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DISSERTATION

The Place We Live, the Health We Have

A Multi-Level, Life Course Perspective on the Effects of Residential Segregation and Neighborhood Poverty on Health and Racial Health Disparities

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This document was submitted as a dissertation in September 2006 in partial fulfillment of the requirements of the doctoral degree in public policy analysis at the Pardee RAND Graduate School. The faculty committee that supervised and approved the dissertation consisted of Brian K. Finch (Chair), Richard J. Buddin, and Nicole Lurie.

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[*] For more information on the nonprofit program, please visit www.westlapal.org.

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Chapter 1: Introduction

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Background

Quite contrary to Margret Thatcher's individual-centric view that "…there is no such thing as society. There are [only] individual men and women…", sociologists and more recently, public health investigators contend that societal forces not only do exist but nontrivially impact our lives by shaping our experiences, social interactions, and opportunities. And although our choices and behaviors are inherently expressed at the individual-level, they are often influenced and constrained by the "macro" social and economic context to which we are exposed. These social and economic contexts are significantly defined by the place where we live. Consequently, place can play an influential role in shaping our culture, our lifestyle, our behavior, and our aspirations in life.

As maintained by Fitzpatrick and LaGory (2000, 2003), "place matters". Among other socioeconomic outcomes, place matters for education, for employment, and for marriage prospects (Wilson 1987; Cutler & Glaser 1997). And as supported by a growing body of literature, place also matters for health (Yen & Syme 1999; Kawachi & Berman 2003; Pickett & Pearl 2001; Robert 1999a). The availability of goods and services, and exposure to hazards and opportunities are increasingly distributed spatially, underscoring the growing connection between place and health.

Evidence that the social and structural environment influences life-chances, and ultimately health outcomes, suggests that health policy, traditionally targeted at the individual-level with little regard to neighborhood context, should consider underlying constraints or opportunities present in the residential environment in designing and implementing the most effective and efficient health care strategies. In addition, the connection between place and health suggests that health policy should be broadened to include housing and urban design so that agencies such as the EPA, HUD, and the Department of Transportation deliberately consider possible health impacts, as well as environmental and economic impacts, when designing and developing the urban environment.

However, while the possible policy implications are far-reaching, the current evidence of a causal link between neighborhood context and health is tenuous. Although ample evidence confirms that living in an economically disadvantaged neighborhood is associated with

adverse health outcomes, the reliance on cross-sectional data and inadequate attention to problems of self-selection make causal inferences problematic.

As such, this dissertation seeks to contribute to a better understanding of the connection between place and health through a series of examinations that target specific weaknesses in the neighborhood-health literature. I begin with a rather broad perspective and investigate the role of racial and economic residential segregation, by creating spatial divisions between the advantaged and the disadvantaged, in perpetuating health disparities between Blacks and Whites. I then narrow my focus to the neighborhood level and investigate whether accounting for the duration of exposure to disadvantaged environments can help explain racial health disparities and health outcomes. Lastly, I use longitudinal data and employ statistical strategies that attempt to recover causal estimates of the effect of neighborhood disadvantage on health.

Objectives

The specific objectives of the dissertation include:

1. To investigate the relationship between metropolitan-level segregation measures and individual-level health outcomes, net of individual-level adjustments.
    a. Are Blacks differentially impacted by segregation compared to Whites?
    b. Do racial and economic segregation differentially impact health?
    c. Do different dimensions of segregation differentially affect health?
2. To distinguish between transient versus persistent exposure to individual and neighborhood poverty in estimating individual and neighborhood poverty effects on health and racial health disparities.
    a. Does the inclusion of duration of poverty, either at the individual or neighborhood level, further explain the Black/White health gap that remains after accounting for single-point-in-time SES measures?
    b. Do multi-point-in-time measures of neighborhood context reveal a stronger connection between neighborhood poverty and health compared to single-point-in-time measures?
3. To estimate the causal impact of neighborhood disadvantage on health.
    a. Are estimates of neighborhood effects larger when mediating factors (e.g., employment status) are allowed to play through?

b. Are there significant neighborhood effects after accounting for unobserved
c. individual-level heterogeneity?

Organization of the Dissertation

The organization of the dissertation is as follows. In Chapter 2, I describe the datasets that I use in the dissertation. In Chapter 3, I investigate the relationship between racial and economic segregation and health through a series of multilevel models. In Chapter 4, I examine the importance of accounting for the temporal dimension of both individual and neighborhood conditions in explaining current health status and racial health disparities. In Chapter 5, I use longitudinal data and employ fixed-effect and propensity-score models to address major sources of bias that are present in cross-sectional neighborhood-health estimates. In Chapter 6, I conclude with a summary of my findings and discussion of policy implications.

Contribution to Knowledge

This dissertation makes a number of contributions to the neighborhood-health literature. First, a multilevel model is used to examine individual-level associations between different dimensions of racial and economic segregation on individual health and racial health disparity. The approach improves on earlier segregation health studies that relied on aggregate data. Second, this dissertation extends the conventional cross-sectional neighborhood-health models that relied on single-point-in-time measures of neighborhood context by accounting for the cumulative effect of an individual's neighborhood environment. Because Blacks and Whites tend to have different rates of neighborhood stability, the addition of this temporal dimension provides insights into the nature of observed racial health disparities. Third, this dissertation employs modeling strategies that represent critical steps towards recovering causal estimates of neighborhood effects on health. These estimates will better equip health policymakers to judge the extent and magnitude of neighborhood impacts on health, thereby aiding in designing appropriate strategies to improve population health and eliminate health disparities.

Chapter 2: Data

Chapter 2: Data

Introduction

The analyses in this dissertation are based on four sets of data: the Current Population Survey, the Panel Study of Income Dynamics, the Neighborhood Change Database, and the Census racial segregation data. I describe each dataset in detail below.

Current Population Survey

I use the 2000 Current Population Survey (CPS) in Chapter Three, for analyses investigating the relationship between metropolitan-level racial residential segregation and individual-level health outcomes. The CPS, conducted by the Bureau of the Census for the Bureau of Labor Statistics, is a monthly survey that collects employment and earnings information from approximately 50,000 households. The sample is representative of the US civilian non-institutionalized population. Analyses for this study are based on the Annual Demographic Survey, or March CPS supplement, which includes individual-level socioeconomic and demographic information, metropolitan area of residence, and respondent-rated health status.

Panel Survey of Income Dynamics

I use The Panel Study of Income Dynamics (PSID) in Chapters Four and Five. Begun in 1968, the PSID is a longitudinal study of a representative sample of the non-immigrant U.S. population with an over-sample of low-income families. From 1968 to 1997, the PSID interviewed individuals from families in the original sample every year, whether or not they were residing in the same location; in 1997, the PSID began administrating their surveys biannually. When individuals moved out and started their own families, the PSID followed them into their new environments, adding these newly formed families to the sample. Consequently, what started out to be 4,800 families in 1968 has grown to more than 7,000 families in 2003. The PSID has a current sample size of approximately 65,000 individuals, with information spanning as much as 36 years of their lives.

Although mostly known for its economic and demographic content, the PSID also contains health related questions that have been considerably expanded during the late 1990s. Available health data include information on current general health status,

retrospective health status as youths, specific chronic conditions, health behaviors, and health insurance status. My sample is restricted to non-Hispanic Black and White individuals throughout.[1] However, various models rely on different sample subsets of the PSID, as appropriate. Consequently, detailed descriptive tables are deferred to the empirical chapters where I will present relevant sample statistics for each individual analysis.

Neighborhood Change Database

Throughout my analyses, census tracts serve as proxies for neighborhoods. Although there are alternative methods to capture neighborhood geographic boundaries (e.g., via residents' perception), the usage of census tracts to proxy for neighborhoods has been widely used in the neighborhood literature and is one of the few feasible strategies when using national data. In addition, census tracts boundaries respect major roads and rivers and are originally demarcated to capture a homogenous population. Census tracts have an average population size of approximately 4,000 residents.

For measures of neighborhood context, I rely on Geolytics' Neighborhood Change Database (NCDB). The NCDB contains decennial census long form data for years 1970, 1980, 1990, and 2000 with over 1,000 variables for each decade. These variables include details on household composition, housing characteristics, income, poverty status, education level, and employment. All of these variables are available only at the census tract level.

A unique feature of the NCDB is that it offers tract level information for all four decades normalized to 2000 tract boundaries. Because tract boundaries change across decennial censuses, variations in tract demographics or housing conditions due to boundary adjustments may be spuriously attributed to compositional changes. Consequently, normalization of tract boundaries is a critical control to ensure that neighborhood trends are estimated accurately. I estimate neighborhood conditions between decennial census years by linearly interpolating across decennial tract measures.[2]

[1] Because of low sample size, individuals from the Hispanic and Immigrant samples that were subsequently added to the PSID during the 1990s were not included.

[2] Using decennial tract measurements as support points, I assume a constant rate of change between each decennial census and linearly interpolate between each pair of decennial census (e.g., 1980/1990 & 1990/2000).

Metropolitan Segregation Measures

For racial segregation measures, I use the metropolitan-level racial segregation indices that were calculated by the U.S. Census. Using 2000 Census tract level data, the US Census calculated racial segregation data for each (primary) metropolitan statistical area in the United States.[3] These racial segregation measures include those that require spatial data (e.g., distance from the central city to individual tracts) to calculate and thus offer data on various segregation indices that have not been widely used in the segregation-health literature.

Economic segregation measures, also based on 2000 Census data, are derived from the NCDB tract-level data to produce MSA level economic segregation indices.

Data Linkage

In Chapter 3, I use individual-level CPS data and metropolitan-level segregation data to investigate the association between segregation and health. Metropolitan racial and segregation measures are linked to CPS respondents via MSA code identifiers that are available in both the Census segregation and CPS datasets.

In Chapters 4 and 5, I use PSID data to investigate the association between neighborhood context and individual health. To link neighborhood context to each individual across time, I rely on 2000 census tract identifiers (available from the PSID only through special contract) that have been linked to each family for each year. Neighborhood-level variables, derived from the NCDB, are merged to the PSID data via census 2000 tract identifiers.

[3] The Census racial segregation data can be accessed at http://www.census.gov/hhes/www/housing/housing_patterns/housing_patterns.html (accessed March 2006).

Chapter 3: Residential Segregation and Health

Chapter 3: Residential Segregation and Health

Introduction

In this chapter, I investigate the link between racial and economic residential segregation, measured at the metropolitan-level, and individual health. I focus on whether the relationship between segregation and health differs across race and economic status. I use multilevel models that adjust for individual-level characteristics and explore the independent effects of different dimensions of segregation.

Background

Investigations into the determinants of racial health disparities have traditionally focused on differences in socioeconomic status to account for the consistent health disadvantage experienced by racial/ethnic minorities, particularly Black Americans, in the US. Although differences in the distribution of socioeconomic indictors, including education, labor force participation, income, and marital status across racial/ethnic groups have been shown to be strong and robust predictors of health outcomes (Adler et al. 1993), these differences have generally not been able to fully account for health disparities between Blacks and Whites (Williams & Collins 1995, 2001). Moreover, in spite of general declines in rates of morbidity and mortality over the past century, Black/White disparities in health have remained fairly stable over time (Williams & Collins 2001). The persistence of this unexplained gap has led many to direct their attention towards differences in residential living conditions as a contributor to health disparities between racial/ethnic groups (House & Williams 2000).

Segregation is one particular aspect of the social and structural environment that has received increasing attention, (Acevedo-Garcia & Lochner 2003; Acevedo-Garcia et al. 2003; Schulz et al. 2002), with Williams and Collins (2001) arguing that racial residential segregation is a fundamental cause of racial disparities in health. As opposed to more proximal causes such as individual health behaviors, segregation may be considered as a fundamental cause of disease because it "embodies access to important resources, [and] affects multiple disease outcomes through multiple mechanisms" (Link & Phelan 1995). There is consistent empirical evidence, grounded in sociological theory, that racial residential segregation undermines the socioeconomic attainment of Blacks. Socio-economic status, in turn, has been shown to be a robust predictor of health outcomes. Additionally, in imposing spatial divisions between the advantaged and the disadvantaged, segregation creates a social

and physical environment in which hazards, risks, and the availability of goods and services become differentially distributed spatially (Fitzpatrick & LaGory 2003). These disadvantaged segregated communities tend to suffer from deteriorating physical environments and public infrastructure, high levels of personal and property crime, and isolation that depress the overall quality of life for its residents. Not surprisingly, the pernicious effects of segregation are disproportionately borne by poor minorities who comprise the vast majority of the segregated population. The racial differences in exposures and opportunities that are being reinforced by segregation restrict racial minorities from access to quality education and employment opportunities --- effectively ensuring a cycle of poverty across generations (Massey & Denton 1993; Wilson 1987).

Racial segregation and Its Impact on Neighborhood Quality for Blacks

An overly simplified perspective of residential segregation may conclude that racial segregation is an inevitable consequence of the disparate distribution of income across racial groups. Although the variation in socioeconomic status between Blacks and Whites explain some of the differences in spatial patterns, it cannot account for the extreme degree of racial segregation experienced by Black minorities; Blacks across all socioeconomic levels are highly segregated (Wilkes & Iceland 2004; Denton & Massey 1988; Massey & Fischer 1999; South & Crowder 1998; Rosenbaum & Friedman 2001). Empirical studies investigating the relationship between residential segregation, race, and socioeconomic status find that Blacks, contrasted to other minority groups such as Asians and to a lesser extent Hispanics, are least likely to be able to translate socioeconomic success into residential mobility to more affluent neighborhoods (Alba & Logan 1993; Rosenbaum & Friedman 2001). As middle class Blacks attempt to distance themselves from high poverty areas by moving to the outskirts of core ghetto areas, racial dynamics (e.g., White out-migration) and institutional disinvestments operate together to diminish property values, ultimately resulting in deteriorating neighborhood conditions (Pattillo-McCoy 2000; Quillian 1999). As a result, middle class Blacks are as segregated as poor Blacks. Thus race, not the differences in the socioeconomic distribution between Blacks and Whites, is the driving factor of residential segregation (Alba & Logan 1993; South and Crowder 1997; Rosenbaum & Friedman 2001).

One readily visible economic consequence of racial segregation, manifested at the place level, is the depression of neighborhood quality for Blacks. Massey & Denton (1993)

postulate that, for a given poverty rate at the metropolitan area level, racial segregation disproportionately exposes Blacks to concentrated poverty. Racial segregation, argue Massey & Fischer (2000), interacts with structural shifts (e.g., rising income inequality, falling income, increasing socioeconomic stratification) in society to spatially isolate the poor. However, the exposure to poverty concentration falls disproportionately on groups that experience high levels of racial segregation. Consequently, most poor Blacks live in areas of concentrated poverty while most poor Whites reside in nonpoor neighborhoods (Wilson 1987, Jargowsky 1997). The spatial patterns of racial segregation that disproportionately expose Blacks to distressed social and economic environments has led Sampson & Wilson (1995 - as cited in Williams & Collins 2001) to conclude that "the worst urban context in which Whites reside is considerably better than the average context of Black communities".

Hypersegregation of Blacks

As conceived by Massey and Denton (1989), segregation consists of five distinct dimensions: evenness, exposure, clustering, centralization, and concentration. The dimension of evenness reflects the degree to which minority members are evenly distributed across a city area; exposure is the degree of potential contact between minority and majority groups; clustering is the extent to which minority areas border one another; centralization is the degree to which minority members reside in and around the center of an urban area; and concentration is the relative amount of physical space occupied by a minority group. The multidimensional property of segregation reflects the multiple ways in which groups can be sorted and separated. Segregation along several dimensions simultaneously (hypersegregation) reflects more severe separation than segregation along a single dimension.

As such, the pervasiveness and depth of racial segregation experienced by Blacks in the US becomes only more apparent when one considers that hypersegregation is a persistent and distinctive Black experience (Massey & Denton 1989; Wilkes & Iceland 2004). Despite a trend in the decline of racial segregation since 1980 (Iceland et al. 2002), Blacks are still hypersegregated in 29 metropolitan areas (Wilkes & Iceland 2004). Considering that eighty-five percent of Blacks live in metropolitan areas (Iceland et al. 2002), residential segregation along racial lines may play a pivotal role in shaping Black/White disparities.

Empirical Evidence of Racial Segregation and Health

Despite the proliferation of studies investigating neighborhood effects on health during the last decade (Yen & Syme 1999; Kawachi & Berkman 2003), the number of empirical studies on the impact of residential segregation on health and health disparity remain modest. A recent review of the sociological and social epidemiology literature identified only twenty-nine relevant studies (Acevedo-Garcia et al. 2003).

Investigations into racial segregation and health have generally found a positive relationship between Black mortality rates and racial residential segregation (LaVeist 2003; Jackson et al. 2000; Leclere et al. 1997; Acevedo-Garcia et al. 2003) across U.S. cities and metropolitan areas. There is more mixed evidence on the direction or extent to which racial segregation affects the health of Whites. While some studies have found either no or advantageous associations between Black segregation and health outcomes among White individuals (e.g., Guest et al. 1998; LaVeist 1989; Polednak 1996), others have found detrimental associations (Collins 1999; Collins & Williams 1999).

An important limitation to all of these studies is that their analyses were based on aggregate-level data, relying on only a few macro-level socioeconomic measures, (e.g., proportion poor, proportion unemployed in an MSA), in an indirect attempt to adjust for compositional differences across areas. Consequently, findings of health variations across metropolitan type may be an artifact of inadequate adjustment of differences in individuals across cities. In addition, though aggregate analyses have enriched our understanding of the possible impacts of segregation on health, inferences from their findings must be tempered with the knowledge that they are susceptible to ecological fallacies (Robinson 1950) in which the relationship between segregation and metropolitan-level health outcomes may not be in the same direction as segregation and individual-level health. Relying on aggregate data may lead to improper inferences to the relationship between segregation and individual health.

To date, only two studies (i.e., Ellen 2000; Subramanian et al. 2005) that examined the impact of racial segregation on health have adjusted for individual-level socioeconomic factors. Ellen (2000) used the 1990 national birth and death files, linked to mother's metropolitan area of residence at the time of birth, to investigate racial segregation on infant mortality. She found moderate evidence that the segregation, as captured by the dimension of centralization, is detrimental to both Black and White infants, net of a set of socioeconomic controls. There was less convincing support for the deleterious effects of

racial evenness on infant health. Subramanian's et al. (2005) study took the initial step towards employing a multilevel modeling strategy to investigate health disparities due to variations between metropolitan area segregation levels. The study employed a two-level hierarchal model to examine whether two dimensions of Black/White racial residential segregation, evenness and interaction, are associated with health after adjusting for key individual-level socioeconomic and demographic factors. In contrast to results from aggregate level analyses, no significant effect was found to link racial evenness to health outcomes. Higher levels of racial isolation, in contrast, were found to be negatively associated with health for Blacks (OR=1.05, CI [1.00-1.12]). No association between racial isolation and health was found for Whites.

