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Assessing the Influence of Redlining on Intergenerational Wealth and Body Mass Index Through a Quasi-experimental Framework

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Abstract

Background: Higher levels of body mass index (BMI), particularly for those who have obesity defined as class II and III, are correlated with excess risk of all-cause mortality in the USA, and these risks disproportionately affects marginalized communities impacted by systemic racism. Redlining, a form of structural racism, is a practice by which federal agencies and banks disincentivized mortgage investments in predominantly racialized minority neighborhoods, contributing to residential segregation. The extent to which redlining contributes to current-day wealth and health inequities, including obesity, through wealth pathways or limited access to health-promoting resources, remains unclear. Our quasi-experimental study aimed to investigate the generational impacts of redlining on wealth and body mass index (BMI) outcomes.

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Ethics Approval Given careful consideration of ethical standards, this study underwent a thorough ethical review process, resulting in its classification as minimal risk for participants. Consequently, the study received expedited review and a consent waiver.

Consent to Participate Due to the minimal risk of using PSID data for a secondary analysis, the University of Washington waived the collection of consent for this study.

Competing Interests The authors declare no competing interests.

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Methods: We leveraged the Panel Study of Income Dynamics (PSID) and Home Owners' Loan Corporation (HOLC) maps to implement a geographical regression discontinuity design, where treatment assignment is randomly based on the boundary location of PSID grandparents in yellowlined vs. redlined areas and used outcome measures of wealth and mean BMI of grandchildren. To estimate our effects, we used a continuity-based approach and applied data-driven procedures to identify the most appropriate bandwidths for a valid estimation and inference.

Results: In our fully adjusted model, grandchildren with grandparents living in redlined areas had lower average household wealth ($\beta = -\$35,419$; 95% $CI_{\text{rbc}} = \$37,423, -\$7615$) and a notably elevated mean BMI ($\beta = 7.47$; 95% $CI_{\text{rbc}} = 4.00, 16.60$), when compared to grandchildren whose grandparents resided in yellowlined regions.

Conclusion: Our research supports the idea that redlining, a historical policy rooted in structural racism, is a key factor contributing to disparities in wealth accumulation and, conceivably, body mass index across racial groups.

Keywords

Residential segregation; Social determinants of health; Intergenerational wealth; Quasi-experimental; Body mass index

Introduction

In the United States (US), structural inequities give rise to an unequal distribution of resources and are associated with pervasive racial disparities in wealth accumulation and health outcomes [1, 2]. These inequities are intertwined with multiple dimensions of racism, particularly structural racism, which encompasses historical and contemporary systems that perpetuate inequities based on the socially constructed concept of race [1–5]. Research has investigated the influence of social determinants of health (SDOH) on racial health disparities [6–8], revealing an association between historical redlining — a form of codified structural racism that restricted mortgage access to neighborhoods disproportionately inhabited by racialized minorities — and elevated rates of breast cancer mortality, preterm birth, and poor physical and mental health outcomes [9–12]. Moreover, Nardone et al. (2021) reported evidence of reduced greenspace in historically redlined areas [13]. These studies suggest that the legacy of redlining continues to reinforce current health inequities. Considering the persistent and profound racial inequalities in the US, it is imperative to understand the historical mechanisms that have led to multiple generations of wealth and health inequities among racial and ethnic minorities. Thus, this study aims to investigate the consequences of redlining on two specific outcomes — household wealth and obesity (or higher body mass index (BMI)).

Structural Racism and Obesity

Obesity is a significant public health concern that is socially patterned and influenced by a complex interplay of social, economic, and environmental factors, which are deeply intertwined with structural access to resources. As defined by a high body mass index, obesity is associated with an increased risk of all-cause mortality in the US [14]. In addition, it increases the risk of developing several chronic conditions, including kidney disease,

hypertension, diabetes, cardiovascular disease, osteoarthritis, stroke, and certain preventable cancers [15–19]. Approximately 41.9% of adults have obesity, with 9.2% having severe obesity [20]. Obesity imposes a significant financial burden to US healthcare systems, with annual obesity-related medical expenditures estimated at \$170 billion [21].

The risk of obesity or a high body mass index has previously been associated with various SDOH, including neighborhood socioeconomic disadvantages, income, and racialized status. Research has shown that the highest prevalence of obesity is among Black and Hispanic populations and those with lower socioeconomic status (SES) [22–25]. Disparities in obesity among racialized groups are marked; however, only limited literature has investigated the role of structural racism in creating these disparities [26–31]. Structural racism may contribute to obesity risk through a lack of investment in or disenfranchisement of racially segregated areas that have primarily Black or racially minoritized residents, by reinforcing neighborhoods with fewer health-promoting and more obesogenic factors [13, 32, 33]. Wealth inequality has also been linked to obesity, with individuals in lower-wealth quintiles having a higher risk of developing obesity than those in wealthier quintiles [34]. There is a high theoretical potential for redlining to explain disparities in obesity through social and economic pathways, including wealth accumulation and neighborhood economic deprivation.

Structural Racism, Wealth, and Obesity

Research investigating the interplay between structurally racist policies, SDOH, and obesity outcomes is scarce [27, 35]. Although some studies have shown that individuals with lower wealth have a higher prevalence of obesity [36–38], even fewer have measured wealth accumulation over multiple generations [39]. There is a gap in the literature that applies generational or life-course approaches to understanding the complex relationship between obesity and wealth, as well as the impact of structural racism on wealth and health [39, 40].

Research indicates a possible connection between structural racism and wealth, which may be linked to obesogenic environments and home values [26, 41]. Drewnowski et al. found that areas near crime, liquor stores, and fast-food stores were associated with lower property values [42]. Many families in redlined neighborhoods may have faced obstacles in building wealth through homeownership due to barriers created by redlining to obtain low-interest loans. Home equity is a significant source of wealth for many Americans, accounting for over a quarter of the portfolio assets of middle-class Americans [43]. Structural racism is also hypothesized to decrease household wealth for racialized minorities because of lower property values in segregated areas [44, 45], which may contribute to a lack of health-promoting amenities, such as safe walkable areas, green spaces, and healthy food access [13, 26, 46–50]. Greenspace, which includes tree canopy coverage, significantly contributes to physical activity, and thus the maintenance of a healthy weight, as this environmental resource commonly encourages residents to engage in outdoor activities [13, 46, 51]. Evidence supports the notion that structural racism and neighborhood segregation contribute to the racial wealth gap. However, no study has used redlining policies as a specific measure of structural racism to quantify their contribution to the gap in generational wealth accumulation [52, 53].

Background of Redlining

In the 1930s, President Roosevelt's New Deal included the creation of the Federal Housing Administration (FHA). The FHA sponsored a federal agency called Home Owners' Loan Corporation (HOLC) [54], which developed a discriminatory system to appraise homes and assess neighborhoods across 239 of America's largest cities using maps to determine perceived credit risk level for home mortgages based on neighborhood characteristics, including demographic composition [55]. The grading scale ranged from the following: A (green — “best” *deemed the lowest credit risk*), B (blue — “still desirable”), C (yellow — “definitely declining”), and D (red — “hazardous” *deemed the highest credit risk*) [54–59]. The racial and ethnic composition of a neighborhood played a significant role in determining the grade an area received, as historical accounts revealed that HOLC graders remarked when “subversive racial elements” were present or increasing in a graded area [60]. Notably, neighborhoods with a higher proportion of racialized minorities, specifically “Negros” (i.e., Blacks), were often labeled as “hazardous” and given a D grade or colored red (hence the term redlining) [58]. The deployment of HOLC maps for assessing mortgage risk has been outlawed since the mid-1970s [54, 61], but the legacy of this practice is still evident in the persistent residential segregation and long-run decline of once redlined neighborhoods throughout the US [55, 58, 62].

We leverage the geographical nature of HOLC's assigned credit security ratings (i.e., red, yellow, blue, and green) as a manifestation of neighborhood-level structural racism. These designations allowed us to evaluate the long-term effects of the redlining. Research has shown that HOLC grading is associated with lower home prices in neighborhoods that were previously redlined, indicating a potential disinvestment in these areas [55, 58, 62, 63]. Furthermore, redlining has been shown to strengthen segregation in many neighborhoods, thereby solidifying racial residential segregation as places previously redlined have become fairly immutable in terms of racial composition over time [55, 58, 62, 63]. The HOLC security maps, endorsed by the federal government through the Federal Housing Authority (FHA), provide a unique opportunity for a natural experiment.

