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Neighborhood Racial Diversity and Metabolic Syndrome: 2003–2008 National Health and Nutrition Examination Survey

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Abstract

This study investigated the independent association between neighborhood racial/ethnic diversity and metabolic syndrome among US adults, and focused on how this association differed across individual and neighborhood characteristics (i.e., race/ethnicity, sex, age, urbanity, neighborhood poverty). Objectively-measured biomarker data from 2003 to 2008 National Health and Nutrition Examination Survey were linked to census-tract profiles from 2000 decennial census (N = 10,122). Multilevel random intercept logistic regression models were estimated to examine the contextual effects of tract-level racial/ethnic diversity on individual risks of metabolic syndrome. Overall, more than 20% of the study population were identified as having metabolic syndrome, although the prevalence also varied across demographic subgroups and specific biomarkers. Multilevel analyses showed that increased racial/ethnic diversity within a census tract was associated with decreased likelihood of having metabolic syndrome (OR 0.71, 95% CI 0.52–0.96), particularly among female (OR 0.64; 95% CI 0.43–0.96), young adults (OR 0.60; 95% CI 0.39–0.93), and residents living in urban (OR 0.67; 95% CI 0.48–0.93) or poverty neighborhoods (OR 0.54; 95% CI 0.31–0.95). The findings point to the potential benefits of neighborhood racial/ethnic diversity on individual health risks.

Keywords

Health disparities; Neighborhoods; Race; Ethnicity; Biomarkers; Metabolic syndrome

Introduction

Metabolic syndrome (MetSyn) refers to a specific cluster of biomarkers that can directly prompt individual risks for developing health problems such as cardiovascular diseases and diabetes. These biomarkers include a large waistline, a high tryglyceride level, a low HDL cholesterol level, high blood pressure, and high fasting blood sugar. People with MetSyn are twice as likely to develop heart disease, and five times as likely to have type 2 diabetes [1]. Common factors for developing MetSyn include abdominal obesity, physical inactivity,

atherogenic diet, and insulin resistance [2]. Many of these underlying risks are behavioral and lifestyle factors, suggesting that public health efforts can be extended to prevent individuals from MetSyn risks.

Prevalence of MetSyn is high among US adults and has increased in the past decades. Nationwide analyses showed that age-adjusted prevalence rose from 29.2% during the years 1988–1994 to 34.2% during 1999–2006 [3]. Further examination across demographic subgroups revealed that younger women had seen the greatest increase in recent years [3]. Evidence also suggested that observed racial/ethnic differences in MetSyn prevalence were not substantially attenuated by individual predictors [4]. There is much need to look beyond individual characteristics and to explore whether contextual or environmental mechanisms may possibly shape these disparities at the population level.

Race-based residential segregation is arguably a fundamental cause of racial disparities in health in the United States (US) [5]. Empirical research on neighborhood racial/ethnic contexts and biomarkers, however, are limited. One study analyzing 1988–1994 National Health and Nutrition Examination Survey (NHANES) data found that both the unequal distribution of minority groups and the degree of potential social contact between minority and majority group members were associated with allostatic load [6]. The authors pointed out that both whites and blacks paid a health penalty for racial residential segregation. Another nationwide study also confirmed the deleterious effects of metropolitan-level segregation on hypertension, net of individual and spatial socioeconomic status (SES) [7]. A recent study in Pennsylvania found that Latino co-ethnic concentration was associated with elevated risks of high blood pressure and cholesterol level [8]. Such evidence suggests that neighborhood racial/ethnic contexts do play salient roles in shaping individual biomarkers.

Other dimensions of community racial/ethnic context, racial/ethnic diversity in particular, warrant further examination. There are several underlying mechanisms explaining why racially and culturally diverse neighborhoods may protect individuals from health risks. Net of area deprivation and neighborhood adversity, diverse neighborhoods in urban areas may provide diverse housing types and mixed land use, probably as a response to differential needs of social and demographic heterogeneity [9]. Such land use and urban design can provide favorable resources for physical activities, especially for non-leisure time activities [10]. Local food environment also shapes residents' energy intake, and neighborhoods with larger share of immigrants tend to have healthier food environment [11]. This may be attributable to immigrants' low energy-dense diet in their original cultures, which results in the availability of healthier ethnic food. Residents in less segregated and immigrant-concentrated neighborhoods both witness beneficial dietary intake [11, 12].

