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Racial/Ethnic Residential Segregation, Obesity, and Diabetes Mellitus

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Abstract Persistent racial/ethnic disparities in obesity and type 2 diabetes mellitus seen in the US are likely due to a combination of social, biological, and environmental factors. A growing number of studies have examined the role of racial/ethnic residential segregation with respect to these outcomes because this macro-level process is believed to be a fundamental cause of many of the factors that contribute to these disparities. This review provides an overview of findings from studies of racial/ethnic residential segregation with obesity and diabetes published between 2013 and 2015. Findings for obesity varied by geographic scale of the segregation measure, gender, ethnicity, and racial identity (among Hispanics/Latinos). Recent studies found no association between racial/ethnic residential segregation and diabetes prevalence, but higher segregation of Blacks was related to higher diabetes mortality. Implications of these recent studies are discussed as well as promising areas of future research.

Keywords Diabetes mellitus · Obesity · Residential segregation · Race/ethnicity

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Introduction

It is well established that the burden of obesity and type 2 diabetes in the US is not evenly distributed by race/ethnicity, with non-Hispanic Blacks, Native Americans, and certain Hispanic/Latino and Asian background groups having much higher rates than their non-Hispanic White counterparts [1–3]. These disparities are likely due to a complex constellation of social, biological, and environmental factors. Many of these factors, such as socioeconomic position, chronic stress, and access to healthy foods, are influenced by racial/ethnic residential segregation, the systematic separation of groups into different neighborhoods by race/ethnicity. The neighborhood an individual grows up in can impact the quality of the education they receive and their subsequent earning potential, which is associated with obesity and type 2 diabetes. In addition, several studies have shown that the physical resources available in a neighborhood can impact obesity and type 2 diabetes [4, 5], and that many of these resources are lacking in highly segregated neighborhoods [6, 7]. For these reasons, Williams and Collins called racial residential segregation a fundamental cause of racial health disparities [8], a cause of the unequal distribution of the resources (e.g., money, knowledge, power) that protect health regardless of the relevant mechanisms of the time [9].

Several studies have examined associations of racial residential segregation with adverse birth outcomes (preterm birth [10–13], low birthweight [14–18], and infant mortality [19–21]) and all-cause mortality [22–26] in Blacks across metropolitan areas, but until recently, few had evaluated relationships with metabolic conditions like obesity or diabetes [27–29]. In addition, far less is known about the role of segregation in health among other race/ethnic groups. The long legacy of housing discrimination and discriminatory lending practices is a major contributor to the persistent segregation of

Blacks from Whites [30]. While Asian and Hispanic/Latino populations do face housing discrimination [31], the continuous influx of Asian and Hispanic/Latino immigrants from Asian countries and Latin America into the US means the forces driving segregation may be different for these groups than those driving the spatial distribution of Blacks in the US.

In this review, we discuss the more recent studies of relationships of segregation of Blacks, Hispanics/Latinos, and Asians with obesity and diabetes, and we identify gaps in our understanding of how and whether segregation influences these conditions. Given the unique historical context of segregation in the US, we limited our review to studies of US populations.

Identification of Relevant Studies

In an effort to identify relevant studies, we conducted a comprehensive review of articles published between January 2013 and December 2015 using MEDLINE, PsychINFO, and Web of Science. We utilized search terms that included both segregation-related terms (residential segregation, ethnic density, immigrant density, ethnic enclave, immigrant enclave, racial composition, ethnic composition) and terms related to either obesity (obesity, body mass index, and weight) or type 2 diabetes (diabetes, glucose, and hemoglobin a1c). The initial obesity search yielded 141 articles. From these, titles and abstracts were reviewed, and only relevant studies published in a journal were retained. Duplicates ($n = 33$), irrelevant topics ($n = 99$), and dissertations ($n = 8$) were excluded. This yielded six relevant articles. The same approach was applied to type 2 diabetes. The initial diabetes search yielded 37 articles. Duplicates ($n = 16$) and irrelevant topics ($n = 15$) were excluded. This yielded eight relevant papers. In sum, our literature review yielded 14 relevant articles on obesity (Table 1) or diabetes (Table 2) pertaining to Black, Hispanic, or Asian residential segregation.

Assessing Residential Segregation

Segregation measures are typically derived using U.S. Census or American Community Survey data based on geocoded participant addresses. In health studies, segregation is typically measured at one of two geographic scales: the metropolitan area level or the neighborhood level. Studying segregation at the metropolitan level is based on the notion that metropolitan areas are the level at which job opportunities and housing markets are shaped. A key limitation of metropolitan-level measures is that they do not account for variations in segregation exposure within metropolitan areas. Examining segregation at the neighborhood level overcomes this limitation, but these studies are susceptible to the same methodological challenges of other neighborhood-level studies [32].

