Neighborhoods and Obesity in Older Adults
The Baltimore Memory Study

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Background: Obesity has reached epidemic proportions. Although its causes are not well understood, its increasing prevalence is not likely to be due to genetic factors or underlying biology. This has led to interest in the role of environmental factors, although few studies have focused on the role of the social environment. This study investigated whether neighborhood psychosocial hazards independent of individual risk factors were associated with increased odds of obesity.

Methods: Baseline data were analyzed in 2005 from a cohort study of 1140 randomly selected community-dwelling men and women aged 50 to 70 years from 65 contiguous neighborhoods in Baltimore MD. Body mass index (BMI in kilograms/meters squared) was calculated from measured height and weight at baseline (2001–2002). People with a BMI of 30 and higher were considered obese. Multilevel logistic regression was used to examine associations between a 12-item scale of neighborhood psychosocial hazards and the odds of obesity.

Results: Thirty-eight percent of the cohort were obese. Residents of neighborhoods in the highest quartile of the Neighborhood Psychosocial Hazards scale were nearly twice as likely to be obese compared to residents in the least-hazardous neighborhoods (53% vs 27%). After adjustment for age, gender, race/ethnicity, education, household wealth, alcohol consumption, tobacco use, self-reported physical activity, and dietary intake, living in more hazardous neighborhoods was associated with a graded increase in the odds of obesity. This association was partially mediated by physical activity.

Conclusions: Even after controlling for a large set of demographic, behavioral, and socioeconomic individual-level risk factors, living in a neighborhood with greater psychosocial hazards was independently associated with obesity.
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Introduction

Rates of obesity have increased dramatically in the last several decades resulting in an emerging global epidemic.[1–3] The rate of growth of the epidemic over this timeframe renders implausible explanations that involve changes in biology or genetics. Although several individual-level risk factors for obesity have been identified, population rates of obesity are determined by a complex interplay of genetic, environmental, behavioral, and cultural factors. A comprehensive understanding of how these factors interact is currently lacking.

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Treatment and prevention efforts continue to be costly and have limited effect.[4,5] It is unlikely that treating those who are obese on an individual basis will have a significant public health impact if each treated case is replaced by large numbers of new cases.[6] A recent report by the Institute of Medicine[7] summarizes an emerging consensus: Environmental factors play a role, and environmental solutions will be required to address the epidemic.

Several models have been proposed to explain the "obesogenic environment."[8–12] One set of studies has focused on the built environment (urban design, transportation, land use, and physical activity)[13–17] or the local food environment.[18–22] Among these, the strongest findings have been from studies of suburban or exurban communities.[16,23] A second group of studies looks at socioeconomic deprivation at the community or neighborhood level.[24–32] These studies show that places in which people are poor are associated with obesity. The reasons for this association, however, are unclear. This is partly because most previous studies have used single and relatively crude summary measures of economic depriva-

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tion that do not allow for testing of specific hypotheses about what underlies the association. Much of the existing literature has been hampered by other problems including same-source bias (getting information on neighborhoods and outcomes from the same people), and using census tracts or postal districts as proxies for residential neighborhoods.

The purpose of this study was to test the hypothesis that living in a neighborhood with more psychosocial hazards is associated with individual obesity, independent of known risk factors (race/ethnicity, individual education, and income) and health behaviors (physical activity and diet). This hypothesis was motivated by animal and human research showing a link among environmental stress, cortisol production, and increasing body mass.[33–36] This study overcomes the limitations of previous studies in four ways: (1) the use of sociologically real neighborhoods (rather than administrative units), (2) state-of-the-art measurement of one specific aspect of neighborhood conditions, (3) collection of data on persons and places separately, and (4) the ability to contrast competing hypotheses about what makes neighborhoods important. Data from a population-based cohort of adults aged 50 to 70 years recruited from strategically chosen neighborhoods in Baltimore City (MD) are used in the analysis.

See related Commentary on page 530.

Methods

Study Design

The Baltimore Memory Study is a multilevel cohort study of risk factors for cognitive decline in Baltimore City residents. Residents were sampled at random from a predetermined study area consisting of 65 contiguous urban neighborhoods selected to contain the full spectrum of socioeconomic conditions. The methods are described elsewhere.[37] A cross-sectional analysis of baseline data was performed.

