

Racial residential segregation and weight status among US adults

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Available online 16 May 2006

Abstract

While the segmentation of residential areas by race is well known to affect the social and economic well-being of the segregated minority group in the United States, the relationship between segregation and health has received less attention. This study examines the association between racial residential segregation, as measured by the isolation index, and individual weight status in US metropolitan areas. Multi-level, nationally representative data are used to consider the central hypothesis that segregation is positively associated with weight status among African Americans, a group that is hyper-segregated and disproportionately affected by unhealthy weight outcomes. Results show that among non-Hispanic blacks, higher racial isolation is positively associated with both a higher body mass index (BMI) and greater odds of being overweight, adjusting for multiple covariates, including measures of individual socioeconomic status. An increase of one standard deviation in the isolation index is associated with a 0.423 unit increase in BMI ($p < 0.01$), and a 14% increase in the odds of being overweight ($p < 0.01$). Among whites, there is no significant association between the isolation index and weight status. These findings suggest that in addition to differences among people, differences among places and, in particular, differences in the spatial organization of persons may be relevant to health policy and promotion efforts.

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Keywords: Racial segregation; Obesity; Body mass index; Socioeconomic factors; Contextual analysis; Health inequalities; USA

Introduction

In the US, residential segregation by race is well known to affect the social and economic well-being of the segregated minority group, particularly that of African Americans. Given relatively higher rates of black poverty, segregation acts to concentrate

poverty in space, creating a distinctive milieu in segregated black neighborhoods, one that is characterized by factors such as housing deterioration, lower quality schools, inferior public services, and higher rates of crime, unemployment, families on public assistance, and single parenthood (Massey & Denton, 1993). These and other features associated with segregation have been linked to lower educational and occupational attainments, diminished prospects for socioeconomic mobility, and higher risks for individual social dislocations (e.g., teen pregnancy, welfare dependency, unemployment,

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criminal behavior, etc.) (Massey, Gross, & Eggers, 1991; Massey & Denton, 1993; Wilson, 1987; Wilson, 1997).

The relationship between racial segregation and health, however, has received less attention, and most empirical work has focused on mortality. Half a century ago, Yankauer (1950) found that black and white infant mortality rates are higher in the more segregated residential areas of New York City. Since then, other ecological studies have demonstrated that black infant mortality is positively associated with segregation at the metropolitan area level (LaVeist, 1989, 1993; Polednak, 1996b) and at the state level (Bird, 1995). Adult mortality has also been examined, with ecological studies showing that metropolitan area segregation is positively associated with black mortality rates (Collins & Williams, 1999; Hart, Kunitz, Sell, & Mukamel, 1998), as well as with the differential between black and white mortality rates (Polednak, 1993, 1996a). More recently, a multi-level study found that metropolitan area segregation is associated with increased odds of poor self-rated health among blacks (Subramanian, Acevedo-Garcia, & Osypuk, 2005). Additionally, a few studies have found that residence in a census tract with a higher proportion of blacks is associated with a higher risk of mortality (Leclere, Rogers, & Peters, 1997, 1998; Jackson, Anderson, Johnson, & Sorlie, 2000).

Though not addressing segregation itself, there are now numerous studies demonstrating that neighborhood-level poverty is inversely associated with health (Pickett & Pearl, 2000). The segmentation of residential areas by race, however, is a key mechanism by which such neighborhood conditions are formed. While Black men in Harlem have a lower life expectancy than their counterparts in Bangladesh (McCord & Freeman, 1990), it is segregation that ultimately gives rise to the concentration of poverty in Harlem.

Segregation and weight status

This study examines the relationship between racial segregation and weight status in US metropolitan areas. In the context of prior work supporting an association between segregation and mortality as well as self-rated health, weight status may function as an intermediary link to such general endpoints. Obesity, which has reached a prevalence of 30% among US adults (Hedley et al., 2004), is well known to be associated with various

morbidities (Must et al., 1999), and may also increase the risk of mortality (Manson et al., 1995). There are many pathways by which segregation and the concentration of poverty could affect weight outcomes. For example, a poor consumer income base can negatively influence the quantity and quality of food retail options; a lower tax base can decrease the provision of parks and public recreational facilities; high crime rates can be prohibitive of outdoor activity; and impoverished, disenfranchised neighborhoods may be severely limited in the provision of medical services (Williams & Collins, 2001). Recent studies find that black neighborhoods may have considerably lower access to supermarkets and offer fewer healthy food selections relative to white neighborhoods (Lewis et al., 2005; Morland, Wing, Diez Roux, & Poole, 2001; Zenk et al., 2005).

Aside from the provision of such material services and amenities, the geographic isolation of segregation can translate into *social isolation*, creating a distinctive sociocultural milieu that may have more direct effects on individual well-being. Not only does racial segregation isolate poor blacks, it spatially concentrates the correlates of individual poverty. Social dislocations such as unemployment, teenage childbearing, and welfare dependency can become the norm rather than the exception, creating a dearth of normatively successful role models and perpetuating attitudes and behaviors that are non-conducive to the fulfillment of conventional expectations. Given that weight status is also correlated with poverty, the status of being overweight may also be fast approaching the norm in segregated enclaves, shifting local weight standards and mitigating the broader social stigma of obesity.

In a related vein, many have argued that alternative status and value schemes may develop to compensate for the disjuncture between dominant socioeconomic goals and structured opportunities. These schemes often reflect a deliberate inversion or disavowal of mainstream counterparts, and can be described as an oppositional subculture (Anderson, 1990; Massey, 1996; Shihadeh & Flynn, 1996; Wilson, 1997). As speech patterns, educational aspirations, and values concerning marriage and childbearing have grown increasingly distant from the mainstream in many poor, predominantly black neighborhoods (Massey & Denton, 1993), we might also expect divergent value schemes in the realm of health promotion efforts and weight preferences. Indeed, some have noted the rise of