One notable challenge for neighborhood-level studies is selection bias, which occurs when the characteristics that influence where individuals decide to live are not independent of the outcome of interest. For example, if leaner individuals are more likely to choose to live in a neighborhood that promotes physical activity, then the relationship between individual weight and neighborhood physical activity resources might be mistaken for a neighborhood effect on obesity. Another is off-support inferences, or structural confounding, which occurs when associations are based on extrapolations rather than actual data. As will be described in more detail later in the review, this methodological challenge is particularly relevant to segregation-health studies, where there may be little overlap in exposure to segregation across race/ethnic groups.

Massey and Denton identified five dimensions of segregation at the metropolitan area level: evenness (spatial distribution of a group), exposure (propensity for contact between groups), clustering (groups of interest located in close proximity, or neighboring areas), centralization (the extent to which a group lives in or near the center of an urban area), and concentration ("relative amount of physical space occupied") [33]. The isolation index, a measure of the exposure dimension, was the most commonly used measure of metropolitan-level segregation in the literature reviewed here. It measures the extent to which race/ethnic groups within neighborhoods of a metropolitan area come into contact with each other. The isolation index is defined as the percentage of the population that is a certain race/ethnic group in the neighborhood in which the average person in that race/ethnic group lives [33]. Scores range from approximately 0 to 1, where a higher score indicates a greater likelihood of being isolated from other race/ethnic groups.

The most commonly used measure of neighborhood-level segregation in these recent studies was racial/ethnic composition, with census tracts or block groups usually serving as proxies for neighborhoods. This measure is not considered a direct measure of segregation because it does not provide any information on how racial/ethnic groups are distributed in space. In addition, the measure does not compare the racial/ethnic composition of the neighborhood to the composition of the larger surrounding area. Spatial and more contextualized measures of neighborhood-level segregation do exist, but they require calculations or the use of specialized software. Further work is needed to determine the extent to which proxy measures like racial/ethnic composition correlate with direct measures, particularly for Asian and Hispanic/Latino groups, who are not as highly segregated as non-Hispanic Blacks.

One issue that emerged from this review of the recent literature is that inconsistencies in the operationalization of residential segregation made comparisons of findings

Table 1 Summary of recent research focusing on residential segregation and obesity in the United States

Study	Sample	Design	Geographic scale	Measure of segregation	Outcome variable	Covariates	Findings
Bower et al. 2015 [34]	Non-Hispanic White and non-Hispanic Black women >20 years old participating in the National Health and Nutrition Examination Survey (1999–2004)	Cross-sectional	Metropolitan area	Black Isolation Index—continuous	Obesity, measured height and weight	Macro level: – Region – Neighborhood percent living in poverty Individual level: – Age – Family income – Marital status – Educational level	Positive association for Black women Negative association for White women
Corral et al. 2014 [36]	Hispanic participants of the Behavioral Risk Factor Surveillance System (2000)	Cross-sectional	Metropolitan area	Hispanic Isolation Index—categorized as high (≥60), moderate, and low (<50) exposure	Obesity, self-reported height and weight	Metropolitan level: – Poverty Individual level: – Gender – Age – Education	Positive association
Kershaw et al. 2014 [37]	Hispanic participants of the 2003–2008 Behavioral Risk Factor Surveillance System age ≥25 years living in metropolitan or micropolitan areas self-identified as White, Black, or "other race"	Cross-sectional	Metropolitan/ micropolitan area	Hispanic Isolation Index—continuous	BMI, self-reported height and weight	Metropolitan level: – Poverty – Population size Individual level: – Age – Age-squared – Race – Language of examination	Null association for men Positive association for Hispanic White women Negative association for Hispanic Black women Null association for Hispanic women identifying as other race
Kershaw et al. 2013 [35••]	Non-Hispanic Black and Mexican American participants of the National Health and Nutrition Examination Survey (1999–2006)	Cross-sectional	Metropolitan area	Black Isolation Index for Blacks and Hispanic Isolation Index for Mexican Americans—categorized as high (>0.6), medium, and low (≤0.3)	Obesity, measured height and weight	Neighborhood level: – Poverty – Negative income incongruity Individual level: – Age – Age-squared – Education – Income – Nativity	Null association for Black and Mexican-American men Positive association for Black women Negative association for Mexican-American women
Li et al. 2014 [38]	Non-Hispanic Black and Non-Hispanic White participants of the Southeastern Pennsylvania Household Health Survey (2006 and 2008)	Cross-sectional	Neighborhood	Percent Black (census tract)—dichotomized at ≥25 % Black	Obesity, self-reported height and weight	Neighborhood level: – Social cohesion – Socioeconomic status – Street connectivity – Park accessibility – Residential stability Individual level: – Age – Age-squared – Marital status – Nativity	Null association for White women, Black women, and Black men Negative association for White men

Table 1 (continued)

