatening, or bothersome [16, 17] to the individual. Psychosocial stress is embedded in social structures, roles, and relationships that persist over time [33]. Physiological responses to psychosocial stress may, over time, lead to changes in metabolic functioning [34] that can influence the distribution of fat in the body [35], particularly in the internal, visceral adipose tissue regions [36–41]. As excess visceral fat accumulates in the abdominal region—called central adiposity—it places individuals at higher risk for certain health outcomes such as cardiovascular disease and diabetes [42]. Therefore, given suggested associations between psychosocial stress and central adiposity, we examine central adiposity, rather than overall body mass index (BMI). In this paper, we focus on exposure to neighborhood poverty, psychosocial stress, and heightened risk for central adiposity.

Relatively few studies have examined the effect of multiple indicators of psychosocial stress on obesity, and only a handful of these examined associations over time. In the prospective Epidemiology of Diabetes Complications (EDC) study, Lloyd and colleagues [43] found no association between self-reported stress and change in waist-to-hip ratio over a 2-year period in either men or women with type I diabetes, adjusting for age, education, and BMI. In contrast, a 13-year prospective study by Fowler-Brown and colleagues [44] in a sample of Black adults found higher levels of perceived stress at baseline predicted a higher percentage increase in BMI over time among women but not men, controlling for age, smoking, education, occupation, and financial strain [44]. Finally, Block and colleagues, in a 9-year prospective study that included Black, White, and Hispanic participants, found that job-related demands and difficulty paying bills were associated with weight gain among both women and men [45]. In addition, among women, perceived constraints in life and strains in family relationships were associated with greater weight gain, as were lack of skill discretion and lack of decision authority at work among men [45]. Together, these findings generally suggest that exposure to stress may be associated with increases in obesity over time, with some variation. However, there are notable gaps in this body of research. First, none of these studies accounted for neighborhood level poverty, and the role it may play in shaping exposure to multiple sources of psychosocial stress. Similarly, studies have not considered the question of whether psychosocial stress may mediate associations between neighborhood poverty and central adiposity, a key question in establishing this pathway.

To address these gaps in the literature, this study examined the influence of neighborhood poverty and multiple indicators of psychosocial stress on increases in obesity over time. Individuals who live in neighborhoods with higher concentrations of poverty may experience heightened levels of multiple types of psychosocial stress [46, 47]. To the extent that psychosocial stress results in physiological responses that modify metabolic functioning, these exposures may shape trends in central adiposity over time, independent of effects on weight or body mass index (BMI). To disentangle the extent to which the effects of neighborhood poverty on central adiposity may be mediated by psychosocial stress, our models control for health-related behaviors (e.g., diet, physical activity) and weight (and thus BMI) that may also be influenced by neighborhood poverty through pathways distinct from the physiological effects of stress on metabolic functioning and central adiposity. Understanding the contributions of neighborhood poverty and psychosocial stress to risk of obesity will help to identify more clearly the factors contributing to racial inequities in obesity risk, and inform interventions that can promote more equitable health outcomes.

Hypotheses

In order to assess the broader research questions outlined above, we tested a series of specific hypotheses related to the independent effects of neighborhood poverty and psychosocial stress on obesity over time. Specifically, we tested the following hypotheses.

1. Residents of neighborhoods with higher levels of poverty will experience greater increases in obesity over time than residents of neighborhoods with lower levels of poverty.
2. Neighborhood poverty and psychosocial stress will be independently and positively associated with obesity over time.
3. Associations between neighborhood poverty and obesity will be mediated by psychosocial stress.

Methods and Procedures

Data

Healthy Environment Partnership Surveys This paper used a prospective 6-year follow up design and drew upon data from three sources: The Healthy Environments Partnership (HEP) community surveys, Wave I 2002–2003 and Wave II 2007–2008, and the 2000 Decennial Census [11]).

The Healthy Environments Partnership is a community-based participatory research partnership made up of community-based organizations, health service providers, and academic partners. HEP members worked together to design the HEP Wave I Community Survey [11], which was conducted in 2002–2003. The survey was based on a stratified two-stage probability sample of occupied housing units in Detroit, designed for 1000 completed interviews with NHB, NHW, and Hispanic adults aged ≥ 25 years. At each household unit, a listing of eligible residents was completed, and one eligible adult was selected randomly for inclusion in the study. Of the 2517 housing units in the initial sample, 1297 were invalid (e.g., vacant), unable to be screened after repeated attempts (i.e., 12+ attempts), or contained no eligible respondent. The final sample consisted of 919 people: face-to-face interviews were completed with 75% of households in which an eligible respondent was identified (919 of 1220), 55% of households with a known or potential respondent (919 of 1663), and 90% of households in which an eligible respondent was contacted (919 of 1027) [11].

The 2008 HEP Wave II community survey followed up on the 2002–2003 survey and included re-interviews with 219 of the 2002–2003 survey respondents, as well as new residents of the same housing units included in the 2002 sample (n = 241). For this paper, we analyzed data from the sample of 219 participants interviewed at both baseline (2002–2003) and follow-up (2007–2008).

Measures

The *dependent variable* was a continuous measure of *waist circumference* in centimeters, assessed at two time points: 2002–2003 and 2007–2008. *Independent variables* included measures at both the individual and the neighborhood levels, derived from the HEP surveys and 2000 Decennial Census, respectively.

