

**Segregation and Cardiovascular Illness: The Role of Individual and Areal  
Socioeconomic Status**

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Antwan Jones and Franklin Goza\*

Department of Sociology and  
Center for Family and Demographic Research  
Bowling Green State University  
Bowling Green, OH 43403  
Phone: (419) 372 – 2294  
Fax: (419) 372 – 8306  
E-mail: [antwanj@bgsu.edu](mailto:antwanj@bgsu.edu)

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## **Introduction**

Racial residential segregation is a persistent feature across the U.S. Blacks are most highly segregated from whites, followed by Hispanics and Asians (2006; Massey and Denton 1989; Massey and Mullan 1984). In fact, black-white segregation levels appear to be converging to levels experienced in South Africa during apartheid (Clark et al. 1999; Massey 2001; Massey 2004; Massey and Denton 1993). As in South Africa, U.S. segregation is the direct result of institutional racism, designed to protect Whites from social interaction with minorities, particularly blacks (Williams and Collins 1995; Williams and Collins 2001).

Differential exposure of racial groups in segregated environments translates to differential exposure to stressors and differential access to resources. These differences in exposures and stress are directly related to racial differences in health. However, equally compelling, segregation is also a cause of racial differences in socioeconomic status (i.e., education and employment opportunities). This relationship is predicated on the notion that communities are also embedded with socioeconomic statuses that are more than the sum or average of the individual members living in a particular area (Bajari and Kahn 2005; Fisher and Massey 2002).

The purpose of this research is to explore how living in potentially stressful areas (i.e., segregated areas) affects cardiovascular illness. More importantly, this research will also examine the effects of individual and areal socioeconomic status in moderating the effect of segregation on health. Using a large, national dataset, we hope to answer three questions previously unanswered in the literature: First, does level of segregation impact cardiovascular illness? Second, how is areal SES related to an individual's SES in predicting health? Finally, does the effect of SES (individual and areal) differ across levels of segregation?

## **Literature Review**

Social scientists' preoccupation with racial and ethnic segregation derives from the familiar connection between a group's spatial position and its corresponding economic position in society. However, whether location and environment impact economic standing or if economic status dictates area of residence seems to be unanswered in the literature (Massey 2004). Much of the literature on assimilation indicates the relative importance of minorities attaining higher levels of income, which enables residents in segregated areas to move and assimilate into a suburban area (Iceland and Wilkes 2006; Massey and Mullan 1984; South and Crowder 1998). Even though the literature has not fully established the causal link between economic status and residence, what is most evident is the strong association between the two (Apodoca 2006; Blanchard et al. 2004).

Regardless of this relationship, residential segregation has many implications for general health and well-being. The primary mechanism operating in residential segregation is the concentration of poverty (Massey and Eggers 1990). In areas where poverty is concentrated, residents are more likely to be exposed to adverse conditions that impact health and well-being, among them, high rates of teenage pregnancy, school dropouts, and crime and unemployment (Jargowsky and Bane 1991; Jencks and Mayer 1990; Wilson 1987). In areas of high poverty concentration, income greatly impacts healthcare options and the quality of healthcare that can be attained (Bharmal and Thomas III 2005). While segregation is tied to specific outcomes, in the subsequent sections, we discuss how the effects of segregation on health follow throughout the life course.

*Untangling Race, Socioeconomic Status, Segregation and Health*

The link between segregation and health has been longstanding in the literature, first appearing in Yankauer's (1950) work that linked residential segregation of African Americans and infant mortality rates in the 1940s. What is less clear is the mediating effects of socioeconomic status (SES) and race on health. In particular, House and Williams (2000) find that low income Whites have rates of heart disease that are about twice as low as those of high income African Americans. Yet, racial disparities persist at every level of SES. The researchers argue that the economic circumstances of African Americans do not have an enveloping effect that would deter them from having less deleterious health outcomes when acquiring higher SES. Essentially, an intergenerational transmission of health through economic disadvantage is purported that is not corrected for by upward social mobility. Their second argument is that because of discrimination, African Americans are less able than their White counterparts to translate economic status into resources that could enable better health. As such, House and Williams' argument relies heavily on the inextricability of race and SES in determining health (Balibar and Wallerstein 1992).

Recent studies have incorporated various kinds of segregation, races, and health outcomes. What is clear from the literature is that segregation has a longitudinal effect on health for those individuals who live in segregated areas. The next section concentrates on the initial, longstanding impact of segregation on health. There, we focus on the literature on segregation and health outcomes and discuss health without specifically examining illness or morbidity.

### *Segregation and Health at Early Ages*

Research literature posits that segregation effects are cumulative and initially experienced at very early ages. LaVeist (1989; 1993) used the Infant Mortality Rate (IMR) to assert that segregation is directly associated with health. The African American IMR was directly associated with segregation, controlling for poverty level and political power. In addition, increasing levels of segregation correspond to higher IMRs, but this effect was larger and more robust for African Americans. In addition, Bird (1995) found the same to be true in his research, even controlling for state-level structural variables unrelated to political power or poverty. Polednak (1996) also found that segregation was associated with IMR. Using MSA level data, Polednak nuanced the longstanding negative relationship between segregation and health by finding that in thirty-eight MSAs segregation was not related to IMR, while in ninety-two MSAs segregation and health were directly related. Polednak thus found that geographic locale was important in predicting the effect of segregation on health.