Study	Sample	Design	Geographic scale	Measure of segregation	Outcome variable	Covariates	Findings
						– Education – Income – Examination year – Cigarette smoking	
Cozier et al. 2014 [39•]	Participants of the Black Women's Health Study (1997–2009)	Cohort	Neighborhood	Percent Black (census block group)—categorized as quartiles	Incident obesity, self-reported height and weight	Macro level: – Geographical region – Neighborhood socioeconomic status – Neighborhood housing density Individual level: – Age – Questionnaire cycle – Education – Household income – Number of people in household – Cigarette smoking – Alcohol consumption – Vigorous exercise – Walking for exercise – Hours of television viewing – Parity – Prudent dietary pattern – Western dietary pattern	Positive association (only statistically significant for women who consistently lived in segregated neighborhoods over follow-up)
Le-Scherban et al. 2014 [40•]	Foreign-born Chinese and Hispanic Multi-Ethnic Study of Atherosclerosis Participants (2000–2012)	Cohort	Neighborhood	Change in percent Asian for Chinese participants and percent Hispanic for Hispanic participants (census tract)—decrease from 75th to 25th percentile	Change in BMI, measured height and weight Change in waist circumference, measured	Neighborhood level: – Household income Individual level: – Moved since previous examination – Age – Years living in US – Annual family income	Null association
Ullman et al. 2013 [41]	Participants of Los Angeles Family and Neighborhood Study (2000–2001 and 2006–2008)	Cohort	Neighborhood	Percent race/ethnic group (census tract)—categorized as: High (≥35 %) Asian/Pacific; predominantly (≥75 %) White; Hispanic and Black; predominantly Hispanic (≥75 %); and White and other group	Annual weight change, self-reported weight	Neighborhood level: – Socioeconomic disadvantage – Collective efficacy Individual level: – Age – Race/ethnicity/immigrant generation – Gender – Marital status – Height	Null association

Table 2 Summary of recent research focusing on residential segregation and diabetes in the United States

Study	Sample	Design	Geographic scale	Measure of segregation	Outcome variable	Covariates	Findings
Grigsby-Toussaint et al. 2015 [46]	Non-Hispanic White and Hispanic participants of the Behavioral Risk Factor Surveillance System (2005)	Cross-sectional	Metropolitan area	All 5 Massey and Denton segregation measures—categorized as hypersegregated (score >0.55 on ≥4 of the 5); segregated (score >0.55 on 1–3 of the 5); and non-segregated (score >0.55 on 0 of the 5)	Diabetes, self-reported	Metropolitan level: – Poverty – Population size – Percent Hispanic Individual level: – Age – Gender – Marital status – Education – Household income – Employment status – BMI – Insurance status – Fruit/vegetable intake – Physical activity	Null association
Lim et al. 2015 [47]	Asian participants of the New York City Community Health Survey (2009–2012)	Cross-sectional	Community district	Dissimilarity index and isolation Index—classified as Asian enclave if score >0.35 on both indices	Diabetes, self-reported	Individual-level – Age – Gender – Language spoken at home – Marital status – Education – Household poverty – Employment – Nativity – Years living in the US – Physical activity – Fruit/vegetable intake – BMI – Healthcare access characteristics	Null association
Rosenstock et al. 2014 [48]	Non-Hispanic Blacks and non-Hispanic Whites living in the 50 most populous US cities (2005–2007)	Ecologic	City	Percent Black (city)—continuous Black Isolation Index—continuous	Age-adjusted diabetes mortality rates	None	Positive association (only statistically significant for percent Black)
Gaskin et al. 2014 [45]	Non-Hispanic Black and non-Hispanic White participants of the National Health and Nutrition Examination Survey (1999–2004)	Cross-sectional	Neighborhood	Percent Black, percent White, and percent other (census tract)—categorized as predominantly Black, White, or other (>65 %) or integrated (at least 2 groups	Diabetes, measured fasting glucose, hemoglobin A1c, and/or self-reported diabetes medication use	Neighborhood level: – Neighborhood poverty Individual level: – Individual poverty – Race – Gender – Age	Null associations

Table 2 (continued)

Study	Sample	Design	Geographic scale	Measure of segregation	Outcome variable	Covariates	Findings
				were each more than 35 % of the census tract's population)		– Age-squared – Family history – Education – Health insurance status	
Hunt et al. 2014 [49]	Non-Hispanic Blacks, Non-Hispanic Whites, and Hispanics living in Chicago (2006–2008)	Ecologic	Neighborhood	Percent Black (community area)—continuous	Age-adjusted diabetes mortality rate	None	Positive association
Piccolo et al. 2015 [44]	Non-Hispanic Black, Hispanic, and non-Hispanic White participants of the Boston Area Community Health Survey (2010–2012)	Cross-sectional	Neighborhood	Percent Black (census tract)—continuous	Diabetes, measured glucose, hemoglobin A1c, and/or self-reported diagnosis	Neighborhood level: – Socioeconomic status – Poverty – Property crime – Violent crime – Built environment – Open space – Physical disorder – Social disorder Individual level: – Age – Gender – BMI – Diet – Physical activity – Income – Education – Occupation	Null association

challenging. A cutpoint of 0.6 (i.e., 60 % Black) is a commonly used in the segregation literature to represent high segregation of Blacks [8], but as our review shows, different cutpoints are often used in studies of segregation and health with little justification offered. This contributes to the difficulty of understanding why findings are null in some studies but significant in others. The cutpoints were more consistent for metropolitan area-level studies, which may explain the more consistent findings at that geographic scale.

