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The Racism-Race Reification Process: A Mesolevel Political Economic Framework for Understanding Racial Health Disparities

Abigail A. Sewell[1,2]

Abstract

The author makes the argument that many racial disparities in health are rooted in political economic processes that undergird racial residential segregation at the mesolevel—specifically, the neighborhood. The dual mortgage market is considered a key political economic context whereby racially marginalized people are isolated into degenerative ecological environments. A multilevel root-cause conceptual framework, the racism-race reification process (R^3p), is proposed and preliminarily tested to delineate how institutional conditions shape the health of racially marginalized individuals through the reification of race. After reviewing and critiquing the conceptual and theoretical roots of R^3p, the key components of the synergistic framework are detailed and applied to clarify extant understandings of the upstream (i.e., macrolevel) factors informing racial health disparities. Using aggregated data from the 1994 Home Mortgage Disclosure Act and Neighborhood Change Database merged at the mesolevel (i.e., the neighborhood cluster) with microlevel data from the Project on Human Development in Chicago Neighborhoods, exploratory analysis is presented that links dual mortgage market political economies to ethnoracial residential segregation at the mesolevel and to childhood health inequalities at the microlevel. The author concludes by considering how racial inequality is an artifact of the political economic reality of race and racism manifested from the neighborhood-level down.

Keywords

political economy, race, racism, real estate, neighborhood effects, health

INTRODUCTION

"Upstream" approaches to race hold that racism produces racial inequality through the etching of racial ideologies into societal institutions (Bonilla-Silva 1997; Carmichael and Hamilton 1967; Feagin 2000; Stewart 2008; Zuberi 2001). Such theories of race suggest that institutional policies, processes, and practices shape the arrangements and consequences of mesolevel contexts, such as the neighborhood. Upstream race theories implicate racial discrimination in institutions in the creation and ongoing presence of health disparities (Darity 2003; LaVeist 1993). Scholars cite institutional racism as a cause of racial residential segregation and its

[1]Emory University, Atlanta, GA, USA
[2]University of Pennsylvania, Philadelphia, PA, USA

Corresponding Author:
Abigail A. Sewell, Emory University, Department of Sociology, 1555 Dickey Drive, 204 Tarbutton Hall, Atlanta, GA 30322, USA
Email: abigail.a.sewell@emory.edu

consequences (Charles 2003; Feagin and McKinney 2003; Massey and Denton 1993; Oliver and Shapiro 1995; Williams and Collins 2001). Yet, health disparities researchers often conceive of segregation as a measure of institutional racism itself. Thus, a quandary is present: the effect cannot be the cause.

This essay opens the "black box" of segregation by delineating how ghettoization (i.e., the segregation of people by ethnoracial group across residential space) is harmful to health via mesolevel political economies. I situate the origins of racial inequalities in disease and illness burden in a collectivity of power dynamics that disadvantage marginalized people within a racist order of social relations rather than in the social status of race itself. Indeed, racism's multiple dimensions and levels create racial disparities in quality of health and timing of death (Harrell et al. 2011; Jones 2000; Williams and Sternthal 2010): the biological ramifications of the sociopolitical construct of race. Yet, the health consequences of the actions, and inactions, of *institutional gatekeepers* (e.g., lenders, police, politicians) in the segregation-health link are not made explicit. Overlooking the political economy of health disparities allows the white racial frame of institutional gatekeepers to never be implicated in the consequences of institutional (in)actions (Feagin 2013). Instead, researchers take residential segregation as a sufficient operationalization of institutional racism, leaving specification of the institutional processes, policies, and practices that undergird the health effects of residential segregation to the imagination, as well as their white supremacist assumptions.

Through focusing on the (in)actions of institutional gatekeepers in the dual mortgage market, this essay specifies the multilevel mechanisms that link institutional conditions to racial health disparities. The dual mortgage market is a delivery system for residential loans whereby lower income borrowers and minority consumers "are served with a different mix of products and by different types of lenders than commonly serve higher-income markets" (Apgar and Calder 2005:102). In this essay, racial health disparities denote health status differences between people at the top of the ethnoracial hierarchy (the reference population) and those at the bottom of the ethnoracial hierarchy (Braveman 2006; Carter-Pokras and Baquet 2002). The term *racial health disparities* is used to denote health outcomes in which panethnic racial minorities (e.g., blacks, American Indians, Latinos) fare worse than whites, while the term *ethnoracial health disparities* is used to denote health outcomes in which racialized ethnic minorities (e.g., African Americans, Mexicans, Puerto Ricans) fare worse than whites.