“health-related subcultures” in such settings (Fitzpatrick & Lagory, 2000). Furthermore, many studies have shown that blacks tend to be more tolerant than whites of heavier weight statuses, and differences in weight preferences or standards of ideal weight may contribute to racial differences in weight outcomes (Chang & Christakis, 2003; Kumanyika, 1998). Although a host of historical and sociocultural factors other than segregation may contribute to racial differences in attitudes with respect to weight and bodily aesthetics, racial isolation would still act to concentrate such standards, shifting the overarching community norm away from mainstream counterparts. Lastly, social isolation may also impede the diffusion of health-related information, and the multifarious nature of stress associated with living with concentrated poverty may precipitate both physiological and coping-type behavioral reactions that contribute to weight gain.^{1,2}

This study uses nationally representative data to examine the relationship between US metropolitan area segregation and individual weight outcomes. While some prior studies on segregation and health do utilize individual-level data (e.g., Leclere et al., 1997, 1998; LaVeist, 2003; Robert & Reither, 2004; Subramanian et al., 2005), most employ ecological analyses, making the contribution of individual-level covariates difficult to ascertain and potentially overstating the role of contextual-level factors. For example, an association between segregation and health may be confounded by the fact that individual SES is correlated with both place of residence and health, reflecting a compositional effect of individual residents rather than an independent, contextual effect of segregation per se. It should be noted, however, that racial segregation likely affects health, in part, *through* a negative effect on individual

socioeconomic attainments, which can ultimately influence health (Goldman, 2001). In this sense, factors such as individual SES would be on a causal pathway between segregation and health, and their adjustment may lead to an underestimation of the total effect of segregation. Hence, this study will use multi-level data to examine of the influence of segregation on weight status both with and without adjustments for individual-level SES.

The principal hypothesis of this study is that a greater degree of metropolitan area segregation will be associated with higher (heavier) weight status among blacks. For whites, a definitive expectation is more difficult to formulate. On the one hand, segregation (measured at the metropolitan level) may be beneficial to whites, especially poor whites, by isolating or buffering them from the correlates of black poverty (Massey, 1990). On the other hand, empirical results for health have been mixed. For example, while some prior studies do find an inverse association between segregation and mortality among whites (LaVeist, 1989, 1993), others show that segregation is either unrelated or positively associated with mortality among whites (Bird, 1995; Collins & Williams, 1999; Jackson et al., 2000; Polednak, 1996a). Lastly, little is known about the relationship between segregation and more specific health outcomes such as weight status.

The isolation dimension

Massey and Denton (1988) conceptualize segregation along five distinct dimensions: unevenness, isolation, centralization, concentration, and clustering. This study will focus on the isolation dimension, which speaks most directly to proposed theoretical links between black social isolation and weight status. Moreover, an analysis of the isolation dimension is in keeping with several prior studies on segregation and health (Collins & Williams, 1999; Fang, Madhavan, Bosworth, & Alderman, 1998; Guest, Gunnar, & Hussey, 1998; Jackson et al., 2000; Leclere et al., 1997; Subramanian et al., 2005). Massey and Denton (1988) recommend the isolation index as the preferred measure of racial isolation. This concept refers to the degree to which minority group members come into contact primarily with other minority group members, indexing the percentage of neighborhood co-residents who are also minority group members.

Some studies rely on the dissimilarity index, a measure of unevenness in the distribution of blacks

¹The human neuroendocrine system responds to stress by releasing the steroid hormone cortisol, which is well known to be associated with weight gain and fat accumulation.

²Racial or ethnic isolation could relate to health benefits for some groups. Among US Hispanics, limited acculturation may bolster cultural protective factors and within-group support networks, factors that are frequently invoked as explanations for better health outcomes compared to whites, an advantage that is “paradoxical” in the context of Hispanic socioeconomic disadvantage (Leclere et al., 1997; Palloni & Arias, 2004). Nevertheless, such a paradox has not been observed among non-Hispanic blacks, and previous segregation studies on health have generally shown a detrimental effect of black isolation. Fang et al. (1998) found that blacks living in white areas of New York City suffer a mortality disadvantage relative to those living in black areas, but this was true only among the elderly.

and whites. It has been noted, however, that the dissimilarity index is typically chosen arbitrarily or without justification, serving as a default measure with inadequate attention to relevant linkages between specific conceptualizations of segregation and the outcome of interest (Acevedo-Garcia & Lochner, 2003; Collins & Williams, 1999; Shihadeh & Flynn, 1996; Subramanian et al., 2005). Here, the isolation index is used to best capture potential pathways involving the effects of racial social isolation on weight status. Furthermore, in contrast to the dissimilarity index, the isolation index explicitly accounts for the relative sizes of the groups being compared (Massey & Denton, 1988), a property that is critical to this study. As a hypothetical illustration, consider a city where blacks are evenly (or perfectly) distributed, with each neighborhood containing an equal percentage of black residents. In this case, the dissimilarity index would be zero, registering no segregation. The isolation index, however, could still be high if blacks constitute a very high proportion of the total residents in the city, resulting in a relatively high percentage of blacks and little contact with whites in individual neighborhoods, despite even distribution. Lastly, in this regard, the isolation index is also effective in capturing suggested pathways involving the concentration of poverty per se (e.g., insufficient services and amenities). This is because segregation leads to concentrated poverty via an interaction between high minority poverty rates and a high proportion of minority group members in individual neighborhoods, and the isolation index speaks most directly to neighborhood minority proportion.

Methods

Data and measures

Individual-level data are from the 2000 *Behavioral Risk Factor Surveillance System* (BRFSS), a cross-sectional, nationally representative survey administered by the Centers for Disease Control and Prevention. The BRFSS collects data on a wide variety of health-related behaviors and outcomes in the US adult population and utilizes a probability sample of non-institutionalized adults for each state through random-digit-dial telephone surveys. The survey includes data on self-reported height and weight and standard sociodemographic information. Weight status, the outcome of interest, is assessed with BMI (height in meters divided by the

square of weight in kilograms) as well as the status of being overweight (BMI ≥ 25) and obese (BMI ≥ 30) as defined by conventional clinical standards. Individual-level covariates include age, sex, marital status, education, household income, healthcare coverage (insurance), and a measure of physical activity and fruit/vegetable intake.

