

## Is Segregation Bad for Your Health?

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For decades, racial residential segregation has been observed to vary with health outcomes for African Americans, although only recently has interest increased in the public health literature. Utilizing a systematic review of the health and social science literature, the authors consider the segregation-health association through the lens of 4 questions of interest to epidemiologists: How is segregation best measured? Is the segregation-health association socially or biologically plausible? What evidence is there of segregation-health associations? Is segregation a modifiable risk factor? Thirty-nine identified studies test an association between segregation and health outcomes. The health effects of segregation are relatively consistent, but complex. Isolation segregation is associated with poor pregnancy outcomes and increased mortality for blacks, but several studies report health-protective effects of living in clustered black neighborhoods net of social and economic isolation. The majority of reviewed studies are cross-sectional and use coarse measures of segregation. Future work should extend recent developments in measuring and conceptualizing segregation in a multilevel framework, build upon the findings and challenges in the neighborhood-effects literature, and utilize longitudinal data sources to illuminate opportunities for public health action to reduce racial disparities in disease.

demography; ethnic groups; health status disparities; public health; social isolation; socioeconomic factors; United States

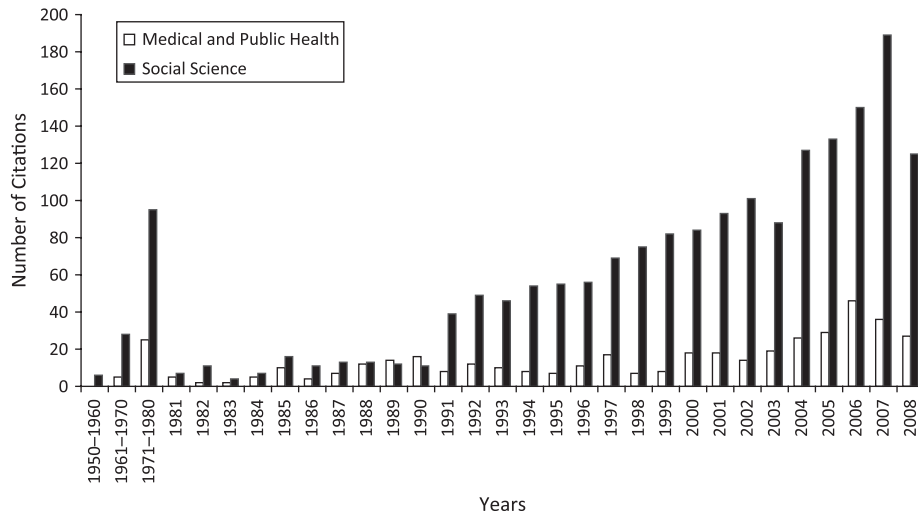
### INTRODUCTION

In 1950, Dr. Alfred Yankauer observed that the infant mortality rate for black babies and white babies in New York City increased as the concentration of blacks in the mother's neighborhood of residence increased (1). While social scientists had been observing racial and ethnic residential settlement patterns for some time before this, Yankauer was the first to link racial residential segregation with population health. Only in the past 15 years has public health interest rekindled in considering whether segregation can explain longstanding racial and economic disparities in health, as evidenced by the growth in the number of publications in biomedical and social science journals (Figure 1).

David Williams has called segregation a "fundamental cause" of health disparities because of the manner in which it differentially sorts individuals into social and economic environments on the basis of race and class (2). Widespread health disparities in the United States remain difficult to explain (3, 4), and thus far difficult to ameliorate, making claims of a "fundamental cause" appealing. This review assesses the evidence for this claim by considering 4 broad

questions of interest to epidemiologists: How is segregation best measured? Is the segregation-health association socially or biologically plausible? What evidence is there of segregation-health associations? Is segregation a modifiable risk factor?

With an eye toward these 4 areas of measurement, mechanism, association, and policy implications, we searched MEDLINE, CINAHL, EMBASE, ERIC, PsycINFO, and the Web of Science databases in September 2008 using variants of the term "residential" combined with variants of the term "segregation," resulting in 2,564 citations. Important literature exists (and more is needed) to enable understanding of economic segregation, segregation of Hispanics and Asians, and the role of segregation in rural areas, but these topics were not the subject of this literature review. The largest body of literature concerns health and the residential segregation of blacks and whites in metropolitan areas of the United States; we therefore focused primarily on these studies and reports. A brief review of the history of black-white segregation in the United States precedes attention to the 4 questions structuring this paper.



**Figure 1.** Numbers of “segregation” and “health” citations in the MEDLINE, CINAHL, EMBASE, ERIC, PsycINFO, and Web of Science databases as of September 2008.

## HISTORY OF SEGREGATION IN THE UNITED STATES

Residential segregation is the degree to which groups of people categorized on a variety of scales (race, ethnicity, income) occupy different space within urban areas, and the process that creates this differential spatial distribution (5). Segregation in US cities is neither new nor unique to any one ethnic or racial group. New European immigrants to US urban areas frequently resided in relatively homogenous ethnic enclaves, a process that may be a critical component of assimilation (6). This segregation of new immigrants typically subsides within a generation as economic opportunity and upward mobility lead to fuller integration. Yet, for black Americans, segregation increased throughout much of the 20th century.

Cutler et al. (7) portray black-white residential segregation in the 20th century in 3 distinct periods. The first, the *birth of the ghetto*, spanned from 1890 to 1940 (Cutler et al. distinguish “ghetto” as a largely black, segregated area, as opposed to a slum, which denotes quality of living conditions). Large-scale migration of rural southern blacks to urban areas in the Northeast was driven by changes in agricultural practices in the South and demand for manual labor in the industrial North. While the average urban black in 1890 lived in a neighborhood that was 27% black, by 1940 this black percentage had increased to 43% (7). This period of increasing racial density parallels the pattern of any new immigrant group and results largely from an affinity of newcomers to live near other newcomers. Congregating in neighborhoods offered opportunity for job leads, connections to cultural and religious institutions, and social support. Limited evidence of housing markets during this period suggests this early segregation may have been driven as much by black choice as by structured limitation to other options.

The period from 1940 to 1970 was one of consolidation and expansion of the urban black ghetto. While further mi-

gration of rural blacks into southern and northern urban areas continued to expand the size of the black population in many cities, racial tensions were increasing (8). In a process Cutler et al. call *collective action racism*, housing markets were manipulated by law, restrictive covenant, and overt acts of intimidation by whites to maintain and increase separation. According to one estimate, 80% of housing deeds in some areas included restrictive covenants regarding race (7, 9). Massey and Denton (10) argue that it was this period of sanctioned and institutionalized racism, which they likened to South-African apartheid, that formed the segregation that persists to today. By most measures, black-white residential segregation peaked in 1970, when the average urban black person lived in a neighborhood that was 68% black (7).