Racial wealth inequality and health disparities highlight the need to assess the causal role of structural racism on both wealth accumulation over multiple generations, and health risk, such as BMI. Our study addresses a crucial gap in the literature by identifying the degree to which redlining is implicated in producing wealth and obesity disparities among racialized minorities over multiple generations. Therefore, this study adopts a temporal causal pathway framework wherein exposure is experienced by the first generation (hereafter called grandparents) and the outcome is measured in the third generation (hereafter called grandchildren). HOLC credit ratings, commonly known as redlining, had a profound impact on mortgage rates by systematically denying loans or offering them at higher (or rather predatory) rates to residents of neighborhoods deemed risky, primarily due to the race or ethnicity of a particular neighborhood. Government policies, originating at the local level, enforced racial zoning ordinances that isolated White families in all-white urban areas, aligning with the discriminatory objectives of redlining [64]. These ordinances aimed to block lower-income African Americans from middle-class White neighborhoods and impede middle-class African Americans from purchasing homes there

[64]. Zoning practices, distinct from redlining but aligned with its discriminatory goals, further entrenched segregation by ensuring that many colored families were ineligible for FHA-insured mortgages, thus perpetuating racial disparities at both local and federal levels. There is evidence that this discriminatory practice led to suppressed property values in redlined areas, reinforced neighborhood segregation, and contributed to the divestment of Black neighborhoods [10, 61, 65, 66]. Consequently, the inability to access equitable and fair loan credit and diminished property values due to redlining appraisals potentially hindered wealth accumulation for marginalized communities and ultimately exacerbated racialized wealth disparities [67].

We posit that structural racism acts as an exposure that accumulates risk clustered across multiple generations through family links, which affects access to social and economic health resources and influences inequities in the risk of diseases, particularly BMI.

Methods

Overview

This study aimed to investigate the long-term effects of redlining on intergenerational outcomes related to wealth and health indicators, particularly BMI. To achieve this, a geographical regression discontinuity (GRD) design was employed to determine whether HOLC-defined discontinuities in perceived lending credit risk levels affect intergenerational wealth and health outcomes. To undertake this study, we utilized the genealogical design of the Panel Study of Income Dynamics (PSID). First, a sample of grandparents (first generation of PSID) for whom the location of their family home was recorded in 1968 was identified. The “treatment” status was assigned by overlaying digitized HOLC category boundaries onto these census blocks and classifying the families as “treated” or exposed to redlining if their census block fell within a D category (red) and as “comparison” if their census block fell within the C category (yellow). The distance from the census block to the nearest D-C HOLC boundary line was calculated and used as the running variable in the continuity-based regression discontinuity analysis.

Data Sources

Panel Study for Income Dynamics: We used data from the PSID, a nationally representative longitudinal survey initiated in 1968 that collects data on various topics, including health and wealth. The survey was conducted annually from 1968 to 1997 and biennially thereafter [68]. Our study used data from 1968 to 2019, spanning a period of 51 years.

The original PSID sample comprised a nationally representative sample and oversampling of low-income families [69]. As of 2017, the PSID sample contained up to 9607 families with almost 81,000 descendants [69, 70]. In 1984, the PSID began collecting data on wealth, incorporating both debt and assets, to calculate household net worth. Health data, such as height and weight, were first collected in 1986 and have been consistently collected with each survey wave since 1999 [71, 72].

Mapping Inequality Redlining Maps: To identify regions graded as C (yellowlined) or D (redlined) by the HOLC, we used the University of Richmond's Mapping Inequality Redlining Maps, which include detailed descriptions of neighborhood-level assessment criteria for 225 US cities [60]. These maps are a digitized repository of paper archive maps, and additional information on the mapping methods used in this study can be found in the "Geographical Method" section.

NHGIS Census: Our study used US Census summary statistics and geographical information system data from the IPUMS National Historical Geographic Information System (NHGIS) [73–75] to conduct falsification analyses. We used decennial census tract data from 1940 to 1960. To reaggregate polygonal data, we used the 2010 census block boundaries to cross-walk those boundaries within tracts from 1940, 1950, and 1960. We then conducted areal weighting to predict the population-level characteristics of the census blocks. This involved downscaling count data to predict the population-level characteristics of the census blocks [76]. The data used included information on total occupied dwelling units, total population, White population, Black population, homeownership rate, median home value, employment rate (males), high school completion rate (males), and vacancy rate.

Study Population: Our study sample consisted of three generations from one family tree, with exposure to redlining practices, as the quasi-experimental variable, in the first generation and wealth and BMI measured in the third generation. We used PSID's family identification mapping system (FIMS) to link children to their biological or adoptive parents, starting with the original 1968 family as the first generation, followed by the second and third generations [68]. Eligibility criteria limited the first-generation sample to those individuals with 2010 census block locations affiliated with their initial 1968 survey responses and who lived in census blocks located in touching HOLC areas categorized as C (yellow) or D (red) across various cities throughout the US. Grandparents without grandchildren and those residing in zones A (green) and B (blue) were excluded from this study. Therefore, as seen in the demographic characteristics (Table 1), the final analytical sample only included those who were third-generation descendants of the first-generation family from the 1968 initiation of the PSID, who had formed their own households, were the reference person or spouse/partner, and had nonmissing values of wealth and BMI.

Measures

Key Exposure: To quantify exposure to structural racism, we utilized PSID to identify grandparents residing in areas classified as HOLC C (yellowlined) or HOLC D (redlined) in the 1960s. We designated grandparents living in yellowlined areas as the comparison group and those residing in redlined areas as the treatment group. Our analysis was further focused on participants who lived in close proximity to a red-yellow HOLC boundary, described in further detail in the "Analytical Method" section.

Outcome — Wealth: Our first primary outcome is the average household wealth accumulated by grandchildren with data in available waves from 1984 to 2019, as represented by the PSID's measure of familial net worth, adjusted for inflation to 2019

constant dollars using the Consumer Price Index (CPI) [77]. All outcome values are expressed in 2019 constant dollars for the study period.

Outcome — BMI: The second primary outcome was the mean body mass index (BMI), which was expressed as a continuous variable based on self-reported weight and height (BMI: weight (kg) / [height (m)]²) [78, 79]. Although BMI is widely used as a simple, cost-effective tool for screening weight status, it is recognized as an imprecise measure of adiposity, often producing false-negatives [80]. Compared to other measures of adiposity, BMI has a high pooled specificity of 0.90 and low sensitivity at 0.50 [81]. Dual energy x-ray absorptiometry (DEXA) is one of the most accurate measures of adiposity [82]. When comparing DEXA to BMI, BMI often underestimates the prevalence of obesity [80]. Although BMI can be imprecise, it is widely used because it is a simple, low-cost tool to measure body fat. The National Institutes of Health (NIH) defines obesity as ≥ 30 BMI kg/m² [83]. However, researchers have indicated that the relationship between BMI and body fat varies by demographics [83, 84]. These differences result in wide variability in BMI thresholds by race and ethnicity, gender, and age. For instance, Black women have lower body fat percentages at a set BMI point than White and Hispanic women and tend to also have higher body weight values [84]. Thus, defining specific BMI cutoff values for body fat by race and ethnicity is more plausibly accurate for determining obesity than current NIH cutoff values. For example, a study conducted by Rahman and Berenson (2010) found varied BMI cutoff values for obesity that were more applicable by race/ethnicity and gender, with ≥ 25.5 kg/m² for White women, ≥ 28.7 kg/m² for Black women, and ≥ 26.2 kg/m² for Hispanic women that are of reproductive age [84]. This difference in obesity thresholds for BMI also potentially translates into differential risks of comorbidities due to higher BMI.

The differences in BMI and obesity values, as well as the differences in risk factors for BMI by race and ethnicity, provide compelling reasons to carefully consider how BMI is applied and categorized in this study. The designation of a general BMI category may not be useful in understanding how obesity operates as a risk factor for disease by race and ethnicity due to exposure to structural racism. Therefore, we employed a continuous variable in our models and expand on the reasons below.

Given the complexity of the BMI tool in terms of accurately measuring adiposity across race, ethnicity, and sex, we chose to keep BMI continuous. Since BMI is a dynamic variable that can be time-varying in a population, we focused our analysis on the BMI distribution rather than ad hoc categorization of BMI values (i.e., underweight, normal, overweight, obese, etc.) [85, 86]. Categorization of continuous variables can lead to misinformation by oversimplifying the data. By keeping the BMI variable as continuous, we retain the full distribution of BMI value outcomes [87–89]. Moreover, at nearly every level, BMI gain is associated with an increase in negative cardiometabolic biomarkers of disease risk [90–92]. Therefore, we used the mean BMI observed in waves between 1986 and 2019 in the PSID as the outcome for grandchildren. Furthermore, we performed additional analyses using the highest BMI observed, and results are presented in the online appendix (Table S4).

Covariates: We included grandchild covariates, such as age, gender, race, ethnicity, and year, in our full models to enhance the precision of our estimates [93]. These covariates are

precision variables that are not causally associated with the exposures and therefore are not confounders [4, 94, 95]. Gender was coded as a dummy variable with females equal to 1, race as a dummy variable with non-Hispanic Black labeled as 1, non-Hispanic White labeled as 0, and age as centered around the mean and specified as continuous. For the construction of gender, race, and ethnicity, we used terms identified by the PSID survey data. In our secondary analysis, stratified by race and ethnicity, we included only the covariates of age, gender, and year in the models.