Independent variables derived from the 2002–2003 HEP Wave I survey are self-reported *neighborhood physical environment*, a mean scale of seven items (e.g., "My neighborhood has a lot of vacant lots or vacant houses."), with higher scores indicating a more positive ranking of neighborhood environment (range 1 = strongly agree to 5 = strongly disagree) (Cronbach's alpha = 0.69) ([18]). *Family stress* is a mean scale of three items (e.g., "Did problems experienced by a parent or other relative put extra burden on you?") (range 1 = never to 5 = always) (Cronbach's alpha = 0.63) ([18]). *Safety stress* is a mean scale of three items (e.g., "How often did you worry about your safety in your home?") (range 1 = never to 5 = always) (Cronbach's alpha = 0.85) ([18]). *Everyday unfair treatment* was constructed as a mean scale of five items (e.g., "How often were you treated with less courtesy or respect than other people in the previous 12 months?") (range 1 = never, 5 = always) (Cronbach's alpha = 0.77) [48].

Finally, because individuals often experience more than one type of psychosocial stress simultaneously, a composite indicator summing each of these individual stressors was created. The *cumulative stress index* (CSI) was created by dichotomizing the scores for each of the above indicators of psychosocial stress, scored as 0 for those falling below the median and 1 for values at and above the median. Scores were summed with a high score indicating a greater number of indicators of stress above the median cut point (range = 0–17).

Controls consisted of a dummy variable representing *time* (0 = 2002, 1 = 2008), *age* (years), *gender* (1 = female, 0 = male); *self-reported race/ethnicity* (Non-Hispanic Black = referent, non-Hispanic White, and Hispanic); *education* (< 12 years, 12 years, ≥ 12 years = referent); the *ratio of income to poverty* was calculated by dividing the household income by the federal poverty threshold for the related family size, with the resulting ratios dichotomized (1 = households at or below the poverty line, 0 = households above the poverty line) [49]; *marital status* (1 = married, 0 = single, widowed, or divorced); *car ownership* (1 = owns or leases car, 0 = no car); and *home ownership* (1 = owns home, 0 = does not own home), and *BMI* (continuous). We allowed age and BMI to vary over time, while the other controls where invariant over time. *Alcohol intake* was constructed by mean daily frequency intake of alcoholic beverages reported on the modified Block 98 questionnaire [50]: beer, red wine, wine, and liquor. For the four alcoholic beverages, reported intake frequencies, ranging from never to every day, were converted into the number of drinks per month ranging from 0 to 300. Because the variable was skewed, with 50% indicating zero drinks in the last month, the variable was converted to a binary variable that represents individuals with less than one drink per month = 0 and individuals with one or more drinks per month = 1. *Current*, *never*, or *former smoker* (e.g., "Do you currently smoke cigarettes") was constructed by using the self-report of whether the individual never smoked, currently smoked, or formerly smoked (1 = current, 0 = never, 2 = former) [51]. The *healthy eating index* (HEI) was constructed by taking the sum of mean daily frequency of intake of foods that consist of grains, meat, milk, vegetables, fruit, fat, saturated fat, sodium, and cholesterol reported on the modified Block 98 semi-quantitative food frequency questionnaire [52]. For the ten food categories, reported intake frequencies, ranging from never to six or more times per day, were converted to daily frequencies using the following weights: "never or less than once a month" = 0, "1–3 times a month" = 0.1, "4–6 times a month" = 5/7, "1 time every day" = 1, "2–3 times every day" = 3, "4–5 times every day" = 5, and "6 or more times every day" = 6. The value of the ten items was summed. The final modified-HEI ranged from 0 to 90, with a higher number representing greater consumption of healthy foods. Physical activity (PA) was measured by asking how many days and the amount of time an individual reported moderate-intensity activities (vacuuming, gardening, or anything else that causes small increases in breathing or heart rate) or vigorous activities (such as fast walking, running, dancing, or participating in strenuous sports that cause large increases in breathing or heart rate) in a usual week for at least 10 min at a time [53]. *Metabolic equivalent of task* (MET) minutes of PA per week were calculated for participants for whom data were available: the frequency and duration of physical activity [54] was scaled (divided) by the standard deviation to create a standardized PA score (range 0–4.2), utilizing guidelines based on the International Physical Activity Questionnaire [55, 56].

Neighborhood Level Independent Variables The time-invariant independent variable *neighborhood percent poverty* (i.e., percent poverty) was derived from the 2000 census. Because previous studies have found that associations between neighborhood poverty and health outcomes are not necessarily linear [17], this variable was categorized into quartiles of poverty at the census block group level: Quartile 1 = 0–20% of households below poverty, Quartile 2 = 20–30%, Quartile 3 = 30–40%, and Quartile 4 > 40% of households below poverty. The use of quartiles enabled the neighborhood poverty variable to be included as dummy variables in models, allowing for non-linear associations with the dependent variable.

Analysis

Preliminary exploratory data analysis techniques were used to assess the distribution of dependent variables and to determine if transformations were needed to address a possible lack of normality. Histograms, QQ plots, and Box-plot were examined. Means, medians, and standard deviations were calculated. Based on findings from those assessments, no transformations were used. Weighted 3-level hierarchical linear regression models for a continuous outcome were estimated to account for both the longitudinal and nested structure of the data. Individuals who reported that they were pregnant or breastfeeding (n = 23), and those missing a measure for waist circumference (n = 60) were removed from the analysis. In addition, since hierarchical linear modeling (HLM) can only handle balanced data for the time varying measures, individuals with missing data were removed from the analysis (n = 5 individuals). Further, block groups that no longer contained individuals at time 2 were excluded from the analyses (n = 13).

