Investigating the Intergenerational Impact of Redlining on Intergenerational Wealth, Employment Quality, and Obesity: A Quasi-Experimental Study

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A dissertation

submitted in partial fulfillment of the

requirements for the degree of

Doctor of Philosophy

University of Washington

2024

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Program Authorized to Offer Degree:

Health Services

University of Washington

Abstract

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Obesity, the second leading cause of preventable deaths in the United States (U.S.), disproportionately impacts marginalized communities especially those impacted by systemic racism. Redlining, a form of structural racism, is a practice by which federal agencies and banks disproportionality assigned high credit risk and less favorable loan terms to neighborhoods where predominantly racialized minorities lived, contributing to residential segregation. Communities affected by structural racism are more likely to live in unhealthy, obesogenic environments. This dissertation employed data from the Panel Study of Income Dynamics (PSID) and the Mapping Inequality project to implement a quasi-experimental design, known as a geographical regression discontinuity design, to explore the generational impact of the Home Owners' Loan Corporation (HOLC) redlining policies on wealth, employment, and body mass index (BMI) outcomes. The study's specific aims included: (1a) identifying the impact of neighborhood-level structural racism on intergenerational wealth accumulation, (1b) testing the

effect of neighborhood-level structural racism on BMI over multiple generations; (2) examining the relationship between neighborhood-level structural racism and employment quality; and (3) assessing the mediating role of intergenerational wealth and employment quality on the relationship between structural racism and BMI outcomes in adults. The study's findings indicated that the grandchildren of individuals who had resided in redlined neighborhoods exhibit lower average household wealth, consistently lower quality employment outcomes, and higher mean BMI measurements when compared to their peers (grandchildren with a grandparent who resided in yellow-lined areas). Moreover, the study showed no evidence of a mediating generational effects of grandparents' experience with redlining on grandchildren's mean BMI through the intergenerational wealth and employment quality of grandchildren. These findings suggest that policymakers should invest in further research that targets understanding the consequences of historical discrimination and explore policies aimed at rectifying intergenerational harm to promote restorative justice for families impacted by discriminatory federal policies such as redlining.

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ACKNOWLEDGMENTS

I would like to begin by extending my deepest gratitude to my dissertation committee members for your time, energy, and support. Each of you provided mentorship that was instrumental in helping me complete this work. Your guidance gave me the courage, knowledge, and skills necessary to carry out this research. I would especially like to thank my dissertation chair, Jesse Jones-Smith. From the very beginning, you stood by my side and believed in my ability to conduct this research. Thank you for being both a collaborator and a mentor, turning this proposed research into reality. Dr. Paul Fishman, thank you for being a constant source of guidance and wisdom throughout my time at UW. Dr. Anjum Hajat, from the moment I met you, you inspired me. I cannot thank you enough for believing in me and for being the kind of person and researcher I aspire to be. Dr. Edmund Seto, this dissertation would not have been possible without your willingness to support me through my GIS journey, without expecting anything in return. I am honored and grateful that you were happy to help me complete this work. Finally, to my Graduate School Representative, Ahoua Koné, you have become such a pivotal part of my experience and Seattle family. Thank you for your kindness and encouragement over the past five years.

Thank you to my cohort, the Fab5—Ammarah Mahmud, Anna Localio, Carolyn Fan, and Maddie Frost. It has been a wonderful journey having you all by my side. Our cohort dates brought me so much joy and happiness, and I am forever grateful to have had you with me on this journey. Thank you to my peer mentors, Andrew Bossick, Meagan Brown, and Canada Parrish, who cheered me on every step of the way.

Thank you to the faculty, students, and staff at SPH (especially Dr. Emily Williams, Emily Bernet, Gloria Rayo, Dr. Jeanne Sears, Lindsay Pysson, Mary Saucier, Ria Francisco, Michele Apo, Dr. Hilary Godwin, and Suzanne Yates) for your dedication to my success throughout my PhD experience. The guidance you provided to a first-generation student like me has been invaluable, and I deeply appreciate that you all went above and beyond for me. For my SPH Aunties—JeShawana Schmidt, Juanita Ricks, Ahoua, Victoria Gardner, and rukie hartman—I am forever grateful for the laughs, hugs, tissues, and love. You all kept my heart going when I wanted to give up.

A huge thank you to all the funders of awards and training grants that made my doctorate possible. Thank you to the Department of Health Systems and Population Health for the Health Systems Excellence, Equity Distinction (HSEED) Award. Thank you to the National Institute for Occupational Safety and Health (T42OH008433) for the Occupational Health Services Training Grant, which funded me through three years of my program. Thank you to the UW Office of Graduate Student Equity & Excellence (GSEE) Dissertation Fellowship for supporting the final stages of my dissertation writing. Additionally, thank you to the National Institutes of Health, National Institute on Minority Health and Health Disparities (1F31MD017449-01) pre-doctoral grant, and to my co-sponsors on my F31 award, Dr. Jones-Smith and Dr. Hajat, for supporting the development of the grant, including the heavy load of paperwork. Lastly, thank you to the research participants and staff at the Panel Study of Income Dynamics for the use of their data for this work.

Finally, to all of my close friends and family, I am grateful for the love and graciousness you have shown me over the years. To my parents, Shurlene and Eric, and my stepmom Lilian, thank you for always supporting and believing in me every step of the way. To my big cousins, I am grateful to have you in my life. You have shown me what it truly means to be family, and I am forever thankful for your unwavering support.

DEDICATION

This work is dedicated to my grandparents for sacrificing so that I can dream. And to my dear friend, Emily Somers, whose love and laughter will forever be carried in my heart. ♡

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.

CHAPTER 1. Introduction

1.1 Overview

Obesity increases the risk for developing chronic conditions and is a contributing factor to preventable deaths in the United States (U.S.) (1,2). Risk for obesity has been previously associated with various social determinants of health (SDOH), including neighborhood disadvantage, socioeconomic status (SES) and racialized status. For example, research has shown that racialized minority groups and those with lower socioeconomic status (SES) are more likely to have higher rates of obesity (3–7).

Emerging research on racialized health disparities indicates historical redlining, which was a manifestation of structural racism that excluded individuals, primarily racialized minorities, from obtaining federally-insured mortgages, has a significant impact on SDOH, such as economic opportunities, educational attainment, and neighborhood resources (8–11). These studies highlight that the remnant of redlining is associated with present-day health inequities (8–11). According to Oliver and Shapiro, racialized policies may contribute to differentials in wealth building opportunities, such as steady employment and homeownership, and they assert that these disparities in wealth accumulation can be seen as contemporary evidence of the "sediment of racial inequality" as it captures the legacy of organizational discrimination and institutionalized racism (12). Research on wealth inequality and obesity demonstrates that those in lower wealth quintiles, when compared to the wealthiest quintile groups, have a 40% to 89% higher risk of developing obesity (13). The trend in wealth inequality and obesity disparities underscores the need for thorough assessments of the causal role of structural racism in wealth accumulation and other SDOH, such as employment, across multiple generations and its impact on the risk of obesity.

Studies examining the connection between employment characteristics and obesity show that employment-related stressors, such as job insecurity, employer-employee power relations and shift work, act as risk factors for increased body mass index (BMI) (14–18). Research shows observable demographics such as educational attainment, do not explain the higher and more cyclical unemployment and underemployment among Blacks compared to Whites (19,20), pointing to a need to understand how structural racism influences precarious employment conditions, and decipher their impact on health outcomes (21,22).

There is high theoretical potential for redlining to explain disparities in obesity, through multiple pathways, including diminished wealth accumulation, inadequate access to high- quality employment opportunities, and neighborhood deprivation. However, to the best of our knowledge, no studies have investigated the extent to which redlining contributes to obesity disparities among marginalized communities over the life course, as well as the mechanisms by which this relationship is mediated by intergenerational wealth accumulation and employment quality outcomes.

Therefore, this research aims to elucidate the part "redlining" – a particular form of structural racism that involves classifying neighborhoods based on race-based credit risk levels, thereby leading to discriminatory mortgage lending practices (23,24) – plays in fostering disparities in wealth and employment quality, as well as BMI outcomes (25).

1.2 Background

Inequalities in obesity

Obesity has been conclusively linked to heightened risk of several serious chronic conditions including kidney disease, hypertension, diabetes, cardiovascular disease, osteoarthritis, stroke, and certain preventable cancers (26–31). The National Institutes of Health

(NIH) defines obesity as ≥ 30 BMI kg/m^2(32). BMI is widely used as a simple, cost-effective tool for screening weight status. However, BMI is recognized as an imprecise measure of adiposity and often produces false-negatives (33). Compared to other adiposity measures, BMI has high specificity (0.90) but low sensitivity (0.50) (34). Dual energy x-ray absorptiometry (DEXA) is a more accurate measure of adiposity (35). BMI often underestimates obesity prevalence when compared to DEXA (33). Despite its imprecision, BMI is widely used due to its simplicity and low cost. Additionally, BMI can also potentially translate into differential risks of comorbidities due to higher BMI.

Obesity poses a significant burden on individuals and healthcare systems. Recent estimates, indicate that approximately 70% of adults in the U.S. have obesity or are overweight (36,37). Obesity is a significant cost burden, with the direct and indirect obesity-related expenses in the U.S. totaling an estimated $140 billion annually (38–41). The substantial health and financial burden of obesity underscores the urgent need for effective solutions to address obesity and related disparities. There is growing consensus among experts in the field of obesity research that upstream factors should be the primary focus of these solutions (42–44).

Disparities in obesity by racialized groups are marked, yet only a small body of literature has interrogated the role of structural racism or segregation in the creation of obesity disparities (45–50). Here, we define structural racism as historical, sociopolitical systems that reinforce inequitable treatment on the basis of race (51,52). For instance, Dougherty et al. (2020), conducted a study that used five domains of discrimination, including education, housing, employment, criminal justice, and health, to create a county-level index of structural racism. Their results revealed higher index scores, indicating greater county-level structural racism, were

associated with increased BMI for Blacks, whereas for their White counterparts it was associated with a decrease in BMI (46).

It is worth noting that among the few studies that have examined the connection between structural racism and obesity, most do not consider the multidimensional nature and long-term social, economic, and health consequences of structural racism over an individual's lifespan (46). As a result, these studies are unable to determine the extent to which racial disparities in BMI persist over time (6,46,48,53–56). Structural racism may impact obesity risk by causing a lack of investment or disenfranchisement of racially segregated areas that primarily house Black or other racialized minority residents, resulting in fewer health-promoting and more obesogenic factors (57,58). This study is of particular significance as it addresses the gap in the literature by examining the relationships between structural racism and BMI across multiple generations and identifying the pathways that may contribute to obesity disparities.

Structural racism, wealth accumulation, and obesity.

Discriminatory structural forces permeate throughout social and economic systems, and they often result in disparate resources that influence health outcomes. Many studies show that having lower (vs higher) income or educational attainment is associated with a higher prevalence of obesity (4,5,38,59–63). Studies have also shown that having lower wealth is associated with a higher prevalence of obesity, although measures of wealth have been limited and have not examined intergenerational aspects of wealth accumulation (64). Relatively few studies explore the relationship between obesity and wealth, as defined by net worth, (63–66) and even fewer measure wealth over multiple generations (67). This ultimately neglects the generational or life course approach to wealth and health (38,66–68). Furthermore, additional research indicates that

structural racism and wealth may be connected to obesogenic environments as well as home values (45,69).

In a study by Drewnowski et al., (2015), it was observed that areas near crime, liquor stores, and fast-food establishments had lower property values (70). Due to difficulties in obtaining low-interest loans, many families residing in redlined neighborhoods faced challenges in building wealth through homeownership. For a significant portion of Americans, home values constitute a substantial part of their portfolio assets, accounting for around 29% of middle-class Americans' wealth (69,71–73). Structural racism is also hypothesized to diminish household wealth for racialized minorities due to lower property values in segregated areas (74). Neighborhoods characterized by racial segregation and a higher proportion of Blacks and other minority populations tend to have lower home values, and limited financial resources to fund neighborhood amenities such as safe walkable areas, green space, and healthy food access (45,75–78). The result of this divestment is the creation of obesogenic environments, which are characterized by elements that promote obesity rather than healthier weight (45,76,79–81). Although there is evidence to support the notion that structural racism, through neighborhood segregation and divestment, contributes to the racial wealth gap, no study has yet used redlining policies as a specific measure of structural racism to quantify its contribution of the generational wealth accumulation gap (82,83).

Structural racism, employment quality, and obesity.

There is a compelling reason to investigate the relationship between structural racism and employment quality (EQ), and its subsequent influence on obesity – employment serves as tangible focal point where structural and social institutions operate as a fundamental cause to produce health inequities (18,84). Structural racism operates by limiting opportunities to jobs

with Standard Employment Relationships (SER) conditions, which are a basis for employment quality and are characterized by consistent, full-time employment with wages and benefits along with control over time and labor processes (85,86). Historically, due to systemic discrimination, women and people of color have been left out of the SER labor market (87–89).

Employment quality embodies the various attributes of a job that contribute to the overall well-being and satisfaction of the employee (90). This includes factors such as job security, income level, working conditions, opportunities for career advancement, work-life balance, job stability, benefits, and the degree of respect and fairness in the workplace (86). High employment quality typically enhances an individual's economic security and overall quality of life, whereas low employment quality can contribute to stress, instability, and various negative health outcomes (91,92). However, there is a paucity of evidence on how exposure to structural racism is associated with employment quality, and to our knowledge no current studies explore how structural racism and precarious employment (i.e., worse, or lower employment quality) contribute to inequality in BMI levels.

Neighborhood-level structural racism is a distal sociopolitical force that is hypothesized to shape the unequal distribution of personal resources or "opportunities" to obtain standard employment quality (93). This phenomenon also contributes to a racialized labor market, which is part of a larger systemic problem that results in worse quality employment for racialized minority groups. This subsequently leads to health disparities, such as obesity, by increasing stress and reducing access to health-promoting assets among certain populations (86,94). There are multiple features of employment that are associated with an individual's ability to accumulate health advantages over their lifetime including, health insurance, vacation, career trajectory, control of time, prestige, and social connectedness, (18,84,86,95). More specifically, there are

several domains that play a significant role in determining the pathways by which employment quality affects health outcomes, these encompass employment stability, material rewards, work-time arrangements, collective organization, and employer-employee power relations (96,97), including unequal BMI outcomes in disenfranchised populations (98).

Precarious employment is often a sign of poor employment quality and is characterized by employment insecurity, unpredictable work schedules, limited opportunities for career advancement, such as training or promotion, low wages, and minimal to no benefits, protection, or power (22,86,99). Long-term exposure to precarious employment can have negative effects on both physical and mental health (22,97–101). The primary mechanisms through which precarious employment leads to disparities in health outcomes include material deprivation, psychosocial stress, and exposure to workplace hazards (102). Furthermore, macro-level discriminatory systems, such as redlining, contribute to social stratification, which in turn leads to unequal exposure to lower levels of employment quality, resulting in health (102). The current study seeks to bridge the existing gap in the fields of population health, health disparities, and occupational health by investigating the direct role of neighborhood-level structural racism on employment quality outcomes, while simultaneously exploring EQ as a determinant of health on the mechanistic (or causal) pathway connecting structural racism and BMI outcomes.

1.3 Study Aims

Data Source

We leveraged the data from the Panel Study on Income Dynamics (PSID) to implement our quasi-experimental design and intergenerational approach. Initiated in 1968, the PSID spans across five generations of families, and encompasses wealth and health data over multiple waves

(103). Additionally, it includes geolocations of households across the U.S., which allows us to assess neighborhood-level structural racism in the form of redlining**.**

Additionally, we use shapefile data from the Mapping Inequality project, which digitized the Home Owners' Loan Corporation (HOLC) discriminatory maps. In the 1930s HOLC, a government-sponsored agency under the Federal Housing Authority (FHA), developed security maps designed to assign 239 of America's largest cities a grade to determine their perceived credit risk level based upon the characteristics of a neighborhood (24,104–106). The grading scale was as follows: A (green – "Best" *deemed the lowest credit risk*), B (blue – "Still Desirable"), C (yellow – "Definitely Declining"), D (red – "Hazardous" *deemed highest credit risk*) [Figure 1.1] (24,106). Neighborhoods comprising of a higher proportion of African Americans or Blacks were graded "hazardous" or graded D (i.e., red) (10,24).

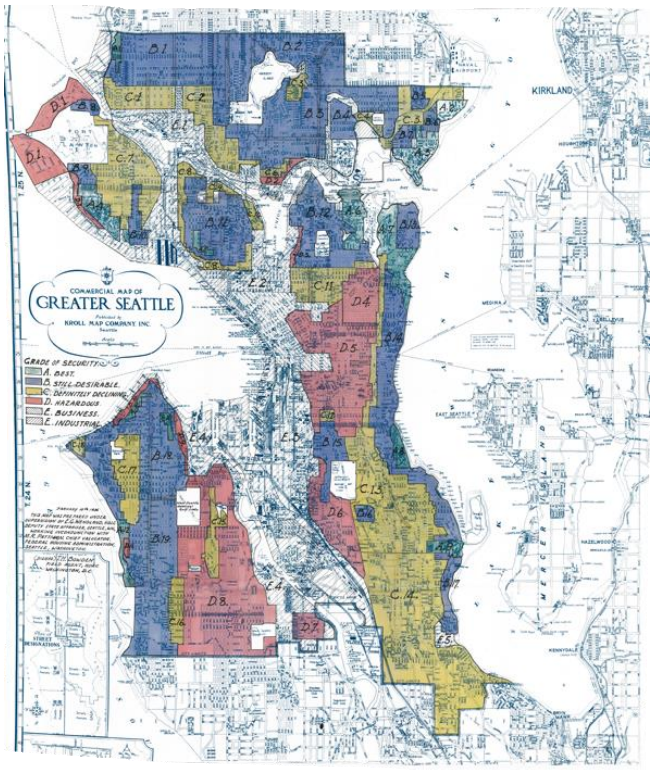


Figure 1.1. Example of Home Owners' Loan Corporation Graded Security Maps: Greater Seattle Area

Our central hypothesis for this research study is federally supported racialized "redlining" policies effectively prohibited marginalized families, particularly Black Americans, from accumulating wealth in their homes, thereby hindering inheritance for future generations and suppressing property values. This has resulted in several decades of divestment in predominately Black neighborhoods, limiting pathways to access opportunities such as high-quality employment, and ultimately increasing the risk of obesity over the course of one's life. In order to examine this overarching hypothesis, we have identified three study aims.

For Aim 1 (Chapter 2), we sought to investigate the effects of neighborhood-level structural racism, specifically redlining, on (a) intergenerational wealth accumulation and (b) body mass index. We posited that grandchildren with a grandparent who resided in the lowest

HOLC grade D – redlined region, as opposed to a yellow-lined one (i.e., HOLC C), would exhibit lower levels of intergenerational wealth accumulation and higher mean BMI.

In Aim 2 (Chapter 3), we examined the connection between redlining and employment quality. Our hypothesis posited that exposure to structural racism in the form of redlining, particularly among grandchildren whose grandparents resided in HOLC D (or redlined areas) compared to those in HOLC C (yellowlined areas) would be associated with higher precarious employment (i.e., low employment quality) for grandchildren.

For Aim 3 (Chapter 4), the mediating influence of intergenerational wealth and employment quality in the connection between redlining and high BMI outcomes in third-generation adults was evaluated. It was hypothesized that a) there exists a mediating effect of intergenerational wealth on the association between redlining and BMI outcomes, and b) there exists a mediating effect of employment quality on the association between redlining and BMI.

1.4 Theoretical Premise

The premise of this research study is guided by multiple theoretical approaches including, Public Health Critical Race Praxis (PHCRP), fundamental cause theory, cumulative inequality theory, and life course theory.

Public Health Critical Race Praxis

First, we incorporate Ford and Airhihenbuwa's praxis for antiracism research (107), which applies Critical Race Theory, by utilizing an antiracism lens to evaluate public health and health equity research (107,108). Public Health Critical Race Praxis (PHCRP) is an iterative methodology that gives a guiding framework for scientists investigating the impact of structural racism or racism-related factors on the risk of disease in communities of color (94,109). PHCRP is born out of Critical Race Theory which establishes a methodology for scholars to illuminate

causes of racial injustice through four principles – 1) race consciousness, 2) contemporary orientation, 3) centering in the margins, and 4) a praxis for application (108,110). PHCRP has four phases and 10 principles. We center on the elements that specifically apply to this research – two essential phases of PHCRP, (Focus 1) contemporary patterns of racial relations and (Focus 2) knowledge production, and three principles: primacy of racialization, race as a social construct, and structural determinism (107).

Using PHCRP's contemporary racial relations (Focus 1), we identify key characteristics of the racialization for the study's period (107). Since the manner in which racism operates in a racialized society changes over time it is important to establish the conceptualization of racism based upon how it operated during the period the study took place. During the era of the sample's exposure, initially experienced by the first-generation PSID participants (i.e., grandparents). Any person with African descent or heritage would have been considered a Negro by the U.S. Census (111). This is due to the one-drop rule which effectively consider any person with a single drop of "Black (or African) blood" a Negro by the Census from 1930 until 1970 (when the term Black was introduced) (111–113). Knowledge production (Focus 2) is another important focus incorporated into this study's design. Knowledge production identifies the norms for producing empirical research within a field to avoid any inadvertent biases that may arise from racialized disciplinary conventions (107).

While staying within the framework of scientific and statistical methods is vital to ensure quality research, it is also critical to examine how constructs, concepts, knowledge, and methodologies can be rooted in racialized disciplinary conventions. Therefore, we take a critical approach to each disciplinary field applied to this study and allow voices from various disciplines and communities to inform the construction of the study aims, design, and analysis.

For instance, research has shown the typical designated obesity categorization of BMI to be inaccurate for racialized groups (25,33,114). Therefore, to avoid use of a categorical variable with cutoff points for various levels of obesity (32), we implement BMI as a continuous measure in our models.

Finally, we apply three other principles from PHCRP, primacy of racialization, race as a social construct, and structural determinism, to this research (107). By acknowledging race is a social construct, yet it has many implications in social, economic, and political arenas (51). In other words, we do not focus on studying race as a risk factor or exposure, and instead use structural racism in housing policies to examine the discriminatory basis for which race is used to exclude certain populations for accessing federally-funded resources (115). Likewise, rather than identifying interpersonal factors related to racism, we specifically call out and identify how historical structural racism at the federal level, through Home Owners' Loan Corporation (HOLC) racial discriminatory loan security maps, is a cause of cumulative, persistent harm through time (i.e., through generational wealth) and across different settings (i.e., employment quality).

