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Does place explain racial health disparities? Quantifying the contribution of residential context to the Black/white health gap in the United States

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ABSTRACT

The persistence of the black health disadvantage has been a puzzling component of health in the United States in spite of general declines in rates of morbidity and mortality over the past century. Studies that have focused on well-established individual-level determinants of health such as socio-economic status and health behaviors have been unable to fully explain these disparities. Recent research has begun to focus on other factors such as racism, discrimination, and segregation. Variation in neighborhood context—socio-demographic composition, social aspects, and built environment—has been postulated as an additional explanation for racial disparities, but few attempts have been made to quantify its overall contribution to the black/white health gap. This analysis is an attempt to generate an estimate of place effects on explaining health disparities by utilizing data from the U.S. National Health Interview Survey (NHIS) (1989–1994), combined with a methodology for identifying residents of the same blocks both within and across NHIS survey cross-sections. Our results indicate that controlling for a single point-in-time measure of residential context results in a roughly 15–76% reduction of the black/white disparities in self-rated health that were previously unaccounted for by individual-level controls. The contribution of residential context toward explaining the black/white self-rated health gap varies by both age and gender such that contextual explanations of disparities decline with age and appear to be smaller among females.

Introduction

The traditional focus of research examining the determinants of health status has been on individual-level differences in socio-economic status (SES) and the health behaviors associated with them. Though SES and health behaviors explain a large portion of the health disparities between non-Hispanic blacks and non-Hispanic whites (henceforth referred to as blacks and white, respectively), in the United States, they do not fully account for the gap (Williams & Collins, 1995). Moreover, health care has been found to contribute little to health status (House & Williams, 2000). The persistence of the unexplained health gap between blacks and whites has led many to direct their attention to differences in residential environments as a contributor to observed health disparities (Kaplan, 1996). This increasing

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recognition that the social and structural environment where one lives may independently contribute to the formation and development of human conditions, including health outcomes, suggests a research area that has yet to be fully exploited. In this paper, we generate quantitative estimates of the contribution of local residential context toward explaining the black/white health gap in the United States, net of individual-level characteristics.

Background

Black/white differences in health and mortality

Despite a marked increase in life expectancy in the United States over the last century, a gap in life expectancy between blacks and whites remains (NCHS, 2007). Although this gap has narrowed from seven to just over five years between 1990 and 2004, mortality differences between blacks and whites have grown or have remained unchanged for a number of causes such as heart disease, HIV, diabetes mellitus, and several types of cancer (NCHS, 2007). These trends in mortality are indicative of most of the rates of major morbidities including breast, lung, and colorectal cancer rates, which are 8.5–35% higher among blacks than whites (U.S. DHHS, 2005) and diabetes prevalence, which is 60% more common among blacks than among whites (U.S. DHHS, 2006).

Determinants of racial health disparities

Although some research suggests that racial disparities in health and mortality are completely accounted for by individual-level SES (e.g., Rogers, 1992), most research indicates otherwise. That is, racial disparities in health are substantially reduced, but not completely eliminated when variation in individual-level SES is accounted for (House & Williams, 2000).

Another suggested contributor to these persistent disparities has been the differential rates in deleterious and health-enhancing behavioral profiles across racial groups; however, the inclusion of health behaviors has not been able to fully account for the remaining disparities (Finch, Frank, & Hummer, 2000; Lantz et al., 2001). Lastly, findings from the sociology literature suggest that job availability and quality (Huffman & Cohen, 2004), racism (LaVeist, 2000), and discrimination (Williams, Neighbors, & Jackson, 2003) may be important contributors to racial health disparities.

Genetics and population health

Genetics may play an important role in determining the health of individuals. However, in most cases, the genetic component of adult illnesses is not deterministic; rather, the actual consequences of genetic predispositions are likely highly influenced by their interactions with the environment and life experiences (Baird, 1994). Moreover, as most genetic variation is within—rather than between—populations (Evans, Hodge, & Pless, 1994), genetics alone is unlikely to be a *major* determinant of *population* health. This must be recognized when interpreting the race residual (i.e., race coefficient) in health disparities as race is often so confounded with different definitions based on language commonalities and on regional and national origins, that it is an ineffective construct of genetic variability (Cooper, 1984; Goodman, 2000; Shields et al., 2005; Smedley & Smedley, 2005). Hence, race is likely to capture many social and cultural characteristics that are often unmeasured or incorrectly measured in survey research (Frank, 2007).

Neighborhood context and its impacts on health

Social environment

In addition to individual-level influences on health, a substantial body of literature indicates that where one lives also has important implications for health (Morenoff & Lynch, 2002; Robert, 1999a, 1999b). Most of the existing literature has focused on the social environment including neighborhood racial segregation (Williams & Collins, 2001), neighborhood demographic or socio-economic characteristics (Robert, 1999b), and social cohesion (e.g., social capital, trust, crime) (Sampson, Morenoff, & Gannon-Rowley, 2002).

