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Race Differentials in Obesity: The Impact of Place*

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Abstract

This article reveals race differentials in obesity as both an individual- and neighborhood-level phenomena. Using neighborhood-level data from the 1990–1994 National Health Interview Survey, we find that neighborhoods characterized by high proportions of black residents have a greater prevalence of obesity than areas in which the majority of the residents are white. Using individual-level data, we also find that residents of neighborhoods in which at least one-quarter of the residents are black face a 13 percent increase in the odds of being obese compared to residents of other communities. The association between neighborhood racial composition and obesity is completely attenuated after including statistical controls for the poverty rate and obesity prevalence of respondents' neighborhoods. These findings support the underlying assumptions of both institutional and social models of neighborhood effects.

The causes of obesity, including the social relationships that mediate and moderate the relationship between various risk factors and obesity, are complex (Weinsier et al. 1998; Whitaker 2002; Bloomgarden 2002). As research consistently demonstrates that behaviors affecting health are rooted within individuals' social environments (Berkman and Kawachi 2000), it is critical to broaden the scope of inquiry such that health is understood not only as a function of individual traits, but also as related to the environments in which people live (MacIntyre and Ellaway 2003; Robert 1999). These environments include physical space as well as community attitudes and behaviors that characterize these places (Frolich, Corin, and Potvin 2001). Although literature linking residential context to health has increased sharply in recent years (see Kawachi and Berkman 2003 for an overview), little work has examined obesity as an outcome. This article contributes to the growing body of work focusing on the ecological correlates of health and makes a timely contribution to the recent focus on obesity in both academic and popular settings.

It is important to fully understand obesity because obesity prevalence among U.S. adults has increased to epidemic levels; has contributed to an increased risk of disease, disability, and death; and has led to an escalation in health care costs (Allison, Zannolli, and Narayan 1999; Wang et al. 2002). Over the last 25 years, the prevalence of adult obesity has more than doubled; in 1976 only 15 percent of the adult population was obese, but by 2000 adult obesity rates surpassed 30 percent (NCHS 2003). This increase is particularly problematic because obesity is associated with an increased risk of serious health problems including type-2 diabetes, gallbladder disease, high blood pressure, and heart disease (Flegal et al. 2002; Calle et al. 2003; Kaplan 2000; Must et al. 1999). More importantly, obesity increases the risk of a number of causes of death and is believed to contribute to approximately 14

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percent of all deaths per year in the United States (Allison and Fontaine et al. 1999; McGinnis and Foege 1993; Rogers, Hummer, and Krueger 2003; Sturm 2002).

Of particular concern to social epidemiologists is consistent evidence demonstrating a substantially higher rate of obesity among non-Hispanic blacks compared to non-Hispanic whites in the United States (Denney et al. 2004; Flegal et al. 2002; Durazo-Arvizu et al. 1998; Mokdad et al. 2003; Schoenborn, Adams, and Barnes 2002). In large part, studies examining the elevated risk of obesity among black adults focus almost exclusively on individual-level resources; however, it is clear from previous research that racial differences in body mass persist despite statistical controls for known risk factors at the individual level (Lakdawalla and Philipson 2002; Sundquist and Johansson 1998). Accordingly, researchers have begun to focus their attention on various aspects of adult's neighborhoods (MacIntyre, Ellaway, and Cummins 2002), and there is consistent evidence that various aspects of neighborhoods are independently associated with the health (Robert 1999; Yen and Syme 1999) and health-related behaviors (Boardman et al. 2001; Ross 2000; Duncan, Jones, and Moon 1996) of persons residing in the neighborhood.

In this article, we examine several aspects of individuals' residential areas as potential determinants of their physical size. We first describe the social ecology of obesity by examining racial and socioeconomic correlates of obesity prevalence across residential areas. We then estimate the independent association of three residential characteristics (race, class, and health) on the risk that an individual will be obese. We pay particular attention to the possibility that neighborhood-level racial composition, poverty rates, and obesity prevalence are positively associated with the risk of obesity among adults. These models are used to provide an empirical examination of the epidemic, institutional, and collective socialization models of neighborhood-health relationships (Jencks and Mayer 1990).