In addition, given the different historical context of segregation among Asians and Hispanics/Latinos described above, the challenge of defining segregation adequately for these groups remains unsolved. For example, a study of residential segregation and birthweight found ethnic composition and immigrant composition were differentially associated with birthweight in the offspring of US-born Mexican-origin women [17]. They found higher US-born Mexican-origin residential segregation (i.e., ethnic enclaves) was associated with lower birthweight, but higher foreign-born Mexican-origin residential segregation (i.e., immigrant enclaves) was associated with higher birthweight. This differential impact by nativity may reflect differences in the drivers of segregation which may in turn have different health consequences. Future research is needed to explore the most salient measures of segregation for different ethnic groups, particularly as they pertain to the patterning of obesity and diabetes.

Racial/Ethnic Residential Segregation and BMI/Obesity

There were eight studies that examined residential segregation and BMI/obesity, and all were in adults. There was little overlap in the geographic scale of segregation (metropolitan versus neighborhood), race/ethnicity of the population, and cutpoints used to assess segregation, which makes comparisons of findings challenging. Two studies of metropolitan-level segregation in non-Hispanic Black women found a higher Black isolation index score was related to higher obesity prevalence [34, 35••], but both studies used National Health and Nutrition Examination Survey (NHANES) data collected around the same time. One of these studies also showed a higher Hispanic isolation index score was associated with lower obesity in Mexican-American women, and that segregation was unassociated with obesity prevalence in men [35••]. Higher segregation was related to higher obesity prevalence in Hispanic Behavioral Risk Factor Surveillance System (BRFSS) participants [36], but a study using the same data source found this association varied by gender and racial identity [37]. In the latter study, using more recent data, researchers found segregation was unassociated with BMI in men. They found a positive association for Hispanic White women, consistent with the earlier BRFSS paper, but they found a negative association for Hispanic Black women and a null association for Hispanic women self-identifying as "other race."

Recent studies of neighborhood-level segregation and BMI/obesity were in racially/ethnically diverse populations, and findings were largely null. The only cross-sectional study was in southeastern Pennsylvania, and investigators found White men living in a neighborhood ≥25 % Black were less likely to be obese than White men living in a neighborhood <25 % Black [38]. This measure was not associated with obesity for Black men, or for Black or White women. The other three studies were longitudinal, and they each focused on a different racial/ethnic population. A study in the Black Women's Health Study found women living in neighborhoods with the highest quartile percent Black were more likely to become obese, but only if they remained in a highest quartile neighborhood over the follow-up period [39•]. A study of foreign-born Chinese and Hispanics participants of the Multi-Ethnic Study of Atherosclerosis (MESA) found decreases in neighborhood percent Asian and Hispanic, respectively, were not associated with changes in BMI or waist circumference [40•]. Similarly, a multi-ethnic Los Angeles study found no association between percent race/ethnic group and annual weight change [41].

Our review of recent studies of metropolitan area-level segregation and obesity revealed the complex relationship this social process has with health. We found heterogeneity in the impact of segregation on obesity by gender, ethnicity, and racial identity among Hispanics. This heterogeneity may reflect differences in the underlying forces driving segregation among different groups (e.g., of non-Hispanic Blacks versus Hispanics/Latinos) and differences in the susceptibility to the effects of segregation on obesity (e.g., men versus women).

There are several potential reasons for the largely null findings in the recent studies examining associations of neighborhood-level racial/ethnic residential segregation with BMI/obesity. These studies focused on different race/ethnic groups living in different parts of the country. Thus, it is possible that these associations vary by race/ethnic group and geographic location within the US. Furthermore, many of these studies relied on self-report, which tends to lead to underestimates in BMI and obesity prevalence. This source of measurement error could result in biased associations of segregation with obesity if the misreporting was different for those living in segregated versus non-segregated areas. The potential for this differential misclassification bias has not been examined, but there is some evidence non-Hispanic Whites are more likely to under-report BMI than non-Hispanic Blacks or Hispanics [42].

In addition, the very nature of residential segregation makes it difficult to produce on-support inferences (i.e., ones based on actual data rather than extrapolations) regarding relationships with health, particularly at the neighborhood level.