Perhaps more insightful is the explanation of why racial disparities exist at birth. A high African American IMR for the most-segregated MSAs persists and contributes to the rising African American-White IMR ratio. In addition, tremendous variation exists in regional trends in African American post neonatal infant mortality rates, suggesting that social and medical-care differences among Blacks are likely to explain this phenomenon. These medical-care differences are manifested in access and quality, a theme constantly argued in social epidemiological research on IMR and race. However, medical-care differences could mask the consequences of living in a segregated environment, such as poor neighborhood quality, extreme poverty, crime and psychosocial stressors (Polednak 1996).

### *Segregation and Health throughout the Life Course*

Segregation and health outcomes have a more tenuous relationship during the adult ages than at any other time. Segregation is uniquely tied to neighborhood social capital (Huie 2001). Quality of schools, housing conditions and safe recreation areas are indicators of neighborhood socioeconomic status. In addition, informal indicators such as the extent of social support and social regulation are associated with neighborhood SES. Thus, unlike the association between segregation and health at early ages (which primarily is the result of individual SES and health infrastructure), the association between segregation and health throughout adulthood is more the result of community socioeconomic status. Indeed, segregation and SES are highly correlated, as segregated areas tend to be low in neighborhood socioeconomic status (Jackson et al. 2000).

Segregated areas also tend to be directly associated with deleterious socioenvironmental conditions and indirectly associated with structural determinants of health such as health care access (Farley and Frey 1994). In addition, the sociological literature has pointed out that segregation is not only associated with health outcomes, but also mortality outcomes. Specifically, segregation and homicide rates are positively related to one another (Potter 1991). This research underscores a link between segregation and health, illustrating that behaviors within the neighborhood are also associated with health outcomes.

For the most part, segregation research has focused more on mortality outcomes than morbidity consequences such as hypertension. In addition, the issue with these studies is that segregation was measured as one construct. Thus, the unit-increase interpretation of the estimates can only capture one level of segregation. To illustrate, LaVeist (1989; 1993) used the index of dissimilarity, which is the percentage of one of the two groups included in the calculation that would have to move to different geographic areas in order to produce a completely even

distribution. Therefore, increasing segregation to its highest value only indicates a high percentage of a racial group living in an area. However, we know that residential segregation can be in the form of de facto segregation or self-segregation (enclaves). Thus, we can more adequately measure segregation when using a number of different indices, which, when simultaneously evaluated, can illustrate the degree to which segregation is occurring. The advantage of using more than one index to measure segregation is presented in the discussion of hypersegregation that follows.

### *Hypersegregation: Understanding Levels of Segregation*

A growing body of research is aimed at explaining how poverty manifests and creates health disparities in environments where certain ethnic and racial minorities have exclusive interactions with members of their own ethnic or racial group. Termed hypersegregation by Massey and Denton (1989), this phenomenon statistically occurs when a racial group scores high (0.60) on four of five indices of segregation: evenness, exposure, centralization, clustering, and concentration (Massey and Denton 1989). All indices have a range from 0.0 to 1.0. *Evenness* refers to the differential distribution of groups across neighborhoods. *Exposure* measures the probability of interaction between groups. *Centralization* indicates the distance to the center of the urban area. *Clustering* indicates the degree to which minorities live in areas that adjoin one another in space. *Concentration* refers to the amount of physical space occupied by the minority group.

In practical terms, however, hypersegregation refers to an accumulation of segregation (Massey and Denton 1989) that consistently isolates a racial group from resources and opportunities that affect social and economic well-being (Logan 1978; Schneider and Slogan 1982). Recent work on hypersegregation found that 29 metropolitan areas can be considered

extremely segregated (Wilkes and Iceland 2004). Moreover, hypersegregated communities are more prone to detrimental ecological processes such as the dumping of toxic waste (Anderton et al. 1994; Davidson and Anderton 2000). As a result, African Americans in hypersegregated communities experience negative health conditions, such as air toxin exposure (Lopez 2002; Pastor Jr. et al. 2004), unhealthy food choices (Dodson et al. 2006) and crime (Charles et al. 2004; Krueger et al. 2004). In addition, Hummer (1995) suggests that hypersegregated areas expose children to overcrowding and unsanitary living conditions, where disease becomes rampant. Living in these conditions will raise the odds of poor health through direct (toxins) and indirect (crime) stresses on the individual. As such, hypersegregation may be linked to mortality, morbidity and health outcomes.

### **Hypotheses**

A review of prior research suggests three hypotheses to be examined herein. First, based on the research that suggests a tie between segregation and health, we hypothesize that areal context is related to hypertension. Specifically, residence in non-segregated areas is associated with much lower odds of being diagnosed with hypertension than is residence in hypersegregated environments. Second, because individual-level socioeconomic status may differ from community-level socioeconomic status, we expect that the latter effects will supersede the former in predicting hypertension and be the stronger predictor of the diagnosis of hypertension.