Study Design: Regression discontinuity design (RDD) is a quasi-experimental technique that employs a cutoff score to assign participants to treatment or comparison groups [96]. By utilizing this threshold, participants who fall above and below the cutoff can be compared to estimate the causal effect of the treatment. The implementation of an RDD study involves three components: cutoff, running variable (or score), and treatment assignment rule.

Using a quasi-experimental approach, we employed a GRD design to identify individuals residing on either side of HOLC C (yellow) and D (red) demarcated lines, which, as previously described, were assigned varying levels of creditworthiness. We included families within close proximity of touching yellowlined and redlined areas due to minimal differences in participant characteristics, except for their residential location. Therefore, we identified individuals residing in designated redlined areas as the treatment group and drew the comparison group from areas classified as one grade higher than HOLC D (redlined)–HOLC C yellowlined areas. To ensure exchangeability, we focused on families residing proximate to the boundary between redlined and yellowlined zones [58, 97, 98]. Exchangeability refers to the assumption that individuals or groups are comparable or interchangeable with respect to the variables under investigation [99, 100]. Given this assumption, we limited the focus of our analysis to HOLC grades C (yellow) and D (red), rather than including HOLC A (green) and B (blue) areas, which often consisted of homogenous racial and ethnic demographics, as well as other neighborhood and housing characteristics.

Our GRD methodology capitalizes on the demarcation of HOLC-designated boundaries, particularly the demarcation between redlined and yellowlined areas, as determined by HOLC assessors. These boundaries do not typically coincide with other boundaries, such as school districts or census tracts [56, 58]. Additionally, residents were usually unaware of the specific HOLC grading assigned to their locality or the delineation between where their graded area ended and an adjacent area with an approximate superior grade, such as the difference between HOLC grade D and C, began [58, 61, 63].

Moreover, while there were differences in average characteristics comparing between entire areas of HOLC C and D grades, our design leverages the fact that the boundary was likely not a perfect demarcation of population or neighborhood difference and particularly so for neighborhood grades of one level up or down, i.e., characterized by sharp jumps in levels. Using block level 1940s characteristics from the US Census, Appel (2016) specifically shows that there were not statistically significant discontinuities in neighborhood characteristics such as rental values, overall racial composition, percent of properties in disrepair, or vacancy rates around the HOLC shared boundaries, which align

with our identifying assumption that other characteristics varied smoothly around these boundaries [58]. Additionally, previous reports have suggested that the HOLC graders often redrew the boundary lines at different places, being unable to decide where the mortgage risk level should change [61]. This evidence supports the use of a regression discontinuity design (RDD) by suggesting that the treatment status changes discretely at the threshold, while other characteristics do not.

Geographical Method: Spatial analysis was conducted using geographical information system tools and techniques. Initially, grandparent locations were mapped using the 2010 census block-level data from the PSID. Subsequently, using census block data, grandparents were overlaid onto their HOLC designated areas using shapefiles made publicly available by the Mapping Inequality Project [60].

We identified grandparents within the HOLC C (yellowlined) and HOLC D (redlined) areas. We then determined which areas had shared borders based on the yellow and red boundaries. We calculated the Euclidean distance (in meters (m)) between each grandparent's census block location and the nearest yellow–red boundary segment line (Fig. 1). We then used distance (m) as the score in our RD analysis, as described in detail below [101, 102]. Finally, in the rare case where a census block fell into two HOLC categories, we assigned the category based on the location of the largest proportion of the block.

Analytical Method

Sharp RD Design: In this study, the HOLC yellowlined and redlined areas, along with their respective borders, served as our exogenous treatment assignment rule. The boundary between these two areas ultimately created the comparison (yellowlined/HOLC C) and treatment (redlined/HOLC D) groups. The sharp regression discontinuity approach is appropriate when the groups receiving treatment and those not receiving treatment are not aware of the specific threshold or cannot take actions to alter their treatment status [103, 104]. This assumption pertains to HOLC ratings, as historical information suggests that while HOLC security maps were known within the housing industry, there is no evidence of their widespread public knowledge or distribution, including among residents in HOLC-graded areas [64]. Therefore, it is reasonable to assume that PSID grandparents, who were not involved in HOLC map design, likely had no knowledge of these designated areas or ability to influence their position relative to these boundaries. A sharp RDD necessitates the selection of an estimation approach, regression function, weighting approach, and optimal bandwidth, as described below.

Continuity-Based Approach: We use a continuity-based regression discontinuity approach, which relies on a sharp or sudden change in a variable of interest, specifically, a policy threshold. This leads to a discontinuous jump in the observed outcome at threshold [105, 106]. We apply this approach by fitting a linear or polynomial regression function to the data separately on either side of the threshold. The difference between the estimated values of the outcome at the threshold for each side represents the local average treatment effect (LATE), which was calculated by comparing the average observed outcomes of the third generation of first generation relatives who are similar in specific characteristics within

a narrow section near the boundary between those who are redlined (treated) and those who are not (comparison) [103, 106]. These estimates provide insights into the causal effects of the policy thresholds.

We define our parameter of interest as follows:

$$\tau = \text{dist}_{x \downarrow c} E[Y_{i_{G3}}^{a=1} | X_{i_{G1}} = c] - \text{dist}_{x \uparrow c} E[Y_{i_{G3}}^{a=0} | X_{i_{G1}} = c]$$

where τ = difference between two intercepts (LATE), cutoff (c) = 0, and i_{G1} = first-generation, i_{G3} = third-generation, dist = distance.

Regression Function and Weighting: To estimate LATE, we applied a local polynomial regression [103, 107]. In addition, we incorporated triangular kernel weights, similar to geographically weighted regression, where the outcomes are a function of weights assigned to observations based on their location relative to the threshold [98, 108]. We used triangular kernel regression in our models to assign the highest weight to the observations closest to the yellow–red boundary. We estimated the effect by fitting two regressions on either side of the boundary for treated (redlined) and comparison (yellowlined) observations and taking the difference between the two regression estimates of the predicted value at $c = 0$.

Given that the smallest level of geographic aggregated data available in the PSID database was the census block level, we applied methods that allowed the analysis of a discrete score. Using the discretized score, we fitted a local polynomial of the outcome as a function of our score and applied clustered standard errors using discrete score values to address the mass points (aggregated units that share the same coordinates) in our score [109–111]. We employed a first-degree polynomial, also known as local linear regression, to our primary regression models [103, 105, 112]. This choice was made because it offered an optimal approximation of the relationship between exposure and outcome variables [113, 114]. Lower-order polynomials, as opposed to higher-order polynomials, mitigate the risks associated with overfitting and erratic behavior near boundaries [103, 115]. A key component in approximating the effect estimate is identifying the bandwidth required to estimate our regressions.

Bandwidth Selection: In a regression discontinuity analysis, bandwidth is utilized to derive the LATE. Specifically, a bandwidth is defined by a specified score range that falls within the full support of the data; this range is used to conduct the estimation and inference process [103, 104, 107]. In this study, two optimal bandwidth methods were implemented: estimation and inference [116, 117]. Calonico et al. (2020) demonstrated that bandwidth methods can balance the bias-variance tradeoff; these bandwidth methods rely on different ranges of the score for optimal point estimation and valid inference [118].

To determine the optimal bandwidth for our analysis, a data-driven approach was employed [104, 105, 119]. We used a mean square error (MSE)-optimal bandwidth procedure, which is dependent on our selection of polynomial and kernel functions, to minimize the mean square of the point estimator [104]. In our study, we report only the effective

observations used in our estimator. An optimal bandwidth was then adopted to produce robust bias-corrected confidence intervals with a minimal coverage error (CER) [105, 118]. CER-optimal bandwidths are centered around the bias-corrected point estimator and use a slightly larger bias-corrected standard error, which enables us to conduct hypothesis testing with minimal probability of errors [116, 118].

Race and Ethnicity Stratified Analysis: To ensure that the overall results did not mask important heterogeneity by race and ethnicity, which might be expected due to racism and its pervasive presence and effects, we conducted a secondary analysis with data stratified by race and ethnicity, namely by Black and other minority groups (i.e., persons of color — POC, including Hispanic) and non-Hispanic White respondents. We used the same outcomes and methods as those used in the primary analysis. We recognize that there were a limited number of individuals across the full support of our data in our race-based stratified samples and interpreted these models with caution. The results from our race- and ethnicity-stratified analyses are available in the online appendix under supplementary material.

Falsification and Validation Tests

Furthermore, we carried out falsification tests to evaluate the plausibility of our assumptions, the validity of our regression discontinuity design, and the robustness of our primary results. Specifically, we implemented three tests: (1) the balance of predetermined covariates, (2) the density of observations across the score, and (3) the sensitivity of observations near the cutoff (all described below). Additionally, we conducted a sensitivity analysis by examining our primary outcome(s) wealth adjusted for family size and BMI using the highest value observed. The details of these analyses are available in the online appendix (Tables S1 and S4).