For example, a study of segregation and preterm birth in two counties in North Carolina found that there were no census tracts in either county that were in the highest quartile for percent Black and the lowest quartile for neighborhood poverty (i.e., predominantly Black, low poverty tracts) and only one in the lowest quartile for percent Black and the highest quartile for neighborhood poverty (predominantly White, high poverty tracts) [43].

The problem with this lack of overlap is that a regression model will still provide an estimate of the association between segregation and health after adjusting for neighborhood poverty, but it will be based on extrapolations rather than real data points. In addition, associations may remain confounded due to the absence of actual data. This type of confounding attributable to social sorting mechanisms like segregation has been called structural confounding [32], and it is especially problematic because it cannot be overcome simply by adding more data. Careful attention to the distribution of key variables in a dataset will help minimize off-support inferences.

Racial/Ethnic Residential Segregation and Diabetes

There were six studies that examined associations of segregation with diabetes, once again all in adults. Of these, four studies focused on diabetes prevalence and two focused on diabetes mortality. Only one study defined diabetes specifically as type 2 diabetes [44]; the rest did not differentiate between type 1 and type 2 diabetes. As with BMI/obesity, studies varied widely in terms of geographic scale, race/ethnicity of the study population, and categorizations of the measures. However, findings were consistently null for diabetes prevalence and consistently positive for diabetes mortality. A Boston study [44] and a NHANES study [45] were the only two to assess relationships of segregation with diabetes prevalence in non-Hispanic Blacks. The same Boston study, as well as a BRFSS study [46], examined segregation and diabetes prevalence in Hispanics, and a New York City study looked at diabetes prevalence in Asians [47]. The two studies of segregation and diabetes mortality were ecologic in design, and both found higher percent Black was correlated with higher age-adjusted diabetes mortality. One compared percent Black and diabetes mortality across the 50 most populous US cities [48], while the other compared rates across neighborhoods in Chicago [49].

This review of the more recent literature does not provide strong evidence supporting residential segregation as a contributor to diabetes diagnosis. The significant findings for diabetes mortality but not prevalence in Blacks may suggest segregation influences the severity of disease but not the development. The ecologic nature of the segregation-diabetes mortality studies preclude us from drawing inferences on the impact of segregation on diabetes mortality at the individual level. However, these findings suggest future studies may want to focus on diabetes-related management and complications.

Previous studies have shown that minorities are less likely to meet guidelines for glycemic control or self-monitoring of diabetes, and they are more likely to suffer from microvascular diabetes complications [50–52]. There is some evidence that diabetic Blacks living in segregated metropolitan areas receive comparable routine diabetes care (defined as receipt of a HbA1c test, low-density lipoprotein test, diabetic eye examination, and diabetic foot examination at least once in the past year) to those living in less segregated metropolitan areas [53], but no studies to our knowledge have examined relationships of segregation with the efficacy of diabetes treatment regimens or control of diabetes. In addition, it is unknown whether those living in more segregated areas are more likely to develop diabetes complications. These types of studies have important implications for our understanding of the role of place in shaping health care disparities.

Implications and Future Directions

The mixed findings we see for studies of residential segregation with obesity and diabetes highlight the difficulties in assessing the impact of this upstream, macro-level process on health. Cross-sectional studies, and even most longitudinal studies, cannot adequately capture the hypothesized impact of segregation on health. Several studies we reviewed adjusted for potential mediators, such as health behaviors and the neighborhood built and socioeconomic environment. Another more problematic potential mediator to handle in these analyses is socioeconomic attainment which begins in childhood and impacts adult levels. Thus, while adult socioeconomic conditions could confound associations of segregation with health, earlier conditions could also mediate these associations. Differences in adult socioeconomic attainment have been shown to account for substantial portions of race/ethnic differences in type 2 diabetes prevalence [54, 55], so not being able to properly attribute this to segregation may result in an underestimation of its impact on disparities in type 2 diabetes. Alternative regression modeling approaches such as marginal structural modeling may help address this issue [56].

This issue of potential overadjustment, and the problem of structural confounding, mean that alternative modeling strategies and study designs will need to be applied to improve our understanding of the role of segregation in health. For example, one of the studies we reviewed used fixed-effects models to estimate associations of within-person changes in exposure to ethnic enclaves with within-person changes in BMI and waist circumference [40•]. The advantage of this approach over other longitudinal modeling strategies is that by examining within-person changes, one is able to tightly control for measured and unmeasured time-invariant confounders. One drawback of this approach, though, is a limited ability to assess heterogeneity between individuals. In addition, this modeling approach does not account for unmeasured time-varying confounders (e.g., changes in residential preferences over time).

Alternative study designs may also offer some insights into the impact of segregation on health. For example, the Exploring Health Disparities in Integrated Communities (EHDIC) study addresses issues of structural confounding by studying health disparities in Blacks and Whites living in integrated communities [57, 58]. They showed that Blacks and Whites living in integrated communities in Baltimore with similar income levels had more similar diabetes and obesity prevalence compared with Black and Whites in national studies. Studies designed like the EHDIC offer unique insights into the role of the environment in health and health disparities [57].