HEALTH, PLACE, AND RACE

The relationship between residential context and well-being is broadly understood in reference to the neighborhood effects paradigm (Jencks and Mayer 1990). Research in this area addresses a relatively straightforward question: Are neighborhood characteristics associated with people's life chances, regardless of their personal characteristics (Robert 1998)? Although recent work has elaborated the relationship between neighborhood context and health (see Frolich et al. 2001 for a useful discussion), three models of neighborhood effects presented by Jencks and Mayer (1990) remain the most widely cited and popularly understood mechanisms through which the social, cultural, and economic characteristics of individuals' neighborhoods might affect their well-being. The *institutional model* claims that residents of relatively disadvantaged communities will present more deleterious health profiles compared to residents of more affluent communities—regardless of their personal characteristics—because an important health-promoting infrastructure (Morland, Wing, and Diez-Roux 2002; Lee and Cubbin 2002; Saelens et al. 2003) and health-related services (Perloff and Jaffee 1999; O'Loughlin et al. 1999) are inaccessible, ineffective, or unavailable in impoverished communities. For example, researchers draw on institutional explanations for the increased risk of alcohol use and abuse in predominantly black communities when they highlight the disproportionate concentration of alcohol outlets in these areas (Scribner et al. 1999). Similarly, the institutional model of neighborhood effects may highlight the concentration of polluting industries or congestion within major transportation networks within disadvantaged communities (Downey 2003) as a potential explanation for elevated risk of poor health in these areas.

Whereas the institutional model focuses on risks and resources that are external to the residents, social models focus on norms, values, and beliefs that are unique to the residents

of the neighborhoods. These models hypothesize that neighborhoods provide a social context in which otherwise subcultural values regarding health-related behaviors become normative (Jencks and Mayer 1990). Neighborhood context can influence behavior directly via imitation processes, or indirectly through the internalization of norms and attitudes present within the collective lifestyles of the neighborhood. For example, the concentration of individuals within a neighborhood with elevated weight may lead to an overarching acceptance of obesity as collective attitudes toward obesity discredit or dismiss (directly or indirectly) the potential harms of being obese. These social interactions occurring within neighborhoods may ultimately place all residents of the community at an elevated risk of being obese.

Two of the most widely cited social models are the epidemic and collective socialization models. The models differ one from another with respect to the functional form that each is expected to take. Specifically, the epidemic model suggests that the risk of obesity for a randomly selected individual from a neighborhood increases exponentially with the prevalence of obesity within the area; the estimated net effect of neighborhood obesity rates on the risk of an individual being obese increases with increasing obesity prevalence within neighborhoods. Crane (1991) summarizes the epidemic model as follows: “if the incidence stays below a critical point, the frequency or prevalence of the problem tends to gravitate toward some relatively low-level equilibrium. But if the incidence reaches a critical point, the process of spread will explode” (p. 1227). Borrowing from traditional models of disease progression in populations, the epidemic model is unique because the risk associated with contextual characteristics, is believed to increase without bounds beyond some “tipping point” (Granovetter 1978).

The collective socialization model also predicts a nonlinear specification in the relationship between neighborhood context and the risk of social problems, but the model differs from the epidemic model because it predicts stabilization in the risk of some deleterious outcome beyond the hypothesized tipping point. The collective socialization model differs in that the normative environment is believed to be discrete; changes in the obesity composition of the neighborhoods denote qualitative rather than quantitative shifts in the meaning of health problems. Therefore, if the relationship between neighborhood obesity prevalence and the risk of obesity is best described by the collective socialization model, then we would expect increases in the obesity prevalence to more strongly impact the risk of obesity at or near the average levels of neighborhood obesity prevalence. Said differently, according to the collective socialization model, increasing levels of neighborhood obesity should have the weakest effects on individual residents’ risk of obesity at the lowest and highest levels of obesity prevalence, respectively.

The neighborhood epidemic and collective socialization models are important for understanding race differences in obesity because black adults may prefer larger body sizes (Flynn and Fitzgibbon 1998; Hebl and Heatherton 1998), which may in turn affect motivations to lose weight (Kumanyika 1993). These preferences may also translate into different sanctions when aggregated to more abstract social levels; the increased acceptance of elevated weight among non-Hispanic blacks decreases the costs associated with obesity, subsequently increasing the likelihood that black individuals will be obese (Dawson 1988). For example, a study of undergraduate female college students revealed that, compared to whites, African American females reported higher current body masses, higher desired body masses, and higher body masses perceived to be desired by others (DiGioacchino, Sargent, and Topping 2001). Further, Stevens, Kumanyika, and Keil (1994) found that, compared to obese white women, obese black women were more than twice as likely to be satisfied with their weight.

To date, little work has investigated the ways in which adults' immediate residential context factors into their perceptions of normal weight and their subsequent likelihood of engaging in health-promoting activities to maintain or attain a particular physical weight. More importantly, given the observed differences among black and white adults with respect to the risk of obesity as well as perceived body size, it is possible that the heightened prevalence of obesity within black communities is independently associated with the risk of obesity among the residents of the immediate area. This is an important aspect of the notion of residential concentration that has been overlooked. That is, the concentration of racial and ethnic minorities is deemed largely problematic because of group-level differences in socioeconomic well-being (Jargowsky 1997). Specifically, because black Americans are three times more likely than whites to have family incomes below the official poverty line (Proctor and Dalaker 2003), the concentration of non-Hispanic blacks within particular neighborhoods also concentrates poverty and a host of poverty-associated social problems. We consider the possibility that residential racial concentration leads to elevated rates of obesity among black compared to white adults because of race differences in the prevalence of obesity (epidemic or collective socialization model) and race differences in neighborhood socioeconomic status (institutional model).