In addition to the physical presence of health-promoting resources, community subcultural orientation may influence residents' health behavior decisions [13]. For instance, percentage of residents walking to work and percentage being obese in a neighborhood are both associated with obesity risks [14, 15]. If a racially diverse neighborhood has more residents engaging in transportation-related or other types of physical activity, or obesity prevalence is lower in this neighborhood, it is possible that such pro-health environment can influence residents' behaviors and lifestyle choices. From the psychosocial perspective, increased

share of the minority populations within residential neighborhoods may prevent minorities from discrimination by reducing their exposure to such stressors that might heighten their physiological dysfunction [16]. Taken together, these mechanisms suggest that neighborhood racial diversity may prevent individuals from MetSyn-related risk factors such as physical inactivity, atherogenic diet intake, and stress.

There is also increasing consensus that the neighborhood–health link is not homogeneous, and more research are needed to examine differential associations across population subgroups [17, 18]. Much evidence shows that neighborhood effects are stronger and more robust among women across a range of health outcomes [15, 19, 20]. A common explanation is that women tend to spend more time at home than men do, so they are more exposed to, hence are more influenced by, various aspects of neighborhood environment. Yet some evidence has suggested inconsistent patterns in this gender dynamic [21]. Furthermore, because biomarkers are considered pre-disease conditions and usually reach to a risky level well ahead of actual physiological change, identifying the environment by age interaction can advance our understanding of residential neighborhoods' different impacts on individuals over the life course.

Neighborhood SES is shown not only to impact individuals directly, but can also condition the effects of other contextual predictors on health [22, 23]. This is especially true with regard to racial diversity considering the intertwined patterns of residential economic and racial segregation in the US [24, 25]. For example, one study of blacks living in New York City found that black concentration was detrimental for physical health and life satisfaction when neighborhood income was low, but this association was reversed in high income black neighborhoods [22]. Another study in Texas also showed that the association between Hispanic concentration and obesity prevalence varied by county-level educational attainment [23]. Finally, given different racial makeup in urban and rural areas [26, 27], it is important to examine whether the association between racial diversity and Met-Syn is likely to differ by levels of urbanization. Research suggested that renter-occupied and low-income households, along with individuals who appreciate social heterogeneity, have stronger preference for compact development [9]. Such urban and compact design may in turn encourage residents to take public transportation or engage in other types of daily activities, and further reduce their risk factors for MetSyn.

In sum, there is clear need to investigate residential ethno-racial diversity and pre-disease biomarkers such as MetSyn to further our understanding of how changing demographic patterns at the societal level shape health risks, and how these social forces impact various subgroups differentially. This study seeks to examine (1) the independent association between neighborhood racial/ethnic diversity and MetSyn among US adults and (2) how this association differs by race/ethnicity, sex, age, urbanity, and neighborhood poverty.

Methods

Data and Sample

Individual-level data in the current study were drawn from the 2003–2008 National Health and Nutrition Examination Survey (NHANES), a series of pooled cross-sectional surveys of

about 5000 American children and adults conducted each year by the National Center for Health Statistics of the Centers for Disease Control and Prevention. The NHANES survey design was based on stratified, multistage probability sampling of the civilian non-institutionalized US residents. More detailed sampling and data collection procedures are provided on the NHANES website: http://www.cdc.gov/nchs/data/series/sr_02/sr02_161.pdf. Because NHANES is a nationally representative survey, results are generalizable to the whole US population. In the analytical sample, we excluded pregnant women and only included respondents aged 20–64 years.

The individual-level data were then linked to the 2000 decennial census, where census-tract socioeconomic and demographic profiles were obtained. The size of residential boundaries has been a debated issue in researching neighborhood and health [28], as variations in spatial scale may encompass different contextual processes underlying health risks. While contextual features of small aggregation such as census tract may be more salient on individual behaviors as they represent more immediate and relevant social and built environment, larger geographic units like county and Metropolitan Statistical Area (MSA) may better capture structural forces resulting from policy influences and levels of social hierarchy. Empirical evidence suggested that contextual influences such as income inequality and residential segregation could be more robust in larger contexts [6, 29–31]. However, because we speculated that local physical and social environment were the primary pathways linking racial diversity and MetSyn through behavioral and lifestyle factors [32], we used census tract as the unit of analysis at the neighborhood level.