Fundamental Cause Theory

Second, we use two premises from fundamental cause theory by Phelan and Link. Link and Phelan (1995) coined the term "fundamental causes" of disease to identify access to macrosocial resources that help individuals prevent risk and maintain their health through various mechanisms. Identifying intervening mechanisms that help to explain the causal direction between exposures and health outcomes, clarifies how distal factors such as social and economic conditions exert indirect effects on risk of disease (84). Link and Phelan (1995) highlight how without a basic understanding of how exposure to social conditions leads to

individually-based risk factors, interventions will consistently fail to improve population health (84).

In 2015, Phelan and Link posit that racism is a fundamental cause of health inequalities by race due to its connection with producing racial differences in SES (116). This research study's hypothesis builds off their concept of racism as a fundamental cause of health inequities. We hypothesize that structural racism, in and of itself, is independently connected to health outcomes, in particular BMI, over generations and that racism operates through various mechanisms to produce disparities. We argue that these resources not only advantage the health of privileged, often White, populations, but that racialized minorities were systematically prohibited from accumulating these health promoting resources resulting in generations of health inequities. We continue to build upon this foundational premise through recognizing that health inequities happen over space and time. Following this thought process, cumulative inequality and life course theory are also incorporated into this work.

Cumulative Inequality Theory

Third, we use cumulative inequality theory to highlight how inequalities are generated by social systems and manifested over time (or the life course) and are not a result of individual choices. From conception macrosocial forces shape life and have the greatest influence during childhood in shaping adult outcomes (117). Consequently, familial lineage and structural forces play a significant role in understanding disadvantage and the accumulation of inequality.

According to cumulative inequality theory, disadvantages and advantages are not opposite sides of one coin, instead they should be thought of as social positions in a hierarchy (117). Under this theory, disadvantage is defined as an unfavorable position on the societal hierarchical level due to structural determinants increasing the probability of exposure to risk.

This risk results in a negative outcome and perpetuates further disadvantage and increased risk (117,118). Life course trajectories are not only shaped by the accumulation of risk but by available resources and human agency (117). Although inequality may accumulate over the course of life, resources and agency play a critical role in shaping life trajectories. Cumulative inequality theory informs the development of our mediation analysis (Aim 3 – Chapter 4) by illuminating how health promoting resources are on the causal pathway between structural racism and BMI disparities, and to illuminate how racism plays a role in influencing an individual's agency for access to health promoting resources.

Life Course Theory

Fourth and final, we utilize life course theory, an approach that takes a broad view over time and encompasses impact of both biological and social transmissions that happen over generations allowing researchers to understand the influence of early-life precursors to disease in adulthood (119). By taking a life course approach we are able to use an interdisciplinary framework that focuses on the long term, intergenerational effects of structural racism on later health outcomes or disease risk. Focusing on three main concepts in life course theory - an intergenerational life course approach, accumulation of risk, and chains of risk - to expand on the premise of how structural racism can be cumulative over time and effectively chain risks together to influence social inequalities that ultimately produce health inequities over multiple generations.

This study focuses on a causal pathway framework where the exposure is experienced by the grandparent and the outcome by the grandchild. This allows us to specifically apply the life course intergenerational approach to understanding population determinants of health and puts the life course approach into historical context. Using life course theory, we can elucidate how

socioeconomic factors act cumulatively and interactively throughout generations to influence the health of an individual over their life course (120). Accumulation of long-term exposure(s), such as structural racism, may result in long-term damage and produce differential health effects for specific groups. The accumulation model with risk clustering due to linked lives assess how the effects of an exposure can persist over multiple generations and poses as an accumulation of risk that cluster within a family system (119). We posit that structural racism acts as an exposure that accumulates risk that are clustered across multiple generations through family links which impact access to social determinants of health (i.e., resources such as, wealth and employment) that influence inequities in risk for disease (i.e., BMI).

Rutter (1989) put forth the idea of a chains of risk (or protective chains) where a sequence of linked exposures over time either raises (or lowers) disease risk, depending on whether the exposures are positive or negative, this is also known as the pathways model (121). Chains of risk may be social, economic, biological, and/or psychological and can be mediating factors between the exposure and health outcome. This pathways or chains of risk model incorporates mediators that can act as an additive risk or protective factor in the development of the causal relationship between the exposure and health outcome.

To illustrate this for this study, exposure to structural racism happens in the first generation (or grandparents) which raises risk of disease over each generation. In other words, exposure to structural racism by the first generation may compound the effects on mediating factors, namely wealth and employment quality, over each generation, and ultimately adding chains of risk for disparities in obesity-related health outcomes across each generation. In other words, exposure to structural racism in one family not only independently affects health risk, but it also increases the risk of subsequent additional exposures to risk through mediating factors.

1.5 Conceptual Model

Theoretical premises outlined above inform the development of our conceptual model, which illustrates the hypothesized causal pathways of each aim (or chapter) [Figure 1.2]. The first hypothesized causal pathway (p1a – Aim 1a) shows the link from structural racism (redlining) experienced by grandparents to intergenerational wealth outcomes in grandchildren [Aim 1a, Chapter 2]. Next, we examined the causal pathway (p1b – Aim 1b) between structural racism and a health risk factor (BMI) [Aim 1b, Chapter 2]. The second causal pathway (p2 – Aim 2) displays exposure of neighborhood-level structural racism to employment quality outcomes in grandchildren [Aim 2, Chapter 3]. Finally, for the mediation pathway (p3 – Aim 3), we hypothesized that intergenerational wealth and employment quality mediates the relationship between structural racism and BMI outcomes (in the third generation) [Aim 3, Chapter 4].

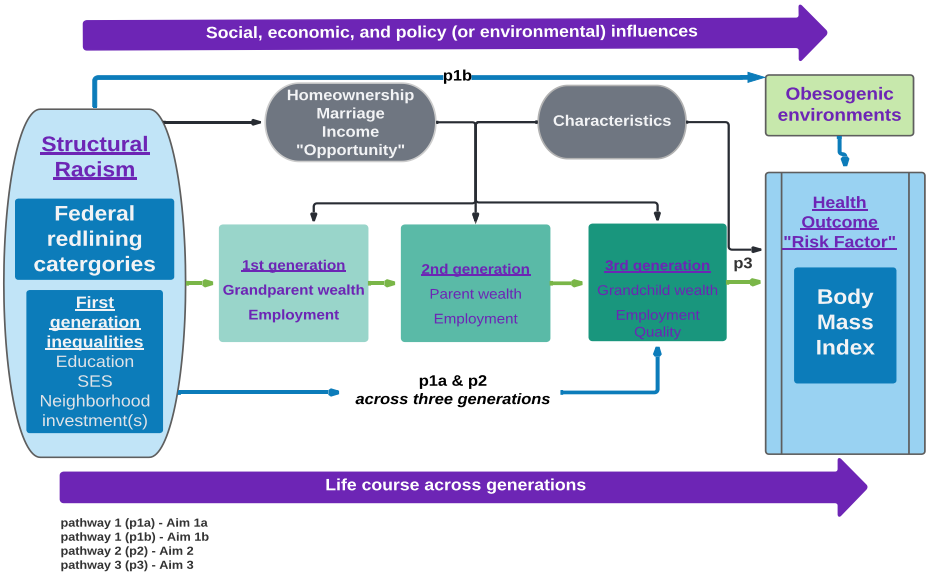


Figure 1.2. Conceptual Model Illustrating the Causal pathway of Structural Racism on Generational Wealth, Employment Quality, and Body Mass Index Outcomes (Aims 1-3)

1.6 Summary

This research examines the intricate relationship between structural racism, social determinants of health (SDOH), and obesity, focusing on the ongoing impact of historical policies, such as redlining, on present-day health disparities. Our study concentrates on obesity as a significant health risk factor, as it is linked to various chronic conditions and preventable deaths, which disproportionately affect racial and ethnic minority groups and those with lower socioeconomic status. Recent research indicates that redlining, a manifestation of structural racism, influences SDOH, including wealth accumulation and employment quality, which subsequently contribute to obesity disparities. However, previous studies often overlook the multidimensional and long-term effects of structural racism across the life course, failing to fully capture the persistence of racial disparities in health outcomes and social determinants of health.

This research seeks to address this gap by investigating the role of redlining in generating wealth and employment quality inequities, and consequently obesity disparities, across generations. Employing data from the Panel Study on Income Dynamics (PSID) and Mapping Inequality project, the study explores intergenerational wealth accumulation, employment quality, and BMI outcomes among racially marginalized communities.

Our theoretical frameworks inform our choice to use a quasi-experimental design and an intergenerational approach to explore and illuminate the complex pathways through which structural racism influences health outcomes. In the subsequent chapters, we detail how we implemented these approaches to elucidate the causal relationships between redlining, intergenerational wealth, employment quality, and BMI and contribute to a deeper understanding of the mechanisms underlying social, economic, and health inequities.

CHAPTER 2. Assessing the Influence of Redlining on Intergenerational Wealth and Body Mass Index through A Quasi-Experimental Framework

2.1 Introduction

In the United States (U.S.), 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 (115,122–125). Research has investigated the influence of social determinants of health (SDOH) on racial health disparities (126–128), revealing an association between historical redlining – a form of codified structural racism that restricted mortgage access to neighborhoods predominately inhabited by racialized minorities, and elevated rates of breast cancer mortality, preterm birth, and poor physical and mental health outcomes (8,9,11,129).

Moreover, Nardone et al. (2021) reported evidence of reduced greenspace in historically redlined areas (130). These studies suggest that the legacy of redlining continues to reinforce current health inequities. Considering the persistent and profound racial inequalities in the U.S., 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).

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 U.S. (2). In addition, it increases the risk of developing several chronic conditions, including kidney disease, hypertension, diabetes, cardiovascular disease, osteoarthritis, stroke, and certain preventable cancers (26–28,131,132). Approximately 41.9% of adults have obesity, with 9.2% having severe obesity (133). Obesity imposes a significant financial burden to U.S. healthcare systems, with annual obesity-related medical expenditures estimated at $170 billion (134).

The risk of obesity or a high body mass index (BMI) 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) (3–5,7). Disparities in obesity among racialized groups are marked, however, only limited literature has investigated the role of structural racism in creating these disparities (45,46,49,50,135,136). 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 (57,58,130). 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 (137). 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 (46,60). Although some studies have shown that individuals with

lower wealth have a higher prevalence of obesity (64,138,139), even fewer have measured wealth accumulation over multiple generations (67). 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 (67,140).

Research indicates a possible connection between structural racism and wealth, which may be linked to obesogenic environments and home values (45,69). Drewnowski et al. (2015) found that areas near crime, liquor stores, and fast-food stores were associated with lower property values (70). 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 (73). Structural racism is also hypothesized to decrease household wealth for racialized minorities because of lower property values in segregated areas (74,141), which may contribute to a lack of health-promoting amenities, such as safe walkable areas, green spaces, and healthy food access (45,75–77,79,130,142). 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 (75,130,143). 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 (82,83).

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) (144), 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 (145). The grading scale ranged from: A (green – "Best" *deemed the lowest credit risk*), B (blue – "Still Desirable"), C (yellow – "Definitely Declining"), D (red – "Hazardous" *deemed the highest credit risk*) (144–149). 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 (150). 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) (148). The deployment of HOLC maps for assessing mortgage risk has been outlawed since the mid-1970s (144,151), but the legacy of this practice is still evident in the persistent residential segregation and long-run decline of once redlined neighborhoods throughout the U.S. (145,148,152).

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 (145,148,152,153). 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 (145,148,152,153). 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 (154). These ordinances aimed to block lower-income African Americans from middle-class White neighborhoods and impede middle-class African Americans from purchasing homes there (154). 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 (9,151,155,156). 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 (157).

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.

2.2 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 (103). 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 (158). As of 2017, the PSID sample contained up to 9,607 families with almost 81,000 descendants (158,159). 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 (160,161).

Mapping Inequality Redlining Maps

To identify regions graded as C (yellow-lined) 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 U.S. cities (150). 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 U.S. Census summary statistics and geographical information system data from the IPUMS National Historical Geographic Information System (NHGIS) (162–164) 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 crosswalk 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 (165). 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, as the quasi-experimental variable, practices 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 (103). 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 U.S. 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 non-missing 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 (yellow-lined) or HOLC D (redlined) in the 1960s. We designated grandparents living in yellow-lined 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 1984-2019, as represented by the PSID's measure of familial net worth, adjusted for inflation to 2019 constant dollars using the Consumer Price Index (CPI) (166). 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}x 703]) (167,168). 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 (33). Compared to other measures of adiposity, BMI has a high pooled specificity of 0.90 and low sensitivity at 0.50 (34). Dual energy x-ray absorptiometry (DEXA) is one of the most accurate measures of adiposity (35). When comparing DEXA to BMI, BMI often underestimates the prevalence of obesity (33).

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/m^2(32). However, researchers have (83,84) indicated that the relationship between BMI and body fat varies by demographics. 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 tend to have higher body weight values (25). 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 for White women, ≥28.7 for Black women, ≥26.2 for Hispanic women of reproductive age (25). These differences in obesity thresholds for BMI 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.) (169,170). 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 (171–173). Moreover, at nearly every level, BMI gain is associated with an increase in negative cardiometabolic biomarkers of disease risk (174–176). Therefore, we used the mean BMI observed in waves between 1986-2019 in the PSID as the outcome for grandchildren.

Covariates

We included grandchild covariates, such as age, gender, race, ethnicity, and year, in our full models to enhance the precision of our estimates (177). These covariates are precision variables that are not causally associated with the exposures, and therefore are not confounders (115,178,179). 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 (180). 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 yellow-lined and redlined areas due to minimal differences in participant characteristics, except for their residential location. Therefore, we identified individuals residing in designated redlined as the treatment group and drew the comparison group from areas classified as one grade higher than HOLC D (redlined) – HOLC C yellow-lined areas. To ensure exchangeability, we focused on families residing proximate to the boundary between redlined and yellowlined zones (148,181,182). Exchangeability refers to the assumption that individuals

or groups are comparable or interchangeable with respect to the variables under investigation (183,184). Given this assumption, we limited the focus of our analysis to HOLC grades C and D, rather than including HOLC A (green) and B (blue) areas, which often consisted of different 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 yellow-lined areas, as determined by HOLC assessors. These boundaries do not typically coincide with other boundaries, such as school districts or census tracts (146,148). 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 (148,151,153).

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 U.S. 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 aligns with our identifying assumption that other characteristics varied smoothly around these boundaries (148). 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 (151). 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 (150).

We identified grandparents within the HOLC C (yellow-lined) 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 (Figure 1). We then used distance(m) as the score in our RD analysis, as described in detail below (185,186). 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 yellow-lined 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 (yellow-lined/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 (187,188). 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 (154). 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 (189,190). 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 (control) (187,190). These estimates provide insights into the causal effects of the policy thresholds.

We define our parameter of interest as:

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

Where, $\tau = difference\ between\ two\ intercepts\ (LATE)$*, 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 (187,191). 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 (182,192). 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 (yellow-lined) 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 (193–195). We employed a first-degree polynomial, also known as local linear regression, to our primary regression models (187,189,196). This choice was made because it offered an optimal approximation of the relationship between exposure and outcome variables (197,198). Lower-order polynomials, as opposed to higher-order polynomials, mitigate the risks associated with overfitting and erratic behavior near boundaries (187,199). 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 (187,188,191). In

this study, two optimal bandwidth methods were implemented: estimation and inference (200,201). 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 (202).

To determine the optimal bandwidth for our analysis, a data-driven approach was employed (188,189,203). 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 (188). 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) (189,202). 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 (200,202).

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 data.

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 cut-off (all described below). Additionally, we performed a sensitivity analysis by analyzing our primary outcome wealth adjusted for family size, the details of which are provided in the online appendix (Table S1).

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 (187,189). 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 (189,204). 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 (187,189,205). 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 (187,189).

Regression Discontinuity (RD) plots

We used plots to graphically depict discontinuity in our overall data (187,191). The horizontal x-axis corresponds to the score "distance(m)," which signifies treatment assignment, while the vertical or y-axis represents the outcome (wealth or 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 plot, or discontinuity, as to whether treatment has had an effect on the outcome (187,189,191). The plots are comprised 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 (187,189,206). 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 cut-off point (189).

Geographical and statistical analyses were performed using RStudio and the RD design was implemented using the rdrobust package (207–209).

2.3 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 (yellow-lined) 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:$1,300±$59,800 versus D:$1,300±$9,600), and mean labor income (C:$4,900±$2,800 versus D:$4,600±$3,100). However, a substantial difference emerged in the racial composition of the yellow-lined 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 yellow-lined areas.

Compared to grandchildren whose grandparents resided in yellow-lined 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 ±4.2 vs. D:28 ±6.1). However, the proportion of grandchildren with financially well-off parents was similar in both yellow-lined (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 yellow-lined areas, the RD plots reveal an abrupt change in both wealth and 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 yellow-lined areas. The unadjusted model(1a) indicates that having a grandparent who lived in a redlined area versus a yellow-lined 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 yellow-lined 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 yellow-lined region. The unadjusted model 1b reveals a higher average BMI (β= 5.16; 95%

CI_{rbc}: -2.54, 9.81) for redlined grandchildren versus yellow-lined 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, reveals a substantial effect size that is much greater than the observed effect size of other models (β= 7.46 95% CI_{rbc}: -4.00, 16.60). 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 yellow-lined neighborhood. Race- and ethnicity-based stratified BMI analyses are available as an online supplemental data (Table S3).

Falsification & 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 Figure 4. Additional predetermined covariate assessment results using census data from 1950 and 1960 can be found in the supplementary material (Appendix A). (1950 – Table S4, 1960 – Table S5).

Density Test

Figure 5 displays histograms of the density of the score for wealth and BMI samples, with the number of observations greater on the treatment (redlined side) than the comparison (yellow-lined 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 (189). 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 (158,210).

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 10m (Table 6). The point estimates are in the same direction and continue to be statistically significant. Moving further out to 50m for the donut-hole approach led to fewer observations for both wealth and BMI analyses.

2.4 Discussion

In this study, we explored the factors contributing to racial disparities in generational wealth and BMI outcomes in the U.S. 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 yellow-lined. 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 yellow-lined 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 (211–213). 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 (82). While our analysis included individuals of all races whose grandparents had lived in redlined versus yellow-lined 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 yellow-lined grandchildren demonstrates that redlining likely contributes to the racialized wealth gap (148,152). 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 yellow-lined 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 U.S. and the manifestation of the racialized intergenerational wealth gap, making this a significant contribution to the literature.

Killewald et al. (2017) highlights 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 (82). 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 (214), 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 (82,215). 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 (137). 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 yellow-lined areas. Additionally, studies have shown a positive relationship between housing segregation, socioeconomic status (SES), and BMI, particularly among Black women (54,136,216,217). These findings were consistent with the results of our primary and racially stratified models. 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 U.S., 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) (9). 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 limitations (182). 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 (182). 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 that we had a limited sample for our racially stratified analyses and 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, in terms of sample size the density of our participants in data were sparse on the yellow-lined side.

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 (218,219). 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 (220,221). 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 (221).

Moreover, structural racism and disparities in access to healthcare and resources, may exacerbate the impact of obesity-related conditions on minority populations (222). 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 (223). 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 (224).

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 (33,34,225,226). 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 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.

Table 2.1. Characteristics of Grandparents and Grandchildren

Grandparents (Head of Households)			
	HOLC Grade C (yellow-lined)	**HOLC Grade D (redlined)**	**Total**
n (%)	n=40	n=81	N=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			
	HOLC Grade C (yellow-lined)	**HOLC Grade D (redlined)**	**Total**
n (%)	n=52	n=185	N=237
Age mean(sd)	30 (± 5.1)	28 (± 5.4)	29 (± 5.3)
Gender			
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 Poor Head			
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			
Owns (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 (± 4.2)	28 (± 6.1)	28 (± 5.8)
Family Income mean(sd)	71000 (± 63000)	48000 (± 39000)	53000 (± 47000)
Wealth Equity mean(sd)	82000 (± 190000)	53000 (± 230000)	59000 (± 220000)

Note: First-generation labor income is expressed in 1968 dollars; Third-generation family income and wealth equity are expressed in 2019 constant dollars; Education is highest level attained.