Subject Selection and Recruitment

Using a sampling frame based on the Baltimore Department of Assessments and Taxation, all residential addresses in the study area were identified and linked to telephone numbers after carefully accounting for apartment buildings and other multiresident dwellings. Six random samples were drawn from this sampling frame, and households were called up to ten times to determine eligibility and recruitment. Eligibility was determined on 2351 subjects (aged 50 to 70 years, living at selected household, lived in Baltimore at least 5 years), and of these subjects, 60.8% were scheduled for an enrollment visit. Of the 1403 scheduled for an appointment, 1140 (81.3%) were enrolled and subsequently tested. The study was approved by the Committee for Human Research of the Johns Hopkins Bloomberg School of Public Health. All participants provided written, informed consent before testing and were paid $50 for their participation.

Data Collection

Data on individual study participants were collected at the study clinic (in northern Baltimore City) by trained research assistants during three study visits, averaging 16 months apart. During the first study visit, a structured interview obtained information on self-reported date of birth and age in years, gender, race/ethnicity (using the Census 2000 method[38]), housing and residential history, and smoking and alcohol consumption history. Body mass index (BMI) was computed based on measured weight (in kilograms) divided by measured height (in meters) squared. Respondents were classified as overweight (BMI >24.9) and obese (BMI >29.9) based on Centers for Disease Control and Prevention guidelines (www.cdc.gov/nccdphp/dnpa/bmi/adult_BMI/about_adult_BMI.htm).

Individual-Level Measures

Socioeconomic status was assessed using an instrument specifically designed for the study to assess individual and household educational attainment and wealth.[37] Variables used included an ordinal index of educational attainment (nine levels) and household wealth (income plus assets) in dollars (five levels).

Physical activity was measured using the Yale Physical Activity Survey (YPAS), a 43-item self-report instrument designed for epidemiologic studies of older adults.[39] The YPAS has been shown to have favorable reliability and validity as assessed by correlation with objective methods.[40] Based on previous studies of obesity, two variables were used: vigorous physical activity and leisure-time walking intensity. Dietary behavior (usual eating habits in the past year) was obtained from the Block 98.2 Food-Frequency Questionnaire,[41] which was filled out by study participants at home before the second study visit. Based on previous studies[40,42] and strength of association with obesity, percentage of calories from fat and total kilocalories of dietary intake were used in the analysis.

Neighborhood Measures

Neighborhood boundaries were created by the Baltimore City Department of Planning based on community definitions of existing neighborhoods. Thus, the spatial unit is named "city neighborhood," not census units or ZIP codes that are used as proxies for neighborhoods in most studies. Data on neighborhood characteristics came from the 2000 U.S. Census, and the Baltimore City Departments of Planning, Public Works, and Police. Block-level census data were recombined into neighborhoods by the U.S. Census Bureau. Block-level census data, the smallest level of aggregation available, is not normally made available to researchers for confidentiality reasons. By special request, the Census Bureau tabulated this lowest-level data to the precise boundaries of the Baltimore neighborhoods. Number and location of violent crimes, off-site liquor licenses, and 911 emergency telephone calls were individually mapped and aggregated at the neighborhood level using a geographic information system (GIS). Participants were linked to their neighborhood of residence by their home address at baseline.

Three summary scales were examined: the Townsend Index of Socioeconomic Deprivation and two variables created specifically for this study. The Townsend Index is a standard summary measure of neighborhood socioeconomic deprivation developed specifically for use in public health research that combines information on crowding, home ownership, unemployment, and availability of a car.[43] Secondly, an Index of Neighborhood Affluence was created from information on neighborhood wealth based on prior studies.[44,45] This index combined information on percentage of residents with a bachelor's degree or higher, percentage with per capita income of ≥$20,000, ≥30% of residents working in professional, administrative, or management occupations, median family income of ≥$50,000, median house value of ≥$75,000, and one quarter or more of school-aged children enrolled in private school.

In order to test whether psychosocial hazards were more closely associated with obesity than these two socioeconomic variables, the Neighborhood Psychosocial Hazards scale (NPH) was also created. Using a two-step method, theory and previous studies were used to choose 20 variables hypothesized to indicate (or measure) psychosocial hazards, defined as stable and visible features of neighborhood environments that give rise to a heightened state of vigilance, alarm, or fear in residents.[46,47] Second, factor analysis identified a final set of 12 items that were found to be measuring one underlying concept (or factor). These included both census variables and noncensus indicators of psychosocial hazards. To create the final scale, each variable was standardized (z-score) and the 12 values were summed. This yielded a normally distributed scale (range of −19.0 to 19.2, mean=0.1, standard deviation [SD]=9.6). For modeling, the NPH was divided into quartiles to test across levels of neighborhood psychosocial hazards. A fuller description of the 12 final indicators in the NPH is shown in an online appendix (www.ajpm-online.net).