Taking advantage of natural experiments is another alternative study design that might be fruitful for examining the contributions of segregation to health. For example, examining the health impacts of housing voucher programs that give residents in segregated, high poverty neighborhoods the opportunity to move into better quality neighborhoods may provide insights into how segregation impacts health and, perhaps more importantly, whether segregation is a modifiable exposure. Though findings for health outcomes are limited, one notable health-related study of a housing mobility program is the long-term follow-up study of the Moving to Opportunity program [59]. Researchers found participants (low income women, predominantly minority) who received vouchers to move into low poverty neighborhoods between 1994 and 1998 were less likely to be morbidly obese and had lower HbA1c levels than the control group (those who did not receive vouchers to move) in 2008–2010.

In addition, systems science approaches have the potential to aid in our understanding of how residential segregation impacts health in different segments of the US population and also provide insights into the types of policies that might best reduce the negative impact of segregation on health. Systems science methodologies can be used to generate models designed to be simplified versions of reality in order to better understanding complex, dynamic relationships between individuals, their environments, and each other [60, 61]. The benefit of these models over traditional regression models is that they are built to identify and quantify nonlinear relationships, bidirectional relationships, and emergent phenomena. A major challenge with these modeling approaches is making the models simple enough to answer specific questions about social processes but still informative and useful for decision making and policy.

Conclusions

It has been 15 years since Williams and Collins called racial residential segregation a fundamental cause of racial health disparities for its influence on socioeconomic attainment and access to health-promoting environmental resources. Since then, there has been a steady increase in publications on residential segregation and an expansion from mortality and birth outcomes into chronic conditions like obesity and type 2 diabetes. While still largely cross-sectional, the number of longitudinal studies is increasing, as evidenced in this review. In addition, the field has expanded from a focus on segregation in African-Americans to include other ethnic groups including Hispanics/Latinos and Asians. Further work is needed to better capture the role of residential segregation patterns in obesity and type 2 diabetes, both by using existing cohort data and by utilizing alternative study designs. Continued efforts in this area have the potential to elucidate the contributions of place to health and guide efforts to develop more effective interventions to meaningfully reduce health inequities in the US.

Compliance with Ethical Standards

Conflict of Interest Kiarri N. Kershaw and Ashley E. Pender declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
• Of importance
•• Of major importance

1. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014;311(8):806–14.
2. Zhang Q, Wang Y, Huang ES. Changes in racial/ethnic disparities in the prevalence of type 2 diabetes by obesity level among U.S. adults. Ethn Health. 2009;14(5):439–57.
3. Kirtland KA, Cho P, Geiss LS. Diabetes among Asians and Native Hawaiians or other Pacific Islanders—United States, 2011–2014. MMWR Morb Mortal Wkly Rep. 2015;64(45):1261–6.
4. Auchincloss AH, Diez Roux AV, Mujahid MS, Shen M, Bertoni AG, Carnethon MR. Neighborhood resources for physical activity and healthy foods and incidence of type 2 diabetes mellitus: the Multi-Ethnic study of Atherosclerosis. Arch Intern Med. 2009;169(18):1698–704.
5. Christine PJ, Auchincloss AH, Bertoni AG, Carnethon MR, Sanchez BN, Moore K, et al. Longitudinal associations between neighborhood physical and social environments and incident type

2 diabetes mellitus: the Multi-Ethnic Study of Atherosclerosis (MESA). JAMA Intern Med. 2015;175(8):1311–20.