Table 2.2. Continuity-based RD Analysis: Effect of Redlining on Grandchildren's Average Household Wealth Using Covariate-Adjusted Local Polynomial Regression

			Robust Inference					
N=173	RD Estimator ($)	MSE-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	CER-Optimal Bandwidth [meters]	N*l*	N*r*
Outcome: Wealth (mean)								
Model 1a	-96104	828.83	[-243710, -23539]	56167	0.017**	685.95	19	90
Model 2a	-105199	807.05	[-285228, -3432]	71888	0.045**	667.93	19	86
Model 3a	-95124	984.23	[-260168, -23875]	60280	0.018**	814.56	22	94
Model 4a	-35419	399.49	[-37423, -7615]	7604	0.003***	331.79	7	65

Note: 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*l* - *(left)*, N*r* - *(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 2.3. Continuity-based RD Analysis: Effect of Redlining on the Mean BMI in Third-Generation Adults Using Covariate-Adjusted Local Polynomial Regression

			Robust Inference					
N=210	RD Estimator (kg/m2)	MSE-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	CER-Optimal Bandwidth [meters]	N*l*	N*r*
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

Note: 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; N*l* - *(left)*, N*r* - *(right)* indicate the effective number of observations within the MSE-bandwidth used for estimation.
*p < 0.10, **p < 0.05, ***p < 0.01

Table 2.4. Continuity-based RD Analysis: Predetermined Census 1940 Covariates Using Local Polynomial Regression

			Robust Inference				
Covariates	Coefficient	CER-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	N*l*	N*r*
Total Occupied Dwelling Units/m2	0.00	589.729	[0.00, 0.003]	0.00	0.439	57	88
Total Population/m2	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

Note: 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 2.5. Continuity-based Approach Density Test

Number of available observations = 657

n (left)	**n (right)**	**statistic**	**p-value**
173	484	-1.0723	0.2836

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

Table 2.6. Continuity-based Analysis: Primary Outcomes Applying the Donut Hole Approach

			Robust Inference					
Donut-Hole Radius	RD Estimator	MSE-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	CER-Optimal Bandwidth [meters]	N*l*	Nr
Outcome: Average Household Wealth ($)								
10	-157882	593.148	[-345971, -10895]	85480	0.037**	488.513	17	66
50	-328490	497.881	[-814696, 171742]	251647	0.201	732.984	7	42
Outcome: Mean BMI								
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

Note: 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*l* - *(left)*, N*r* - *(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

Figure 2.1. Hypothetical PSID Grandparent Census Block within HOLC Grade C and D Areas, Showing Calculated Distance from Polygon to Adjacent Red-Yellow Border

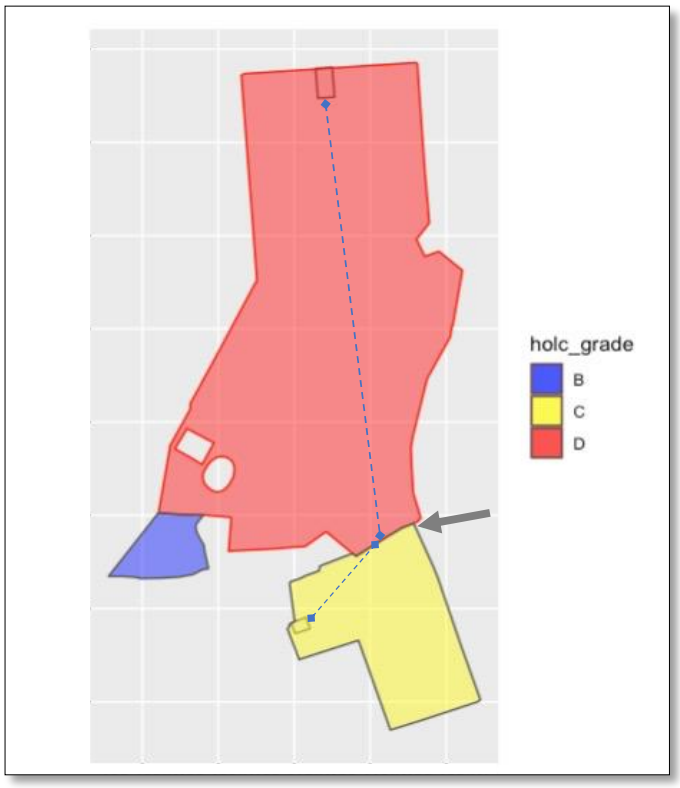
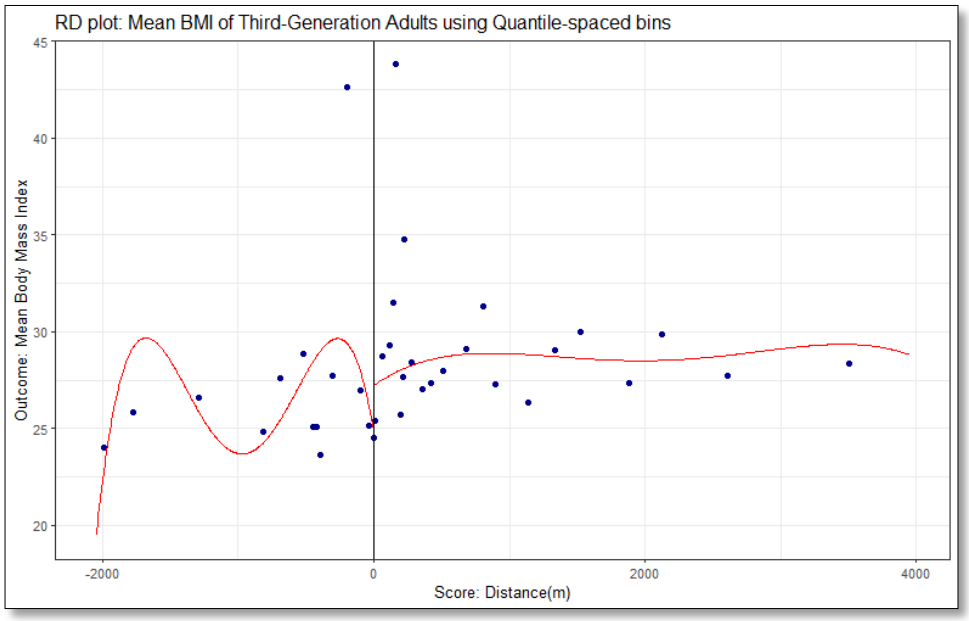


Figure 2.2. Regression Discontinuity Plot: Average Wealth of Grandchildren Households Using Quantile-Spaced Bins

Note: Regression Discontinuity (RD) plot using quantile-spaced bins and triangular kernels

Figure 2.3. Regression Discontinuity plot: Mean Body Mass Index of Grandchildren Adults Using Quantile-Spaced Bins



Note: Regression Discontinuity (RD) plot using quantile-spaced bins and triangular kernels

Figure 2.4. Regression Discontinuity Plot: 1940 Predetermined Covariates Using Quantile-Spaced Bins

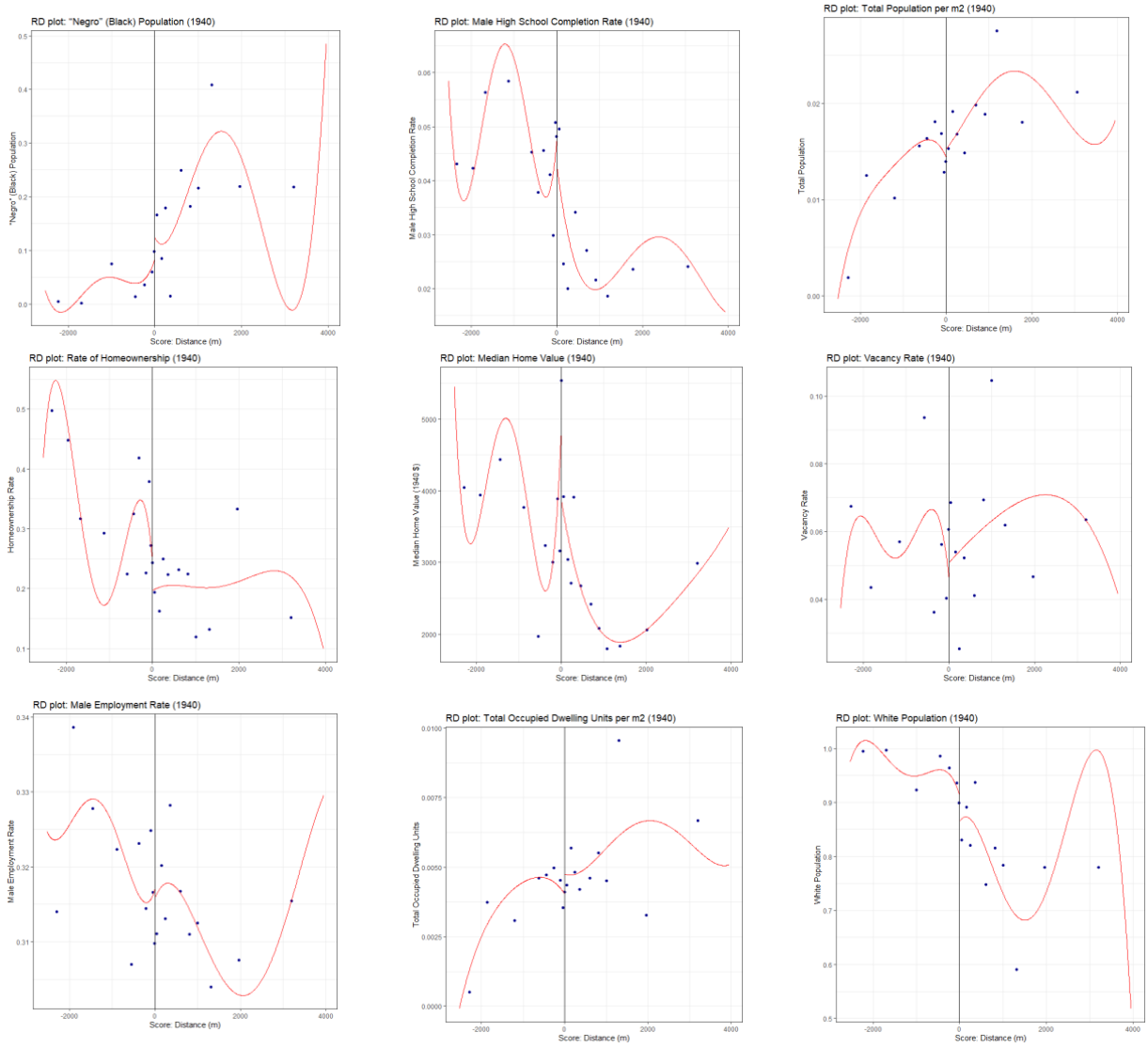
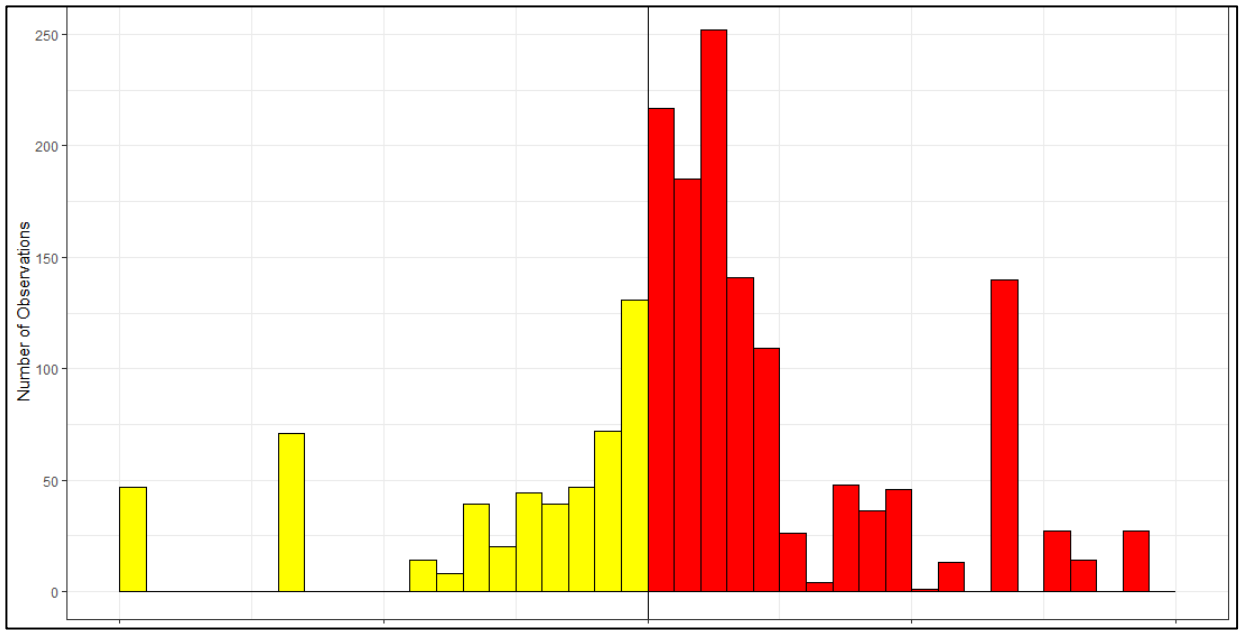


Figure 2.5. Density Plots: Sample Size of Grandchildren for Average Household Wealth and Mean Body Mass Index



2.5 Appendix A. Supplementary Materials

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I. Results: *Race and Ethnicity – Stratified Models*

Race-based stratification for non-Hispanic Black grandchildren with grandparents living in redlined areas compared to those in yellow-lined areas revealed a similar pattern as our primary analysis, lower wealth, and higher BMI, although the sample size was smaller, and the differences were not statistically significant. For non-Hispanic White grandchildren, we observed lower average household wealth and lower BMI outcomes for those with a grandparent in a redlined region. The results are presented in detail below:

Average Household Wealth

Our secondary analyses involved race- and ethnicity-stratified models, which were implemented using methods similar to our primary analysis. The results of our POC models examining the association between grandparent HOLC designated grade location and generational wealth accumulation are presented in Table A2.1. Model 1a, unadjusted, (β= -

$10,814; 95% CI_{rbc}: -$53,195, $48,820) and Model 2a, adjusted for age and year, (β= -$18,552; 95% CI_{rbc}: -$56,060, $44,925) demonstrated a lower household wealth accumulation for POC grandchildren whose grandparent lived in a redlined area when compared to those with a grandparent in a yellow-lined area, although not statistically significant. In the fully adjusted model, POC grandchildren whose grandparents lived on the redlined side of the boundary had a $20,525 (95% CI_{rbc}: -$56,460, $44,210) lower average household wealth accumulation than POC grandchildren whose grandparents lived on the yellow-lined side of the boundary, although this association was not statistically significant. The confidence intervals across all POC models were wide and none of the models were statistically significant.

The unadjusted association for White non-Hispanics indicates that grandchildren with a grandparent who lived in a redlined area have lower wealth compared to those with a grandparent in a yellow-lined area (β= -$8,386; 95% CI_{rbc}: -$160,334.65, $140,615.41). Similarly, the remaining models show that White descendants have lower generational wealth accumulation associated with grandparents residing in redlined areas than those with a grandparent in yellow-lined areas (Table A2.2). Models 2a and 3a reveal a significant discontinuity at the threshold, indicating lower wealth accumulation for White grandchildren with a grandparent living in redlined areas compared to those with a grandparent connected to yellow-lined areas, at -$149,279 (95%$CI_{rbc}$: -$332,074.99, -$65523.06) and -$141,464 (95% CI_{rbc}: -$315,613.88, -$50,386.20), respectively.

Highest Body Mass Index

Table A2.3 presents the results of our secondary analysis regarding body mass index (BMI). In our unadjusted PoC models, we observed a lower mean BMI at -0.35 (kg/m^2) (95% CI_{rbc}: -17.70, 18.34). Our PoC adjusted models indicate that grandchildren who have a

grandparent residing in redlined areas have a higher mean BMI range than those in yellow-lined areas, with an effect size of 1.21 to 2.37 (kg/m^2). Although no models displayed statistical significance, this suggests a positive association between living in redlined areas and mean BMI. Conversely, the White non-Hispanic models show a negative effect size ranging from -0.88 to -3.99 (kg/m^2), and all adjusted models are statistically significant, potentially indicating that have a grandparent living in a redlined versus yellow-lined area is associated with a lower BMI.

II. Background on Falsification and Validation Test

A few assumptions must be met or nearly met for a geographical regression discontinuity (GRD) design to yield plausible causal estimates. First, differential response bias must be ruled out (190,227). This means that the response cannot be influenced by the treatment or a specific covariate. To test this assumption, we examined our data for any systematic responses along the treatment assignments (227). Second, exchangeability is assumed in the RD design. This assumption holds when there is a similarity in characteristics and no sorting around threshold (228). Variability in the distribution of covariates for individuals by treatment assignment can be removed by analyzing only the individuals closest to the threshold in the RD analysis. The third assumption is the continuity of conditional regression functions. Meaning that treatment cannot be contaminated by other interventions at the cutoff point in a way that triggers or affects treatment assignment (194,204). Therefore, potential outcomes must remain continuous at our threshold.

In general, it is difficult to fully satisfy the assumptions of GRD using data alone. However, historical evidence, such as the work of Hillier (2005), provides insights into the creation and design of the Home Owners' Loan Corporation (HOLC) security maps and their lack of adherence to administrative boundaries, such as census tracts (146). Furthermore,

historical accounts suggest that these maps were selectively shared among agencies responsible for loan allocation, further diminishing the likelihood that families would have knowledge about the maps and manipulated their home location to influence their treatment assignment (147).

Additionally, the maps have undergone several revisions, indicating that identifying precise neighborhood regions for security maps was challenging (146). While two studies have rigorously examined the continuity assumption by identifying discontinuities related to neighborhood and resident characteristics for red-yellow borders (148,229), there is still a need for further assessment. Therefore, we created regional comparison maps for the purpose of falsification and validation tests. While the literature suggests that HOLC security maps meet most of the assumptions for a GRD design (148,152,229), our analysis seeks to provide further insights into the validity of these assumptions.

We conducted falsification tests to assess the plausibility of our assumptions and validity of our GRD design methods. These tests include balance tests of predetermined covariates, density tests, and bandwidth sensitivity approaches. We evaluated the distribution of predetermined grandparent neighborhood characteristics for the treatment and control groups to determine whether they were similar before treatment assignment (190,230). We rejected the null hypothesis if the mean distribution of the observed covariates was not the same, indicating that our predetermined covariates were not comparable before treatment assignment (189).

To assess this assumption, we conducted predetermined covariate balance tests on 1940 census neighborhood characteristics (Table 4 in the main paper) because the HOLC maps were initially implemented in the late 1930s and the 1940s. We also carried out this test through 1950 and 1960, and the results are shown below.

A density test aims to determine whether a score is continuous at the cut-off point (231). Using a density test, one would anticipate observing some type of clustering if individuals could manipulate their treatment assignment. The McCrary density test examines whether there is a jump in the threshold mark that might indicate some type of sorting occurring at or around the threshold (204,205,232). The results from our density test can be found in the main paper (Table 5 and Figure 5). A final validation test was used to assess our bandwidth selection by conducting multiple sensitivity tests with bandwidth windows of various sizes to assess the effects of our outcome (190). Applying spatial techniques to a regression discontinuity design provides an opportunity to operationalize structural racism using HOLC discriminatory security maps. This approach is not without its limitations, and we carefully considered our assumptions, estimation procedures, and falsification tests to determine the credibility and interpretation of our findings.

III. Supplemental Tables and Figures

Supplemental - Table A2.1. Continuity-based Regression Discontinuity Analysis: Effect of Redlining on Average Wealth of Third-Generation Households Adjusted for Family Size Using Covariate-Adjusted Local Polynomial Regression

			Robust Inference					
N=173	RD Estimator ($)	MSE-Optimal Bandwidth [meters]	95% CIrbc	SE	p-value	CER-Optimal Bandwidth [meters]	Nl	Nr
Outcome: Wealth (mean) adjusted for family size								
Model 1a	-48901	928.26	[-121826, -4826]	29847	0.034**	768.24	22	89
Model 2a	-44298	922.39	[-114190, -4117]	28080	0.035**	763.39	22	89
Model 3a	-41824	1026.73	[-108972, -3531]	26899	0.037**	849.73	22	95
Model 4a	-10264	487.72	[-3945129, -3149511]	202968	<0.001***	539.34	12	73

Note: 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; Nl - (left), 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

Supplemental - Table A2.2. Continuity-based Regression Discontinuity Analysis: Effect of Redlining on Grandchildren's Average Household Wealth Using Covariate-Adjusted Local Polynomial Regression

Race-Stratified Models			Robust Inference					
N=173	RD Estimator ($)	MSE-Optimal Bandwidth [meters]	95% CIrbc	SE	p-value	CER-Optimal Bandwidth [meters]	Nl	Nr
Outcome: Wealth (mean)								
Persons of Color								
Model 1a	-10814	584.20	[-53195, 48820]	26205	0.933	487.05	13	87
Model 2a	-18552	582.48	[-56060, 44925]	25762	0.829	485.61	13	87
Model 3a	-20525	588.66	[-56460, 44210]	25681	0.811	490.77	13	87
Non-Hispanic White								
Model 1a	-8386	712.70	[-160335, 140615]	76774	0.898	620.44	15	17
Model 2a	-149279	709.62	[-332075, -65523]	67999	0.003***	617.76	15	17
Model 3a	-141464	736.09	[-315614, -50386]	67661	0.007***	640.80	15	17

Note: 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; Nl - (left), Nr - (right) indicate the effective number of observations within the MSE-bandwidth used for estimation; Wealth is rounded to the nearest whole dollar and expressed in 2019 dollars. *p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table A2.3. Continuity-based Regression Discontinuity Analysis: Effect of Redlining on the Mean BMI in Third-Generation Adults Using Covariate-Adjusted Local Polynomial Regression

Race-Stratified Models			Robust Inference					
N=210	RD Estimator (kg/m2)	MSE-Optimal Bandwidth [meters]	95% CIrbc	SE	p-value	CER-Optimal Bandwidth [meters]	Nl	Nr
Outcome: Mean Body Mass Index								
Persons of Color								
Model 1b	-0.35	615.40	[-17.70, 18.34]	9.20	0.972	513.06	10	88
Model 2b	1.21	596.07	[-16.71, 20.40]	9.47	0.845	474.43	6	83
Model 3b	2.37	567.66	[-15.51, 20.26]	9.49	0.75	473.26	6	83
Non-Hispanic White								
Model 1b	-0.88	774.66	[-2.55, 0.87]	0.87	0.337	678.90	13	14
Model 2b	-3.21	783.26	[-5.91, -0.46]	1.39	0.022**	686.43	13	14
Model 3b	-3.99	746.91	[-6.48, -1.47]	1.28	0.002***	654.58	13	14

Note: 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; Nl - (left), Nr - (right) indicate the effective number of observations within the MSE-bandwidth used for estimation. *p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table A2.4. Continuity-based RD Analysis: Predetermined Census 1950 Covariates Using Local Polynomial Regression

			Robust Inference				
Covariates	Coefficient	CER-Optimal Bandwidth [meters]	95% CIrbc	SE	p	Nl	Nr
Total Occupied Dwelling Units/m2	0.00	572.697	[0.00, 0.004]	0.00	0.257	57	83
Total Population/m2	0.003	585.037	[-0.004, 0.011]	0.00	0.319	57	85
White Population	-0.057	476.372	[-0.204, 0.116]	0.08	0.59	54	77
"Negro" Population	0.06	473.239	[-0.12, 0.205]	0.08	0.58	53	77
Homeownership Rate	-0.01	401.794	[-0.13, 0.143]	0.07	0.94	46	73
Median Home Value	-2706.65	833.966	[-5731, -211]	1408.14	0.035**	67	107
Male Employment Rate	-0.02	523.756	[-0.04, 0]	0.01	0.048**	56	81
Male High School Completion Rate	-0.02	452.796	[-0.05, 0.014]	0.02	0.291	53	76
Vacancy Rate	0.00	633.668	[-0.01, 0.014]	0.01	0.4	59	87

Note: 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

Supplemental - Table A2.5. Continuity-based RD Analysis: Predetermined Census 1960 Covariates Using Local Polynomial Regression

			Robust Inference				
Covariates	Coefficient	CER-Optimal Bandwidth [meters]	95% CIrbc	SE	p	Nl	Nr
Total Occupied Dwelling Units/m2	0	604.663	[-0.001, 0.004]	0.001	0.373	60	96
Total Population/m2	0.003	577.162	[-0.003, 0.009]	0.003	0.365	60	95
White Population	-0.076	426.355	[-0.264, 0.116]	0.097	0.447	50	83
"Negro" Population	-690.22	389.793	[-2583.12, 1030.216]	921.79	0.4	46	81
Homeownership Rate	0.15	470.267	[-0.449, 0.686]	0.29	0.682	56	86
Home Value $35k +	-0.9	593.693	[-17.44, 14.01]	8.021	0.831	60	96
Male Employment Rate	0.01	535.49	[-0.01, 0.03]	0.011	0.419	59	91
Male High School Completion Rate	-208.24	385.941	[-436.35, -12.14]	108.22	0.038**	45	78
Vacancy Rate	0	408.429	[-0.01, 0.02]	0.007	0.542	49	83

Note: Discrete analysis using cluster standard errors; All models use first-degree polynomial. *p < 0.10, **p < 0.05, ***p < 0.01

CHAPTER 3. Examining the Generational Impact of Redlining on Employment Quality: Findings from the Panel Study for Income Dynamics

3.1 Introduction

Employment quality is now recognized as an essential factor in achieving a secure, healthy, and fulfilling life (18,86). Typically, high-quality employment embodies the Standard Employment Relationship (SER), which is characterized by consistent, full-time employment with wages and benefits along with control over time and labor processes (85,86). Research has shown that women and people of color have historically been excluded from SER-type jobs due to systemic discrimination (87–89). Additionally, structural racism limits opportunities for SER-related jobs and has a significant impact on communities of color (233,234).