Statistical Analyses

The main objectives of this analysis were to evaluate whether: (1) NPH was associated with odds of obesity, (2) the association was independent of individual-level risk factors for obesity, and partially independent of hypothesized mediators (physical activity and diet), and (3) associations of the NPH differed when compared to two other measures of neighborhoods that focus more narrowly on socioeconomic characteristics. Bivariate associations were evaluated using chi-squared tests for pairs of ordinal variables, and one-way analysis of variance for unbalanced designs for pairs of continuous and discrete variables.

Random effects (multilevel) logistic regression models were used to examine associations of the three neighborhood metrics with obesity, controlling for individual-level confounders and mediators. By fitting a random intercept for each neighborhood, the models took account of the correlation among persons within a neighborhood, yielding more accurate standard errors and unbiased maximum likelihood parameter estimates.[48] The interpretation of parameters is analogous to the standard logistic regression model.

Ten subjects were missing height or weight data and were excluded from the analysis. Another eight subjects were excluded because they lived in a neighborhood where it was impossible to calculate the NPH due to small sample sizes. Models were constructed sequentially based on a priori theory. Model 1 included known sociodemographic confounders (e.g., age, gender, and race/ethnicity), Model 2 included individual socioeconomic status (education and household wealth), Model 3 added health behaviors known to be associated with risk of obesity (smoking and alcohol consumption), and Models 4 and 5 included two hypothesized mediators of the influence of the NPH on obesity: physical activity and diet (total kilocalorie intake and percent of calories from fat). All models included three dummy variables to indicate increasing levels of NPH by quartile (lowest level of psychosocial hazards served as the reference category). A final set of models was estimated replacing the NPH with the Townsend Index and the Index of Neighborhood Affluence (Table 4). All statistical analyses were performed using SAS, version 8.2 (SAS Institute, Cary NC, 2001).

Results

Study subjects were 66% female, 41% non-Hispanic black/African American, and 54% non-Hispanic white or white/Native American, and had a mean (SD) age of 59 (6) years. Overall, 79% of study participants with valid data on height and weight were overweight (BMI >24.9) and 38% were obese (BMI >29.9) (Table 1). Women, African Americans, those with less education, nonsmokers, less-frequent drinkers, and those with lower household wealth were more likely to be obese (Table 1). Significant trends were also seen for those reporting less-vigorous physical activity and a higher percent of calories from fat.

Next, differences in individual characteristics across quartiles of the NPH were examined (Table 2). Those living in neighborhoods with more psychosocial hazards were more likely to be overweight and obese. Residents of the most-hazardous neighborhoods had average BMI scores 3.7 points higher than the least-hazardous neighborhoods. Other than gender, all variables showed trends in association with obesity across levels of the NPH. While a strong association was observed between race/ethnicity and NPH, there was still reasonable overlap in the distributions; 11% of residents in the highest quartile neighborhoods were white and 8% and 41% of residents in the least- and next-to-least hazardous neighborhoods were African American, respectively. Finally, higher NPH scores were associated with less physical activity and less healthy diets.

Random effects logistic regression was next used to evaluate the association between NPH and obesity controlling for individual covariates (Table 3). Controlling for age, gender, and race/ethnicity (Model 1), the odds of obesity increased across all levels of NPH compared to the lowest level. Obesity prevalence ranged from 70% higher in the low–moderate quartile to more than twice as high in the two highest quartiles. After adjusting for individual and household socioeconomic status, a signifi-

