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Invited Commentary

Invited Commentary: Residential Segregation and Health—The Complexity of Modeling Separate Social Contexts

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When researching racial disparities in health, residential segregation cannot be ignored. Because of segregation, contextual differences by race are so pronounced that ignoring them may lead to misestimating the effect of individual-level factors. However, given the stark racial separation of social contexts, researching how residential segregation and neighborhood inequality contribute to racial health disparities remains methodologically challenging. Estimating the contribution of neighborhood effects to health disparities would require overlap in the racial distributions of neighborhood environment, for example, in the distributions of neighborhood poverty. Because of segregation, though, the extent of such overlap is extremely restricted. Previous analyses of the 2000 US Census found, on average, only a 24% overlap between the distribution of neighborhood poverty for black children and that for white children in metropolitan areas. Propensity score methods may be 1 useful tool for addressing limited overlap or exchangeability. However, as shown by their application to the segregation and health relation, their use should be informed by a sound conceptualization of the scale of the social exposure of interest, the hypothesized pathways between the exposure and the health outcome, and possible unmeasured confounders.

African Americans; infant mortality; residence characteristics

In this issue of the *Journal*, Hearst et al. (1) contribute a paper on the relation between racial residential segregation and infant mortality. From a methodological perspective, the innovative aspect of this paper is the use—to our knowledge, for the first time—of propensity score analysis in a study of segregation and health. We agree with Oakes and Church that ''the benefits of propensity score methods . . . lie not only with potentially improved effect estimation but with conceptualization and practice as well'' (2, p. 1119). Hearst et al.'s excellent paper helps us illustrate the complexity of conceptualizing (and then modeling) the relation between segregation and health.

RESIDENTIAL SEGREGATION AND RACIAL HEALTH DISPARITIES

Racial residential (i.e., housing) segregation is one of the most pervasive patterns in American metropolitan areas and a powerful engine of racial stratification. Whites and blacks are separated into different residential neighborhoods with markedly different resources to support health and well-being over the life course (3–6). The question of interest to social epidemiologists is whether segregation is associated with health patterns among blacks and with black/white health disparities.

In the last 2 decades, social epidemiologists have increasingly turned to the health effects of ''place'' above and beyond the health effects of individual-level factors, since the latter do not fully account for black/white health disparities (7, 8). Research on place influences on health has largely focused on neighborhoods. However, a focus exclusively on neighborhoods limits our understanding of health disparities. Individual neighborhoods—and their qualities, risks, and resources—are part of metropolitan-area-wide neighborhood distributions. Neighborhoods are influenced by the larger economic and social context (e.g., housing and labor markets) of their metropolitan area (9, 10). For instance, an inner-city neighborhood may have a large proportion of unemployed minorities because job creation happens primarily in the suburban portion of the metro area, while minority neighborhood choices are largely limited to the inner city (11).

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Although this metropolitan context has been well documented in sociology, demography, and urban studies, the health literature remains focused primarily on the health effects of individual neighborhoods. While sociologists and demographers have theorized about and measured segregation in metropolitan areas since the beginning of the 20th century, public health research began to establish a link between segregation and poor health among blacks starting in only the late 1980s. Conceptual and methodological refinements of the relation between segregation and health have come about since 2000 (3, 4, 12–21). The work of Hearst et al. (1) is 1 example.

METHODOLOGICAL IMPLICATIONS OF RESIDENTIAL SEGREGATION FOR ESTIMATING HEALTH DISPARITIES

The social reality of high racial residential segregation complicates quantitative analyses of neighborhoods' contribution to racial health disparities. In an ideal research world, estimating the effect of neighborhood context or residential segregation on health outcomes would require that we randomly assign individuals to different types of neighborhoods (e.g., poor vs. nonpoor) or metro areas (segregated vs. nonsegregated). With the exception of the Moving to Opportunity study, which randomly assigned individuals to receive housing vouchers redeemable in low-poverty neighborhoods (vs. not), attempts to estimate neighborhood or metropolitan-area effects on health have relied on nonexperimental (i.e., observational) data (22).