Literature reviews on the social environment and health reveal there is substantial evidence suggesting constructs such as community SES (e.g., neighborhood poverty rate, unemployment rate), social structures (e.g., racial segregation, income inequality), and the quality of the environment (e.g., services, crime, traffic) are associated with health outcomes and health behaviors including low birthweight, morbidity, activity limitations, and physical activity (Riva, Gauvin, & Barnett, 2007; Yen & Syme, 1999).

One frequently investigated measure is neighborhood disadvantage, commonly quantified using neighborhood-level poverty rate or income level. Areas of concentrated poverty, typically defined as neighborhoods with greater than 20% poverty rate, are associated with a diminished quality of the neighborhood's social and physical environment, high rates of neighborhood turnover and mobility, crime, social disorder (Sampson, Raudenbush, & Earls, 1997), and the attenuation of both individual socio-economic attainment and upward mobility (Collins & Williams, 1999). A host of studies link neighborhood disadvantage to a wide range of detrimental health outcomes, including low birthweight, infant mortality, asthma, tuberculosis, depression, and poor self-rated health (Barr, Diez-Roux, Knirsch, & Pablos-Méndez, 2001; Morenoff & Lynch, 2002; Yen & Kaplan, 1999a, 1999b).

Built environment and urban form

More recently, attention has been paid to the built environment, such as housing conditions, ambient air quality, and urban form (Frank, Engelke, & Schmid, 2003). These studies have found an association between: physical quality of the residential environment and poorer self-reported health status (Cummins, Stafford, Macintyre, Marmot, & Ellaway, 2005; Krause, 1996); higher levels of ambient air pollution and increased use of medical care (Fuchs & Frank, 2002; Fuchs, McClellan, & Skinner, 2001) and mortality

(Jerrett et al., 2005); suburban/urban sprawl and higher rates of motor vehicle fatalities (Ewing, Schieber, & Zegaer, 2003) and homicide-by-stranger deaths (Lucy, 2003); neighborhood problems (e.g., excessive noise, heavy traffic, limited access to public transportation) and functional decline (Balfour & Kaplan, 2002). An over-abundance of fast-food restaurants and small grocery stores have been found to be associated with greater rates of overweight and obesity (Brownell, 2004; Wang, Kim, Gonzalez, MacLeod, & Winkleby, 2007), while density of alcohol outlets has been linked with violence (Gruenewald & Remer, 2006). Finally, access to parks or walking/biking trails is associated with physical activity levels (Huston, Evenson, Bors, & Gizlice, 2003; King et al., 2003) and neighborhoods with a more compact grid design and multiple intersecting streets are positively correlated with the amount of walking of its residents (Ewing, Schmid, Killingsworth, Zlot, & Raudenbush, 2003; Frank et al., 2003).

Hypothesis

Neighborhood context and health disparities

The level of risk and concentration of resources in the social and physical space are clustered in patterns that mimic the larger patterns of stratification in society (Fitzpatrick & LaGory, 2003). That is, individuals of lower SES and racial minorities are segregated spatially (Jargowksy, 1997; Massey & Fong, 1990; Massey & Shibuya, 1995) into areas that contain higher levels of contextual risk factors that promote the creation of urban sub-cultures that lead to higher rates of risky health behaviors (Wilson, 1987), and most importantly, isolation from socio-economic support, resources, and services necessary for health maintenance and upward mobility (Fitzpatrick & LaGory, 2003).

Because exposure to disadvantaged neighborhood environments is delineated strongly along racial lines, it is plausible that residential context is responsible for a significant portion of the black/white disparities in health. However, very few attempts have been made to estimate the proportion of racial health disparities that can be explained by differences in residential context (c.f., Sastry & Hussey, 2003). The intent of this paper is to produce a general estimate of how much residential context, as a whole, might contribute to black/white disparities in self-rated health.

Data and measures

Data set

Our analysis is based on data from the 1989–1994 National Health Interview Survey (NHIS) cross-sections. Since 1957, the NHIS has annually conducted nationwide household interviews to collect information concerning the health of the U.S. civilian non-institutionalized population. The 1989–1994 NHIS sample design is a multi-stage probability sample of U.S. households with new households interviewed each year (Massey, Moore, Parsons, & Tadros, 1989). The survey collects information on race, socio-economic characteristics, and various health measures including self-reported health status.