Age is modeled as a continuous variable and includes a squared term, as preliminary analyses indicate significant non-linear effects on BMI. Remaining covariates are modeled as categorical variables with dichotomous indicators. Household income includes a missing data category for those who refused or could not answer the survey item. Healthcare coverage includes government plans such as Medicare and Medicaid. Since many of the pathways between segregation and weight status imply differences in physical activity and nutritional intake, the potential mediating role of such behaviors is assessed. Using the measures of activity and intake available in the BRFSS, a four-category variable is constructed: (1) physically active *and* consumes five or more servings of fruits/vegetables per day, (2) active only, (3) five servings only, and (4) neither. These contrasts are created to allow for synergistic effects between activity and intake. Physically active is defined as physical activity for 20 or more minutes, three or more times per week at 50% of functional capacity. The consumption of at least five servings of fruits/vegetables per day roughly corresponds to federal dietary recommendations (US Department of Agriculture, 2000).

All metropolitan statistical area (MSA) measures are based on data from the 2000 US Census. Here, metropolitan statistical areas and primary metropolitan statistical areas (PMSAs) are collectively referred to as metropolitan areas or MSAs. MSAs are defined to identify a large population nucleus and surrounding communities with which the nucleus has a high degree of economic and social integration. Segregation between blacks and whites is computed using census tracts as a proxy for neighborhoods. The isolation index varies between 0 and 1 (with 1 denoting maximal isolation) and is computed as the average fraction of black residents in each census tract, weighted by the proportion of blacks in each tract. It is mathematically represented by the following formula:

$$\text{Isolation index} = \sum_{i=1}^n \left[\frac{x_i}{X} \right] \left[\frac{x_i}{t_i} \right].$$

Given n census tracts in a metropolitan area, x_i is number of blacks in tract i ; X is the total population of blacks in the metropolitan area; and t_i is the total population in tract i . The index attempts to measure “the experience of segregation as felt by the average minority” person rather than a “departure from some abstract ideal of ‘evenness’” (Massey & Denton, 1988:287). For example, an index of 0.40 means that, on average, a black person lives in a neighborhood where 40% of his or her co-residents are also black. Metropolitan-level covariates, which include median household income, proportion of families in poverty, and population size, are modeled continuous variables, and population is logged due to a distribution skewed to the right.

Analyses

Given well known racial differences in the nature of body image and weight-related preferences, and given the expectation that metropolitan-level segregation differentially influences the health of minority versus majority group members, analyses are stratified by race.³ This is in keeping with prior studies on mortality. The sample is limited to non-Hispanic black and white adults (aged 18 and over) identified to 130 metropolitan areas where blacks constitute at least 10% of the total population. Of the 46,881 BRFSS respondents in this category, 567 women are excluded because they are pregnant and 1839 persons are excluded because they are missing data on weight status. Additionally, 78 persons are excluded because they are missing data on education, followed by 100 missing data on marital status and 87 missing data on healthcare coverage. This leaves an overall sample size of 44,210, consisting of 8800 blacks and 35,410 whites, with an average of 340 persons per MSA.

Two-level hierarchical linear regression is used to assess the effect of metropolitan area segregation on individual BMI, adjusting for covariates at both levels. With individuals at level one nested within metropolitan areas at level two, hierarchical modeling can take into account the influence of different sample sizes across MSAs as well as the non-independence of persons clustered within the same MSA. Using BMI as the level-one dependent variable, level-one predictors are centered on their grand means, and the variance components of their

slopes are fixed at level two. This allows for assessment and modeling of the variation in BMI between metropolitan areas (adjusted for differences between areas in level-one variables), which is reflected in a random effect for the level-one intercept (Raudenbush & Bryk, 2002). Metropolitan-level variables are modeled as predictors for the level-one intercept. Census regions are included as fixed effects to address potential confounding of the association between metropolitan area segregation and weight status by unmeasured regional factors; both segregation and weight tend to show strong variation by region. Hence, the focus is on modeling a fixed effect of region, or absorbing between-MSA variation in weight status due to region, rather than modeling a random effect of region, clustering within regions, or variance between regions. Overweight (BMI ≥ 25) and obese (BMI ≥ 30) are also modeled as dependent variables using hierarchical generalized linear models. Analyses are conducted using STATA 8.2 and HLM 6.02 software; all results account for BRFSS sampling weights, which adjust for unequal selection probabilities and response rates.

Results

Sample characteristics

Table 1 displays descriptive statistics for the BRFSS sample and the metropolitan areas. Whites have a lower mean BMI, percentage overweight, and percentage obese compared to blacks, but both groups have a mean BMI that is in the overweight range, and the majority of both groups is overweight. Both groups show relatively high proportions residing in the South because the sample is restricted to persons residing in metropolitan areas where blacks constitute at least 10% of the population. For the 130 metropolitan areas included in the analyses, the mean value for the isolation index is 0.53, with a range of 0.25–0.83. Examples along this range are given by the following cities: Charlottesville, VA (0.27), Orlando, FL (0.46), Oakland, CA (0.56), Los Angeles, CA (0.65), Chicago, IL (0.78), and Detroit, MI (0.81). Compared to US MSAs as a whole, the sample MSAs have higher means on segregation, population, and percentage of families below poverty, reflecting the fact that areas with a low black population have been excluded from the sample.

³Stratification by individual race also removes potential confounding from the compositional effects of individual race.

Table 1
BRFSS and MSA Sample Characteristics

BRFSS	Mean (SD) or Frequency %		
	Blacks (<i>N</i> = 8800)	Whites (<i>N</i> = 35,410)	
BMI	28.1 (6.1)	26.1 (5.1)	
Overweight (BMI ≥ 25)	68.1	54.5	
Obese (BMI ≥ 30)	30.1	18.3	
Age (years)	43.2 (16.6)	46.9 (17.5)	
Female	54.6	50.2	
Married/coupled	41.0	63.0	
<i>Education:</i>			
HS or less	14.4	8.1	
HS graduate	36.4	28.3	
Some college or tech.	29.4	27.7	
College graduate	19.8	35.9	
<i>Household Income</i>			
<\$20	24.9	10.5	
20 to <35	27.8	19.0	
35 to <50	15.3	16.7	
50 to <75	10.9	18.3	
75 or more	8.7	23.0	
Don't know/refused	12.3	12.5	
Health care coverage	81.8	91.0	
<i>Activity and fruit/vegetable intake</i>			
Physically active and 5+ servings/day	3.4	5.6	
Physically active	6.2	10.0	
5 servings/day	18.0	17.9	
Neither	68.9	63.0	
Don't know/refused component question	3.7	3.5	
<i>Region</i>			
Northeast	17.2	16.1	
Midwest	19.9	27.4	
West	7.8	6.2	
South	55.1	50.3	
MSA	Sample (<i>N</i> = 130) Mean (SD)	Sample Range	US (<i>N</i> = 331) Mean (SD)
Isolation index	0.53 (0.13)	0.25–0.83	0.32 (0.22)
Population size (100 K)	9.9 (16.0)	0.8–95.2	6.8 (11.4)
Med. household inc. (\$K)	40.1 (6.6)	29.1–62.2	41.2 (7.9)
% Families below poverty	9.8 (2.9)	5.0–17.1	8.8 (3.7)
% Black	21.6 (9.8)	10.0–51.3	11.2 (10.6)

Note: Data reflect sampling weights.