Finally, because individuals living in segregated and hypersegregated areas may differ in levels of socioeconomic status, we hypothesize that the effect of individual and areal SES differs across levels of segregation. In this case, we expect that socioeconomic status is more protective against hypertension in nonsegregated areas than in segregated areas.



## **Data and Methods**

Two data sources are used in this study, the 2005 Behavioral Risk Factor Surveillance System (BRFSS) and the 2005 American Community Survey (ACS). The BRFSS is a collaborative project of the Centers for Disease Control (CDC) and U.S. states and territories. The BRFSS is an ongoing data collection program designed to measure behavioral risk factors in the adult population (18 years of age or older) living in households. The objective of the BRFSS is to collect uniform, state specific data on preventive health practices and risk behaviors that are linked to chronic diseases, injuries, and preventable infectious diseases in the adult population. Factors assessed by the BRFSS include tobacco use, health care coverage, HIV/AIDS knowledge and prevention, physical activity, and fruit and vegetable consumption. Data are collected from a random sample of adults (one per household) through a telephone survey (Mokdad et al. 2003). The BRFSS is a rich dataset that can adequately test the proposed hypotheses. This dataset also contains geopolitical indicators at both the micro- and meso-level of analysis. On average, missing data accounted for about 7% of each measure. Unless otherwise specified, missing cases were dropped from the analysis, leaving an analytical sample of N=200,102.

In addition to the BRFSS, estimates of community-level data are used. These data come from the 2005 American Community Survey (ACS). The ACS is a monthly household survey developed by the U.S. Census Bureau to provide data users with annual estimates of household, social, and economic characteristics for geographies and populations of at least 65,000 people. In addition, the ACS annually updates their multi-year demographic estimates for geographies down to the block group. The ACS data will be used to supplement the BRFSS with community-level indicators that are associated with health.

*Cardiovascular illness.* We assess cardiovascular illness based on reports of hypertension. Hypertension is measured by a question that asks whether or not respondents have ever been told by a health professional that they have high blood pressure. Approximately 32% of the total sample has been told that they have high blood pressure.

*Individual SES.* We assess individual SES using three standard measures. First, respondent's *education* is divided into four categories: less than high school, high school graduate, some college and graduated college. Second, *income* is constructed to include household total income, excluding income from interest, dividends, and other investments. This measure includes other income for each person such as disability assistance, social security, and public assistance, which are contributed to the household but are not necessarily from earnings. Poisson iterative regression replacement was used to substitute any missing data in the original variables and sample selection tests via maximum likelihood were performed to assess whether a selection effect of income was present in the sample (Miranda and Rabe-Hesketh 2006; Rencher 2002). We found no sample selection bias with regards to hypertension. Income is represented by series of dummy variables represented by the categories: less than \$15,000, \$15,000 to \$20,000, \$20,000 to \$25,000, \$25,000 to \$35,000, \$35,000 to \$50,000, \$50,000 to \$75,000 and more than \$75,000. Third, all respondents were asked to indicate their *employment status*. This dummy variable indicates whether or not the respondents worked full- or part-time during the previous year.

*Individual controls.* Health correlates and demographic measures are used as controls to isolate the effect of individual SES on cardiovascular illness. *Body Mass Index* (BMI) is a continuous measure of body fat based on height and weight that applies to both adult men and women. *Smoker status* is measured with a series of dummy variables that correspond to

respondents being current smokers, former smokers or nonsmokers at survey date. *Alcohol consumption* indicates the number of days per month respondents had at least one alcoholic drink. *Physical activity* assesses the number of days per week respondents perform at least 10 minutes of exercise excluding work-related activities. The last health measure, *insured*, is a dummy variable that indicates whether or not the respondent has any kind of public or private health insurance.

In addition to health correlates, demographic measures are also used as controls. *Race* is captured by a series of dummy variables (White, Black and Other). A probable limitation of the dataset is that the sample is overwhelmingly White (63%), which may create constraints in sample variability within the analyses (Agresti 2002). Among those classified as Other, approximately 87% were Asian.

*Age* at time of survey is measured in complete years. *Gender* is coded as a dummy variable with males as the contrast category. Lastly, *marital status* is coded with a set of dummy variables indicating if the respondent is married, separated, never married or coupled with an unmarried partner.

*Segregation.* Wilkes and Iceland (2004) identified 29 MSAs as hypersegregated areas in their research. Wilkes and Iceland (2004) also found that five of the 29 hypersegregated MSAs scored high on all five indices of segregation, indicating extreme hypersegregation. We use this finding to distinguish four types of segregation: *extreme hypersegregation*, *hypersegregation*, *segregation* and *racial integration*. Extremely hypersegregated MSAs correspond to the 5 MSAs that scored high on all five indices of segregation, as revealed by Wilkes and Iceland's (2004) research. Likewise, hypersegregated MSAs correspond to the MSAs that scored high on four of the five segregation indices, as outlined by the researchers. Segregated MSAs were thus, by

definition, those remaining MSAs that scored high on 1, 2 or 3 of the 5 segregation measures. Lastly, nonsegregated MSAs were those that did not score high on any of the segregation indices.