Observational studies are limited for understanding neighborhood effects because, in our metropolitan areas, the distribution of individuals across neighborhoods is not random. Demographers have used segregation measures to estimate how much the actual distribution of individuals across neighborhoods deviates from a hypothetical random distribution. One of the most commonly used measures of segregation, the dissimilarity index, captures the degree of departure from randomness. If individuals of white race and black race were randomly distributed across the neighborhoods that make up a metropolitan area or city, we would expect the racial composition of each neighborhood to approximately mirror the racial composition of the metropolitan area. The dissimilarity index measures what proportion of blacks (or whites) would need to move across the metropolitan area to approximate such hypothetical random distribution. As of the last available data (2000 US Census), the average dissimilarity index between blacks and whites was 0.64, indicating that, on average, 64% of blacks (or whites) would need to move across neighborhoods to approximate a random distribution (23). This very high level of segregation, or dissimilarity, suggests that our social reality is very distant from one that would approximate an experimental design. The question then is whether, absent experimental studies, there is hope in trying to estimate the effects of neighborhood environment on health—and whether methods are available that would enable us to do a better job of estimating such effects.

This question is not only a methodological one; it also has implications for our ability to estimate the causes of racial health disparities. Estimating the contribution of neighborhood effects to health disparities would require overlap in the racial distributions of neighborhood environment, for example, in the distributions of neighborhood poverty. Because of segregation, the extent of such overlap is extremely restricted. In our analyses of the 2000 US Census, we found limited overlap in the distributions of neighborhood poverty between blacks and whites. Neighborhoods with the highest poverty rates for whites overlapped neighborhoods with the lowest poverty rates for blacks, while the middle part of the distributions for whites and blacks did not overlap at all (6, 24). On average, in metropolitan areas, there is only a 24% overlap between the distribution of neighborhood poverty for black children and the one for white children. In other words, on average, the poverty rates in the lowest-poverty neighborhoods for black children are equivalent to those found in the 24% poorest neighborhoods for white children. This lack of overlap is not due to racial differences in the distribution of family poverty. When we limit our analysis to children living in poor families, we find that, on average, the extent of overlap in the racial distributions of neighborhood poverty is only 26%. The above figures are average for the 100 largest metropolitan areas. However, in many metropolitan areas, there is virtually no overlap in the racial distributions of neighborhood poverty (6, 24).

APPROPRIATENESS OF PROPENSITY SCORE ANALYSIS

Hearst et al. (1) used propensity scores to balance covariates between individuals in segregated and nonsegregated areas in order to improve estimation of the effect of living in segregated areas—the exposure of interest. Although propensity scores are an appealing solution for addressing limited overlap to improve exchangeability, we need to evaluate their appropriateness based on what we know about how segregation operates and how it may impact health.

Operation of segregation at the metropolitan-area level

In conducting research on racial disparities in health, we cannot ignore residential segregation. Because of segregation, contextual differences by race are so pronounced that ignoring them may lead to a misestimation of the effect of individual-level factors.

Thus, we need to understand the scale at which segregation operates. Residential segregation is a metropolitan-area phenomenon and has been conceptualized as such in the demographic, sociologic, and urban planning literatures because metropolitan areas approximate housing and labor markets. A metropolitan area comprises a central city and surrounding municipalities (i.e., suburbs). In US metropolitan areas, the divide between the central city and suburban areas is a major axis of racial residential segregation (9, 11).

By focusing on only the central city portion of a metropolitan area, we may be distorting the picture of segregation's impact on health by introducing a form of selection bias. In general, blacks are substantially more likely than

whites to live in the central city of their metropolitan area than in the suburbs. On average, in the 100 largest metropolitan areas, 23% of the non-Hispanic white population lives in the central city compared with 55% of the non-Hispanic black population. Hearst et al. (1) do avoid the issue of possible race-based selection bias in central cities by restricting their analysis to only blacks. However, across metro areas, there is also considerable variation in the proportion of blacks who live in the central city. For example, in the Atlanta, Georgia, metro area, only 19% of blacks live in the central city compared with 79% in Detroit, Michigan (authors' calculations from the US 2000 Census; not shown). Therefore, in those metro areas in which a large fraction of blacks lives in the central city, we are observing outcomes for nearly the entire distribution of blacks, while, in those areas in which only a relatively small fraction lives in the central city, we may be observing outcomes for a selected population of blacks such as the poorest blacks or those with worse health. Applying propensity scores across the central city portion may balance the distribution of observable covariates across the births that occur within the limits of the central city, but, within each central city, we are observing a truncated birth distribution, and the extent of truncation varies across metro areas. So, selection bias may still be operating.