Multivariate analyses

Body mass index

Table 2 displays fixed effect and random effect estimates for non-Hispanic blacks from hierarchical linear regression models with BMI as the dependent variable. Model 1 includes the following individual-level factors: age, sex, marital status, education,

household income, and healthcare coverage. BMI increases in curvilinear fashion with age, is higher among black females, and is significantly higher in the lowest education and income categories compared to the highest. Consistent with prior work, the gradient across income and educational categories is non-monotonic among blacks (Chang & Lauderdale, 2005). Healthcare coverage is also positively

Table 2
Models for body mass index: non-hispanic blacks (N = 8800)

Independent variables	Fixed effects γ (SE)					
	(1)	(2)	(3)	(4)	(5)	(6)
Models						
MSA isolation ^a		0.261* (0.113)	0.433** (0.152)	0.444** (0.153)	0.423** (0.144)	0.402* (0.154)
MSA population		-0.347** (0.106)	-0.378** (0.123)	-0.386** (0.135)	-0.352** (0.127)	-0.350** (0.131)
MSA median income (\$10 K)			-0.201 (0.304)	-0.268 (0.300)	-0.174 (0.286)	-0.094 (0.321)
MSA poverty (families %)			-0.093 (0.061)	-0.104 (0.058)	-0.093 (0.055)	-0.081 (0.061)
<i>Region</i>						
Northeast		0.302 (0.255)	0.377 (0.249)	0.241 (0.251)	0.190 (0.243)	0.119 (0.272)
Midwest		-0.005 (0.279)	-0.222 (0.315)	-0.317 (0.312)	-0.333 (0.290)	-0.280 (0.350)
West		0.464 (0.586)	0.769 (0.580)	0.518 (0.674)	0.506 (0.614)	0.491 (0.662)
South		—	—	—	—	—
Age	0.340** (0.023)			0.326** (0.022)	0.341** (0.023)	0.343** (0.023)
Age ²	-0.003** (<0.001)			-0.003** (<0.001)	-0.003** (<0.001)	-0.003** (<0.001)
Female	0.660** (0.165)			0.680** (0.163)	0.656** (0.163)	0.686** (0.167)
Married/coupled	0.049 (0.175)			-0.145 (0.171)	0.026 (0.178)	0.010 (0.178)
<i>Education</i>						
High school or less	1.472** (0.342)				1.416** (0.355)	1.384** (0.362)
High school graduate	0.746** (0.269)				0.724** (0.271)	0.697* (0.272)
Some college or tech.	0.805** (0.257)				0.795** (0.261)	0.806** (0.267)
College graduate	—				—	—
<i>Household Income</i>						
Under \$20,000	0.865* (0.422)				0.857* (0.437)	0.809 (0.452)

Table 2 (continued)

Independent variables	Fixed effects γ (SE)					
	(1)	(2)	(3)	(4)	(5)	(6)
Models						
\$20,000–\$34,999	0.485 (0.351)				0.474 (0.356)	0.432 (0.364)
\$35,000–\$49,999	0.654 (0.340)				0.626 (0.329)	0.603 (0.329)
\$50,000–\$74,999	0.224 (0.364)				0.218 (0.364)	0.232 (0.369)
Don't know/refused	0.235 (0.386)				0.233 (0.400)	0.149 (0.411)
\$75,000 or more	—				—	—
Health care coverage	0.678** (0.254)				0.689** (0.254)	0.676** (0.253)
<i>Physical activity and fruit/vegetable intake</i>						
Active and 5+ servings/d						–1.356** (0.387)
Active						–0.614 (0.393)
5+ servings/d						–0.120 (0.264)
Don't know/refused						–1.002** (0.348)
Neither						—
Intercept	28.239	31.794	33.216	33.850	32.941	32.553
Int variance component (random effect)	0.189**	0.189**	0.197**	0.164**	0.128**	0.196**

* $p < 0.05$.** $p < 0.01$.^a γ = change in BMI estimated for a one standard deviation increase in black isolation.

Table 3
Models for body mass index: non-hispanic whites (N = 35,410)

Independent variables	Fixed Effects γ (SE)					
	(1)	(2)	(3)	(4)	(5)	(6)
Models						
MSA isolation ^a		0.139 (0.085)	0.211* (0.107)	0.171 (0.102)	0.168 (0.097)	0.164 (0.094)
MSA population		-0.119 (0.067)	-0.106 (0.083)	-0.110 (0.076)	-0.081 (0.074)	-0.082 (0.070)
MSA median income (\$10 K)			-0.246 (0.207)	-0.268 (0.197)	-0.085 (0.194)	-0.100 (0.187)
MSA poverty (families %)			-0.056 (0.049)	-0.051 (0.046)	-0.032 (0.044)	-0.038 (0.042)
<i>Region</i>						
Northeast		-0.358 (0.214)	-0.305 (0.208)	-0.263 (0.120)	-0.228 (0.192)	-0.200 (0.190)
Midwest		0.143 (0.170)	0.079 (0.180)	0.144 (0.174)	0.134 (0.165)	0.125 (0.158)
West		-0.408 (0.511)	-0.175 (0.542)	-0.092 (0.444)	-0.084 (0.432)	0.009 (0.396)
South		—	—	—	—	—
Age	0.327** (0.012)			0.304** (0.012)	0.327** (0.012)	0.327** (0.012)
Age ²	-0.003** (<0.001)			-0.003** (<0.001)	-0.003** (<0.001)	-0.003** (<0.001)
Female	-1.679** (0.068)			-1.581** (0.072)	-1.677** (0.068)	-1.635** (0.072)
Married/coupled	0.205** (0.065)			-0.076 (0.066)	0.202** (0.065)	0.210** (0.065)
<i>Education</i>						
High school or less	1.307** (0.171)				1.302** (0.172)	1.144** (0.168)
High school graduate	0.774** (0.095)				0.766** (0.095)	0.665** (0.095)
Some college or tech.	0.560** (0.104)				0.555** (0.103)	0.507** (0.101)
College graduate	—				—	—
<i>Household Income</i>						
Under \$20,000	1.407** (0.168)				1.404** (0.170)	1.288** (0.170)