Predetermined Covariates: We evaluated the distribution of observed covariates before implementing the treatment assignment to determine whether our selected randomization mechanism resulted in a balance of covariates between the treatment and comparison groups [103, 105]. To test our covariate balance, we applied the same methodology as our primary analysis to evaluate the null hypothesis of comparable units and neighborhood characteristics across both the treatment and comparison groups using 1940 census data [105, 120]. Additional predetermined covariates were assessed using the 1950 and 1960 census data and can be found in the online appendix.

Density of the Score: We conducted McCrary's (2008) density test of the score to assess for sorting or any manipulation by the units near the cutoff, employing the same methodology as our primary outcomes [103, 105, 121]. The null hypothesis tested was continuity in the density function across the treatment and control units at the cutoff.

Observations Near the Cutoff (Donut-Hole Approach): To further reinforce the validity of our findings, we performed a sensitivity analysis called the donut-hole approach, using the same methods as in our primary analysis. A donut-hole approach entails excluding observations closest to the threshold to assess whether these observations have an excessive influence on the results of the study [103, 105].

Regression Discontinuity (RD) Plots

We used plots to graphically depict discontinuity in our overall data [103, 107]. The horizontal x -axis corresponds to the score “distance(m),” which signifies treatment assignment, while the vertical or y -axis represents the outcome (average wealth or mean BMI). In an RD plot, a sudden and significant change in the outcome as the variable crosses the cutoff point (which in this case is equal to zero) signifies a pattern in the plot, or discontinuity, as to whether treatment has had an effect on the outcome [103, 105, 107]. The plots are composed of a global polynomial fit to show a smooth approximation of the regression functions and the local means are constructed with mimicking variance using quantile-spaced bins [103, 105, 122]. The quantile-spaced bins ensured consistent observation counts within each bin. The quantile-bin method adapts the bin length based on the data density along the score, resulting in more observations near and fewer away from the cutoff point [105].

Geographical and statistical analyses were performed using RStudio and the RD design was implemented using the `rdrobust` package [123–125].

Results

Sample Characteristics

Our primary analysis involved a sample of 237 grandchildren, who were descendants of 121 grandparents. As shown in Table 1, the sample statistics for both grandparent and grandchild participants were categorized by the HOLC graded classification. Among grandparents identified as heads of households in the PSID, similarities in characteristics exist between those living in HOLC grade C (yellowlined) and HOLC D (redlined) areas. These commonalities include mean age (C 39 years versus D 40 years), the proportion of females (C 48% versus D 43%), the percentage of those in marital or cohabitating relationships (C 50% vs. D 54%), individuals who grew up with parents of low socioeconomic status (C 60% versus D 56%), mean home value (C \$1300 ± \$59,800 versus D \$1300 ± \$9600), and mean labor income (C \$4900 ± \$2800 versus D \$4600 ± \$3100). However, a substantial difference emerged in the racial composition of the yellowlined and redlined areas among grandparent residents. Redlined areas have a higher concentration of Blacks (C 52% versus D 67%) and other racialized ethnic groups (C 2% versus D 8%), coupled with lower education levels (C 45% versus D 23%) than the yellowlined areas.

Compared to grandchildren whose grandparents resided in yellowlined regions, grandchildren whose grandparents lived in redlined areas exhibited a lower likelihood of attaining a college degree (C 35% vs. D 21%) and a higher likelihood of not completing high school (C 8% vs. D 23%) (Table 1). Furthermore, grandchildren with grandparents from redlined areas tend to have lower mean family income (C \$71,000 ± \$63,000 vs. D \$48,000 ± \$39,000) and wealth (C \$82,000 ± \$190,000 vs. D \$53,000 ± \$230,000), but a higher average BMI (C 26 kg/m² ± 4.2 kg/m² vs. D 28 kg/m² ± 6.1 kg/m²). However, the proportion of grandchildren with financially well-off parents was similar in both yellowlined (27%) and redlined areas (31%).

RD Plots — Primary Outcomes

Figure 2 displays RD plots for the primary outcome, wealth. A noticeable discontinuity emerges at the threshold for wealth outcome. Figure 3 presents RD plots for mean BMI outcomes. A small discontinuity arises at the threshold when examining the sample variance along the score. In general, when comparing the outcomes of grandchildren in redlined and yellowlined areas, the RD plots reveal an abrupt change in both average wealth and mean BMI.

Average Household Wealth

Table 2 details the findings on average household wealth for the study. The results from the models show a substantial, statistically significant reduction in household wealth across generations of grandchildren whose grandparents lived in redlined areas compared with those in yellowlined areas. The unadjusted model (1a) indicates that having a grandparent who lived in a redlined area versus a yellowlined area is associated with a lower family wealth of \$96,100 (95% CI_{rbc} – \$243,710, – \$23,539). The adjusted models (2a and 3a), which include age, calendar year, and gender, yielded similar results with narrower confidence intervals (Table 2). The fully adjusted model(4a), which also included race and ethnicity, revealed a similar association, but lower in magnitude, indicating a persistent association between grandparent residence in a redlined area and their grandchildren having lower household wealth by – \$35,419 (95% CI_{rbc} – \$37,423, – \$7,615) compared to yellowlined area grandchildren. Further wealth analyses, considering family size adjustments, revealed a similar pattern (Table S1). Additional race- and ethnic-stratified analyses are available in the online appendix (Table S2).

Mean Body Mass Index

Results of the local linear regression models with the mean BMI as the continuous outcome are presented in Table 3. Overall, when examining the relationship between HOLC grade location for grandparents and the mean BMI of grandchildren, we observed that the mean BMI was notably elevated in our treatment group — grandchildren with a grandparent residing in a redlined region, in contrast to the comparison group — grandchildren with a grandparent living in a yellowlined region. The unadjusted model 1b reveals a higher mean, but not significant, BMI ($\beta = 5.16 \text{ kg/m}^2$; 95% CI_{rbc} – 2.54 kg/m², 9.81 kg/m²) for redlined grandchildren versus yellowlined grandchildren. The associations in models 2b and 3b are positive, yet not statistically significant. The fully adjusted model(4b) includes covariates age, year, gender, and race/ethnicity and reveals a substantial effect size that is much greater than the observed effect size of other models ($\beta = 7.46 \text{ kg/m}^2$; 95% CI_{rbc} – 4.00 kg/m², 16.60 kg/m²). This suggests a consistent association between grandchildren having a grandparent residing in a redlined neighborhood and an overall higher mean BMI than those with a grandparent residing in a yellowlined neighborhood. Race- and ethnicity-based stratified and highest BMI analyses are available in the online supplementary material.

Falsification and Validation Test

Predetermined Covariates: Table 4 presents the results of the census-level neighborhood 1940s covariate balance tests. All neighborhood-level characteristics

determined prior to treatment assignment failed to reject the null hypothesis of comparable predetermined covariates across the treatment and comparison groups, which provides evidence that there are no systematic differences among neighborhoods for grandparents, ultimately validating our RD design. A visual representation of our covariate balance analysis is presented in Fig. 4. Additional predetermined covariate assessment results using census data from 1950 and 1960 can be found in the online supplementary material (1950 — Table S5, 1960 — Table S6).

Density Test: Figure 5 displays histogram of the density of the score for the grandchildren's sample, with the number of observations greater on the treatment (redlined side) than the comparison (yellowlined side) group. The formal analysis identifies that we fail to reject the null hypothesis that the density of the score is continuous at threshold for the full dataset [105]. Therefore, there is no evidence of “sorting” near the neighborhood around the cutoff in our sample. The statistic was -1.072 , and the associated p -value was 0.284 (Table 5). Therefore, the number of observations was consistent with what would be expected for the treatment mechanism, particularly given the fact that the PSID oversampled the Black population in the initial 1968 enrollment of the PSID study [69, 126].

Donut-Hole Approach: Results from the donut-hole approach, a sensitivity analysis used to validate our results, show that our conclusions from our primary analyses for wealth and BMI are robust to the exclusion of observations within 10 m (Table 6). The point estimates are in the same direction for both average wealth and mean BMI and continue to be statistically significant for our wealth variable. Moving further out to 50 m for the donut-hole approach led to fewer observations for our wealth analyses. For BMI, we observed very far bandwidths for both the estimate and inference, and an effect estimate that is in the opposite direction, yet still remains insignificant.

Discussion

In this study, we explored the factors contributing to racial disparities in intergenerational wealth and BMI outcomes in the US. These findings suggest that the historical discrimination in the housing and real estate markets experienced by PSID grandparents has had a persistent and far-reaching impact on subsequent generations. Our findings suggest a plausible causal relationship between grandparents' exposure to redlining and lower intergenerational wealth accumulation by their descendants, compared to those whose grandparents lived in areas designated as yellowlined. We also observed a lasting generational effect of redlining on BMI, which is an important health marker. Although the study findings lacked statistical significance, our data support a possible connection between grandparents who resided in a redlined area and higher mean BMI in their grandchildren, compared to those with grandparents who lived in yellowlined areas.