Since 1970, segregation has decreased modestly. This trend resulted mostly from the movement of some black families to previously all-white areas rather than from the integration of largely black areas. While segregation may have decreased overall, the results of previous decades have persisted in terms of isolation and poverty concentration for many urban black residents. There are some areas (particularly in the South and West) in which middle-class black families have integrated into white neighborhoods, while poor black families have become increasingly isolated physically and economically in areas that suffer from infrastructure disinvestment (11).

The Civil Rights Act (Fair Housing Act) of 1968 prohibited discrimination in housing sales and rentals and thus theoretically stopped the collective-action racism that shaped segregation during the preceding decades. Nevertheless, ongoing financial and interview audit studies demonstrate that redlining (the illegal process of systematically denying loans to certain portions of a city) and outright racial discrimination persist today in shaping urban housing markets (12). Cutler et al. (7) argue that, in this third historical period from 1970 forward, *decentralized racism* became the operative process maintaining segregation.

During this phase, white avoidance (due to racism or to escape from economically crumbling central cities) may be most operative in maintaining segregation, as evidenced by whites paying more for comparable housing in predominantly white neighborhoods and survey data that blacks more than whites desire greater residential integration (64% of blacks vs. 40% of whites in 2007) (13).

This brief review of the history of 20th-century segregation in the United States points out 2 important features relevant to the health researcher. First, the black-white urban residential segregation seen today is distinct from any other ethnic or group segregation in the United States, and perhaps elsewhere in the world, and therefore reflects a process of social stratification that is both historically situated and uniquely American. Second, because the mechanisms driving segregation varied over time, the health implications of segregation may vary temporally as well. For example, the health effects associated with the early 20th century ethnic enclaves of new black immigrants to the North were likely different from those experienced by residents of the hyper-segregated cities in the latter 3 decades of the 20th century, where poverty concentration and infrastructure decay dominate.

## MEASURING RESIDENTIAL SEGREGATION

The nonrandom clustering of social groups in space is not inherently good or bad. To affect population health, segregation must have more to it than the departure from random distribution of housing, and yet it is precisely this patterning of residents that is typically measured in segregation indices. One challenge in conceptualizing segregation is that its social and health-relevant effects are often described in terms of the *process* of segregation—a series of forces that differentially allot individuals into residential environments and economic opportunities on the basis of race (10, 14)—as opposed to the *condition or state* of segregation, which is the description of spatial residential patterns at a point in time (15). Many studies essentially estimate the degree to which measuring the *state* approximates what we believe the *process* to be.

The crudest measure of segregation (used by Yankauer (1) in 1950, and by many investigators currently) is the proportion of a group (e.g., whites) in a given neighborhood (e.g., census tract), often termed neighborhood racial composition. It is easily operationalized and appears intuitive to the reader, but it says nothing about the distribution of people in space, is invariant to population density in a neighborhood, and does not specify a reference against which to measure the neighborhood (e.g., a neighborhood that is 30% black means different things in a city that is 1% black and one that is 30% black). In other words, the composition of a given neighborhood is independent of the residential patterning of the larger city.

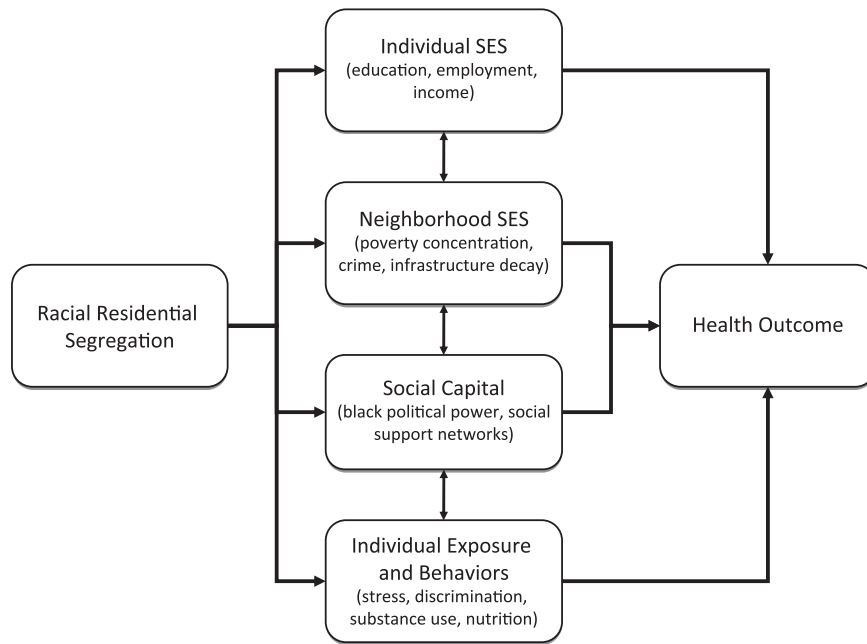
For these reasons, most social scientists utilize measures that acknowledge 2 scales of geography: subareas (e.g., neighborhoods) situated within larger overall geographic areas (e.g., cities or metropolitan areas). Segregation is then expressed as a comparison of the subareas to the overall area, commonly in the form of a population-weighted aver-

age across all neighborhoods. Although central cities, counties, and even states have been used as the larger geographic context, the Metropolitan Statistical Area could arguably be the best larger context in which to situate neighborhoods. The notion of sorting individuals into residential environments is largely a function of housing markets, and the consequences of segregation discussed below are largely related to labor markets. Metropolitan Statistical Areas are units constructed by the Office of Management and Budget to define counties clustered around a central city defined by their degree of economic integration (16); in other words, one goal of the Metropolitan Statistical Area definition is to describe a discrete regional housing and labor market (17).

Segregation is most commonly conceived of as having 5 dimensions described by Massey and Denton (5): evenness (the degree to which groups are evenly distributed in space), isolation (the probability for interaction between members of same vs. different racial groups in a given neighborhood), concentration (the spatial density of a minority group in an area), centralization (the degree to which a group is primarily located in the city core), and clustering (the grouping of racially similar neighborhoods in space). Numerous indices are intended to proxy each dimension, but 2 frequently encountered in the segregation literature are the dissimilarity index—which measures evenness—and the isolation index (sometimes represented as  ${}_xP^*_x$ ). The dissimilarity index can be interpreted as the proportion of the minority group that would have to move to another neighborhood to achieve complete integration. This index ranges from 0 (complete integration or evenness) to 1 (complete segregation). The isolation index, on the other hand, measures the probability that 2 individuals randomly drawn from the same neighborhood are of the same race, essentially estimating exposure or isolation of one group to another. It also ranges from 0 (complete exposure) to 1 (complete isolation).