Structural racism is a distal sociopolitical force which contributes to the unequal distribution of personal resources or "opportunities" to obtain standard employment relationship (93). This unequal access to resources results in an inequitable labor market, which continues the cycling of lower quality employment for marginalized groups. Structural racism operates through several mechanisms to exacerbate inequitable employment outcomes for disenfranchised populations, including by hindering access to socioeconomic advantages within neighborhoods and restricting social networks and avenues for intergenerational upward mobility (235–237). The generational outcomes of systemic discrimination, espoused by historical institutional racism in housing and employment quality, remain unexamined. This study aims to explore the link between employment quality and redlining, a form of institutional racism that was the basis of racially discriminatory federal policies in housing, over multiple generations.

Employment as a determinant of health

Employment quality has a significant impact on population health and equity. Employment conditions, such as health insurance, time off, job security, and social status, can affect an individual's ability to maintain good health throughout their lifetime (18,86,95,238). In addition, the relational and contractual aspects between the employer and employee, including employment stability, material rewards, work-time arrangements, collective organization, and employer-employee power relations, make up the multidimensional construct of employment quality (97,239). Collectively, these aspects of employment determine the specific pathways through which employment quality contributes to disparate health outcomes (98,102), among disenfranchised and privileged populations.

There is strong motivation to demonstrate the relationship between structural racism and employment quality, as work serves as a domain for which structural racism manifests to produce health inequities (18,126,240). Systemic inequalities based on race may intertwine with the instability and economic vulnerability inherent in precarious work arrangements, perpetuating a cycle of disadvantages and limited opportunities for marginalized communities. Precarious employment is often indicative of subpar (or low) employment quality, and is typically characterized by job insecurity, irregular work schedules, limited opportunities for advancement such as through training or promotion, low wages, and a lack of protection benefits, and power (22,86,99). Prolonged exposure to precarious employment has been linked to negative effects on both physical and mental health (22,86,97,99,102).

Cumulative Inequality theory

Utilizing the concept of the cumulative inequality theory, we argue that social systems, not just individual choices, create and worsen inequalities over time. This "cumulative inequality" begins at birth, with childhood experiences and grandparents' socioeconomic

position heavily shaping one's future outcomes (83,215,241). Therefore, intergenerational processes and structural forces play crucial roles in understanding the disadvantages and accumulation of inequality. Disadvantages, caused by structural determinants such as institutional racism, may increase the likelihood of exposure to risk such as lower employment quality. Additionally, early exposure to disadvantages such as institutional racism places individuals on a more precarious trajectory, restricting their access to good jobs and health-protective resources (242). These disadvantages are not simply cancelled out by advantages. Instead, they accumulate like weights, pushing individuals further down the socioeconomic hierarchy and making it harder for subsequent generations to climb up (241). A high accumulation of risk over generations, such as through structural racism in federal policies like redlining, can lead to stark health disparities between groups, as those with more disadvantages struggle to accumulate health-promoting resources (118,241).

Redlining

In the late 1930s, the U.S. federal government sponsored the Home Owners' Loan Corporation (HOLC) to assess neighborhoods and determined their eligibility for federally backed mortgages (23,145). The HOLC created discriminatory color-coded maps that outlined the grade given to each neighborhood based on its characteristics, including racial demographics. The grading scheme was as follows: A) the highest grade, colored green, and identified as a minimal risk for banks when determining which neighborhoods within a city were 'safe investments'; B) the second highest, colored blue, and identified as 'low risk' for home loans; C) the second lowest, labeled as 'definitely declining' and colored yellow; and D) the lowest grade, assigned the colored red – hence the term redlining. It is noteworthy that most areas with predominantly "Negro" (i.e., Black) populations were deemed to have the lowest grade, D

(145,147,149,243). HOLC grades served as a catalyst for codifying residential segregation on the basis of race and social class; for instance, raters classified several areas based on the "infiltration" of Jews, Italians, and Negros (among other immigrants) (145). An example of statements used to characterize or describe redlined areas:

> "This large rambling area is occupied almost entirely by negroes, there being a smattering of whites between 5th and 7th Streets… It is spotty, there being between 5th and 7th Streets, from State north to Freeman, some good houses occupied largely by white people, with values depressed and sales very poor due to the negro influence and continued infiltration…" (150)

Although redlining was legally prohibited after the 1970s, there is evidence of the continuation of this discriminatory practice (144,151,244).

This study aimed to investigate the impact of "redlining", a manifestation of structural racism at the neighborhood level, across multiple generations. Specifically, we seek to understand its role in shaping intergenerational disparities in a crucial determinant of health – the quality of employment.

3.2 Method

This study employs generational and life-course perspectives to describe the interaction between discriminatory federal policies and intergenerational mobility, in terms of employment quality outcomes. Specifically, our objective was to estimate the potential causal impact of structural racism, as measured by the HOLC maps, on the employment quality outcomes of grandchildren of people who lived in the 1960s in redlined compared with yellow-lined areas. To achieve this, we used a geographical regression discontinuity (GRD) design and longitudinal

data to assess its effects. In the following section, we outline the data and the methods employed in the analysis.

Data Sources

We utilize the Panel Study for Income Dynamics (PSID) and the University of Richmond's Mapping Inequality project digitized HOLC maps to incorporate both geographical and genealogical components to employ our GRD design (150). The PSID is a nationally representative longitudinal study that has collected economic, social, and health and well-being data on families across multiple generations since 1968 (158). To obtain each PSID first-generation (henceforth called grandparent) HOLC location, we merged geocoded HOLC maps with the geocoded census block-level data of the PSID grandparents. We further merged these data with the IPUMS Decennial Census Data to identify neighborhood summary statistics from 1940 and conducted falsification and validation tests, as described in more detail in Appendix B, Supplementary Materials.

Study population

Grandchildren (i.e., third-generation PSID respondents) were eligible for our study if their grandparents resided in a yellow-lined area (HOLC grade C) touching a redlined area (HOLC grade D) or vice versa in the 1960s, with those whose grandparents resided in a touching yellow-lined area classified as ‘controls’ and touching redlined area as ‘treated’. We restricted our sample of grandchildren to those interviewed between 1999-2019 when all items contributing to our EQ outcome variable were measured. Additionally, we limited our dataset to grandchildren aged 25-64 years old who had available data on their demographics, income, educational attainment, and employment status during the study’s outcome period. For two

participants with missing demographic data, we applied carry forward/backward imputation methods to impute missing data.

Study design

Geographical Regression Discontinuity

Geographical regression discontinuity (GRD) design is a specialized form of regression discontinuity design in which a geographical boundary acts as a cutoff or treatment assignment mechanism to differentiate between the treatment and control groups (181,182,193). We used the spatial distance/separation between a grandparent's census block location and the HOLC grade boundary line as our exogenous exposure variable, allowing our results to be interpreted causally within the regression discontinuity framework (181,182). This is justified in the historical context of redlining maps, where it is unlikely the distance between a grandparent's home and the HOLC grade boundary line was influenced by the placement of the HOLC grade boundary line (i.e., treatment) itself, as evidenced in previous studies (148,152,229).

Moreover, we delineated the distance variable by categorizing the grandparent's residence (using census block rather than exact address) based on their distance: with those residing on the "yellow-lined" side of the boundary as families below the threshold or cutoff, and those residing on the "redlined" side as those above the cutoff. This delineation allows us to establish distinct control and treatment groups respectively (181,182). A visual example of our home-to-boundary distance measure can be found in the Supplementary Materials. Importantly, it is highly improbable that the PSID grandparents were aware of these maps, given the 'behind-the-scenes' application of HOLC grading and historical evidence that only those in the housing

industry were aware of HOLC maps and hence their borders (154). For this reason, we assumed full compliance and employed a sharp RD design with binary treatment assignment (187,189).

Exposure

As previously stated, we applied the GRD approach to define treatment and control groups. We used a running variable in our analysis to implement our exposure. The running variable, or score, in a GRD is a continuous measure that determines treatment assignment based upon a predetermined threshold or cutoff point along a geographic boundary. Specifically, we assigned grandchildren whose grandparents resided in redlined areas to the treatment group, and those whose grandparents lived in yellow-lined areas to the control group.

Outcome

The primary outcome of our study was a latent multidimensional concept of employment quality (EQ). As previously mentioned, SER is used as a benchmark for assessing the characteristics of EQ, and it entails stable and permanent full-time work that is regularly scheduled with benefits (85,245–247). EQ is considered multidimensional, as it embodies several different aspects of employment conditions and relations (245). Using a multidimensional EQ construct better captures the aspects of employment contracts and employee-employer relations. In our study, these dimensions are operationalized through a set of indicators based on job characteristics that serve as a proxy for measuring each dimension (92,248).

We draw from previous literature to consider seven dimensions that describe the employment arrangement and employee-employer relationship: (1) employment stability, (2) material rewards, (3) workers' rights and social protections, (4) standardized working time arrangements, (5) collective organization (e.g., unions), (6) employability opportunities (or professional development), and (7) interpersonal power relations (248,249). We used data from

the PSID to identify proxy indicators of EQ dimensions, encompassing precarious employment (or low-quality jobs) and high-quality jobs.

Following the existing literature (250), Table 1 outlines the dimensions, indicators, and specific constructs that inform the development of our employment quality score. Building on earlier research (251,252), we employed a principal component analysis (PCA) procedure to construct this score. PCA is a data-driven statistical technique aimed at parsimoniously summarizing a set of correlated variables into a smaller, more manageable set of summary indices or components, called principal components, while preserving the essential information contained within the original set of data (253).

In this study, we utilized five EQ dimensions available for analysis in the PSID: (1) employment stability, (2) material rewards, (3) workers' rights and social protection, (4) standardized working time arrangements, and (5) collective organization (e.g., unions) (252). However, we did not aim to reduce the data dimensionality through variable elimination. Instead, we leveraged PCA to optimally weigh individual indicators based on the proportion of shared variance they explain, reflecting their contribution to the overall construct of latent employment quality. Principal components with eigenvalues exceeding or equal to one were summed, encompassing all indicators and domains present in our data. By identifying the key latent components behind the five established dimensions, this approach helped us distill the multifaceted concept of EQ, as reflected in our dataset. For the purposes of our analysis, we calculated the average EQ score across numerous observations for each individual to obtain their mean EQ. We additionally standardized the length of employment and labor income by age using z-scores.

Covariates

To enhance the precision of our estimates, we integrated variables such as age, sex, and race/ethnicity of grandchildren into our models as covariates, as these variables are hypothesized to affect only the EQ outcome variable (177,189). Sex was operationalized as a dummy variable, with females assigned a value of 1, while race and ethnicity were also represented as dummy variables, with Non-Hispanic Black designated as 1 and Non-Hispanic White designated as 0. The age variable was considered a continuous variable and centered around the mean. Additionally, calendar year in which the data were collected was treated as an ordered categorical variable and incorporated into the models.

Analytical Method

Using a continuity-based approach in our GRD analysis, we treated home-to-boundary distance, measured in meters as a discrete score. To estimate the local average treatment (LATE) effect, we used polynomial regression with triangular kernels (189,192). Employing triangular kernels enabled us to apply higher weights to observations that lie closest to HOLC C and D touching borders. To compute our effect estimate for each model, we subtracted the regression results for the control (yellow-lined) and treated (redlined) areas on either side of the boundary.

A critical aspect of approximating the effect estimate is identifying the bandwidth required to estimate the regressions. Employing a mean squared error (MSE)-optimal bandwidth approach aims to balance bias and variance by obtaining estimates with minimal bias (189). We applied MSE-optimal bandwidth methods to compute our estimates, and reported the effective observations used in the calculation. Alternatively, a coverage error rate (CER)-optimal bandwidth approach aims to minimize the coverage error rate, and we employed this approach for inference to construct robust, bias-corrected confidence intervals (189,202).

Sensitivity Analysis

For a sensitivity analysis, we applied a linear approach to develop an EQ outcome variable, where each indicator was scored as 0 or 1 and then summed to provide an unweighted summative score (Table 1) (252). We employed this linear approach to examine whether the overall outcome of our analysis changed when each EQ subscale or indicator was allowed to contribute equally to the total EQ score regardless of the number of items included within a given indicator. We report this analysis in the results section. Additionally, we implemented multiple falsification and validation test, including an evaluation of the distribution of predetermined covariates, a density test of the score, and a bandwidth sensitivity test. These tests are further described under Appendix B, Supplementary Materials.

Regression Discontinuity plots

A fundamental element of any regression discontinuity design is the graphical depiction of the predicted outcome values along the running variable, or score, for the control and treatment groups (189,206). We visually explored the relationship between the two groups by examining fitted polynomial regression lines. A significant effect should manifest as a noticeable change or discontinuity in the plot at the threshold at which treatment assignment shifts. In our study, we generated our regression discontinuity (RD) plots by mimicking the variability in our data and utilizing quantile-space bins (189). We created RD plots for both the PCA and Linear EQ outcome variables.

Creation of the EQ PCA was performed in Stata version 17, and all other analyses were performed with R Statistical Software version using RStudio IDE (208,254).

3.3 Results

Table 2 displays the sample characteristics of both the grandparents and grandchildren. The average age of grandparents in the redlined areas was seven years younger than those

individuals in yellowed-lined areas. Additionally, in areas that were subject to redlining, there were more likely to be residents of Black (HOLC D: 78% vs. HOLC C: 69%) and Puerto Rican/Mexican descent (HOLC D: 3% vs. HOLC C: 0%), with a lower proportion of White residents (HOLC D: 19% vs. HOLC C: 31%), compared to areas that were subject to yellow-lining. The distributions of education and sex were similar between the two groups. The labor income of grandparents in redlined areas was not substantially higher at $5100 (SD: ± 3300) compared to those in yellow-lined areas at $5000 (SD: ± 3000). Moreover, a substantial proportion of grandparents in both the redlined and yellow-lined areas reported that their parents were poor. Regarding employment status, a larger proportion of grandparents in the redlined areas were housewives than in the yellow-lined areas.

Table 2 presents detailed demographic data for the 113 grandchildren, with 29 participants in the yellow-lined control group and 84 in the redlined treatment group. Generally, the ages of grandchildren were comparable (Table 2). Similar to their grandparents, a larger percentage of grandchildren from Black and other racially diverse groups resided in redlined areas, while yellow-lined regions had a more balanced racial distribution with 52% White and 48% Black. Furthermore, grandchildren in yellow-lined areas were more likely to be married than those in redlined areas. In terms of education and employment, a large proportion of grandchildren in the yellow-lined regions were college-educated and held salaried positions.

Conversely, redlined grandchildren had a larger proportion of individuals with less than a high school education and hourly jobs. Moreover, a larger proportion of individuals in redlined areas reported that their parents were poor and that they rented their homes. Lastly, the average labor income for grandchildren in yellow-lined areas (70000; SD: ± 47000) was substantially higher than that of those in redlined areas (60000; SD: ± 67000).

Employment Quality Outcome

Primary Analysis

Table 3 presents the results of the primary analysis. In our unadjusted analysis (Model 1a), we observed a significant relationship between redlining and lower employment quality among the grandchildren. Specifically, we found that grandchildren with a grandparent in a redlined area had a lower average employment quality by 0.21 (95% CI_{rbc}: -0.62, -0.07), a large difference in magnitude compared with yellow-lined grandchildren. In Model 2a, which adjusts for covariates age and year, we continued to observe the same pattern of lower employment quality for grandchildren with redlined grandparents compared to those with yellow-lined grandparents, with a slightly wider MSE-optimal bandwidth and a confidence interval excluding zero (β: -0.27; 95%CI_{rbc}: -0.69, -0.01).

In our final two adjusted models, we observed a slightly larger magnitude point-estimated effect. In Model 3a, which adjusts for age, year, and gender, we also found a substantial difference in employment quality with a score of -0.32 (95%CI_{rbc}: -0.766, -0.057). In our fully adjusted Model 4a which includes covariates age, year, sex, and race/ethnicity, we observed a consistent association between redlining and grandchildren's employment quality outcomes. Specifically, we found that grandchildren with a grandparent who lived on the redlined side had a 0.30 lower employment quality score compared to grandchildren with a grandparent on the yellow-lined side (95%CI_{rbc}: -0.91, 0.01). These findings suggest that the effects of redlining policies may persist and influence employment quality outcomes of future generations.

Figure 1 presents the RD plot for our primary analysis using a PCA EQ variable. A clear discontinuity can be observed in the plot at the threshold point delineating control and treatment groups. This discontinuity in the RD plot demonstrates the key finding of our primary analysis:

that is, there is a discernible effect on the EQ outcomes of grandchildren with a grandparent who resided in a redlined neighborhood compared to those with a grandparent in a yellow-lined neighborhood, which becomes evident at the boundary line of the respective neighborhoods.

Sensitivity Analysis

Our sensitivity analysis using an unweighted summative EQ outcome variable provided similar results to our primary analysis, with findings suggesting that having a grandparent who resided in a redlined area, in contrast to a yellow-lined area, is linked to lower employment quality scores for grandchildren (Table 4). Our unadjusted Model 1b indicates a difference of 0.46-point lower employment quality (95%CI_{rbc}: -1.24, 0.24). Models 2b and 3b exhibit similar patterns, demonstrating a negative association between grandparents' exposure to redlining and grandchildren's employment outcomes, -0.72 (95%CI_{rbc}: -2.22, 0.18) and -0.76 (95%CI_{rbc}: -2.282, 0.144) respectively. In our final model (Model 4b), again including age, year, sex, and race/ethnicity as covariates, we found a similar pattern where grandchildren with a grandparent who resided in a redlined area experienced a 0.74-point lower employment quality score compared to those with a grandparent living in a yellow-lined area (95%CI_{rbc}: -1.60, -0.01). While statistical significance at the 0.05 level was not observed across all tests, the persistent alignment in the direction of association and the presence of p-values <0.10 in numerous specifications contribute to our understanding of the results as consistently negative and meaningfully different.

The results of our sensitivity analysis are presented visually in an RD plot in Figure 2, which reveals the presence of a discontinuity jump at the threshold between the treatment group (grandchildren with grandparents in a redlined area) and the control group (grandchildren with grandparents in a yellow-lined area). As depicted in our RD plots, the results from our sensitivity

analyses align with the findings from our primary analyses, identifying an emerging effect at the juncture where the control transitions to treatment. Note that while visual inspection of the data reveals a sufficient number of participants right at the boundary, there is a decrease in the number of individuals in the yellow-lined area between 750 and 1400 meters. This is likely attributable to the smaller sample size in the yellow-line area.

Ultimately, the insufficient quantity of grandchildren with comprehensive employment quality data may have hindered our sample from fully satisfying the density test. This test evaluates the density of observations on both sides of threshold (189), and our sample had a higher density within the sample for those close to the threshold. Again, there were more individuals near the threshold on the yellow-lined area than those further away, and thus, those closest to the threshold were within the range of bandwidths used for the analysis. Further details on the density test including the table and figure can be found in Appendix B under Supplementary Materials.

3.4 Discussion

This study highlights the profound historical influence of redlining policies on multigenerational outcomes in terms of employment quality. The results of our study demonstrate that the employment quality of grandchildren who have a grandparent who resided in a redlined neighborhood is lower than that of grandchildren whose grandparents resided in yellow-lined neighborhoods. Our primary analysis revealed a plausible causal relationship between grandparents redlining exposure and employment outcomes for grandchildren, and a significant disparity between the two groups. We presume that the observed contrast between yellow-lined and redlined grandchildren might be more pronounced by comparing the employment quality outcomes of blue- (HOLC grade B) and green-lined (HOLC grade A)

grandchildren within a causal study design, alongside those in yellow- and redlined areas. We posit that these EQ findings may contribute to the intergenerational transmission of cumulative inequality – first, the relationship between neighborhoods, social networks, and the intergenerational mobility of labor outcomes, and second, employment as a determinant of access to resources that promote and shape health and wellbeing.

Our findings shed light on the lasting effects of neighborhood-level discriminatory policies cementing the downward or stagnant trajectory of social mobility across multiple generations within the realm of EQ. Our findings of lower EQ among grandchildren whose grandparents lived in a redlined versus yellow lined neighborhood are consistent with previous work that found redlined neighborhoods are more likely to have lower access to jobs due to reduced social and economic capital within the community, which compounds the effect of lower real estate and building neighborhood economic opportunities (255). According to Elliott (1999), the socioeconomic status of a neighborhood has a significant impact on the quantity and quality of job contacts and referrals available to residents (256). Additionally, informal networks within impoverished areas tend to lead to lower-paying jobs that are less likely to have a standard employment relationship or high employment quality than contacts or networks within more affluent neighborhoods or communities (256)**.** Bayer, Ross, and Topa (2008), examined the impact of social interactions by analyzing the Census block where people who work together live (257). Their results showed that living on the same block increased the probability of working together by 33%. Moreover, there is evidence that referral networks are linked to an individual's neighborhoods and can influence labor market outcomes, including expected earnings (236,257).

Understanding the influence of intergenerational dynamics that shape EQ outcomes requires a nuanced view of intergenerational social mobility and its relationship to employment

trajectories over generations. Social connections and neighborhood resources such as transportation are an important factor in understanding EQ outcomes (237,257,258), as they can potentially facilitate acquiring higher-quality employment. In the context of intergenerational social mobility, access to certain high-quality jobs or careers often reflects persistent patterns in economic positions and occupational standing over time. The results of this study on disparate employment quality outcomes among grandchildren from redlined and yellow-lined build upon research aimed at understanding the generational consequences of a discriminatory policy on socioeconomic mobility over multiple generations. Assessing intergenerational mobility through multiple lenses, including EQ, allows for a comprehensive understanding of inequality of opportunity and the level of inflexibility or openness of social and economic class boundaries within a society (93,259). Ultimately, social networks within a neighborhood influence an individual's labor market outcomes, and our findings suggest that this occurs over multiple generations when previous generations are exposed to neighborhood-level structural racism such as redlining policies.