HYPOTHESES

Based on the literature reviewed above, we test the following eight hypotheses:

Hypothesis 1: Residents of black communities face an increased risk of obesity compared to residents of nonblack communities.

Hypothesis 2: The elevated risk of obesity among black adults is due in part to residential racial concentration.

Hypothesis 3: Neighborhood poverty rates are positively associated with the risk of obesity among adults.

Hypothesis 4: The elevated risk of obesity in predominantly black communities is due to disparate levels of socioeconomic resources across black and white communities (institutional model).

Hypothesis 5: Individuals residing in relatively obese communities will face an increased risk of being obese themselves.

Hypothesis 6: The effect of neighborhood obesity prevalence on the risk of obesity among individual residents will increase rapidly and continue to increase (epidemic model).

Hypothesis 7: The effect of neighborhood obesity prevalence on the risk of obesity among individual residents will increase rapidly but then plateau (collective socialization model).

Hypothesis 8: The elevated risk of obesity in predominantly black communities is due to neighborhood differences in obesity rates.

METHODS

Data

Individual-level and neighborhood-level data come from the 1990–1994 National Health Interview Survey (NHIS; NCHS 1990, 1992, 1993, 1994, 1995). NHIS is an annual, nationally representative survey of over 100,000 noninstitutionalized individuals used to examine national trends in illness and disability and to track progress toward achieving national health objectives (Schoenborn, Adams, and Schiller 2003). In our analyses we

include information on respondent's height, weight, age, sex, race, educational attainment, and poverty status. Data were limited to 1994 due to the reconstructed sampling design that occurred in 1995.

Neighborhood-level data are derived from a methodology developed by Wells and Horm (1998) of the National Center for Health Statistics (NCHS). To protect respondents, NCHS excludes small geographic identifiers from public release data sets. Because the NHIS sampling frame is based on Census blocks and block groups, Wells and Horm use the term "very small area" (VSA) to denote the geographic areas referred to as neighborhoods in the present analyses. VSAs provide three unique advantages in contextual analyses. First, because VSAs are smaller than Census tracts, they may provide a more accurate indicator of neighborhoods (Bond Huie 2001; Brooks-Gunn, Duncan, and Aber 1997; Booth and Crouter 2001). Second, the NHIS is available on an annual basis and the geographic identifiers for the VSAs do not vary, which provides neighborhood data for each year of the survey (Bond Huie, Hummer, and Rogers 2002). Last, these areas are defined so that they capture similar social, economic, and cultural characteristics of the area.

NHIS collected information from 402,154 respondents over the five-year period. Because VSA-level information is derived from the aggregation of individual respondents and multiple records per household were collected in the NHIS, the prevalence of obesity in a neighborhood may be exaggerated or decreased because of differences in the number of respondents per household. Accordingly, we randomly selected one adult per household for these analyses. This process provided a reduced sample of 220,347 respondents. Of these adult respondents, we dropped 2.9 percent or 6,302 adults from the analyses because small VSA-level sample sizes (i.e., less than 10 respondents per VSA) may lead to unreliable neighborhood-level measures (Sampson, Raudenbush, and Earls 1997). This produced a final data set of 220,347 respondents from 7,953 neighborhoods. Overall, there was a mean of 29.6 respondents per neighborhood (SD = 9.8), with a minimum of 10 and a maximum of 76.

Measures

The dependent variable, obesity, is a binary measure, coded 1 if an individual is obese and 0 if not obese. Obesity is determined if an individual's body mass index (BMI) is greater than or equal to 30. BMI is calculated by dividing an individual's weight (in kilograms) by his or her height in meters squared (World Health Organization 1997). Previous research finds self-reported data such as these to be reliable measures of physical size (Stewart 1982).

Because of our interest in black-white differences in the risk of obesity, only respondents who described themselves as non-Hispanic white (reference group) or non-Hispanic black were included.1 Additional individual-level measures include demographic and socioeconomic variables that are associated with both race and obesity (Flegal et al. 2002; Durazo-Arvizu et al. 1998). First, given the nonlinear relationship between age and obesity (Himes 2000; Krueger et al. 2004), we use four age categories in all analyses: 18–29 (the referent), 30–49, 50–69, and 70 and older. Sex is coded 1 for males and 0 for females. Educational attainment includes 13 or more (the referent), 12, and less than 12 years of schooling (for similar coding see Rogers, Hummer, and Nam 2000). Finally, poverty is a dichotomous measure, coded 1 for individuals who are living at or below the poverty threshold. The poverty threshold is based on family size, number of children under 18 years

[1]Although it would be informative to examine other ethnic groups, their small sample sizes, especially at the neighborhood level, would lead to analytic problems, particularly with the multilevel analyses.

of age, and family income, using poverty levels from the particular NHIS year derived from the subsequent year's Current Population Survey.