While the indices discussed by Massey and Denton (5) dominate segregation research, critiques of the measures exist (18). One important criticism is the reliance on census tracts as proxies for neighborhoods. Although tracts have been demonstrated to be reasonable small-area approximations for understanding health disparities (19, 20), in the case of the inherently spatial concept of segregation, tracts may be too arbitrary in terms of their boundaries and scale (21–23). Specifically, mechanisms by which segregation impacts social and health outcomes may be operative at a different scale than is represented by the census tract.

Several investigators have proposed newer “spatial” versions of the traditional indices that utilize geographic information system software to estimate segregation by varying neighborhood scales without relying on the tract boundaries (21, 23–26). Approaches have included creation of new indices as well as extending traditional indices such as the isolation index and dissimilarity index to account for scale and spatial orientation of one neighborhood to the next. Early results demonstrate that both the absolute estimation of segregation and the relative ranking of cities change when segregation is measured at different scales (23, 27), suggesting potential misclassification of exposure if the scale measured is not conceptually wed to the intended hypothesis.



**Figure 2.** Possible pathways between segregation and health. SES, socioeconomic status.

One interesting result of eliminating the arbitrary reliance on census tracts as the default scale of neighborhoods is that the 5 dimensions of segregation collapse into 2 (18, 21). Reardon (21) argues that the distinction between evenness and neighborhood clustering is simply a matter of the scale at which measures are calculated. Similarly, centralization and concentration can be seen as special cases of the general spectrum of evenness versus clustering of households, which results in spatial evenness and spatial isolation as 2 general dimensions of residential segregation. Few studies have utilized a spatial measure of segregation (28, 29), and it remains to be seen whether these measures will prove to be meaningfully different tools for understanding the association between segregation and health.

Although most studies identified used segregation measures for 2 groups, indices do exist for alternate conceptualizations. With increasingly multicultural cities, researchers' interest may lie in the residential patterns of multiple racial and ethnic groups simultaneously or the black-white patterns in the context of other groups (30). Multigroup segregation indices are logical extensions of the 2-group indices mentioned above (31, 32). Segregation can also be measured along an ordinal scale, as would be the case for understanding segregation across levels of family income (33, 34).

#### POTENTIAL SOCIAL/BIOLOGIC PATHWAYS FROM SEGREGATION TO POPULATION HEALTH

A primary concern of epidemiologists regarding any exposure-disease relation is its biologic (or social) plausibility: through what causal pathway could an association be mediated? Four interconnected mechanisms (Figure 2) are com-

monly hypothesized: 1) residential segregation begets individual socioeconomic status, which itself is related to health; 2) segregation perpetuates and reproduces unhealthy neighborhood environments; 3) segregation modifies social capital for a city overall or for specific racial groups within a city; and 4) segregation modifies individual risk behaviors or exposure to stressful stimuli (2, 35–37). Segregation is often construed to affect minority communities differently from majority communities, and, as such, these mechanisms relate to possible mechanisms for varying racial disparities in health. Evidence for each mechanism is reviewed in turn below.

#### Segregation and individual socioeconomic status

A leading hypothesis is that the toxic effects of residential segregation are due in part to the association of racial segregation with economically related consequences (11). Strong evidence exists for an interaction of racial and economic segregation through spatial concentration of minority poor people in urban areas. The majority of poor people in the United States are white, yet most poor whites live in economically integrated neighborhoods. In contrast, most poor blacks live in poor neighborhoods (38). The propensity for poor blacks to live in high-poverty neighborhoods has been termed “double jeopardy” (39). In the United States in 2000, 1.4% of white children lived in poor families inside poor neighborhoods, while 16.8% of black children experienced this double jeopardy. The average black child spends 50% of his or her first 18 years of life in high-poverty neighborhoods, while the average white child spends 5% (40).

One of the most direct consequences of this spatial concentration of the poor is reduced educational opportunities,

because school options are primarily a function of neighborhood of residence. If all schools were equal, this issue would be inconsequential, yet there is substantial evidence that poor urban schools perform worse than suburban schools on nearly all markers of quality, including curricular variety, test scores, teacher and administrator experience, high school completion, and the social environment including violence, drugs, and teen pregnancy (2, 41, 42). In a study on the racial gap in Scholastic Aptitude Test (SAT) scores, metropolitan-level segregation explained one quarter of the gap (approximately 45 points) (43). Growing up in more-compared with less-segregated neighborhoods negatively affects adult educational attainment (44) and may influence the academic performance of those who enter college (45).

One approach to estimating the causal component of segregation on the education gap is to use an instrumental variable analysis, in which a variable causally associated with segregation, but unlikely to be associated with education, is substituted in models. Cutler and Glaeser (46) used number of rivers in a metropolitan area as an instrumental variable for segregation, acknowledging the likely causal manner in which topographic features such as rivers reduce intracity migration and increase segregation. They found that segregation measured with the dissimilarity index was negatively associated with adults having obtained high school and college degrees, and that the pattern of association held when segregation was instrumented by rivers.

Segregation also reduces employment opportunities and lowers income through a spatial mismatch of workers and jobs. When education and skills are controlled for, black residents in highly segregated cities are more likely to be unemployed than black persons living in less-segregated cities (47) and to spend more time searching for jobs (48), and they are less likely to be self-employed (11, 49, 50). These effects are particularly strong for extremes of the isolation and clustering dimensions. A review of urban employment and industry over 40 years concluded that the degree of spatial mismatch was less explanatory of racial disparities in employment than was the overall decrease in urban manufacturing (51). However, another study testing the spatial mismatch theory used employment records of the US Postal Service, which, as an institution, has had large processing facilities geographically fixed in urban centers throughout the post-World War II decades (52). In this analysis, degree of metropolitan segregation was unassociated with racial composition of the US Postal Service workforce in the 1940s–1960s. When centrally located urban jobs declined with the exodus of manufacturing in the 1970s, the degree of city segregation and black employment at central postal facilities became correlated, suggesting that racial segregation in the past 30 years is associated with decreased access to employment opportunity for many black families.