Empirical Evidence of Economic Segregation and Health

In spite of the widely held position that residential segregation undermines the outcomes of Blacks predominantly through the adverse affects of concentrated poverty (Williams 1996; Acevedo-Garcia 2000), only two studies have examined the relationship between economic residential segregation and health outcomes (Lobmayer & Wilkinson 2002; Waitzman & Smith 1998). Both of these studies found increased mortality rates in more economically segregated urban areas. Lobmayer & Wilkinson (2002) used two measures of income segregation: the ratio of between tract inequality to within tract inequality and Jargowsky's neighborhood sorting index (NSI)[4]. Separate analyses using these two measures yielded similar results, showing a significant, positive relationship between economic segregation and mortality rates. The strongest associations for both males and females were for infant mortality and those aged over 15 years at the time of death. Waitzman and Smith (1998) used the pooled 1986-1994 cross-sectional samples of the National Health Interview Surveys (NHIS), matched to death data from the National Death Index (NDI) from 1986 to 1995 to model the association between segregation and all-cause mortality. They investigated the impact of economic segregation using four measures: concentration index, dissimilarity index, isolation index, and the NSI. Results across all indices were generally similar and suggest that economic segregation is associated with elevated mortality risk. Their findings

[4] The formula for the Jargowsky Index is the standard deviation of the mean incomes of neighborhoods divided by the standard deviation of individual household income in the entire MSA (Jargowsky 1996). Unlike other economic segregation measures, the Jargowsky Index is independent of the mean and variance of income within the area of interest.

were robust to the inclusion of individual-level factors, including income. Although Waitzman and Smith (1998) adjusted for race, they did not investigate whether the association between economic segregation and risk of mortality varied between Blacks and Whites.

Dimensions of Segregation and Health

As aforementioned, residential segregation is a multidimensional construct that consists of several distinct spatial patterns. Massey and Denton (1988) determined the existence of five dimensions of segregation that capture different aspects of residential sorting: evenness, interaction, concentration, centralization, and clustering. Each dimension of segregation captures conceptually distinct patterns of racial sorting within metropolitan areas that may, in turn, have varying degrees of significance for health outcomes.

The dimension of evenness reflects the differential spatial distribution of different groups within a metropolitan area. Segregation is low when different groups are distributed equally across neighborhoods and higher otherwise. Among the five segregation dimensions, evenness is the least associated empirically with indicators of neighborhood deprivation (Denton 1994 --- as cited in Subramanian et al. 2005). However, despite the unclear conceptual justification to link the degree of spatial evenness to health, the vast majority of studies have focused solely on the dimension of evenness, most commonly measured by the dissimilarity index, to establish an association between segregation and health.

Interaction, the second most commonly investigated segregation dimension in the health literature, reflects the potential level of contact between individuals from minority and majority groups and is influenced by the degree to which the two groups share common residential neighborhoods. Minority members may experience little exposure to majority members even if there is minimal segregation on the evenness dimension if minority members comprise a large proportion of the metropolitan area. Conversely, minority members may have high levels of exposure to majority members in areas of low evenness if they comprise a very small proportion of the metropolitan area. As such, indices measuring exposure take into account the relative size of minority and majority groups.

The dimension of interaction is most commonly operationalized via the Black isolation index. This reflects the degree of isolation experienced by minority group members. Residential isolation of Blacks has been hypothesized to affect the health of Blacks through

its concentration of disadvantage into Black neighborhoods (Subramanian 2005) and its influence on transmission patterns for infectious diseases (Acevedo-Garcia 2000; Acevedo-Garcia 2001). However, while exposure and evenness theoretically capture two distinct spatial patterns, the indices tend to be highly correlated empirically (Massey & Denton 1988).[5] Consequently, although the link between the dimension of exposure and health may rest on a stronger theoretical argument, it is unclear, given its high correlation to the dimension of evenness, whether modeling strategies utilizing the isolation index would result in substantially different results from those using the dissimilarity index. Moreover, given the conceptually distinct spatial patterns between these two dimensions of segregation, making proper inferences without first disentangling the large empirical overlap is difficult.

The remaining segregation dimensions: concentration, centralization, and clustering have been largely neglected in the health literature (c.f., Ellen 2000). This omission may be due, in part, to the difficulty of obtaining and manipulating the spatial data that is necessary to calculate these dimensions. Nonetheless, their theoretical relevance to health outcomes strongly warrants such investigations. As a consequence of discrimination, equitable access to residential neighborhoods has been denied to Blacks, restricting their choices of neighborhoods to those that are often less desirable and often divergent from their socioeconomic attainment (Alba et al. 1994; Logan & Alba). That is, Blacks tend to live in neighborhoods that have higher crime rates and lower quality of housing than do comparable Whites. These restrictions may lead to the concentration of Blacks into more densely populated, mostly poor neighborhoods. Moreover, these neighborhoods tend to be located nearer to the central city where the residential environments are often more polluted, violent, and unsanitary (Fitzpatrick & LaGory 2000); this geographic pattern of segregation would be more directly captured by the centralization index, which measures the degree to which Blacks reside in the central cities. In addition, given that the Black ghetto environment which has been shown to be detrimental to socioeconomic progression (Wilson 1987) tends to develop when these neighborhoods are adjoined to one another, investigating the degree of clustering among Black neighborhoods within a metropolitan area may provide more insightful information. To the extent that concentrated poverty is hypothesized to be the main mediator to which segregation detrimentally impacts the health of Blacks and that the

[5] Massey & Denton (1988) calculated a correlation of 0.77 (weighted by the minority population) between the dissimilarity and isolation indices.

geographic patterns of racial concentration, centralization, and clustering mirror the geographic patterns of disadvantaged residential environments, examinations of these dimensions may provide a stronger empirical link between segregation and health than the previously explored dimensions of evenness and exposure.

Objectives

As previously mentioned, the overwhelming majority of previous segregation studies, relying exclusively on aggregate-level data, suffer from the well-recognized shortcomings of ecological analyses. Although Ellen's (2000) and Subramanian's et al. (2005) analyses represent a substantial methodological improvement over aggregate studies, there is still little empirical evidence supporting the hypothesis that higher levels of segregation are detrimental to the health of Blacks, net of individual-level socioeconomic and demographic factors. The dearth of support is due not to contrary evidence suggesting a null effect, but to the lack of studies that have utilized multilevel data to investigate these theoretical relationships. Consequently, the empirical evidence supporting the deleterious role of residential segregation on health is far from conclusive. Moreover, scant attention has been directed towards examining whether the dimensions of segregation other than evenness and interaction are associated with health.

Hence, through a series of hierarchal models, I investigate the association between residential segregation and health. There are several objectives, motivated by gaps in the segregation health literature. First, I extend Subramanian's et al. (2005) multilevel study that investigated the impacts of the segregation dimensions of evenness and isolation on health, and explore the link between segregation and health using segregation indices that reflect the dimensions of concentration, clustering, and centralization. Second, I examine the association between metropolitan-level economic segregation on individual health. Third, I test whether these associations differ between Blacks and Whites, as a whole and by economic status.

Data

The segregation-health analyses are based on data drawn from several sources: the 2000 March Supplement of the Current Population Study (CPS), the Geolytics National Change Database (NCD), and the Census MSA-level segregation files.

The CPS, conducted by the Bureau of the Census for the Bureau of Labor Statistics, is a monthly survey that collects employment and earnings information from approximately 50,000 households. The sample is representative of the US civilian non-institutionalized population. Analyses for this study are based on the Annual Demographic Survey, or March CPS supplement, which includes individual-level socioeconomic and demographic information, metropolitan area of residence, and respondent-rated health status.

Racial segregation measures are obtained directly from files prepared by the US Census Bureau that are publicly available on the Bureau's website.[6] These files include metropolitan racial segregation measures for all five dimensions of segregation. Economic segregation indices are computed using the Geolytics National Change Database's nation-wide tract-level data. Since indices for the dimensions of clustering and centralization require spatial data (e.g., distance between tracts and the central city) that are not available in the database, only economic segregation measures for the dimensions of evenness, interaction, and concentration are calculated. The segregation measures from both the US Census and NCD are merged onto the CPS data by matching them to respondents' metropolitan area of residence. MSA boundaries are based on 1999 MSA/PMSA definitions. Racial and economic segregation indices are based on 2000 US Census tract-level distributions.

The sample used in the analyses is composed of non-Hispanic Black and non-Hispanic White adults aged 18+ who reside in metropolitan areas with a total population of over 100,000. Because segregation indices for metropolitan areas with small minority populations are less reliable than those with larger ones, the sample is further restricted to metropolitan areas that have a Black population totaling over 5000.[7] The resulting sample consists of approximately 51,000 respondents.

Health Outcome Measure

A five scale (poor to excellent) respondent-rated health, dichotomized to fair/poor health, serves as the health outcome.[8] Although not an objective measure, respondent-rated

[6] The data can be accessed at http://www.census.gov/hhes/www/housing/housing_patterns/housing_patterns.html. (Accessed on March 17, 2006).

[7] The smaller the minority proportion, the more likely that perceived segregation patterns may be due to random fluctuations rather than social and structural forces (Cortese et al 1976; Massey & Denton 1988), making intercity comparisons misleading.

[8] For brevity, fair/poor health is henceforth referred to as poor health.

health has been shown to be a strong predictor of mortality, net of adjustments for clinical measures of health status (Benyamini and Idler, 1999; Idler and Benyamini, 1997). Moreover, current evidence suggests that the relationship between mortality and respondent-rated health are consistent for Blacks and Whites (McGee et al. 1999).

Segregation Indices

For racial segregation, all five distinct dimensions of segregation: evenness, interaction, concentration, clustering, and centralization are investigated. As aforementioned, since calculations for segregation indices for the dimensions of clustering and centralization require spatial data that are not readily available, investigations of economic segregation are restricted to the dimensions of evenness, interaction, and concentration. Racial segregation measures the spatial sorting between Blacks and Whites and economic segregation measures the spatial sorting between the poor and nonpoor.[9]

Each segregation measure used in the analyses, as defined by Massey and Denton (1988), are as follows[10]:

---

[9] A poor individual is defined as one whose family income falls below the poverty threshold. A nonpoor individual is defined as one whose family income is at or above the poverty threshold.

[10] Formula's and definition terms were borrowed from
http://www.census.gov/hhes/www/housing/housing_patterns/app_b.html
(Accessed on March 17, 2006)
The definition of each term used in the formulas is:
n = the number of areas (census tracts) in the metropolitan area, ranked smallest to largest by land area
m = the number of areas (census tracts) in the metropolitan area, ranked by increasing distance from the Central Business District (m = n)
x_i = the minority population of area i
y_i = the majority population of area i
y_j = the majority population of area j
t_i = the total population of area i
X = the total population of area j
Y = the sum of all x_i (the total minority population)
T = the sum of all y_i (the total majority population)
T = the sum of all t_i (the total population)
p_i = the ratio of x_i to t_i (proportion of area i's population that is minority)
P = the ratio of X to T (proportion of the metropolitan area's population that is minority)
a_i = the land area of area i
A = the sum of all a_i (the total land area)
n1 = rank of area where the sum of all t_i from area 1 (smallest in size) up to area n1 is equal to X
T_1 = sum of all t_i in area 1 up to area n1
n2 = rank of area where the sum of all t_i from area n (largest in size) down to area n2 is equal to X
T_2 = the sum of all t_i in area n2 up to area n
d_{ij} = the distance between area i and area j centroids, where $d_{ii} = (0.6a_i)^{0.5}$
c_{ij} = the exponential transform of $-d_{ij}$ [= $\exp(-d_{ij})$]

Dissimilarity Index: $$\frac{\sum_{i=1}^{n}(t_i|p_i - P|)}{2TP(1-P)}$$ EQ 3.1

The dissimilarity index (D-Index) ranges from 0.0 (complete integration) to 1.0 (complete segregation). A dissimilarity index of 0.6, for example, indicates that 60 percent of the minority population must relocate to achieve an even distribution of minority population across neighborhoods. A value above 0.6 is considered to be high.

Isolation Index: $$\sum_{i=1}^{n}\left[\left(\frac{x_i}{X}\right)\left(\frac{x_i}{t_i}\right)\right]$$ EQ 3.2

The isolation index (P-Index) ranges from 0.0 (maximum contact) to 1.0 (complete isolation). An isolation index of 0.6, for example, indicates that there is a 60 percent probability that a randomly drawn minority member resides in the same neighborhood as another minority member.

Delta Index: $$0.5\sum_{i=1}^{n}\left|\frac{x_i}{X} - \frac{a_i}{A}\right|$$ EQ 3.3

The delta index (Del) ranges from 0.0 (uniform density) to 1.0 (complete unevenness). Higher levels of segregation on this dimension reflect a higher degree of sorting of minority members into a relatively small portion of the total metropolitan area. The higher the index, the smaller the relative area accorded to members of the minority group. Analogous to the interpretation of the dissimilarity index, a delta index of 0.6 indicates that 60 percent of minority members must relocate to achieve a uniform density of minority members across neighborhoods.

Absolute Clustering Index: $$\frac{\sum_{i=1}^{n}\left(\frac{x_i}{X}\sum_{j=1}^{n}c_{ij}x_j\right) - \left(\frac{X}{n^2}\sum_{i=1}^{n}\sum_{j=1}^{n}c_{ij}\right)}{\sum_{i=1}^{n}\left(\frac{x_i}{X}\sum_{j=1}^{n}c_{ij}t_j\right) - \left(\frac{X}{n^2}\sum_{i=1}^{n}\sum_{j=1}^{n}c_{ij}\right)}$$ EQ 3.4

The absolute clustering index (ACL) ranges from 0.0 to 1.0 and reflects the proportion of the population in nearby neighborhoods that belong to the minority group. A high level

on the clustering dimension implies that the spatial pattern of minority residency form areas of adjoining minority neighborhoods, creating racial or economic enclaves. An absolute clustering index of 0.6, for example, indicates that, on average, minority members represent 60 percent of the population in nearby neighborhoods.

Absolute Centralization Index: $\sum_{i=1}^{m}(X_{i-1}A_i)-\sum_{i=1}^{m}(X_iA_{i-1})$ EQ 3.5

The dimension of centralization reflects the degree to which minority members reside near the center of a metropolitan area. Higher values on the index indicate that minority members tend to reside more in the urban center while lower values on the index indicate that minority members tend to reside in the outlying areas. The absolute centralization index (ACE) ranges from –1.0 (maximum tendency of minority members living in outlying areas) to 1.0 (maximum tendency of minority members residing in the central city). The absolute centralization index only takes into account the distribution of the minority group relative to the city's center. A value of 0.6, for example, indicates that 60 percent of members of the minority group must change neighborhoods to achieve a uniform distribution around the central city. A value of 0.0 indicates that the minority group has a uniform distribution throughout the metropolitan area.

For the purposes of calculating segregation indices, "minority" refers to non-Hispanic Blacks for racial segregation measures and to individuals whose family income fall below the poverty threshold for economic segregation measures. Accordingly, "majority" refers to non-Hispanic Whites and to individuals whose family income is above the poverty threshold for racial and economic segregation measures, respectively.

Individual-Level and Metropolitan-Level Adjustments

Individual-level socioeconomic and demographic factors that are included in the model to adjust for confounding are age (continuous), gender, race (Black or White), marital status (single, married, separated/divorced, or widowed), educational attainment (no high school, high school, college, four-year college, or graduate school), labor force status (employed, unemployed, not in the labor force), and ratio of family income to poverty threshold (categorical and demarcated at 100%, 125%, and 150% of the poverty threshold).

Table 3.1 Individual-level Socio-Economic and Demographic Characteristics

	Full Sample		Black		White	
	N	%	N	%	N	%
Sample Size	51,316	100.00	7,437	14.49	43,879	85.51
Black	7,437	14.49				
Poor Health	6,976	13.59	1,499	20.16	5,477	12.48
Female	27,206	53.02	4,291	57.70	22,915	52.22
Mean Age	46.32		43.91		46.73	
Marital Status						
Married	29,345	57.18	2,773	37.29	26,572	60.56
Single	11,708	22.82	2,715	36.51	8,993	20.49
Separated/Divorced	6,457	12.58	1,291	17.36	5,166	11.77
Widowed	3,806	7.42	658	8.85	3,148	7.17
Education Level						
No High School Degree	6,386	12.44	1,618	21.76	4,768	10.87
High School Degree	26,994	52.60	4,162	55.96	22,832	52.03
Some College	3,831	7.47	466	6.27	3,365	7.67
Four-Year College	9,336	18.19	821	11.04	8,515	19.41
Graduate School	4,769	9.29	370	4.98	4,399	10.03
Family Income						
Family Income Below Poverty Level	4,010	7.81	1,359	18.27	2,651	6.04
100 to 125% Poverty Level	1,617	3.15	394	5.30	1,223	2.79
125 to 150% Poverty Level	1,822	3.55	443	5.96	1,379	3.14
Above 150% Poverty Level	43,867	85.48	5,241	70.47	38,626	88.03
Labor Force Status						
Employed	33,725	5.99	4,543	61.38	29,182	66.78
Not Employed	1,149	2.25	338	4.57	811	1.86
Not In the Labor Force	16,229	31.76	2,521	34.06	13,708	31.37

Table 3.1 provides descriptive statistics of the sample as a whole and by race. Age is measured as a continuous value while all other individual-level factors are categorical. Unconditional comparisons of respondent-rated health between Blacks and Whites show that Blacks are much more likely to rate their health as fair or poor compared to Whites (20% vs 12%). Reflecting their socioeconomic disadvantaged status, Blacks are more likely than Whites to be poor (18% vs 6%), unemployed, (5% vs 2%), and less educated (22% vs 11% with no high school education). In addition, a higher proportion of Blacks in the sample are single (37% vs 20%), female (58% vs 52%), and young (average age of 44 vs 47 years).

At the metropolitan-level, total population (including non-Whites and non-Blacks) and proportion poor are included to adjust for metropolitan size and economic level. Metropolitan-level statistics, as described in Table 3.2, show that on average, Blacks tend to live in more highly populated areas than Whites (average city population of 3.3M versus 2.4M) and in cities that are characterized by higher levels of racial segregation, as measured by the isolation and clustering indices. There is no difference in the metropolitan-level poverty rates between Blacks and Whites.

Table 3.2 Metropolitan-level Variables Statistics

	Total Sample			Black	White
	Mean	Min	Max	Mean	Mean
Total Population (1K)	2546	112	9519	3323	2414
Percent Poor	11.14	0.45	23.91	11.88	11.01
Racial Segregation Indices					
Dissimilarity Index	0.62	0.30	0.85	0.65	0.61
Isolation Index	0.49	0.04	0.83	0.61	0.47
Delta Index	0.81	0.40	0.97	0.80	0.81
Absolute Centralization Index	0.73	-0.33	0.96	0.73	0.73
Absolute Clustering Index	0.30	0.00	0.71	0.39	0.28
Economic Segregation Indices					
Dissimilarity Index	0.34	0.17	0.51	0.37	0.36
Isolation Index	0.20	0.08	0.36	0.21	0.19
Delta Index	0.72	0.42	0.92	0.72	0.72

Analytical Strategy

In their study, Subramanian et al. (2005) specified two-level models with individuals at level one and metropolitan characteristics at level two. However, considering that multiple individuals reside within the same household and therefore may not be statistically independent observations, not accounting for the clustering at the household level may underestimate standard errors and lead to inappropriate inferences.