Intergenerational mobility and quality of employment are closely related to educational outcomes, which are in turn influenced by parental and neighborhood socioeconomic status (SES) and social connections (260,261). Previous research demonstrates that the legacy of redlining persists through generations, as grandchildren of residents in historically redlined neighborhoods exhibit lower wealth than those in yellow-lined neighborhoods (262), which may limit access to quality education and impede upward mobility into communities with greater social and economic opportunities. Individuals from high-SES backgrounds have greater access to high-mobility rate colleges (235). Many of the top educational institutions are dominated by those who can leverage the advantages of social connectedness and higher neighborhood

economic status passed down through generations (235). Numerous studies have demonstrated that individuals inhabiting socioeconomically disadvantaged neighborhoods exhibit lower levels of educational attainment (particularly in higher education), poorer health outcomes, and less success in securing stable employment relationships, ultimately resulting in precarious employment or low-quality job opportunities-(236,237,263–265). Our findings indicate that, although yellow-lined grandparents may have faced some disadvantages, they likely had slightly better neighborhood network advantages, which may have led to improved economic, educational, and employment quality outcomes across generations.

Access to employment with benefits that fall under the umbrella of employment quality is an important factor in employee well-being and economic mobility (263). Research applying a simple descriptive approach using the PSID from 1984 to 1994 to examine the relationship between job quality and economic mobility, as measured through pension plans, insurance and leave benefits, and paid vacation, found that access to sick leave was positively and significantly associated with upward mobility (266). Our study takes this one step further by capitalizing on the longitudinal and geographical nature of PSID to explore a plausible causal explanation for the relationship between discriminatory redlining policies and the multidimensional concept of employment quality.

Our findings suggest that, when examined through a collection of job characteristics, employment quality is affected by generational exposure to structural racism. The disparities in Covid-19 risk and outcomes highlight the role of employment quality and occupation type play in determining an individual's ability to access health-promoting resources and level of exposure to risk in their job (18,101,267,268). Our analysis reveals that redlining, an early example of racially discriminatory government policy that contributed to residential

segregation(145,152,255), perpetuates inequality in high-quality jobs. We posit that this may work by weakening the economic capability of neighborhoods, leading to disparities in education, wages, and employment opportunities, particularly for Black Americans who may face other forms of discrimination in the labor market.

Limitations & Strengths

This study has several limitations. First, out of the seven dimensions that define the construct of EQ, we only had adequate data available from the PSID to analyze five EQ dimensions: employment stability, material rewards, working time arrangements, collective organization, and workers' rights. The PSID lacks sufficient data to measure employment opportunities and training, and interpersonal power relations. Previous research has indicated that the two dimensions lacking PSID data might have some correlation with other dimensions (252,269). Ultimately, we used the PSID because it represents the best option for both geographical and longitudinal data, along with the most complete data, to measure employment quality in the United States.

Moreover, our analysis used aggregated geographical locations rather than precise locations (i.e., addresses) of grandparents in yellow-lined and redlined areas. While the use of Census block, the smallest spatial unit available from the PSID, rather than addresses restricts our ability to precisely capture the spatial variability of each unit, prior research has demonstrated its validity for use in assigning HOLC grades. Researchers have determined that using Census blocks, as opposed to Census tract or block-groups, for the accurate assignment of HOLC grades diminishes the misclassification of matching spatial area data to HOLC graded areas (270).

Furthermore, our findings may not be generalizable to other areas or regions outside of the yellow-lined/redlined areas, as we are limited to only those families closest to the boundary. Additionally, while we recognize that redlining policies have impacted multiple marginalized communities, including immigrants and other ethnic groups. We were unable to specifically identify these groups due to a lack of disaggregated information on individuals identified as "Other" in the racial category within the PSID. Finally, as previously mentioned in light of the smaller sample size employed in this study, in particular within the yellow-lined area, likely led to a less robust test of the sample distribution.

A key advantage or strength of our study was the potential causal interpretation of the results. We mitigated concerns regarding confounding effects arising from interval events or other confounding dynamics by focusing on a specific subset of grandparents residing near the yellow/red border in the 1960s. This proximity to the border is crucial for fulfilling the identification assumptions for causal inference. Leveraging this proximity allowed us to compare families on either side of the yellow/red border who exhibited similar characteristics, as demonstrated in Table 2. This approach enabled us to apply causal inference techniques to our analysis, ultimately strengthening the internal validity of our findings.

Conclusion

This study endeavored to explore the ramifications of inequities in opportunities (a result of historical discriminatory housing policies) on multigenerational outcomes. Our data indicate that residing in economically and socially disadvantaged neighborhoods can have detrimental, long-lasting effects that transcend generations, leading to stagnant or subpar employment prospects.

While our data reveals the generational story of harm, EQ holds the potential to be modified through the implementation of labor protection policies. By promoting stronger labor protections for workers through policies such as, minimum wage, secure scheduling, and curtailing right to work policies could help reduce the negative impacts of PE felt today.

In pursuing the goal of enhancing population health and eradicating health disparities, it is essential to identify and comprehend the causal factors that contribute to these inequities within historically marginalized communities. A more thorough understanding of these factors can inform both national and statewide interventions and policies designed to rectify past injustices, such as affirmative action. Despite the fact that the U.S. has yet to provide reparations to communities that have been deliberately harmed by federal policies, there is an immediate and ongoing need to uncover and understand the extent of the harm inflicted upon Black and brown communities in order to advance the cause of restorative justice.

Table 3.1. Conceptualization of Employment Quality Dimensions using the Panel Study of Income Dynamics

		Operationalization	
Employment Quality (EQ) Dimension	**Proxy indicators**	PCA-derived EQ score	Linear EQ Score (0 to 5 pts)
[1] Employment stability	[1] Length of employment	Continuous: Age-standardized z-score	Categorical: Employed for ≥ 12 months – No, Yes [1 pt]
[2] Material rewards	[2a] Total annual labor income	Continuous: Age-standardized z-score	Categorical: ≥ Median age-standardized z-score – No, Yes [1/3 pts]
	[2b] Employer-provided health insurance	Categorical: No, Yes	Categorical: No, Yes [1/3 pts]
	[2c] Employer-provided pension contributions	Categorical: No, Yes	Categorical: No, Yes [1/3 pts]
[3] Workers' rights & social protections	[3a] Salaried employment	Categorical: No, Yes	Categorical: No, Yes [1/2 pts]
	[3b] Employer paid extra for overtime	Categorical: No, Yes	Categorical: No, Yes [1/2 pts]
[4] Working time arrangements	[4a] Number of hours worked annually	Continuous: Linear	Categorical: ≥ 32 hrs per week on average – No, Yes [1 pt]
[5] Collective organization	[5a] Employee union membership	Categorical: No, Yes	Categorical: No, Yes [1 pt]
[6] Employability opportunities	No available data		
[7] Interpersonal power relations	No available data		

Note: Labor income is adjusted to reflect 2019-value prior to standardization (Blaikie et al., 2023)

Table 3.2. Grandparent and Grandchildren Characteristics

Grandparents (Head of Households)			
	HOLC Grade C (yellow-lined)	HOLC Grade D (redlined)	Total
n (%)	n=13 (27%)	n=36 (73%)	N=49 (100%)
Age – Mean (SD)	46 (± 12)	39 (± 11)	41 (± 11)
Gender			
Male	7 (54%)	20 (56%)	27 (55%)
Race			
"Negro" (Black)	9 (69%)	28 (78%)	37 (76%)
Other	0 (0%)	0 (0%)	0 (0%)
Puerto Rican/Mexican	0 (0%)	1 (3%)	1 (2%)
White	4 (31%)	7 (19%)	11 (22%)
Marital Status			
Divorced	0 (0%)	2 (6%)	2 (4%)
Married/Cohabit	7 (54%)	20 (56%)	27 (55%)
Separated	4 (31%)	9 (25%)	13 (27%)
Single	1 (8%)	4 (11%)	5 (10%)
Widowed	1 (8%)	1 (3%)	2 (4%)
Education			
<High School	8 (62%)	25 (69%)	33 (67%)
High School	4 (31%)	11 (31%)	15 (31%)
Some College	0 (0%)	0 (0%)	0 (0%)
College	0 (0%)	0 (0%)	0 (0%)
Missing	1 (7%)	0 (0%)	1 (2%)
Labor income – mean (SD)	5000 (± 3000)	5100 (± 3300)	5100 (± 3200)
Parents SES status			
Poor	9 (69%)	21 (58%)	30 (61%)
Pretty Well Off	0 (0%)	7 (19%)	7 (14%)
Missing	4 (31%)	8 (23%)	12 (25%)
Employment status			
Working, temp laid off	7 (54%)	22 (61%)	29 (59%)
Housewife	3 (23%)	12 (33%)	15 (31%)
Retired, disable	3 (23%)	1 (3%)	4 (8%)
Student	0 (0%)	0 (0%)	0 (0%)
Unemployed	0 (0%)	1 (3%)	1 (2%)
Grandchildren			
	HOLC Grade C (yellow-lined)	HOLC Grade D (redlined)	Total
n (%)	n=29 (26%)	n=84 (74%)	N=113 (100%)
Age – Mean (SD)	39 (± 5.6)	37 (± 6.7)	38 (± 6.4)
Gender			
Male	13 (45%)	43 (51%)	56 (50%)

Race			
Black	14 (48%)	65 (77%)	79 (70%)
Other	0 (0%)	3 (4%)	3 (3%)
White	15 (52%)	16 (19%)	31 (27%)
Ethnicity			
Hispanic	0 (0%)	3 (4%)	3 (3%)
Marital Status			
Divorced	1 (3%)	5 (6%)	6 (5%)
Married/Cohabit	18 (62%)	35 (42%)	53 (47%)
Separated	2 (7%)	6 (7%)	8 (7%)
Single	7 (24%)	38 (45%)	45 (40%)
Widow	1 (3%)	0 (0%)	1 (1%)
Education			
<High School	1 (3%)	11 (13%)	12 (11%)
High School	3 (10%)	20 (24%)	23 (20%)
Some College	9 (31%)	26 (31%)	35 (31%)
College	16 (55%)	27 (32%)	43 (38%)
Parents Poor			
Average	20 (69%)	26 (31%)	46 (41%)
Poor	2 (7%)	31 (37%)	33 (29%)
Pretty Well Off	7 (24%)	26 (31%)	33 (29%)
Missing	0 (0%)	1 (1%)	1 (1%)
Housing status			
Owns (or buying)	15 (52%)	27 (32%)	42 (37%)
Rents	14 (48%)	52 (62%)	66 (58%)
Neither	0 (0%)	5 (6%)	5 (4%)
Wage or Salaried			
Hourly	10 (34%)	46 (55%)	56 (50%)
Salaried	16 (55%)	29 (35%)	45 (40%)
Other	3 (10%)	9 (11%)	12 (11%)
Labor Income – Mean (SD)	70000 (± 47000)	60000 (± 67000)	62000 (± 63000)
EQ (PCA) – Mean (SD)	-0.09 (0.5)	-0.04 (0.44)	-0.065 (0.52)
EQ (Linear) – Mean (SD)	3.20 (1.08)	3.14 (1.00)	3.16 (1.02)

Note: First-generation labor income is expressed in 1968 dollars; Third-generation labor income is expressed in 2019 constant dollars; Education is highest level attained; SD: Standard Deviation.

Table 3.3. Impact of Redlining on Grandchildren's Employment Quality Using Principal Component Analysis-Based Measure of Employment Quality and Covariate-Adjusted Local Polynomial Regression

			Robust Inference					
N=113	RD Estimator	MSE-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	CER-Optimal Bandwidth [meters]	N*l*	N*r*
Outcome: Mean Employment Quality (PCA)								
Model 1a	-0.21	710.74	[-0.62, -0.07]	0.14	0.015**	591.03	20	57
Model 2a	-0.27	770.58	[-0.69, -0.01]	0.17	0.041**	640.79	20	59
Model 3a	-0.32	767.44	[-0.77, -0.06]	0.18	0.023**	638.18	20	59
Model 4a	-0.30	751.56	[-0.91, 0.01]	0.23	0.057*	624.97	20	59

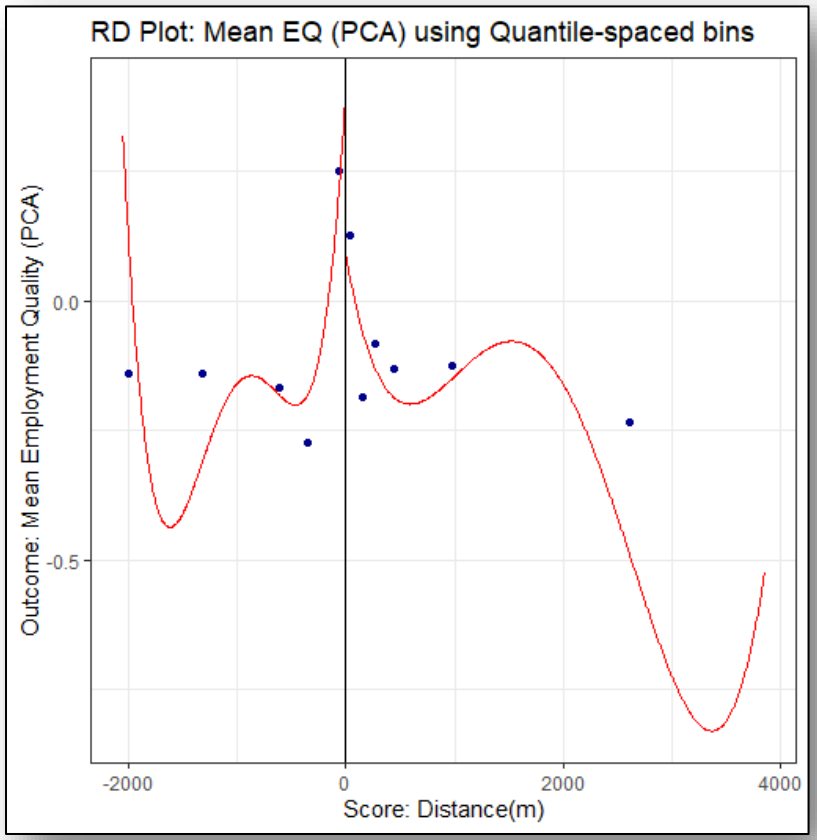
Note: Discrete analysis using cluster standard errors; Bandwidth is the distance (in meters) from yellow/red border to grandparent's census block; Adjusted models: 2a - age, year, 3a - age, year, gender, 4a - age, year, gender, race; All models use a first-degree polynomial; N*l* - *(left)*, N*r* - *(right)* indicate the effective number of observations within the MSE-bandwidth used for estimation; RD: Regression Discontinuity, MSE: Mean-Squared Error; CI: Confidence Interval, rbc: Robust bias-corrected; CER: coverage error, SE: Standard Error. *p < 0.10, **p < 0.05, ***p < 0.01

Table 3.4. Impact of Redlining on Grandchildren's Employment Quality Using Linear Specification of Employment Quality and Covariate-Adjusted Local Polynomial Regression

			Robust Inference					
N=113	RD Estimator	MSE-Optimal Bandwidth [meters]	95% CIrbc	SE	p-value	CER-Optimal Bandwidth [meters]	N*l*	N*r*
Outcome: Mean Employment Quality (Linear)								
Model 1a	-0.46	1529.46	[-1.24, 0.24]	0.38	0.183	1271.85	23	74
Model 2a	-0.72	789.87	[-2.22, 0.18]	0.61	0.096*	656.83	20	59
Model 3a	-0.76	787.46	[-2.28, 0.14]	0.62	0.084*	654.83	20	59
Model 4a	-0.74	1309.83	[-1.60, -0.01]	0.40	0.046**	1089.21	20	69

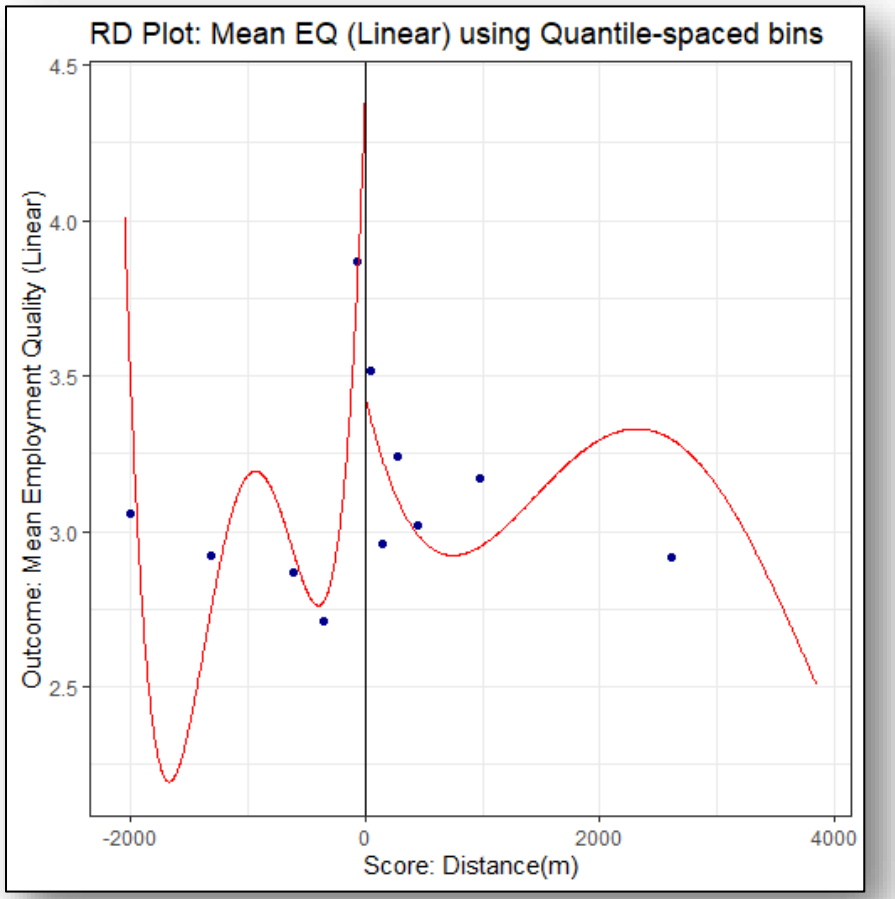
Note: Discrete analysis using cluster standard errors; Bandwidth is the distance (in meters) from yellow/red border to grandparent's census block; Adjusted models: 2a - age, year, 3a - age, year, gender, 4a - age, year, gender, race; All models use a first-degree polynomial; N*l* - *(left)*, N*r* - *(right)* indicate the effective number of observations within the MSE-bandwidth used for estimation; RD: Regression Discontinuity, MSE: Mean-Squared Error; CI: Confidence Interval, rbc: Robust bias-corrected; CER: coverage error, SE: Standard Error. *p < 0.10, **p < 0.05, ***p < 0.01

Figure 3.1. Regression Discontinuity Plot: Average EQ Score (Principal Component Analysis) Using Quantile-Spaced Bins



Note: RD plot applying the full support of the data applying a global polynomial.

Figure 3.2. Regression Discontinuity Plot: Average EQ Score (Linear) Utilizing Quantile-Spaced Bins



Note: RD plot applying the full support of the data applying a global polynomial

3.5 Appendix B. Supplementary Materials

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I. Overview of Falsification and Validation of the GRD

This study aims to investigate the causal effects of racial discrimination using a geographical regression discontinuity (GRD) design based on the Home Owners' Loan Corporation (HOLC) security maps. However, certain assumptions need to be met for valid causal inference.

Assumptions:

No Differential Response Bias: It's crucial to ensure that individuals' responses aren't directly influenced by the treatment or specific factors (e.g., knowledge of the maps). Data analysis and historical evidence were used to check for any systematic differences along the treatment boundary. **Exchangeability:** This assumption signifies that treatment assignment has not caused systematic differences in characteristics between groups. Examining characteristics near the cutoff point helps address this assumption by focusing on individuals most likely to be affected. **Continuity of Conditional Regression Functions:** Treatment shouldn't be influenced by other interventions at the cutoff, and potential outcomes for both treated and untreated groups should remain continuous around the threshold. Historical evidence suggests the maps weren't strictly aligned with administrative boundaries.

This research recognizes the inherent difficulties in fully adhering to GRD assumptions solely through data. However, it draws upon historical findings from previous researchers, such as Hillier (2005), which provide additional insights into the development of HOLC maps and their disregard for administrative boundaries, such as census tracts (146). Moreover, historical accounts indicate that these maps were selectively shared among agencies responsible for loan allocation, further reducing the likelihood that families would have known about the maps and manipulated their home location to affect their treatment assignment (147). Two studies have systematically examined the continuity assumption by identifying discontinuities related to neighborhood and resident characteristics for red-yellow borders (148,152,229). Figure B3.1 is a hypothetical example of our distance measured between yellow-lined and redlined areas.

This study has conducted additional falsification tests (balance tests of predetermined covariates, density test, bandwidth sensitivity) to evaluate the design's validity. These tests aim to detect imbalances in predetermined covariates across treatment groups, identify any

discontinuities at the cutoff point, and assess the impact of different bandwidth sizes on results. Although previous research suggests that HOLC maps generally meet GRD assumptions, this study aims to provide insights of the data used through comprehensive sensitivity analyses and falsification tests.

II. Predetermined Covariates

Pre-Treatment Covariate Balance Assessment

This study employed a rigorous approach to ensure covariate balance between the treatment and control groups prior to treatment implementation (189). This is crucial to establish the internal validity of the study and bolster confidence in the causal interpretation of any observed outcome differences.

Drawing upon established practices in the field (189), we evaluated the distribution of **predetermined covariates,** defined as observable characteristics unlikely to be influenced by the intervention. We specifically utilized data from the 1940 census to test the null hypothesis of comparable units and neighborhood characteristics across both groups. This mirrored the methodology employed in our primary analysis, ensuring consistency, and facilitating direct comparison. The detailed results of this analysis are presented in Table 1 and Figure 2. By meticulously examining covariate balance before the introduction of the intervention, we mitigated the potential for pre-existing group differences confounding our interpretation of the treatment's effects. This strengthens the internal validity of our study and enhances the reliability of our conclusions.

The outcomes of the census-level neighborhood 1940s covariate balance tests are presented in Table B3.1. The null hypothesis of equivalent predetermined covariates between the treatment and control groups was not rejected for all neighborhood-level characteristics

determined prior to treatment assignment. This indicates that there are no systematic differences among neighborhoods for grandparents, thereby validating our RD design. Figure B3.2 provides a visual representation of our covariate balance analyses.

III. Density of the Score Test

Evaluating Continuity at the Cutoff with McCrary's Density Test

To investigate potential manipulation or sorting behavior by units near the treatment assignment threshold, we adopted McCrary's (2008) density test. The null hypothesis tested posits the continuity of the density function across treatment and control units at the cutoff point (189). Any significant deviation from this null hypothesis would indicate discontinuities suggestive of non-random sorting or manipulation.