The final models included the remaining 314 repeated measures (level 1), nested in 157 individuals (level 2), and 56 census block groups (level 3).

To examine hypothesis 1, residents of neighborhoods with higher levels of poverty will experience greater increases in obesity over time; neighborhood percent poverty measure was included at level 3. At level 2, models included non-time changing varying covariates such as gender, race/ethnicity, education, ratio of income to poverty, marital status, car ownership, home ownership, alcohol intake, smoking, HEI, and METs. Level 1 included time-changing measures such as a time indicator dummy variable (0 = baseline and 1 = follow up), age, and BMI (Model 1).

Model 1
Level 1 (repeated measures): waist circumference$_{ijk}$ = β_{000+} β_{100*}Age$_{ijk+}$ β_{200*}BMI$_{ijk+}$ β300*Time$_{ijk}$
Level 2 (individual): $\beta_0 = \gamma_{000} + \gamma_{010}$ * Female$_{jk}$ + γ_{020} * Hispanic$_{jk}$ + γ_{030} * White$_{jk}$ + …
Level 3 (neighborhood): $\gamma 000 = \delta_{000} + \delta_{001}$ * Poverty$_k$

Variables in the models were grand mean centered.

To test hypothesis 2, whether poverty and psychosocial stress were associated with obesity over time, each individual indicator of psychosocial stress and the cumulative stress index was added separately to level 2 (Model 2–6) in two different settings: (1) as a time-invariant covariate and (2) as a time-varying covariate. Although patterns were similar, the coefficients for time-varying covariates where not significant, thus we report only the time-invariant covariates.

Models 2–6 tested the hypothesis that each indicator of psychosocial stress will be independently associated with central adiposity over time: self-reported neighborhood physical environment, family stress, safety stress, everyday unfair treatment, and cumulative stress index (CSI).

Model 2: Model 1 + neighborhood physical environment
Model 3: Model 1 + family
Model 4: Model 1 + safety
Model 5: Model 1 + everyday unfair treatment
Model 6: Model 1 + CSI

Finally, we analyzed the mediating effect of indicators of psychosocial stress on the association between neighborhood percent poverty and central adiposity, using the recommendation of Zhang and colleagues [57] for testing multilevel models and avoid confounding. Hierarchal linear models were analyzed with group mean centered independent variables to decompose between-group from within group variation in model 7–11. We used test statistics proposed by Freedman and Schatzkin [58], to examine the difference in point estimates for neighborhood percent poverty with and without the mediator. The formula can be written as follows:

$$t_{N-2} = \frac{c - c'}{\sqrt{\sigma_c^2 - \sigma_{c'}^2 - 2\sigma_c\sigma_{c'}\sqrt{1-\rho_{xm}}}}$$

where ρ_{xm} refers to the correlation between the independent variable X and the mediator M.

Results

The sample demographics are illustrated in Table 1. The mean age at baseline was 49.1 (s.e. = 0.82) years, mean self-reported neighborhood physical environment was 3.1 (s.e. = 0.04), mean family stress was 2.0 (s.e. = 0.03), mean safety stress was 2.5 (s.e. = 0.13), mean everyday unfair treatment was 1.6 (s.e. = 0.00), mean cumulative stress index was 5.9 (s.e. = 0.16), and mean waist circumference was 98.9 cm (s.e. = 0.90) at baseline. In addition, the mean neighborhood percent poverty was 31.3 (s.e. = 10.94).

Is Neighborhood Poverty Associated with Change in Central Adiposity Over Time?

Table 2, Model 1, shows results from multilevel regression models testing the hypothesis that neighborhood poverty was associated with change in central adiposity over time, accounting for individual level demographics and behavioral controls. Participants who lived in neighborhoods in with poverty levels between 20 and 30% (second quartile) (β = 3.79, p = 0.025) or with poverty levels between 30 and 40% (third quartile) (β = 3.73, p = 0.024) had greater increases in central adiposity over time, compared with those in neighborhood with less than 20% poverty (the lowest poverty quartile).

Table 1 Weighted baseline descriptive characteristics

	Mean ± SE	Percent	Range
Individual (Levels 1 and 2)			
Age	49.1 ± 0.82		26.0–87.0
Gender, %Female		51	
Race/Ethnicity, %			
White		22	
Black		45	
Hispanic		31	
Marital status, %Married		32	
Education attainment, %			
Less than high school		42	
High school		24	
Beyond high school		33	
Below poverty		36	
Car ownership		73	
Home ownership		67	
Alcohol use		47	
Currently smoking		39	
Healthy eating index	64.6 ± 0.37		32.0–88.0
Physical activity (METs)	1.0 ± 0.04		0.0–4.2
Waist circumference	98.9 ± 0.90		72.0–141.0
Body mass index	32.0 ± 0.50		17.5–57.9
Neighborhood physical environment	3.1 ± 0.04		1.4–5.0
Family stress	2.0 ± 0.03		1.0–5.0
Safety stress	2.5 ± 0.13		1.0–5.0
Everyday unfair treatment	1.6 ± 0.00		1.0–3.6
Cumulative stress index	5.9 ± 0.16		0.0–17.0
Block group (Level 3)			
Percent poverty	31.3 ± 10.94		8.0–54.0
Quartile 1		18	
Quartile 2		29	
Quartile 3		32	
Quartile 4		21	

Are Neighborhood Poverty and Psychosocial Stress Jointly Associated with Increases in Central Adiposity Over Time?