The likelihood of a strong correlation in responses between individuals within households is further increased because the CPS interview procedure relies on a single household member to answer all questions, including those that pertain to other members of

the household. Although this survey strategy may capitalize on the willingness of a single respondent within a household and be time and cost efficient, it also introduces a design effect that accentuates the correlation of health responses within households that may violate the modeling assumption of independence across observations. A two-level hierarchal analysis with households at the second level reveals an intraclass correlation of 0.631 for respondent-rated health. The unusually high correlation of health levels within household may reflect common attributes at the household level (e.g., health behaviors, genetics); however, a more probable source is respondent bias. Compared to more objective measures of health, respondent-rated health may be more sensitive to respondents' personal perception and internal benchmark, making assessments of health within households more consistent than health assessments across households. For example, Elliott et al. (2006) estimated intra-class correlations within families for a variety of health outcomes, including respondent-rated health, using the pooled 1989-1994 NHIS dataset. Intraclass correlations for respondent-rated health, body mass index, and number of doctor's visit in the last 12 months were estimated to be 0.521, 0.207, and 0.198, respectively. The intraclass correlation for respondent-rated health is twice as high when compared to the ICCs for more objective health measures. This suggests that utilizing a subjective criterion as a health outcome require statistical adjustments to account for the correlation of health conditions within family members due to intra-family context and particularly due to respondent bias that artificially reduces the variation of health status within households. Hence, to appropriately address this issue, all analyses account for the clustering at the household level as well as the MSA level.

As such, a series of three-level (individuals nested within households nested within MSAs) random intercept logit models are conducted to investigate the relationship between each dimension of segregation and respondent-rated health. Each segregation dimension is modeled separately, with two model specifications. For the racial segregation models, the first specification allows for a cross-level interaction between the MSA-level segregation index and race (Models 1A-5A) to allow the relationship between racial segregation and health to vary across race. To test whether segregation is more detrimentally associated with health for poor Blacks, the second specification accounts for the economic level of Blacks. That is, poor Blacks and nonpoor Blacks are considered separately in Models 1B-5B.

The strategy for the economic segregation models follows a similar sequence. For each dimension, the first model specification examines whether there are differential effects of economic segregation on health between Blacks and Whites (Models 6A-8A) via a cross-level interaction between race and the MSA-level segregation index. In the second specification, each race category is divided between poor and nonpoor and interacted with the MSA-level segregation index. This allows the effect of economic segregation to vary across socioeconomic status for Blacks as well as Whites.

Results

Table 3.3 presents results from the racial segregation models (Models 1A-5A and Models 1B-5B). All segregation estimates are net of a set of socioeconomic and demographic factors including gender, age, marital status, education, family income/poverty threshold level, employment status, metropolitan population size, and metropolitan poverty rate.

Consistent with the results of Subramanian's et al. (2005) study, no significant relationship between metropolitan Black/White dissimilarity index and respondent-rated health for either Blacks or Whites was found. However, in contrast to their results indicating a significant detrimental association between the Black isolation index and health for Blacks (but not for Whites), results from Model 2A show no statistically significant association. Further examination reveals that the deviating results are due to the addition of labor force status to the set of individual-level controls.[11] Hence, the association between the isolation index and health is rather tenuous, being sensitive to the inclusiveness of individual-level confounders in the model specification.

Review of results for the less studied racial segregation dimensions of concentration, centralization, and clustering, indicate that each of these dimensions has a stronger

[11] As a base of comparison, the same model specification as Subramanian's et al. (2005) was conducted yielding identical results presented in their paper. With the addition of individual-level labor force status, the association between Black isolation index and respondent-rated health was no longer significant. However, it should be noted that socioeconomic status, including labor force status, may be considered as mediating mechanisms through which segregation impacts health. Adjusting for these factors may underestimate the relationship between segregation and health.

Table 3.3 Results of Racial Segregation Models for Poor Respondent-Rated Health[±]

Evenness		Exposure		Concentration		Centralization		Clustering	
(Model 1A)		(Model 2A)		(Model 3A)		(Model 4A)		(Model 5A)	
	OR		OR		OR		OR		OR
Black	1.19	Black	1.30	Black	0.43	Black	0.90	Black	1.50***
Dissimilarity Index	0.98	Isolation Index	1.01	Delta Index	0.89***	Absolute Centralization Index	0.99	Absolute Clustering Index	0.99
Black* Dissimilarity Index	1.08	Black* Isolation Index	1.07*	Black* Delta Index	1.21**	Black* Absolute Centralization Index	1.12**	Black* Absolute Clustering Index	1.08**
(Model 1B)		(Model 2B)		(Model 3B)		(Model 4B)		(Model 5B)	
	OR		OR		OR		OR		OR
Black	1.20	Black	1.30	Black	0.43	Black	0.91	Black	1.50***
Dissimilarity Index	0.98	Isolation Index	1.01	Delta Index	0.89***	Absolute Centralization Index	0.99	Absolute Clustering Index	0.99
Poor Black* Dissimilarity Index	1.09	Poor Black* Isolation Index	1.08*	Poor Black* Delta Index	1.22**	Poor Black * Absolute Centralization Index	1.13**	Poor Black * Absolute Clustering Index	1.10**
NonPoor Black* Dissimilarity Index	1.08	NonPoor Black* Isolation Index	1.07*	NonPoor Black* Delta Index	1.21**	NonPoor Black* Absolute Centralization Index	1.11**	NonPoor Black* Absolute Clustering Index	1.08**

[±] ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level,
*Statistically Significant at the 10% Level

Coefficients for the dissimilarity, isolation, delta and absolute clustering indices are based on a 10 percentage point change.
Coefficients for the absolute centralization index are based on a 10 point change.
All models adjust for gender, age, marital status, education, family income/poverty threshold level, employment status, metropolitan population size, and metropolitan-level poverty rate.

association with health than the dimensions of evenness and exposure. Higher levels of Black concentration, as measured by the delta index, are advantageous for the health of Whites, with each ten percentage point increase on the index is associated with an eleven percent decrease in the odds of reporting poor health. In contrast, there is a differential and detrimental association between higher Black concentration and health for Blacks; each 10 percentage-point increase in the delta index is associated with a twenty one percent increase in the odds of poor health.[12] Neither the dimensions of centralization nor clustering had a statistically significant association for the health of Whites. However, higher segregation levels in each of these dimensions are related to statistically significant poorer health for Blacks. A ten point increase in the absolute centralization index is associated with a twelve percent increase in the odds of reporting poor health for Blacks. Similarly, a ten percentage point increase in the absolute clustering index is associated with an eight percentage point increase in the odds of reporting poor health. Results from Models 1B-5B show that the point estimates for poor Blacks are minimally larger than those for nonpoor Blacks. However, these differences are not statistically significant, suggesting that Blacks across all socioeconomic levels are equally harmed by racial segregation.

Figures 3.1-3.4 plot the predicted probability of reporting poor health as a function of metropolitan racial segregation levels for the delta index, absolute centralization index, and absolute clustering index. The predicted probabilities are based on results from Models 3A-5A and are calculated at the means of the Black sample population. These plots show that the marginal change in probability of reporting poor health is relatively constant, suggesting a linear relationship between segregation levels and health. Racial segregation along the dimensions of concentration, centralization, and clustering are detrimental for Blacks and either neutral of beneficial for Whites.

[12] The net effect of racial concentration for Blacks, however, is statistically insignificant.

Figure 3.1 Predicted Probability of Reporting Poor Health as a Function of Racial Segregation Level, as Measured by the Black/White Delta Index

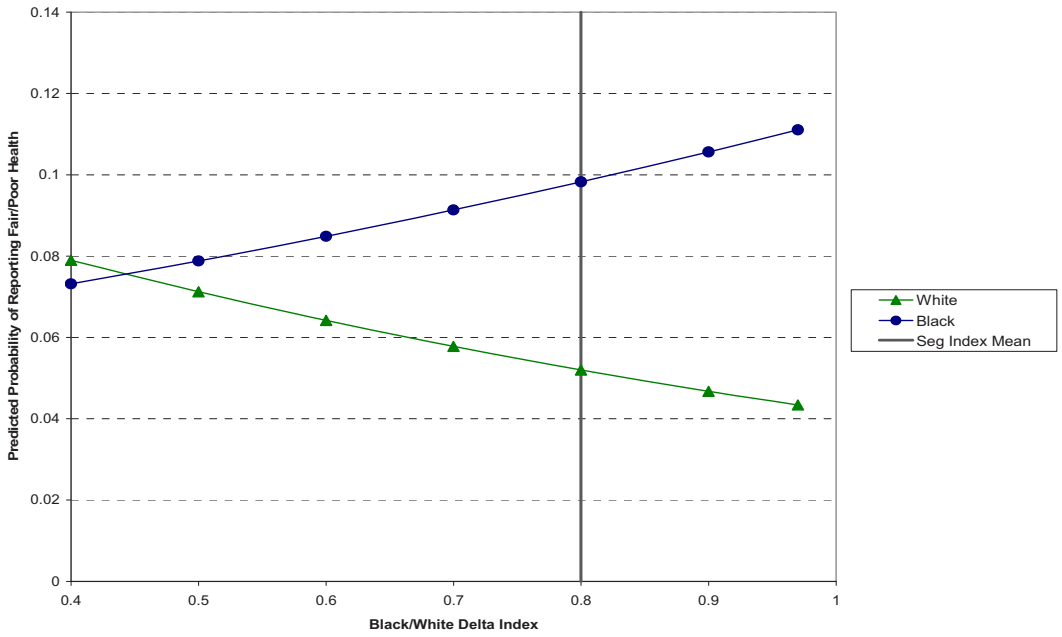
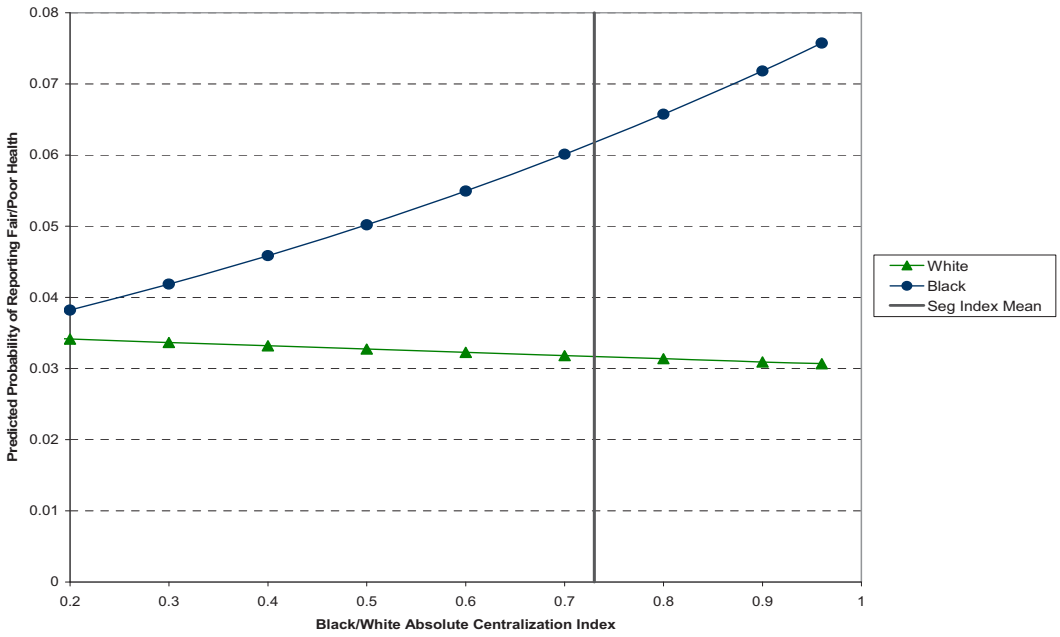


Figure 3.2 Predicted Probability of Reporting Poor Health as a Function of Racial Segregation Level, as Measured by the Black/White Absolute Clustering Index



Results from the economic segregation models presented in Table 3.4 reveal a somewhat different pattern between race, segregation, and health. Economic segregation is consistently associated with advantageous health outcomes for Whites. For example, a ten percentage point increase in the spatial concentration of the poor is associated with a twelve percent decrease in reporting poor health for Whites.

Table 3.4 Results of Economic Segregation Models for Poor Respondent-Rated Health

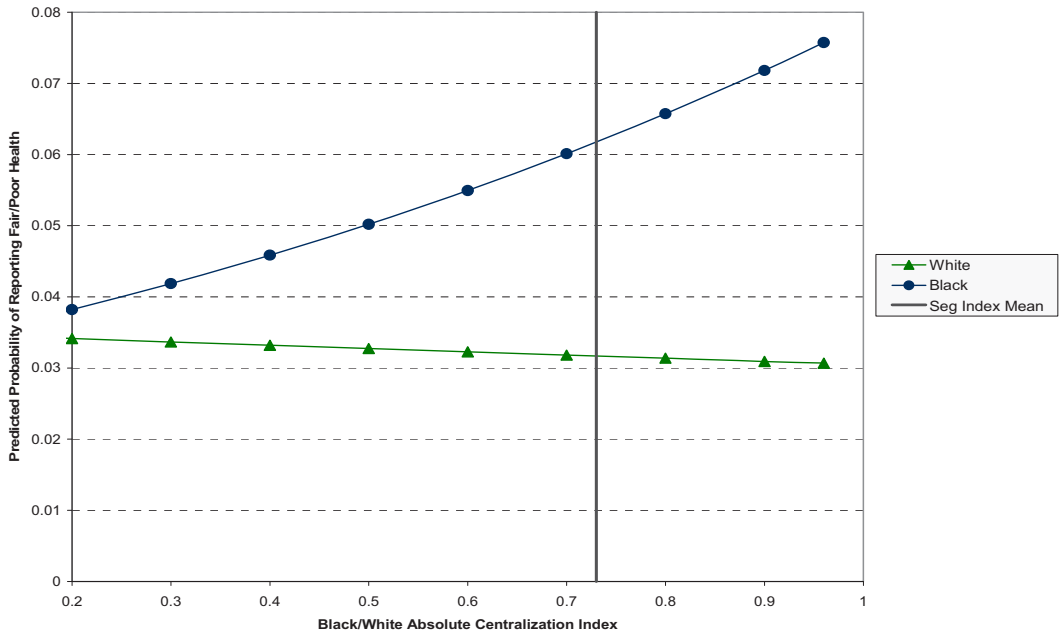
Evenness		Exposure		Concentration	
(Model 6A)		(Model 7A)		(Model 8A)	
	OR		OR		OR
Black	0.53	Black	1.84**	Black	0.59
Dissimilarity Index	0.82***	Isolation Index	0.79**	Delta Index	0.88***
Black* Dissimilarity Index	1.43***	Black* Isolation Index	1.05	Black* Delta Index	1.19**
(Model 6B)		(Model 7B)		(Model 8B)	
	OR		OR		OR
Black	0.54	Black	1.89**	Black	0.61
Dissimilarity Index	0.82***	Isolation Index	0.78**	Delta Index	0.87***
Poor Black* Dissimilarity Index	1.48***	Poor Black* Isolation Index	1.14	Poor Black * Delta Index	1.22**
NonPoor Black* Dissimilarity Index	1.42***	NonPoor Black* Isolation Index	1.03	NonPoor Black* Delta Index	1.18**
Poor White* Dissimilarity Index	1.03	Poor White* Isolation Index	1.10	Poor White* Delta Index	1.03

± ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level, *Statistically Significant at the 10% Level
Coefficients for the dissimilarity, isolation, delta and absolute clustering indices are based on a 10 percentage point change.
All models adjust for gender, age, marital status, education, family income/poverty threshold level, employment status, metropolitan population size, and metropolitan-level poverty rate.

The salutary link between economic segregation and health for Whites does not appear to be greater for nonpoor Whites compared to poor Whites (Models 6B-8B). For Blacks as a whole, spatial patterns of economic evenness and concentration are associated with poorer

health outcome while there is no differential significant relationship between economic isolation and health. Although the larger point estimate for poor Blacks compared to nonpoor Blacks (Models 6B-8B) are suggestive of an even greater detrimental association of spatial isolation based on poverty status, it is not statistically significant.[13] Figures 3.4 and 3.5 show plots of the predicted probabilities of reporting poor health as a function of poor/nonpoor dissimilarity and delta indices, respectively.

Figure 3.3 Predicted Probability of Reporting Poor Health as a Function of Economic Segregation Level, as Measured by the Black/White Absolute Clustering Index



[13] The (even smaller) differences in point estimates between poor and nonpoor Blacks for the economic segregation dimensions of evenness and concentration are also nonsignificant.

Figure 3.4. Predicted Probability of Reporting Poor Health as a Function of Economic Segregation Level, as Measured by the Poor/Non-Poor Dissimilarity Index

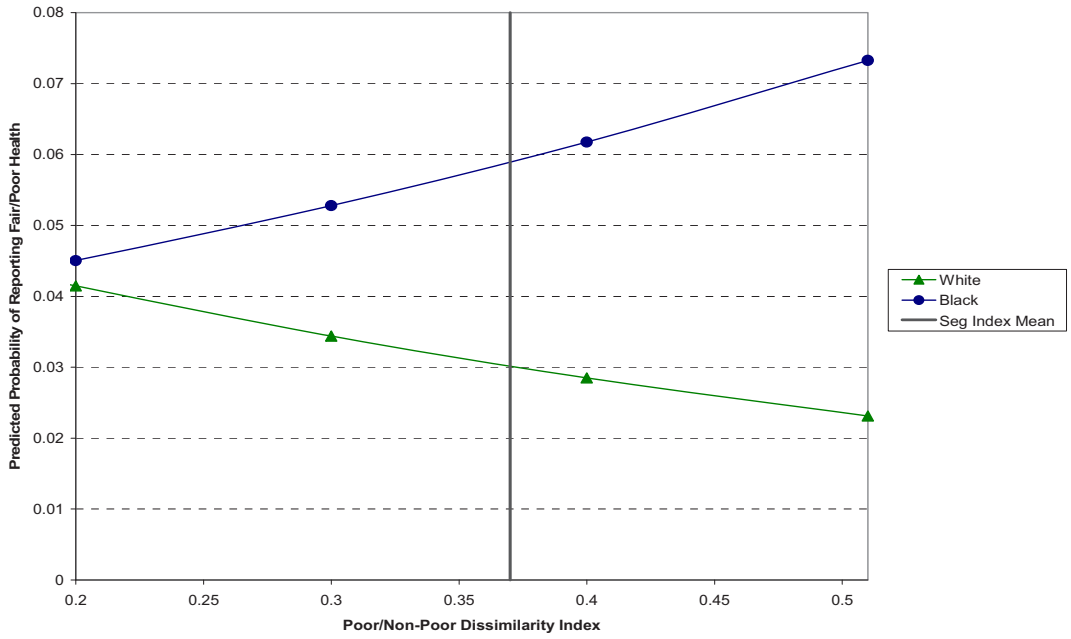
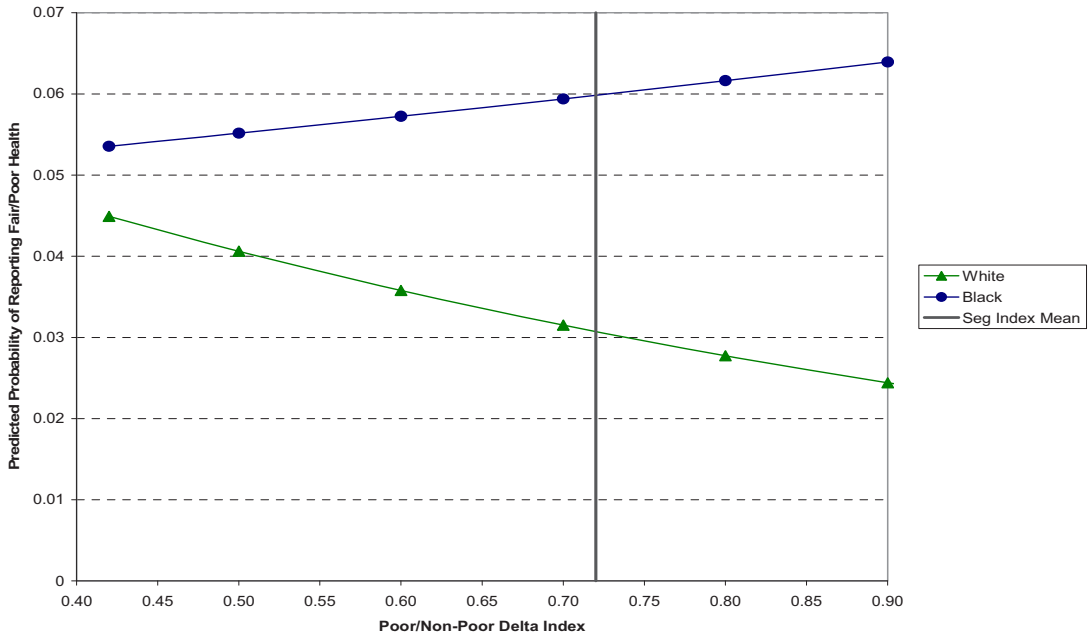


Figure 3.5. Predicted Probability of Reporting Poor Health as a Function of Economic Segregation Level, as Measured by the Poor/Non-Poor Delta Index



Discussion

The significant associations between segregation and health, after adjusting for individual-level socioeconomic and demographic factors, lend support to the relevance of segregation on individual health outcomes. Results from the series of three-level hierarchal models reveal that the two most commonly investigated dimensions of racial segregation, evenness and exposure, have the weakest link to health outcomes. Indices for racial concentration, centralization, and clustering are much more strongly associated to health outcomes. Higher levels of segregation along these dimensions are found to be detrimental for the health of Blacks. Contrary to some aggregate studies (e.g., Collins & Williams 1999) that had found poorer health for Whites in more segregated areas, results across both racial and economic segregation models consistently found no negative association between segregation and health for Whites. Moreover, there is evidence to suggest that the separation of Blacks from Whites and the poor (who are more likely to be Black) from the nonpoor (who are more likely to be White) has beneficial health effects for White individuals.