*Areal SES.* In order to fully exhaust the spatial nature of hypersegregation, a few measures at the MSA level are used to examine the effect of hypersegregation on health. These measures are linked to the MSA the respondent resided in during 2005. The first is *population size*, which is logged. The *proportion of residents who are below the poverty line* is also used as a control measure. The *proportion of African Americans* in the MSA is used to control for racial concentration. Lastly, the *proportion of female headed households*, a measure of concentrated poverty determined for each MSA, is employed. These covariates are derived from the 2005 American Community Survey estimates and are merged onto the person-level BRFSS data file.

### *Analytic Strategy*

Multilevel statistical models are used to estimate the parameters of interest since the structure of the data is two-level (individual and MSA) and hierarchical. These models allow us to estimate the within-group and between-group variations and examine individual-level and MSA-level effects (Raudenbush and Bryk 2002; Wong and Mason 1991). Because hypertension is employed as a binary dependent variable, we used the PROC NLMIXED procedure in SAS 9.1 statistical software. In these analyses, the level-1, or individual-level model, can be written as

$$Y_{ij} = \beta_{0j} + \sum_k \beta_k X_{(ij)k} + \varepsilon_{ij}$$

where  $Y_{ij}$  is whether or not the individual respondent  $i$  in area  $j$  has been diagnosed with hypertension.  $\beta_{0j}$  is the intercept,  $\beta_k$  is the coefficient of the variable  $k$  and  $X_{(ij)k}$  is the value of the variable  $k$ .  $\varepsilon_{ij}$  is the error term associated with individual (or fixed effects) variance.

The level-2, or MSA-level model, can be written as

$$\beta_{0j} = q_{00} + \sum_{z=1}^7 q_{0z} W_{0z} + u_{0j}$$

where  $q_{00}$  is the random effects intercept,  $q_{0z}$  is the neighborhood-level coefficient of one of the 7 MSA-specific variables used,  $W_{0z}$  corresponds to the value of the variable  $z$  and  $u_{0j}$  is the error term associated with the MSA (or random effects) variance.

## Results

### *Is an individual's SES related to cardiovascular illness?*

Before exploring the differential effects between individual and areal socioeconomic status, we must first assess whether an individual's SES is associated with diagnoses with hypertension. Table 2 presents the results of the multilevel analyses of hypertension diagnosis for the entire sample. Model 1 includes all measures of individual SES, and the model estimates the variance without including control variables. In Model 1, we find that education is highly associated with hypertension. Having higher levels of education is protective against being diagnosed with hypertension. This association is also present in both income and employment status. However, for income, a positive relationship at the two lowest levels of income emerged. Compared to individuals who make less than \$15,000, individuals who earn between \$15,000 and \$20,000 have significantly higher odds of being diagnosed with hypertension. This result appears to illustrate that the "working poor", i.e. individuals who simultaneously fall short of being below the poverty line and do not qualify for Medicare benefits are more health disadvantaged than persons who fall below the poverty line (less than \$15,000).

Even though the threshold difference between working poor and individuals below the poverty line is compelling, the effect diminishes in Model 2. Model 2 adds individual-level controls (i.e., health correlates and demographic controls). While education and employment

status follows the same pattern as seen in Model 1, the effect sizes of income (i.e., the parameter estimates) decrease. Thus, net of health correlates and demographic controls, income diminishes in effect in predicting hypertension diagnosis.

Health correlates as control variables have independent effects on hypertension diagnosis in the expected way. Increasing levels of BMI (body mass index) and alcohol consumption are positively associated with hypertension. Not smoking at the survey date, and engaging in physical activity are associated with not being diagnosed with hypertension. Having insurance is associated with being medically diagnosed with hypertension. Demographic controls also have independent effects on hypertension. Age, gender and marital status are all positively associated with hypertension in the expected ways. That is, those older, male or separated are more likely to be diagnosed with hypertension. To summarize, an individual's SES is related to hypertension and this relationship remains when controlling for health correlates and demographic profiles.

*Net of individual-level characteristics, does level of segregation impact heart health?*

The effects of segregation are added in Model 3 of Table 2. An auxiliary zero-order model containing only the four segregation measures revealed all measures to be positive, significant and increasing monotonically, as expected. Model 3, which includes the individual measures, is extremely similar. Net of individual SES, health correlates and demographic controls (which in this model changed slightly but retained their significance), residence in a segregated, hypersegregated, or extremely hypersegregated area is associated with a higher risk of being diagnosed with hypertension, relative to living in a nonsegregated area.

In addition, the level 1 error (which corresponds to the model variance explained by the individual-level error) is significant. This indicates that there is significant variation in hypertension that is unaccounted for by individual level SES and individual controls. Thus, in

Models 3 and 4 we add level 2 (i.e., MSA-specific) variables to account for the variance left unexplained.