Wealth

Our study, which employs a quasi-experimental approach, aligns with existing research on the racialized wealth gap. Previous research has shown that the racial wealth gap cannot be attributed to differences in saving rates, educational attainment, income, or

labor opportunities among racialized groups but is intricately linked to structural racism [127–129]. To shed light on this, we leveraged the HOLC mortgage loan security maps to investigate the effects of codified neighborhood-level structural racism on wealth. We capitalize on the longitudinal nature of the PSID to capture genealogical and cumulative wealth data, a robust approach supported by the literature for studying the racialized wealth gap [52]. While our analysis included individuals of all races whose grandparents had lived in redlined versus yellowlined neighborhoods, previous literature has demonstrated that Black families were substantially more likely to have lived in neighborhoods graded red by HOLC. Furthermore, the difference in wealth between redlined and yellowlined grandchildren demonstrates that redlining likely contributes to the racialized wealth gap [58, 62]. In our fully adjusted model (4a), which incorporated race as a precision variable, we observed a smaller disparity in average household wealth between redlined and yellowlined grandchildren. However, it is unclear from our data how including race as an additional adjustment affected our effect size. Nonetheless, our study establishes a plausible causal relationship between discriminatory policies in the US and the manifestation of the racialized intergenerational wealth gap, making this a significant contribution to the literature.

Killewald et al. (2017) highlight several methodological concerns when studying wealth inequality and accumulation, including the unexplained differences in wealth compared to income levels and the use of transformation when analyzing highly skewed measures such as wealth [52]. Our research addresses these concerns by utilizing a historical policy lever to implement a natural experiment that examines the effect of wealth accumulation over multiple generations, accounting for debt and zero net worth by averaging wealth over multiple years [130], and using an intergenerational framework to analyze the effects of historical redlining practices on the accumulation of household wealth. Research on intergenerational wealth indicates that wealth acquired by grandchildren through early life investments, such as access to advantageous neighborhoods, homeownership, and other forms of social and cultural capital, may account for the racial wealth gap as much as direct gifts and bequests [52, 131]. Our findings demonstrate the solidifying effects of historical disadvantages on contemporary inequities in intergenerational wealth accumulation and social mobility between Black and White populations, ultimately strengthening the argument that social origins and historical structural racism have lasting effects on wealth outcomes across multiple generations.

Body Mass Index

Previous research has found that wealth is inversely associated with obesity [34]. Our study design provides an opportunity to delve deeper into the relationship between wealth and BMI disparity. By examining the impact of generational exposure to structural racism on BMI outcomes, we discovered a greater disparity in BMI outcomes for grandchildren whose grandparents were exposed to redlining compared to those with grandparents in yellowlined areas. Additionally, studies have shown a positive relationship between housing segregation, socioeconomic status (SES), and BMI, particularly among Black women [29, 132–134]. These findings are consistent with the outcomes of our primary and racially stratified models, albeit our results fall short of achieving statistical significance. Our

research suggests that race-based segregation of neighborhoods, operating over multiple generations, may shape the social and economic environment of communities, particularly those composed mostly of Blacks and other historically disadvantaged groups, leading to neighborhoods that are disproportionately exposed to obesogenic environments. We maintain that race-based bifurcation of neighborhoods in the US, perpetuated and cemented by discriminatory HOLC maps, has contributed to systematic disinvestment in health-promoting neighborhood amenities and the emergence of obesogenic environments. We speculate that these factors likely contributed to poorer health outcomes, as indicated by higher BMI in the current study and the documented worse physical and mental health outcomes for racially segregated minority neighborhood, as identified by Lynch et al. (2021) [10]. Our work advances research on the causal link between BMI and neighborhood-level structural racism. Since redlining was based on the credit risk assigned to neighborhoods, this policy likely had far-reaching impacts on the economic and social trajectories of neighborhood resources.

Limitations

This study has several limitations that must be considered. First, the regression discontinuity design utilized in this study was limited to individuals who were closer to the border of the HOLC red and yellow thresholds, which restricted our ability to generalize our findings to those who were farther away. Additionally, the use of fine-scaled geocoded data, such as address or longitude/latitude, is ideally suited for regression discontinuity design, and we were unable to utilize such data because of technical and privacy limitations [98]. Furthermore, we did not have a precise location for the grandparents and used aggregated areal units that may not accurately reflect the spatial variation in the units being measured [98]. To address these geographical limitations, we utilized the most granular spatial census unit available, census blocks, to identify the proximal location of the first-generation PSID participants in the HOLC areas.

Another limitation is the limited sample for our racially stratified analyses; we were unable to obtain disaggregated data for all racialized groups, particularly those classified as “Other” within the PSID, which limited our ability to understand the impact of redlining on these groups. Additionally, the density of our data for participants on yellowlined side, particularly for the BMI analysis, was sparse.

Finally, we acknowledge the use of body mass index as a measure of obesity is suboptimal, although it is the only available measure of obesity currently captured by the PSID [135, 136]. Additionally, since we lacked adequate data on information that may influence BMI such as medication and pregnancy, we used an average of BMI for outcome. The use of body mass index (BMI) as a measure of adiposity and its association with morbidity and mortality has been extensively studied, particularly regarding its application across different racial and ethnic groups. Studies, such as those conducted by Seo and Torabi (2006) and Jackson et al. (2014), have shed light on racial disparities in BMI and its relationship with health outcomes, especially among Black individuals [137, 138]. While Seo and Torabi’s (2006) findings revealed a higher mean BMI among non-Hispanic Black women compared to non-Hispanic White women, Jackson et al. (2014) suggested a weaker association between

BMI and mortality risk in Black individuals, particularly Black women, compared to their White counterparts. Several factors may contribute to this observed difference, including the possibility that BMI may be a weaker indicator of adiposity in Black populations [138]. Moreover, structural racism and disparities in access to healthcare and resources may exacerbate the impact of obesity-related conditions on minority populations [139]. The study by Park et al. (2012) further emphasizes the complexity of BMI-mortality associations across ethnic groups, indicating variations in the strength of these associations and underscoring the importance of considering early adulthood BMI and ethnic-specific pathways to obesity-related diseases [140]. Furthermore, researchers have highlighted the differential trends in obesity-related cardiovascular mortality by race, sex, and place of residence, underscoring the need for targeted structural interventions to address disparities [141].

Overall, while BMI remains a widely used measure of adiposity, its usefulness across diverse racial and ethnic populations warrants careful consideration, taking into account variations in body composition, healthcare access, and societal factors. Despite these variations, there is strong evidence that generally high BMI (rather than directly measured adiposity) is highly correlated with a multitude of chronic diseases and is highly correlated with directly measured adiposity among non-elderly adults [80, 81, 142, 143]. Future research should continue to explore the complex interplay between BMI, race, and health outcomes to inform more tailored structural approaches to obesity prevention and healthy weight management in minority communities.

Conclusion

The intricate relationship between structural racism, economic inequities, and population health is the central focus of this study. Structural racism perpetuates the idea that racialized minorities, particularly Black people, are inferior and detrimental to everyone. While discriminatory redlining practices were primarily aimed at excluding Black families from participating in the economic benefits from homeownership, these policies may have had broader effects on other racialized groups with grandparents residing in redlined areas, including White populations. Our primary objective is to investigate the foundations of intergenerational disparities in wealth and health. We employ empirical evidence to scrutinize the significant role of federal policies in the creation and exacerbation of structural racism and inequalities. Our research findings lend credence to the notion that racial disparities in wealth accumulation and potentially body mass index can be attributed to the historical policy of redlining, which is a prime example of structural racism. This historical policy has left a legacy of intergenerational harm with profound implications for marginalized racial groups. Further research is imperative to advance our efforts to address the underlying structural causes of these inequities. It is essential to shed light on the most effective strategies for implementing and assessing social and economic structural interventions aimed at mitigating the harm experienced by Black and other racially marginalized communities.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Evaluates the role of redlining on present-day inequities in wealth and BMI.
- Examines impact of structural racism using quasi-experimental method.
- Reveals historical discrimination persists, affecting wealth gaps for generations.
- Highlights need to address and rectify generational impact of structural racism.