Neighborhood definition

To capture neighborhood characteristics and processes that may affect health, we rely on "very small areas" (VSAs) in lieu of the traditional census tract proxy for neighborhoods. VSAs are the secondary sampling units used in the NHIS sampling design between 1985 and 1994. (We restrict our sample years to 1989 and later in order to include nativity, initially collected in 1989, in our models. We end our sample year in 1994, as a redesigned sampling framework was implemented after that year.) The approximately 6500 VSAs in our sample represent a wide range of neighborhood contextual environment, as measured by poverty level (mean poverty = 12%, SD = 15). VSAs are smaller than census tracts and are more similar in size to census blocks or block groups. However, these VSAs should be viewed as independent of census areas, as they may overlap census block demarcations.

The VSA strategy, which capitalizes on a unique feature of the NHIS sampling design was developed by Wells and Horm (1998) and later applied by Bond-Huie, Hummer, and Rogers (2002) in their analysis of death rates across racial/ethnic groups. By concatenating the unique NHIS geographic identifiers (random recodes of the primary sampling unit and segment number) and the temporal identifier (calendar quarter of interview), we are able to construct and identify the same VSAs that the NHIS repeatedly sampled across survey years. In short, VSA identifiers allow us to identify residents of the same residential area in multiple years of the NHIS sample. Wells and Horm (1998) estimate that combining survey years provides a sample that captures up to 60% of the block or block group. Using this methodology, we create a VSA identifier for each respondent in our study.

Sample characteristics

Table 1 provides descriptive statistics of our sample as by gender. Race/ethnicity includes seven categories: non-Hispanic white, non-Hispanic black, Asian, other non-Hispanic, Mexican, Puerto Rican, and other Hispanic. Age is measured as a continuous variable and nativity is a binary indicator (U.S. born vs. non-U.S. born).

SES is measured by marital status, family income, education, and labor force status. Educational attainment represents the years of schooling attended (fewer than 12, 12, 13–15, 16, and 17+). Labor force categories include the employed, the unemployed, and the labor force non-participants. Because the reasons for not being in the labor force are likely to differ by age, we divide this status into two categories according to whether the respondent is under 65 or 65 or older. Respondents in the younger group are more likely to be discouraged workers, students, and homemakers, while those in the older group are more likely to be retired. Adjusted family income is in 1986 dollars and was calculated by taking the mean of the income categories and adjusting for inflation and family size (Rogers, Hummer, & Nam, 2000).

Table 1
Descriptive statistics

Variable	Male ($N = 172,753$), %[a]	Female ($N = 196,152$), %[a]
Race/ethnicity		
White	77.66	74.87
Black	10.65	13.58
Asian	2.56	2.52
Other non-Hispanic	1.36	1.32
Mexican	4.55	4.14
Puerto Rican	0.80	0.99
Other Hispanic	2.41	2.58
Nativity		
Foreign born	10.28	10.26
Age (years)		
18–44	58.42	56.47
45–54	15.65	14.82
55–64	11.65	11.41
Above 65	14.28	17.31
Socio-economic demographics		
Marital status		
Single, never married	20.50	16.30
Married	69.70	61.47
Divorced	5.79	8.82
Separated	1.56	2.75
Widowed	2.45	10.66
Income		
Mean adjusted family income (1986 $)	17,1189	15,664
Education		
Non-high school graduate	20.94	20.79
12 Years of school	36.11	40.05
13–15 Years of school	20.44	21.51
16 Years of school	12.23	10.68
17+ Years of school	10.28	6.97
Labor force		
Employed	74.22	57.02
Unemployed	3.58	3.09
Not in the labor force, age under 65	10.52	24.39
Not in the labor force, age 65+	11.68	15.50
Health status		
Weight		
Underweight	1.33	4.91
Normal weight	42.54	55.62
Overweight	42.06	24.23
Obese	14.06	15.25
Activity limitation		
No activity limitation (including unknown)	82.77	81.26
Limited in non-major activities	5.27	6.27
Limited in major activities	5.59	7.28
Unable to perform major activities	6.37	5.19
Self-reported health		
Poor health	3.55	3.63
Fair health	7.96	10.12
Good health	22.92	26.94
Very good health	28.54	28.97
Excellent health	37.02	30.34

[a] Unless otherwise indicated.

Health measures used as additional controls are captured by respondents' weight status (underweight, normal weight, overweight, obese) as categorized by their body mass index level and self-reported level of activity limitation (no activity limitation, limited in non-major activities, limited in major activities, and unable to perform major activities).

Health outcome measure

We use self-rated health (SRH) as our health outcome measure of interest. In our models, SRH status is a continuous numeric indicator of five levels: poor, fair, good, very good, and excellent health, with poor health status reflecting the lowest rank of 1 and excellent health status reflecting the highest rank of 5.