Table 2, Models 2–6, shows results from multilevel regression analyses testing whether each of the indicators of psychosocial stress (i.e., neighborhood physical environment, family stress, safety stress, everyday unfair treatment) was independently associated with central adiposity, after accounting for neighborhood poverty, and demographic and behavioral controls. Models show no association between neighborhood physical environment ($\beta = -0.01$, $p = 0.985$) (Model 2), family stress ($\beta = -0.11$, $p = 0.860$) (Model 3), or safety stress ($\beta = 0.65$, $p = 0.170$) (Model 4) and change in central adiposity over time. Everyday unfair treatment was positively associated with change in central adiposity over time ($\beta = 2.36$, $p = 0.020$) above and

Table 2 Waist circumference regressed on multiple indicators of psychosocial stress, accounting for demographic variables and health-related behaviors

N = 157	Model 1		Model 2		Model 3		Model 4		Model 5		Model 6	
	β	SE	β	SE	β	SE	β	SE	β	SE	β	SE
Intercept	105.58	2.34	106.99	2.36	105.52	2.31	105.67	2.34	105.55	2.28	107.32	2.18
Level 3 (BG)												
Poverty Quartile 2	3.79*	1.65	2.44	1.76	3.86*	1.71	3.78*	1.66	3.62*	1.70	2.87	1.62
Poverty Quartile 3	3.73*	1.61	1.80	1.83	3.82*	1.66	3.54*	1.61	3.64*	1.76	1.83	1.70
Poverty Quartile 4	3.15	1.73	1.05	2.07	3.21	1.74	3.43	1.76	3.61*	1.78	1.56	1.79
Neighborhood physical environment			2.86	1.74								
Family stress					0.37	1.34						
Safety stress							0.82	1.22				
Everyday unfair treatment									−1.62	2.22		
Cumulative stress index											0.65	0.53
Levels 1 and 2 (individual) baseline and follow-up												
Neighborhood physical environment			−0.01	0.56								
Family stress					−0.11	0.65						
Safety stress							0.65	0.47				
Everyday unfair treatment									2.36*	1.00		
Cumulative stress index											0.28	0.14
Sigma square	16.55	2.09	16.56	2.09	16.54	2.09	16.57	2.10	16.59	2.11	16.62	2.10
Tau pi	23.88	4.41	23.29	4.33	23.87	4.40	22.70	4.43	22.28	4.27	21.91	4.23
Tau beta	0.05	1.97	0.03	1.93	0.05	1.97	0.44	2.08	0.27	1.97	0.15	1.90

Adjusted for time, age, BMI, gender, race/ethnicity, marital status, education, ratio of income to poverty, car ownership, homeownership, alcohol intake, smoking, alcohol intake, dietary intake, and physical activity

*≤ 0.05; **< 0.01; ***`≤ 0.001

beyond the effects of neighborhood poverty (Model 5). This association remains significant in models that include neighborhood, family, and safety stress (results not shown, β = 2.48, $p = 0.019$). Finally, the cumulative stress index was marginally associated with change in central adiposity over time (β = 0.28, $p = 0.056$) (Model 6) again, above and beyond the effects of neighborhood poverty. In Models 2 (neighborhood physical environment) and 6 (cumulative stress index), neighborhood poverty is no longer statistically significant. Because these effects are visible after accounting for BMI, they suggest that stress mediates associations between neighborhood poverty and central adiposity, above and beyond effects on BMI. This finding is consistent with the hypothesis that cumulative stress may be associated with metabolic changes that influence the deposition of adipose tissue in the mid-section of the body.

Does Psychosocial Stress Mediate the Association between Neighborhood Poverty and Central Adiposity Over Time?

To assess whether psychosocial stress mediated associations between neighborhood poverty and change in central adiposity over time, we used Freedman Schatzkin statistics [57]. Findings suggest that the association between neighborhood poverty and central adiposity over time is mediated by neighborhood physical environment (Freedman Schatzkin statistic = 1.867, $p = 0.031$) and by the cumulative stress index (Freedman Schatzkin statistic = 1.853, $p = 0.032$) (models not shown). There was no evidence that family stress (Freedman Schatzkin statistic = −0.09, $p = 0.535$), safety stress (Freedman Schatzkin statistic = −0.02, $p = 0.506$), or everyday unfair treatment (Freedman Schatzkin statistic = −0.07, $p = 0.529$) mediated the association

between neighborhood poverty and central adiposity over time.

Discussion

There were three main findings of this study. First, we found a significant association between neighborhood poverty and central adiposity, with residents of neighborhoods with higher concentrations of poverty more likely to experience greater increases in central adiposity over time. Second, we found partial support for the hypothesis that psychosocial stress may be positively associated with increases in central adiposity over time, above and beyond the effects of neighborhood poverty. Specifically, everyday unfair treatment was positively associated with increases in central adiposity over time, after accounting for multiple individual level demographic and behavioral indicators, and for neighborhood poverty level. Finally, formal tests for mediation suggest that perceptions of the neighborhood physical environment and cumulative stress mediate associations between neighborhood poverty and change in central adiposity over time. We discuss each of these findings in greater detail below.

Neighborhood Poverty and Central Adiposity Over Time.