Outcome Variable

Metabolic Syndrome (MetSyn)—We followed the criteria proposed by the American Heart Association and National Heart, Lung, and Blood Institute [2], and determined MetSyn if a respondent had at least three of the following five biological risk factors:

1. Elevated blood pressure (systolic blood pressure \geq 130 mm/Hg or diastolic blood pressure \geq 85 mm/Hg);
2. Central adiposity (waist circumference \geq 102 cm for men; \geq 88 cm for women);
3. Low serum HDL ($<$ 40 mg/dL for men; $<$ 50 mg/dL for women);
4. Elevated triglycerides (\geq 150 mg/dL);
5. Elevated fasting glucose (\geq 100 mg/dL).

Thus the outcome variable used in our analyses was a binary measure indicating whether a respondent had MetSyn (yes vs. no).

Neighborhood-Level Variables

Racial Diversity—The index of racial/ethnic heterogeneity was used to represent racial/ethnic diversity. It was defined as $1 - \sum_i p_i^2$, where p_i is the fraction of the population in a

given census tract. This index takes into account both the relative size and number of groups in the populations, with a value of 1 reflecting maximum diversity, and a value approaching

0 reflecting the presence of only one racial/ethnic group within a census tract. The calculation of the index was based on proportions of non-Hispanic whites, non-Hispanic blacks, Asians/Pacific Islander, Hispanics, American Indians/Alaska Natives, and other racial/ethnic groups in a census tract.

Poverty Concentration—Neighborhood poverty concentration was investigated as both a control variable and a moderating factor in the current study. We chose to focus on poverty instead of other aspects of neighborhood SES because spatial concentration of poverty oftentimes intersects with residential segregation and individual poverty among racial/ethnic minorities [24, 25]. Following the categorization of US Census Bureau, poverty concentration was dichotomously measured by whether a census tract had at least 20% of residents living below the poverty line [33].

Urbanity—We used the U.S. Department of Agriculture (USDA) 2000 primary rural–urban commuting areas (RUCA) codes to distinguish urban and non-urban neighborhoods in this study. The RUCA codes were based on measures of population density, urbanization and daily commuting. We classified census tracts into 10 categories, with categories 1–3 being metropolitan tracts (areas with 50,000 or more people), categories 4–6 being micropolitan tracts (areas between 10,000 and 49,999 people), categories 7–9 being small towns (areas between 2500 and 10,000 people), and category 10 being small rural (areas less than 2500 people) [34]. Following the White House Office of Management and Budget (OBM) definition, we defined urban neighborhoods as all metropolitan tracts (RUCA codes 1–3) and the rest as non-urban neighborhoods (RUCA codes 4–10).

Individual-Level Variables

Socio-demographic Characteristics—They included race/ethnicity, age, sex, marital status, nativity status, educational attainment, and household income. In addition to the continuous measure of *age*, an age-squared term was added in the models to account for possible curvilinear relationship between age and biomarkers. Self-reported race and ethnicity included non-Hispanic whites (hereafter “whites”), non-Hispanic blacks (hereafter “blacks”), US-born Hispanics, and foreign-born Hispanics. We specifically distinguished nativity status among Hispanics because prior studies suggested that US-born Hispanics had higher biological risks than foreign-born Hispanics [35]. Respondents identified themselves as other racial/ethnic categories were excluded in the analysis. *Sex* (male vs. female), *marital status* (married or living with partner vs. single, separated, divorced or widowed), *nativity* (US born vs. foreign born) were all coded as binary variables. *Educational attainment* included four categories: less than high school, high school graduate, some college, and college degree or higher. Besides educational attainment, a continuous variable of household *income-poverty ratio* was included in the analysis as another indicator of individual SES. This measure was based on the ratio of total household income divided by the federal poverty threshold for the appropriate family size.

Prescribed Medication Use—Because patients whose biomarkers were diagnosed at risky levels were likely to use drug treatment to control their elevated risks, all models adjusted for medication use available in the NHANES interview data. This included self-

reported responses to two survey questions asking whether the respondent was taking prescribed medicine to control for high blood pressure or high cholesterol level, respectively (yes vs. no).