Table 1. Summary of selected study variables in BMS cohort by obesity status

Variable	Full sample n (%)	Percent obese[a] (BMI>29.9)
Entire BMS cohort[b]	1119 (100)	421 (38)
Age (years)		
50–55	359 (32)	42
56–60	294 (26)	42
61–65	233 (21)	42
66–70	247 (22)	45
		p=0.93
Gender		
Male	390 (34)	34
Female	743 (66)	47
		p<0.0001***
Race/ethnicity		
African American	470 (41)	53
Other/multiple	55 (5)	40
White	608 (54)	35
		p<0.0001***
Years of education		
<12	164 (15)	52
12	251 (22)	53
13–15	228 (20)	48
16–17.5	215 (19)	31
>17.5	274 (24)	33
		p<0.0001***
Household wealth (income and assets, log transformed)		
Low quartile	286 (25.1)	45
Mid–low quartile	283 (24.8)	53
Mid–high quartile	286 (25.1)	45
High quartile	285 (25.0)	27
		p<0.0001***
Currently smoking?		
Yes	241 (21)	34
No	892 (79)	45
		p<0.0034**
Alcoholic drinks/month		
None	465 (41)	53
1–10	334 (30)	42
10–40	206 (18)	27
>40	121 (11)	33
		p<0.0001***
Percent calories from fat		
Low quartile	252 (25)	35
Low–moderate quartile	257 (25)	43
High–moderate quartile	257 (25)	45
High quartile	255 (25)	48
		p=0.02*
Total dietary intake in kilocalories		
Low quartile	252 (25)	42
Low–moderate quartile	256 (25)	45
High–moderate quartile	256 (25)	38
High quartile	257 (25)	46
		p=0.21
Vigorous physical activity		
None	266 (26)	50
Moderate	392 (38)	46
High	368 (36)	34
		p<0.0001***
Leisure walking intensity		
None	257 (25)	49
Moderate	370 (36)	42
High	398 (39)	39
		p=0.03*

[a]p values are for chi-square tests of whether the groups within each cell are significantly different.
[b]Total sample size used in this table excludes 10 subjects missing information on body mass index and 8 subjects missing neighborhood data (total missing=18).
*$p < 0.05$;
**$p < 0.01$;
***$p < 0.0001$ (all bolded).
BMI, body mass index; BMS, Baltimore Memory Study.

cant increased odds of obesity was seen across each level of the NPH (odds ratio $[OR]_{low–moderate}$=1.58, $OR_{high–moderate}$=1.91, OR_{high}=1.93) (Model 2). Adjustment for smoking and alcohol consumption resulted in no reduction in the magnitude of associations with NPH (Model 3). Controlling for diet (Model 4) attenuated the NPH association slightly ($OR_{moderate–low}$=1.60, $OR_{moderate–high}$=2.00, OR_{high}=2.03). Adjustment for vigorous physical activity and leisure walking resulted in a further small decline in the NPH odds ratios, suggestive of some degree of mediation. However, even after controlling for the entire set of confounders and mediators, living in a neighborhood with a higher NPH remained associated with 52% to 96% increased odds of obesity (Model 5).

Finally, Table 4 presents results comparing the associations of the NPH with obesity to those of two other measures of neighborhood socioeconomic characteristics. The three models shown are estimated using the same model and covariates as Model 5. The Townsend Index showed a weaker association with obesity across all levels compared to the NPH. A significantly increased OR was seen for the high–moderate quartile compared to the lowest quartile and for the test of linear trend. The index of neighborhood affluence was unrelated to odds of obesity.

Discussion

Although associations between individual-level socioeconomic status and obesity have been repeatedly observed, the relationship between neighborhood characteristics and obesity is less clear. This study adds to the evidence that neighborhood social and economic factors may be related to adult obesity. The main finding is that after adjusting for a large set of important individual-level risk factors, including household socioeconomic status, a significant association was found between the NPH and obesity in an older, community-based, adult population in a major

Table 2. Associations of Neighborhood Psychosocial Hazards scale (in quartiles) with obesity and selected covariates