The finding that neighborhood poverty at baseline is positively associated with changes in central adiposity is consistent with previous research [59]. It extends previous studies by showing that individuals residing in areas with high concentrations of poverty are at greater risk of increases in central adiposity over time, over and above the effects of household poverty, individual demographics, and individual behaviors associated with obesity. Thus, living in neighborhoods with higher concentrations of poverty may contribute to increases in central adiposity over time. Because NHBs and Hispanics disproportionately reside in neighborhoods with higher concentrations of poverty [60], these patterns may contribute to racial and ethnic disparities in central adiposity and associated chronic health conditions in the USA. Models used in these analyses controlled for BMI, thus suggesting associations specifically with central adiposity, above and beyond potential effects of neighborhood poverty on BMI.

Are Neighborhood Poverty and Psychosocial Stress Independently Associated with Central Adiposity?

Only one of the four measures of psychosocial stress included in our models was independently associated with change in central adiposity over time, over and above the effects of neighborhood poverty. The findings reported here demonstrate an association between everyday unfair treatment and increases in central adiposity extend those previously reported (Kwarteng, J., et al. (2016). "Neighbourhood poverty, perceived discrimination and central adiposity in the USA: Independent associations in a repeated measures analysis." Journal of biosocial science: 1–14.). This study shows that this association remains significant when accounting for neighborhood poverty and for several other indicators of psychosocial stress (i.e., neighborhood physical environment, family stress, and safety stress), and after accounting for BMI. Our finding that everyday unfair treatment and neighborhood poverty (when included simultaneously in models) each exert independent effects on change in central adiposity over time, reinforces the importance of examining multiple pathways through which inequalities may be linked to health [61].

Does Psychosocial Stress Mediate the Association between Neighborhood Percent Poverty and Central Adiposity?

We found some support for the hypothesis that psychosocial stress mediates the relationship between neighborhood poverty and increases in central adiposity over time. Specifically, a formal test for mediation suggests that self-reported neighborhood physical environment and the index of cumulative stress each mediate this association. There was no evidence that family stress, safety stress, or everyday unfair treatment mediate associations between neighborhood poverty and central adiposity. These findings are consistent with the hypothesis that neighborhood poverty may influence central adiposity through stress process pathways [59]. Previous studies have suggested that neighborhood poverty may be associated with obesity through physical environmental characteristics (e.g., recreation facilities, sidewalk condition) [10, 62, 63] that affect health-related behaviors, particularly diet and physical activity [5, 16], which are likely to be associated with BMI. Given that our models controlled for physical activity and dietary intake, these findings suggest that physical activity and dietary intake may not completely capture pathways

linking neighborhood physical environment with obesity. In addition, our finding that the cumulative stress index used in these analysis mediated the association between neighborhood poverty and change in central adiposity over time contributes to a growing body of research on associations between stress and central adiposity [43, 44]. Ours is the first study of which we are aware that has reported evidence that a measure of the combined effects of stress across multiple domains mediates associations between neighborhood poverty and central adiposity. These findings are consistent with evidence that residents of neighborhoods with higher poverty may confront a greater number of stressful life conditions [20, 23], and that those conditions may exert a greater toll on health (i.e., central adiposity) compared to those living in lower poverty neighborhoods.

Limitations and Strengths

This study has a few of limitations. Among these is the relatively modest sample size. Studies with larger sample sizes may have greater statistical power to assess more nuanced associations than those observed here. The dataset used for this analysis was drawn from predominantly NHB and Hispanic, low- to moderate-income urban neighborhoods. Additional research that encompasses neighborhoods with a broader range of socioeconomic status and different distributions of racial and ethnic characteristics, as well as rural and suburban neighborhoods, is warranted to more clearly understand these associations. Furthermore, this dataset offered measures of psychosocial stress across four domains—neighborhood environments, safety, family, and unfair treatment. These are, at best, a partial assessment of psychosocial stressors that may emerge across many additional domains and life circumstances, many of which emerge outside of, and independently from, the neighborhoods in which people reside. Further exploration of psychosocial stress across additional domains, and their independent and joint contributions to changes in central adiposity, may help to further inform interventions to reduce obesity.

A key strength of this study is the examination of central adiposity and the independent, conjoint, and composite associations of multiple indicators of psychosocial stress. Reviews of the literature on the relationship between stress and obesity have recommended the inclusion of a wide range of indicators of psychosocial stress. Our findings may provide insight on the influence of multiple indicators of psychosocial stress on excess central adiposity risk. In particular, our finding that everyday unfair treatment and neighborhood poverty are independently associated with increases in central adiposity suggests that residents of neighborhoods with high rates of poverty may experience stressors in multiple domains of life that exert independent effects on risk for central adiposity. Furthermore, the finding that both neighborhood physical environment, and the indicator of cumulative stress, mediated associations between neighborhood poverty and change in central adiposity over time, suggests that there are specific pathways within high poverty neighborhoods that may influence risk of central adiposity, and that these pathways extend beyond their specific effects on health-related behaviors such as physical activity and dietary quality. The inclusion of BMI in the models is also a strength, as it allows us to disentangle specific effects of poverty and psychosocial stress on central adiposity, above and beyond effects of BMI on waist circumference.