However, when comparing the variances for the intercepts in Model 2 (individual-level model) with Model 3 (individual-level and segregation model), we note a numerically small difference (0.02 to 0.01), but a powerful result. This difference indicates that about 50% of the variance explainable by MSA is accounted for by after controlling for what type of segregated area individuals live. In addition, the significant variance in Model 3 indicates a significant difference in hypertension diagnoses across MSAs. Thus, net of individual-level characteristics, segregation impacts hypertension diagnosis. Not only does living in a segregated or hypersegregated environment seem to be predictive of hypertension, segregated and hypersegregated environments show significant variation in who is diagnosed with hypertension. The socioeconomic conditions of a given MSA may help to explain why this variation would exist. We examine this possibility in the next section.

*How is areal SES related to an individual's SES in predicting cardiovascular health?*

Model 4 adds measures of areal SES to explore how different kinds of SES affect hypertension. In a zero order model with only the four areal SES measures, we found that all measures except the proportion of African Americans living in the MSA were significant (results not shown). In this model, which controls for individual variables and segregation measures, only the proportion of MSA residents in poverty and the proportion of female-headed households (the two more proximate measures of areal poverty) remain significant. In both cases, these variables, net of all controls, are positively related to hypertension diagnosis. Indeed, the more people in poverty and the more female-headed households in an area, the greater the chance of being diagnosed with hypertension.

Adding the areal SES measures attenuates the effect that living in an extremely hypersegregated area has on hypertension. The proportion of female-headed households and the proportion in poverty most contribute to explaining away extreme hypersegregation. However, residence in a segregated or hypersegregated area is still significant in predicting hypertension.

Lastly, most of the individual levels of socioeconomic status retained their significance from Model 3 to 4, which indicates that both areal and individual SES impact hypertension. The only category to become insignificant is high school graduate. In the previous models, being a high school graduate is associated with a lower likelihood of being diagnosed with hypertension relative to having less than a high school education. However, in the final model, which controls for the socioeconomic status of area, the likelihood of having hypertension is the same for high school graduates and those with less than a high school education. Thus, areal SES has independent effects in predicting hypertension *and* can help explain some of the relationships between individual SES and health.

*Does the effect of SES (individual and areal) differ across levels of segregation?*

Table 3 presents the multilevel analyses of hypertension diagnosis for each area type. Each model presented is a full model, with all measures controlled for simultaneously. Because the segregation variables were significant in the final model of Table 2, we would expect to find areal-specific differences in the effects that individual and areal SES measures have on being diagnosed with hypertension.

In nonsegregated environments, the individual-level SES effects are similar to those of the combined sample. That is, being highly educated (i.e., having at least some college), having more than \$20,000 in annual income, or being employed at least part-time are all protectors against hypertension. In addition, at the lowest levels of education (high school graduation or



less) and income (less than \$20,000), the likelihood of being diagnosed with hypertension is similar. The nonsegregated areal SES effects indicate that having more people and having more African Americans are protective against hypertension diagnosis, while having more people in poverty is predictive of being diagnosed with hypertension. In analyses not shown we found that 80.9% of residents living in nonsegregated areas were also in rural areas. In that case, increasing population and the percentage of minorities in rural, nonsegregated areas would be tied to increasing development and access to health care, factors which would lower hypertension propensity in these areas (Cort and Fahs 2001).

In segregated areas, individual and areal SES measures are not strong in predicting hypertension. For individual SES, only high levels of education (i.e., graduating college), high levels of income (i.e., more than \$75,000), or working at least part-time seem to be protective against hypertension. Thus, in these locations, the highly educated, the employed, and the wealthy to benefit the most (see Jackson et al. 2000). Regarding areal SES measures, only the proportion of individuals in poverty is predictive of hypertension in segregated areas, as increased poverty in these areas is associated with a higher likelihood of being diagnosed with hypertension.

In hypersegregated areas (i.e., areas where minority residents may not encounter members outside of their own race) the effects of individual-level SES are also rather sparse. Regarding education, only those with at least some college are less likely to be diagnosed with hypertension than are those with less than a high school education. Also, those who are employed enjoy a significantly reduced likelihood of being diagnosed with hypertension. However, no association exists between income and cardiovascular illness in hypersegregated areas. Thus, somewhat unexpectedly, income does not appear to be a factor in developing

hypertension in these hypersegregated areas. Instead, what is more important in these locales is whether people work and the extent to which they are well-educated.

The areal SES measures reveal that the proportion of African Americans and the proportion of female-headed households in hypersegregated areas are powerful predictors of hypertension. Increasing numbers of African Americans in these areas correspond to a decreased risk of being diagnosed with hypertension, while increasing numbers of female-headed households increase the risk of diagnosis. At least in this areal context, the presence of non-Black members may heighten stress levels due to a perceived threat that African Americans may have to compete with limited resources or marriageable partners (Demo and Hughes 1990; Lichter et al. 1992). This threat creates anxiety (Pearlin et al. 1981), which can increase the likelihood of developing hypertension. Thus, having more same-race members join an area work as a buffer to protect against hypertension. Likewise, an increase the number of female-headed households in a hypersegregated area may also correspond to a lack of resources and increased stress among residents.