The relative magnitude and statistical significance of the point estimates for the delta, absolute centralization, and absolute clustering indices compared to those for the dissimilarity and isolation indices suggest that prior studies which have been almost exclusively relying on the latter two indices are overlooking the more influential racial spatial patterns as it pertains to health outcomes. To the extent that racial segregation negatively affects the health outcomes of Blacks through restricting Blacks to lower quality neighborhoods, the dimensions of concentration, centralization, and clustering may more readily capture the spatial patterns of neighborhood quality compared to the dimensions of evenness and interaction. Poorer neighborhoods tend to have a higher concentration of Blacks, neighborhoods in downtown central areas tend to be older and poorer, and contiguous Black neighborhoods are more likely to reflect poor Black ghetto areas. Moreover, the similar results from the racial concentration and economic concentration indices provide some evidence that racial concentration is highly correlated with economic concentration.

The stronger link between economic segregation and health do suggest that neighborhood poverty is an important mediator between segregation and health. Higher economic segregation (and racial concentration) levels along all the three dimensions examined are associated with more detrimental health outcomes for Blacks but also with

more salutary health outcomes for Whites. Segregation, thus, widens the health gap between Blacks and Whites through its positive health impacts for Whites, to the detriment of health for Blacks.

The magnitude of the associations between segregation and health is nontrivial and their robustness to the inclusion of individual-level socioeconomic factors --- factors that may be considered as mediators --- underscores the profound impact of residential racial and economic segregation on racial health disparity and, in turn, population health.

Chapter 4: Dynamics of Income and Neighborhood Context on Health and Racial Health Disparities

Chapter 4: Dynamics of Income and Neighborhood Context on Health and Racial Health Disparities

Introduction

Socioeconomic status and its differential distributions across race are key determinants of health and racial health disparity. However, socioeconomic status is often treated as a static factor with only single-point-in-time estimates of both individual and neighborhood conditions. These cross-sectional measures fail to account for possible heterogeneous histories within groups who may share similar characteristics at a given point in time. Given that long exposures to poverty have more profound impacts than short exposures and that the duration of exposure greatly differs between Blacks and Whites, ignoring the dynamic nature of these factors may lead to the underestimation of their importance in explaining health and racial health disparities. In this chapter, I investigate the relationship between individual and neighborhood poverty on health, focusing on whether the addition of a temporal dimension reveals: 1. a stronger relationship between neighborhood poverty and health, and 2. a greater explanatory power for the health gap between Blacks and Whites that has persisted even after adjustments of single-point measures of socioeconomic status.

The Dynamic Nature of Individual and Neighborhood Characteristics

The importance of the temporal dimension over a life-course is especially salient if one considers the dynamic nature of financial resources, both in terms of absolute monetary levels and poverty status. Although a person's real income is expected to rise as he accumulates experience and expertise in the workforce, there may be considerable volatility over a lifetime (Duncan 1988; Solon 1992; & Zimmerman 1992). Rank and Hirschl (2001) estimate that half of Americans will experience either poverty or affluence at least one year between the ages of twenty-five & seventy-five while only 20 percent will experience neither ends of the economic spectrum. Temporary and short-term fluctuations may be a consequence of job loss or exit from the labor force for various reasons, including returning to school. Similarly, income spikes may reflect a transition into a different field, re-entry into the labor force after a spell of unemployment, or initial employment upon completion of school. Cross-sectional data is ill-equipped to adjust for these life-course changes.

Within a lifetime, drops in income may push some to fall into poverty. However, the condition of poverty is not an absorbing state. To the extent that income fluctuates,

individuals may also cross the threshold into and out of the dichotomous classification of poverty multiple times over the life-course (Stevens 1994). For example, thirty-five percent of those who were poor in 1996 were not poor the next year and almost half climbed out of poverty after three years (Iceland 2003). Thus, a single-point-in-time measurement of even an extremely coarse category such as poverty status may not accurately reflect an individual's long-term situation.

As individual-level characteristics may change, so may the context of neighborhoods in which individuals reside. This may be due to a slow process of neighborhood gentrification or deterioration over time or a more abrupt change when individuals move residential locations. Evidence with respect to the stability of neighborhood environment an individual experiences over time is mixed. Kunz, Page, & Solon (2001) estimate that childhood neighborhood characteristics exhibit great consistency over their observed five year time-frame. The sample correlation between the five-year average of log mean neighborhood income and single-year value is between 0.83 and 0.96, depending on the restriction of the sample to only movers versus all children, respectively. The high correlations suggest that a snap shot of an individual's neighborhood context may not be such an inaccurate reflection of his long-term neighborhood environment. In contrast, several other investigations into neighborhood stability, using longer time frames, suggest a different conclusion (Quillian 2003; Timberlake 2003; Massey et al. 1994; Gramlich et al. 1992). Gramlich et al. (1992) find a great deal of neighborhood heterogeneity even in poor adults, with a quarter of them entering and leaving poor urban neighborhoods (tract poverty rate 30%+) in a year. However, using a dichotomous measure of neighborhood poverty may give a misleading picture if individuals who cross over the neighborhood poverty threshold into or out of poor neighborhoods experience only a marginal change in neighborhood circumstance. This does not seem to be the case. Quillian (2003) estimates that among individuals who move into or out of poor neighborhoods (tract poverty rate 20%+), they usually move into our out of neighborhoods with substantially different poverty rates. For those moving into or out of poor neighborhoods, the average change in neighborhood poverty rate is approximately 18 percent; changes among non-movers were smaller, from 0.6 to 2 percent. However, it is important to note that these minor single year changes usually represented a progression towards neighborhood gentrification or deterioration that took several years to complete.

The Temporal Dimension of Individual and Neighborhood Poverty and Its Impact on Estimates of Neighborhood Effects and Racial Disparities

The dynamic nature of both individual-level economic means and neighborhood context may be a crucial component to understanding racial health disparities, neighborhood context, and their intersection. An extensive line of research has documented a strong association between individual-level socioeconomic status and health outcomes (Haan & Kaplan 1986, Adler et al. 1993, 1994; Pappas et al. 1993). Those with less education and financial resources exhibit disproportionately worse health outcomes across all levels of the socioeconomic gradient, not just at the left end of the distribution (Adler et al. 1993). The distinction between chronic and transient spells of poverty is essential. The handful of studies that have investigated the impact of varying durations of income or poverty on individuals' well-being consistently find that longer term spells of financial hardship have a greater negative impact on health than shorter term spells (Malat et al. 2005; Mcdonough et al. 2005; McDonough & Berglund 2003; Duncan et al. 1994; Korenman et al. 1994). Equally important, current economic circumstances did not erase the impact of earlier poverty experiences on health (Malat et al. 2005; Mcdonough & Berglund 2003) and estimates based on long-term poverty were greater than those based on singe year measures (Korenman et al. 1995). These findings suggest that long-term measures of financial resources may be better predictors of health than single year measures (Benzeval & Judge 2001; Korenman et al. 1995; but see McDonough et al. 1997).

There has been less work on how varying durations of neighborhood poverty exposure differentially affect health and well-being. However, findings from the few that have done so suggest that the impact of neighborhood context increases with duration of exposure (Turley 2003; Ross, Reynolds, & Geiss 2000). For example, Ross et al. (2000) find that the length of time an individual resides in a poor neighborhood is positively related with increased depression and anxiety of residents. Their findings support the view of conventional wisdom that suggests there are cumulative effects of risk exposure, whether they exist at the individual or neighborhood level, on health. That is, a limited exposure to poor neighborhood environments (e.g., driving through a poor neighborhood) would exert minimal effects on health while a persistent, life-long exposure may exert detrimental health effects (e.g., higher stress levels) that have been compounded over time. Cross-sectional measures that fail to partition short-term exposure to neighborhood poverty from long-term

exposure may be noisy proxies for long-term neighborhood conditions, consequently leading to attenuated estimates of neighborhood effects.

Time and Racial Health Disparity

The temporal dimension of individual and neighborhood poverty is closely intertwined with race and by extension, racial health disparity. Research consistently finds that Blacks are more likely to experience chronic poverty, with significantly longer spells of poverty, lower poverty exit rates, and higher re-entry rates than Whites (Iceland 2003; Stevens 1999; McDonough et al. 2005). The longitudinal pattern of neighborhood poverty exposure across racial groups parallels that for individual-level poverty duration. That is to say, not only do Blacks have longer durations of poverty spells, they also spend a disproportionate time in poorer neighborhoods than comparable Whites (Quillian 2003; Timberlake 2003).

Using the PSID, Quillian (2003) estimates the average tenure for those who enter poor neighborhoods (tract poverty rate 20%+) is 8.5 years for Whites and 11.8 years for Blacks. The exit rate after one year is 30 percent for Whites and only 18 percent for Blacks. South and Crowder (1997), using a shorter time frame of the PSID, also find a differential entry and exit rate between Blacks and Whites. The rate of entry into poor neighborhoods for Blacks residing in nonpoor tracts in any given year is 11 percent, compared to 1.4 percent for Whites. Exit rates are 6.7 percent and 16.7 percent for Blacks and Whites, respectively.

Since life trajectories may be shaped early in life, the importance of childhood environment should not be discounted. As in the case with adulthood, there is great racial inequality in childhood exposure to neighborhood poverty and affluence. Timberlake (2003) finds that Black children spend 60 percent of their childhood in poor neighborhoods (tract poverty rate 20%+), compared to 14 percent for White children. On the other side of the economic spectrum, White children spend almost five times longer in affluent neighborhoods (tract poverty rate 3% or less) than Black children. Even more disturbing is that gap seems to be increasing over time (Timberlake 2003).

The notion that both individual and neighborhood poverty may exert their effects through duration of exposure (Timberlake 2003; McDonough et al. 2005) suggest a potential explanation for why a significant portion of Black/White health disparities, even after adjusting for contemporary socioeconomic differences between the two groups, remain unexplained (Williams & Collins 1995, 2001). If we interpret the race residual as a disparity

that cannot be accounted for by differences in socioeconomic status, then we can view the estimation as a potentially biased estimate of the disparity.

This potential bias can be illustrated with an example of a multivariate regression model that investigates the proportion of Black/White health disparity that can or cannot be explained by differences in socioeconomic factors.

Suppose the true relationship is:

$$health = \beta_0 + \beta_1(black) + \beta_2(poor) + \beta_3(durationpoor) + \sum_{i=4} \beta_i(x_i) + u \qquad \text{EQ 4.1}$$

where (health) and (poor) are dichotomous measures of poor health and poverty status, respectively; (durationpoor) is the percent of time an individual is poor up to the time of measurement; (x_i) are conventional socioeconomic and demographic controls including education, marital status, gender, and age; and u is the error term. All measures, except for (durationpoor), are contemporaneous, single-point-in-time measures.

But only cross-sectional data is available and we can only model:

$$health = \tilde{\beta}_0 + \tilde{\beta}_1(black) + \tilde{\beta}_2(poor) + \sum_{i=3} \tilde{\beta}_i(x_i) + v \qquad \text{EQ 4.2}$$

Because all the regressors can be pairwise correlated, it is difficult to obtain the direction of bias for $\tilde{\beta}_1$ without making further assumptions. However, if we simplify the model by ignoring all the other explanatory variables, then the direction of bias is the sign of β_3 multiplied with corr (Black,durationpoor). Given that β_3 has been found consistently to be positive and that there is evidence to suggest that the corr (Black,durationpoor) is strongly positive, the proportion of racial health disparity unaccounted for by single-point measures of socioeconomic status may be overestimated.

To the extent that neighborhood poverty negatively affects health, the direction of bias due to the omission of duration in poor neighborhoods is likely to be the same. The strength of the bias depends on the strength of the association between race and duration in poor neighborhoods, net of the set of controls included in the regression model. It is probable that after accounting for socioeconomic status, the net correlation between race and duration in poor neighborhood is negligible. However, Quillian (2003) found that only a modest fraction of racial differences in duration of residing in a poor neighborhood is contributable to differences in poverty status or family structure. He observed that Blacks above the poverty line and in male-headed household were more likely to be exposed to

poor neighborhoods than Whites below the poverty line and in female-headed households. Consequently, accounting for the temporal dimension in both individual and neighborhood factors may provide additional explanation to the Black/White health disparity that has been thus far largely ignored.

Chapter Objectives

This Chapter has several objectives:

1. To investigate whether multiple-year measures of neighborhood context are stronger predictors of health than single-year measures.
2. To investigate whether longer durations of neighborhood poverty exposure have a larger effect on health than shorter exposures.
3. To investigate whether the Black/White health disparity, net of single-point-in time measures of SES, is further explained with the inclusion of multiple-year measures of individual income and neighborhood context

Data

To examine these relationships, I use the 1980-1996 subset of the PSID.[14] The sample is restricted to Black and White respondents who are either the household head or spouse and at least 18 years of age in 1980. The resulting sample size is comprised of approximately 11,400 individuals, with information on personal income and neighborhood context[15] as far back as 1980. Table 4.1 provides descriptive statistics of the total sample and by race. Socioeconomic status is measured by marital status, family income, education, and labor force status. Marital status is divided into three categories: single, married, and other (divorced, separated, widowed). Educational attainment represents the years of schooling attended (fewer than 12, 12, 13-15, 16, and 17+). Labor force categories include the employed, the unemployed, and the labor force non-participants. The financial means of a family is a continuous measure of family income to poverty threshold ratio, divided into four categories: family needs ratio less than or equal to 1, family needs ratio between 1 and 2, family needs ratio between 2 and 3, and family needs ratio 3 or higher. Neighborhood

[14] In 1997, the PSID sample was modified and the Core Sample was reduced.
[15] Neighborhood is defined as the census tract. Because tracts did not exist for portions of the U.S. before 1990, any computations using 1980-1989 tract information are restricted to respondents residing in areas that have at least 75 percent tract coverage.

Table 4.1 1996 PSID Sample Statistics

Characteristics	Full Sample		White		Black	
	N	%	N	%	N	%
Sample Size	11,386	100	7,676	64.42	3,710	32.58
Gender						
Female	6,309	55.41	4,087	53.24	2,222	59.89
Male	5,077	44.59	3,589	46.76	1,488	40.11
Mean Age	45.73		46.34		44.68	
Race						
White	7,676	64.42				
Black	3,710	32.58				
Family Income						
1996 Mean Family Income/Poverty Ratio	4.29		5.06		2.68	
Mean 1980-1996 Ave Family Income/Poverty Ratio	3.93		4.60		2.54	
Marital Status						
Married	7,967	69.98	5,996	78.12	1,971	53.13
Single	1,063	9.34	405	5.28	658	17.17
Other	2,355	20.69	1,274	16.60	1,081	29.14
Education Level						
<12 Years	2,525	22.52	1,313	17.35	1,212	33.27
12 Years	3,970	35.41	2,659	35.14	1,311	35.99
13 to 15 Years	2,509	22.38	1,697	22.43	813	22.29
16 Years	1,319	11.77	1,122	14.83	197	5.41
17+ Years	887	7.97	776	10.26	111	3.05
Labor Force Status						
Employed	7,668	68.06	5,359	70.43	2,309	63.12
Unemployed	457	4.06	188	2.47	269	7.35
Not In The Labor Force	3,142	27.89	2,062	27.10	1,080	29.52
Tract Poverty						
1996 Tract <10% Poor			4,332	58.11	688	19.12
1996 Tract 10%to20% Poor			2,375	31.86	1,057	29.38
1996 Tract 20%+ Poor			748	10.03	1,853	51.50
Mean 1980-1996 Average Poverty Rate	14.87		10.38		24.12	
Mean 1980-1996 Percent Time in Poor Tract	25.68		10.95		56.10	
Health Status						
Fair/Poor Health	1,931	16.96	989	12.88	942	25.39
Good to Excellent Health	9,455	83.04	6,687	87.12	2,768	74.61

poverty levels are divided into three categories representing affluent, mixed, and poor neighborhoods. These neighborhood categories are defined as less than 10 percent poor, 10 to 20 percent poor, and 20 percent or more poor, respectively. Percentage of time residing in a poor neighborhood is calculated as the ratio of years residing in a poor tract to the number of years observed during the specified timeframe.

Health Measure

I use respondent-rated health as the outcome measure. Respondent-rated health was collected in a five-point scale: fair, poor, good, very good, excellent in which I dichotomize to fair/poor versus good/very good/excellent health.[16] As previously mentioned, although not an objective measure, respondent-rated health has been shown to be a strong predictor of mortality, net of adjustments for clinical measures of health status (Benyamini and Idler, 1999; Idler and Benyamini, 1997). Moreover, the relationship between mortality and respondent-rated health appears to be consistent for Blacks and Whites (McGee et al., 1999).