Life-course and intergenerational health effects of segregation

Whether propensity scores may help us estimate the effect of residential segregation depends largely on the hypothesized pathways between segregation and specific health outcomes, including clear specification of the causal exposure of interest, mediators, and confounders. Similar to other methods used in social epidemiology, propensity scores may facilitate valid estimation of health effects that result from a limited window of exposure (i.e., proximal causes) but may be more limited for modeling health outcomes such as birth weight or preterm birth for which exposures—such as segregation—across the life course or intergenerationally may matter (25, 26).

Although infant mortality in the postneonatal period may be associated with exogenous causes such as accidents, it has also been linked to maternal exposures prior to the birth of the infant, such as smoking in pregnancy, teenage childbearing, and low socioeconomic status (27, 28), as well as to low birth weight and preterm birth (27, 29), which the propensity score method, as used by Hearst et al. (1), forces us to consider as confounders. However, those earlier maternal exposures might be ''where the action is'' with respect to segregation effects. Segregation may result in different distributions of variables, such as education or teenage childbearing (30), that may be on the causal pathway between segregation and health outcomes, for example, infant mortality. The different distribution of covariates in low- and high-segregation areas may be in itself a product of segregation as well as a cause of health disparities. Thus, using propensity scores and restricting the window of the causal exposure may amount to washing out the effect of segregation because of adjustment for variables that some perinatal health researchers may consider mediators.

Operation of segregation through individual- and neighborhood-level factors

Residential segregation is related to both lower socioeconomic attainment among blacks at the individual level and higher exposure to disadvantaged neighborhood environments (19). Since propensity score matching is only as good as the observed confounders that can be modeled, even after matching on individual variables, there still may be an imbalance in neighborhood variables and thus residual confounding. For instance, in highly segregated areas, blacks are more likely than whites to live in poor neighborhoods. Unfortunately, the birth and infant mortality data from the vital statistics do not include information on neighborhood environment. Therefore, in their multilevel analysis of individuals nested in central cities, where the exposure of interest is city-level segregation, Hearst et al. (1) are limited to balancing individual-level covariates between segregated and nonsegregated areas.

In sum, researching how residential segregation and neighborhood inequality contribute to racial health disparities remains methodologically challenging given the stark racial separation of social contexts. Propensity score methods may be 1 useful tool for addressing limited overlap or exchangeability. However, as shown by their application to the segregation and health relation, their use should be informed by a sound conceptualization of the scale of the social exposure of interest, the hypothesized pathways between the exposure and the health outcome, and possible unmeasured confounders. Ultimately, the question is whether our methods, propensity scores or other, enable us to capture a meaningful picture of the complex social reality we are trying to model.

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REFERENCES

1. Hearst MO, Oakes JM, Johnson PJ. The effect of racial residential segregation on black infant mortality. *Am J Epidemiol*. 2008;168(11):1247–1254.
2. Oakes JM, Church TR. Invited commentary: advancing propensity score methods in epidemiology. *Am J Epidemiol*. 2007;165(10):1119–1121.
3. Acevedo-Garcia D, Lochner KA. Residential segregation and health. In: Kawachi I, Berkman LF, eds. *Neighborhoods and Health*. New York, NY: Oxford University Press; 2003: 265–287.