Neighborhood-level independent variables are calculated by aggregating individuals' responses within each respective VSA. Three characteristics of respondents' neighborhoods are assessed: the proportion of residents who are non-Hispanic black, the proportion of residents who are poor, and the proportion of residents who are obese.2 Non-Hispanic black and poor communities are identified as areas in which at least 25 percent of the residents are black or poor, respectively. Because we are interested in the functional form of the relationship between neighborhood obesity and the risk of individual residents being obese (epidemic or collective socialization), we use the following six dummy variables to capture neighborhood obesity prevalence: (1) 0–4.9 percent (the referent), (2) 5–9.9 percent, (3) 10–14.9 percent, (4) 15–19.9 percent, (5) 20–24.9 percent, and (6) 25 percent and higher.

Statistical Analyses

Due to the multilevel character of the research questions, we use the SAS GLIMMIX macro to estimate multilevel generalized linear models with a logit link and a binary distribution. Error across neighborhoods is captured with a level-2 residual term that is believed to be normally distributed with a mean of zero and an unknown variance of σ_u^2 (Littell et al. 1996; McCulloch and Searle 2001). The multilevel model for binary outcomes is conceptually quite similar to a traditional logistic regression model with the inclusion of the neighborhood-level error component (u_j). The following equation represents a multilevel equation for the probability of being obese, allowing obesity to vary across neighborhoods and including individual-level (x_{ij}) and neighborhood-level (z_j) explanatory variables:

$$\log\left(\frac{P_{ij}}{1-P_{ij}}\right)=\beta_0+\beta_1 x_{ij}+\beta_2 z_j+u_j. \quad (1)$$

The probability (P_{ij}) that the *i*th individual in the *j*th neighborhood is obese is captured in equation 1. The variance of the level-2 residual ((σ_u^2)) can be used to estimate the extent to which residual variation in the log-odds of obesity is situated within or between neighborhoods. The intraclass correlation coefficient is simply the ratio of level-2 residual variance to the overall residual variance ($\sigma_u^2+\sigma_e^2$). Snijders and Bosker (1999) and Guo and Zhao (2000) suggest using $\Pi^2/3$ (the variance of the standard logistic distribution) as an estimate for the level-1 residual variance when modeling binary outcomes in a multilevel framework. Finally, because NHIS uses a clustered, stratified unequal probability sampling frame, we weighted all estimates to reflect the civilian, noninstitutionalized adult population (Massey et al. 1989).

RESULTS

Table 1 presents obesity prevalence rates across neighborhoods in the years 1990–1994. The average neighborhood in the United States had an obesity rate of 14.7 percent during these years, and almost 15 percent of adults in the United States resided in communities where at least one-quarter of the residents were obese. The overall black and nonblack community

[2]Because we are interested in modeling the probability that an individual is obese given the prevalence of obesity in his or her neighborhood, the inclusion of information about that individual in the neighborhood prevalence rate would bias the parameter estimates and artificially lower the standard error estimates. Accordingly, each respondent receives a unique value for the obesity prevalence value as they are removed from the calculation of the neighborhood-level estimate. This same method was used to calculate neighborhood racial and socioeconomic composition.

prevalence rates are strikingly different. In particular, less than 10 percent of nonblack areas had obesity rates that exceeded 25 percent, but nearly two out of every five (37.6 percent) black communities presented similarly high obesity levels. Whereas 14.3 percent of nonblack areas had very low obesity rates, only 3.4 percent of black communities had obesity prevalence rates below 5 percent.

A similar pattern emerges when comparing poor and nonpoor areas: Poor areas were three times more likely than nonpoor areas to have obesity rates that exceeded 25 percent. Given that black communities are overrepresented among relatively impoverished areas (Jargowksy 1997), we also consider the relationship between racial composition and obesity prevalence rates across poor and nonpoor areas. Whereas more than 50 percent of impoverished black communities had obesity prevalence rates beyond 25 percent, only 22 percent of nonblack communities had obesity rates this high. The prevalence of high obesity among black communities was the lowest in nonpoor areas; however, obesity rates in nonpoor black communities were still higher than obesity rates in poor nonblack communities. In addition, the relative risk of high obesity rates across black and nonblack communities indicates that socioeconomic differentials are only partly responsible for these differences. To illustrate, black communities are 3.8 times as likely as white communities to have obesity rates beyond 25 percent, but this relative risk drops to 3.4 when comparing black and white communities in nonpoor areas and even farther (2.3) when comparing black and white communities in poor areas.