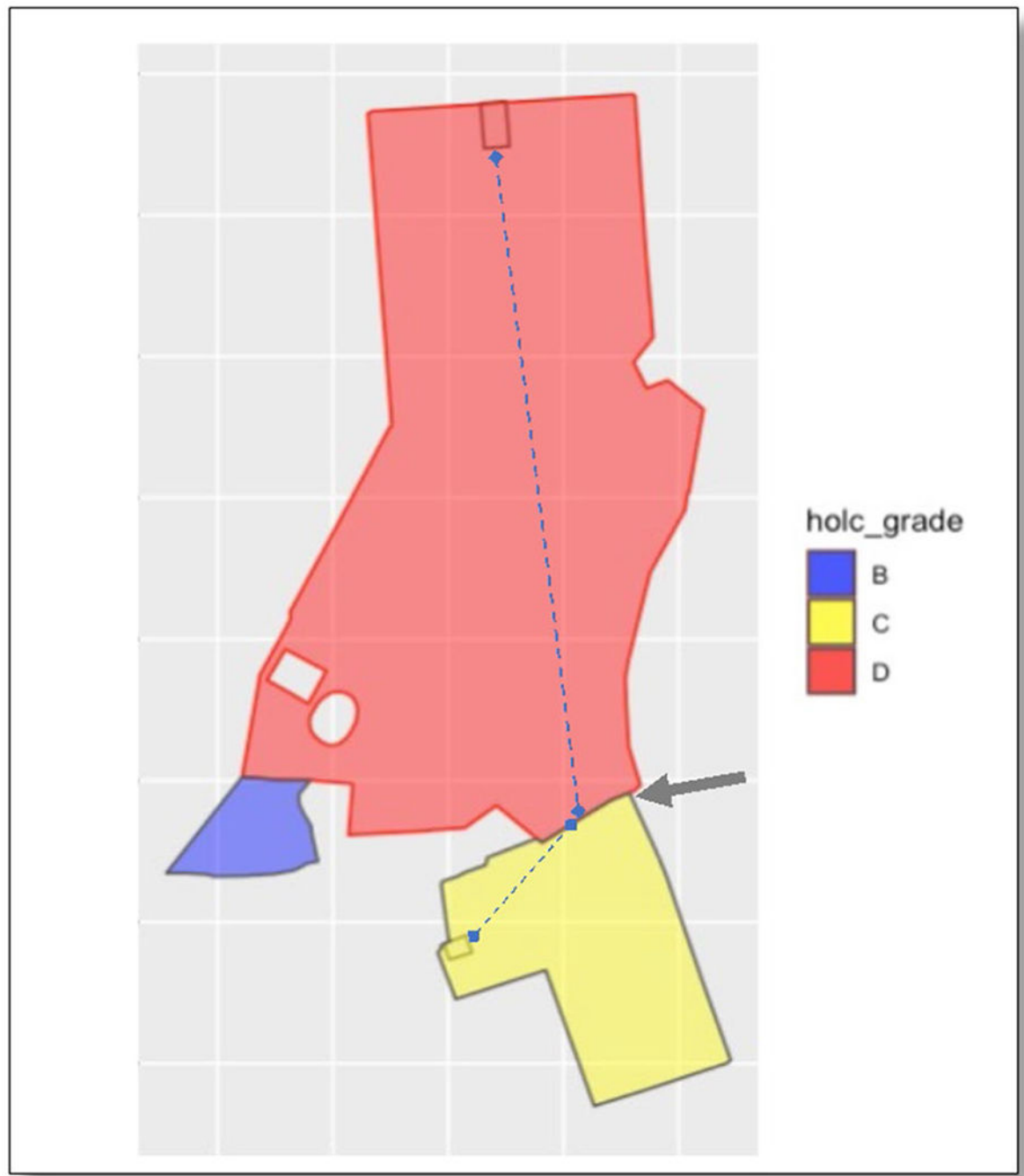


Fig. 1.

A hypothetical PSID grandparent census block within an HOLC grade D and grade C area with calculated distance from polygon to touching red-yellow border

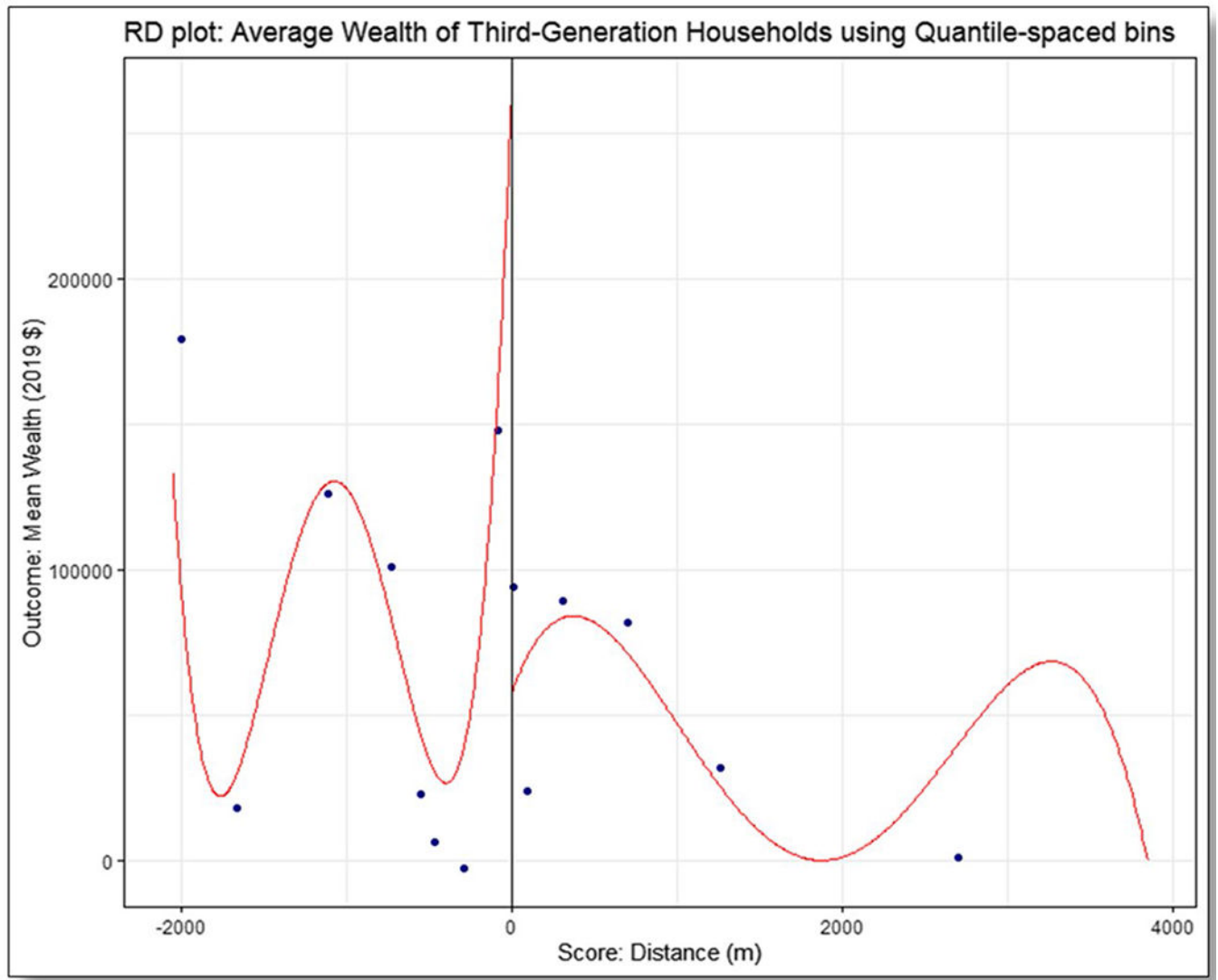


Fig. 2. Regression discontinuity (RD) plot: Average wealth of grandchildren households using quantile-spaced bins. Regression discontinuity (RD) plot using quantile-spaced bins and triangular kernels