Descriptive Analyses

I begin with exploratory descriptive analyses investigating the relationship between race, duration of poverty, and duration of residing in poor neighborhoods. Simple bivariate analyses reveal that single-point adjustments for family income do not account for the very different economic histories between Blacks and Whites. Blacks who fall under the poverty threshold in 1996 had experienced durations of poverty that were twice as long as those experienced by comparable Whites (Figure 4.1). Similarly, only 19 percent of Whites who were poor in 1996 had an average income below the poverty threshold during the previous sixteen years, compared to over 50 percent of Blacks (Figure 4.2.). These statistics suggest a single-point-in-time measures of poverty status may be more accurate for Blacks than for Whites.

Parallel analyses for neighborhood poverty rate show a similar pattern. On average, Black individuals who resided in a poor neighborhood in 1996 had spent over 80 percent of the years between 1980 and 1995 in poor neighborhoods (Figure 4.3). Comparable Whites had spent approximately 62 percent of those years in poor neighborhoods. There is an equal

[16] For brevity, fair/poor health is henceforth referred to as poor health.

Figure 4.1 1980-1995 Percent of Years Poor by 1996 Family Poverty Status

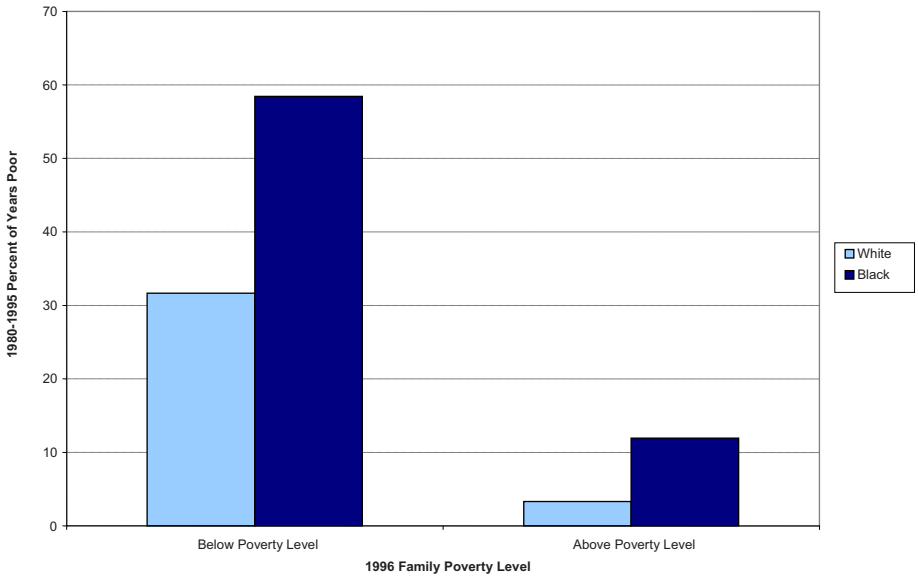


Figure 4.2 1980-1995 Average Family Income/Poverty Ratio Category for Individuals Who Were Poor in 1996

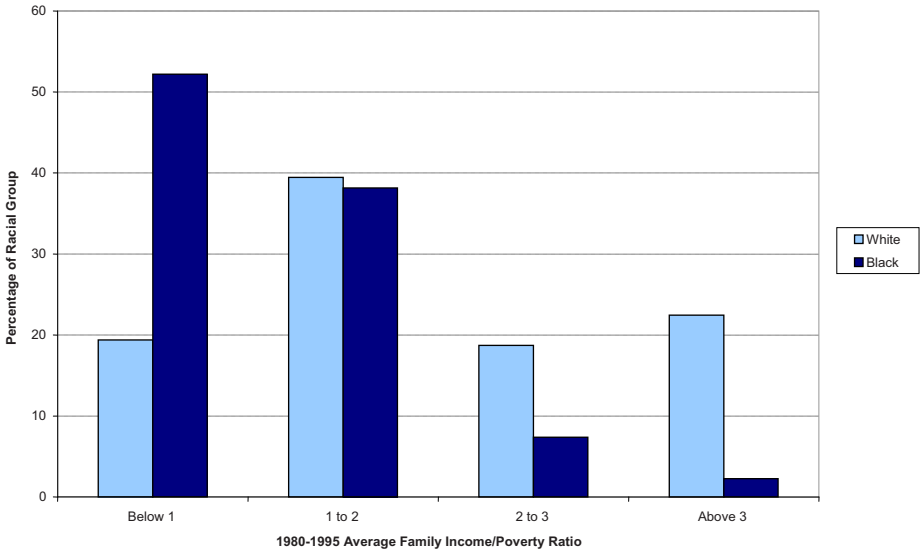


Figure 4.3. 1980-1995 Proportions of Years Resided In a Poor Neighborhood

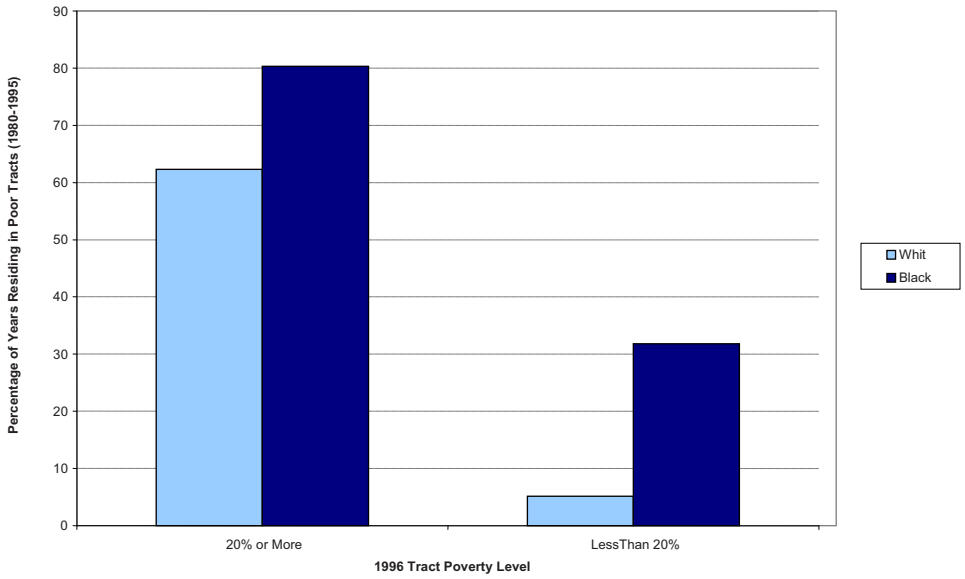
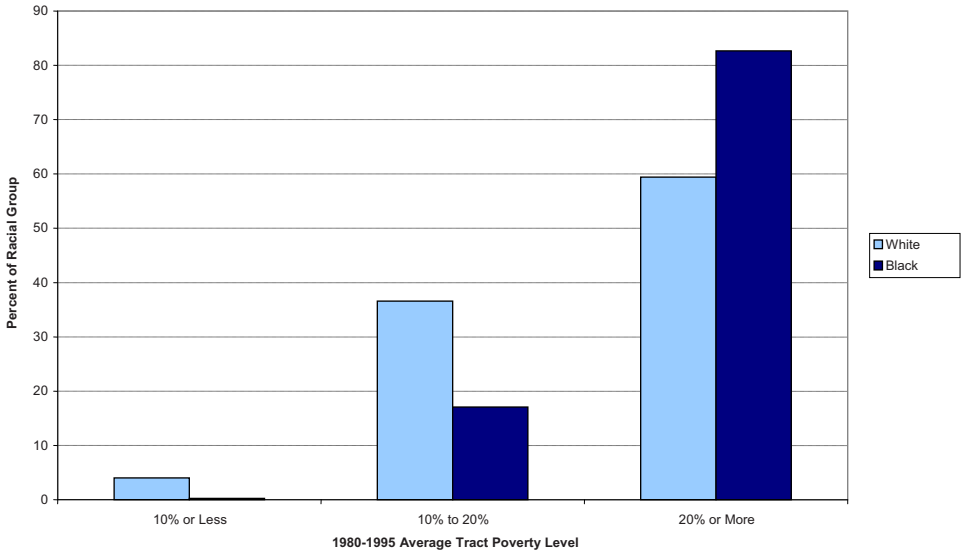


Figure 4.4. Average 1980-1995 Neighborhood Poverty
Category Among Individuals Who Resided In a Poor Neighborhood in 1996



divergence when comparing 1980-1995 average residential neighborhood poverty rates across races (Figure 4.4). Eighty-five percent of Blacks who lived in a poor neighborhood in

1996, lived in neighborhoods between1980-1995 that were on average, poor. In comparison, only sixty percent of Whites residing in poor tract in 1996 had experienced the same conditions.

These descriptive analyses suggest that: 1) the accuracy of single-point measures in capturing long-term income differs across racial group, with a larger proportion of transient poor Whites being classified as chronically poor and 2) the accuracy of single-point measures in capturing long-term neighborhood conditions differs across racial group, with a larger proportion of Whites residing in a poor neighborhood in a given year experiencing better neighborhood environments in the long run. These results support the hypothesis that racial health disparities may be overestimated when only single-point in time measures for individual and neighborhood economic conditions are used to account for the differences in socioeconomic characteristics between Blacks and Whites. However, these are only bivariate statistics and do not adjust for other socioeconomic characteristics (e.g., education and employment status) between Blacks and Whites that are associated with poverty and residing in a poor neighborhood. The magnitude in the heterogeneity of the socioeconomic histories between Blacks and Whites may be attenuated once other single-point measures are included.

In the following sections, I adjust for a set of socioeconomic factors in a series of regression models to empirically examine whether the inclusion of a temporal dimension further accounts for the racial health gap. For each of my analysis, I conduct a logistic regression model with a binary outcome measure of respondent reported poor health. Controls for gender, age, education, employment, and marital status, as previously defined, are included throughout. For models that include multiple point measures of family income or neighborhood poverty, I include a series of dummies that adjust for the number of years family income or neighborhood poverty are observed.

Single-Point versus Long-Term Measures of Individual Income

To test whether including long-term economic measures account for a larger portion of the racial health disparity than single-point-in-time measures, I begin with a base model (Model 1) with only year 1996 measures of socioeconomic and demographic conditions. In the next model (Model 2), I account for past economic conditions by adding the 1980-1995 average family income/poverty threshold categories to examine the relative strengths of

historic and current economic levels in predicting health status. In Model 3, I use the 1980-1996 average family income/poverty threshold ratios as a long-term measure of economic means to compare estimates of single-point versus long-term measures of income on health. Results, presented in Table 4.2, reveal that past economic conditions are strong indicators of current health, even after adjusting for current income levels. Moreover, income estimates based on long-term measures are greater than those based on single year measures. These patterns are consistent with results from previous studies (Benzeval & Judge 2001; Korenman et al. 1995).

Table 4.2. Individual-level Poverty Results for Respondent-Rated Poor Health±

Characteristics	Model 1	Model 2	Model 3
	OR	OR	OR
Race			
Black	1.58***	1.44***	1.45***
White	1.00	1.00	1.00
1996 Family Income/Poverty			
100% Or Less	2.59***	1.71***	
100%to200%	2.28***	1.60***	
200%to300% [17]	1.42***	1.21*	
Over 300%	1.00	1.00	
1980-1995 Ave Family Income/Poverty Ratio			
100% Or Less		1.93***	
100%to200%		2.02***	
200%to300%		1.30***	
Over 300%		1.00	
1980-1996 Ave Family Income/Poverty Ratio			
100% Or Less			3.01***
100%to200%			2.85***
200%to300%			1.65***
Over 300%			1.00

± ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level, *Statistically Significant at the 10% Level
All models adjust for gender, age, education, employment, and marital status.

Accounting for the economic history of individuals only modestly reduced the unexplained racial health disparity observed in Model 1. The inclusion of both current and average past income in Model 2 and long-term 1980-1996 average income measures in Model 3 yielded identical reductions in the Black/White disparity estimates, from OR=1.58

[17] 1996 and 1980-1995 average categories jointly significant at the 1% level for Model 1B.

to 1.44 and 1.45, respectively.[18] The remaining racial health gap is still highly statistically significant, indicating that adjustments for long-term measures of individual socioeconomic factors still do not fully account for why Blacks disproportionately experience poor health.

In the next section, I adjust for the differential neighborhood environment between Blacks and Whites to examine whether this inclusion provides further explanation for the racial health gap.

Single-point versus Long-Term Measures of Neighborhood Context

The modeling sequence to determine the impact of adjusting for long-term measures of neighborhood context versus single-point measures follows the same strategy used in the family income models. The base model accounts for only current family income and current neighborhood context (Model 4A) which I then augment by adding an adjustment for past long-term neighborhood poverty level (Model 5A). Including both past and current neighborhood context measures allows me to gauge the relative strengths of association between historic and current neighborhood environment on health. To test the hypothesis that a multiple-year measure of neighborhood context is a stronger predictor of health than a single-year measure, I compare estimates of the impact of single-point-in-time measures of neighborhood poverty categories to 1980-1996 average measures (Model 6A). In addition, each primary model (i.e., Models 4A, 5A, 6A), is followed by a secondary model (e.g., Model 4B) that uses an alternative measure for personal economic sufficiency. In the secondary models, single-point measures of family income are replaced with 1980-1996 averages. By accounting for long-term economic conditions, this specification reduces the likelihood that the health consequences of previously unobserved family economic fluctuations are being attributed to neighborhood context. Coefficients of the key predictors are presented in Table 4.3.

Results from the base model show statistically significant associations between neighborhood context and health; the odds of reporting poor health increases by 21 percent for individuals residing in poor neighborhoods, relative to those residing in affluent neighborhoods. Comparing results from Model 1, the addition of neighborhood poverty adjustments minimally reduces the relationship between current family income and health

[18] The differences in the racial disparity coefficients (compared to the coefficient for the base model) are statistically different at the 1% level.

and does not provide any additional account for the Black/White health gap. However, single-point measures of neighborhood context may be poor proxies for long-term neighborhood environment. With the addition of past neighborhood context is included (Model 5A), current measures of neighborhood context are no longer statistically significant and the impact of long-term neighborhood poverty measures is not only significant but larger (OR=1.51 vs OR=1.21). With measures of only long-term neighborhood conditions, the estimate for the impact of mixed neighborhood becomes significant, with comparable effects to those in the base model (OR=1.23). Replacing single-point measures of family income with multiple-year averages reveal a similar pattern in the relative estimates of neighborhood context on health with most of the estimates retaining their statistical significance, although the magnitudes of the relationships are slightly attenuated, as expected.

Table 4.3. Tract Poverty Results for Respondent-Rated Poor Health[±]

Characteristics	Model 4A	Model 4B	Model 5A	Model 5B	Model 6A	Model 6B
	OR	OR	OR	OR	OR	OR
Race						
Black	1.51***	1.42***	1.40***	1.34***	1.37***	1.33***
White	1.00	1.00	1.00	1.00	1.00	1.00
1996 Family Income/Poverty Ratio						
100% Or Less	2.42***		2.32***		2.33***	
100%to200%	2.19***		2.14***		2.11***	
200%to300%	1.38***		1.36***		1.35***	
Over 300%	1.00		1.00		1.00	
1980-1996 Ave Family Income/Poverty Ratio						
100% Or Less		2.86***		2.68***		2.63***
100%to200%		2.73***		2.65***		2.58***
200%to300%		1.60***		1.57***		1.56***
Over 300%		1.00		1.00		1.00
1996 Tract Poverty Level						
10% Or Less	1.00	1.00	1.00	1.00		
10%to20%	1.22**	1.17*	1.10	1.09		
20% Or More	1.21**	1.14	0.95	0.93		
1980-1995 Ave Tract Poverty Level						
10% Or Less			1.00	1.00		
10%to20%			1.16	1.11		
20% Or More			1.51***	1.42***		
1980-1996 Ave Tract Poverty Level						
10% Or Less					1.00	1.00
10%to20%					1.23***	1.17*
20% Or More					1.49***	1.38***

[±] ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level, *Statistically Significant at the 10% Level
All models adjust for gender, age, education, employment, and marital status.

With respect to racial disparity, accounting for longer-term conditions of both family income and neighborhood poverty reduces the unexplained racial health gap from OR=1.58 to OR=1.33. However, the gap is still statistically significant; indicating that there still remains a real difference between the health of Blacks and Whites that have yet to be explained. One possible explanation for the remaining racial health gap is that long-term averages may mask large variances in neighborhood context and may not be the most appropriate strategy to summarize an individual's exposure to neighborhood poverty. As such, in the next section I use an alternative strategy to capture the dynamics of neighborhood context by disaggregating residence in neighborhood poverty into categories demarcated by duration of exposure.

Transient versus Persistent Exposure to Neighborhood Poverty

I define a transient exposure to neighborhood poverty as residing in a poor neighborhood for less than half of the time within the timeframe of 1980 and 1996; conversely, residing in a poor neighborhood for over half of the time is considered to be a persistent exposure. I conduct three sets of models that explore the relationships between neighborhood poverty exposure, heath, and health disparities (results presented in Table 4.4). I begin with a model that includes both current neighborhood poverty levels and 1980-1995 durations in poor neighborhood categories (Model 7A). To disaggregate the effects of transient versus persistent neighborhood poverty exposures, I interact the durations in poor neighborhoods measures with the 1996 poor neighborhood category in the next model (Model 8A). The next model adjusts for the amount of exposure to poor neighborhoods between 1980 and 1996, without separately including the current neighborhood context (Model 9A). As in the previous section, each primary model is followed by a secondary model that replaces 1996 single-point measures of family income with 1980-1996 averages.

Estimates of persistent exposure to neighborhood poverty on health are consistently significant across all models. These estimates are slightly larger than those using only single-point measures of exposure to neighborhood poverty but are not as strong as the average neighborhood context measures. None of the estimates for transient neighborhood poverty exposure were statistically significant. In addition, the duration in neighborhood poverty specification left more of the racial health gap unexplained.

Table 4.4. Duration in Poor Tracts Results for Respondent-Rated Poor Health±

Characteristics	Model 7A OR	Model 7B OR	Model 8A OR	Model 8B OR	Model 9A OR	Model 9B OR
Race						
Black	1.44***	1.39***	1.51***	1.44***	1.46***	1.40***
White	1.00	1.00			1.00	1.00
1996 Family Income/Poverty Ratio						
100% Or Less	2.34***		2.39***		2.42***	
100%to200%	2.15***		2.18***		2.18***	
200%to300%	1.37***		1.37***		1.38***	
Over 300%	1.00				1.00	
1980-1996 Ave Family Income/Poverty Ratio						
100% Or Less		2.72***		2.76***		2.74***
100%to200%		2.67***		2.70***		2.67***
200%to300%		1.58***		1.60***		1.60***
Over 300%		1.00		1.00		1.00
1996 Tract Poverty Level						
10% Or Less	1.00	1.00				
10%to20%	1.18**	1.14				
20% Or More	1.00	0.98				
1980-1995 Duration in Poor Tract						
Never	1.00	1.00				
Transient	1.33	1.09				
Persistent	1.33**	1.26*				
1980-1996 Duration in Poor Tract						
Never					1.00	1.00
Transient					1.11	1.09
Persistent					1.26***	1.18*
1996 Tract Poverty Level*Duration in Neighborhood Poverty Interaction						
10% Or Less			1.00	1.00		
10%to20%			1.21**	1.17*		
20% Or More* Transient Neighborhood Poverty			0.98	0.97		
20% Or More* Persistent Neighborhood Poverty			1.26**	1.18		

±***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level, *Statistically Significant at the 10% Level.