Table 3 (continued)

Independent variables	Fixed Effects γ (SE)					
	(1)	(2)	(3)	(4)	(5)	(6)
Models						
\$20,000–\$34,999	0.855** (0.096)				0.846** (0.097)	0.767** (0.096)
\$35,000–\$49,999	0.806** (0.116)				0.799** (0.117)	0.742** (0.119)
\$50,000–\$74,999	0.566** (0.119)				0.560** (0.119)	0.515** (0.120)
Don't know/refused	0.095 (0.116)				0.088 (0.115)	0.033 (0.116)
\$75,000 or more	—				—	—
Health care coverage	0.153 (0.135)				0.147 (0.136)	0.167 (0.133)
<i>Physical activity and fruit/vegetable intake</i>						
Active and 5+ servings/d						–1.257** (0.143)
Active						–1.204** (0.095)
5+ servings/d						–0.158 (0.133)
Don't know/refused						
Neither						
Intercept	26.083	27.219	28.299	28.537	27.159	27.304
Int. variance component (random effect)	0.157**	0.212**	0.209**	0.172**	0.138**	0.123**

* $p < 0.05$.** $p < 0.01$.^a γ = change in BMI estimated for a 1 SD increase in isolation.

associated with weight status. The estimated intercept variance component (0.189) indicates significant ($p < 0.01$) variation in BMI between metropolitan areas, even after adjusting for compositional differences from these individual-level characteristics. Using the variance component from a one-way ANOVA model with random effects (0.244) as a basis of comparison, these demographic factors account for about 23% ($1 - 0.189/0.244$) of the baseline variation between MSAs in BMI.

Model 2 includes the MSA isolation index, population, and region, and Model 3 additionally adjusts for MSA median income and poverty levels. The isolation index is scaled so that its regression coefficient represents the change in BMI estimated for an increase of one standard deviation in MSA isolation. For blacks, segregation shows a significant positive association with BMI even after adjustments for measures of aggregate income status in Model 3. In Model 3, a one standard deviation increase in the isolation index is associated with a 0.433 unit increase in BMI. A significant positive association remains after adjustments for age, sex, and marital status in Model 4, and adjustments for education, income, and health-care coverage in Model 5. As noted previously, it can be argued that individual socioeconomic factors are conceptually on the pathway between segregation and weight status. Hence, one may be underestimating the total effect of segregation when statistically adjusting for individual SES. In Model 5, which includes such factors, the coefficient for segregation (0.423) shows only slight attenuation relative to the estimate from Model 4 (0.444), suggesting a significant association with BMI independent of individual SES and a relatively minor contribution through such indirect effects. A comparison of the estimated variance component from Model 5 (0.128) to that from Model 1 (0.189) shows that the inclusion of the MSA-level variables and region explains about 32% ($1 - 0.128/0.189$) of the remaining variance between metropolitan areas after adjustment for individual sociodemographics.

As units of BMI are somewhat abstract, one can consider the association between BMI and segregation relative to the association between BMI and well known individual-level correlates such as education and income. In Model 5, BMI is estimated to increase by 1.416 units when comparing the lowest (less than HS graduate) to highest (college graduate) levels of education, and BMI is estimated to increase by 0.857 units comparing the lowest (under \$20,000) to highest

(\$75,000 and over) levels of income. From the same model, BMI is estimated to increase by 1.845 units comparing the lowest (0.25) to highest level of segregation (0.83) in the sample, by 1.209 units comparing the 10th to the 90th percentile of segregation, and by 0.668 units comparing the 25th to the 75th percentile of segregation.

Models were checked for sensitivity to the use of MSA % black. When using MSA % black rather than MSA isolation in Model 5, a one standard deviation increase in MSA % black is associated with a 0.333 (SE = 0.131; $p = 0.012$) unit increase in BMI. Like MSA isolation, MSA % black is also positively associated with BMI. When both measures are included in the model, the coefficient for MSA black isolation is 0.300 (SE = 0.192; $p = 0.120$); the coefficient for MSA % black is 0.183 (SE = 0.162; $p = 0.261$); and a test of their joint contribution is significant at $p < 0.01$ ($\chi^2 = 10.8$; $df = 2$). In sum, each measure shows a significant and positive association with BMI when modeled alone. Together, the coefficients are attenuated and not individually significant, but they remain positive and are jointly significant. These results are consistent with collinearity between the two measures and considerable overlap in what they measure. MSA % black is the crude percentage of blacks across the MSA as a whole, while the MSA isolation index is the average percentage of blacks across census tracts, weighted by the proportion of blacks that reside in each tract.

Lastly, Model 6 adjusts for physical activity and fruit/vegetable intake. Compared to those who are neither physically active nor consume at least five or more servings of fruits/vegetables per day, all other groups are estimated to be lower in average BMI. This difference is statistically significant, however, only for those who meet both requirements. With respect to segregation, the adjustment for these measures of activity and intake results in a relatively minor attenuation of its estimated association with weight status.

Table 3 displays the same sequence of models in non-Hispanic whites. Here, Model 1 estimates lower BMI among females and a monotonically inverse relationship with the education and income categories.⁴ Again, there is significant variation (0.157;

⁴The inclusion of interactions between sex and the terms for income and education precipitated little change with respect to estimates and conclusions concerning segregation, so models excluding them, which are more parsimonious, are presented. No significant interactions were found between sex and the isolation index.