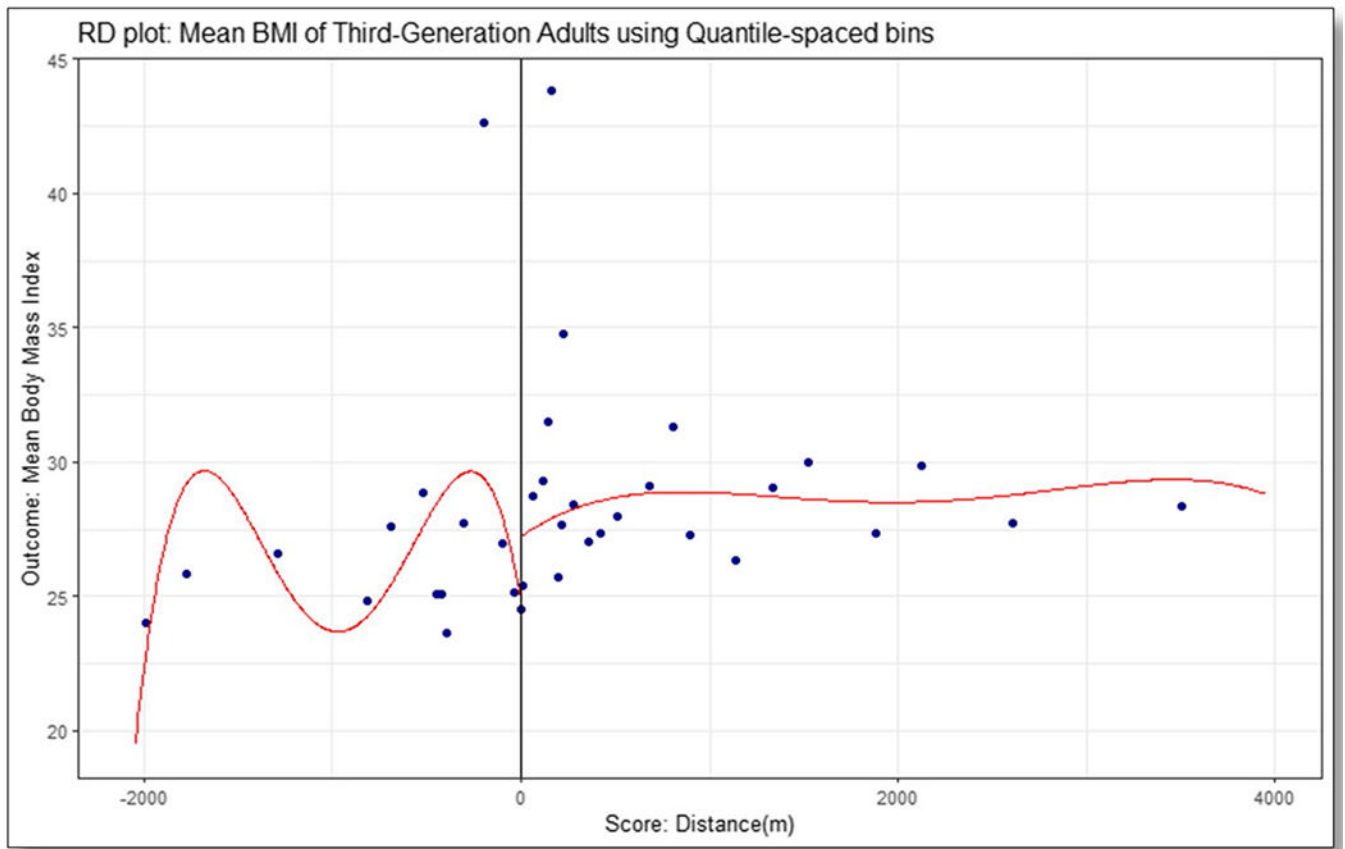


Fig. 3. Regression discontinuity (RD) plot: Mean body mass index in grandchildren adults using quantile-spaced bins. Regression discontinuity (RD) plot using quantile-spaced bins and triangular kernels

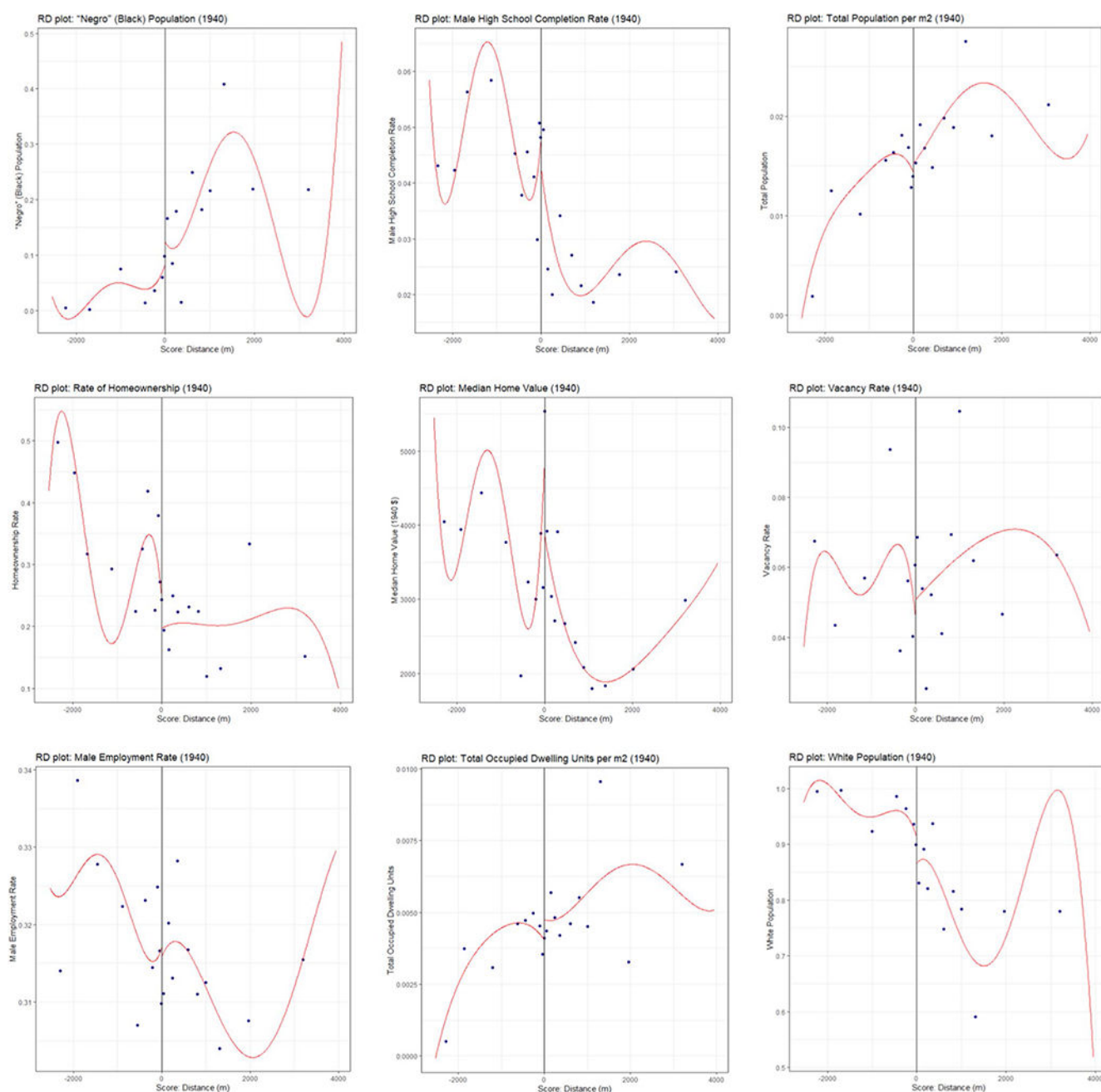


Fig. 4.
Regression discontinuity (RD) plot: 1940 predetermined covariates using quantile-spaced bins

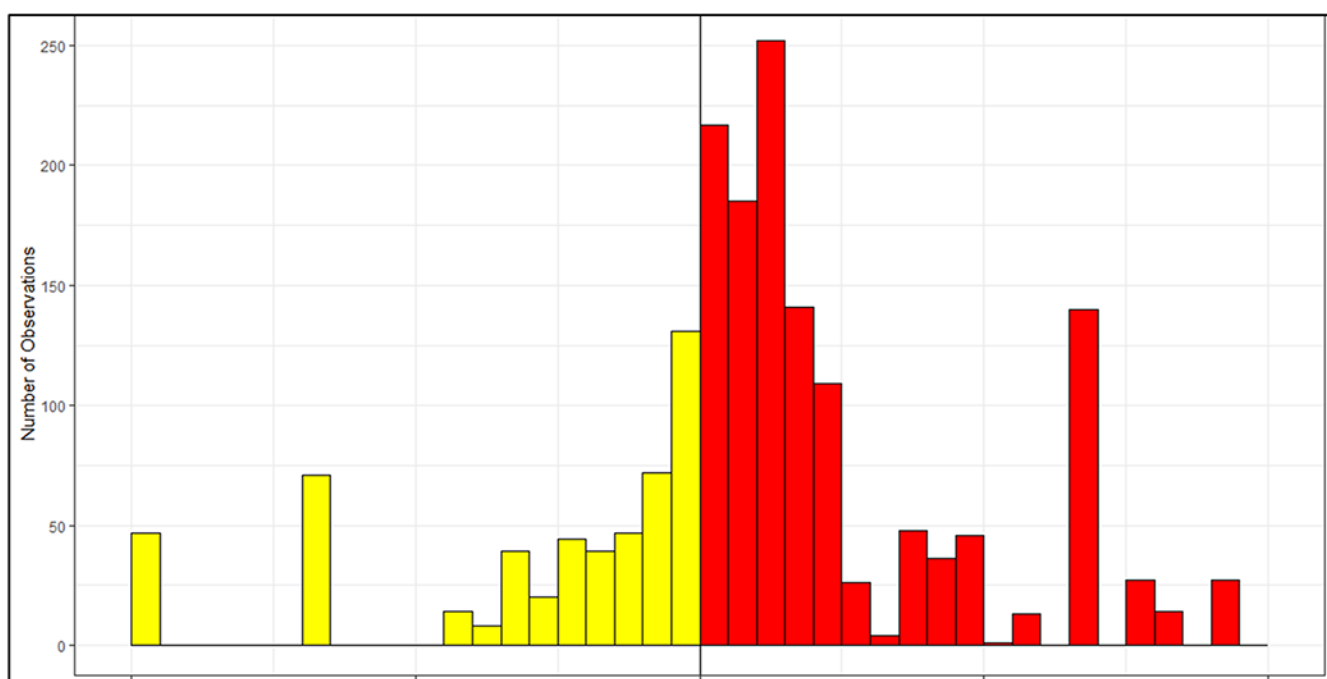


Fig. 5.
Density plots: sample size of grandchildren for average household wealth and mean BMI