Variable	Low quartile % or mean (SD)	Low–moderate quartile % or mean (SD)	High–moderate quartile % or mean (SD)	High quartile % or mean (SD)	Test for differences across columns
All participants	219 (19%)	446 (39%)	225 (20%)	245 (22%)	
Obese (%)	27.4	43.3	46.9	52.5	**<0.0001****
Body mass index (mean)	27.5 (5.5)	30.0 (6.8)	30.0 (7.3)	31.2 (7.2)	**<0.0001****
Age in years (mean)	59.3 (5.7)	59.1 (6.0)	59.6 (6.1)	59.6 (6.1)	**<0.0001****
Female (%)	62.6	67.5	61.8	68.6	0.259
Race/ethnicity (%)					
African American	7.8	40.8	32.4	81.6	
Other/multiple	2.7	3.1	7.1	6.9	
White	89.5	56.1	60.4	11.4	**<0.001***
Years of education (mean)	17.1 (3.1)	15.0 (3.8)	13.7 (3.7)	12.8 (3.5)	**<0.0001****
Household wealth (median income plus assets, $)	426,300	149,000	84,285	58,200	**<0.0001****
% currently smoking	11.9	19.3	25.8	29.0	**<0.0001****
Drinks per month	22.4 (35)	12.5 (24)	16.1 (43)	9.5 (23)	**<0.0001****
Calories from fat (%)	37.4 (8.6)	36.9 (7.9)	36.0 (7.5)	37.8 (7.2)	**<0.0001****
Total dietary intake (kilocalories)	1723 (621)	1731 (914)	1885 (1022)	1940 (1177)	**<0.0001****
Vigorous physical activity score	20.2 (15.9)	15.9 (16.1)	14.4 (16.4)	13.1 (16.4)	**<0.0001****
Leisure walking intensity	14.3 (9.7)	11.8 (10.0)	13.1 (10.8)	13.1 (11.7)	**<0.0001****

*$p<0.001$;
**$p<0.0001$ (all bolded).
SD, standard deviation.

U.S. urban area. Because these results are based on cross-sectional analyses, it is not possible to establish causal effects. However, given that people living in their current neighborhood for less than 5 years were excluded from participating in the study, these findings are unlikely to result from recent moves of people into or out of their neighborhood as a function of body mass. Importantly (although not shown), neighborhood factors were found to be more strongly related to obesity than to overweight in this adult population. This provides additional evidence that the risk factors for overweight and obesity may differ in adults.

Although the study of neighborhoods and health has grown rapidly over the past decade, several important weaknesses have been identified. First, the vast majority of studies use administrative areas (like ZIP codes, postal districts, or census tracts) as proxies for residential neighborhoods. These fail to capture true neighborhood boundaries and may lead to misclassification of exposure to environmental factors. Second, previous studies have been limited to crude measures like poverty or median income. This has impeded the testing of specific hypotheses about how neighborhoods matter.[49,50] This study addresses both these limitations. A measure of neighborhood psychosocial hazards was developed and validated to test a specific, biologically plausible hypothesis about how neighborhood conditions might be related to obesity. The NPH is composed of a variety of types of variables, taken from sources other than study participants that were found in factor analysis to be closely related to one another. The material and psychosocial aspects of neighborhoods are assumed to be complex and closely interrelated. However, the contribution of psychosocially mediated processes (like stress) and material deprivation are not mutually exclusive. Due to this considerable conceptual overlap, the three neighborhood measures were not included in a single model. Correlations among these factors range from a −0.65 between the Townsend Index and the Index of Neighborhood Affluence to 0.87 for the Townsend Index and the NPH. These represent three batches of covariance that emphasize different but interrelated facets of the neighborhood atmosphere, not distinct analytically separable phenomenon.

The association between the NPH and obesity was not substantially mediated through dietary intake or physical activity. Dietary behavior was only assessed at the second study visit, and although it was designed to assess usual eating patterns in the previous year, these data may be biased assessments of baseline diet. Further, obese individuals who are attempting to lose weight may consume fewer calories, or may underreport intake. Differential measurement error in this self-report instrument is a possibility, making this test of mediation inconclusive. The association between NPH and obesity was reduced slightly when self-reported physical activity was added (Model 5), indicating that this association may be mediated, at least in part, by the impact of neighborhoods on physical activity. These findings corroborate other studies showing that area of

Table 3. Associations of individual- and neighborhood-level (bolded) risk factors for obesity (BMI>29.9) in 50- to 70-year-old subjects in Baltimore Memory Study using random-effects logistic regression