$p < 0.01$) in BMI between metropolitan areas even after adjustment for the individual-level variables. Given an estimated variance component of 0.250 from a one-way ANOVA model with random effects, these demographic factors explain about 37% ($1 - 0.157/0.250$) of the BMI variation between metropolitan areas. In general, the estimated association between segregation and BMI is also positive in whites, but this estimate is statistically significant ($p < 0.05$) only in Model 3, prior to the inclusion of individual-level covariates. The estimate is attenuated and rendered non-significant in Model 4 (which adjusts for individual age, sex, and marital status) and remains non-significant in subsequent models. Comparing the variance components between Models 1 and 5, the inclusion of the MSA-level variables and region explains about 12% ($1 - 0.138/0.157$) of the remaining variance after adjustment for individual sociodemographics. Lastly, results in both whites and blacks are not altered in any meaningful fashion with further adjustments for either current smoking status or self-rated health status.

Overweight and obesity

Table 4 shows fixed effect estimates for the isolation index from hierarchical logistic regression models for the odds of being overweight (BMI ≥ 25) and obese (BMI ≥ 30). In contrast to the models for BMI, these models assess whether or not persons are actually in a weight category defined, according to clinical and public health standards, as problematic for health. These models adjust for all the covariates used in Model 5 of the regressions for BMI. Estimates for both weight outcomes are positive in blacks, but the association is significant only for the odds of being overweight. Here, blacks are estimated to have 1.139 times higher odds of being overweight with a one standard deviation

increase in the isolation index, independent of individual socioeconomic status. Thus, compared to a person living at the lowest level of segregation in the sample—with isolation index of 0.25—the odds of being overweight increase by 16% for a person living in an MSA with an index of 0.40; by 41% for a person living in an area with an index of 0.60; and by 77% for a person living in an area with an index of 0.83 (the highest level of segregation in the sample), adjusting for multiple covariates. As with the model for BMI, adjusting for physical activity and diet resulted in a minor attenuation of the estimated coefficient for segregation (OR:1.133 in the overweight model, $p < 0.01$). Consistent with the models for BMI, the estimates for segregation are also positive among whites, but are not significant for either overweight or obesity despite a considerably larger sample size relative to blacks.

Discussion

To date there have been a limited number of studies on racial residential segregation and health, and most prior work has focused on mortality. This study narrows the scope to weight status, a more specific health outcome that disproportionately affects the most segregated minority group in the US—African Americans—and functions as a potential risk factor for mortality (Hedley et al., 2004; Massey & Denton, 1989). Findings show that for non-Hispanic blacks, racial isolation is positively associated with BMI and greater odds for being overweight. Blacks who live in more segregated metropolitan areas have, on average, a higher weight status and risk of being in a clinically adverse weight category. These relationships do not appear to be confounded by region or metropolitan area characteristics such as aggregate income status and population size. Moreover, these findings extend prior work limited to ecological analyses, demonstrating that these relationships occur above and beyond the effects of individual-level factors such as income and education. Among whites, there was no significant association between segregation and weight status. If segregation is in fact beneficial to whites, as some have argued, this benefit does not appear to manifest itself in healthier weight outcomes.

Black segregation is well known to restrict education, income and employment opportunities for the residents of segregated black neighborhoods, and individual socioeconomic well-being is well

Table 4
Multivariate adjusted odds ratio of overweight and obesity

Dependent variable	OR for MSA Isolation Index [95% CI]	
	Blacks ($N = 8800$)	Whites ($N = 35,410$)
Overweight	1.139 [1.045–1.242] **	1.073 [0.999–1.152]
Obese	1.102 [0.987–1.230]	1.072 [0.979–1.173]

Note: All models adjust for age, sex, marital status, household income, education, health care coverage, MSA population, MSA median income, MSA family poverty, and region. OR = change in odds estimated for a 1 SD increase in isolation.

** $p < 0.01$.

known to bear an inverse relationship to health measured along numerous dimensions (Goldman, 2001). Hence, segregation may affect health in part through an effect on individual socioeconomic well-being. An association with weight status that is independent of individual SES, however, suggests that segregation can also have a more direct contextual influence on weight status. These findings show that blacks experience a higher risk of being overweight when living in a more segregated metropolitan area, even with equivalent levels of individual education and (household) income. Furthermore, given that estimates for the association between segregation and weight status were minimally attenuated with adjustments for individual income and education, individual SES may play a relatively small role in mediating this relationship.

As previously discussed, segregation and the concentration of poverty could affect weight outcomes through many pathways. Racial isolation, a distinctively contextual phenomenon, is known to influence values and aspirations relating to various sociological outcomes, and it may similarly influence goals or preferences with respect to health and body weight. The prevalence of overweight may be quite high in hyper-segregated black urban enclaves, and heavier average weight among neighborhood peers can shift the local standard of comparison, potentially blunting any broader, normative stigma associated with being overweight. Recent empirical work is consistent with this pathway (Boardman, Saint Onge, Rogers & Denney, 2005). Furthermore, segregation, by creating a context of mismatch between mainstream expectation and actual opportunity, is thought to promote the development of subcultures where value schemes are formed in direct opposition to wider expectations. As such, there might also be a greater tendency to reject any normative emphasis on personal health promotion endeavors, as well as mainstream directives with respect to nutrition, exercise, and ideal weight. Hence, in addition to pathways between segregation and health that are mediated by an effect on individual SES, and to those mediated by the provision of local services and amenities (e.g., supermarkets, parks, recreational facilities, etc.), isolation may also be capturing a more direct sociocultural effect from segregation itself.

The findings of this study differ from those of a recent study by Robert and Reither (2004) where community (census tract) percent black was

not associated with BMI. Their study uses data from the 1980s, resulting in a 20-year time difference from this study and potential changes in segregation patterns. Furthermore, the distribution of weight status has shifted quite dramatically since the 1980s, with large increases in the prevalence of overweight and obesity (Chang & Lauderdale, 2005; Hedley et al., 2004). Given the host of environmental changes that are thought to contribute to such shifts, and a concomitantly changing cultural context, the various weight-promoting pathways hypothesized to be exacerbated or induced by segregation may be more salient now than they were in the past.