Statistical Analysis

We first presented weighted crude prevalence of MetSyn along with its five specific biomarkers for the full sample and subsamples (Table 1). Then we estimated a series of multilevel random intercept logistic regression models with individual predictors at Level 1 and tract-level predictors at Level 2 [36]. We started by analyzing the full sample and examined the independent effects of neighborhood racial/ethnic heterogeneity, while adjusting for individual-level covariates and neighborhood-level poverty concentration (Table 3). Then we conducted stratified analyses across subsamples to detect whether the racial diversity-MetSyn association differed by gender, age, urbanity, and neighborhood poverty (Tables 4 and 5). In each set of the stratified analyses, Model 1 tested the crude effect of neighborhood ethnic heterogeneity on MetSyn, while adjusting for individual-level controls and neighborhood urbanity. Then in Model 2, neighborhood poverty concentration was included to see if the effect of ethnic heterogeneity remained statistically significant net of area deprivation. All analyses were performed in SAS software with GLIMMIX procedure, and were remotely accessed through the National Center for Health Statistics Research Data Center. The study was approved by the Institutional Review Board at the University of Utah.

Results

Descriptive Statistics

Table 1 presented weighted prevalence of having MetSyn along with its specific biomarkers among US adults aged 20–64. Overall, about 20.5% of our study population had MetSyn. Specific to demographic subgroups, men (21.8%), the middle-aged (28.5%) and non-urban residents (22%) witnessed higher MetSyn prevalence compared to women (19.2%), young adults (14.6%) and urban residents (20%). Among the five MetSyn biomarkers, low serum HDL (56.1%) and waist obesity (49%) were most prevalent among US adults, and elevated fasting glucose (17.9%) and elevated triglycerides (13.4%) were less prevalent. This pattern was similar across subsamples. Overall, women had more favorable outcomes in MetSyn, but waist obesity was more prevalent among women (57.8%) than among men (40.3%). Compared to the middle-aged and non-urban residents, young adults and urban residents in general witnessed more favorable MetSyn outcomes.

Weighted sample characteristics of individual and neighborhood covariates were presented in Table 2. As the NHANES surveys were designed to be nationally representative, socio-demographic characteristics in our analytical sample were largely comparable to the US population. The present study limited respondents to ages 20–64, so the average age was about 41 years old. Our sample consisted slightly more male (50.4%) than female (49.6%). The majority were white respondents (73.4%), and blacks accounted for 12.7%. There were more foreign-born Hispanics (9.1%) than US-born Hispanics (4.9%). At the neighborhood level, most respondents lived in urban areas (75%). The measure for racial diversity (index

of racial/ethnic heterogeneity) was at an average of 0.29 in the full sample, but was much higher for urban residents (0.32) compared to rural residents (0.20). As to poverty concentration, about 15.4% of the respondents lived in census tracts where at least 20% of their neighbors in the same tract were in poverty. There was no notable difference regarding neighborhood poverty concentration across gender or urbanity groups, but more younger adults (17.6%) lived in poverty neighborhood than the middle-aged (12.4%).

Multilevel Analyses Predicting Metabolic Syndrome

Turning to regression analyses, Table 3 presented results from two-level random intercept logistic models predicting individual odds of having MetSyn in the full sample. This full model predicted the odds ratio (OR) of MetSyn as a function of a set of individual- and neighborhood-level covariates. Compared to whites, blacks had lower risks of having MetSyn in this adjusted model (OR 0.84, 95% CI 0.72–0.97), but neither US-born nor foreign-born Hispanics showed any significant differences compared to whites. Increased age (OR 1.13; 95% CI 1.09–1.16), being married (OR 1.13; 95% CI 1.01–1.26), and being US-born (OR 1.34; 95% CI 1.01–1.76) were all positively associated with MetSyn. Socioeconomic indicators such as having a college degree (OR 0.69; 95% CI 0.58–0.82) and higher income (OR 0.95; 95% CI 0.92–0.99) were significantly associated with decreased risks of having MetSyn. At the neighborhood level, both economic profiles and racial/ethnic composition of a community were significantly associated with residents' MetSyn risks. After adjusting for neighborhood poverty concentration, which was associated with higher odds of having MetSyn itself (OR 1.19; 1.03–1.37), racial diversity was associated with lower odds of having MetSyn (OR 0.71; 95% CI 0.52–0.96). This result suggested that racial diversity of a community was significantly associated with residents' risks of having MetSyn, and this relationship was independent of a community's economic structure.