Conclusion

This study is one of only a few that has tested the independent effects of neighborhood poverty and multiple indicators of psychosocial stress on change in central adiposity over time. Our findings suggest that neighborhood poverty is significantly associated with changes in central adiposity over time, and that this association persists when several individual indicators of psychosocial stress are included in the models. Associations between neighborhood poverty and central adiposity were attenuated when self-reported neighborhood physical environment and when the combined effects of multiple stressors are accounted for. Tests of mediation effects suggested that self-reported neighborhood physical environment and cumulative stress index mediated, or helped to explain, associations between neighborhood poverty and central adiposity. Our findings are consistent with the idea that neighborhood poverty is associated with greater increases in central adiposity over time, and that these effects occur through pathways above and beyond their influence on physical activity and dietary practices. Our findings are consistent with the idea that neighborhood poverty may influence central adiposity at least in part through its effects on residents self-reported neighborhood physical environment. Higher poverty neighborhoods have been shown to have higher levels of both social and physical disorder [64], and those observed environments are

linked to residents' perceptions as well as levels of psychosocial stress [65–67] lending further evidence to support this pathway. In addition, the findings reported here suggest that the combined effects of multiple stressors may be important contributors to increases in central adiposity, and that these combined effects may be greater among those who reside in neighborhoods with higher rates of poverty.

This study extends previous research on the associations between psychosocial stress and increases in obesity-related measures over time [30, 32, 43], by showing evidence that both neighborhood poverty and the combined effects of multiple indicators of psychosocial stress are associated with increases in central adiposity over time. The results reported here suggest the importance of understanding exposure to stressful life conditions as a risk factor for increases in central adiposity over time. People living in high concentrations of poverty may encounter neighborhoods with higher levels of physical environmental risks that, in addition to their potential effects on physical activity, may also contribute to changes in central adiposity over time through physiologic changes resulting from psychosocial stress. In addition, our finding of an independent effect of unfair treatment suggests that those who experience racism, discrimination, or other forms of unfair treatment associated with social statuses may experience heightened risk of central adiposity over time, above and beyond risks associated with poverty. Together, these findings suggest the importance of extending analyses of central adiposity beyond a conversation about diet and physical activity, to encompass an analysis of broader social processes that contribute to excess levels of psychosocial stress among some subgroups of the population.

These findings suggest that adverse effects of concentrated poverty and of experiences of unfair treatment contribute to persistent inequalities in risks associated with obesity, and that the pathways linking these factors extend beyond commonly conceptualized behavioral pathways (e.g., dietary practices, physical activity) to encompass stress process pathways that influence obesity through changes in metabolic functioning. They suggest that economic investments and increased access to resources focused on reducing concentrations of poverty in urban communities, as well as interventions that reduce experiences of unfair treatment, may have important public health benefits. Future interventions and policies to reduce central adiposity should consider improvements in neighborhood physical environments, access to employment opportunities, quality education, increased wages, and mixed income housing to reduce concentrations of poverty. Further, interventions should work to reduce experiences of discrimination and unfair treatment.

Acknowledgements The Healthy Environments Partnership (HEP) (www.hepdetroit.org) is a communitybased participatory research partnership affiliated with the Detroit Community-Academic Urban Research Center (www.detroiturc.org). The authors thank the members of the HEP Steering Committee for their contributions to the work presented here, including representatives from Detroit Department of Health and Wellness Promotion, Detroit Hispanic Development Corporation, Friends of Parkside, Henry Ford Health System, Warren Conner Development Coalition, University of Michigan School of Public Health and community members. The study and analysis were supported by the National Institute of Environmental Health Sciences (NIEHS) (R01ES10936, R01ES014234), the Promoting Ethnic Diversity in Public Health Research Education Project (5-R25-GM058641-11), the Rackham Merit Fellowship, Rackham Graduate School, University of Michigan, and a Summer Mentored Writing Award through the Rackham Faculty Allies program at the University of Michigan. The results presented here are solely the responsibility of the authors and do not necessarily represent the views of NIEHS, the Promoting Ethnic Diversity in Public Health Research Education project, Rackham Merit Fellowship or the Rackham Faculty Allies program. This analysis was also supported by the Aetna Foundation, a National Foundation based in Hartford, Connecticut, that supports projects to promote wellness, health and access to high quality health care for everyone. The views presented here are those of the authors, and not necessarily those of the Aetna Foundation, its directors, officers or staff.

References

1. Kumanyika SK, Obarzanek E, Stettler N, et al. Population-based prevention of obesity the need for comprehensive promotion of healthful eating, physical activity, and energy balance: a scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the Expert Panel on Population and Prevention Science). *Circulation*. 2008;118(4):428–64.
2. Boardman JD, Onge JMS, Rogers RG, Denney JT. Race differentials in obesity: the impact of place. *J Health Soc Behav*. 2005;46(3):229–43.
3. Sallis JF, Glanz K. Physical activity and food environments: solutions to the obesity epidemic. *Milbank Q*. 2009;87(1):123–54.
4. Morland K, Diez Roux AV, Wing S. Supermarkets, other food stores, and obesity: the atherosclerosis risk in communities study. *Am J Prev Med*. 2006;30(4):333–9.