To more fully appreciate these relationships, we merged neighborhood-level data with individual-level records from the NHIS. Table 2 presents descriptive statistics for black and white respondents. Of particular interest in this table are the observed race differentials in obesity when measured at the individual-level and the neighborhood-level. Specifically, the relationship between race, obesity, and residential context becomes clearer when one considers that black respondents are 1.7 times as likely as whites to be obese, but they are 3.1 times as likely to reside in relatively obese communities. What remains unclear is the extent to which individual-level and neighborhood-level differences between blacks and whites account for the elevated rates of obesity within black communities and the increased risk of obesity among individual black adults.

To investigate these relationships further, Table 3 presents estimates from a series of multilevel logistic regression models in which odds ratios are estimated for each risk factor. These estimates are derived from individual-level data nested within neighborhoods where the dependent variable is coded 1 if respondents are obese and 0 if otherwise. Model 1 presents the null model estimates in order to calculate the unadjusted intraclass correlation coefficient for obesity. According to this model, 8 percent of the variation in the risk of obesity is due to the unobserved characteristics associated with respondents' neighborhoods:

$$\left(\frac{.30}{.30+\frac{\pi^2}{3}}\right)=.08.$$

As with the percentages presented in Table 2, the parameter estimate in model 2 suggests that black adults face an 88 percent increase in the odds of being obese compared to whites ($p_b = .228$; $p_w = .136$; $o_b = .295$; $o_w = .157$; $o_b / o_w = OR = 1.88$). Model 3 examines the extent to which the elevated risk of obesity among black adults is due to sociodemographic differences between blacks and whites. According to these results, less educated ($OR_{<HS} = 1.71$; $OR_{HS} = 1.35$) and poor (OR = 1.36) adults are significantly more likely to be obese compared to adults from higher socioeconomic positions, and these controls reduce the effect of race by roughly 17 percent. That is, 17 percent of the elevated risk of obesity

among black compared to white adults is due to sociodemographic differences in the two groups.

Model 4 controls for the racial composition of respondents' neighborhoods and provides a test of hypotheses 1 and 2. As hypothesized, residing in a black community is positively and significantly associated with an increased risk in obesity among adults (hypothesis 1).3 Those residing in communities in which at least one-quarter of the residents are black face a 13 percent increase in the odds of being obese compared to residents of communities with smaller proportions of blacks. Equally important, as expected (hypothesis 2), differences among black and white respondents in the racial composition of their neighborhoods attenuated the estimated net effect of race by roughly 19 percent. In other words, black adults face an increased risk of obesity compared to whites in part because they are more likely to reside in neighborhoods with high concentrations of black residents.

Models 5 and 6 are designed to account for the elevated risk of obesity among residents of black communities (hypotheses 3 and 4). By controlling for socioeconomic characteristics and obesity profiles of respondents' neighborhoods, the goal is to reduce the net effect associated with residing in a black community to zero. Model 5 controls for the prevalence of poverty in respondents' neighborhoods. As hypothesized (hypothesis 3), residence in a relatively impoverished community is positively associated with an increased risk of obesity. Further, in support of the institutional frame work of neighborhood dynamics (hypothesis 4), socioeconomic differences among white and nonwhite neighborhoods explains approximately 30 percent of the effect associated with neighborhood racial composition. In other words, residents of black communities face an increased risk of obesity because important health-promoting infrastructural resources may be absent in these relatively disadvantaged communities.

Model 6 tests the epidemic and collective socialization models (hypotheses 5–8) by including statistical controls for the prevalence of obesity in respondents' neighborhoods. We are interested in three aspects of this relationship: (1) the direction and significance (hypothesis 5), (2) the functional form (hypotheses 6 and 7), and (3) the extent to which these controls attenuate the residual effect of the neighborhood racial composition variable from model 5 (hypothesis 8). First, we find support for hypothesis 5: Each level of neighborhood obesity rate is strongly and positively associated with an increased risk of obesity for individual residents. Keeping in mind that the prevalence of obesity excludes the respondent, these findings suggest that individuals are more likely to be obese if they reside in a community in which a relatively high proportion of the residents are obese.