In Tables 4 and 5, we presented results from multilevel models stratified by individual gender, age, and neighborhood urbanity and poverty concentration. Because this study focused on the contextual influence of neighborhoods, we only presented regression estimates for neighborhood-level predictors and omitted individual-level covariates in the tables. Across all the four stratified analyses, Model 1 estimated the crude effect of neighborhood racial diversity on MetSyn, and Model 2 was the full model that further adjusted for neighborhood poverty concentration (as in Table 3).

The first part of Table 4 presented differential associations between women and men. Among women, living in a racially diverse neighborhood was associated with decreased MetSyn risks (Model 1: OR 0.64; 95% CI 0.43–0.96), although adding neighborhood poverty rendered the effect of racial diversity marginally significant in Model 2 (OR 0.68; 0.45–1.02). Neighborhood poverty concentration itself was associated with women's MetSyn risks (OR 1.27; 1.06–1.53). Results did not show any significant effect of either racial diversity or neighborhood poverty among men.

Table 4 also presented stratified analyses between younger adults and the middle-aged. It showed that increased racial diversity in a neighborhood was consistently and significantly associated with decreased risks of having MetSyn among younger adults between 20 and 44 years old, both before (OR 0.60; 0.39–0.93) and after (OR 0.61; 0.40–0.94) adjusting for

neighborhood poverty. But this association did not seem to exist among middle-aged adults aged between 45 and 64 years. Living in a poverty neighborhood was not significantly associated with the likelihood of having Met-Syn in this set of analyses.

Results for stratified analyses by urbanity and neighborhood poverty were reported in Table 5. As shown, increased racial diversity in neighborhood was significantly associated with lower risks of MetSyn among urban residents, regardless of controlling for neighborhood poverty (Model 1: OR 0.67, 95% CI 0.48–0.93; Model 2: OR 0.69, 95% CI 0.50–0.96). Neighborhood poverty concentration was only marginally associated with MetSyn in urban areas (OR 1.17; 1.00–1.36). In contrast, among non-urban neighborhoods, neither racial diversity nor poverty concentration was significantly associated with MetSyn. Finally, with regard to neighborhood poverty concentration, racial diversity was significantly and inversely associated with having MetSyn in high poverty neighborhoods (OR 0.54; 95% CI 0.31–0.95). This association was not statistically significant among residents living in neighborhoods of low poverty concentration.

Discussion

Using objectively measured biomarker data from 2003 to 2008 NHANES survey, this nationwide study examined contextual effects of residential racial/ethnic diversity on MetSyn, a cluster of biomarkers that prompt health problems like cardiovascular diseases and diabetes. Our analyses particularly focused on potential modifiers such as gender, age, urbanity, and poverty concentration. Results suggested that increased racial/ethnic diversity in a neighborhood was significantly associated with lower risks of having MetSyn for US adults, particularly among women, young adults, and residents living in urban and poverty neighborhoods.

Past research has often used single-group segregation indices or co-ethnic concentration. The present study has expanded this line of work by applying the index of ethnic heterogeneity to operationalize multi-group composition within census tracts; thus, it allows the opportunity to assess the influence of diversity on individuals' immediate residential environment. Focusing on racial diversity as a distinct dimension of neighborhood racial/ethnic context enables us to better detect the health impact of living in a diverse social environment, which are often shadowed by using measures such as co-ethnic concentration. Our findings point to the salutary benefits of residential racial diversity on MetSyn, a set of cardiovascular disease-related biomarkers.

Our examination of differential associations in the neighborhood-health link was motivated by the inconsistent findings in the past literature as well as the lack of evidence in a few understudied modifiers. Neighborhood effects are complex, and it is crucial to test whether the effects of one contextual predictor would change according to other independent variables [18]. An important finding of the current study is the differential associations of neighborhood racial diversity and MetSyn by poverty concentration. This finding echoes a previous study of linguistic diversity and neighborhood violence, where Sampson reported that the protective effects of linguistic diversity on neighborhood violence were stronger in high disorder and high poverty neighborhoods and implied that diversity and immigration

might have re-energized historically disadvantaged neighborhoods [37]. Such pattern was also detected in health outcomes. For example, a nationwide study of adolescents found that higher immigrant concentration particularly buffer against obesity risks in poor neighborhoods [38].