All models adjust for gender, age, education, employment, and marital status.

As an alternative specification to account for the amount of exposure to poor neighborhoods, I repeat the previous three sets of models and replace the durations to poor neighborhood categories with a continuous linear term that measures the percentage of years an individual resided in a poor neighborhood (results not shown). Results were generally consistent with the previous section with longer durations in neighborhood poverty being associated with detrimental health. The magnitudes of the unexplained racial health gap were comparable to the results of models that aggregated neighborhood poverty duration into discrete categories.

Sensitivity to Model Specification

The addition of a temporal dimension to both individual and neighborhood poverty, although reducing the racial health disparity between Blacks and Whites, still left a statistically significant racial health gap unexplained. There are several factors omitted from the models (e.g., health behaviors, insurance coverage, genetics, health care quality) which may further explain the Black/White health gap. However, another potential explanation to the persistent unexplained racial health disparity may be model misspecification. Although often totally ignored, model misspecification has been found to be an even greater source of bias than unobserved heterogeneity (Heckman, Ichimura, & Todd 1998; Lalonde 1986). When groups, such as Blacks and Whites, differ greatly in observed characteristics, there may be insufficient overlap between the two groups to make accurate comparisons. As a result, "comparability is only achieved by imposing linearity and extrapolating over different regions" (Heckman, Ichimura, Smith, & Todd 1998). Thus, conventional regression adjustments for the differential characteristics between Blacks and Whites may be extremely sensitive to model specification.

To examine the robustness of the unexplained racial health disparity to model misspecification, I use a propensity score strategy to adjust for the differential distribution of socioeconomic factors between Blacks and Whites.[19] This strategy is less sensitive to model misspecification (Drake 1993) and allows for a simple and direct method to test whether covariate balance has been achieved between Blacks and Whites.

In this particular case, the propensity score is defined as the probability of an individual to be Black, based on a set of observed characteristics. The propensity score is used as

[19] A more detailed description of the propensity score strategy may be found in Chapter 5.

weights that transform the distribution of covariates for the White population to be comparable to those of the Black population. Blacks have a full weight of one and Whites have a weight of P/(1-P), where P is the estimated probability of an individual to be Black. The more a White individual shares the same set of characteristics of the Black population, the higher the probability the individual is Black and the larger the weight an individual is assigned.

Once the weight is calculated, it is employed in a second-stage logistic regression model in which the dependent variable is poor health and the independent variables are the same set of covariates employed in the propensity score model with the addition of the key variable of interest, "Black". Typically, a weighted difference in the outcome variable (e.g., health) between Blacks and Whites is sufficient to recover the racial health disparity estimate since the usage of the propensity score weight achieves covariate balances between the two groups. However, the addition of the covariates in the second-stage model serves as an additional measure of robustness as consistent estimates are achieved if either the propensity score model or the regression model is appropriately specified (Bang & Robins 2005).

I conduct several propensity score models, all of which include gender, age, educational attainment, labor force status, and marital status as covariates.[20] The strategy is to progressively include more information on family income and neighborhood context into the model and examine the minimum amount of information required to fully explain the Black/White health gap. The series of propensity score models are conducted in the following sequence:

Base covariates + 1996 family needs ratio (Model 10)

Base covariates + 1980-1996 average family needs ratio (Model 11)

Base covariates + 1996 family needs ratio
+ 1996 neighborhood poverty level (Model 12)

Base covariates + 1980-1996 average family needs ratio
+ 1996 neighborhood poverty level (Model 13)

Base covariates +1980-1996 average family needs ratio
+ 1980-1996 average neighborhood poverty level (Model 14)

The first propensity score model adds the 1996 family needs ratio to the list of basic covariates (Model 10). Second, I investigate whether a longer term measure of income

[20] These are the same set of covariates included in all the previous regression models.

accounts for the racial health disparity by replacing the 1996 single-point-in-time estimate of family economic well being with the 1980-1996 average family needs ratio (Model 11). Third, I account for single-point-in-time estimates of family income and neighborhood context (Model 12). Fourth, I include long term family income measures but only the 1996 neighborhood poverty information (Model 13). Lastly, I use long term measures of both family income and neighborhood poverty (Model 14). Results from the propensity score models are presented in Table 4.5, along with estimates for the unexplained Black/White health gap from comparable regression models that used the same set of controls for comparison.

Table 4.5. Propensity Score Results for Respondent-Rated Poor Health[±]

Characteristics	Model 120	Model 11	Model 12	Model 13	Model 14
	OR	OR	OR	OR	OR
Black (from regression model)	1.58***	1.45***	1.51***	1.42***	1.33***
Black (from propensity score model)	1.36**	1.17**	1.16	1.08	0.93
Black (from propensity score + regression model)	1.40***	1.21**	1.11	1.06	0.92
1996 Family Income/Poverty Ratio	✓		✓		
1980-1996 Ave Family Income Poverty Ratio		✓		✓	✓
1996 Tract Poverty Level			✓	✓	
1980-1996 Ave Tract Poverty Level					✓

[±] ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level,
*Statistically Significant at the 10% Level.
All models adjust for gender, age, education, employment, and marital status.

The magnitudes of the unexplained racial health gap from the propensity models are consistently smaller than those from comparable conventional regression models. Compared to the results from a regression model that used the same set of covariates, the propensity score results for the base model that adjusted for the only the 1996 single-point measures of family income and base covariates reduced the unexplained health gap from OR=1.58 to OR=1.40. The propensity model explained as much of the Black/White health gap as did regression model 5A which, in addition to the 1996 family income, also adjusted for both 1996 and 1980-1995 average neighborhood contexts. The racial health gap becomes statistically insignificant with adjustments for current family income and neighborhood context (Model 12, OR=1.11). However, because the use of weights in the propensity score

strategy may result in a decrease in precision, the statistical insignificance may be more a reflection of the increased variability than a true explanation of the racial health gap. The additions of long-term family income and long-term neighborhood context yielded estimates for the racial health gap that were both statistically insignificant and less than 1.00 (Model 14, OR=0.93). These results suggest that the racial health gap that has persisted even after adjustments for socioeconomic factors may be more an artifact of measurement error and model misspecification rather than the result of omitted variable bias.

Discussion

This chapter had two main objectives: to ascertain whether single-point measures of neighborhood context underestimate the effects of neighborhood poverty on health and to determine whether the inclusion of long-term measures of socioeconomic and neighborhood context helps to further explain the racial health gap.

Consistent with the few studies that investigated duration of neighborhood poverty on health, results show that longer exposures to neighborhood poverty have a larger detrimental effect on health than shorter exposures. Moreover, results indicate that long-term neighborhood measures are stronger predictors of health outcomes than single-point measures. The simultaneous inclusion of both single and historic neighborhood context measures reveals that long-term measures are more important, as measured by the relative magnitude of the point estimates, than current neighborhood context on current health. Hence, past studies relying on cross-sectional measures of neighborhood context are underestimating the association of neighborhood poverty on individual health. However, the underestimation is only moderate and the magnitude of neighborhood poverty estimates on health is still considerably less than the estimates for individual-level socio economic conditions.

Accounting for long-term family income and neighborhood poverty also reduces the observed unexplained racial health disparity, as compared to the estimates based on cross-sectional models (e.g., from OR=1.51 to OR=1.33). However, the inclusion of long-term family income, long-term neighborhood poverty rate, and neighborhood poverty duration is unable to fully explain the health disparity between Blacks and Whites and a statistically significant racial health gap remains. Results from the propensity score adjustment strategy suggest that a major source of bias that may have led to unexplained racial health disparities

is the reliance of model specification to accurately extrapolate over areas where there is little overlap in observed characteristics between racial groups. While conventional regression adjustments failed to fully explain the racial health gap, the propensity score strategy, with the same set of covariates, fully accounted for the observed health disparity between Blacks and Whites. These results suggest that the elusiveness of being able to explain why Blacks persistently suffer worse health outcomes than Whites stems not from having insufficient information on the determinants (e.g., socio-economic level) of the health disparity between Blacks and Whites but from the inadequate adjustment of these determinants across race. Hence, while health researchers seek additional information (e.g., genetics, health behaviors, and racism) to include in their regression models, it may be that the source of the unexplained Black/White health disparity is more an artifact of measurement error and model misspecification, rather than the omission of key determinants. As such, reaching our goal of understanding the sources of racial health disparities may closer than we first thought.

Chapter 5: Neighborhood Poverty and Health: Context or Composition?

Chapter 5: Neighborhood Poverty and Health: Context or Composition?

Introduction

Despite rather consistent evidence of neighborhood effects on health, current neighborhood-health analyses pay inadequate attention to problems of self-selection, almost exclusively relying on cross-sectional data to adjust for compositional differences across neighborhoods. Hence, causal inferences from these studies are problematic.

In this chapter, I use longitudinal data and employ statistical modeling strategies that address the possible sources of neighborhood effect bias present in previous neighborhood-health studies. The objective of this chapter is to investigate the causal effects of neighborhood economic deprivation on individual health. I begin with first presenting a brief summary of the state of the neighborhood-health literature, focusing on the limitations of their analytical strategies in recovering causal estimates. I then describe the motivation and setup of two modeling strategies that will be employed: a propensity score model and a fixed-effect model, to recover causal estimates of neighborhood effects on health. After reviewing and summarizing the results from both models, I conclude with a discussion on what inferences can be made.

Background

A growing body of literature has documented a positive association between living in economically disadvantaged neighborhoods and adverse health outcomes. Among the strongest causal evidence thus far has come from the Moving to Opportunity (MTO) intervention study. Between 1994 and 1997, families living in high-poverty public housing projects in five cities across the United States, were randomly offered, through a lottery, housing vouchers to aid them to move into lower poverty neighborhoods. Families that were qualified for the study but were not offered vouchers served as the control group. In 2002, after an average of five years since random assignment, over half of the experimental group had lived in neighborhoods with an average poverty rate of less than 20 percent, compared to the average poverty rate for the control group of over 40 percent (Kling, Liebman, and Katz 2005). Although there was no evidence of improvements in general health for the

overall study population,[21] significant mental health benefits and obesity reduction were observed. The magnitude of the benefits was nontrivial. The experimental group experienced a 20 percent reduction in relative risk of being obese, a 30 percent reduction in relative risk of a diagnosis of a major depressive episode, and a 45 percent reduction in relative risk for severe mental illness.[22] The mental health benefits were larger than many of the most effective clinical interventions for depression (Kling, Liebman, & Katz 2005). Moreover, larger effect sizes were observed for those who experienced greater reduction in neighborhood poverty rates, indicating a gradient relationship between neighborhood poverty and health

The body of evidence based on observational data also generally supports the existence of a link between neighborhood economic disadvantage and negative health consequences. Both Robert's (1999a) and Picket & Pearl's (2001) reviews found generally consistent evidence that economically disadvantaged neighborhoods, as primarily identified by measuring the proportion poor in an area, have a detrimental affect on a wide range of health outcomes, including mortality, chronic diseases, mental health, and health behaviors (c.f., Reijneveld & Schene, 1998; Sloggett & Joshi, 1994). These associations remained statistically significant even after adjusting for various individual-level socioeconomic characteristics. However, causal inferences from these studies are tenuous, at best. Because individuals tend to sort into different types of neighborhoods based on observed and most likely unobserved characteristics, differences in health outcomes between individuals across different types of neighborhoods may be a product of neighborhood composition rather than neighborhood context. For example, individuals living in poor neighborhoods may have opted to live there because they value the resources and safety offered in more affluent neighborhoods less than those who live in nonpoor neighborhoods. Or they may have inherent health limitations, limiting their ability to work and earn money, which prescribe them to live in more affordable residential areas characterized by higher poverty rates. To the extent that these characteristics are also correlated to poorer health outcomes, not accounting for these differential individual-level characteristics would spuriously attribute health differences to neighborhood context. Current studies almost exclusively rely on cross-

[21] Significant physical health benefits were observed for individuals less than 33 years old at time of random assignment.
[22] These estimates reflect the effect of treatment on the treated.

sectional data and attempt to account for these differences by controlling for socioeconomic factors such as income and education in regression models. Equation 5.1 represents the conventional modeling strategy used in neighborhood-health studies:

$$HealthOutcome = \beta * NeighborhoodContext + \alpha * SES + \varphi * Behaviors \qquad \text{EQ 5.1}$$

where (SES) and (Behaviors) may consist of a collection of socioeconomic (e.g., education, labor force status) and behavioral characteristics (e.g., smoking and alcohol consumption), respectively. However, these often represent a limited set of controls and cannot account for all potential factors that are often un-measured (and even un-measurable) in survey research. The reliance on cross-sectional data and only a handful of individual-level variables to control for *all* the compositional heterogeneity across neighborhoods makes the claim of (conditional) exogenous variation in neighborhood context in the models difficult to justify. More recent studies have employed hierarchal linear modeling variants to account for the multilevel structure of neighborhood-health data. However, the main advantage of this strategy is that it accounts for the clustering of individuals within neighborhoods and adjusts the standard errors accordingly; it does not account for unobserved confounders anymore than conventional regression models.

Sufficiently adjusting for baseline differences between groups to be compared is a traditional problem in all observational studies. However, estimating the causal effect of neighborhood context is further complicated by the fact that 1) neighborhood context is a life-long exposure starting from birth and 2) neighborhood context is a dynamic experience. I discuss each factor in detail below.

Difficulties in Estimating Neighborhood Effects

It is somewhat ironic that at the same time neighborhood-health studies have been criticized for under-adjustment of individual confounders, they are as likely to be susceptible to bias due to over-adjustment. Beginning from birth, individuals are exposed to the context of their neighborhood environment. The environment in which a child is exposed to may be especially significant as it may influence educational attainment, labor force attachment, and adoption of behavioral norms (Wilson 1987). To the extent that socioeconomic status is a strong determinant of health and neighborhood context influences socioeconomic

attainment, socioeconomic factors may be considered as mediators, rather than confounders. However, in neighborhood-health studies, these factors are often adjusted for in the models, thereby eliminating possible critical pathways through which neighborhoods affect health. The ability to justify which characteristics should be considered as confounders versus mediators is hampered by the usage of cross-sectional data. Because single-point measures of neighborhood context preclude researchers from clearly demarcating the timeframe of neighborhood context that is being investigated, investigators cannot adjust for baseline characteristics (i.e., observed characteristics just before being exposed to the neighborhood context being investigated) as neighborhood context is a continuous exposure that began from birth. Consequently, cross-sectional models compel investigators to adjust for any and all characteristics that can be considered as confounders, including socioeconomic factors that were affected by past neighborhood context. Thus, only direct or instantaneous impacts of neighborhood context are recovered. If recovering the total neighborhood effect is the objective, adjusting for these mediating factors would likely lead to downwardly biased estimates of neighborhood effects. It is not surprising then that estimates of neighborhood impact are consistently quite small, in comparison to the impact of individual-level socioeconomic factors.

The dynamic nature of neighborhood context adds an additional layer of complication. Even though exposure to neighborhoods is continuous, the environment in which individuals are exposed to may be far from static. Over time, neighborhoods may experience socioeconomic transformations as characterized by gentrification or deterioration. Additionally, individuals may move in and out of different types of neighborhoods. This may be especially true for individuals on the margin between poverty and self-sufficiency as they struggle to escape poverty-stricken environments. For example, Gramlich et al. (1992) estimate that a quarter of poor adults enters and leaves poor neighborhoods (tracts with poverty rate of 30%+) in a given year. Thus, single-point-in-time measures of neighborhood context may be noisy proxies for life-long neighborhood context and bias estimates of neighborhood effects towards zero.[23]

While researchers examining health consequences of neighborhood context have largely left the issues of unobserved confounders and mediating factors unexamined, researchers

[23] If single-point-in-time estimates are interpreted as the impact of one year's worth of exposure, then, under the assumption that longer exposures lead to greater impacts, estimates may be biased up.

investigating neighborhood impacts on socioeconomic attainment such as marriage, high school graduation and employment have, using an array of different econometric strategies, have made more considerable strides towards casual inferences. Cutler and Glaeser (1997), for example, used instrumental variables to examine the effects of racial segregation on employment rates and Aaronson (1998) used variation in neighborhood context between siblings to estimate a fixed-effect model on neighborhood poverty on educational outcome. More recently, Harding (2003) used propensity score adjustment and sensitivity analysis to estimate the impact of neighborhood poverty on high school graduation and teenage pregnancy. Results from all of these studies suggest that neighborhood context does have real and substantively strong impacts on individual socioeconomic attainment.[24] Cutler and Glaeser's results indicated that a one-standard-deviation reduction in metropolitan racial segregation (13 percent), as measured by the dissimilarity index, would eliminate approximately one-third of the observed gap between Blacks and Whites in high school graduation rates, income, and rate of single motherhood. Aaronson found that an increase in the neighborhood poverty rate of 10 percent decreases the likelihood of graduating from high school by 7 percent. Harding found that residing in a high poverty neighborhood (census tracts with poverty rate of 20%+) compared to low poverty neighborhood (census tracts with poverty rate less than 10%) approximately doubles the odds of high school dropout. The effect of neighborhood poverty on teenage pregnancy was even greater, with residency in high poverty neighborhoods more than doubling the odds of teenage childbirth.

The modeling strategies applied by Harding and Aaronson each focused on a different source of bias in neighborhood effects models. Harding's strategy addressed the problematic issue of adequately adjusting for compositional differences while allowing for the impact of possible mediating factors to play out. In his study, Harding defined and constrained the duration of neighborhood context under investigation to be within a restricted timeframe. Consequently, he was able to adjust for time-varying covariates as measured just immediately preceding the timeframe window. No adjustment was performed for factors that changed during the window in which individuals were exposed to the neighborhood context, as any *differential* changes in individual factors between the two groups were attributed to the effect of neighborhood environment. Thus, the modeling strategy accounts for the compositional

[24] Cutler and Glaeser's, Aaronson's, and Harding's models are conditioned on individual-level socioeconomic characteristics.

differences as observed before exposure while at the same time avoiding over-adjustment of time-varying covariates (e.g., labor force status, family income) that may be affected by neighborhood context.

However, the propensity score approach used by Harding is not without its limitations. To make an argument for causal inference, the propensity model, as in the case of conventional regression models, relies on the absence of omitted variables that are correlated to *both* the treatment and the outcome.[25] For example, educational attainment is correlated to both neighborhood poverty level and health. If the propensity score model lacked that key predictor, estimates of neighborhood poverty on health would wrongly attribute the effect of education on health to neighborhood context, thereby overstating the magnitude of the neighborhood effect. Although sensitivity analyses can address how strong the correlations must be to render the point estimate statistically insignificant, it does not address the issue of unobserved heterogeneity directly.

In contrast, Aaronson's fixed-effect modeling strategy is designed to directly account for unobserved heterogeneity. By capitalizing on time-varying factors within a person (or group as in the case of sibling fixed-effect models), the fixed-effect model relies on the variation in the neighborhood poverty rate within a person across time to estimate the impact of neighborhood context. This strategy eliminates any bias on estimates of neighborhood effects due to individual unobserved non-time-varying factors. However, as in the propensity score approach, the fixed-effect strategy has limitations. Although it effectively eliminates all unobserved heterogeneity within an individual that is constant across time, it does not account for any time-varying unobserved factors that may be related to neighborhood context. In addition, it requires adjustment for time-varying factors that may be considered as mediators between neighborhood deprivation and health outcomes. This disadvantage results in the same issue of over adjustments that have plagued conventional cross-sectional modeling strategies and would likely bias downward the total impact of the neighborhood effects estimates.