6. Bower KM, Thorpe RJ, Rohde C, Gaskin DJ. The intersection of neighborhood racial segregation, poverty, and urbanicity and its impact on food store availability in the United States. Prev Med. 2014;58:33–9.
7. Osypuk TL, Diez Roux AV, Hadley C, Kandula NR. Are immigrant enclaves healthy places to live? The Multi-ethnic Study of Atherosclerosis. Soc Sci Med. 2009;69(1):110–20.
8. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. Public Health Rep. 2001;116(5):404–16.
9. Link BG, Phelan J. Social conditions as fundamental causes of disease. J Health Soc Behav. 1995; Spec No:80–94.
10. Anthopolos R, James SA, Gelfand AE, Miranda ML. A spatial measure of neighborhood level racial isolation applied to low birthweight, preterm birth, and birthweight in North Carolina. Spat Spatiotemporal Epidemiol. 2011;2(4):235–46.
11. Anthopolos R, Kaufman JS, Messer LC, Miranda ML. Racial residential segregation and preterm birth: built environment as a mediator. Epidemiology. 2014;25(3):397–405.
12. Britton ML, Shin H. Metropolitan residential segregation and very preterm birth among African American and Mexican-origin women. Soc Sci Med. 2013;98:37–45.
13. Mason SM, Kaufman JS, Daniels JL, Emch ME, Hogan VK, Savitz DA. Neighborhood ethnic density and preterm birth across seven ethnic groups in New York City. Health Place. 2011;17(1):280–8.
14. Debbink MP, Bader MD. Racial residential segregation and low birth weight in Michigan's metropolitan areas. Am J Public Health. 2011;101(9):1714–20.
15. Grady SC. Racial residential segregation impacts on low birth weight using improved neighborhood boundary definitions. Spat Spatiotemporal Epidemiol. 2010;1(4):239–49.
16. 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):661–77.
17. Osypuk TL, Bates LM, Acevedo-Garcia D. Another Mexican birthweight paradox? The role of residential enclaves and neighborhood poverty in the birthweight of Mexican-origin infants. Soc Sci Med. 2010;70(4):550–60.
18. Walton E. Residential segregation and birth weight among racial and ethnic minorities in the United States. J Health Soc Behav. 2009;50(4):427–42.
19. Hearst MO, Oakes JM, Johnson PJ. The effect of racial residential segregation on black infant mortality. Am J Epidemiol. 2008;168(11):1247–54.
20. Polednak AP. Black-white differences in infant mortality in 38 standard metropolitan statistical areas. Am J Public Health. 1991;81(11):1480–2.
21. Polednak AP. Trends in US urban black infant mortality, by degree of residential segregation. Am J Public Health. 1996;86(5):723–6.
22. Fang J, Madhavan S, Bosworth W, Alderman MH. Residential segregation and mortality in New York City. Soc Sci Med. 1998;47(4):469–76.
23. Hart KD, Kunitz SJ, Sell RR, Mukamel DB. Metropolitan governance, residential segregation, and mortality among African Americans. Am J Public Health. 1998;88(3):434–8.
24. Inagami S, Borrell LN, Wong MD, Fang J, Shapiro MF, Asch SM. Residential segregation and Latino, black and white mortality in New York City. J Urban Health. 2006;83(3):406–20.
25. Jackson SA, Anderson RT, Johnson NJ, Sorlie PD. The relation of residential segregation to all-cause mortality: a study in black and white. Am J Public Health. 2000;90(4):615–7.
26. Polednak AP. Poverty, residential segregation, and black/white mortality ratios in urban areas. J Health Care Poor Underserved. 1993;4(4):363–73.
27. Kramer MR, Hogue CR. Is segregation bad for your health? Epidemiol Rev. 2009;31:178–94.
28. White K, Borrell LN. Racial/ethnic residential segregation: framing the context of health risk and health disparities. Health Place. 2011;17(2):438–48.
29. Kershaw KN, Albrecht SS. Racial/ethnic residential segregation and cardiovascular disease risk. Curr Cardiovasc Risk Rep. 2015; 9(3).
30. Massey DS, Denton NA. American apartheid: segregation and the making of the underclass. Cambridge: Harvard UP; 1993.
31. Turner MA, Santos R, Levy DK, Wissoker D, Aranda C, Pitingolo R, et al. Housing discrimination against racial and ethnic minorities 2012. Washington, D.C: Department of Housing and Urban Development; 2013.
32. Oakes JM. Commentary: Advancing neighbourhood-effects research—selection, inferential support, and structural confounding. Int J Epidemiol. 2006;35(3):643–7.
33. Massey DS, Denton NA. The dimensions of residential segregation. Soc Forces. 1988;67(2):281–315.
34. Bower KM, Thorpe Jr RJ, Yenokyan G, McGinty EE, Dubay L, Gaskin DJ. Racial residential segregation and disparities in obesity among women. J Urban Health. 2015;92(5):843–52.
35.•• Kershaw KN, Albrecht SS, Carnethon MR. Racial and ethnic residential segregation, the neighborhood socioeconomic environment, and obesity among Blacks and Mexican Americans. Am J Epidemiol. 2013;177(4):299–309. **First to examine associations of residential segregation with obesity in Hispanics/Latinos.**
36. Corral I, Landrine H, Zhao L. Residential segregation and obesity among a national sample of Hispanic adults. J Health Psychol. 2014;19(4):503–8.
37. Kershaw KN, Albrecht SS. Metropolitan-level ethnic residential segregation, racial identity, and body mass index among U.S. Hispanic adults: a multilevel cross-sectional study. BMC Public Health. 2014;14:283.
38. Li K, Wen M, Henry KA. Residential racial composition and black-white obesity risks: differential effects of neighborhood social and built environment. Int J Environ Res Public Health. 2014;11(1): 626–42.
39.• Cozier YC, Yu J, Coogan PF, Bethea TN, Rosenberg L, Palmer JR. Racism, segregation, and risk of obesity in the Black Women's Health Study. Am J Epidemiol. 2014;179(7):875–83. **First to examine longitudinal association of residential segregation with obesity.**
40.• Le-Scherban F, Albrecht SS, Osypuk TL, Sanchez BN, Diez Roux AV. Neighborhood ethnic composition, spatial assimilation, and change in body mass index over time among Hispanic and Chinese immigrants: Multi-Ethnic Study of Atherosclerosis. Am J Public Health. 2014;104(11):2138–46. **Illustration of use of fixed-effects regression modeling to examine associations of within-person changes in segregation with within-person changes in a health outcome.**
41. Ullmann SH, Goldman N, Pebley AR. Contextual factors and weight change over time: a comparison between U.S. Hispanics and other population sub-groups. Soc Sci Med. 2013;90:40–8.
42. Wen M, Kowaleski-Jones L. Sex and ethnic differences in validity of self-reported adult height, weight and body mass index. Ethn Dis. 2012;22(1):72–8.
43. Messer LC, Oakes JM, Mason S. Effects of socioeconomic and racial residential segregation on preterm birth: a cautionary tale of structural confounding. Am J Epidemiol. 2010;171(6): 664–73.
44. Piccolo RS, Duncan DT, Pearce N, McKinlay JB. The role of neighborhood characteristics in racial/ethnic disparities in type 2 diabetes: results from the Boston Area Community Health (BACH) survey. Soc Sci Med. 2015;130:79–90.