Second, the observed increase in the odds of being obese as respondents move from neighborhoods with average obesity levels (OR = 1.34) to above average levels (OR = 1.62) is consistent with both the epidemic and collective socialization models. According to the collective socialization model, this increase should subside rapidly with increasing levels of obesity. Although this pattern is evident, we also see slight increases in the relative odds of individuals being obese when they reside in communities where 20–25 percent (OR = 1.68) and more than 25 percent (OR = 1.77) of their neighbors are obese. Thus, these estimates

[3]It is important to consider the large sample sizes used in these analyses when interpreting statistical significance. As with most inferential statistics, the test statistic here is simply the ratio of the parameter estimate (the slope) and its corresponding standard error. Standard error estimates in this study are quite small because of the large sample sizes; even odds ratios near 1.0 may prove to be statistically significant. Moreover, the magnitude of these estimates should be considered carefully when interpreting the results. Nevertheless, all of the statistically significant results are significant at the $p < .01$ if not $p < .001$ level. Moreover, it is also important to consider that contextual predictors, although smaller in magnitude, may have important substantive significance because these effects are believed to impact all of the residents of a particular community. Small changes in the contextual aspects or the sociodemographic composition of a neighborhood may have a greater social impact compared to similar changes among individuals or families.

support key assumptions of both the epidemic (hypothesis 6) and the collective socialization (hypothesis 7) models of socially driven neighborhood effects.

Third, and most importantly, statistical controls for the prevalence of obesity in respondents' neighborhoods reduce the effect of the racial composition of respondents' neighborhoods from 1.09 ($p < .05$) in model 5 to 1.01 ($p > .05$) in model 6. In other words, while model 5 describes a 9 percent increase in the odds of being obese for residents of black neighborhoods compared to residents of nonblack neighborhoods, when comparing black and white neighborhoods with similar levels of obesity, residing in a black neighborhood is no longer associated with an individual's risk of being obese. This suggests that black-white differences in neighborhood obesity prevalence entirely mediate the relationship between neighborhood racial composition and the elevated risk of obesity among residents of black communities. Thus, in support of hypothesis 8, the elevated risk of obesity among residents of black neighborhoods is due in part to institutional characteristics of these neighborhoods, but also because obesity levels within these communities cross important, yet undefined, thresholds.

DISCUSSION

The results presented above effectively demonstrate that contextual predictors at the neighborhood level provide valuable insight into the risk of obesity among adults in the United States. Indeed, individuals living in neighborhoods characterized by relatively high proportions of obese residents are significantly more likely to be obese themselves, net of individual-level differences. These findings contribute to a growing body of literature involving neighborhood effects on health (Kawachi and Berkman 2003; Robert 1999) as well as social epidemiological inquiries into race differences in health and well-being (Williams and Collins 1995; Williams 1997). Blacks and whites often inhabit qualitatively dissimilar areas in the United States, and high levels of racial concentration leads to disparate cultural and structural environments for blacks and whites (Alba and Logan 1993; Farley and Frey 1994; Frey and Farley 1996; Massey and Denton 1993). These differences have a profound impact on the physical well-being of black and white adults throughout the life course (LaVeist 1996; Williams 1996, 1997). As shown here, the geographic and social positions of whites and blacks affect their obesity levels.

The findings suggest that increased risk of obesity among residents of black communities is due in part to socioeconomic differences in the communities, but more importantly, this risk is attenuated when the obesity rate of the neighborhood is considered. These findings support both institutional and social models of neighborhood effects and suggest that elevated risk of obesity among black adults is due in part to residential racial concentration. These results confirm studies that indicate that residential racial concentration operates as an indirect effect on health outcomes, primarily through concentrated poverty (Williams 1996; Acevedo-Garcia 2000). Living in neighborhoods characterized by high levels of poverty increases the probability of a host of negative health outcomes, including increasing an individual's probability of being obese net of individual characteristics, such as gender, income, and education.

Social models focus on how individuals within a given context influence one another's behaviors and norms. The processes result from imitation behavior and are conditioned by a willingness or susceptibility to submit to the prevailing norms present within a given locale. Our results provide support for contagion arguments that suggest that residing in areas characterized by high levels of obesity may minimize the social costs associated with being obese. These social models are not necessarily short-term and hurried, but rather developing and progressive processes. Health outcomes, and more specifically obesity, are not the result

of immediate imitation processes that result in immediate pandemic proportions. Whereas negative behaviors such as drug activity or teenage sexual behavior might potentially be viewed as a short-term trend or epidemic (Crane 1991), obesity is a result of long-term health behaviors, including physical activities and nutritional intake that are invariably connected to institutions, as illustrated in the results. Health behaviors are not necessarily deep-rooted personality traits but rather reflect the social milieu in which one interacts (Frolich et al. 2001).

Negative health behaviors are slowly disseminated through the social and cultural environment. Individuals are not unexpectedly inflicted by unhealthy behavior, but are continually exposed over time until more and more individuals are exposed. Attitudes are internalized and externalized and continue to influence the residents of a given area at a rapid rate. Rather than being a passive recipient or susceptible to disease, the individual is influenced by his or her neighborhood over time and concomitantly influences the neighborhood (Tienda 1991). All residents have agency and are not necessarily predetermined to become obese, but they are at increased risk with increased exposure levels. Thus, the minimization of body mass concerns results from increased exposure to individuals exhibiting unhealthy practices, buttressed by institutional-level factors, including but not limited to racial concentration.