In recognition that each strategy has their own strengths and weakness, my approach to estimating the impact of neighborhood poverty on health is to employ both the propensity score adjustment and fixed-effect strategies and compare results across models. I suspect

[25] Treatment, in this case, is exposure to neighborhood poverty.

that the neighborhood effects estimates from the fixed-effect model, because it over adjusts for mediating factors will be smaller than the estimates from the propensity score model.

Health Outcome

As in previous chapters, for the health outcome of interest, I use a five-scale (poor to excellent) respondent-rated health measure, dichotomized to fair/poor health.[26] There are several advantages in using respondent-rated health as the outcome measure. First, it is a global measure that captures both physical and mental health. Second, and more importantly for the modeling strategies employed in this chapter, it is a dynamic measure that has two useful properties: it is nonabsorbing in the sense that individuals can recover and lapse back into poor health across time and changes in perceived health has been shown to move in the same direction as changes in reported diseases (Goldberg et al. 2001). The nonabsorbing and time varying aspect of this measure lends itself well to a fixed-effect model approach as well as having it serve as a baseline health measure for the propensity model.

Propensity Score Analytical Framework

As previously mentioned, disentangling confounders from mediating factors is especially difficult in neighborhood effects models because neighborhood context is a life-long exposure starting from birth. Conventional neighborhood-health investigations thus far have (implicitly) treated single-point-in-time neighborhood measures as proxies for life-long exposure. As such, there is no conventional "pre-treatment" phase in which differing baseline factors can be adjusted for prior to the initiation of exposure to neighborhood context. In this section, I circumvent this problematic conceptual framework by explicitly demarcating a finite timeframe in which the effect of neighborhood context is estimated. As such, I allow for the effects of mediating factors to pass through and be accounted for in the estimate of the total impact of neighborhood poverty on health.

Conceptually, the propensity score analytical strategy for recovering the total neighborhood effects attempts to emulate the framework of the MTO Demonstration. In the MTO Demonstration, qualified individuals were randomly assigned to either the experimental or control group. Hence, individuals in the experimental group who were given housing vouchers that enabled them to move into low poverty neighborhoods were, on

[26] For brevity, fair/poor health is henceforth referred to as poor health.

average, no different from individuals in the control/comparison group who were not given any voucher. After several years, health outcomes were measured for each group. Any differences in outcomes between the two groups were attributed to the effect of being exposed to differing neighborhood context, with the duration of exposure measured as from the time the experimental group entered their new low poverty neighborhoods to the time the outcomes were measured.

To emulate the MTO framework, I define a timeframe, $[t_0, t_c]$, for which the effects of neighborhood context are to be measured. Individuals who resided in areas with an average neighborhood poverty rate equal to or over 20 percent within this time frame are considered to have been exposed to high poverty neighborhoods. That is, they are considered to be in the "experimental" group. Individuals who resided in areas with an average neighborhood poverty rate less than 20 percent within this time frame are considered to be in the "control" group. Comparability is achieved by adjusting for observed baseline differences between the two groups as measured at t_0, just before the beginning of the "treatment", by propensity score weighting --- which I explain in full detail in the next section. At the end of the defined timeframe, t_c, I compare the health outcome between the two groups. Therefore, by clearly defining a timeframe of interest, I can adjust for only baseline confounders (pre-treatment characteristics) and allow the effects of time-varying mediating factors (e.g., socioeconomic position) to pass through by not re-adjusting for any characteristics during the defined exposure timeframe. Implicitly, this model assumes that any differential changes in baseline characteristics at the end of the timeframe are due to neighborhood context and should be included in the neighborhood effect estimate.

Propensity Score Adjustment

In a multivariate regression model approach, adjustments for systematic differences between individuals residing in high poverty neighborhoods and those residing in low poverty neighborhoods would entail including characteristics that are known to differ between the two groups as covariates into a model that relates neighborhood type to the health outcome. However, when differences between the two groups are large, as is typically the case between residents of low poverty and high poverty neighborhoods, estimates from parametric regression models may be particularly sensitive to assumptions of model specification. In other words, when there exist substantial differences across the groups in

which comparisons are to be made, "comparability is only achieved by imposing linearity and extrapolating over different regions" (Heckman, Ichimura, Smith, & Todd 1998). Because proper extrapolation requires knowledge of the true relationship (e.g., linear vs quadratic vs log vs interaction terms) between the outcome and *all the* covariates, regression analyses, masking any insufficiencies in the overlap of covariates, may lead to improper inferences --- even in the absence of omitted confounders. In contrast, the propensity score covariate adjustment approach allows for a straightforward t-test to examine whether there remains any statistically significant difference in covariates across the two groups. This allows for transparency in the quality and extent of covariate balance between the two groups.

Unlike the more traditional regression model framework, the propensity score's strategy to account for systematic differences between groups to be compared is to model the selection process directly. For this particular application, the selection process refers to the sorting of individuals into high poverty and low poverty neighborhoods. Thus, the objective of the propensity score model is to estimate the probability of an individual to reside in a high poverty neighborhood as a function of a set of observed characteristics. The estimated probability is called the propensity score and individuals who reside in high poverty neighborhoods (the treatment group) with similar propensity scores to those who do not (the control group) are considered to be directly comparable.

More formally, the propensity score is defined as the probability that an individual with characteristics **X** is assigned to the treatment group, $P(T=1 \mid \mathbf{X})$ where $T=1$ reflects receipt of treatment and **X** represents an array of individual characteristics. Conventionally, a standard logistic regression model is used to estimate the propensity score. However, this approach may not provide sufficient covariate balance when the relationship between the treatment and covariates is complicated, possibly requiring nonlinear transformations and interaction terms to be included in the model. The opaqueness of the optimal propensity model often requires researchers to experiment with different functional forms, testing each time whether sufficient covariate balance is achieved. In this study, I use a multivariate nonparameteric regression technique called generalized boosted models to estimate the propensity scores. This method uses an iterative procedure applied to a series of regression trees and provides an automated, adaptive modeling algorithm that accounts for nonlinear transformations and interactions of the covariates to solve for the optimal $P(T=1 \mid \mathbf{X})$ that

minimizes the differences between the treatment and propensity-score-adjusted control group (McCaffrey, Ridgeway, & Morral, 2004).

There are several strategies, including matching and stratification, to adjust for covariate differences once the propensity score is estimated. In this study, I incorporate the propensity score into the response model as observation weights. Individuals in the treatment group are assigned a full weight of one while individuals in the control group are assigned a weight of:

$$\hat{w}_i = \frac{\hat{P}(T = 1 \mid X_i)}{1 - \hat{P}(T = 1 \mid X_i)}$$

This weighting scheme recovers the treatment effect on the treated or in our case, the effect of living in high poverty neighborhoods on individuals who were exposed to neighborhood poverty, and serves to transform the covariate distribution of the control group to correspond with those of the treatment group. Once accomplished, there should be no differences between the two groups on factors that were included in the propensity score model.

Intuitively, the propensity score strategy may be thought of as a statistical adjustment in which the aim is to emulate as closely as possible the setup of a random control trial. In a conventional random control trial experiment, individuals are randomly assigned to either the treatment group or the control group; for the purposes of this study, individuals exposed to neighborhood poverty are considered to be in the treatment group and individuals not exposed to neighborhood poverty are considered to be in the control group. In principal, individuals in the treatment group should be no different to those assigned to the control group and recovering the treatment effect simply requires differencing the outcome variable between the experiment group and the weighted control group with no further need for statistical adjustment.

However, when differences between the two groups are substantially different, it may be impossible to adequately adjust for the differential distributions of the covariates. While conventional regression models may mask the occurrence of this shortfall, propensity score adjustments allows for a simple and straightforward strategy to compare the quality of covariate balance by statistically testing, for each covariate used to estimate the propensity

score, whether the covariate distributions between the treatment group and control group differ.

Propensity Score Sample and Model Specification

The timeframe for which the effect of neighborhood context is to be measured is 1985 to 1997, inclusive. Hence, [t0, te] = [1985, 1997]. The sample I use for the propensity score model is restricted to PSID heads and wives who were observed in the baseline year of 1984 and at least once again between 1995 and 1997 with valid health outcome and neighborhood poverty measures for both time periods. Respondents were at least 18 years old in 1984.[27]

The health outcome variable is respondent-rated health, as measured in the latest year between 1995 and 1997 for which the respondent is observed. Neighborhood context is a binary measure of neighborhood poverty derived by averaging the neighborhood poverty level of the respondent between 1985 and the year the respondent-rated health is measured. An average neighborhood poverty rate of 20 percent or more during this time period is defined as a high poverty environment and is the "treatment".

The analytical strategy is to adjust for observable individual and neighborhood level differences between the high poverty and low poverty neighborhood groups *before* the exposure to these neighborhood contexts begins. This allows for the adjustment of observable differences between the two groups while refraining from needing to adjust for time-varying factors that may have been affected by neighborhood context.

This strategy is analogous to a random-control trial design in which individuals are randomly assigned into two groups in 1984, the start of the study. For the next 13 years, one group is assigned to live in a neighborhood where, on average, the poverty rate is twenty percent or higher. The other group is the control group and is assigned to live in neighborhoods where the average poverty rate is less than twenty percent. In 1997, the end of the study period, respondent-rated health is collected and compared across the two groups. Differences across the two groups measure the effect of exposure to neighborhood poverty during the 13 years on health.

[27] Because older respondents in the sample may be relatively healthier given that they have not died during the study time period, I also conducted sensitivity analyses by restricting the sample to younger respondents who are less likely to be affected by selective attrition due to death. Results were substantively similar.

I control for an extensive set of individual socioeconomic factors that are observed just prior to the start of the neighborhood exposure duration to be investigated. These factors include employment status, educational attainment, marital status, female-headed household indicator, welfare recipient indicator, and wealth as measured in 1984. In addition, I include 1984 respondent-rated health as a baseline health adjustment and, since current neighborhood quality is a strong predictor of future neighborhood quality, I also adjust for the 1980 to 1984 average neighborhood poverty rate. Non time-varying adjustments include race and gender. Finally, I include adjustments for the number of years on which the average neighborhood poverty rates are based.

As previously mentioned, one of the biggest advantages in applying a propensity score method over conventional regression models is that balance of covariates can be statistically tested, allowing for crucial transparency on the quality of covariate adjustment. Before propensity score adjustment, residents in high and low poverty neighborhoods differ greatly (Table 5.1). Not surprisingly, those residing in high poverty neighborhoods tend to have a history of residing in high poverty neighborhoods, be Black, and have lower socio-attainment attainment. These differences are quite substantial, suggesting that conventional regression model approaches may be sensitive to assumptions of model specifications. After propensity score adjustment, these differences are no longer statistically different across many of the observed characteristics. However, though substantially reduced, the differences on baseline respondent-rated health, neighborhood poverty, family needs ratio, employment status, and educational attainment remain statistically significant at the 5% level. The incomplete balancing of these factors, reflecting the initial large divergence in the observed characteristics between the two groups, indicates that further statistical adjustments are warranted. Although an unfortunate circumstance, this highlights the key advantage of the propensity score approach as the lack of covariate balance is readily discerned; this would not be the case in a regression model.

Because complete covariate balance was not achieved, a simple weighted difference on observed respondent-rated health between the two groups may yield biased results. Hence, I choose to model the treatment effect on health using a weighted logistic regression model with the purpose of including the set of characteristics that was identified to be incompletely balanced. Usually, if covariate balance is achieved across all observed characteristics, no

Table 5.1. 1984 PSID Sample Characteristics: Pre- and Post-Propensity Score Weighting

Characteristics	High Poverty Neighborhood	Low Poverty Neighborhood (No PS Weighting)	P-Value of Difference	Low Poverty Neighborhood (After PS Weighting)	P-Value of Difference
Gender					
Female	0.65	0.56	0.00	0.68	0.23
Race					
Black	0.79	0.18	0.00	0.76	0.08
Age	39.25	39.01	0.57	37.90	0.13
Economic Factors					
Family Needs Ratio	2.00	4.38	0.00	2.17	0.03
Wealth	14281.47	64276.61	0.00	12663.24	0.80
Welfare Recipient	0.13	0.02	0.00	0.10	0.20
Female Headed Household	0.21	0.05	0.00	0.21	0.94
Education					
No High School	0.46	0.18	0.00	0.43	0.28
High School	0.38	0.41	0.05	0.39	0.97
Some College	0.11	0.20	0.00	0.12	0.59
College	0.03	0.14	0.00	0.05	0.04
Graduate School	0.01	0.08	0.00	0.02	0.19
Labor Force Status					
Employed	0.55	0.73	0.00	0.63	0.01
Unemployed	0.13	0.03	0.00	0.11	0.29
Not In The Labor Force	0.33	0.24	0.00	0.27	0.02
Marital Status					
Single	0.21	0.10	0.00	0.23	0.52
Married	0.54	0.77	0.00	0.50	0.16
Widowed	0.07	0.04	0.00	0.28	0.45
Divorced	0.10	0.06	0.00	0.32	0.30
Separated	0.08	0.02	0.00	0.26	0.40
Baseline Factors					
Baseline Neighborhood Poverty	27.54	9.42	0.00	25.36	0.01
Baseline Poor Health	0.27	0.10	0.00	0.40	0.01

additional adjustment is required and the weighted logistic regression model with only the treatment variable on the RHS is sufficient and would yield the same results as the weighted difference. However, when there remain observed statistical differences between the two groups, the addition of these covariates would add an additional layer of adjustment. While regression models are sensitive to model misspecification when the treatment and control groups are substantially different, weighting by the propensity score serves to close that gap -

-- perhaps sufficiently so that additional modeling with covariates can adequately adjust for the remaining differences and provide potential bias reduction. Moreover, the augmented strategy of combining propensity score with regression adjustment may increase the precision of the neighborhood effect estimate and serves to produce a "doubly robust" estimator in so much that the neighborhood effect estimate is unbiased if *either* the propensity or regression adjustment is correctly specified (Bang & Robins 2005).

The second-stage regression model is then:

$$Log\left(\frac{\Pr(PoorHealth=1)}{1-\Pr(PoorHealth=1)}\right) = B0 + B1(poorneighborhood) +$$
$$B2(1984\,familyneedsratio) + B3(1984education) +$$
$$B4(1984neighborhoodpoverty) + B5(employment) + B6(1984health)$$

Individuals residing in high poverty neighborhoods have a full weight of one and individuals residing in low poverty neighborhoods are weighted by

$$\hat{w}_i = \frac{\hat{P}(T=1 \mid X_i)}{1-\hat{P}(T=1 \mid X_i)}.$$

I begin with an unconditional weighted logistic regression model with only the neighborhood poverty measure on the RHS and obtain an estimate of OR=1.75. This estimate is expected to be slightly biased as there remains statistically significant baseline differences between residents of high and low poverty neighborhoods. With the inclusion of the five covariates: baseline respondent-rated health, neighborhood poverty, family needs ratio, educational attainment, and labor force status, the estimate is slightly attenuated. Nonetheless, results (shown in Table 5.2) reveal that there is a significant impact of neighborhood poverty exposure on individual self-respondent health (OR=1.63), indicating that on average, a person who resided in a high poverty neighborhood is 63 percent greater

Table 5.2. Propensity Score Results for 1997 Respondent-Rated Poor Health

	Propensity Score Models			OLS Model
	Model 1	Model 2	Model 3	Model 4
1984 -1997 Average Tract Poverty				
Average Tract Poverty <20%	1.00	1.00	1.00	1.00
Average Tract Poverty 20%+	1.75***	1.63***	1.67***	1.26*
Race				
White			1.00	1.00
Black			1.06	1.42***
Gender				
Female			0.78*	0.75***
Male			1.00	1.00
Age			1.10***	1.09***
Age Squared			1.00*	1.00*
Family Needs/Poverty Ratio				
100% Or Less		1.32	1.76**	1.70***
100%to200%		1.36*	1.62**	1.63***
200%to300%		1.31	1.51**	1.42***
Over 300%		1.00	1.00	1.00
Family Wealth				
Wealth Quintile 1			1.03	1.32*
Wealth Quintile 2			0.79	1.04
Wealth Quintile 3			0.81	1.01
Wealth Quintile 4			1.00	1.00
Education				
No High School Degree		2.24***	1.59***	1.43***
High School Degree		1.00	1.00	1.00
Some College		0.74	0.80	0.83
College		0.46**	0.47**	0.41***
Graduate School		0.94	0.63	0.58***
Labor Force Status				
Employed		1.00	1.00	1.00
Unemployed		1.04	1.19	1.48***
Not In The Labor Force		1.41**	1.20	1.33***
Baseline Health				
Poor Health		5.00***	3.63***	4.42***
NonPoor Health		1.00	1.00	1.00
Baseline Neighborhood Poverty Rate				
Poverty Rate Less Than 10%		1.00	1.00	1.00
Poverty Rate 10-20%		0.95	0.84	0.87
Poverty Rate 20%+		0.76*	0.67**	0.83

odds to report poor health than a comparable individual who did not reside in a high poverty neighborhood. As an additional check for covariate balance, I add the full set of factors that were included in the propensity score model. The inclusion of the remaining covariates into the second-stage regression model does not significantly alter the

neighborhood effects estimate. This is to be expected, as these covariates should no longer be correlated to neighborhood poverty after propensity score weighting. As aforementioned, the inclusion of the full set of covariates also serves to produce a "doubly robust" estimator. In comparison, the neighborhood effect estimate from a conventional logistic regression model without propensity score weighting yielded a smaller effect size (OR=1.26). The divergent results suggest that conventional regression strategies may inadequately adjust for systematic differences between Blacks and Whites, yielding biased results.

Fixed-Effect Model

As in conventional OLS regression models, the propensity score method can only effectively adjust for observed factors. Hence, the validity of a causal inference from the propensity model rests on the strong assumption that there are no unobserved factors that are systematically related to both neighborhood type and health. If such an unobserved factor exists, the estimate of neighborhood poverty may be biased. For example, if individuals who engage in high health risk behavior also tend to reside in poorer communities, to the extent that this unobserved factor is not already captured by observed characteristics (e.g., baseline health status), adjustments based on the propensity score may not adequately achieve comparability between the two groups. Neglecting to account for this preference would spuriously attribute the positive health effects to neighborhood environment rather than inherent individual tendencies. In this section, I account for possible unobserved confounders.

In situations where multiple observations of an individual is available and both the treatment and an outcome of interest (that is nonabsorbing) varies across time, accounting for unobserved individual heteterogenity may be accomplished through the use of a fixed-effect model. In 1984, the PSID began collecting respondent-rated health measures of heads and wives of households annually. Consequently, intra-person variation in this health measure and neighborhood context across time allows for the estimation of a within-person estimator of the impact of neighborhood poverty on health.

Under the assumption that the health production function is:

$$Y_{it} = B1(X_i) + B2(X_{it}) + B3(N_{it}) + \varepsilon_i + \varepsilon_{it} \qquad \text{(EQ 5.2)}$$

where Y_{it} is a measure of health for individual i at time t, X_i are non-time varying individual characteristics (e.g., race, gender), X_{it} are time varying individual characteristics (e.g., family income, marital status), and N_{it} are time varying neighborhood characteristics. There are two components in the error term: an individual-specific error term, ε_i, that is constant across time and an individual-specific error term, ε_{it}, that is time-varying. Note that the ε_i error component includes omitted characteristics that are constant across time.