We chose this particular health measure for several reasons: (1) studies show that a poor self-rating of health has predictive value for future mortality above and beyond clinical assessments (Benyamini & Idler, 1999; Idler & Benyamini, 1997); (2) other studies have found relationships between poor SRH and subsequent functional decline and disability (Idler & Angel, 1990); (3) although evidence that SRH is associated with *specific* disease outcomes is limited (Ferraro, Farmer, & Wybraniec, 1997; Menec, Chipperfield, & Perry, 1999), a recent study suggests that SRH is associated with *current* morbidity (Ferraro & Farmer, 1999); and (4) SRH is often more sensitive to change in response to external factors than are physiologic parameters. In short, these studies find that SRH is a well-validated indicator of adult health and mortality risk, above and beyond physician assessments, and that a single measure on a survey can help to capture a great deal of information about one's health status.

However, because SRH is a subjective and not an objective clinical measure of health status, sub-groups of individuals may interpret and respond to these items in different ways. For example, if Hispanic immigrants do not interpret and/or respond to a given health measure the same way as more-acculturated Hispanic or white respondents, then observed differences between these groups on this measure may be artifactual (see, e.g., Finch, Hummer, Reindl, & Vega, 2002). Therefore, if disparities are found between racial groups based on a given measure such as SRH, it is crucial to know whether this measure reflects true health differences or response artifacts. A recent study demonstrated that fair/poor self-ratings of health have similar mortality risks for blacks and whites and for both men and women within each of these racial classifications (McGee, Liao, Cao, & Cooper, 1999). This finding indicates that SRH is a valid measure of future mortality risk for our populations of interest, suggesting that our use of this measure for analyses of health disparities between blacks and whites is valid.

Analytical strategy

Conventional models that investigate racial health disparities may be subject to bias if they omit neighborhood characteristics that contribute to health outcomes and are correlated to race. While one strategy would be to explicitly include neighborhood characteristics as controls, as a number of studies have done (e.g., Yen & Kaplan, 1999a), the reliance on imprecisely measured units of context, the use of administrative data (and hence a scarcity of potential contextual variables), and the high likelihood of omitting

important neighborhood factors, make accounting for all potential neighborhood-level factors that may be correlated to race extremely difficult. Consequently, models that include neighborhood characteristics may still yield biased estimates of racial health disparities if the neighborhood characteristics included are either non-exhaustive or imprecisely measured.

In our analyses, we sidestep these problematic issues by employing a fixed-effect (FE) modeling approach. Here, we use the expression "fixed-effect" model as it is commonly defined in the econometrics literature, where unobserved heterogeneity across cities, persons, etc., that is constant across time is captured by a series of dummy variables denoting each city, person, etc. In this case, we are accounting for the unobserved heterogeneity across neighborhoods by including a VSA fixed effect which represents all neighborhood factors that do not change across time. (For a more complete discussion of these models see Cameron and Trivedi (2005, chp 21).) That is, we specify all VSAs as a series of dichotomous controls in the regression models. These VSA dummies absorb any characteristics that are shared across residents of the same VSA. Thus, rather than attempting to account for all neighborhood-level characteristics correlated with race through the inclusion of an exhaustive set of VSA-level variables (a virtually impossible task), our strategy is to purge the potential correlation from the race estimates through the inclusion of VSA dummies. This eliminates all potential VSA-level contributions to the race residuals.

Since the inclusion of VSA dummy indicators only captures the aggregate effect of neighborhoods on health, our modeling strategy precludes us from identifying specific neighborhood characteristics that are responsible for the reduction, if any, in the race residual. However, in this study, we view the specific neighborhood characteristics as a nuisance rather than a parameter of interest. The goal of the paper is not to identify effects of specific neighborhood characteristics, but rather to quantify the potential overall contribution of place in explaining racial health gaps. That is, we are interested in neighborhood effects only in so far as it may provide further explanation for racial health disparities, above and beyond individual-level characteristics. We do not seek to recover point estimates of specific neighborhood characteristics per se.

To quantify the explanatory power of neighborhoods on black/white health disparities, we compare the black race estimate across two models: an ordinary least squares (OLS) model, which incorporates no area effects; and an FE model, which incorporates neighborhood effects. As previously mentioned, the race residuals in the OLS models are likely to be biased upward due to the exclusion of contextual factors. However, in the FE models, all potential VSA-level contributions are eliminated, providing estimates of coefficients that reflect only individual-level differences across racial groups.