The collective minimization of obesity affects all individuals living in areas characterized by obesity, but it has particularly damaging effects on blacks because of persistently high levels of residential segregation. This highlights the importance of policies that address specific geographic areas and subpopulations. Blacks and individuals with lower socioeconomic status are less likely to accept norms that encourage weight loss or that stigmatize obesity (Jeffery and French 1996; Allan, Mayo, and Michel 1993). Thus, disproportionately obese and black neighborhoods may be characterized by less confining attitudes toward obesity. Such relaxed attitudes may protect against market discrimination, especially against obese black women, but these attitudes may be unable to prevent increased risks of poor health, functional limitations, and, ultimately, death (Averett and Korenman 1996).

LIMITATIONS

Although the NHIS is especially well-suited for our analyses, we recognize several limitations. First, as discussed earlier, important considerations of confidentiality precluded us from accessing the geocodes for the respondents of this study, and as a result we were unable to include more detailed information regarding institutional factors of respondents' neighborhoods. Said differently, neighborhood socioeconomic status (poverty rate) is used in these analyses as a proxy for institutionally related risks and resources. High poverty areas are less likely to provide structural resources essential to healthy living, such as health care facilities, recreational facilities, and a built environment that encourages exercise (MacIntyre, MacIver, and Sooman 1993; Sooman, MacIn-tyre, and Anderson 1993). Furthermore, in poor areas, population density per food market is greater, the cost of food is higher, and the quality of available food is lower (Troutt 1993). Accordingly, the inclusion of this information could provide a more complete accounting for sociodemographic differences in health, and this information would improve the efficacy of policies aimed at the amelioration of health disparities across social geographies (Berkman and Kawachi 2000; Williams and Collins 1995).

Second, future research should address other aspects of relatively impoverished communities that should be considered. For example, the relatively high levels of disorder and fear in poor neighborhoods may also affect neighborhood health behaviors (Ross and Mirowsky 2001), including obesity. Environments influence physical activity, and certain

environments may discourage walking and exercise and make travel dangerous (Humpel, Owen, and Leslie 2002). More advantaged areas are likely to be safer and thus encourage walking and promote healthier activities. Additionally, it is plausible that residents in economically disadvantaged areas are unaware of important health care information or rely on resources outside of their neighborhoods for health promoting activities such as exercise and social interaction (MacIntyre and Ellaway 1998). Thus, the inclusion of information regarding exercise and the reasons that adults lead active or sedentary lives should be included in future work to supplement the findings presented in this article.4

Finally, it is important to acknowledge the cross-sectional nature of these data. Although multiple years of data are used, each year draws a new cross section of the U.S. population. As physical information of respondents is only collected at one point in time, it is possible that the relationships described in this study are due to the selection of heavier persons into communities with higher rates of obesity. To examine these processes more thoroughly would require longitudinal information on the residential location of adults over repeated observations as well as health-related information at the neighborhood level over time.

CONCLUSION

Location within social and geographic space is a vital component in the examination of adult obesity. Individuals living in disadvantaged neighborhoods characterized by higher rates of obesity are at a greater risk of obesity. Our findings suggest that obesity research is complex and multifaceted, and that studies that do not incorporate context overlook an important component. Adult obesity levels in the United States are affected through both institutional and epidemic pathways, in which socioeconomic disadvantage and obesity at the neighborhood level affect individual-level obesity. Thus, context must be considered in conjunction with individual risk factors to more fully understand obesity, a major contributor to ill health and shortened life among U.S. adults.

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[4]To maintain large sample sizes, especially for the very small area analysis, we used NHIS core questions, which are asked of each respondent every year. Supplemental questions—including such health behaviors as cigarette smoking, alcohol consumption, diet, and physical activity—are asked in selected years and are generally asked of sample individuals (often of one adult person in the household). Thus, the irregularity of the sample coupled with the small sample sizes prevented us from including such supplemental questions in our neighborhood-level analyses.

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Biography

Jason D. Boardman is assistant professor of sociology and research associate in the Population Program and Health Behavior Program in the Institute of Behavioral Science at the University of Colorado, Boulder. His work examines the social determinants of health among children, adolescents, and adults.

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Richard G. Rogers is professor of sociology and director in the Population Program in the Institute of Behavioral Science at the University of Colorado, Boulder. His research examines the demographic, socioeconomic, health, and behavioral factors that influence the quality and length of life. For example, with Fred Pampel, Rogers recently tested three competing theories of cumulative advantage on health and mortality (see the September 2004 issue of *JHSB*).