	Model 1		Model 2		Model 3		Model 4		Model 5	
Parameter	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**	**OR**	**95% CI**
Individual intercept	0.26	0.18–0.36	0.57	0.27–1.19	0.85	0.40–1.84	0.26	0.09–0.76	0.34	0.11–1.06
Age (in years)	0.99	0.81–1.22	0.99	0.80–1.22	0.92	0.74–1.15	0.95	0.75–1.20	0.91	0.72–1.16
Race/ethnicity	1.61	1.21–2.15	1.39	1.02–1.87	1.36	1.00–1.85	1.56	1.13–2.16	1.50	1.08–2.09
African American vs white										
Other vs white	0.92	0.51–1.67	0.86	0.47–1.57	0.98	0.53–1.80	1.28	0.66–2.47	1.27	0.65–2.46
Female gender	1.70	1.31–2.21	1.70	1.30–2.21	1.54	1.17–2.03	1.35	1.00–1.81	1.29	0.96–1.74
NPH scale										
Low–moderate vs low	1.70	1.18–2.46	1.58	1.07–2.31	1.60	1.09–2.36	1.60	1.07–2.41	1.52	1.01–2.30
High–moderate vs low	2.13	1.41–3.20	1.91	1.25–2.93	1.93	1.25–2.97	2.00	1.26–3.17	1.91	1.20–3.05
High vs low	2.04	1.30–3.20	1.93	1.21–3.10	1.97	1.22–3.17	2.03	1.22–3.37	1.96	1.18–3.28
Respondent education	—	—	0.81	0.67–0.97	0.76	0.63–0.92	0.76	0.62–0.93	0.78	0.64–0.96
Household wealth										
(Low quintile)[a]	—	—	0.79	0.55–1.13	0.86	0.59–1.24	0.82	0.55–1.22	0.80	0.53–1.20
(3rd quintile)	—	—	1.51	1.06–2.16	1.43	0.99–2.05	1.45	0.99–2.14	1.45	0.98–2.14
(4th quintile)	—	—	0.81	0.56–1.17	0.79	0.54–1.14	0.71	0.48–1.06	0.73	0.49–1.09
(5th quintile)	—	—	0.70	0.43–1.14	0.68	0.42–1.11	0.65	0.39–1.09	0.65	0.39–1.08
Current smoking (yes vs no)	—	—	—	—	0.55	0.39–0.76	0.51	0.36–0.73	0.49	0.35–0.71
Alcoholic drinks per month	—	—	—	—	0.96	0.89–1.03	0.96	0.89–1.04	0.96	0.89–1.04
Percent of calories from fat	—	—	—	—	—	—	1.39	1.17–1.66	1.40	1.17–1.66
Total dietary intake (kilocalories)	—	—	—	—	—	—	1.00	0.76–1.32	1.01	0.76–1.32
Vigorous physical activity										
Moderate vs none	—	—	—	—	—	—	—	—	0.98	0.69–1.38
High vs none	—	—	—	—	—	—	—	—	0.69	0.48–1.00
Leisure walking										
Moderate vs none	—	—	—	—	—	—	—	—	0.87	0.61–1.24
High vs none	—	—	—	—	—	—	—	—	0.80	0.56–1.14
−2 log likelihood		1477.3		1453.5		1430.2		1267.7		1258.5
Change in model fit				23.8		23.3		162.5		9.2

[a]The second quintile of household wealth was used as the reference group.
BMI, body mass index; CI, confidence interval; NPH, Neighborhood Psychosocial Hazards; OR, odds ratio; PA, physical activity.

Table 4. Contrast of associations[a] between Neighborhood Psychosocial Hazards scale, Townsend Index, and Index of Neighborhood Affluence with odds of obesity

	Neighborhood measure		
	Neighborhood psychosocial hazard scale Adjusted OR (95% CI)	**Townsend index of socioeconomic deprivation Adjusted OR (95% CI)**	**Index of neighborhood affluence Adjusted OR (95% CI)**
Low quartile	1.00	1.00	1.00
Low–moderate quartile	1.52 (1.01–2.30)	1.04 (0.70–1.55)	0.91 (0.60–1.38)
High–moderate quartile	1.91 (1.20–3.05)	1.53 (1.09–2.15)	0.83 (0.56–1.24)
High quartile	1.96 (1.18–3.28)	1.48 (0.95–2.31)	0.63 (0.39–1.02)
Test of linear trends[b]	***p*=0.008****	***p*=0.015***	***p*=0.061**

[a]All models were adjusted for the following covariates: age, race/ethnicity, gender, education, household wealth, smoking, alcohol consumption, dietary fat intake, total kilocalories of energy intake, vigorous physical activity, frequency of walking, and nesting of persons within neighborhoods
[b]The test for a linear trend is based on fitting a separate analysis using the same model and the same covariates, but replacing the separate quartile-specific effects with a single degree of freedom test with four levels of the neighborhood measure.
*$p<0.05$;
**$p<0.01$ (all bolded).
CI, confidence interval; OR, odds ratio.

residence is associated with physical activity.[14,51] However, the main finding is that neighborhood characteristics may exert an independent influence on risk of obesity. This suggests that pathways other than food availability or physical activity may be involved.