The beneficial influences of racial diversity also seem to only exist among urban neighborhoods. This finding is in accordance with our expectation as the racial and demographic makeup largely differ between urban and non-urban neighborhoods [26, 27]. As noted above, racially diverse urban residence not only provides residents with denser and mixed housing types with better street connectivity, a health-promoting built environment; they may further create a subculture with higher prevalence of walking and lower obesity prevalence [14, 15]. As metropolitan cities also serve as major destinations for most immigrants, they may benefit from the fact that immigrants can form a healthier food environment with various options of ethnic foods and stores. These are all mechanisms contributing to the risks of developing MetSyn and can explain why urban neighborhoods observed stronger effects of racial diversity in this study.

Few studies in the past literature have explored how neighborhood effects on health vary by age groups. Contrary to an early study of neighborhood SES and physical health that showed stronger contextual effects among older adults [39], this study found that protective effects of racial diversity were stronger among younger adults aged between 20 and 45 years old. As speculated, because the biological “wear and tear” process starts early in the life course, perhaps even traced back to the childhood and adolescence [40–42], neighborhood environment can exert influences on individual physiological dysregulation such as MetSyn during early life stages, as compared to later life stages when the morbidity process actually occurs. Another possibility for this age variation may relate to the specific contextual predictor examined in the analysis. As the “new urbanism” has been an explanation for the protective effects of neighborhood diversity [37], and the younger generation is perhaps the major group particularly attracted to its glamour, it is not surprising to find the positive influences of diversity only among this age group.

Our finding is also consistent with previous studies of residential segregation showing stronger neighborhood effects among women [15, 19, 20], we found that protective effects of racial diversity were more salient among women than among men. This result may be due to gender differences in their responses to a homogeneous or a diverse residential environment. It may also be explained by different employment or occupational status and lifestyle factors that vary between urban and non-urban residents across gender groups. Future research may further explore whether differences in occupation, mode of transportation and diet pattern have contributed to such sex differences.

This study is not without limitations. First, the cross-sectional design of this analysis has limited the possibility in handling selection bias as a result of the nonrandom nature of individuals’ neighborhood choice, thus disallowing any causal inference of contextual influences on MetSyn risks. The selection bias may vary systematically across social groups leading to differential effects of racial diversity among different groups. Second, although we have speculated several underlying mechanisms linking neighborhood racial diversity

and MetSyn, such as local built environment and healthy food availability, this study only focused on effect modification and did not directly test potential mediators. Future research may inquire into such mechanisms to further our understanding of the values of diversity.

Current racial discourse and ongoing debate on immigration in the US have stimulated soaring scholarship to examine influences of neighborhood racial/ethnic composition on various aspects of social life and population well-being in general. As the US is moving towards a minority-majority society, it will undoubtedly expect continuing debates surrounding race and immigration. This study joins others and provides fresh and important evidence confirming the salutary effect of racial diversity. It demonstrates that residential racial diversity can have potential benefits preventing residents' health risks, especially among certain social groups. Findings from this research can serve as an important basis for relevant policy makers, public health practitioners, and urban designer in their efforts to prevent chronic diseases while incorporating multi-dimensional societal factors and demographic changes.

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Prevalence of metabolic syndrome and specific biomarkers, full and subsamples (NHANES 2003–2008)

Table 1

	Full sample	Women	Men	Age 20–44	Age 45–64	Urban	Non-urban
Metabolic syndrome (have at least three of the following five biomarkers)	20.48%	19.18%	21.79%	14.56%	28.49%	19.96%	22.04%
Specific biomarkers							
Elevated blood pressure (systolic blood pressure ≥ 130 mm/Hg or diastolic blood pressure ≥ 85 mm/Hg)	24.47%	20.30%	28.64%	15.17%	37.05%	23.54%	27.27%
Waist obese (waist circumference ≥ 102 cm for men; ≥ 88 cm for women)	49.02%	57.76%	40.29%	41.48%	59.21%	47.04%	54.94%
Low serum HDL (< 40 mg/dL for men; < 50 mg/dL for women)	56.10%	57.04%	55.16%	58.91%	52.28%	56.83%	53.90%
Elevated triglycerides (≥ 150 mg/dL)	13.43%	10.34%	16.52%	11.38%	16.21%	13.51%	13.21%
Elevated fasting glucose (≥ 100 mg/dL)	17.92%	14.35%	21.50%	12.60%	25.12%	17.41%	19.47%
Observations	10,122	5019	5103	5697	4425	7986	2136