While poor blacks disproportionately experience the negative effects of segregation (53), middle-class blacks may also suffer from residential segregation through limited housing choices (54, 55), limited wealth accumulation (56), and restrictions on upward mobility (57). Middle-class blacks are more likely than poor blacks to live in less-segregated neighborhoods, yet they often do not achieve income-matched parity with whites in terms of neighbor-

hood quality, with blacks living in neighborhoods that are older and have lower tax bases, and living among whites who have a lower mean income than they do (58). In fact, for blacks (compared with Hispanics or Asians), degree of residential segregation is relatively independent of individual socioeconomic status (59).

### Segregation and neighborhood socioeconomic environment

Although segregation may limit individuals' economic opportunities, it could also produce neighborhoods that are in and of themselves unhealthy. This hypothesized pathway links the segregation-health literature with the increasingly rich neighborhood effects-health literature in a manner that may complement each. In recent years, a large body of research has struggled to distinguish between the population health impacts of neighborhood context versus neighborhood composition (60–62). One important criticism of this literature is the limited ability to account for selection into neighborhoods, a plausible confounder of the contextual-health association (63). However, metropolitan-level segregation may serve as one such distal sorting mechanism that accounts for the differential assignment of neighborhood environment.

Segregation may propagate negative social environments in multiple ways. Highly segregated cities suffer from higher levels of violent and property crime (64–66). This association seems particularly true for cities with high income inequality, poverty concentration, and segregation on the isolation dimension, suggesting that social isolation and corresponding economic inequality may be particularly important in this regard (65, 67, 68). Neighborhood health is thereby worsened, not only by risk of victimization but also by alterations to individual behavior and social networks associated with living in a dangerous environment (69, 70). Finally, a combination of increased crime and systematic differences in policing and arrests results in high male incarceration rates in many segregated black urban communities (71, 72). This large-scale incarceration of young black men affects the health of the men but has also been implicated in destabilizing family support systems, thereby impacting the health of women and children (73, 74).

Both isolation and concentration probably enhance the spread of infectious disease such as tuberculosis (75, 76), human immunodeficiency virus (77), and gonorrhea (78) and possibly lead to racially disparate exposure to environmental toxins. There is conflicting evidence on differential toxic exposure, with 2 studies suggesting an increased health risk for poor and minority communities from toxin releases in highly segregated cities (79–81) but 1 study observing racial differences in exposure to toxic hazards that were not explainable by degree of segregation (82).

Also of concern is differential access to local health-relevant resources. Segregated and poor neighborhoods tend to have fewer options for purchasing healthy food and more options for purchasing alcohol (83–87). Simply residing in economically deprived neighborhoods may also be associated with important health outcomes such as obesity and heart disease (88–90). There is mixed evidence regarding whether Medicaid

participation by physicians varies with degree of residential segregation, with 2 studies suggesting it does (91, 92) but 2 others suggesting that the effect is small to none (93, 94).

### Segregation and social capital

Social capital has been defined as the degree of social trust, extent of social networks, and willingness to provide mutual aid and reciprocity between individuals in a given area (95, 96). Kawachi (97) and others have proposed that variations in social capital may explain geographic variations in population health, with a relative health-protective effect of living in a social-capital-rich area. Although Putnam (95) suggests that increased social capital is associated with increased equality, it has been argued that the social capital in a given area does not necessarily cross race or class lines and in some cases may be inversely associated with indicators of racial equity (98).

Whether degree of metropolitan segregation increases or decreases social capital is unclear. There is some evidence that high-isolation segregation and concomitant poverty concentration decrease black social capital and reduce interracial trust (99–101). However, a body of evidence also suggests health-protective effects for blacks who live in racially homogenous ethnic enclaves, a phenomenon attributed to enhanced social support and ties. For example, Bell et al. (102) found that isolation segregation increases risk of low-birth-weight infants but that, when conditioning on isolation, higher clustering segregation reduces risk of low birth weight. It is posited that, given the adverse environment posed by isolation segregation, increased clustering may provide social support and enhance political power for black communities. Laveist (103, 104) suggests that the black political empowerment that sometimes results from high-clustering segregation can counter the negative effects of segregation on health outcomes.

### Segregation and individual behaviors and exposures

It is also plausible that some effect of segregation on health could be mediated through individual-level behaviors and exposures patterned by the socioeconomic and neighborhood environmental effects previously described. While much research has looked at individual behaviors to understand racial disparities in health generally, relatively few studies have considered individual behaviors as mediating variables between segregation and health. Increasing residential segregation is associated with eating less fruit (105) and being less physically active (106). For black women, living in neighborhoods with either relatively low or relatively high segregation has been associated with increased smoking during pregnancy (107). Black women typically smoke less than whites during pregnancy (108), and the authors attribute the increased smoking to influence of the majority group in the integrated neighborhoods and response to stressors in the segregated neighborhoods.

A different category of individual exposure now receiving increased attention is the role of psychosocial stress (109–111)—including adverse life events and perceived racism

(112)—in chronically “weathering” immune and neuroendocrine systems, thereby increasing susceptibility to disease (113, 114). Whereas segregation can be seen as a form of institutionalized racism (115), it may interact with personally mediated racism or discrimination, thus representing a truly individual-level exposure. This hypothesis has perhaps been most widely tested in understanding determinants of pregnancy outcomes, in which increased segregation and perceived racism have been found to increase risk for black women (102, 116, 117).

### Refining causal pathways through the lens of the dimensions of segregation

Each of the 5 dimensions of segregation described by Massey and Denton (5) serves as a proxy for overlapping, but distinct types of residential patterning. To the degree that the dimensions diverge, health researchers have an opportunity to test more refined causal hypotheses for the effects of segregation on health. Going hand in hand with this notion is the expectation that researchers clearly describe a hypothesized causal path by which the effects may be mediated. Different health outcomes are sensitive to different mechanisms; thus, the dimension of segregation that is most relevant is likely to vary by outcome of interest.

For example, Acevedo-Garcia (76) suggests that segregation on the concentration and isolation dimensions may be conducive to the spread of infectious diseases, whereas Dickerson (47) reports that clustering and evenness are most predictive of the degree of black unemployment. Either unevenness or the contiguous clustering of predominantly black neighborhoods could similarly associate with these negative exposures; however, conditional on degree of isolation, they may represent the ethnic enclave effect, which provides social support and increases relative political power (103, 118). As previously discussed, this possibly *protective* effect of one kind of segregation, conditioning on others, suggests a richer complexity that can be leveraged in hypothesizing causal pathways. One other pattern deserving of attention is the hypersegregated area, where segregation is deemed high on all 5 dimensions, possibly incurring a unique health effect not seen in cities considered high regarding only some dimensions (119, 120).