4. Acevedo-Garcia D, Lochner KA, Osypuk TL, et al. Future directions in residential segregation and health research: a multilevel approach. *Am J Public Health*. 2003;93(2): 215–221.
5. Acevedo-Garcia D, Osypuk T. Impacts of housing and neighborhoods on health: pathways, racial/ethnic disparities, and policy directions. In: Carr J, Kutty N, eds. *Segregation: The Rising Costs for America*. New York, NY: Routledge; 2008: 197–235.
6. Acevedo-Garcia D, Osypuk TL, McArdle N, et al. Towards a policy relevant analysis of geographic and racial/ethnic disparities in child health. *Health Aff (Millwood)*. 2008;27(2):321–333.
7. Macintyre S, Ellaway A, Cummins S. Place effects on health: how can we conceptualise, operationalise and measure them? *Soc Sci Med*. 2002;55(1):125–139.
8. Williams D, Jackson P. Social sources of racial disparities in health. *Health Aff (Millwood)*. 2005;24(2):325–334.
9. Briggs XdS, ed. *The Geography of Opportunity: Race and Housing Choice in Metropolitan America*. Washington, DC: Brookings Institution Press; 2005.
10. Galster GC, Killen SP. The geography of metropolitan opportunity: a reconnaissance and conceptual framework. *Housing Policy Debate*. 1995;6:7–43.
11. Altshuler A, Morrill W, Wolman H, et al, eds. *Governance and Opportunity in Metropolitan America*. Washington, DC: National Academy Press; 1999.
12. Grady SC, Ramirez IJ. Mediating medical risk factors in the residential segregation and low birthweight relationship by race in New York City. *Health Place*. 2008;14(4):661–677.
13. Masi CM, Hawkley LC, Piotrowski ZH, et al. Neighborhood economic disadvantage, violent crime, group density, and pregnancy outcomes in a diverse, urban population. *Soc Sci Med*. 2007;65(12):2440–2457.
14. Bell JF, Zimmerman FJ, Almgren GR, et al. Birth outcomes among urban African-American women: a multilevel analysis of the role of racial residential segregation. *Soc Sci Med*. 2006;63(12):3030–3045.
15. Grady SC. Racial disparities in low birthweight and the contribution of residential segregation: a multilevel analysis. *Soc Sci Med*. 2006;63(12):3013–3029.
16. Osypuk TL, Acevedo-Garcia D. Are racial disparities in preterm birth larger in hypersegregated areas? *Am J Epidemiol*. 2008;167(11):1295–1304.
17. Subramanian SV, Acevedo-Garcia D, Osypuk TL. Racial residential segregation and geographic heterogeneity in black/white disparity in poor self-rated health in the US: a multilevel statistical analysis. *Soc Sci Med*. 2005;60(8):1667–1679.
18. Collins C, Williams DR. Segregation and mortality: the deadly effects of racism. *Sociol Forum*. 1999;14:495–523.
19. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep*. 2001;116(5):404–416.
20. Acevedo-Garcia D. Residential segregation and the epidemiology of infectious diseases. *Soc Sci Med*. 2000;51(8): 1143–1161.
21. Acevedo-Garcia D. Zip code level risk factors for tuberculosis: neighborhood environment and residential segregation, New Jersey, 1985–1992. *Am J Public Health*. 2001;91(5):734–741.
22. Acevedo-Garcia D, Osypuk TL, Werbel RE, et al. Does housing mobility policy improve health? *Housing Policy Debate*. 2004;15:49–98.
23. Iceland J, Weinberg D, Steinmetz E. US Census Bureau. *Racial and Ethnic Residential Segregation in the United States: 1980–2000*. Washington, DC: US Census Bureau, US Government Printing Office; 2002. (CENSR no. 3).
24. Osypuk TL, Galea S, McArdle N, et al. Quantifying separate and unequal: racial/ethnic distributions of neighborhood poverty in metropolitan America. *Urban Aff Rev.* In press.
25. Conley D, Strully KW, Bennett NG. *The Starting Gate: Birth Weight and Life Chances*. Berkeley, CA: University of California Press; 2003.
26. Misra DP, Guyer B, Allston A. Integrated perinatal health framework: a multiple determinants model with a life span approach. *Am J Prev Med*. 2003;25(1):65–75.
27. Little RE, Peterson DR. Sudden infant death syndrome epidemiology: a review and update. *Epidemiol Rev.* 1990;12: 241–246.
28. Kraus J, Greenland S, Bulterys M. Risk factors for sudden infant death syndrome in the US Collaborative Perinatal Project. *Int J Epidemiol*. 1989;18(1):113–120.
29. Kramer M, Demissie K, Yang H, et al. The contribution of mild and moderate preterm birth to infant mortality. Fetal and Infant Health Study Group of the Canadian Perinatal Surveillance System. *JAMA*. 2000;284(7):843–849.
30. Cutler DM, Glaeser EL. Are ghettos good or bad? *Q J Econ*. 1997;112:827–872.