Statistics are weighted and are reported in percentages

Table 2
 Descriptive statistics of individual and neighborhood variables, full and subsamples (NHANES 2003–2008)

	Full sample	Women	Men	Age 20–44	Age 45–64	Urban	Non-urban
Individual-level variables							
Age	41.17 (0.25)	41.61 (0.26)	40.74 (0.30)	32.33 (0.18)	53.14 (0.13)	40.63 (0.25)	42.81 (0.49)
Race/ethnicity							
White	73.37%	73.31%	73.43%	69.15%	79.09%	68.42%	88.21%
Black	12.65%	13.58%	11.71%	13.27%	11.81%	14.86%	6.01%
US-born Hispanic	4.87%	5.20%	4.55%	6.09%	3.22%	5.58%	2.76%
Foreign-born Hispanic	9.11%	7.91%	10.31%	11.49%	5.88%	11.14%	3.02%
Married	65.41%	63.63%	67.19%	60.99%	71.38%	63.44%	71.33%
US born	86.10%	87.60%	84.61%	83.68%	89.38%	83.24%	94.70%
Education							
Less than high school	16.88%	15.52%	18.25%	17.89%	15.52%	16.44%	18.22%
High school	25.64%	23.93%	27.34%	25.49%	25.84%	28.36%	32.49%
Some college	32.44%	34.74%	30.14%	32.61%	32.21%	32.12%	33.39%
College degree or higher	25.04%	25.81%	24.27%	24.01%	26.43%	23.08%	15.90%
Income-poverty ratio	3.10 (0.05)	3.03 (0.05)	3.17 (0.05)	2.84 (0.05)	3.45 (0.06)	3.19 (0.05)	2.82 (0.09)
Medication for blood pressure	15.03%	15.50%	14.55%	5.25%	28.25%	13.78%	18.76%
Medication for cholesterol	9.71%	8.99%	10.44%	2.87%	18.97%	9.48%	10.42%
Neighborhood-level variables							
Racial diversity	0.29 (0.02)	0.29 (0.02)	0.29 (0.02)	0.30 (0.02)	0.27 (0.02)	0.32 (0.02)	0.20 (0.04)
Poverty concentration 20%	15.37%	15.15%	15.58%	17.56%	12.41%	15.49%	15.01%
Observations	10,122	5019	5103	5697	4425	7986	2136

Descriptive statistics are weighted and are reported in percentages or means. Standard errors are in parentheses

Table 3

Odds ratio from multilevel logistic regression models predicting metabolic syndrome, full sample (NHANES 2003–2008)

	Full sample
Individual-level variables	
Race/ethnicity (ref. white)	
Black	0.84 (0.72–0.97) *
US-born Hispanic	1.09 (0.90–1.32)
Foreign-born Hispanic	1.27 (0.93–1.73)
Age	1.13 (1.09–1.16) ***
Age-squared	1.00 (1.00–1.00) ***
Men (vs. women)	1.00 (0.91–1.11)
Married (vs. single/divorced/widowed)	1.13 (1.01–1.26) *
US born	1.34 (1.01–1.76) *
Education (ref. high school)	
Less than high school	1.05 (0.91–1.22)
Some college	1.01 (0.88–1.16)
College degree	0.69 (0.58–0.82) ***
Income-poverty ratio	0.95 (0.92–0.99) *
Medication for blood pressure (vs. no)	2.17 (1.91–2.48) ***
Medication for cholesterol (vs. no)	1.32 (1.13–1.55) ***
Neighborhood-level variables	
Racial diversity	0.71 (0.52–0.96) *
Poverty concentration 20% (vs. <20%)	1.19 (1.03–1.37) **
Urban (vs. non-urban)	1.10 (0.94–1.30)
Intra-class correlation coefficient	0.06
Goodness-of-fit indexes	
Log Pseudo-likelihood	50222.73
Generalized χ^2	9585.48
Observations	10,523
Number of tracts	1620