Results of the density test are presented in Table B3.2. We observe the test statistic is 6.4915, and the p-value is <0.001. This indicates we reject the null hypothesis of no difference between the density of the treated and control observations at the cutoff. In Figure B3.3, shows the density of the observations to be much greater on the redlined (treatment) side in comparison to the yellow-lined (control) side. This is mostly a function of our sample size and the number of individuals with available data to compute an employment quality score, rather than evidence of sorting behavior by the participants. Previous research has proven that it would be implausible for families to sort themselves into redlined and yellow-lined neighborhoods (148). Notably, there are more density of the observations on both sides adjacent to the cutoff point.

IV. Observations near the Cutoff (Donut-hole approach)

Strengthening Internal Validity with the Donut-Hole Sensitivity Analysis

To bolster the internal validity of our findings and mitigate concerns about potential biases arising from observations near the treatment threshold, we implemented a sensitivity

analysis known as the "donut-hole" approach (189). This technique, consistent with the methods employed in our primary analysis (189), involves systematically excluding observations progressively closer to the threshold. Each iteration then estimates the treatment effect, allowing us to assess whether observations in this proximity exert undue influence on the overall results.

This sensitivity analysis serves two primary purposes. Firstly, it allows us to evaluate the robustness of our findings to potential manipulation or sorting behavior near the threshold, a key assumption of the regression discontinuity design. Secondly, it helps to ensure that our conclusions are not driven by idiosyncratic characteristics of a limited subset of observations near the cutoff point. By demonstrating that our key findings persist across various donut-hole sizes, we strengthen confidence in the internal validity of our study and the generalizability of our conclusions beyond the immediate threshold region.

Our results from the bandwidth sensitivity analysis test (shown in Table B3.3) are in the same direction as our primary analysis. Therefore, it continues to tell the story of lower employment quality among grandchildren from redlined grandparents in comparison to grandchildren from yellow-lined backgrounds. Albeit the magnitude is significantly larger, and our confidence intervals are much wider. Additionally, the lack of significance in these estimates may largely be a function of our sample size.

VI. Supplemental Figures & Tables

Figure B3.1. Hypothetical PSID Grandparent Census Block Within An HOLC Grade D And Grade C Area With Calculated Distance From Polygon To Touching Red-Yellow Border

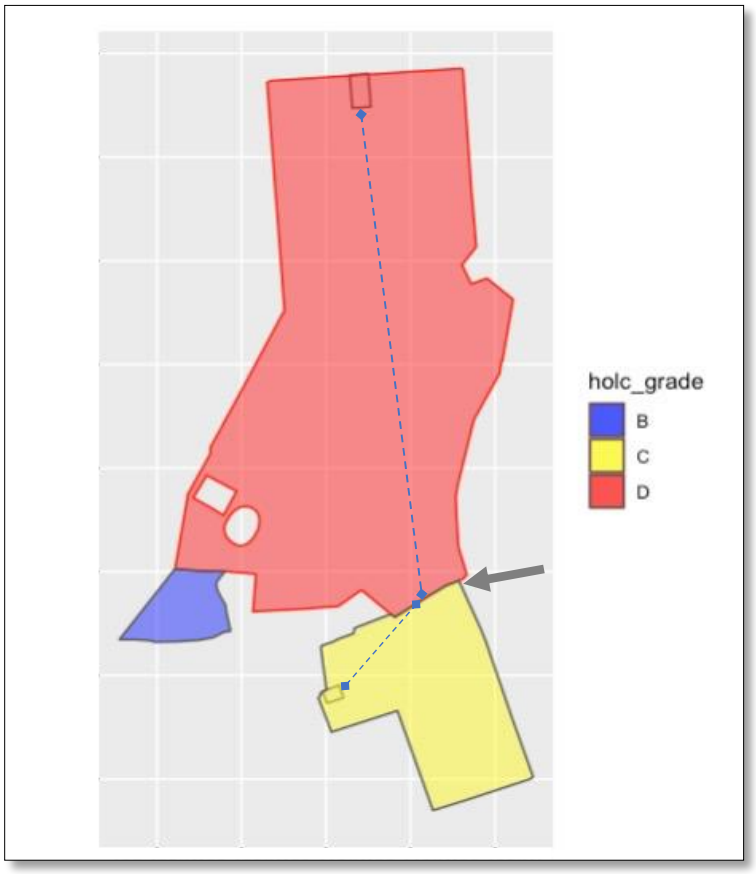


Figure B3.2. Regression Discontinuity Plot: 1940 Predetermined Covariates Using Quantile-Spaced Bins

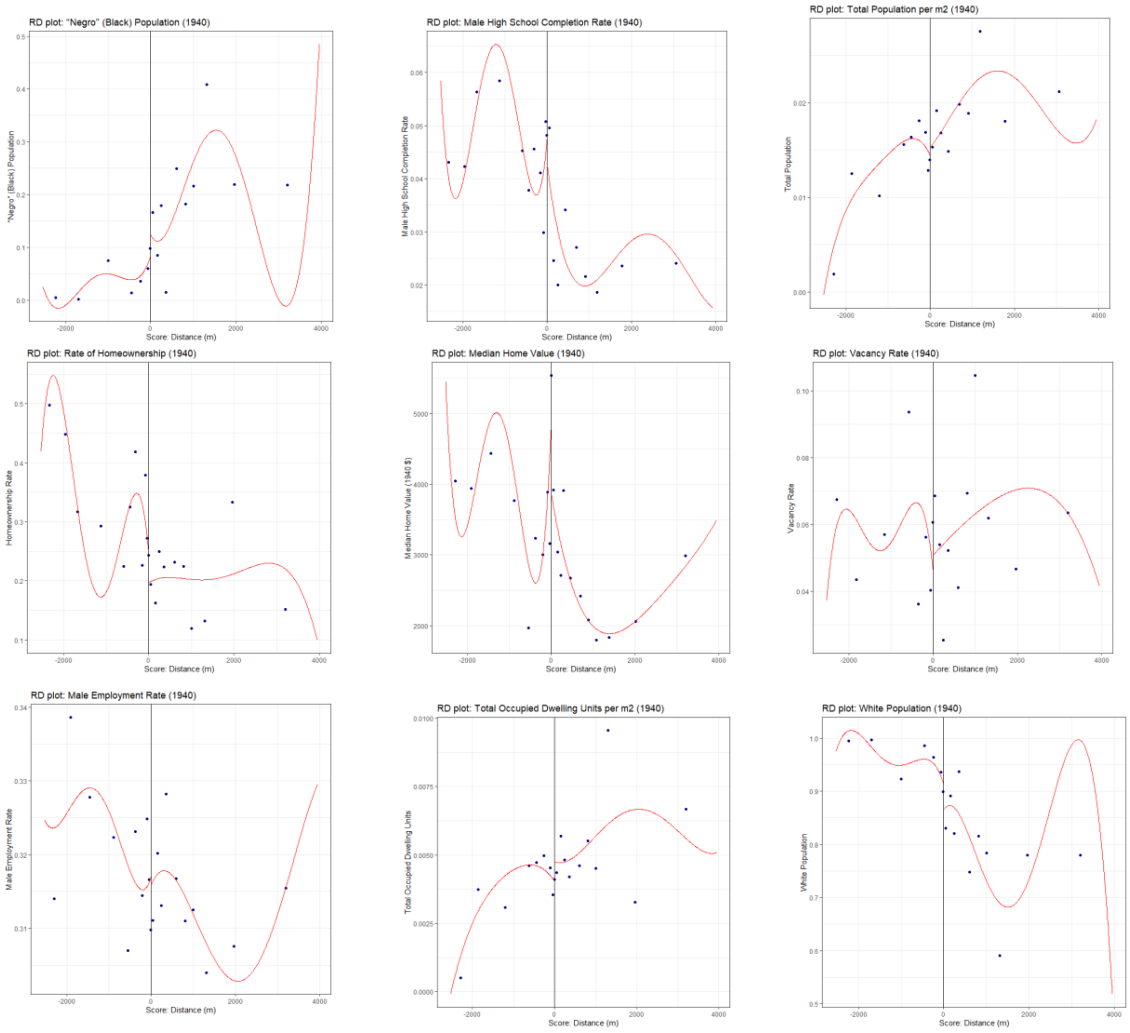
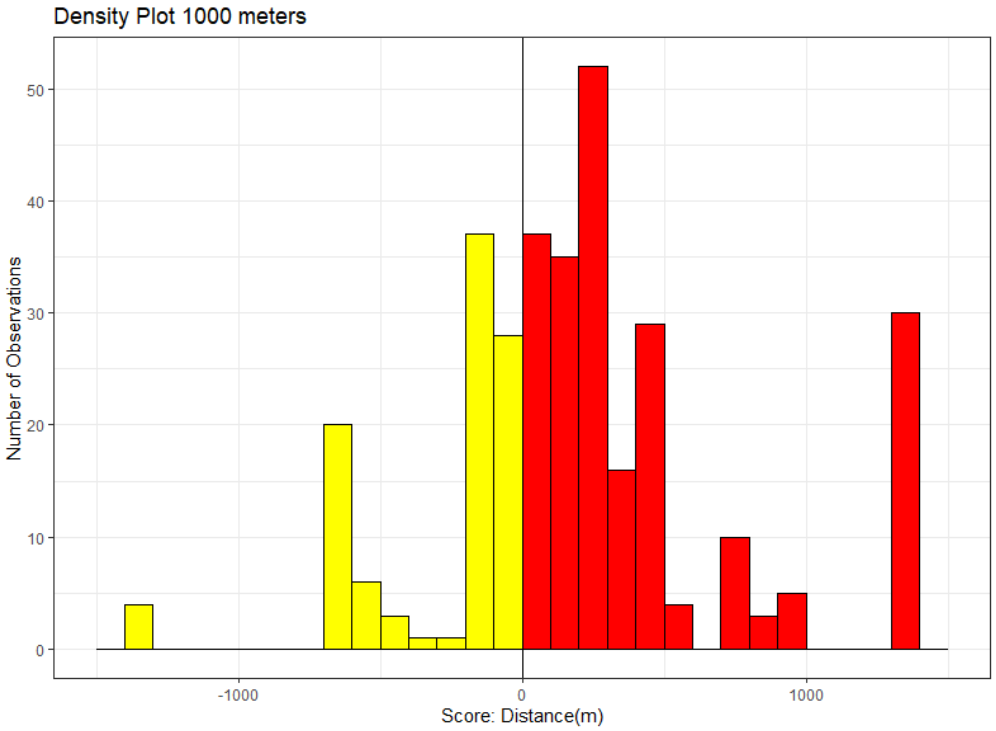


Figure B3.3. Density Plots: Sample Size of Grandchildren for Average Household Wealth and Mean Body Mass Index



Supplemental - Table B3.1. Continuity-based Regression Discontinuity Analysis: Predetermined Census 1940 Covariates Using Local Polynomial Regression

			Robust Inference				
Covariates	Coefficient	CER-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	N*l*	N *r*
Total Occupied Dwelling Units/m2	0.00	589.73	[0.00, 0.00]	0.00	0.439	57	888
Total Population/m2	0.00	566.81	[-0.01, 0.01]	0.00	0.802	57	884
White Population	-0.04	552.11	[-0.15, 0.09]	0.06	0.621	57	884
"Negro" Population	0.04	562.39	[-0.09, 0.15]	0.06	0.607	57	884
Homeownership Rate	-0.07	411.15	[-0.17, 0.05]	0.06	0.308	47	758
Median Home Value	-706.36	528.97	[-2494.90, 806.89]	842.31	0.316	56	883
Male Employment Rate	0.00	573.19	[-0.02, 0.01]	0.01	0.601	57	885
Male High School Completion Rate	0.01	396.64	[-0.01, 0.02]	0.01	0.361	45	737
Vacancy Rate	0.01	484.64	[-0.02, 0.04]	0.01	0.428	54	880

Note: Discrete analysis using cluster standard errors; All models use first-degree polynomial. Median home value is rounded to the nearest whole dollar; CI: Confidence Interval, rbc: Robust bias-corrected; CER: coverage error, SE: Standard Error.
*p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table B3.2. Continuity-based Approach Density Test

Number of available observations = 372

n (left)	n (right)	statistic	p-value
128	244	6.4915	<0.0001

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

Supplemental - Table B3.3. Continuity-based Analysis: Primary Employment Quality Outcomes Applying the Donut-Hole Approach

			Robust Inference					
Donut-Hole Radius	RD Estimator	MSE-Optimal Bandwidth [meters]	95% CI_{rbc}	SE	p-value	CER-Optimal Bandwidth [meters]	N *l*	N r
Outcome: Employment Quality (PCA)								
10	-2.43	301.88	[-7.00, 2.09]	2.32	0.289	252.713	3 8	9 9
50	-2.15	315.29	[-6.15, 1.84]	2.04	0.289	264.320	3 8	8 7
Outcome: Employment Quality (Linear)								
10	-6.44	288.98	[-18.12, 5.08]	5.92	0.271	241.912	3 8	9 9
50	-6.35	299.91	[-17.06, 4.27]	5.44	0.240	251.419	3 8	8 7

Note: 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*l* - *(left)*, N*r* - *(right)* indicate the effective number of observations within the MSE-bandwidth used for estimation; RD: Regression Discontinuity, MSE: Mean-Squared Error; CI: Confidence Interval, rbc: Robust bias-corrected; CER: coverage error, SE: Standard Error. *p < 0.10, **p < 0.05, ***p <0.01

CHAPTER 4. Understanding the Mediating Mechanisms of Structural Racism: Intergenerational Influences of Redlining on BMI Disparities through Wealth and Employment Quality

4.1 Introduction

Disparities in obesity and obesity-related conditions, such as cardiovascular disease, diabetes, hypertension, and specific types of cancer, among individuals of different racial groups and socioeconomic status (SES) are well known (1,60,271,272). These disparities have been linked to a variety of factors, including institutional and structural racism (45,46). This study aims to understand the mechanisms that link structural racism to health inequities. Our goal is to determine whether the generational effects of redlining, a federal policy supporting racially discriminatory home lending practices, on body mass index (BMI) operate through two particular social determinants of health – intergenerational wealth and employment quality (EQ).

Recognizing the intergenerational connections among structural racism, social determinants of health (i.e., wealth and employment quality), and BMI outcomes can provide valuable insights into how historical policies and decisions have long-lasting consequences for populations. Wealth and employment are two key SDOHs that reflect the accumulation of social, economic, and environmental resources over time and can either increase or decrease an individual's likelihood of experiencing health risks or benefits (234,242,273). These determinants are often shaped by policies implemented by decision makers, which have been influenced by racism, and can impact how racialized individuals have access to different resources. Evidence suggests that structural racism and neighborhood segregation contribute to the racial wealth gap (274), and health inequities (46). Previous research has identified a relationship between grandparents who have experienced redlining practices and their

grandchildren's lower multigenerational wealth and poorer employment quality outcomes (262,275). However, no studies have yet used redlining policies as a specific measure of structural racism to explore how generational wealth and employment quality, as primary drivers of determinants of health, may mediate the relationship between structural racism and body mass index. The analysis presented in this study aims to elucidate the mechanisms through which historical discriminatory systems exacerbate adverse social and economic conditions within marginalized communities that often lead to unequal health outcomes among racialized subgroups. These systems are posited as one of the underlying causes of racialized health disparities.

Phelan and Link (2015) asserted that systemic racism is a fundamental cause of health inequality (116). They identified three key associations between racism and health disparities: a) racism is a fundamental cause of racialized differences in socioeconomic status (SES), b) SES is a direct fundamental cause of disparities in health and mortality, and c) racism is a fundamental cause of health and morality inequalities, independent of SES (116). They reached these conclusions using empirical evidence that demonstrated racial differences in health risks, access to health-promoting resources, and ultimately, health outcomes (116). Moreover, they identified multiple flexible resources (i.e., prestige, power, social connections, and freedom), of which SES is a part, which operates through multiple replaceable mechanisms to produce racial differences in health outcomes (116,276). In our study, we specifically examine a form of systemic or structural racism – redlining, a practice institutionalized by the federal government – to understand how wealth and employment quality may act as mechanisms or mediators on the causal pathway between structural racism and body mass index outcomes across multiple generations.

Although several studies report that lower (as opposed to higher) levels of wealth are typically associated with increased prevalence of obesity, the measures of wealth utilized in these studies have been limited and have not considered the intergenerational aspects of wealth accumulation (64). Researchers have demonstrated that grandparental wealth plays a crucial role in wealth accumulation for their grandchildren, beyond the influence of parental wealth (83,277)**.** These advantages contribute to social and economic mobility for grandchildren of wealthier grandparents, enabling them to achieve advancements in realms such as home buying (83), which can influence neighborhood exposures that affect health outcomes.

Due to the pervasive issue of structural racism, the wealth generated through homeownership is lower for racialized minorities, particularly Blacks and Hispanics, when compared to Whites (278,279). Given that home equity is a significant component of American wealth portfolios, homeownership serves as a critical pathway for wealth building, especially later in life when ownership is sustained over time. Moreover, studies have demonstrated that neighborhoods are associated with opportunities or characteristics, such as quality of education, crime rates, property value stability, employment opportunities, and neighborhood accessibility, that contribute to wealth accumulation. Those living in lower opportunity neighborhoods, which are characterized by fewer positive characteristics compared to higher opportunity neighborhoods, are more likely to face disparities in wealth building opportunities (141). A study that examined neighborhood opportunities and race using the Panel Study of Income Dynamics (PSID) found that Black families with children were almost four times as likely to reside in lower opportunity neighborhoods compared to White families, and nearly half of the PSID families who identified as White resided in high opportunity neighborhoods (141).

In communities where there is a higher proportion of Black and other racialized minority populations, there is a deficiency of investment in health-promoting neighborhood amenities, such as walkable areas, green spaces, and healthy food access (80,130,280,281). This lack of investment is often a result of systemic racism (74,76,77). Consequently, these areas often become obesogenic environments, which are characterized by factors that promote obesity rather than healthy weight (45,76,79–81). Based on this, we propose that intergenerational wealth could serve as a mediator in the causal relationship between redlining and higher BMI among marginalized communities. Furthermore, the resources available in one's neighborhood can directly impact the quality of social connections, which in turn influence access to a range of high-quality employment opportunities that affect health and contribute to health inequities (237,257,258).

Employment in the United States (U.S.) often exhibits racial patterns, and certain aspects of employment quality may contribute to disparities in health outcomes among different racial and ethnic groups (18,276,282). Studies have shown that residents of socioeconomically disadvantaged neighborhoods have lower educational achievements, especially in higher education, and struggle to maintain stable employment, often leading to precarious or low-quality employment which has been linked to poorer health outcomes (86,236,237,263–265). Precarious employment, characterized by factors such as job insecurity, irregular work schedules, limited career prospects, low wages, and inadequate benefits, has a significant impact on an individual's health and well-being (99). There are three mechanisms by which precarious employment impacts health and well-being through three primary mechanisms: material deprivation, psychosocial stress, and exposure to workplace hazards (102). When this form of employment results in material deprivation, it can lead to poor housing conditions, inadequate

access to healthcare, and a poor quality of diet, all of which can result in adverse physical and mental health outcomes (102,276,283). In many disadvantaged neighborhoods, particularly those with high concentrations of racialized minorities such as Black families (284), these challenges are particularly pronounced (102,276,285). There is a growing need to examine the nexus between structural racism and health inequalities, as well as the potential role of employment quality as a locus wherein structural racism engenders health inequities (18,126,240). As systemic racial disparities may intersect with the instability and economic precariousness inherent in precarious employment arrangements produced through lower opportunity neighborhoods, perpetuating cycles of disadvantage and limiting opportunities for marginalized communities, thereby resulting in health inequities, such as disparities in BMI outcomes.

This study extends the scope beyond individual risk factors to include intergenerational social, economic, and environmental circumstances, such as wealth and employment quality. Its aim is to uncover the fundamental mechanisms that perpetuate disparities caused by structural racism. Specifically, the study examines the relationship between structural racism, exemplified by redlining and BMI. It seeks to identify the pathways through which structural racism affects BMI outcomes and to assess the extent to which familial wealth and employment quality mediate BMI disparities.

4.2 Methods

Operationalization of structural racism

Redlining

During the late 1930s, amidst President Roosevelt's New Deal initiatives, the United States (U.S.) government established the Home Owners' Loan Corporation (HOLC) under the Federal Housing Administration (FHA). HOLC was mandated with evaluating neighborhoods

across 239 of America's largest cities to determine their eligibility for federally backed mortgages (23,145). To achieve this, HOLC institutionalized color-coded maps and a grading system ranging from A (green) to D (red), where each grade represented the perceived credit risk level for home mortgages based on various neighborhood characteristics, including demographic composition. The grading system started with A) the highest grade, colored green, and considered sound investments; B) the second highest, colored blue, and identified as minimal risk; C) the second lowest, colored yellow, and often labelled as declining with a moderate risk level; and D) being the lowest grade, and the highest risk, was assigned the color red (145,147,149,243). Significantly areas with a higher density of Black populations were often designated as "hazardous" and received the lowest grade, D – and colored red. This practice led to the coining of the term "redlining" to describe this discriminatory practice (145). Leveraging HOLC's geographical security ratings, particularly the redlining designation, provides a unique opportunity to investigate the long-term impacts of this discriminatory practice. Thus, continued examination of the effects of redlining is crucial for informing policies aimed at promoting equitable access to social and economic determinants to address persistent racial disparities in health inequalities.

Dataset

Our research examines individuals whose grandparents resided in areas rated by the HOLC for their home loan credit worthiness during the 1960s. To accurately associate each grandparent with their respective HOLC area, we utilized census block-level data from the Panel Study of Income Dynamics (PSID) and merged them with the University of Richmond's Mapping Inequality HOLC redlining maps (150,158). The PSID is a nationally representative multi-generational sample of Americans, with an additional oversampling of low-income

families (158). The PSID began collecting data on household wealth in 1984, and subsequently began collecting data on health outcomes such as height and weight in 1986 (160,161). Therefore, we utilized PSID data ranging from 1986 to 2019.

Inclusion criteria

The criteria for inclusion in our study sample were limited to individuals whose grandparents resided in areas that shared a touching redlined or yellow-lined border, in order to meet the requirements for our mediation analysis. To examine the potential pathways through which redlining might impact health outcomes, we conducted two separate mediation analyses using different analytical datasets. The first dataset was based on intergenerational wealth as the mediator, while the second dataset utilized mean employment quality score as the mediator. Additionally, we only included families in our analysis if they had available data for our exposure, mediator(s), and outcome variables.

Variables

Key Treatment Indicator – Redlining

We derived grandparents' designated HOLC location from the PSID and the Mapping Inequality project. The treatment indicator distinguished individuals whose grandparents resided in a redlined area, while families with a grandparent in a yellow-lined area were designated as the comparison group.