### SEGREGATION-HEALTH ASSOCIATIONS

We identified 39 studies that used quantitative approaches to measuring an association between racial segregation and a health outcome (Table 1). The vast majority demonstrated statistically significant observed associations, although the evidence for a causal association is limited by study designs. Seventeen studies (most conducted before 2000) were cross-sectional ecologic (1, 75, 78, 103, 112, 121–132), 16 studies were cross-sectional multilevel (accounting for various individual-level covariates while acknowledging segregation as an inherently contextual variable) (28, 29, 37, 69, 102, 120, 133–142), 5 utilized follow-up data collected either prospectively or retrospectively (143–147), and 1 utilized a time-series, cross-sectional ecologic design (148). In terms of health outcomes, the majority of the earlier studies

**Table 1.** Population Studies of Racial Residential Segregation and Health, United States

Author, Year (Reference No.)	Study Design (Population)	Measure of Segregation (Geographic Unit (Neighborhood Unit)) <sup>a</sup>	Outcome	Results
<b>Mortality studies</b>				
Hearst, 2008 (142)	Cross-sectional, multilevel (infants born to black mothers in 64 central cities with $\geq 250,000$ population)	Isolation dichotomized at 0.6 into "segregated" or "not segregated" (central city (census tracts))	Infant mortality	Propensity score matched analysis demonstrated no significant infant mortality rate difference in segregated vs. nonsegregated cities
Inagami, 2006 (121)	Cross-sectional, ecologic (black, white, and Hispanic adults in New York City, 1999–2000)	"Black areas" ( $\geq 70\%$ black), "white areas" ( $\geq 70\%$ white); "Hispanic area" ( $\geq 70\%$ Hispanic) (New York City (zip code areas))	All-cause mortality	Mortality lowest for blacks, whites, and Hispanics living in neighborhoods of the same ethnicity
Laveist, 2003 (143)	Prospective cohort (National Survey of Black Americans respondents enrolled in 1979–1980)	Multidimensional segregation index compiled from self-reported segregation in school, work, residence, and church	Survival over 13 years of follow-up	Segregation in 3-level ordinal variable, with increasing segregation-associated mortality; adjusted HR = 1.2 (95% CI: 1.02, 1.41) for 1-unit change in segregation
Cooper, 2001 (122)	Cross-sectional, ecologic (white adults and black adults in 267 MSAs, 1989–1991)	Dissimilarity (metropolitan area (census tract))	Premature mortality (prior to age 65 years)	Increasing dissimilarity associated with premature mortality
Jackson, 2000 (144)	Retrospective cohort (National Longitudinal Mortality Study, enrolled in 1978–1985)	% Black in 1980 in census tract of residence at enrollment	All-cause mortality	2- to 3-fold increased mortality rate for blacks aged 25–44 years living in predominantly black neighborhoods compared with $< 10\%$ black neighborhoods; associations modest to null for older blacks and for whites
Collins, 1999 (112)	Cross-sectional, ecologic (black adults and white adults in cities with $> 100,000$ total population and $> 10\%$ black, 1990)	Isolation and dissimilarity (metropolitan area (census block group))	Age-adjusted all-cause, leading cause, and homicide mortality	Isolation associated with increased all-cause, cancer, and heart disease mortality in blacks; associated with higher cancer rates in white males
Fang, 1998 (123)	Cross-sectional, ecologic (non-Hispanic black adults and white adults, New York City, 1988–1994)	"Black areas" ( $\geq 75\%$ black), "white areas" ( $\geq 75\%$ white) (New York City (zip code areas))	All-cause and cause-specific mortality	Higher mortality for older blacks living in predominantly white areas; higher mortality for whites living in predominantly black areas; lower mortality for blacks living in predominantly black areas
Guest, 1998 (124)	Cross-sectional, ecologic (blacks and nonblacks in Chicago, Illinois, 1989–1991)	Isolation (Chicago (census tract))	Infant and adult mortality	Nonsignificant, positive effect between increasing black isolation and black infant and working adult mortality; no effect for nonblacks
Hart, 1998 (125)	Cross-sectional, ecologic (black adults and white adults aged 25–64 years in 124 MSAs with $> 200,000$ population, 1990–1991)	Dissimilarity (metropolitan area (census tract))	Metropolitan age- and race-adjusted mortality rates	Higher dissimilarity associated with higher black mortality for males and females, but no association for whites
LeClere, 1997 (145)	Prospective cohort (National Health Interview Survey respondents, 1986–1990)	% Black in census tract	Survival over follow-up (maximum 6 years of follow-up)	Increasing concentration of blacks in census tract associated with lower survival for whites and blacks, with moderate dose response