MSA-level poverty was not significantly associated with weight status, suggesting that the main effect of black isolation on weight status may operate more through the consequences of social isolation than through the concentration of poverty. As previously discussed, the sequelae of racial segregation are not limited to the concentration of poverty and the provision of material services and amenities. On the other hand, MSA-level poverty does not speak to how poverty is concentrated across individual neighborhoods. Lastly, adjustments for basic indicators of individual-level physical activity and fruit/vegetable intake resulted in only minor attenuation of the association between metropolitan area isolation and weight status. While these measures are based on survey variables designed to approximate national health guidelines, diet and physical activity are complex forms of behavior, and such simple measures may not capture sufficiently detailed differences between persons.

This study is limited by the fact that individuals are not linked to specific neighborhoods and by the fact that the isolation index does not measure specific neighborhood characteristics. Hence, additional research is needed not only to capture the effects of segregation at a smaller spatial level, but also to study potential intermediary pathways between segregation and health such as specific local neighborhood characteristics. With respect to weight status, a number of studies have provided preliminary evidence that more specific features of neighborhoods, such as the quality and quantity of various food outlets and recreational spaces, may influence diet and physical activity (Humpel, Owen, & Leslie, 2002; Morland, Wing, & Diez Roux, 2002). As previously noted, there is also new empirical work linking segregation to specific neighborhood features such as supermarkets and the nutritional quality of local food selections.

Ideally, future research would empirically illustrate the full pathway from segregation to neighborhood to health behavior and health. Despite the importance of delineating and intervening on the more proximal elements of residential life, the amelioration of population-level health disparities will also require attention to underlying structural forces—such as segregation—that ultimately contribute to the configuration of local conditions. Racial segregation, like other social inequities, has been conceptualized as a “fundamental” cause of disparities in health and disease (Williams & Collins, 2001) and can provide a much broader context for existing studies on the influence of detailed neighborhood characteristics. It should also be noted that this study, which is cross-sectional, cannot address the possibility that weight status may influence place of residence. Racial discrimination, however, is thought to be the predominant force driving racial segregation (Massey & Denton, 1993).

A second limitation to this study is that the BRFSS relies on self-reported height and weight. If, however, error occurs predominantly at the higher end of the weight spectrum due to underreporting of weight (e.g., some portion of overweight persons are misclassified as normal weight), then this would lend a conservative bias to the findings of this study. Nationally representative data on measured weight status (e.g., the National Health and Nutrition Examination Survey) does not permit the identification of respondents into a sufficient number of metropolitan areas for this type of analysis. Lastly, this study, like much research on segregation, is limited by the fact that standard measures of segregation do not assess actual social contact and rely entirely on place of residence. Future work could address actual contact between persons and non-residential settings such as school and work. LaVeist (2003) has recently used a measure of self-reported exposure to segregation that addresses such issues.

In the context of much prior work linking segregation to an increased risk of mortality among blacks, weight status may function as an intermediary health outcome in this relationship. Conventional medical models of risk assessment and intervention frequently focus on the characteristics of individuals, typically focusing on lifestyle behaviors. This study contributes further evidence to the fact that known risk behaviors at the individual-level may be conditioned by risk factors understood on a spatial level, risk factors reflecting structural

conditions that are quite beyond individual control. Given that a rise in income inequality has been mirrored by an increasing divergence of residential environments (Massey, 1996), differences among places and the spatial organization of persons may be as critical to health policy and promotion efforts as differences among people.

Acknowledgements

This research was supported by a Career Development Award (K12-HD-043459) from the National Institutes of Health, National Institute of Child Health and Human Development, the Measy Foundation, and the University of Pennsylvania and Cheney University EXPORT Center of Excellence for Inner City Health. I thank Shiriki Kumanyika for insightful discussions on this topic, Dawei Xie and Michael Elliot for consultation on the analyses, and Jason Schnittker and Theodore Iwashyna for helpful comments on the manuscript.

References

- Acevedo-Garcia, D., & Lochner, K. A. (2003). Residential segregation and health. In I. Kawachi, & L. F. Berkman (Eds.), *Neighborhoods and health* (pp. 265–281). New York: Oxford University Press.
- Anderson, E. (1990). *Streetwise: Race, class, and change in an urban community*. Chicago: University of Chicago Press.
- Bird, S. T. (1995). Separate black and white infant mortality models: Differences in the importance of structural variables. *Social Science & Medicine*, 41(11), 1507–1512.
- Boardman, J. D., Saint Onge, J. M., Rogers, R. G., & Denney, J. T. (2005). *Journal of Health and Social Behavior*, 46(3), 229–243.
- Chang, V. W., & Christakis, N. A. (2003). Self-perception of weight appropriateness in the United States. *American Journal of Preventive Medicine*, 24(4), 332–339.
- Chang, V. W., & Lauderdale, D. S. (2005). Income disparities in body mass index and obesity in the United States, 1971–2002. *Archives of Internal Medicine*, 165(18), 2122–2128.
- Collins, C., & Williams, D. R. (1999). *Segregation and mortality: The deadly effects of racism? Sociological Forum*, 14(3), 495–523.
- Fang, J., Madhavan, S., Bosworth, W., & Alderman, M. H. (1998). Residential segregation and mortality in new york city. *Social Science & Medicine*, 47(4), 469–476.
- Fitzpatrick, K., & Lagory, M. (2000). *Unhealthy places: The ecology of risk in the urban landscape*. New York: Routledge.
- Goldman, N. (2001). Social inequalities in health: Disentangling the underlying mechanisms. *Annals of the New York Academy of Sciences*, 954, 118–139.
- Guest, A. M., Gunnar, A., & Hussey, J. M. (1998). The ecology of race and socioeconomic distress: Infant and working-age mortality in Chicago. *Demography*, 35(1), 23–34.