95% confidence intervals are in parentheses

 $p < 0.001$,

**
 $p < 0.01$,

*
 $p < 0.05$ (two-tailed test)

Table 4

Odds ratios from multilevel logistic regression models predicting metabolic syndrome, stratified by gender and age groups (NHANES 2003–2008)

	Women		Men	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level variables				
Racial diversity	0.64 (0.43–0.96) *	0.68 (0.45–1.02) ⁺	0.80 (0.53–1.20)	0.80 (0.53–1.21)
Urban ^a	1.01 (0.82–1.24)	1.00 (0.81–1.22)	1.14 (0.93–1.40)	1.14 (0.93–1.40)
Poverty concentration 20% ^b		1.27 (1.06–1.53) *		1.09 (0.90–1.32)
Intra-class correlation coefficient	0.04	0.04	0.06	0.06
Goodness-of-fit indexes				
Log Pseudo-likelihood	24976.92		25349.27	
Generalized χ^2	4776.28		4861.31	
Observations	5186	5186	5337	5337
Number of tracts	1489	1489	1484	1484
<hr/>				
	Age 45–65		Age 18–44	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level variables				
Racial diversity	0.60 (0.39–0.93) *	0.61 (0.40–0.94) *	0.77 (0.54–1.18)	0.83 (0.56–1.22)
Urban ^a	1.18 (0.94–1.48)	1.18 (0.94–1.48)	1.01 (0.83–1.23)	1.00 (0.82–1.22)
Poverty concentration 20% ^b		1.17 (0.96–1.43)		1.18 (0.98–1.42)
Intra-class correlation coefficient	0.06	0.06	0.06	0.06
Goodness-of-fit indexes				
Log Pseudo-likelihood	29397.47	29420.51	20498.71	20505.46
Generalized χ^2	5262.15	5278.99	4281.60	4281.96
Observations	5932	5932	4591	4591
Number of tracts	1428	1428	1331	1331

95% confidence intervals are in parentheses. All models adjusted for individual-level variables of age, age-squared, race/ethnicity, marital status, nativity status, education, income-poverty ratio, and medication use for blood pressure and cholesterol

 $p < 0.001$,

**
 $p < 0.01$,

*
 $p < 0.05$,

⁺
 $p < 0.10$

^aReference group is non-urban ^bReference group is poverty concentration < 20%

Table 5

Odds ratios from multilevel logistic regression models predicting metabolic syndrome, stratified by urbanity and neighborhood poverty (NHANES 2003–2008)

	Urban		Non-urban	
	Model 1	Model 2	Model 1	Model 2
Neighborhood-level variables				
Racial diversity	0.67 (0.48–0.93) *	0.69 (0.50–0.96) *	0.73 (0.30–1.79)	0.72 (0.30–1.73)
Poverty concentration 20% ^a		1.17 (1.00–1.36) ⁺		1.28 (0.90–1.81)
Intra-class correlation coefficient	0.07	0.07	0.06	0.06
Goodness-of-fit indexes				
Log Pseudo-likelihood	39882.72	39897.63	10354.53	10360.75
Generalized χ^2	7484.13	7488.31	2049.68	2056.64
Observations	8334	8334	2189	2189
Number of tracts	1475	1475	172	172
<hr/>				
	Poverty concentration < 20%		Poverty concentration 20%	
	Model 1		Model 1	
Neighborhood-level variables				
Racial diversity	0.54 (0.31–0.95) *		0.74 (0.51–1.07)	
Urban ^b	1.22 (0.83–1.80)		1.10 (0.92–1.32)	
Intra-class correlation coefficient	0.08		0.06	
Goodness-of-fit indexes				
Log Pseudo-likelihood	11560.34		38742.57	
Generalized χ^2	2171.09		7377.00	
Observations	2455		8068	
Number of tracts	472		1221	

95% confidence intervals are in parentheses. All models adjusted for individual-level variables of age, age-squared, race/ethnicity, marital status, nativity status, education, income-poverty ratio, and medication use for blood pressure and cholesterol

 $p < 0.001$,

**
 $p < 0.01$,

*
 $p < 0.05$,

⁺
 $p < 0.10$

^aReference group is poverty concentration < 20%

^bReference group is non-urban