Table continues

Table 1. Continued

Author, Year (Reference No.)	Study Design (Population)	Measure of Segregation (Geographic Unit (Neighborhood Unit)) <sup>a</sup>	Outcome	Results
Polednak, 1996 (148)	Time-series, ecologic (black births and white births in 38 MSAs with $\geq 1$ million population, 1982–1991)	Dissimilarity with MSAs divided into quintiles of segregation (metropolitan area (census tract))	Yearly black infant and white infant mortality rates for 1982–1991	For the highest quintile of segregated cities: higher than average black infant mortality for every year; for the lowest quintile: lower black infant mortality for 1982–1988, but similar black infant mortality risk between the lowest and highest quintiles in 1988–1991; no effect for whites
Bird, 1995 (126)	Cross-sectional, ecologic (black infants and white infants born in 1986–1988 in 34 US states)	Dissimilarity (state (metropolitan area (census tract)))	Infant mortality	Dissimilarity associated with black but not white infant mortality
Laveist, 1993 (103)	Cross-sectional, ecologic (births to black women and white women in MSAs with $>50,000$ population and $>10\%$ black, 1981–1985)	Dissimilarity (metropolitan area (census tract))	Black-white disparity (risk ratio) for infant mortality for each city	Increase in the black-white gap as dissimilarity increases
Polednak, 1993 (127)	Cross-sectional, ecologic (black adults and white adults in 38 MSAs with $\geq 1$ million population, 1982–1986)	Dissimilarity (metropolitan area (census tract))	Black-white adult mortality risk difference	Increasing dissimilarity associated with increasing black-white mortality gap
Polednak, 1991 (128)	Cross-sectional, ecologic (black births and white births in 38 MSAs with $\geq 1$ million population, 1982–1986)	Dissimilarity (metropolitan area (census tract))	Black-white infant mortality risk difference	Increasing dissimilarity associated with larger difference between black infant and white infant mortality
Yankauer, 1950 (1)	Cross-sectional, ecologic (black women and white women delivering in New York City, 1945–1947)	% Black in 318 “residential areas” of New York City	Infant mortality	Increase in black infant and white infant mortality as proportion black in neighborhood of residence increased
<b>Pregnancy outcome studies</b>				
Kramer, 2008 (129)	Cross-sectional, ecologic (black women and white women with livebirths in 168 MSAs, 2002–2004)	Isolation and dissimilarity (metropolitan area (census tract))	Very preterm birth ( $<32$ weeks)	Increased risk of very preterm birth with isolation but decreased risk with unevenness for black women; no effect for white women
Osypuk, 2008 (120)	Cross-sectional, multilevel (black women and white women delivering livebirths in 237 MSAs, 2000)	Hypersegregation defined as highly segregated on $\geq 4$ of 5 dimensions (metropolitan area (census tract))	Preterm birth ( $<37$ weeks)	Increased odds of preterm birth to black women in hypersegregated cities compared with not; black-white disparities also larger in hypersegregated cities
Vinikoor, 2008 (134)	Cross-sectional, multilevel (black women delivering livebirths in Wake and Durham Counties, North Carolina, 1999–2001)	Predominantly black census tracts ( $>75\%$ ) vs. mixed census tracts ( $\leq 75\%$ )	Low birth weight ( $<2,500$ g); preterm birth ( $<37$ weeks)	Income incongruity (living in a higher median income tract than expected based on individual education and marital status) protective against low birth weight and preterm birth in predominantly black neighborhoods but not in mixed tracts
Grady, 2007 (29)	Cross-sectional, multilevel (foreign- and US-born black women delivering livebirths in New York City, 2000)	Local spatial segregation index (New York City (census tract))	Low birth weight ( $<2,500$ g)	Increased isolation associated with increased low birth weight risk for US-born black women after control for individual and neighborhood poverty; for foreign-born women, excess risk explained by individual risk factors

Table continues



Table 1. Continued

Author, Year (Reference No.)	Study Design (Population)	Measure of Segregation (Geographic Unit (Neighborhood Unit)) <sup>a</sup>	Outcome	Results
Masi, 2007 (135)	Cross-sectional, multilevel (black, white, and Hispanic women delivering live singleton births in Chicago, Illinois, in 1991)	Census tracts categorized as <10% black, 10%–90% black, >90% black	Birth weight (continuous); preterm birth (<37 weeks)	No association of racial concentration on birth weight or preterm birth for black women; modest association for white women, with higher risk in predominantly black tracts
Bell, 2006 (102)	Cross-sectional, multilevel (livebirths to black women in 225 US MSAs, 2002)	Isolation and spatial proximity (metropolitan area (census tract))	Birth weight (continuous); preterm birth (<37 weeks); intrauterine growth restriction	Decreased birth weight (68 g) and increased preterm birth for high vs. very low isolation (OR = 1.27); high vs. very low clustering associated with higher birth weight (25 g) and lower preterm birth (OR = 0.86)
Grady, 2006 (28)	Cross-sectional, multilevel (black women with livebirths in New York City, 2000)	Local spatial segregation index (New York City (census tract))	Low birth weight (<2,500 g)	Segregation associated with low birth weight independent of neighborhood poverty and individual SES
Pickett, 2005 (37)	Cross-sectional, multilevel (black women delivering livebirths in Chicago, Illinois, 1991)	Predominantly black census tracts (>90%) vs. mixed census tracts (≤90%)	Low birth weight (<2,500 g); preterm birth (<37 weeks)	Income incongruity (living in a higher median income tract than expected based on individual education and marital status) protective against low birth weight and preterm birth in predominantly black neighborhoods, but not in mixed tracts
Ellen, 2000 (136)	Cross-sectional, multilevel (black women and white women delivering in 261 MSAs with >100,000 population and >5,000 black, 1990)	Dissimilarity and centralization (metropolitan area (census tract))	Low birth weight (<2,500 g)	Increasing segregation associated with increased risk of low birth weight for black women but not white women
Sucoff, 1998 (146)	Retrospective cohort (black female Panel Study of Income Dynamics participants born in 1953–1968)	Neighborhoods (tracts) categorized by racial concentration	Time to teenage premarital first birth	Black girls in highly segregated neighborhoods 50% more likely than black girls in racially mixed tracts to have premarital births before age 20 years
<b>Other health outcomes</b>				
Haas, 2008 (133)	Cross-sectional, multilevel (black, white, and Hispanic adults aged >65 years in the SEER database, 1992–2002)	Isolation (counties (census tract)); categorized into high- vs. low-segregated counties	Early- vs. late-stage diagnosis of primary lung, colorectal, breast, or prostate cancer	Black-white disparity in early-stage diagnosis smallest in high-segregation, low-income neighborhoods
Cooper, 2007 (130)	Cross-sectional, ecologic (blacks in MSAs with ≥500,000 population in 1993)	Isolation and concentration (metropolitan area (census tract))	Injection drug use prevalence	Isolation, but not concentration, associated with black injection drug use prevalence
Do, 2007 (137)	Cross-sectional, multilevel (NHANES III participants, 1988–1994)	% Black or % Hispanic (census tract)	Body mass index	Proportion black and Hispanic in a neighborhood marginally associated with body mass index in black and Hispanic males but not females
Rodriguez, 2007 (147)	Retrospective cohort (black adults and white adults beginning dialysis between 1995 and 2002)	% Black in patient zip code area divided into quartiles	Time to death or kidney transplant	Increased mortality for whites but not blacks as black concentration increased; time to transplantation longer for both blacks and whites living in predominantly black neighborhoods