5. Lovasi GS, Hutson MA, Guerra M, Neckerman KM. Built environments and obesity in disadvantaged populations. *Epidemiol Rev*. 2009;31(1):7–20.
6. Waldstein SR, Moody DLB, McNeely JM, et al. Cross-sectional relations of race and poverty status to cardiovascular risk factors in the Healthy Aging in Neighborhoods of Diversity across the Lifespan (HANDLS) study. *BMC Public Health*. 2016;16:258.
7. Franco M, Diez Roux AV, Glass TA, Caballero B, Brancati FL. Neighborhood characteristics and availability of healthy foods in Baltimore. *Am J Prev Med*. 2008;35(6):561–7.
8. Larson NI, Story MT, Nelson MC. Neighborhood environments: disparities in access to healthy foods in the US. *Am J Prev Med*. 2009;36(1):74–81. e10
9. Powell L, Slater S, Mirtcheva D, Bao Y, Chaloupka F. Food store availability and neighborhood characteristics in the United States. *Prev Med*. 2007;44(3):189–95.
10. Kwarteng JL, Schulz AJ, Mentz GB, Zenk SN, Opperman AA. Associations between observed neighborhood characteristics and physical activity: findings from a multiethnic urban community. *J Public Health*. 2013;36(3):358–67.
11. Schulz A, Kannan S, Dvonch J, et al. Social and physical environments and disparities in risk for cardiovascular disease: the healthy environments partnership conceptual model. *Environ Health Perspect*. 2005;113(12):1817–25.
12. Sallis JF, Floyd MF, Rodríguez DA, Saelens BE. Role of built environments in physical activity, obesity, and cardiovascular disease. *Circulation*. 2012;125(5):729–37.
13. Troped PJ, Wilson JS, Matthews CE, Cromley EK, Melly SJ. The built environment and location-based physical activity. *Am J Prev Med*. 2010;38(4):429–38.
14. Ding D, Gebel K. Built environment, physical activity, and obesity: what have we learned from reviewing the literature? *Health & Place*. 2012;18(1):100–5.
15. Mackenbach JD, Rutter H, Compernolle S, et al. Obesogenic environments: a systematic review of the association between the physical environment and adult weight status, the SPOTLIGHT project. *BMC Public Health*. 2014;14(1):233.
16. Diez Roux AV, Mair C. Neighborhoods and health. *Ann N Y Acad Sci*. 2010;1186(1):125–45.
17. Schulz AJ, Mentz G, Lachance L, Johnson J, Gaines C, Israel BA. Associations between socioeconomic status and allostatic load: effects of neighborhood poverty and tests of mediating pathways. *Am J Public Health*. 2012;102(9):1706–14.
18. Schulz AJ, Zenk SN, Israel BA, Mentz G, Stokes C, Galea S. Do neighborhood economic characteristics, racial composition, and residential stability predict perceptions of stress associated with the physical and social environment? Findings from a multilevel analysis in Detroit. *Journal of Urban Health*. 2008;85(5):642–61.
19. Evans GW. The environment of childhood poverty. *Am Psychol*. 2004;59(2):77.
20. Santiago CD, Wadsworth ME, Stump J. Socioeconomic status, neighborhood disadvantage, and poverty-related stress: prospective effects on psychological syndromes among diverse low-income families. *J Econ Psychol*. 2011;32(2):218–30.
21. Baum A, Garofalo J, YALI A. Socioeconomic status and chronic stress: does stress account for SES effects on health? *Ann N Y Acad Sci*. 1999;896(1):131–44.
22. Steptoe A, Feldman P. Neighborhood problems as sources of chronic stress: development of a measure of neighborhood problems, and associations with socioeconomic status and health. *Ann Behav Med*. 2001;23(3):177–85.
23. Boardman JD. Stress and physical health: the role of neighborhoods as mediating and moderating mechanisms. *Soc Sci Med*. 2004;58(12):2473–83.
24. Lantz PM, House JS, Mero RP, Williams DR. Stress, life events, and socioeconomic disparities in health: results from the Americans' Changing Lives study. J *Health Soc Behav*. 2005;46(3):274–88.
25. Lohman BJ, Stewart S, Gundersen C, Garasky S, Eisenmann JC. Adolescent overweight and obesity: links to food insecurity and individual, maternal, and family stressors. *J Adolesc Health*. 2009;45(3):230–7.
26. Bennett GG, McNeill LH, Wolin KY, Duncan DT, Puleo E, Emmons KM. Safe to walk? Neighborhood safety and physical activity among public housing residents. *PLoS Med*. 2007;4(10):e306.
27. Williams DR, John DA, Oyserman D, Sonnega J, Mohammed SA, Jackson JS. Research on discrimination and health: an exploratory study of unresolved conceptual and measurement issues. *Am J Public Health*. 2012;102(5):975–8.
28. Williams D, Mohammed S. Discrimination and racial disparities in health: evidence and needed research. *J Behav Med*. 2009;32(1):20–47.
29. Pascoe E, Richman L. Perceived discrimination and health: a meta-analytic review. 2009.
30. Pearlin LI. The sociological study of stress. *J Health Soc Behav*. 1989:241–56.
31. Williams DR, House JS. Stress, social support, control and coping: a social epidemiological view. *WHO Reg Publ Eur Ser*. 1991;37:147–72.
32. Williams DR, Spencer MS, Jackson JS. Race, stress, and physical health. Self, social identity, and physical health 1999:71–100.
33. Pearlin LIPearlin LI. Stress and mental health: A conceptual overview. In: Horwitz AV, ed. Handbook for the study of mental health: Cambridge University Press; 1999:161–175.
34. Björntorp P, Rosmond R. The metabolic syndrome—a neuroendocrine disorder? *Br J Nutr*. 2000;83(S1):S49–57.
35. Dallman MF, la Fleur SE, Pecoraro NC, Gomez F, Houshyar H, Akana SF. Minireview: glucocorticoids—food intake, abdominal obesity, and wealthy nations in 2004. *Endocrinology*. 2004;145(6):2633–8.
36. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci*. 2006;896(1):30–47.
37. Björntorp P. The associations between obesity, adipose tissue distribution and disease. *Acta Med Scand*. 1987;222(S723):121–34.
38. Björntorp P. Stress and cardiovascular disease. *Acta Physiol Scand Suppl*. 1997;640:144–8.
39. Koch FS, Sepa A, Ludvigsson J. Psychological stress and obesity. *J Pediatr*. 2008;153(6):839–44. e833