- Hart, K. D., Kunitz, S. J., Sell, R. R., & Mukamel, D. B. (1998). Metropolitan governance, residential segregation, and mortality among African Americans. *American Journal of Public Health, 88*(3), 434–438.
- Hedley, A. A., Ogden, C. L., Johnson, C. L., Carroll, M. D., Curtin, L. R., & Flegal, K. M. (2004). Prevalence of overweight and obesity among us children, adolescents, and adults, 1999–2002. *JAMA, 291*(23), 2847–2850.
- Humpel, N., Owen, N., & Leslie, E. (2002). Environmental factors associated with adults' participation in physical activity. *American Journal of Preventive Medicine, 22*, 188–199.
- Jackson, S. A., Anderson, R. T., Johnson, N. J., & Sorlie, P. D. (2000). The relation of residential segregation to all-cause mortality: A study in black and white. *American Journal of Public Health, 90*(4), 615–617.
- Kumanyika, S. K. (1998). Obesity in African Americans: Biobehavioral consequences of culture. *Ethnicity and Disease, 8*, 93–96.
- LaVeist, T. A. (1989). Linking residential segregation to the infant-mortality race disparity in U.S. Cities. *Social Science Research, 73*(2), 90–94.
- LaVeist, T. A. (1993). Segregation, poverty and empowerment: Health consequences for African-Americans. *The Milbank Quarterly, 71*(1), 41–64.
- LaVeist, T. A. (2003). Racial segregation and longevity among African-Americans: An individual level analysis. *Health Services Research, 38*(6, Part II), 1719–1733.
- Leclere, F. B., Rogers, R. G., & Peters, K. (1997). Ethnicity and mortality in the United States: Individual and community correlates. *Social Forces, 76*(1), 169–198.
- Leclere, F. B., Rogers, R. G., & Peters, K. (1998). Neighborhood social context and racial differences in women's heart disease mortality. *Journal of Health and Social Behavior, 39*, 91–107.
- Lewis, L. B., Sloane, D. C., Nascimento, L. M., Diamant, A. L., Guinyard, J. J., Yancey, A. K., & Flynn, G. (2005). African Americans' access to healthy food options in south los angeles restaurants. *American Journal of Public Health, 95*(4), 668–673.
- Manson, J. E., Willet, W. C., Stampfer, M. J., Colditz, G. A., Hunter, D. J., Hankinson, S. E., Hennekens, C. H., & Speizer, F. E. (1995). Body weight and mortality among women. *New England Journal of Medicine, 333*, 677–685.
- Massey, D. S., & Denton, N. A. (1988). The dimensions of residential segregation. *Social Forces, 67*(2), 281–315.
- Massey, D. S., & Denton, N. A. (1989). Hypersegregation in U.S. Metropolitan areas: Black and hispanic segregation along five dimensions. *Demography, 26*(3), 373–391.
- Massey, D. S. (1990). American apartheid: Segregation and the making of the underclass. *American Journal of Sociology, 96*(2), 329–357.
- Massey, D. S., Gross, A. B., & Eggers, M. L. (1991). Segregation, the concentration of poverty, and the life chances of individuals. *Social Science Research, 20*, 397–420.
- Massey, D. S., & Denton, N. A. (1993). *American apartheid: Segregation and the making of the underclass*. Cambridge, MA: Harvard University Press.
- Massey, D. S. (1996). The age of extremes: Concentrated affluence and poverty in the twenty-first century. *Demography, 33*(4), 395–412.
- McCord, C., & Freeman, H. P. (1990). Excess mortality in harlem. *New England Journal of Medicine, 322*(3), 173–177.
- Morland, K., Wing, S., Diez Roux, A., & Poole, C. (2001). Neighborhood characteristics associated with the location of food stores and food service places. *American Journal of Preventive Medicine, 22*, 23–29.
- Morland, K., Wing, S., & Diez Roux, A. (2002). The contextual effect of the local food environment on residents' diets: The atherosclerosis risk in communities study. *American Journal of Public Health, 92*(11), 1761–1767.
- Must, A., Spadano, J., Coakley, E., Field, A., Colditz, G. A., & Dietz, W. H. (1999). The disease burden associated with overweight and obesity. *JAMA, 282*, 1523–1529.
- Palloni, A., & Arias, E. (2004). Paradox lost: Explaining the hispanic adult mortality advantage. *Demography, 41*(3), 385–415.
- Pickett, K. E., & Pearl, M. (2000). Multilevel analysis of neighborhood socioeconomic context and health outcomes: A critical review. *Journal of Epidemiology and Community Health, 55*, 111–122.
- Polednak, A. P. (1993). Poverty, residential segregation, and black/white mortality ratios in urban areas. *Journal of Health Care for the Poor and Underserved, 4*(4), 363–373.
- Polednak, A. P. (1996a). Segregation, discrimination, and mortality in U.S. Blacks. *Ethnicity and Disease, 6*, 99–108.
- Polednak, A. P. (1996b). Trends in us urban black infant mortality, by degree of residential segregation. *American Journal of Public Health, 86*(5), 723–726.
- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models*. Thousand Oaks, CA: Sage Publications.
- Robert, S. A., & Reither, E. N. (2004). A multilevel analysis of race, community disadvantage, and body mass index among adults in the us. *Social Science & Medicine, 59*, 2421–2434.
- Shihadeh, E. S., & Flynn, N. (1996). Segregation and crime: The effect of black social isolation on the rates of black urban violence. *Social Forces, 74*(4), 1325–1352.
- Subramanian, S. V., Acevedo-Garcia, D., & Osypuk, T. L. (2005). Racial residential segregation and geographic heterogeneity in black/white disparity in poor self-rated health in the us: A multilevel statistical analysis. *Social Science & Medicine, 60*, 1667–1679.
- US Department of Agriculture. (2000). Nutrition and your health: Dietary guidelines for americans. Washington, DC: US Government Printing Office.
- Williams, D. R., & Collins, C. (2001). Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Reports, 116*, 404–416.
- Wilson, W. J. (1987). *The truly disadvantaged: The inner city, the underclass, and public policy*. Chicago: University of Chicago Press.
- Wilson, W. J. (1997). *When work disappears*. New York: Vintage Books.
- Yankauer, A. (1950). The relationship of fetal and infant mortality to residential segregation: An inquiry into social epidemiology. *American Sociological Review, 15*(5), 644–648.
- Zenk, S. N., Schulz, A. J., Israel, B. A., James, S. A., Bao, S., & Wilson, M. L. (2005). Neighborhood racial composition, neighborhood poverty, and the spatial accessibility of supermarkets in metropolitan detroit. *American Journal of Public Health, 95*(4), 660–667.