Table continues

Table 1. Continued

Author, Year (Reference No.)	Study Design (Population)	Measure of Segregation (Geographic Unit (Neighborhood Unit)) <sup>a</sup>	Outcome	Results
Chang, 2006 (138)	Cross-sectional, multilevel (black respondents and white respondents to BRFSS, 2000)	Isolation index (metropolitan area (census tract))	Body mass index (continuous) % with body mass index $\geq 30$ kg/m <sup>2</sup>	One standard deviation increase in isolation associated with a 0.42 increase in body mass index, and obesity OR = 1.14 for blacks; no association for whites
Mobley, 2006 (69)	Cross-sectional, multilevel (uninsured, low-income women enrolled from 5 US states)	Isolation index, although scale of neighborhood and broader area not defined	Body mass index 10-year predicted heart disease risk	No association between segregation and body mass index for any group, and reduced coronary heart disease risk for blacks and Hispanics as segregation increased
Robert, 2006 (139)	Cross-sectional, multilevel (black adults and white adults aged >65 years from the Americans Changing Lives survey and the National Survey of Families and Households, 1986–1988)	Dissimilarity and isolation (counties (census tract))	Self-rated health	No association for whites or blacks between segregation and self-rated health
White, 2006 (140)	Cross-sectional, multilevel (adults in New York City Social Indicator Survey, 1999–2002)	% Minority by zip code area, categorized into tertiles	Self-rated health (fair/poor vs. excellent/very good/good)	Poor self-rated health associated with higher density minority in zip code area (aOR = 1.7, 95% CI: 1.1, 2.7)
Subramanian, 2005 (141)	Cross-sectional, multilevel (black adults and white adults living in MSAs with >100,000 population and >5,000 black, 2000)	Dissimilarity and isolation (metropolitan area (census tract))	Self-rated health (fair/poor vs. excellent/very good/good)	High isolation associated with increased odds of poor self-rated health among blacks but not whites
Fabio, 2004 (131)	Cross-sectional, ecologic (Pennsylvania, 1995–1997)	Gini coefficient of racial unevenness (counties (census tract))	Intentional injury diagnosis on hospital discharge	Increasing segregation (unevenness) associated with increased intentional injury discharge
Skinner, 2003 (132)	Cross-sectional, ecologic (black beneficiaries and white beneficiaries of Medicare, 1998–2000)	Dissimilarity (metropolitan area (census tract))	Rates of knee arthroplasty	Smaller difference in black and white knee arthroplasty for women living in low-segregation cities (rate difference = 0.46/1,000) vs. high-segregation cities (rate difference = 1.05/1,000); no effect for men
Thomas, 2003 (78)	Cross-sectional, ecologic (counties in 14 southeastern US states, 1986–1995)	Dissimilarity and isolation (counties (census tract))	County gonorrhea rates categorized as endemically high or endemically low	Black isolation index >0.2 associated with endemically high counties controlling for proportion black and poverty indicators (aOR = 248, 95% CI: 22, 999)
Acevedo-Garcia, 2001 (75)	Cross-sectional, ecologic (New Jersey adults)	Isolation and concentration (New Jersey (zip code area))	Annual incidence of tuberculosis by race	Higher isolation associated with tuberculosis in blacks, and, to a lesser extent, Hispanics; no association for whites

Abbreviations: aOR, adjusted odds ratio; BRFSS, Behavioral Risk Factor Surveillance System; CI, confidence interval; HR, hazard ratio; MSA, Metropolitan Statistical Area; NHANES, National Health and Nutrition Examination Survey; OR, odds ratio; SEER, Surveillance, Epidemiology, and End Results.

<sup>a</sup> Geographic units for measuring segregation are displayed in terms of the nesting of 2 scales: (macro area (neighborhood subarea)).

focused on infant and adult mortality, while studies of the past decade broadened to include a wide variety of reproductive, infectious, and chronic disease endpoints.

A third of the studies conducted in the past decade relied on racial composition of neighborhoods to define segregation rather than indices that place those neighborhood com-

positions in the context of the wider city. As previously noted, the proportion of blacks in a neighborhood is an incomplete description of residential patterns, although several studies used custom-defined thresholds of proportion black to describe “predominantly black” and “predominantly white” neighborhoods (37, 121, 123, 134,

135), which may coarsely serve as a proxy for citywide segregation.

Of the remaining two-thirds of the studies, the dissimilarity index was the most common measure, although several studies also used the isolation index. Acevedo-Garcia (36) suggests that health researchers have opted for the ease of interpreting the dissimilarity index without appreciating the conceptual complexities represented by differing measures. The dissimilarity index may be the conceptually weakest in terms of understanding ill-health effects of segregation, whereas isolation and concentration may be stronger in patterning unhealthy environments and exposures. In fact, it is segregation measured on the evenness scale that sometimes appears to be health protective (102, 121, 123, 129), although, in most of these cases, it was evenness, controlling for isolation, suggesting that the hypothesized ethnic enclave effect is visible only after conditioning on isolation.

Studies varied greatly in model specifications. Because the hypothesized effect of segregation is mediated through many socioeconomic and behavioral processes, distinguishing between a variable that confounds the association and one that is a mediator is by no means simple. Increasingly, studies of the past few years have explicitly stated the underlying conceptual model tested and have discussed conditioning and interacting effects of individual and neighborhood variables with segregation (28, 29, 37, 69, 102, 107, 120, 130, 134–139, 141, 142, 149).

While there are important geographic variations in white health outcomes, the spatial determinants of health appear to be quite different for whites and blacks (126, 129). With few exceptions (112, 123, 135, 145, 147), the sometimes-profound effects of segregation on black health outcomes are absent for whites in the same cities. When a negative association has been found, it is usually among poor whites living in predominantly black neighborhoods.

### ARE SEGREGATION-RELATED HEALTH EFFECTS MODIFIABLE?

There is an intrinsic benefit in better understanding pathways to population health, regardless of whether the pathways are easily modifiable. However, there is a more pressing public health benefit in focusing on exposures that are at least potentially modifiable. Theoretically, residential segregation is a modifiable exposure, yet effective instrumental attempts to reduce segregation in the United States have yet to be developed.

The Fair Housing Act of 1968 is the only coordinated policy initiative taken to reduce racial residential segregation, and its scope is arguably incomplete (150). There were small, but steady declines in racial residential segregation between 1970 and 2000 (40, 151), although one study noted that black-white segregation declined at the micro level only but that macro segregation has been unchanged (27). Despite possible improvements over 30 years, black Americans remain the most segregated minority group in the United States.

In terms of intervention research on housing or neighborhood choice and health or social outcomes, 2 studies are

frequently cited. The 1976 court decree establishing the Gautreaux Assisted Housing Program in Chicago, Illinois, was in response to a preceding housing discrimination lawsuit against the Chicago Housing Authority claiming systematic discrimination in public housing. The Gautreaux program represents a natural experiment wherein 7,000 housing vouchers were made available to residents of highly segregated Chicago neighborhoods with the requirement that recipients be placed in racially integrated, low- to moderate-poverty neighborhoods (152). Subsequent studies of the placement cohort found that, 10–20 years later, the majority continued to live in lower poverty, less-segregated neighborhoods than prior to placement (153); second-generation boys placed in the suburbs had fewer drug offenses (154); and, among males relocating to areas with higher proportions of college graduates, all-cause and homicide-related mortality was lower (155).