40. Brydon L. Adiposity, leptin and stress reactivity in humans. *Biol Psychol.* 2011;86(2):114–20.
41. Wardle J, Chida Y, Gibson EL, Whitaker KL, Steptoe A. Stress and adiposity: a meta-analysis of longitudinal studies. *Obesity.* 2011;19(4):771–8.
42. Mahabadi AA, Massaro JM, Rosito GA, et al. Association of pericardial fat, intrathoracic fat, and visceral abdominal fat with cardiovascular disease burden: the Framingham Heart study. *Eur Heart J.* 2009;30(7):850–6.
43. Lloyd C, Wing R, Orchard T. Waist to hip ratio and psychosocial factors in adults with insulin-dependent diabetes mellitus: the Pittsburgh Epidemiology of Diabetes Complications study. *Metab Clin Exp.* 1996;45(2):268–72.
44. Fowler-Brown A, Bennett G, Goodman M, Wee C, Corbie-Smith G, James S. Psychosocial stress and 13-year BMI change among blacks: the Pitt County study. *Obesity.* 2009;17(11):2106–9.
45. Block JP, He Y, Zaslavsky AM, Ding L, Ayanian JZ. Psychosocial stress and change in weight among US adults. *Am J Epidemiol.* 2009;170(2):181–92.
46. Geronimus A. Understanding and eliminating racial inequalities in women's health in the United States: the role of the weathering conceptual framework. *J Am Med Womens Assoc* (1972). 2001;56(4):133.
47. Schulz A, Northridge M. Social determinants of health: implications for environmental health promotion. *Health Educ Behav.* 2004;31(4):455.
48. Williams DR, Yu Y, Jackson JS, Anderson NB. Racial differences in physical and mental health socio-economic status, stress and discrimination. *J Health Psychol.* 1997;2(3):335–51.
49. Fisher GM. The development of the Orshansky poverty thresholds and their subsequent histoy as the official US poverty measure. 1997.
50. Block G, Hartman AM, Naughton D. A reduced dietary questionnaire: development and validation. *Epidemiology* 1990:58–64.
51. Frazier E, Franks A, Sanderson L. Behavioral risk factor data. Using chronic disease data: A handbook for public health practitioners 1992:4.1–1.17.
52. Kennedy ET, Ohls J, Carlson S, Fleming K. The healthy eating index: design and applications. *J Am Diet Assoc.* 1995;95(10):1103–8.
53. Ainsworth BE, Wilcox S, Thompson WW, Richter DL, Henderson KA. Personal, social, and physical environmental correlates of physical activity in African-American women in South Carolina. *Am J Prev Med.* 2003;25(3):23–9.
54. Wineman JD, Marans RW, Schulz AJ, van der Westhuizen DL, Max P. Neighborhood design and health: characteristics of the built environment and health related outcomes for residents of Detroit neighborhoods. Paper presented at: Eighth International Space Syntax Symposium, Santiago de Chile, Chile 2012.
55. Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc.* 2000;32(9; SUPP/1): S498–504.
56. Craig C, Marshall A, Sjöström M, the IPAQ Consensus Group and the IPAQ Reliability and Validity Study Group, et al. International Physical Activity Questionnaire (IPAQ): 12-country reliability and validity. *Med Sci Sports Exerc.* 2003;35:1381–95.
57. Zhang Z, Zyphur MJ, Preacher KJ. Testing multilevel mediation using hierarchical linear models: problems and solutions. *Organ Res Methods.* 2009;12(4):695–719.
58. Freedman LS, Schatzkin A. Sample size for studying intermediate endpoints within intervention trials or observational studies. *Am J Epidemiol.* 1992;136(9):1148–59.
59. Jiménez-Cruz A, Castañeda-Gonzalez LM, Bacardí-Gascón M. Poverty is the main environmental factor for obesity in a Mexican-Border City. *JHCPU.* 2013;24(2):556–65.
60. Jargowsky PA. Concentration of poverty in the new millennium: changes in prevalence, composition, and location of high-poverty neighborhoods. New York The Century Foundation; December 2013 2013.
61. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav.* 1995:80–94.
62. Black JL, Macinko J. Neighborhoods and obesity. *Nutr Rev.* 2008;66(1):2–20.
63. Hoehner CM, Brennan Ramirez LK, Elliott MB, Handy SL, Brownson RC. Perceived and objective environmental measures and physical activity among urban adults. *Am J Prev Med.* 2005;28(2):105–16.
64. Neckerman KM, Lovasi GS, Davies S, et al. Disparities in urban neighborhood conditions: evidence from GIS measures and field observation in new York City. *J Public Health Policy.* 2009:S264–85.
65. Austin DM, Furr LA, Spine M. The effects of neighborhood conditions on perceptions of safety. *J Crim Just.* 2002;30(5): 417–27.
66. Boehmer T, Hoehner C, Deshpande A, Ramirez LB, Brownson RC. Perceived and observed neighborhood indicators of obesity among urban adults. *Int J Obes.* 2007;31(6):968–77.
67. Theall KP, Drury SS, Shirtcliff EA. Cumulative neighborhood risk of psychosocial stress and allostatic load in adolescents. *Am J Epidemiol.* 2012;176(suppl 7):S164–74.