For the OLS model, we estimate SRH, k, of individual i, in VSA j, as a function of person-level socio-economic and demographic variables, x_{ij}, and a race indicator, r_{ij}. The error terms, ε_j and ε_{ij}, reflect omitted neighborhood-level and individual-level characteristics, respectively, as well as random variation.

$$k_{ij} = \beta x_{ij} + \gamma r_{ij} + \varepsilon_j + \varepsilon_{ij} \quad (1)$$

γ is the estimate of racial health disparity. These estimates, however, may be biased if ε_j is correlated with race. To net out all of these potential contextual influences, we estimate an FE model by incorporating a series of VSA dummies. Under the highly plausible assumption that ε_j is correlated with race (i.e., race is confounded with place), the VSA indicators will purge the bias in the race estimate that is due to the correlation between race and neighborhood characteristics. This specification yields the FE model:

$$k_{ij} = \beta x_{ij} + \gamma r_{ij} + \varphi n_j + \varepsilon_{ij} \quad (2)$$

where n_j is a vector of dummy variables, one for each VSA, and the error term now consists of only omitted individual-level attributes (and random variation).

Given that residential context likely has non-constant effects on health and mortality over the life-span (Glass & Balfour, 2003; Haan, Kaplan, & Camacho, 1987; Morenoff & Lynch, 2002; Waitzman & Smith, 1998), we include interactions between race and age to allow the level of racial disparity in health to vary by age. In addition, because females are known to have a different health risk profile than males, and area effects may have differential impacts across gender (Stafford, Cummins, Macintyre, Ellaway, & Marmot, 2005), we perform our models separately for males and females.

Our regression models include the full set of variables listed in Table 1 and a set of year dummies – not shown – to control for any temporal correlations to health and neighborhood context. (Year indicators were significant and suggest that self-reported health has declined with time.) We estimate models both with and without controls for two key aspects of health status (i.e., weight status and major activity limitations). Controlling for overweight and activity limitations serves to absorb unobserved individual-level attributes that are correlated to current health and attempts to minimize the selection bias that stems from (1) individuals with poorer health habits residing in more disadvantaged neighborhoods and (2) individuals in poor health being forced to live in poorer neighborhoods due to economic constraints related to their illness.

The exclusion of health controls may upwardly bias our estimates of how much place can explain racial health disparities, as some of these effects will be attributed to residential context, rather than individual-level characteristics that lead to neighborhood selection. However, current health status may also be a mediating factor on the pathway between current residential context and SRH status, and the inclusion of such variables may result in over-controlling. To the extent that current health status has been influenced by prior neighborhood context and that controlling for overweight status and activity limitations eliminates previous contributions of place, estimates from these models may be viewed as producing an overly conservative estimate of place effects. Hence, the second set of OLS/FE models do not include health controls, allowing for all health status differences to be attributed to place.

To summarize, we specify two regression models: a conventional OLS model that is likely biased due to the omission of VSA-level factors, and a neighborhood FE

model that purges the race estimates of any correlation between individual-level and VSA-level variables. We can infer the magnitude of the neighborhood effects on health disparities by calculating the proportion of the disparity explained:

$$\left[\frac{\{\gamma_{OLS}-\gamma_{FE}\}}{\gamma_{OLS}}\right]\times 100 \quad (3)$$

where γ_{OLS} and γ_{FE} are the race residuals from the OLS and FE models, respectively. This simple calculation allows us to place a quantitative value on the proportion of the racial disparity in SRH that can be accounted for by the inclusion of residential context. If the race coefficient from the FE models is the same as the estimate from the OLS models, nothing additional has been explained by including neighborhood context and the proportion explained by doing so is 0%. If the race coefficient from the FE models is zero, all the racial health disparity has been explained with the addition of neighborhood context and the resulting proportion explained is 100%.

As a sensitivity check, we also estimated a series of models with SRH as a binary outcome (poor/fair vs. good, very good, excellent health). As in the continuous models, the FE models significantly reduced the racial health disparity. For simplicity, we only present results from the linear models as the logit scale is unintuitive and comparisons of odds ratios across models are not straightforward.

Results

We present results for male respondents in Table 2 and female respondents in Table 3. In each of these tables, we first present the results of the OLS model and FE models with health controls (models 1 and 2). Next, we present results from the OLS and FE models that excluded health controls (models 3 and 4).

Because all models interact race with age, and age has been centered by the sample mean age, the race coefficients directly estimated from the models reflect the racial health disparities at age 44. As the magnitude of the racial health disparity is allowed to vary by age, we compute the black/white SRH disparity for discrete age categories within the10th and 90th percentiles of the sample age distribution (five year intervals between ages 25 and 70) and report the proportion of the disparities explained by the VSA FE for each gender (Tables 4 and 5).

Results from Tables 4 and 5 show a consistent health disadvantage for blacks across all models with black females suffering a larger health disadvantage compared to black males. Moreover, for the models without health adjustments, the disparities for both genders increase with age, with the gap for black females widening at a faster rate. Though the gaps for both males and females are reduced with the neighborhood FE specifications, significant health gaps remain.