In extremely hypersegregated areas, neither education nor employment matter in predicting hypertension. In fact, only the \$35,000-\$50,000 income category resulted in lower odds of being diagnosed with hypertension. Thus, traditional measures of individual SES do not seem to be protective against hypertension (net of individual-level health correlates and demographic controls) in such highly segregated areas. Areal measures of SES also seem to be poor predictors in these locations. We suggest that socioeconomic well-being can only be protective in environments where health and social infrastructures exist to ensure the physical well-being of residents. In hypersegregated communities, structural conditions such as the lack of healthy food choices (Dodson et al. 2006; Jetter and Cassady 2006), quality medical

professionals and hospitals (Goldberg et al. 2006), noise- and pollution-free zones (Anderton et al. 1994; Davidson and Anderton 2000), and (perceived) safe areas for walking and/or exercise (Bird 1995; Clarke et al. 1994) create an environment where all residents, regardless of education, income or occupation, are at a health disadvantage.

### **Discussion and Conclusion**

This study examined the role of individual and areal socioeconomic status in predicting hypertension, with particular emphasis on level of segregation. We found mixed support for our three hypotheses. Segregation levels were significant in predicting hypertension diagnosis. More specifically, controlling for individual- and MSA-level predictors, we found that living in either a segregated or hypersegregated area was associated with greater likelihood of being diagnosed with hypertension.

We also determined that the effects of individual-level socioeconomic status may differ from one's community-level socioeconomic status. However, we did not find evidence that areal SES explained away the effects of individual-level SES. While education, income and employment status were significantly and negatively related to hypertension, we could not attenuate these effects when adding areal SES measures of population size, percent in poverty, percent African American and percent of female headed households. Only at low levels of education (i.e., a high school graduate or less) and at low levels of income (less than \$20,000) did the areal SES measures attenuate. Thus, individual-level predictors seem to be robust in explaining whether or not an individual is diagnosed with hypertension.

For our final hypothesis, we found that although individual-level SES matters in predicting hypertension, SES only matters in certain environments. Stratifying the sample, we found that in segregated and hypersegregated environments, those individuals who are most

educated and who are high earners seem to be protected against hypertension. In extremely hypersegregated areas, areas where there is little to no interaction with non-Blacks, SES does not seem to have any protective benefit. We suggested that in these areas, structural conditions are probably more revealing as to whether an individual was diagnosed with hypertension.

Four limitations to this research must be acknowledged. The first is sample representation. The sample size for the BRFSS was large, perhaps enabling small differences to become statistically significant ones. The reader is warned that the same results may not be guaranteed if using a smaller sample size. In addition, the sample was collected via telephone interviews, which indicates some class bias regarding sample participants. While we found no sample selection effects with regard to income, by virtue of sampling method, severely impoverished persons may not be adequately represented in this sample. Coupled with a potential class bias is the relatively small representation of African Americans in this large sample. With less than 9 percent in the analytic sample, the sample may be skewed to more affluent African Americans, a demographic that is rather small in representation in the U.S. Future studies may want to improve on ways to estimate those individuals who are least represented in the BRFSS.

The second limitation of this research is the cross-sectional design of the survey. Disentangling causal ordering relationships proves to be nearly impossible because all information is gathered at one specific time. We have modeled associations with certain processes (e.g., SES) and health. However, we cannot determine, for instance, if SES directly impacts health, if SES is impacted by health, or if SES and health are both impacted by each other or another factor. Thus, a longitudinal take on hypertension would illuminate how direct associations (or causal ordering of events) impact health.

The third limitation to this study is that MSA level indicators had to be appended onto individual-level data with a different dataset. Thus, we had to rely on the assumption that MSA characteristics are semistable across time (or in this case, 1 year) which may not be true for all MSAs. Also, MSAs were proxies for neighborhood characteristics but the geographical difference was too great to be substantiated. MSAs are vastly different from neighborhoods and often spatially encompass many neighborhoods. Future research will need to specifically look at community or neighborhood indicators at the time of data collection to better analyze the role of meso-level factors on individual-level outcomes.

The fourth limitation to this study is that health status is self-reported and not based on medical examinations or records. The reporting of health depends on whether patients choose to consult their general practitioner and is based on their own decisions. The self-reporting of health on surveys is conceptually problematic for two reasons. First, bias could be embedded in the study. If this research uses the assumption that people accurately report their morbidities based on a doctor's evaluation, then the assumption fails to acknowledge those people who are unable to seek medical advice. Second, self-reported health status could be self-diagnosed, which could lead to error in misdiagnosis. Misdiagnosis through self-reports could result in significant variability in self-reported health, which problematizes the validity of the measurement.

Despite the aforementioned limitations, this research is not without its strengths. The use of the most recent data with a more sophisticated and more accurate methodology to document a historical trend with individual- and meso-level measures is the most appealing aspect of this research. Again, the lack of focus on hypersegregation, a phenomenon conceptually different than segregation, allows for a new wave of research that will look at the effects of areal context (instead of "segregation levels", i.e., dissimilarity index or percent minority presence) on health.