Neighborhood deprivation may operate through at least three pathways. First, deprivation may reduce walking[52–54] and outdoor physical activity[27,55] due, in part, to perceptions of safety and fear of crime.[47,56] Second, the association may be purely economic (poor people can afford only cheap, calorie-dense foods that are more likely to lead to obesity).[57] Finally, living in a socioeconomically deprived neighborhood may expose residents to psychosocial hazards leading to chronic activation of, and dysregulation of, the hypothalamic–pituitary–adrenal (HPA) axis response.[34] The NPH was independently related to obesity, while the more commonly employed deprivation measures were more weakly associated (Townsend Index) or were not associated (Index of Neighborhood Affluence). These data are consistent with the theory that prolonged exposure to environments that evoke vigilance, threat, and alarm—the a priori hypothesis—may be an important and modifiable contributor to the epidemic of obesity. Beyond the behavioral pathways examined, living in psychosocially hazardous neighborhoods might plausibly lead to increased body weight through stress pathways.[33,36] In humans, the presence of psychosocial hazards results in increased glucocorticoid secretion, which in turn promotes visceral adiposity and the metabolic syndrome.[34,58] This effect operates directly through the central appetite regulation system.[59] These data are consistent with an environmental stress theory for the role of the social environment in obesity, but remains speculative given that biomarkers of HPA axis dysregulation were not included in this analysis.

Strengths of this study include (1) greater precision in the measurement of individual socioeconomic status (with the addition of household assets as well as income), (2) ability to adjust for diet and physical activity, (3) use of multilevel models to account for correlations of persons within neighborhoods, (4) a rich array of neighborhood measures independent of study participants that were combined into a scale using factor analysis, and (5) availability of a neighborhood geography that may be more valid than census tracts. This study is an important advance over previous studies, which were mainly ecologic in design, because it combined information gathered separately on people and places.[60]

A potential criticism is that these results may be explained by the overwhelming influence of socioeconomic status or race/ethnicity on obesity. Neighborhood associations may represent residual confounding due to unmeasured heterogeneity in individual social position or race/ethnicity.[61] It is not possible to completely rule this out. However, several factors mitigate this criticism. First, the multilevel models used adjust for the similarity among people living in the same neighborhood and the effect of individual and household economic status using strong measures of education and household wealth. Second, several indicators that make up the NPH are not likely to be direct measures of socioeconomic status (e.g., street conditions, 911 calls, and rates of marital disruption). Third, recent findings using National Health and Nutrition Examination Survey (NHANES) data suggest that socioeconomic disparities in obesity narrowed significantly between 1971 and 2000,[62] making residual confounding less likely. Finally, stronger associations were observed for the NPH compared to standard measures of deprivation (Townsend Index) or affluence. Drewnowski and Specter[57] argue that obesity is associated with socioeconomic position because healthy food is too expensive compared to energy-dense foods. The absence of an association between neighborhood affluence and obesity is suggestive of alternative pathways.

Another potential criticism is that no adjustment was made for chronic disease or disability. However, a growing literature on the health consequences of living in deprived neighborhoods shows that poor health may lie on the causal pathway.[63–65] Therefore, adjustment for these factors would lead to an under-estimation of the association of neighborhood conditions and obesity. However, these models were re-fit after adjusting for the presence of disability; the main inferences were unchanged.

The public health significance of these findings may lie in the additional evidence provided that neighborhood conditions can alter population patterns of obesity. The urgency of developing environmental strategies to reduce the burden of disease that results directly and indirectly from obesity is increasing. These data support the importance of focusing on places rather than (or in addition to) the individuals who live in those places. These results suggest the potential utility of community-level interventions that might lead to reductions in psychosocial hazards. Several components of the NPH are amenable to change including public safety, crime rates, vacant housing, and educational attainment. Ecologic approaches to obesity prevention have begun to be tested focusing on urban design and food availability.[66]

In previous centuries, major advances in the control of infectious diseases like cholera and tuberculosis came from environmental changes involving public sanitation. The modern epidemics of obesity, diabetes, and cardiovascular disease may require a return to this basic strategy. These studies suggest that community redevelopment, housing policy, and blight reduction, in addition to other benefits, may contribute to improvements in population health.

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