45. Gaskin DJ, Thorpe Jr RJ, McGinty EE, Bower K, Rohde C, Young JH, et al. Disparities in diabetes: the nexus of race, poverty, and place. Am J Public Health. 2014;104(11):2147–55.
46. Grigsby-Toussaint DS, Jones A, Kubo J, Bradford N. Residential segregation and diabetes risk among Latinos. Ethn Dis. 2015;25(4): 451–8.
47. Lim S, Yi SS, Lundy De La Cruz N, Trinh-Shevrin C. Defining ethnic enclave and its associations with self-reported health outcomes among Asian American adults in New York City. J Immigr Minor Health. 2015.
48. Rosenstock S, Whitman S, West JF, Balkin M. Racial disparities in diabetes mortality in the 50 most populous US cities. J Urban Health. 2014;91(5):873–85.
49. Hunt BR, Whitman S, Henry CA. Age-adjusted diabetes mortality rates vary in local communities in a metropolitan area: racial and spatial disparities and correlates. Diabetes Care. 2014;37(5): 1279–86.
50. Spanakis EK, Golden SH. Race/ethnic difference in diabetes and diabetic complications. Curr Diab Rep. 2013; 13(6):10.1007/s11892-013-0421-9.
51. Saaddine JB, Engelgau MM, Beckles GL, Gregg EW, Thompson TJ, Narayan KM. A diabetes report card for the United States: quality of care in the 1990s. Ann Intern Med. 2002;136(8):565–74.
52. Sequist TD, Adams A, Zhang F, Ross-Degnan D, Ayanian JZ. Effect of quality improvement on racial disparities in diabetes care. Arch Intern Med. 2006;166(6):675–81.
53. Gaskin DJ, Dinwiddie GY, Chan KS, McCleary R. Residential segregation and disparities in healthcare services utilization. Med Care Res Rev. 2012;69(2):158–75.
54. Piccolo RS, Subramanian SV, Pearce N, Florez JC, McKinlay JB. Relative contributions of socioeconomic, local environmental, psychosocial, lifestyle/behavioral, biophysiological, and ancestral factors to racial/ethnic disparities in type 2 diabetes. Diabetes Care. 2016;39(7):1208–17.
55. Link CL, McKinlay JB. Disparities in the prevalence of diabetes: is it race/ethnicity or socioeconomic status? Results from the Boston Area Community Health (BACH) survey. Ethn Dis. 2009;19(3): 288–92.
56. Cerda M, Diez-Roux AV, Tchetgen ET, Gordon-Larsen P, Kiefe C. The relationship between neighborhood poverty and alcohol use: estimation by marginal structural models. Epidemiology. 2010;21(4):482–9.
57. LaVeist T, Pollack K, Thorpe Jr R, Fesahazion R, Gaskin D. Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. Health Aff (Millwood). 2011;30(10):1880–7.
58. LaVeist T, Thorpe Jr R, Bowen-Reid T, Jackson J, Gary T, Gaskin D, et al. Exploring health disparities in integrated communities: overview of the EHDIC study. J Urban Health. 2008;85(1):11–21.
59. Ludwig J, Sanbonmatsu L, Gennetian L, Adam E, Duncan GJ, Katz LF, et al. Neighborhoods, obesity, and diabetes—a randomized social experiment. N Engl J Med. 2011;365(16):1509–19.
60. Luke DA, Stamatakis KA. Systems science methods in public health: dynamics, networks, and agents. Annu Rev Public Health. 2012;33:357–76.
61. Mabry PL, Marcus SE, Clark PI, Leischow SJ, Méndez D. Systems science: a revolution in public health policy research. Am J Public Health. 2010;100(7):1161–3.