Lastly, the notion that an individual's SES and corresponding socioeconomic context could be driving health disparities because of their linkages could pave the way for a new line of inquiry that more directly examines the ties between individual, community, and society on health.

Future research should indeed address the limitations of this study. In addition, future research should examine the intersections of race, segregation, socioeconomic well-being and health. Specifically, can racial differences in cardiovascular illness be explained by other variables that link socioeconomic position, areal context and unique psychosocial stressors related to being a minority in the US? Additional future research, much like this project, should couch these health outcomes as conditions of certain health behaviors, which should also be explored. For instance, socioeconomic and areal differentials may exist in condom use or testing, which could influence the HIV/AIDS racial divide. In addition, socioeconomic and areal differences in sugar intake or regulation of sugar could be the driving force behind a racial divide in death due to diabetes. Future research that couches this research in spatial demography would provide a clearly mapped picture of the social facts that operate independently of the individual, yet are a part of an individual's life, namely the constant impact of segregation and hypersegregation.

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**TABLE 1. Means and Standard Deviations for Individual- and MSA-Level Variables**

	Range	Mean / %	SD
<i>Cardiovascular Illness</i>			
Hypertension Diagnosis	0 (no) - 1 (yes)	30.92%	--
<i>Individual SES</i>			
Educational Level			
Less than High School <sup>†</sup>	0 (no) - 1 (yes)	9.93%	--
High School Graduate	0 (no) - 1 (yes)	30.09%	--
Some College	0 (no) - 1 (yes)	27.02%	--
Graduated College	0 (no) - 1 (yes)	32.97%	--
Household Income			
Less than \$15,000 <sup>†</sup>	0 (no) - 1 (yes)	9.59%	--
\$15,000 to \$20,000	0 (no) - 1 (yes)	7.94%	--
\$20,000 to \$25,000	0 (no) - 1 (yes)	9.65%	--
\$25,000 to \$35,000	0 (no) - 1 (yes)	12.73%	--
\$35,000 to \$50,000	0 (no) - 1 (yes)	17.46%	--
\$50,000 to \$75,000	0 (no) - 1 (yes)	18.80%	--
More than \$75,000	0 (no) - 1 (yes)	23.84%	--
Employed	0 (no) - 1 (yes)	88.04%	--
<i>Individual Controls</i>			
Body Mass Index	8.38 - 42.11	27.24	5.68
Smoker Status			
Current Smoker <sup>†</sup>	0 (no) - 1 (yes)	19.59%	--
Former Smoker	0 (no) - 1 (yes)	28.08%	--
Never Smoked	0 (no) - 1 (yes)	52.33%	--
Alcohol Consumption (Days Per Month)	0 - 30	10.38	5.30
Physical Activity (Days Per Week)	0 - 7	4.40	2.37
Insured	0 (no) - 1 (yes)	88.04%	--
Race			
White	0 (no) - 1 (yes)	84.13%	--
Black <sup>†</sup>	0 (no) - 1 (yes)	8.66%	--
Other	0 (no) - 1 (yes)	7.21%	--
Age	18 - 99	51.17	17.06
Male	0 (no) - 1 (yes)	38.24%	--
Marital Status			
Never Married	0 (no) - 1 (yes)	13.55%	--
Cohabiting	0 (no) - 1 (yes)	2.62%	--
Married <sup>†</sup>	0 (no) - 1 (yes)	55.75%	--
Separated	0 (no) - 1 (yes)	28.08%	--
<i>Segregation Measures</i>			
Nonsegregated <sup>†</sup>	0 (no) - 1 (yes)	78.96%	--
Segregated	0 (no) - 1 (yes)	12.24%	--
Hypersegregated	0 (no) - 1 (yes)	6.89%	--
Extremely Hypersegregated	0 (no) - 1 (yes)	1.91%	--
<i>Areal SES</i>			
Total MSA Population	65,076 - 5,193,448	848,169.80	941,084.20
Proportion in Poverty	0.02 - 0.36	0.10	0.03
Proportion African American	0.00 - 0.62	0.10	0.11
Proportion Female-Headed Households	0.05 - 0.26	0.13	0.03
N		200,102	

Source: 2005 BRFSS and 2005 ACS.

Notes: Categories may not sum to 100 due to rounding. Standard deviations of binary variables are excluded.

<sup>†</sup> used in the analyses as contrast variable.