Age adjusted black/white health disparity tables by gender reveal several overarching patterns. First, the FE models

Table 2
Male sample linear regression model results

Variable	OLS model, with health controls (Model 1)	FE model, with health controls (Model 2)	OLS model, with no health controls (Model 3)	FE model, with no health controls (Model 4)
Race/ethnicity [ref = white]				
Black	−0.15**	−0.08**	−0.13**	−0.06**
Age				
Age[a]	−0.01**	−0.01**	−0.02**	−0.02**
Age squared[b]	0.12**	0.11**	0.31**	0.29**
Race/ethnicity × age interaction				
Black × age[b]	−0.82*	−0.50	−2.66**	−2.37**
Health				
Weight [ref = normal weight]				
Underweight	−0.31**	−0.30**		
Overweight	−0.02**	−0.02**		
Obese	−0.24**	−0.23**		
Activity limitation [ref = no activity limitation]				
Limited in non-major activities	−0.66**	−0.67**		
Limited in major activities	−0.87**	−0.87**		
Unable to perform major activities	−1.48**	−1.45**		
Sample size for the male models	172,753	172,753	172,753	172,753
Adjusted R^2	0.3522	0.3667	0.2342	0.2538

All models adjust for marital status, family income level, education level, labor force status, and nativity. All models also adjust for "other race" group (Asian, other non-Hispanic, Mexican, Puerto Rican, other Hispanic) as well as age interactions for each racial group.
*Statistically significant at the 5% level; **statistically significant at the 1% level.
[a] All age variables were centered at the overall sample mean age 44.
[b] Coefficients have been multiplied by 1000.

Table 3
Female sample linear regression model results

Variable	OLS model, with health controls (Model 1)	FE model, with health controls (Model 2)	OLS model, with no health controls (Model 3)	FE model, with no health controls (Model 4)
Race/ethnicity [ref = white]				
Black	−0.22**	−0.17**	−0.26**	−0.20**
Age				
Age[a]	−0.01**	−0.01**	−0.02**	−0.02**
Age squared[b]	0.09**	0.08**	0.17**	0.16**
Race/ethnicity × age interaction				
Black × age[b]	−0.13	−0.36	−3.76**	−4.01**
Health				
Weight [ref = normal weight]				
Underweight	−0.13**	−0.12**		
Overweight	−0.12**	−0.11**		
Obese	−0.30**	−0.28**		
Activity limitation [ref = no activity limitation]				
Limited in non-major activities	−0.70**	−0.70**		
Limited in major activities	−0.99**	−0.98**		
Unable to perform major activities	−1.44**	−1.42**		
Sample size for the female models	196,152	196,152	196,152	196,152
Adjusted R^2	0.3307	0.3455	0.1968	0.2177

All models adjust for marital status, family income level, education level, labor force status, and nativity. All models also adjust for "other race" group (Asian, other non-Hispanic, Mexican, Puerto Rican, other Hispanic) as well as age interactions for each racial group.
*Statistically significant at the 5% level; **statistically significant at the 1% level.
[a] All age variables were centered at the overall sample mean age 44.
[b] Coefficients have been multiplied by 1000.

consistently explain more of the racial health gap than the OLS models, suggesting that place is a significant contributor to the black health disadvantage and that estimates of health disparities are biased when residential context is excluded from regression models. Second, the inclusion of residential context explains more of the black/white gap in SRH at younger ages than at older ages (though the gradient across age is minimal—and statistically non-significant—when overweight status and activity limitations are included). Third, the relative magnitudes of the contribution of place are in the expected direction; that is, residential context appears to explain more of the black/white disparity in the models that do not control for health conditions—at least during the younger years. Interestingly, there is a cross-over point where this pattern reverses at middle-age due to the larger differences in how quickly the health gap widens by age between FE and OLS models without health adjustments. Results from the male-specific models indicate that, at younger ages, place explains a substantial amount of the remaining racial health disparity that was unaccounted for by individual SES. Place explains, for example, over 75% of the black/white health gap at age 25 for males (Table 4); moreover, the racial gap is no longer statistically significant in the

Table 4
Pre-/post-fixed-effect differences at various ages for linear regression models of male sample

Age	Male sample					
	With health controls			Without health controls		
	Black/white differences: no fixed effects	Black/white differences: fixed effects	% Black/white differences explained[a]	Black/white differences: no fixed effects	Black/white differences: fixed effects	% Black/white differences explained[a]
25	−0.132	−0.070	46.87	−0.080	−0.020	75.53
30	−0.135	−0.072	46.68	−0.093	−0.031	66.60
35	−0.139	−0.075	46.50	−0.106	−0.043	59.84
40	−0.143	−0.077	46.33	−0.119	−0.054	54.56
45	−0.147	−0.079	46.18	−0.133	−0.066	50.34
50	−0.151	−0.082	46.06	−0.146	−0.077	46.87
55	−0.156	−0.084	45.94	−0.159	−0.089	43.99
60	−0.160	−0.087	45.83	−0.172	−0.101	41.54
65	−0.164	−0.089	45.72	−0.185	−0.112	39.43
70	−0.168	−0.091	45.61	−0.199	−0.124	37.61

[a] All differences in the black coefficient across the OLS and FE models are statistically significant at the 1% level within the age range presented.