Table 1

Grandparent and grandchildren characteristics

	HOLC grade C (yellowlined)	HOLC grade D (redlined)	Total
Grandparents (head of households)			
<i>n</i> (%)	<i>N</i> = 40	<i>N</i> = 81	<i>N</i> = 121
Age mean(sd)	39 (± 12)	40 (± 10)	40 (± 11)
Gender			
Male	21 (52%)	46 (57%)	67 (55%)
Race			
“Negro” (Black)	21 (52%)	54 (67%)	75 (62%)
Other	0 (0%)	2 (2%)	2 (2%)
Puerto Rican/Mexican	1 (2%)	5 (6%)	6 (5%)
White	18 (45%)	19 (23%)	37 (31%)
Marital status			
Divorced	1 (2%)	6 (7%)	7 (6%)
Married/cohabit	20 (50%)	44 (54%)	64 (53%)
Separated	11 (28%)	18 (22%)	29 (24%)
Single	5 (12%)	8 (10%)	13 (11%)
Widow	3 (8%)	5 (6%)	8 (7%)
Education			
< High school	22 (55%)	55 (68%)	77 (64%)
High school	13 (32%)	23 (28%)	36 (30%)
Some college	1 (2%)	3 (4%)	4 (3%)
College	2 (5%)	0 (0%)	2 (2%)
Labor income mean(sd)	4900 (± 2800)	4600 (± 3100)	4700 (± 3000)
Parents’ SES status			
Poor	24 (60%)	45 (56%)	69 (57%)
Pretty well off	5 (12%)	14 (17%)	19 (16%)
Grandchildren			
<i>n</i> (%)	<i>N</i> = 52	<i>N</i> = 185	<i>N</i> = 237
Age mean(sd)	30 (± 5.1)	28 (± 5.4)	29 (± 5.3)
Gender			

	HOLC grade C (yellowlined)	HOLC grade D (redlined)	Total
Male	25 (48%)	90 (49%)	115 (49%)
Race			
Black	28 (54%)	147 (79%)	175 (74%)
Other	0 (0%)	7 (4%)	7 (3%)
White	24 (46%)	31 (17%)	55 (23%)
Ethnicity			
Hispanic	1 (2%)	10 (5%)	11 (5%)
Marital status			
Divorced	14 (6%)	2 (4%)	12 (6%)
Married/cohabit	88 (37%)	29 (56%)	59 (32%)
Separated	13 (5%)	2 (4%)	11 (6%)
Single	117 (49%)	17 (33%)	100 (54%)
Widow	5 (2%)	2 (4%)	3 (2%)
Education (head)			
< High school	4 (8%)	43 (23%)	47 (20%)
High school	14 (27%)	51 (28%)	65 (27%)
Some college	13 (25%)	53 (29%)	66 (28%)
College	18 (35%)	38 (21%)	56 (24%)
Parents' SES status			
Average	28 (54%)	61 (33%)	89 (38%)
Poor	9 (17%)	62 (34%)	71 (30%)
Pretty well off	14 (27%)	58 (31%)	72 (30%)
Housing status			
Owens (or buying)	23 (44%)	44 (24%)	67 (28%)
Rents	28 (54%)	131 (71%)	159 (67%)
Neither	1 (2%)	10 (5%)	11 (5%)
BMI mean(sd)	26 kg/m ² (± 4.2)	28 kg/m ² (± 6.1)	28 (± 5.8)
Family income mean(sd)	\$71,000 (± 63,000)	\$48,000 (± 39,000)	\$53,000 (± 47,000)
Wealth equity mean(sd)	\$82,000 (± 190,000)	\$53,000 (± 230,000)	\$59,000 (± 220,000)

First-generation (grandparents) labor income is expressed in 1968 dollars; third-generation (grandchildren) family income and wealth equity are expressed in 2019 constant dollars; education is highest level attained

Table 2
Continuity-based RD analysis: effect of redlining on grandchildren’s average household wealth using covariate-adjusted local polynomial regression

N = 173	RD estimator (\$)	MSE-optimal bandwidth [meters]	Robust inference		CER-optimal bandwidth [meters]	N/	Nr
			95% CI _{rbc}	SE			
Outcome: Wealth (mean)							
Model 1a	- 96,104	828.83	[- 243,710, - 23,539]	56,167	685.95	19	90
Model 2a	- 105,199	807.05	[- 285,228, - 3432]	71,888	667.93	19	86
Model 3a	- 95,124	984.23	[- 260,168, - 23,875]	60,280	814.56	22	94
Model 4a	- 35,419	399.49	[- 37,423, - 7615]	7604	331.79	7	65

Discrete analysis using cluster standard errors; bandwidth is the distance (in meters) from yellow/red border to grandparent’s census block; adjusted models; 2a — age, year; 3a — age, year, gender; 4a — age, year, gender, race; all models use a first-degree polynomial; N/(left) and Nr(right) indicate the effective number of observations within the MSE bandwidth used for estimation; wealth is rounded to the nearest whole dollar.

* $p < 0.10$;
** $p < 0.05$;
*** $p < 0.01$

Table 3
Continuity-based RD analysis: effect of redlining on the mean BMI in third-generation adults using covariate-adjusted local polynomial regression

N = 210	RD estimator (kg/m ²)	MSE-optimal bandwidth [meters]	Robust inference			CER-optimal bandwidth [meters]	NI	Nr
			95% CI _{Rob}	SE	p-value			
Outcome: Body Mass Index (mean)								
Model 1b	5.16	290.89	[- 2.54, 9.81]	3.15	0.249	239.21	11	69
Model 2b	5.37	314.26	[- 2.29, 10.73]	3.32	0.204	258.43	11	69
Model 3b	5.92	405.77	[- 2.08, 13.86]	4.07	0.148	333.68	13	86
Model 4b	7.46	374.44	[- 4.00, 16.60]	5.26	0.231	308.87	10	78

Discrete analysis using cluster standard errors; bandwidth is the distance (in meters) from yellow/red border to grandparent's census block; adjusted models: 2b — age, year; 3b — age, year, gender; 4b — age, year, gender, race; all models use a first-degree polynomial; NI/(left) and Nr(right) indicate the effective number of observations within the MSE bandwidth used for estimation

* $p < 0.10$;
** $p < 0.05$;
*** $p < 0.01$

Table 4
Continuity-based RD analysis: predetermined census 1940 covariates using local polynomial regression

Covariates	Coefficient	CER-optimal bandwidth [meters]	Robust inference		N/	Nr
			95% CI _{hc}	SE		
Total occupied dwelling units/m ²	0.00	589.729	[0.00, 0.003]	0.00	0.439	57 88
Total population/m ²	0.001	566.81	[- 0.006, 0.007]	0.00	0.802	57 84
White population	- 0.037	552.109	[- 0.146, 0.087]	0.06	0.621	57 84
“Negro” population	0.04	562.387	[- 0.09, 0.146]	0.06	0.607	57 84
Homeownership rate	- 0.07	411.152	[- 0.17, 0.054]	0.06	0.308	47 75
Median home value	- 706.36	528.97	[- 2494.90, 806.892]	842.31	0.316	56 83
Male employment rate	0.00	573.186	[- 0.02, 0.009]	0.01	0.601	57 85
Male high school completion rate	0.01	396.638	[- 0.01, 0.019]	0.01	0.361	45 73
Vacancy rate	0.01	484.644	[- 0.02, 0.035]	0.01	0.428	54 80

Discrete analysis using cluster standard errors; all models use first-degree polynomial; median home value is rounded to the nearest whole dollar

* $p < 0.10$;
** $p < 0.05$;
*** $p < 0.01$

Table 5

Continuity-based approach density test

<i>Number of available observations = 657</i>			
<i>n (left)</i>	<i>n (right)</i>	<i>Statistic</i>	<i>p-value</i>
173	484	– 1.0723	0.2836

Point estimate and standard errors are based upon the full range of data

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Table 6
Continuity-based analysis: primary outcomes applying the donut-hole approach

Donut-hole radius	RD estimator	MSE-optimal bandwidth [meters]	Robust inference		CER-optimal bandwidth [meters]		N/ Nr
			95% CI _{rb}	SE	p-value		
Outcome: average household wealth (\$)							
10	- 157,882	593.148	[- 345,971, - 10,895]	85,480	0.037**	488.513	17 66
50	- 328,490	497.881	[- 814,696, 171,742]	251,647	0.201	732.984	7 42
Outcome: mean BMI (kg/m ²)							
10	34.16	288.62	[- 18.14, 75.76]	23.95	0.229	239.14	9 49
50	- 0.298	852.939	[- 5.14, 6.07]	2.859	0.87	707.55	27 79

Discrete analysis using cluster standard errors; bandwidth is the distance (in meters) from yellow/red border to grandparent's census block; fully adjusted model including age, year, gender, race; all models use a first-degree polynomial; N/ (left) and Nr (right) indicate the effective number of observations within the MSE bandwidth used for estimation; wealth is rounded to the nearest whole dollar

* $p < 0.10$;
** $p < 0.05$;
*** $p < 0.01$