**TABLE 2. Multilevel Analyses of Hypertension Diagnosis, Combined Sample (N=200,102)**

	Model 1	Model 2	Model 3	Model 4
<i>Fixed Effects</i>				
Intercept	-0.03 ***	-5.60 ***	-5.59 ***	-5.67 ***
Educational Level (Less than High School)				
High School Graduate	-0.08 ***	-0.04 *	-0.05 ***	-0.03
Some College	-0.21 ***	-0.11 ***	-0.10 ***	-0.09 ***
Graduated College	-0.36 ***	-0.25 ***	-0.25 ***	-0.23 ***
Household Income (Less than \$15,000)				
\$15,000 to \$20,000	0.05 ***	0.03	0.03	0.03
\$20,000 to \$25,000	-0.04 ***	-0.06 ***	-0.06 ***	-0.05 ***
\$25,000 to \$35,000	-0.05 ***	-0.07 ***	-0.07 ***	-0.07 ***
\$35,000 to \$50,000	-0.12 ***	-0.07 ***	-0.07 ***	-0.06 ***
\$50,000 to \$75,000	-0.22 ***	-0.10 ***	-0.10 ***	-0.09 ***
More than \$75,000	-0.32 ***	-0.14 ***	-0.14 ***	-0.13 ***
Employed	-0.88 ***	-0.19 ***	-0.19 ***	-0.19 ***
Body Mass Index				
		0.10 ***	0.10 ***	0.10 ***
Smoker Status (Current Smoker)				
Former Smoker		-0.05 *	-0.05 *	-0.04 *
Never Smoked		-0.16 ***	-0.16 ***	-0.16 ***
Alcohol Consumption				
		0.00 ***	0.00 ***	0.00 ***
Physical Activity				
		-0.03 ***	-0.03 ***	-0.03 ***
Insured				
		0.18 ***	0.18 ***	0.19 ***
Race (Black)				
White		-0.57 ***	-0.57 ***	-0.51 ***
Other		-0.56 ***	-0.57 ***	-0.50 ***
Age				
		0.05 ***	0.05 ***	0.05 ***
Male				
		0.12 ***	0.12 ***	0.12 ***
Marital Status (Married)				
Never Married		-0.02	-0.02	-0.01
Cohabiting		-0.08	-0.08	-0.06
Separated		0.06 ***	0.06 ***	0.06 ***
Segregation Measures (Nonsegregated)				
Segregated			0.01 *	0.01 *
Hypersegregated			0.01 ***	0.08 ***
Extremely Hypersegregated			0.02 ***	0.00
Log Total MSA Population				
				-0.01
Proportion in Poverty				
				0.66 ***
Proportion African American				
				0.09
Proportion Female-Headed Households				
				1.32 ***
<i>Random Effects</i>				
	Variance Components			
Intercept	0.04 *	0.02 *	0.01 *	0.01 *
Level 1 error	0.45 ***	0.41 ***	0.41 ***	0.40 ***
AIC	2,135	1,983	1,976	1,970
Pseudo R <sup>2</sup> (level 1)	0.06	0.28	0.29	0.34

Source: 2005 BRFSS and 2005 ACS.

Notes: Contrast categories are in parentheses. AIC = Akaike's information criterion.

\* p < 0.05; \*\* p < 0.01; \*\*\* p < 0.001.

**TABLE 3. Multilevel Analyses of Hypertension Diagnosis, Stratified Sample**

	Nonsegregated	Segregated	Hypersegregated	Extremely Hypersegregated
<b>Fixed Effects</b>				
Intercept	-5.67 ***	-5.84 ***	-4.87 ***	-10.66 ***
<i>Individual SES</i>				
Educational Level (Less than High School)				
High School Graduate	-0.02	0.01	-0.11	-0.03
Some College	-0.08 ***	-0.04	-0.21 **	-0.15
Graduated College	-0.23 ***	-0.16 ***	-0.33 ***	-0.22
Household Income (Less than \$15,000)				
\$15,000 to \$20,000	0.03	0.05	-0.03	-0.29
\$20,000 to \$25,000	-0.05 *	-0.08	-0.07	-0.17
\$25,000 to \$35,000	-0.05 **	-0.05	-0.20	-0.41
\$35,000 to \$50,000	-0.06 ***	-0.04	-0.06	-0.19 ***
\$50,000 to \$75,000	-0.09 ***	-0.10	0.01	-0.35
More than \$75,000	-0.13 ***	-0.11 *	-0.10	-0.29
Employed	-0.19 ***	-0.19 ***	-0.24 ***	-0.14
<i>Areal SES</i>				
Log Total MSA Population	-0.01 *	-0.02	-0.01	0.44
Proportion in Poverty	0.67 ***	0.01 *	1.51	<sup>a</sup>
Proportion African American	-0.53 ***	1.74	-5.59 ***	-7.95
Proportion Female-Headed Households	1.35	2.01	1.83 **	<sup>a</sup>
<b>Random Effects</b>				
	Variance Components			
Intercept	0.01 *	0.03 *	0.00	0.00 *
Level 1 error	0.10 ***	0.55 *	0.35 **	0.00
Pseudo R <sup>2</sup> (level 1)	0.37	0.29	0.31	0.32
N	184,274	44,373	12,784	3,044

Source : 2005 BRFSS and 2005 ACS.

Notes : Contrast categories are in parentheses. AIC = Akaike's information criterion. All models control for individual health and demographic measures seen in Model 4 in Table 2.

<sup>a</sup> Collinearity issues within the subsample did not allow for stable parameter estimates.

\* p < 0.05; \*\* p < 0.01; \*\*\* p < 0.001.