Justin T. Denney is a graduate student in the Department of Sociology and the Population Program in the Institute of Behaviorial Science at the University of Colorado, Boulder, and he is a supervisory statistician with the U.S. Bureau of the Census. His research focuses on racial, ethnic, and sex inequities in health and mortality, with an emphasis on the impact of social and residential context.

TABLE 1

Obesity Prevalence Rates by Racial and Socioeconomic Characteristics of Respondents' Neighborhoods

		Racial Composition		Socioeconomic Composition		Poor Areas		Nonpoor Areas	
		< 25%	≥ 25%	< 25%	≥ 25%	< 25%	≥ 25%	< 25%	≥ 25%
Obesity Prevalence (%)	**Total (%)**	**Black**	**Black**	**Poor**	**Poor**	**Black**	**Black**	**Black**	**Black**
0–4.9	12.3	14.3	3.4	13.4	5.7	10.2	1.5	14.6	4.7
5–9.9	19.9	22.3	8.9	21.4	10.7	14.8	7.0	22.6	10.2
10–14.9	22.4	24.1	14.5	23.9	13.5	19.3	8.2	24.5	18.9
15–19.9	17.8	18.1	16.2	18.0	16.4	19.7	13.5	18.0	18.1
20–24.9	12.9	11.5	19.5	12.2	16.8	13.8	19.5	11.8	19.4
> 25.0	14.7	9.8	37.6	11.1	36.9	22.2	50.3	8.5	28.6
Average	14.7	13.3	21.6	13.7	20.9	16.8	24.6	12.9	19.4
Standard deviation	9.2	8.3	10.0	8.5	10.7	9.7	10.1	8.1	9.4
N	7,953	6,544	1,409	6,840	1,113	528	585	6,016	824

Source: 1990–1994 National Health Interview Survey.

Notes: Neighborhood-level data only. Cell entries represent column-specific percentages.

TABLE 2

Descriptive Statistics for all Variables Used in Multivariate Analyses

	Black Respondents	White Respondents
Obese (body mass index ≥ 30)	22.8	13.6
Sociodemographic characteristics		
Age		
18–29	27.5	21.2
30–49	42.8	40.6
50–69	20.7	23.6
≥ 70	8.9	14.6
Sex		
Male	41.3	46.5
Years of education		
< 12	29.2	19.3
12	38.3	37.5
≥ 13	32.5	43.2
Poverty status		
Poor	24.1	9.1
Neighborhood characteristics		
Black area (≥ 25% black)	76.4	5.7
Poor area (≥ 25% poor)	36.4	9.3
Obese area (≥ 25% obese)	31.9	10.4
N	30,891	183,154

Source: 1990–1994 National Health Interview Survey.

Notes: Cell entries represent percentages. All data have been weighted. Bivariate tests of association indicate statistically significant differences in the distribution of all variables between white and black respondents.

J Health Soc Behav. Author manuscript; available in PMC 2011 September 12.

TABLE 3

Odds Ratios: Individual- and Neighborhood-Level Risk Factors Associated with Obesity among Adults

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Sociodemographic characteristics						
Racial identification [non-Hispanic white]						
Non-Hispanic black		1.88***	1.73***	1.59***	1.58***	1.55***
Age [18–29]						
30–49			1.82***	1.82***	1.84***	1.81***
50–69			2.10***	2.10***	2.12***	2.10***
≥70			1.18***	1.18***	1.19***	1.19***
Sex [female]						
Male			.95***	.95***	.95***	.95***
Years of education [≥ 13]						
< 12			1.71***	1.70***	1.68***	1.55***
12			1.35***	1.35***	1.34***	1.27***
Poverty status [not poor]						
Poor			1.36***	1.36***	1.31***	1.30***
Neighborhood characteristics						
Racial composition [< 25% non-Hispanic black]						
≥ 25% non-Hispanic black				1.13***	1.09**	1.01
Socioeconomic composition [< 25% poor]						
≥ 25% poor					1.15**	1.11***
Body mass composition [< 5% obese]						
5–9.9% obese						1.27***
10–14.9% obese						1.34***
15–19.9% obese						1.62***
20–24.9% obese						1.68***
≥ 25.0% obese						1.77***
σ_u^2	.30	.26	.25	.23	.21	.00

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
ρ (intraclass correlation)	.08	.07	.07	.07	.06	.00
−2 log likelihood	178,627.34	177,218.40	173,824.19	173,796.67	173,744.83	173,070.40
Likelihood ratio test		1,408.94***	3,394.21***	27.52***	51.84***	674.43***

* $p < .05$;

** $p < .01$;

*** $p < .001$ (two-tailed tests)

Source: 1990–1994 National Health Interview Survey.

Note: Cell entries represent odds ratios.

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