Table 5
Pre-/post-fixed-effect differences at various ages for linear regression models of female sample

Age	Female sample					
	With health controls			Without health controls		
	Black/white differences: no fixed effects	Black/white differences: fixed effects	% Black/white differences explained[a]	Black/white differences: no fixed effects	Black/white differences: fixed effects	% Black/white differences explained[a]
25	−0.216	−0.160	25.94	−0.186	−0.121	34.87
30	−0.217	−0.162	25.32	−0.204	−0.141	31.08
35	−0.218	−0.164	24.69	−0.223	−0.161	27.92
40	−0.218	−0.166	24.08	−0.242	−0.181	25.25
45	−0.219	−0.168	23.48	−0.260	−0.201	22.97
50	−0.220	−0.170	22.89	−0.279	−0.220	21.00
55	−0.221	−0.171	22.30	−0.298	−0.240	19.29
60	−0.221	−0.173	21.73	−0.317	−0.260	17.78
65	−0.222	−0.175	21.15	−0.336	−0.280	16.44
70	−0.223	−0.177	20.58	−0.354	−0.300	15.24

[a] All differences in the black coefficient across the OLS and FE models are statistically significant at the 1% level within the age range presented.

FE models for males under age 35. For women, residential context explains a significant, though much smaller portion of the racial disparity (Table 5) than it does among men; at age 25, place explains an additional 34% of the racial gap, net of individual SES. The proportion of the black/white health gap that is explained by residential context declines less with age for females compared to males.

Discussion

Our analyses lead to two important conclusions. One, the exclusion of neighborhood context leads to upwardly biased estimates of racial health disparities that were believed to be independent of socio-economic conditions. As a consequence—our second conclusion is that accounting for place provides further explanation for a moderately large portion of observed racial health disparities. The contribution of neighborhood context to the observed racial health gap varies considerably by age and gender, but is substantively and statistically significant across a host of models that control for both overweight status and activity limitations and models that do not. In short, place explains a significant proportion of racial disparities in health that were previously unaccounted for by individual-level SES.

The observation that place explains a larger proportion of the racial health gap explained in younger age groups is consistent with results from studies that found larger neighborhood associations for younger adults and non-significant associations for older adults (e.g., Chaix, Rosvall & Merlo, 2007; Kling, Liebman, Lawrence, & Sanbonmatsu, 2004). A possible explanation for the pattern is that the health of young adults is relatively robust and less likely to be influenced by individual-level SES. Consequently, variation in external factors such as environmental exposures (e.g., exposure to toxins), built environment (e.g., availability of safe recreational facilities), and social conditions (e.g., exposure to neighborhood violence and drugs) may play a greater role in contributing to the health differences between blacks and whites at younger ages. At older ages, health level and health care may depend more on personal socio-economic resources that have already been accounted for in the models. Another possible reason is that the current neighborhood conditions fail to capture the life-long residential context that resulted in the cumulative health disadvantage experienced by blacks (as supported by the increasing health gap by age) and thus are less likely to explain health difference at older ages. That is, because current residential context is likely to be more representative of long-term neighborhood conditions for younger age groups due to their limited total exposure to neighborhood characteristics and to be less reliable as an indicator of average exposure for older adults, we would expect the underestimation of place effects to be more severe at older ages than at younger ages. Finally, just as socio-economic disparities decrease over the life course due to selective mortality, we might expect the same to happen with respect to the effect of current residential context.

Our finding that residential context explains less of the racial health disparities among women than among men is related to other studies that have found differential neighborhood associations in health across gender. Molinari, Ahern, and Hendryx (1998) found that women's health is more strongly associated with community problems (e.g., crime, poverty, domestic violence), while environmental problems (e.g., quality of outdoor air, drinking water, trash disposal) seem to be associated with men more than women. Other studies have found stronger neighborhood connections with SRH for women than for men (Kavanagh, Bentley, Turrell, Broom, & Subramanian, 2006; Stafford et al., 2005) and differential neighborhood associations with weight outcomes across gender (Chang & Christakis, 2005; Robert & Reither, 2004; Wang et al., 2007). In fact, much of the literature on neighborhood context indicates that women are more influenced by some aspects of neighborhood context than are men and that the patterns vary considerably across health outcomes (Bird & Rieker, 2008). Here however, we are focused not on whether men or women are more affected by neighborhood context, but specifically on whether place explains more of the racial gap in SRH for women than for men.