Mediator – Intergenerational Wealth

In our examination of the intergenerational wealth mediation, we employed grandchildren's average household wealth which was quantified by the net worth of households less any debt over the time period of 1987 through 2019. These values were adjusted for inflation using the Consumer Price Index and are expressed in 2019 standard U.S. dollars (166).

Additionally, this figure comprises business assets, checking/savings, stocks, IRA/private annuities, net worth of vehicles, equity from home(s), other assets, and debts.

Mediator – Employment Quality

Since employment quality is a multidimensional construct, we applied principal component analysis (PCA) to facilitate the creation of an EQ score. PCA has been previously employed in the literature on EQ (251,252) and is a statistical method that is used to reduce the dimensionality of a dataset while still retaining most of the variability by transforming the original variables into a smaller set of uncorrelated components (253). By utilizing the following domains of employment quality: (1) employment stability, (2) material rewards, (3) workers' rights and social protections, (4) standardized working time arrangements, and (5) collective organization (such as unions) (249,286), and using indicators within each of these domains we were able to derive an EQ score for all grandchildren. (59) We used employment data collected by the PSID over the period of 1999 through 2019 and these data were used to formulate our PCA-based EQ score. For further information on the indicators and dimensions included in our mean employment quality score, please see the Appendix for supplementary materials.

Outcome – Body Mass Index

To create our outcome variable, we used grandchildren's most recent recorded body mass index that was collected after their wealth and EQ data. Body mass index was treated as a continuous variable and was calculated based on self-reported weight and height (BMI: weight (kg) / [height (m)]^2) (167,168).

Covariates

In order to mitigate the potential influence of confounding factors, we included several covariates into our model, such as age (continuous; centered around the mean), year (a numeric

variable indicating the year of data collection), gender (a categorical variable where female is true is equal to one), education (an ordered categorical variable coded from 0 to 4, ordered as follows: less than high school, high school, some college, and college). We collapsed our race and ethnicity categories Black, Asian, Native Hawaiian or Pacific Islander, Native American or Alaska Native Black, Other, and Hispanic into a variable indicating Black, Indigenous, and persons of color (BIPOC) equal to one and non-Hispanic White equal to zero. We combined the categories of Black and those designated another race to accommodate for smaller sample size of other racialized minorities besides Blacks in the PSID, and since families who were deemed, racialized minorities were often subjected to redlining (145,149). Additionally, we included childhood poverty, which was determined by whether parents were poor as reported by the grandchildren (coded as one) or not (coded as zero).

Missing data

To address missing data, we utilized carry forward/backward imputation approaches to fill in the gaps for three participants with incomplete data for both wealth and EQ analytical samples on the following variables: racial category, education, and childhood poverty. In addition, for 15 individuals who were missing BMI in the analytical wealth dataset, we employed multiple imputation techniques to analyze a more comprehensive dataset (287).

Study design

More recent advances in mediation analysis have been achieved by incorporating quasi-experimental designs to identify parameters of interest and apply causal interpretation to the mediation analysis. Although several studies have applied instrumental variable methods in mediation analysis, to the best of our knowledge, no prior research to date has applied a causal mediation analysis using a regression discontinuity approach (288). This involves leveraging the

design of a regression discontinuity (RD) study to estimate the direct and indirect effects of an independent variable on a dependent variable through a mediator (288). In an RD design, participants are assigned to treatment or control groups based on a cutoff point of a continuous assignment variable (182). As the methodology for implementing this approach within a mediation analysis has not been fully developed, we classify this study as an exploratory causal mediation analysis (CMA) using a geographical regression discontinuity (GRD) design approach. Previous research examining the impact of redlining on intergenerational wealth and BMI outcomes has demonstrated a plausible causal relationship (262)**.** These findings consistently demonstrated that BMI was higher for grandchildren of individuals residing in redlined areas compared to those living in yellow-lined areas, although that statistical significance was not always present (262). Similarly, findings on the effects of redlining on wealth and employment quality consistently showed a negative direction and were statistically significant for grandchildren of individuals residing in redlined areas compared to those residing in yellow-lined area (262,275) [Figure 4.1]. By understanding the indirect effects of treatment on an outcome, it may be possible to shed light on the potential mechanisms on the causal pathway between redlining and BMI outcomes.

The primary objective of our study design was to pinpoint an analytic sample capable of conducting wealth and employment quality mediation analyses, focusing specifically on grandchildren whose grandparents resided in geographically close yellow-lined or redlined areas. This design was implemented to reflect the likelihood that individuals living in close proximity to one another in these areas would share several similar characteristics, as we have previously demonstrated (262). Furthermore, historical data suggests that only a limited number of government and real estate officials were aware of the HOLC maps and their borders during the

1960s, given this information, we assumed compliance with the treatment for all families under our study design (147,274).

Overview of mediation analysis

Imai, Keele, and Tingley (2010), describe causal mediation analysis as a method by which the treatment assignment (in this case redlining), influences the outcome (BMI), through an intermediate and endogenous variable, referred to as the mediator, which lies along the causal pathway between the treatment and outcome variables (289) (Figure 4.1). Our study utilized mediation with a causal inference framework to uncover the mechanisms contributing to the disparities in BMI. Using this framework allows for a more flexible identification process (290).

Causal mediation analysis allows researchers to evaluate the specific impact of a treatment on the outcome of interest by decomposing the total effect into its natural indirect and direct effects (291).The total effect represents the combined influence of the independent variable on the dependent variable. The natural indirect effect, or average causal mediation effect, represents the portion of the total effect that serves as the proposed mechanism through which the independent variable, such as redlining, operates to influence the outcome (289,291). It measures the extent to which changes in the independent variable results in changes in the mediator, which subsequently affect changes in the dependent variable.

On the other hand, the natural direct effect pertains to the portion of the total effect of the independent variable on the dependent variable that is not transmitted through any specified mediator(s) under the hypothetical scenario where the mediator(s) are held at their natural levels (289,291).

Sensitivity Analysis

Sequential ignorability

The ability to estimate nonparametric outcomes without specifying a functional form or distribution is facilitated by the identification of effects under the assumption of sequential ignorability (289,291). If this assumption holds, there are no unobserved confounders that can impact the relationships between our mediator-outcome, treatment-mediator, and treatment-outcomes (292). While the assumption of sequential ignorability is untestable, we employed sensitivity analyses for both the wealth and employment quality mediations to assess the robustness of our findings with respect to potential unmeasured confounding, by varying the effect sizes of the unmeasured confounder across a range of plausible values and examined the stability of the estimated direct and indirect effects.

Statistical Analysis

In our primary analysis, we utilized a weighted linear structural equation modeling method to estimate causal mediation effects. We employed a structural equation model to define the effects of interest using regression coefficients, constructing two parametric models, one for the mediator and another for the outcome (293,294). Specifically, for our mediator models we included our mediator, treatment, interaction terms with treatment and distance (in meters) of grandparents' location, and covariates. In our outcome models, we included all the same variables as well as the mediator. To emphasize the importance of families that were situated closer to the touching red/yellow border, we incorporated triangular kernel weights of distance from the border to a grandparent's census block location in the model (182,189)**.**

In our secondary analysis, we applied a bandwidth technique derived from a previously established geographical regression discontinuity (GRD) design. This method was based on our earlier GRD approach, in which we modeled BMI an as outcome variable and used redlining as the treatment indicator (262). To effectively test causal mediation assumptions for families

located closer to the border, we employed this GRD approach to select the appropriate bandwidth for determining the distance of 486 meters and 778 meters for the wealth and employment quality analytical datasets, respectively. All models utilized clustered standard errors, which were clustered on the family identifier.

4.3 Results

Wealth meditation sample

Table 1a provides descriptive statistics for the wealth mediation sample which includes approximately 225 grandchildren, among whom 51 had grandparents residing in the yellow-lined areas and 174 who had grandparents in redlined areas. The average age of grandchildren is 34 years (± 8.8 years), with those having a grandparent in yellow-lined areas being slightly older than those with a grandparent in redlined areas (HOLC C: 37 years (± 8.4 years); HOLC D: 34 years (± 8.8 years). A little over half of the total sample are female. In terms of educational attainment, only a quarter of participants have attained college-level education, and a greater proportion of grandchildren with a grandparent residing in the yellow-lined areas have a college education (HOLC C: 37%) compared to those with grandparents in the redlined areas (HOLC D: 21%). Approximately 76% of the overall sample are BIPOC, with 53% of grandchildren with grandparents in yellow-lined areas and 83% of grandchildren with grandparents in redlined areas being BIPOC. Notably, 45% of grandchildren with a grandparent residing in yellow-lined areas owned a home, whereas only 24% of grandchildren with a grandparent who lived in redlined areas either own or plan to buy a home. The mean wealth of grandchildren with a grandparent in yellow-lined areas was $58,000 (± $140,000), and those grandchildren with grandparents in redlined areas it is $57,000 (± $230,000). Furthermore, average BMI of grandchildren with a

grandparent in the yellow-lined areas is 28 kg/m^2(± 5.9 kg/m^2) and 29 kg/m^2 (± 7.4 kg/m^2) in those with a grandparent in the redlined areas.

Employment Quality mediation sample

The characteristics of the EQ sample are presented in Table 1b. The sample comprised a total of 113 grandchildren, among whom 29 grandchildren had a grandparent residing in yellow-lined areas (HOLC C) and 84 had a grandparent residing in redlined areas (HOLC D). The average age of grandchildren in the EQ sample was 38 years (± 6.4 years). Grandchildren with a grandparent in the yellow-lined areas had an average age of 39 years (± 5.6 years), while grandchildren with grandparents in redlined areas had an average age of 37 years (± 6.7 years). When comparing grandchildren with a grandparent residing in yellow-lined areas to those with grandparents in redlined areas, the EQ analytical sample demographics were similar in proportions to the analytical wealth sample in terms of gender, race, education level, and marital status. Only 7% of grandchildren with a grandparent from yellow-lined areas reported their parents being poor, in contrast to the 37% of grandchildren with a grandparent from redlined areas who reported the same. The mean total labor income of grandchildren with a grandparent based in yellow-lined areas was $61,000 (± $35,000), and for those with a grandparent based in redlined areas was $57,000 (± $65,000). The overall mean EQ score of the sample was low, with grandchildren with a grandparent who lived in yellow-lined areas having a slightly lower EQ score at -0.041 (± 0.44) compared to -0.11 (± 0.52) for those with a grandparent who lived in redlined areas. Lastly, BMI units were slightly higher for grandchildren who had a grandparent residing in redlined areas at 30 kg/m^2 (± 6.6 kg/m^2) compared to grandchildren with grandparents in yellow-lined areas at 28 kg/m^2 (± 5.2 kg/m^2).

Wealth mediation

Primary

Table 2 presents the mediation results for our primary models, which encompass the entire wealth sample (N=225). The total effect is estimated to be 1.32 kg/m^2 (95%CI: -1.47, 4.15), indicating that grandchildren of individuals who lived in redlined areas had a higher BMI of approximately 1.32 kg/m^2 on average, but the difference was not statistically significant at the 0.10 level. We decomposed the total effect into the average (natural) direct and indirect effects. The indirect effect, which represents the portion of the effect transmitted through our mediator wealth, is estimated to be -0.02 kg/m^2 (95%CI: -0.31, 0.23). Our average direct effect, which represents the effect of redlining on BMI, independent of our mediator wealth, is 1.34 kg/m^2 (95% CI: -1.42, 4.16).

Secondary

Table 3 presents the outcomes of the secondary analysis for the analytical wealth sample with a bandwidth of 486 meters (N = 128). Again, this bandwidth selection approach is modeled after the one utilized in a previous study that we conducted, where we investigated the relationship between redlining and BMI using a geographic regression discontinuity approach (262). The indirect effect is approximately -0.06 kg/m^2 (95%CI: -0.59, 0.37). Although our mediation results are consistent with our primary analysis, the (natural) direct effect differs from the initial analysis, with an effect estimate of around 5.72 kg/m^2 (95% CI: -0.41, 11.87). This indicates a substantial and significant direct effect at the 0.10 level. Our total effect is 5.66 kg/m^2 (95%CI: -0.57, 11.96).

Employment Quality mediation

Primary

The outcomes of the primary analysis using the full EQ analytical sample (N=113) are summarized Table 2. The estimated total effect indicates a difference of 0.91 kg/m^2 (95%CI: -1.52, 3.34) between participants with grandparents living in redlined versus yellow-lined areas, pointing to higher BMIs on average, but not statistically significant at the 0.10 level. The average causal mediation effect (or indirect effect) through EQ is an estimated -0.05 kg/m^2 (95%CI: -1.00, 0.85), which is not statistically significant at the 0.10 level. The average direct effect is 0.96 kg/m^2 (95%CI: -1.31, 3.25).

Secondary

In the secondary analysis, we used a bandwidth of 778 meters, which reduced the sample size to 79 grandchildren (Table 3). The results from our primary EQ mediation analysis differ substantially from the models presented here in the secondary analysis. Specifically, the indirect effect is estimated to be 1.61 kg/m^2 (95%CI: -0.40, 4.10), and the direct effect, which incorporates all other causal pathway mechanisms, is estimated to be 2.78 kg/m^2 (95%CI: -2.30, 7.92). Additionally, total effect estimate indicates that a grandparent's redlining experience of redlining led to a substantial marginal increase of 4 kg/m^2 in their grandchildren (95%CI: -0.96, 9.69), although these estimates are not statistically significant.

Sensitivity Analysis

The outcomes of our sensitivity analysis are summarized in Tables S1 and S2 which are in the Supplementary Materials under Appendix C. The consistency of our estimates, particularly the direct and indirect effects, was apparent across all effect sizes tested for unmeasured confounding. Generally, our results demonstrated stability in the direct and indirect effect estimates for both the wealth and employment quality mediation analyses, indicating that they are not significantly influenced by potential unmeasured confounding. However, the results still

show that they lack statistical significance for the pathways mediated by our selected mediator(s).

4.4 Discussion

Our primary objective was to investigate the long-lasting consequences of redlining on health outcomes across generations. Specifically, our study aimed to examine the mediating role of two modifiable social determinants of health—intergenerational wealth and employment quality—in the transgenerational effects of redlining on BMI outcomes. Our findings suggest we lack evidence to support the notion that the relationship between grandparent's HOLC grade and grandchildren's BMI outcome was mediated by their intergenerational wealth. Additionally, we observed the absence of a significant effect in our EQ analysis, suggesting insufficient evidence to support the claim that there is a mediating effect between redlining and grandchildren's BMI through employment quality. Moreover, the level of support for evidence for an effect of redlining on BMI is also weak in these samples. Although our results lacked statistical significance to show a mediation effect for either the wealth or the employment quality analytical samples, this study represents an innovative effort as an initial exploratory causal mediation analysis using a regression discontinuity approach to investigate the potential causal mechanisms linking structural racism to health inequities.

Although our analysis lacked strong evidence in support of connecting the mechanisms redlining with BMI outcomes through intergenerational wealth and employment quality, several studies have demonstrated a correlation between historical redlining and contemporary health disparities (8,129,130,295–298). However, there have been limited empirical investigations into the precise mechanisms through which structural discriminatory forces of redlining result in disparities in health outcomes. Research examining the influence of SES on BMI outcomes has

revealed that living in communities with higher levels of SES disadvantage is associated with significant differences in BMI, particularly among women and Black women (299). Notably, Robert and Reither (2004), found that women living in a community with higher socioeconomic disadvantage, as indicated by percentage of families receiving public assistance, incomes <$30,000, and adult employment rates, had a higher BMI independent of age, race and individual income levels (299). However, these researchers discovered that Black men in more socioeconomically disadvantaged communities had a lower BMI (78). This study emphasizes the complexity of examining BMI as an outcome across race and gender and highlights that neighborhood socioeconomic factors can pose as a potential risk for disease for certain social groups.

Evidence suggests that redlining has led to neighborhood segregation, resulting in decreased investment in community resources, often due to lower property values in these areas, which in turn has resulted in fewer health promoting amenities and obesogenic environments within marginalized, and frequently racialized minority communities (281,300). Research examining the effects of neighborhood socioeconomic (nSES) has demonstrated that nSES encompasses multiple domains that are embedded within the social and economic environment in which a person lives and ultimately serves as a mediating factor between race and ethnicity and obesity-related chronic conditions (301). It is crucial to note that race, as a social construct, does not directly cause race-based disparities in health outcomes. Rather, in our study, we argue that discriminatory race-based policies, which use race as an opportunity to separate and treat individuals in a discriminatory manner based on skin color and/or physiological features, are responsible for such disparities (108,302–304). Further investigation on how neighborhood SES is linked to structural racism in obesity outcome may be warranted.

Our research uncovered little to no evidence of a mediating effects of grandchildren's (intergenerational) wealth on the relationship between redlining and BMI. Body mass index was employed a measure of health disparities due to its association with obesity and various chronic conditions, such as cardiovascular health outcomes, which is one of the leading causes of death in the U.S. (271,305–307). Additionally, we discovered several studies linking socioeconomic status to disparities in obesity outcomes. For instance, Newton, Braithwaite, and Akinyemiju (2017) conducted a meta-analysis of the association between life course SES and with obesity, using mean BMI as the measure. They found that those with lower life course socioeconomic status had a 0.65 mean BMI difference, compared to those with higher socioeconomic status (308). Moreover, the gender-specific findings indicated that females were more likely to have a larger mean BMI difference than males, at 1.44 and 0.21, respectively (308). Although our findings did not reveal a significant mediating effect through intergenerational wealth, studies such as the one by Newton et al. (2017), highlight the need to better understand how social and economic resources contribute to creating health inequities, particularly with obesity outcomes. Identifying the root causes of lower SES, such as through inequities in employment outcomes, presents opportunities to comprehend how to implement restorative justice for subpopulations that continue to suffer from the adverse health effects of policies historically entrenched in systemic racism.

Research conducted on the distribution of employment opportunities in economically segregated areas has shown that precarious employment is more common in areas with limited or stagnant economic growth (309–312). In our secondary mediation analysis, we used a more restricted EQ analytic sample to reflect the bandwidth approach applied in our original analysis examining the plausible causal relationship between redlining practices experienced by

grandparents and their grandchildren's BMI outcomes (262). By employing a bandwidth of 778 meters for our secondary mediation EQ models, we found a direct large effect estimate that was not statistically significant at the 0.10 level. However, our analysis did not include those individuals who were unemployed which may have masked the underlying effect of employment seeking (or lack thereof) opportunities on BMI through structural factors. Research on systemic racism and social mobility, demonstrates that individuals who experience racism in some form often exhibit slower or stagnant mobility, which relates to employment opportunities, and poorer health outcomes (260,313,314). Fujishiro et al., (2017) found that ensuring that Black individuals have access to complex employment opportunities that enable them to utilize their full range of skills could reduce race-based mortality disparities, particularly between Black and white Americans. However, this may prove challenging, as Black individuals frequently encounter barriers in securing employment commensurate with their qualifications or skillset (285,315). Moreover, many racial and ethnic minorities, particularly Blacks and Hispanics, are disproportionately represented in certain job sectors such as service, manufacturing, and transportation, which may negatively impact employment quality and, consequently affect health outcomes such as BMI (316). Deeper research is needed to determine which socioeconomic resources, beyond employment quality, can influence health inequities, such as disparities in BMI outcomes.

Limitations of the study

The present study is subject to several limitations that warrant acknowledgment. One challenge in our analysis pertains to adequately capturing the multifaceted contextual factors that are intricately linked to structural racism and that evolve over successive generations to encompass multiple compounding systemic discriminatory processes within various ecological

levels (i.e., marco-, meso-, and micro-levels) that impact individuals. The intricate nature of these intergenerational factors poses a significant challenge in our analysis. To address this issue, our methodology aimed to identify grandparents who exhibited similar characteristics who happened to reside on either side of the yellow/red border by chance. By using this identification strategy in our analysis, we were able to explore how policies imbued with racial bias can exert enduring effects across multiple generations. Although we accounted for confounding with our study design and inclusion of covariates, the interpretation of our results depends largely on the thoroughness of our causal model. Therefore, the possibility of unmeasured confounding in our analysis is highly probable and difficult to address. Despite conducting sensitivity analyses to address concerns about this assumption, it is plausible that residual confounding likely persists within our models. For instance, our inability to account for individual lifestyle factors such as physical activity or medication-related BMI imposes some constraints on the interpretation of our results. Although we have conducted multiple tests to ensure the robustness of meeting causal mediation assumptions, we acknowledge that our mediation analysis still may have violated the causal mediation analysis assumptions of positivity, consistency, and the Stable Unit Treatment Value Assumption (SUTVA), which would also influence the causal interpretation of our findings. These violations could lead to biased estimates and misinterpretations of the mediating pathways (184,317,318).

Furthermore, the potential limitations to the study's sample size may have implications to the statistical power for our causal mediation analyses, wherein the size of the estimates observed, could indicate issues with statistical power. Additionally, sample size can impact the precision of the estimates and the width of the confidence intervals. In the secondary analytical sample using the GRD boundary approach, the magnitude of the point estimates was more

pronounced, resulting in some slightly significant outcomes despite a smaller sample size. It is important to note that our study utilized a GRD approach for the sample design, which renders our results sensitive to bandwidth selection and may reduce their robustness compared to scenarios where outcomes exhibit consistent patterns across varied bandwidth specifications. The smaller sample size observed in both the wealth and employment quality analytic samples may contribute to this underpowered status, highlighting the need for careful consideration when interpreting our findings within the context of these limitations. Lastly, our reliance on self-reported BMI may have introduced measurement error into our analysis.

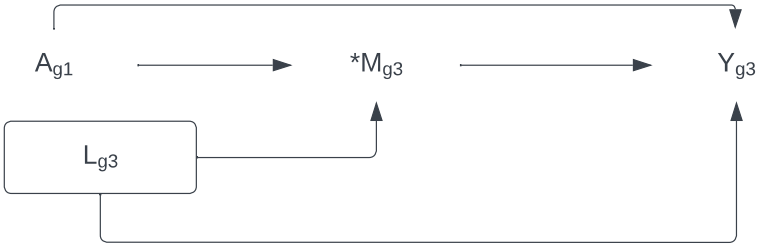
Conclusion

The objective of this study was to uncover the fundamental causes of the pervasive and substantial disparities in obesity. It underscores the significance of identifying modifiable determinants of health that can be targeted by policymakers. Our research aimed to investigate structural racism, rather than the socially-derived concept of race, as an exposure to historical discriminatory policies (109,302,303,319,320). In doing so, we examined various factors contributing to inequalities, including wealth and employment quality linked to housing policies and neighborhood environments.