In the federally funded, randomized Moving To Opportunity (MTO) trial, public-housing-eligible families were assigned either housing counseling with a voucher requiring placement in a low-poverty neighborhood, a voucher without geographic restriction, or no voucher (156). While early evidence supported positive economic effects for treatment families (157), those gains may not persist with time (158, 159). In terms of health outcomes, there is some evidence that families that moved to low-poverty neighborhoods experienced significantly improved mental health (160) and lower obesity rates (161), but other health outcomes such as asthma and self-reported health were not different from those of controls.

In separate reviews of housing-mobility programs and health effects, Acevedo-Garcia et al. (162) and Anderson et al. (45) suggest that there is modest support for rental voucher programs as one approach to improve housing safety, reduce risk of crime victimization, and improve adult and child mental health. The paucity of evidence for other outcomes is in large part due to the absence of health outcomes as originally measured indicators in either study.

Research that continues to pursue the effects of public housing policy interventions on population health is important. Another approach is to view the causal chain from segregation to health as a series of opportunities for intervention, with an overarching goal of increasing access to “opportunity neighborhoods” for all (39). This option opens up opportunities to alter negative effects of segregation that could range from assisting in opening housing markets so that moves out of concentrated poverty are possible (45, 153, 162) to addressing characteristics of segregated neighborhoods that are unhealthy, as seen in the built-environment literature (163, 164). One caution, however, is that researchers seeking to remedy the effects of segregation remain cognizant of the distinction between the process of segregation (e.g., institutionalized racism or inequitable access to health-promoting opportunities) and the state or condition of segregation (living near black families or far from white families) (115). It is most probable that any injurious attributes of segregation result from inequity in the process rather than the condition of close proximity to black families (or distance from white families) *per se*.

## DISCUSSION

The weight of the available evidence is that the process of racial residential segregation is associated with generally deleterious health of African Americans, and particularly for poor pregnancy outcomes, but this evidence is limited in many regards. Although segregation and social outcomes have been studied for decades, analyzing segregation as a useful construct in epidemiologic research is still in its infancy. In 2003, Acevedo-Garcia et al. (36) reviewed the state of residential segregation and health research, making 4 recommendations: develop multilevel research designs, expand segregation research beyond black-white to include other ethnic and racial groups, consider interaction between racial and economic segregation, and further develop the conceptual framework in which to understand segregation and health. Much progress has been made in response to these goals, but much work remains in order to understand how to best measure segregation, understand mechanisms by which distal social forces become proximal social and biologic outcomes, and test amenable paths to intervention. Future research should consider several issues.

First, continued development of methodological and conceptual tools to better understand residential segregation is necessary. While many recent studies use multilevel thinking in both conceptual and statistical approaches, clarity is still needed regarding the relevant scale of effect (e.g., of the measured neighborhood), levels of interest, and mediating variables. The interesting findings of variable segregation effects across different dimensions such as clustering and isolation should encourage researchers to further investigate the multidimensional nature of residential patterns and health. Similarly, use of newer spatial indices of segregation as well as multigroup indices may provide more insight than repetitive use of the US Census–derived dissimilarity index. Although this review focuses primarily on racial residential segregation, it is clear that poverty concentration and economic segregation are closely linked with racial settlement patterns. Research considering the interaction of economic and racial segregation is still needed.

Second, the existing segregation-health literature can be decomposed into 2 broad categories: 1) segregation as an exposure in intercity studies and 2) segregation as a local exposure in intracity research. This distinction is extremely important. The body of work using single cities to explore the health effects of segregation is an extension of the neighborhood-effects literature (46); it posits that the neighborhood context in mostly black neighborhoods is different from that in mixed-race or predominantly white neighborhoods and therefore impacts health. This approach has the strength of finer spatial resolution of individuals nested within neighborhoods, but Oakes (63) has argued that counterfactual reasoning in research on neighborhood effects and health outcomes may suffer from unmeasured confounding by forces that select individuals into neighborhoods. In other words, individuals are not randomly assigned to neighborhoods within a city; thus, groups may not be exchangeable.

Intercity research, on the other hand, has tended to look at the average health of residents of metropolitan areas (although typically controlling for individual-level covari-

ates such as age, gender, and risk behaviors) and uses the heterogeneity of segregation across metropolitan areas to make inferences about the impact of segregation net of individual characteristics. This approach posits that living in a city with higher segregation (e.g., greater exposure to institutionalized racism) negatively impacts the health of all black residents, regardless of the racial composition of their neighborhood of residence. While this approach offers a partial solution to the selection problem (e.g., segregation may be one of the previously unmeasured forces that differentially sort individuals), most such studies lose the spatial resolution to know how outcomes varied by neighborhood within metropolitan areas. Further extending the multilevel framework to include individuals nested within neighborhoods nested within an ample number of heterogeneous metropolitan areas could offer promising new insight.

Finally, the segregation-health literature could be greatly enhanced by utilizing longitudinal in addition to cross-sectional study designs. Longitudinal designs could be applied to individuals, neighborhoods, or metropolitan areas, each with different implications. Most of the pathways hypothesized between segregation and health act across the life course, but there is almost no literature that accounts for different levels of cumulative exposure across the life course and very little literature with prospective information on health outcomes. For some poor and minority individuals, living in highly segregated environments may not be a time-varying exposure but rather a life-long constant (38). However, there is evidence that intercity and intracity migration varies by race and economics *and with regard to metropolitan segregation*, suggesting that longitudinal comparisons could be meaningfully made between those living in high- versus low-segregated cities (30). Alternatively, neighborhoods or metropolitan areas could be followed longitudinally to better understand the relation between residential patterning and health. Such time-series approaches might be particularly fruitful in areas characterized by progressive gentrification or decay over time. Similarly, time-series, cross-sectional analysis of health patterns in cities across the decades could document health changes associated with changing segregation.

The vast majority of black Americans live in urban settings, many but not all of which are highly segregated. It is vitally important to understand how much of their health disparities are a result of specific dimensions of segregation and whether these disparities can be reduced either by policies that reduce segregation or interventions that reduce the impact of segregation.

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