The finding that more of the racial gap in health is explained by context for men than for women is consistent with greater geographic variation in black men's opportunities for a healthy life relative to those of white men than among black women relative to white women.

For example, the risk of unemployment and exposure to violence likely are higher for black men than for white men and for women of either race (Bird & Rieker, 2008). As such, the patterning of these types of risk across neighborhoods and the contribution of place may be much stronger for black men. Consequently, our results suggest that understanding and addressing place effects may be particularly important to improving the health of black males.

Overall, our results suggest that neighborhood context per se explains a moderate to substantial portion of the black/white health gap, net of individual characteristics. To summarize, residential context might account for as much as 76% of the residual black/white disparities in health among 25-year-old males, but in contrast, only accounts for about 15% of the residual black/white disparities among older women. Although the range of the findings is rather wide, it nonetheless indicates that place may be associated with a non-inconsequential portion of the racial health gap. It is unclear whether our results would generalize to other countries. Nonetheless, this study takes an important, albeit limited, step toward quantifying this potential contribution as few attempts have been made to quantify the actual role that residential context plays in producing racial health disparities.

Limitations to our analyses include the assumption that VSA-level effects are equal across residents within the same VSA, an unlikely reality given that stressful environments might have larger effects on the unemployed, for example. Another is the pooling of data across a number of years to ensure large enough numbers of respondents in each VSA to produce stable estimates. As a result, respondents in the same VSA may not have actually lived there concurrently, and the characteristics of the VSA may have changed dramatically over time—a change that we are unable to measure. This is unlikely, however, as analyses of census data indicate that neighborhoods do not usually change dramatically within a small time period.

Importantly, several factors may bias our results in either direction. These findings may have overestimated the contribution of neighborhoods to racial health disparities if omitted variables at the individual-level are correlated with both race and VSA characteristics and/or *included* individual characteristics were poorly measured or misspecified. VSA indicators would absorb all the unmeasured or poorly measured compositional differences across VSAs, erroneously attributing them to contextual effects. Income, for example, is often believed to be poorly measured. Consequently, the VSA fixed effects may be absorbing some of the measurement noise and model misspecification in personal income. Though these factors are not problematic with respect to further explaining the black/white health gap and reducing the race residual per se, it is of sizable consequence to what inferences can be made of the sources that led to the increased explanation of the black health disadvantage. However, this problem is not unique to our FE strategy and the difficulty of disentangling composition vs. context is a common problem in neighborhood-effect studies.

Still, there are reasons to suspect that these estimates might be biased downwards (i.e., they underreport neighborhood contribution to health disparities). First, place effects on health most likely manifest themselves as early as birth (O'Campo, Xue, Wang, & Caughy, 1997), accumulating and persisting through adolescence (Brooks-Gunn, Duncan, & Aber, 1997) and into old age (Yen & Kaplan, 1999b). Models relying on a single point-in-time estimate of current residential context cannot distinguish between individuals with long-time exposures to disadvantaged neighborhoods from individuals with only a limited exposure.

Second, the effects of residential context might be felt much more broadly than at the level in which VSAs are measured. That is, while this small area might capture much larger variation in the social and built environment in neighborhoods than census tracts do, both extra-residential variation and spill-over from neighboring places might have both direct and indirect effects on health that are not captured by such a circumscribed measurement of social and physical space. In fact, recent evidence suggests that failure to control for extra-residential characteristics (in terms of the characteristics of the social space that individuals occupy outside of their homes, such as places of work, worship, shopping, and play) might actually suppress the true effects of residential context on health (Inagami, Cohen, & Finch, 2005).

Third, not all place effects on health work directly through health-related mechanisms. Place might indirectly affect health by affecting an individual's access to quality education, jobs, and higher levels of income (Wilson, 1996)—all factors that have been associated with innumerable health outcomes (Link & Phelan, 1995). As such, controlling for these variables represents at least a partial over-control for potential place effects and may affect how much of the variation in health disparities is actually attributable to residential context. For these reasons, we believe our estimates to be fairly conservative, or downwardly biased estimates of the role of residential context in contributing to racial health disparities.

In conclusion, although this paper has made a substantial attempt at quantifying the role of residential context in generating racial disparities in health, much research remains to be done. First, attempts to quantify the contribution of place should continue, particularly in view of the limitations inherent in the use of the NHIS data set. Second, explanations for why place effects on health might diminish over the life course, or at least their ability to explain disparities, need to be explored. Third, the notion that residential context explains less of the racial disparity among women than among men is worthy of both empirical and theoretical focus. Finally, and most importantly, the precise factors that contribute to racial disparities in health need to be continually generated by careful empirical research, much needed theory, and attention to the methodological shortcomings of so-called "neighborhood-effect" studies.

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