Research indicates the vital role played by upstream determinants of health, particularly factors that influence obesity, and highlights the necessity of implementing policy and programmatic interventions that target these determinants concurrently with innovative clinical approaches aimed at altering health behaviors (116,321). It is crucial to address and rectify the mechanisms that have led to unequal social, economic, and environmental factors (i.e., SDOH) responsible for health disparities is crucial to promoting health equity. Addressing the underlying structural mechanisms that shape "healthy lifestyles" is a strategy for improving the health

outcomes of marginalized populations. However, our study lacks evidence to support a mediating effect of intergenerational and employment quality on the relationship between structural racism (i.e., redlining) and BMI outcomes. Further research is needed to examine the upstream processes through which structural racism, such as with redlining, exerts its influence on racialized health inequities in obesity-related conditions. Understanding these upstream processes can aid policymakers and public health officials in more effectively addressing the growing obesity crisis in the U.S.

Figure 4.1. Directed Acyclic Graph for the Relationship between Redlining and Body Mass Index Outcomes Mediated by Intergenerational Wealth and Employment Quality



*Note: *M represents the mediators Intergenerational Wealth and Employment Quality separately in the mediation analysis. g# is the generation (i.e., first, second, third); A=exposure, L=post-exposure covariates, M=mediator, Y=outcome.*

Table 4.1. Characteristics of Grandchildren in the Analytical Datasets for Intergenerational Wealth and Employment Quality

a. Wealth Dataset

	HOLC Grade C (yellow-lined)	**HOLC Grade D (redlined)**	**Total[a]**
	n=51	n=174	N=225
Age	37 (± 8.4)	34 (± 8.8)	34 (± 8.8)
Female	27 (53%)	93 (53%)	120 (53%)
Race			
BIPOC	27 (53%)	145 (83%)	172 (76%)
White	24 (47%)	29 (17%)	53 (24%)
Marital Status			
Married/Cohabiting	28 (55%)	58 (33%)	86 (38%)
Education			
<High School	4 (8%)	40 (23%)	44 (20%)
High School	15 (29%)	47 (27%)	62 (28%)
Some College	13 (25%)	50 (29%)	63 (28%)
College	19 (37%)	37 (21%)	56 (25%)
Childhood SES			
Average | Well off	42 (82%)	115 (66%)	157 (70%)
Poor	9 (18%)	59 (34%)	68 (30%)
Housing Status			
Owns (or buying)	23 (45%)	42 (24%)	65 (29%)
Rents	27 (53%)	123 (71%)	150 (67%)
Neither	1 (2%)	9 (5%)	10 (4%)
Last BMI[b]	28 (± 5.9)	29 (± 7.4)	29 (± 7.1)
Family Income	$61,000 (± $58,000)	$42,000 (± $36,000)	$47,000 (± $43,000)
Wealth	$58,000 (± $140,000)	$57,000 (± $230,000)	$57,000 (± $210,000)

b. Employment Quality Dataset

	HOLC Grade C (yellow-lined)	**HOLC Grade D (redlined)**	**Total[c]**
	n=29	n=84	N=113
Age	39 (± 5.6)	37 (± 6.7)	38 (± 6.4)
Female	16 (55%)	41 (49%)	57 (50%)
Race			
BIPOC	14 (48%)	68 (81%)	82 (73%)
White	15 (52%)	16 (19%)	31 (27%)
Marital Status			
Married/Cohabit	18 (62%)	35 (42%)	53 (47%)
Education			

<High School	1 (3%)	11 (13%)	12 (11%)
High School	3 (10%)	20 (24%)	23 (20%)
Some College	9 (31%)	26 (31%)	35 (31%)
College	16 (55%)	27 (32%)	43 (38%)
Childhood SES			
Average | Well off	27 93%)	53 (63%)	80 (71%)
Poor	2 (7%)	31 (37%)	33 (29%)
Housing Status			
Owns (or buying)	15 (52%)	27 (32%)	42 (37%)
Rents	14 (48%)	52 (62%)	66 (58%)
Neither	0 (0%)	5 (5%)	5 (4%)
Last BMI[b]	28 (± 5.2)	30 (± 6.6)	30 (± 6.3)
Labor Income	$61,000 (± 35,000)	$61,000 (± $35,000)	$58,000 (± $59,000)
EQ score	-0.04 (± 0.44)	-0.11 (± 0.52)	-0.09 (± 0.5)

Note: *Mean (± sd)* or *n (%)*; Mean family and labor income, as well as wealth are expressed in 2019 constant dollars; BIPOC: Black, Indigenous, and persons of color Education is highest level completed; HOLC: Home Owners' Loan Corporation, SES: socioeconomic status, BMI: body mass index, EQ: employment quality; a) total based on wealth analytic sample, b) Last recorded body mass index, c) total based on employment quality analytic sample.

Table 4.2. Primary Causal Mediation Analysis Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Intergenerational Wealth or Employment Quality

		Robust Inference	
	Estimate	95% CI	p-value
N=225; Mediator: Intergenerational Wealth (mean)			
Indirect	-0.02	[-0.31, 0.23]	0.86
Direct	1.34	[-1.42, 4.16]	0.33
Total Effect	1.32	[-1.47, 4.15]	0.35
N=113; Mediator: Employment Quality (mean)			
Indirect	-0.05	[-1.00, 0.85]	0.91
Direct	0.96	[-1.31, 3.25]	0.42
Total Effect	0.91	[-1.52, 3.34]	0.47

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

Table 4.3. Secondary Causal Mediation Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Intergenerational Wealth or Employment Quality

		Robust Inference	
	Estimate	95% CI	p-value
N=128; bandwidth=486m; Mediator: Intergenerational Wealth (mean)			
Indirect	-0.06	[-0.59, 0.37]	0.77
Direct	5.72	[-0.41, 11.87]	0.07*
Total Effect	5.66	[-0.57, 11.96]	0.07*
N=79; bandwidth=778m; Mediator: Employment Quality (mean)			
Indirect	1.61	[-0.40, 4.10]	0.11
Direct	2.78	[-2.30, 7.92]	0.29
Total Effect	4.39	[-0.96, 9.69]	0.11
Proportion Mediated	0.33	[-1.11, 2.66]	0.17

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

4.5 Appendix C. Supplementary Materials

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Supplemental - Table C4.1. Conceptualization of Employment Quality Dimensions using the Panel Study of Income Dynamics

		Operationalization	
Employment Quality (EQ) Dimension	**Proxy indicators**	PCA-derived EQ score	Linear EQ Score (0 to 5 pts)
[1] Employment stability	[1] Length of employment	Continuous: Age-standardized z-score	Categorical: Employed for ≥ 12 months - No, Yes [1 pt]
[2] Material rewards	[2a] Total annual labor income	Continuous: Age-standardized z-score	Categorical: ≥ Median age-standardized z-score - No, Yes [1/3 pts]
	[2b] Employer-provided health insurance	Categorical: No, Yes	Categorical: No, Yes [1/3 pts]
	[2c] Employer-provided pension contributions	Categorical: No, Yes	Categorical: No, Yes [1/3 pts]
[3] Workers' rights & social protections	[3a] Salaried employment	Categorical: No, Yes	Categorical: No, Yes [1/2 pts]
	[3b] Employer paid extra for overtime	Categorical: No, Yes	Categorical: No, Yes [1/2 pts]
[4] Working time arrangements	[4a] Number of hours worked annually	Continuous: Linear	Categorical: ≥ 32 hrs per week on average - No, Yes [1 pt]
[5] Collective organization	[5a] Employee union membership	Categorical: No, Yes	Categorical: No, Yes [1 pt]
[6] Employability opportunities	No available data		
[7] Interpersonal power relations	No available data		

Note: Labor income is adjusted to reflect 2019-value prior to standardization (Blaikie et al., 2023)

Supplemental - Table C4.2. Sensitivity Analysis for Primary Models for Causal Mediation Analysis Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Intergenerational Wealth with varying Effect Sizes

		Robust Inference	
	Estimate	95% CI	p-value
N=225; Mediator: Intergenerational Wealth (mean)			
Effect size = 0.2			
Indirect	-0.02	[-0.30, 0.24]	0.86
Direct	1.35	[-1.25, 3.98]	0.33
Total Effect	1.33	[-1.33, 3.99]	0.35
Effect size = 0.5			
Indirect	-0.02	[-0.31, 0.25]	
Direct	1.36	[-1.31, 4.06]	0.33
Total Effect	1.34	[-1.40, 4.08]	0.34
Effect size = 0.8			
Indirect	-0.02	[-0.29, 0.23]	0.9
Direct	1.36	[-1.35, 4.03]	0.33
Total Effect	1.34	[-1.38, 4.06]	0.35
Effect size = -0.2			
Indirect	0.02	[-0.30, 0.23]	0.87
Direct	1.38	[-1.27, 4.05]	0.32
Total Effect	1.36	[-1.34, 4.06]	0.34
Effect size = -0.5			
Indirect	-0.02	[-0.30, 0.23]	0.86
Direct	1.32	[-1.34, 3.92]	0.33
Total Effect	1.30	[-1.40, 3.92]	0.35
Effect size = -0.8			
Indirect	-0.02	[-0.30, 0.24]	0.86
Direct	1.32	[-1.35, 3.91]	0.34
Total Effect	1.30	[-1.42, 3.94]	0.35

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table C4.3. Sensitivity Analysis for Primary Models for Causal Mediation Analysis Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Employment Quality with varying Effect Sizes

		Robust Inference	
	Estimate	95% CI	p-value
N=113; Mediator: Employment Quality (mean)			
Effect size = 0.2			
Indirect	-0.05	[-0.93, 0.81]	0.92
Direct	0.94	[-1.15, 2.95]	0.39
Total Effect	0.89	[-1.42, 3.08]	0.44
Effect size = 0.5			
Indirect	-0.06	[-0.94, 0.78]	0.88
Direct	0.93	[-1.21, 3.00]	0.39
Total Effect	0.87	[-1.39, 3.09]	0.46
Effect size = 0.8			
Indirect	-0.05	[-0.93, 0.81]	0.89
Direct	0.97	[-1.14, 3.07]	0.37
Total Effect	0.92	[-1.37, 3.23]	0.41
Effect size = -0.2			
Indirect	-0.05	[-0.97, 0.81]	0.91
Direct	0.97	[-1.12, 3.05]	0.36
Total Effect	0.92	[-1.29, 3.20]	0.43
Effect size = -0.5			
Indirect	-0.05	[-0.93, 0.82]	0.9
Direct	0.95	[-1.21, 3.06]	0.37
Total Effect	0.90	[-1.43, 3.21]	0.44
Effect size = -0.8			
Indirect	-0.06	[-0.96, 0.80]	0.88
Direct	0.94	[-1.15, 3.04]	0.39
Total Effect	0.88	[-1.38, 3.19]	0.46

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table C4.4. Sensitivity Analysis for Secondary Models for Causal Mediation Analysis Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Intergenerational Wealth with varying Effect Sizes

		Robust Inference	
	Estimate	95% CI	p-value
N=128; bandwidth=486m; Mediator: Intergenerational Wealth (mean)			
Effect size = 0.2			
Indirect	-0.06	[-0.58, 0.36]	0.78
Direct	5.75	[-0.33, 11.80]	0.06
Total Effect	5.68	[-0.44, 11.91]	0.07
Effect size = 0.5			
Indirect	-0.06	[-0.58, 0.36]	0.76
Direct	5.67	[-0.65, 11.58]	0.07
Total Effect	5.61	[-0.79, 11.63]	0.08
Effect size = 0.8			
Indirect	-0.06	[-0.58, 0.36]	0.76
Direct	5.66	[-0.53, 11.90]	0.08
Total Effect	5.60	[-0.70, 11.95]	0.08
Effect size = -0.2			
Indirect	-0.06	[-0.58, 0.36]	0.79
Direct	5.73	[-0.48, 11.94]	0.06
Total Effect	5.67	[-0.61, 12.02]	0.07
Effect size = -0.5			
Indirect	-0.06	[-0.59, 0.37]	0.78
Direct	5.84	[-0.30, 12.06]	0.06
Total Effect	5.78	[-0.52, 12.10]	0.07
Effect size = -0.8			
Indirect	-0.06	[-0.59, 0.37]	0.78
Direct	5.68	[-0.37, 11.75]	0.07
Total Effect	5.61	[-0.55, 11.78]	0.07

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

Supplemental - Table C4.5. Sensitivity Analysis for Secondary Models for Causal Mediation Analysis Using Structural Equation Models to Assess the Mediation of the Association Between Redlining and Body Mass Index by Employment Quality with varying Effect Sizes

		Robust Inference	
	Estimate	95% CI	p-value
N=79; bandwidth=778m; Mediator: Employment Quality (mean)			
Effect size = 0.2			
Indirect	1.68	[-0.38, 4.16]	0.11
Direct	2.83	[-2.36, 7.87]	0.27
Total Effect	4.51	[-0.85, 9.79]	0.1
Effect size = 0.5			
Indirect	1.68	[-0.28, 4.12]	0.10
Direct	2.89	[-2.20, 8.00]	0.29
Total Effect	4.56	[-0.62, 9.77]	0.09
Effect size = 0.8			
Indirect	1.64	[-0.27, 3.96]	0.10
Direct	2.88	[-2.34, 8.04]	0.28
Total Effect	4.51	[-1.00, 9.74]	0.10
Effect size = -0.2			
Indirect	1.65	[-0.33, 4.14]	0.10
Direct	2.87	[-2.25, 7.94]	0.28
Total Effect	4.53	[-0.80, 9.81]	0.11
Effect size = -0.5			
Indirect	1.66	[-0.31, 4.15]	0.10
Direct	2.91	[-2.29, 8.01]	0.27
Total Effect	4.57	[-0.79, 9.76]	0.09
Effect size = -0.8			
Indirect	1.64	[-0.30, 4.09]	0.11
Direct	2.90	[-2.30, 8.07]	0.26
Total Effect	4.54	[-1.03, 9.80]	0.10

Note: Linear Structural Equation Modelling; models include kernel weights, clustered standard errors, and covariates in mediator and outcome models: age (centered), year, gender, racial category, education, childhood poverty; prop. = proportion mediated.
*p < 0.10, **p < 0.05, ***p < 0.01

CHAPTER 5. Conclusion

5.1 Summary of Findings

In Aim 1, we uncovered the ongoing and extensive consequences of historical discrimination in the federal home lending market on racial disparities in intergenerational wealth and potentially even BMI outcomes. Our results indicated a reasonable likelihood of a causal link between grandparents' experience with redlining and their descendants' reduced accumulation of intergenerational wealth. Similarly, while our data suggested a possible association between redlining exposure and higher average BMI in grandchildren compared to those with yellow-lined grandparents, the absence of statistical significance emphasized the multifaceted nature of these intergenerational processes underscored a need to further investigate these inequities.

For Aim 2, our research highlighted the profound historical impact of redlining policies on multigenerational outcomes, particularly in terms of employment quality. Our primary analysis demonstrated a plausible causal relationship between grandparents' redlining exposure and inferior employment outcomes for grandchildren, indicating significant disparities between redlined and yellow-lined grandchildren. We propose that the observed disparities may be further accentuated by comparing employment quality outcomes of grandchildren in blue- and green-lined areas within a causal study design. These findings contribute to our understanding of the intergenerational transmission of cumulative inequality, emphasizing the role of neighborhoods, social networks, and labor outcomes in shaping access to resources that influence health and well-being.

In Aim 3, our mediation analysis was designed to investigate the persistent effects of redlining on health outcomes across generations, focusing specifically on the mediating role of

average household wealth and employment quality in the transgenerational impact of redlining on BMI outcomes. Our primary and secondary analyses did not reveal evidence of mediation pathways, However, our secondary analyses showed minimally significant direct and total effects, suggesting a possible direct effect of redlining on grandchildren's BMI through multiple pathways beyond average household wealth. We emphasize that the findings from our secondary analysis should be interpreted with caution, and they highlight the need for further research that examines the complex interplay between historical discriminatory policies, socioeconomic determinants of health, and potential BMI disparities. This additional research should use more nuanced approaches and larger datasets to better understand and address these pervasive inequalities.

5.2 Strengths & Limitations

This study encounters several limitations across its aims that warrant consideration. In Aim 1, our employment of a regression discontinuity design was confined to individuals proximate to the HOLC red and yellow thresholds, constraining the generalizability of our findings to those further from these borders. Moreover, technical and privacy constraints prevented us from utilizing fine-scaled geocoded data, limiting our ability to precisely capture spatial variations. We also acknowledge the challenge of sample sparsity in racially stratified analyses, particularly in disaggregating data for all racialized groups. Additionally, the study's reliance on body mass index (BMI) as a measure of obesity presents certain limitations, as while it is the only measure used in the PSID data, its utility across different racial and ethnic groups requires careful consideration, given variations in body composition, healthcare access, and societal factors. Furthering, height and weight are self-reported by PSID participants potentially leading to some measurement error. While BMI serves as a widely used measure, its

applicability across diverse racial and ethnic populations is potentially a matter of concern in public health research.

Moving to Aim 2, we encountered limitations in the availability of data to analyze all dimensions of employment quality (EQ) as our analysis was limited to those variables available in the PSID. Specifically, dimensions such as job opportunities and training, as well as interpersonal power dynamics, were hindered by these limitations. Despite these limitations, our study's strength lies in its potential for causal interpretation, achieved by focusing on grandparents residing near the yellow/red border in the 1960s, facilitating comparisons of families with similar characteristics on either side of the border and strengthening the internal validity of our findings.

Finally in Aim 3, despite conducting sensitivity analyses, residual confounding may still exist within our mediation models. Additionally, the limited sample size of our study potentially affected the statistical power for conducting causal mediation analyses, leading to less than desirable outcomes and imprecise estimates. These limitations highlight the importance of interpreting our findings with caution, acknowledging the limitations of our study, and recognizing the need for additional research to bridge these gaps and enhance our comprehension of the intergenerational consequences of redlining and discriminatory policies on SDOH and health inequities.

While the limited sample size in our analyses in all three aims may have posed some constraints, the strength of employing novel approaches to elucidate the impact of structural racism on marginalized and racially minoritized communities should not be overlooked. These approaches offer valuable insights and have the potential to inspire additional research on the long-term impacts of federal policies over multiple generations. However, smaller sample sizes

can limit the ability to establish definitive causal relationships, therefore it is essential to have access to (or collect) extensive, historical data from larger datasets to thoroughly investigate the long-term effects of discriminatory policies, such as redlining.

5.3 Implications for Research & Policy

This research offers substantial insights for policymakers in the fields of health services, health policy, and social and economic policies. While there was no statistical significance in the relationship between redlining and Body Mass Index (BMI), the clear link between redlining and adverse outcomes in wealth and employment highlights the long-lasting impact of discriminatory housing policies on future generations. This calls for a comprehensive approach to address the intergenerational consequences of redlining and promote health equity.

In terms of health services, it is essential to develop interventions and programs that specifically target and prioritize communities that have been historically affected by redlining by addressing prevalent health concerns. Efforts should be made to improve access to healthy food options and quality healthcare facilities in marginalized communities that were redlined in the past. This can be achieved by using mobile clinics, partnering with healthy food providers, and providing transportation subsidies for medical appointments. Additionally, policy reforms are needed to address the systemic inequalities perpetuated by historical redlining, such as housing policies and improving lending eligibility for affected families across generations.

In the realm of health policy, it is crucial to advocate for measures that prevent discriminatory housing policies and practices, and to direct public and private investments towards the revitalization of redlined communities. This may involve the provision of grants for infrastructure improvement, the development of affordable housing projects, and the

implementation of business development initiatives aimed at creating wealth and quality employment opportunities.

In terms of social and economic policy implications, it is necessary to implement reparations measures to address the harms inflicted on redlined communities across generations. This may include the creation of targeted financial assistance programs, community development initiatives, and investment in affordable housing (124). It is also crucial to promote employment equity and access to quality job opportunities, recognizing the significant link between employment, access to health insurance, and health services. Closing the gap in employment quality (EQ) is achievable through stronger labor protections. Policies such as increased minimum wage, predictable work schedules, and restrictions on right-to-work laws can empower workers and lessen the burden of economic disparities on marginalized communities. Moreover, government agencies can provide equitable access to low-cost healthcare or universal health care for all (322), expand access to free early education and affordable higher education, establish safety net programs and policies to reduce child poverty and racialized economic inequality (323,324), offer redress to marginalized communities through reparation programs (325–328), and incorporate diverse voices throughout the federal government so that public servants reflect the composition of the U.S. (329).

Grassroots organizations in redlined areas should be empowered to lead policies or initiatives, fostering collective action for health and economic justice. Intergenerational wealth-building programs, such as matched savings accounts for low-income families, should be developed to promote financial stability across generations. For instance, establishing housing reparations commissions, inspired by examples like the restorative housing program in Evanston, Chicago, and similar initiatives in Santa Monica, California, and Portland, Oregon (330).

Potentially creating revisions to the Community Reinvestment Act (CRA) should aim to promote favorable lending practices, support community development, create affordable housing, and address gentrification and displacement in previously redlined communities.

Finally, acknowledgment and apology for the intentional harms caused by historical redlining are essential steps towards healing the communities harmed by these discriminatory policies. Addressing intergenerational disparities requires a multi-faceted approach, encompassing health services, health policy, and social and economic policies to promote the well-being and resilience of future generations.

Redlining's legacy continues to impact the health, wealth, and well-being of marginalized communities. The proposed policy interventions promote health equity by addressing the social determinants of health. By improving access to healthcare, fostering economic opportunities, and building intergenerational wealth, these policies can help heal the intergenerational scars of redlining and create a path towards a healthier future for all.

5.4 Conclusions

The relationship between structural racism, economic inequities, and population health is a highly intricate and multifaceted issue. This study aimed to scrutinize the intergenerational disparities in wealth and health by using empirical evidence to investigate the role of federal policies in perpetuating through neighborhood-level structural racism. It is imperative to address structural racism to improve population health and rectify the intergenerational consequences of historical injustices inflicted upon marginalized communities. The Heckler Report, published in 1985, revealed significant disparities in health status among racialized and ethnic minorities (331). Since then, the U.S. has struggled to bridge the gap on racialized health inequalities, and in some areas of health outcomes, the disparity has only widened (322). Consequently, it is

crucial for public health researchers who continue to focus on racialized health disparities to delve deeper into understanding the issue of prolonged, pervasive race-based health inequalities and examine historical and generational factors, in order to rectify and prevent the exacerbation of health inequities (123,327,332,333). It is imperative for decision-makers to set goals and assess progress toward advancing health equity and racial justice through federal policies using a generational lens that commits to sustained partnership with marginalized communities through long-term investments. To move forward with addressing the structural root causes of inequities, further research is needed to determine the best ways to implement and assess structural interventions to address social and economic determinants.

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