The concern here is that individuals living in high poverty neighborhoods have unobserved characteristics that increase both their likelihood of living in high poverty neighborhoods and having poor health. In other words, a correlation between neighborhood characteristics (N_{it}) and unobserved individual characteristics (ε_i) may exist. Estimating EQ 5.2 under this condition would lead to biased neighborhood estimates.

However, the fixed-effect modeling strategy is designed to eliminate individual-specific unobserved heterogeneity. This is accomplished by performing a fixed-effects transformation of EQ 5.2 to produce a fixed-effects (or within) estimator. That is, average over time EQ 5.2:

$$\overline{Y}_t = B1(X_i) + B2(\overline{X}_{it}) + B3(\overline{N}_{it}) + \overline{\varepsilon}_{it} \qquad \text{(EQ 5.3)}$$

and then subtract this from EQ 5.2. The resulting equation is:

$$Y_{it} - \overline{Y}_t = B2(X_{it} - \overline{X}_{it}) + B3(N_{it} - \overline{N}_{it}) + (\varepsilon_{it} - \overline{\varepsilon}_{it}) \qquad \text{(EQ 5.4)}$$

As a consequence of the transformation, ε_i is removed because it does not vary over time. Thus, estimates of neighborhood effects will no longer be affected by possible correlation between neighborhood conditions and unobserved, non time-varying person-specific factors.

The fixed-effect estimator described above can be readily applied to a linear model. However, the same approach applied to non-linear models has been shown to yield estimates that are, asymptotically, inconsistent as the number of observations increases to infinity for a fixed number of time periods. This is because the number of person-specific

parameters, i.e., person dummy, increases without limit while the amount of information about each person-specific parameter remains fixed (Neyman & Scott 1948). In contrast, the conditional fixed-effect estimator has been shown to be consistent (Chamberlan 1980). Moreover, Monte Carlo simulations indicate that conditional fixed-effect estimates show only a negligible amount of bias for finite samples even with time periods less than twenty (Katz 2001). Thus, the fixed-effect estimates presented here are based on the conditional fixed-effect logistic models.[28]

Fixed-Effect Sample and Model Specification

The fixed-effect sample consists of PSID heads and wives of household who are at least 18 years old in 1984. The time frame for the model is between 1984 and 1997, a span of 13 years. Due to attrition and new family formation, the panel is unbalanced, with individuals entering and exiting the sample at various times. In addition, because individuals whose reported health status does not change within the timeframe studied contribute no information to the conditional maximum likelihood, these individuals are automatically dropped during the model estimation process. The resulting sample size is 45,412 person-years with an average of 10 years of observations per person. Table 5.3 gives descriptive statistics for 1997 cross-section as a whole, and by neighborhood poverty status.

As previously mentioned, the dependent variable is a binary measure of poor respondent-rated health. Adjustments are made for time varying socioeconomic factors which include martial status, age, education, female headed household indicator, welfare recipient indicator, labor force status, and family needs ratio. To mitigate the effects of reverse causation, measures of welfare receipt, labor force status and family needs ratio reflect conditions for the previous year (t-1). Finally, a complete set of year dummies is included to adjust for unobserved factors that are particular to each year. Several specifications of neighborhood poverty are explored: linear, log, and categorical.

[28] I also run linear probability fixed-effect models and the results were consistent with the conditional fixed-effect logistic models.

Table 5.3. 1997 PSID Cross-Section Descriptive Statistics

Characteristics	Full Sample		Low Poverty Neighborhood		High Poverty Neighborhood	
	N	%	N	%	N	%
Sample Size	8,313	100.00	6,700	80.60	1,613	9.40
Gender						
Female	4,604	44.62	3,603	53.78	1,001	62.06
Male	3,709	55.38	3,097	46.22	612	37.94
Mean Age	43.61		46.44		45.53	
Race						
White	5,921	71.23	5,474	81.70	447	27.71
Black	2,392	28.77	1,226	18.30	1,166	72.29
Socioeconomic Factors						
Female Headed Household	664	7.99	349	5.21	315	19.53
Welfare Recipient	225	2.71	86	1.28	139	8.62
Family Income/Poverty Ratio						
100% Or Less	771	9.27	368	5.49	403	24.98
100% to 200%	1,475	17.74	966	14.42	509	31.56
200% to 300%	1,870	22.49	1,420	21.19	450	27.90
Over 300%	4,940	59.42	4,440	66.27	500	31.00
Marital Status						
Single	665	8.00	384	5.73	281	17.42
Married	6,090	73.26	5,186	77.40	904	56.04
Other	1,558	18.74	1,130	16.87	428	26.53
Education Level						
<12 Years	1,568	19.15	1,014	15.36	554	34.97
12 Years	2,885	35.24	2,308	34.95	577	36.43
13 to 15 Years	1,891	23.10	1,610	24.38	281	17.74
16 Years	1,109	13.55	1,017	15.40	92	5.81
17+ Years	734	8.97	654	9.90	80	5.05
Labor Force Status						
Employed	5,669	68.83	4,730	71.21	939	58.91
Not Employed	284	3.45	173	2.60	111	6.96
Not in the Labor Force	2,283	27.72	1,739	26.18	544	34.13
Residential Mobility						
Head Moved Last Year	1,199	14.42	955	14.25	244	15.13
Neighborhood Context						
Mean Neighborhood Poverty Rate	14.87		8.80		30.29	
1997 Health Status						
NonPoor	7,036	84.64	5,846	87.25	1,190	73.78
Poor	1,277	15.36	854	12.75	423	26.22

Table 5.4. OLS and Fixed-effect Results[±]

Neighborhood Poverty Specification	No Fixed-effect	Fixed-effect
	OR	OR
Continuous Measures of Neighborhood Poverty		
Linear	1.17***	1.06**
Log Poverty	1.37***	1.10**
Categorical Measures of Neighborhood Poverty		
Neighborhood Poverty Rate: Below 20%	1.00	1.00
Neighborhood Poverty Rate: Above 20%	1.44***	0.977
Neighborhood Poverty Rate: 0 to5%	1.00	1.00
Neighborhood Poverty Rate: 5 to10%	1.27**	1.16*
Neighborhood Poverty Rate: 10 to20%	1.55***	1.19*
Neighborhood Poverty Rate: Above 20%	1.97***	1.14

[±] ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level, *Statistically Significant at the 10% Level
Coefficients for the linear and long measures of neighborhood poverty represent a hypothetical 10 percentage point change.

Table 5.4 show the neighborhood effects estimates from the fixed-effect models, along with results from a representative cross-sectional logistic regression models based on 1997 data. Estimates from the logistic regression models,[29] having not been adjusted for unobserved individual heterogeneity, are consistently significant and larger than the fixed-effect estimates. For example, the cross-sectional model suggests that a 10 percentage point increase in neighborhood poverty corresponds to 17 percent increase in the probability of a high poverty health rating. In contrast, the fixed-effect estimate suggests a much more moderate impact; a 10 percentage point increase in neighborhood poverty is associated with only a 6 percent increase in the likelihood of reporting high poverty health. Fixed-effect estimates for the categorical specifications are not significant, probably due to insufficient variation across categories within a person.

The critical assumption in the fixed-effect modeling strategy is that the sources of neighborhood poverty variation are uncorrelated with unobserved time-varying factors that affect health. There are two sources of neighborhood variation in the sample: changes in neighborhood context over time due to neighborhood gentrification or socioeconomic deterioration and changes in neighborhood context due to residential moves. Table 5.5 reports the neighborhood poverty variation for movers and non-movers and indicates that

[29] Non fixed-effect logistic models are based on 1997 health outcomes and socioeconomic characteristics reflecting 1996 measures.

the bulk of neighborhood variation is due to individuals who moved at least once during the time period of observation. Given that over a third of the sample moved, this source of neighborhood variation is a concern if residential moves correspond to changes in individual characteristics that also affect health. Changes such as marriage, employment, and family income are accounted for in the model and therefore do not pose a great threat. However, neighborhood moves due to, for example, unobserved declining health or changes in attitudes towards quality of life may be problematic.

Table 5.5 Variation in Neighborhood Poverty

Population	Average Within-Person Standard Deviation of Neighborhood Poverty Rate
Total Sample	1.87
Movers	2.19
Non-Movers	0.63

According to respondents who had moved during the previous year, the two most common reasons for moving were for residential improvement: to purchase a home or to acquire more space and a better home (Table 5.6). However, nearly 16 percent of moves were due to divorce, retirement, or health reasons[30]. Fourteen percent of those in this category had an observed health status change from good to poor in the two years preceding their move. Even if the stated reasons for moving were not directly related to health, to the extent that the move may also correspond with attitudinal changes that may affect health (e.g., the motivation to make a fresh start in a new house may coincide with a renewed commitment to engage in a more active, healthier lifestyle), the impetus for moving may be correlated to health outcomes. Therefore, to test the sensitivity of the fixed-effect neighborhood estimate to unobserved changes that coincide with residential moves, I account for residential moves in the next set of model specifications (Table 5.7). Model 6 includes a dummy for moved/no move while Model 7 breaks the indicator for residential move into moves for voluntary reasons (e.g., closer to work, bigger house) and involuntary reasons (e.g., divorce, retirement, health). The inclusion of residential moves in the set of controls does not significantly alter the estimates of the impact of neighborhood poverty on health.

[30] The PSID did not separate moves due to health from those due to divorce or retirement.

Table 5.6. Reasons for Moving

Reported Reasons for Moving	%
New Job, Nearer Work	12.75
More Space, Better House	19.97
Less Space, Less Rent	6.71
Got Married, Own House	21.68
Better Neighborhood, To Be Near Friends/Relatives	7.27
Involuntary: Got Divorced, Retired, Health, etc	15.67
Other Reasons: Mixed, Ambiguous	15.95

Number of Moves=24639

Results of the fixed-effect models do suggest that, even after accounting for unobserved non time-varying person-specific confounders, neighborhood poverty does have a negative impact on respondent-rated health.[31]

Table 5.7 Fixed-effect Results with Adjustments for Residential Moves±

Neighborhood Poverty Specification	No Adjustment for Residential Moves	Adjustment for Residential Moves	Adjustment for Involuntary/Voluntary Residential Moves
	Model 5	Model 6	Model 7
	OR	OR	OR
Continuous Measures of Neighborhood Poverty[32]			
Linear	1.06***	1.05***	1.06***
Log Poverty	1.10***	1.09***	1.10***
Categorical Measures of Neighborhood Poverty			
Neighborhood Poverty Rate: Below 20%	1.00	1.00	1.00
Neighborhood Poverty Rate: Above 20%	0.98	0.97	0.97
Neighborhood Poverty Rate: 0 to5%	1.00	1.00	1.00
Neighborhood Poverty Rate: 5 to10%	1.16**	1.16**	1.16**
Neighborhood Poverty Rate: 10 to20%	1.19**	1.19**	1.19**
Neighborhood Poverty Rate: Above 20%	1.14	1.11	1.11

± ***Statistically Significant at the 1% Level, **Statistically Significant at the 5% Level,
*Statistically Significant at the 10% Level
Coefficients for the linear and long measures of neighborhood poverty represent a hypothetical 10 percentage point change.

[31] I also examine the sensitivity of neighborhood effect estimates due to attrition (either due to deaths or dropping out of the survey) by shortening the time frame of analyses and restricting the age range to younger individuals. Estimates of neighborhood effects remained relatively stable, albeit with decreasing precision of the point estimates as sample size decreased.

[32] Continuous measures of neighborhood poverty are on a scale of 1 to 10. This yields an interpretation of, for the case of the linear model as an example, a 17% increase in the odds of reporting poor health for 10 percentage increase in neighborhood poverty rate.

Discussion

The propensity score estimate (OR=1.67) is substantially larger than the fixed-effect model estimate (OR=1.05). However, the two estimates are not directly comparable as the propensity score estimate reflects an average threshold effect at the 20 percent level while the fixed-effect model assumed a linear relationship and reflects a 10 percent point change in neighborhood poverty rate.

A rough proxy for the difference in poverty rates between the two categories in the propensity model would be to take the difference in the mean of each category. The average poverty rates within the low poverty and high poverty neighborhoods are 8.55 percent & 31.74 percent, respectively. Hence, the 63 percent increase in the odds of reporting poor health in the propensity model correspond roughly to a 23 percent increase in the neighborhood poverty rate. For an equivalent change in neighborhood poverty rate in the fixed-effect model, this would equate to approximately an increased odds of 12 percent for reporting poor health. The propensity score estimate is still quite a bit larger than the fixed-effect estimate.

The relative magnitude of the propensity score and fixed-effect estimate is not surprising, given that there are reasonable justifications to believe that the fixed-effect model approach yields a downward bias of the effect of neighborhood poverty on health. As other neighborhood-health researchers have noted (e.g., Robert 1999b; Duncan et al. 1997), estimates of neighborhood effects, despite criticisms of under-adjusting for unobserved confounders, may generally have been underestimated. There are several methodological limitations in the conventional neighborhood-health modeling approaches that suggest that the estimates are biased towards zero. First, the conventional usage of single census tracts to define neighborhood boundaries ignore the impact of surrounding communities and importance of areas frequently visited by individuals (e.g., for work, shopping, worship, etc.) that are outside the census tract of residence. Recent work suggests that estimates of neighborhood effects that considered only the tract of residence may have underestimated the total effects of neighborhoods on health (Inagami et al. 2006; Finch et al. 2006). Second, as discussed in Chapter 4, estimates of neighborhood effects based on cross-sectional data fail to consider the impact of length of exposure to specific neighborhood context. Under the assumption that the impacts of neighborhood context are stronger for those with longer exposure times, single-point-in-time measures of neighborhood context may be noisy

proxies for long-term exposures as there is no distinction between residents with long versus short-term exposures to their current neighborhood context. Results in Chapter 4, suggest that estimates of neighborhood effects based on multiple year measures of neighborhood context are larger than those based on a single year measure. Third, given that neighborhood context shapes and influences socioeconomic attainment and health behaviors, the adjustment of these individual characteristics as confounders in neighborhood-health models allow only estimates of independent (or direct) effects of neighborhood context. This precludes estimates of the total (both direct and indirect) impact of neighborhood context on health, thereby underestimating the overall magnitude and importance of neighborhood context on health. As such, the resilience of the neighborhood poverty estimate to unobserved heterogeneity is even more remarkable considering that the majority of the socioeconomic controls included in the model may be considered as mediating factors through which neighborhood poverty impacts health.

The significant results from both the propensity score and fixed-effect approaches do suggest that there is a causal link between neighborhood poverty and health outcomes.

Chapter 6: Summary and Policy Implications

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Summary

In this dissertation, I investigated the intersections of place, health, and racial health disparity. In Chapter One, I investigated the impacts of metropolitan-level racial and economic residential segregation on health disparities between Blacks and Whites. In Chapter Two, I extended conventional cross-sectional analyses of socioeconomic and neighborhood context models by including a temporal dimension that recognized the dynamic nature of both of these determinants of health. In Chapter Three, I applied statistical strategies that focused on recovering the causal effects of neighborhood poverty on health by addressing issues of endogenity and covariate over adjustment.

Through quantitative analyses that improved on the methodological weaknesses in the current neighborhood-health literature, the overarching goal across each of these chapters was to strengthen the understanding of how and to what extent neighborhood context impacts health and racial health disparities. Results provided evidence that:

- Racial residential segregation detrimentally affects the health of Blacks, even after adjustment of individual socioeconomic factors. These associations are more strongly linked with previously unexplored dimensions of concentration, clustering, and centralization.
- Economic residential segregation also detrimentally affects the health of Blacks, with magnitudes of point estimates comparable to or larger than those of racial segregation effects.
- The effects of racial and economic segregation on health for Whites are either neutral or beneficial, suggesting that segregation provides protective environments for Whites, to the detriment of Blacks.
- Single-point measures of neighborhood context underestimates the connection between neighborhood environment and health, although only moderately.
- Multiple-year measurements of individual-level and neighborhood-level socioeconomic factors lead to substantial reduction in the magnitude of the Black/White health gap that was previously unexplained by adjustments of single-point socioeconomic measures.

- A larger source of bias in estimating racial health disparities is model misspecification.
- Higher levels of neighborhood disadvantage are causally linked with detrimental health outcomes.

Policy Implications

Williams and Collins (2001) argue that racial residential segregation is the fundamental cause of racial disparities in health. To truly understand Williams and Collins' claim, the historical context in which segregated communities were originally created and enforced in America must be taken into account. It is not the racial composition of an area per se that so severely undermines the health of its residents; rather, it is how segregation served, through its historical discriminatory housing and labor practices, to isolate Blacks into areas of concentrated poverty. There is very little doubt that segregation played a critical role in the formation of ghetto poverty and emergence of the "underclass" (Massey 1990; Massey & Eggers 1993; Jargowsky 1997). Focusing on segregation (as opposed to the multiple pathways in which segregation can act) in Chapter 3 underscores the role of historical discrimination, resulting from both federal and private initiatives and actions, to segregate communities and thus foster areas of concentrated poverty. In a time of receding funds for social welfare, the recognition that the federal government is in part responsible for the "urban health penalty" (Greenberg 1991) may provide additional motivation to direct limited resources to improving the conditions of inner-city residents. The connection between residential segregation, detrimental health outcomes, and racial health disparities may be a negative finding, but it can also guide public health policies. Since segregation is an upstream determinant that critically influences the formation and distribution of risk factors through multiple pathways, evidence for the connection between segregation and health may increase the recognition and acceptance that the social and structural environments impose both constraints and opportunities which influence individual choices and behaviors that directly and indirectly impact health and other outcomes.

The results from Chapter 3 also suggest that neighborhood poverty is a mediating mechanism between residential segregation and health outcomes. The racial health gap between Blacks and Whites stem from the opposing effects of economic segregation across races. Most likely through the concentration of poverty in Black neighborhoods, economic

segregation has salutary health effects for Whites and detrimental health effects for Blacks. Hence, although racial segregation may be decreasing, the increasing economic segregation in American cities, where the affluent are separated from the poor, pose challenges to reducing racial health disparity. The increasing polarization of American cities by income may have both social and health consequences as residents in affluent neighborhoods enjoy protective effects while residents in poor neighborhoods suffer detrimental effects. Moreover, because minorities are disproportionately represented in the most disadvantaged neighborhoods, strategies to eliminate racial disparities in health and improve population health must expand their efforts to include public policies that may not conventionally be viewed as health policies. Because urban context matters for health, agencies such as the EPA, HUD, and the Department of Transportation must consider the possible health impacts, as well as environmental and economic impacts, when designing and developing the urban environment. This includes both the direct impacts (e.g., increased pollution) as well as the indirect impacts (e.g., changes in the distribution of exposure to risk factors through zoning laws).

Results from Chapter 5, suggesting causal neighborhood effects, underscore the potentially immense impact of housing policies and urban design in reducing racial health disparity. Moreover, given that results from Chapter 4 indicate that the differential distribution of socioeconomic conditions, both at the individual and neighborhood level, plays a greater role in generating the Black/White health gap than currently thought, health policies should augment efforts to target the fundamental causes of these differential distributions.

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