First, I conceptualize the mesolevel as a social context wherein upstream risk factors can be codified using concepts from the dual mortgage market applied to the neighborhood. Next, I specify the conceptual components of the racism-race reification process (R^3p). Finally, I conduct an empirical analysis of key components of the synergistic theoretical framework presented hereafter. I conclude by identifying several challenges to studying the health effects of the political economy of race and racism.

THE REIFICATION OF RACE

Mesolevel Race Reification

I argue that the causal mechanism that propagates segregation's negative consequences is the mesolevel reification of race. The mesolevel refers to the social realms in between the individual and the nation-state, which are commonly referred to as the microlevel and macrolevel, respectively. Figure 1 provides a simplified depiction of the multilevel nature of racial health disparities. Nation-states inform the organization of communities (e.g., neighborhoods, employers, families), in which are nested people of different (ethno)racial groups according to the sociopolitical realities of race, ethnicity, and racism. Each level can contain various representations of its basic unit. For example, nation-states can be represented as national or municipal governments, while the ethnoracial hierarchy can be represented through the (ethno)racial characteristics of individuals or products of individual actions (e.g., a mortgage application, a pedestrian stop). Macrolevel racial ideologies applied at the mesolevel trickle down to shape the lives of the oppressed. The individual is the most basic unit from which all other levels are derived.

The reification of race refers to the fact that racial bias is embedded in the institutional and social arrangements of society through the decision-making processes of disembodied and embodied institutionalized actors. Desmond and Emirbayer's (2009) conception of reification is useful here:

> Racial categories are naturalized when these symbolic groupings—the products of specific historical contexts—are wrongly conceived as natural and unchangeable. We misrecognize race as natural when we begin to think that racial cleavages and inequalities can be explained by pointing to attributes somehow

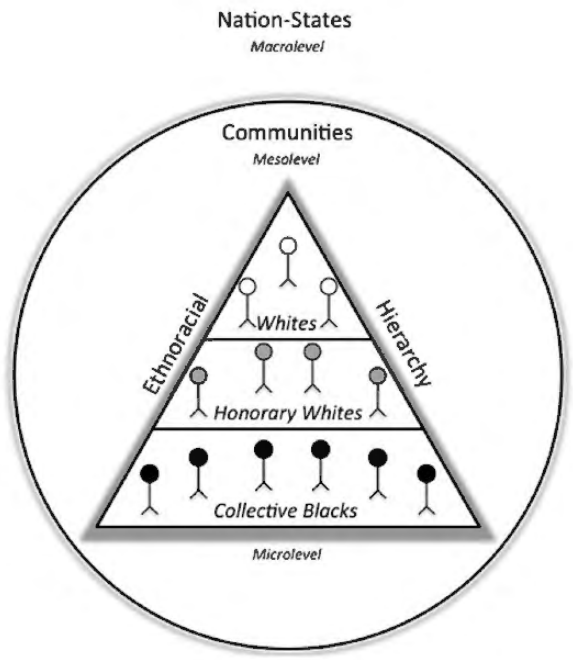

Figure 1. Multilevel Nature of Racial Health Disparities.

> inherent in the race itself (as if they were biological) instead of understanding how social powers, economic forces, political institutions, and cultural practices have brought about these divisions. (p. 339)

Race reification occurs when people act upon the social meanings ascribed to phenotypic differences among humans as if they are real (Desmond and Emirbayer 2009). The people I focus on are institutional gatekeepers. Applying this perspective, race reification, a variable mesolevel feature, can be encapsulated in the political economic processes that undergird segregation. Areal variation in reification is located in the practices of institutional gatekeepers as they distribute mortgage capital throughout the urban landscape. At the mesolevel, the reification of race occurs when institutional gatekeepers act upon understandings of phenotypic signifiers as if they reflect inherent differences among racial groups, regardless of whether they intend to discriminate because of race or not.

For instance, when financial institutions that provide small business loans designate predominately black neighborhoods as neighborhoods unworthy of credit (Immergluck 2002), the mesolevel reification of race is activated: lenders come to see predominately black neighborhoods as "redlined" neighborhoods (Berkovec et al. 1994; Bradford and Marino 1977; Tootell 1996) or areas with a credit "risk premium" (Black 1979). Residential redlining occurs when an area is disinvested by the credit industry because of the perceived and/or actual racial composition of present or future residents of an area (Hillier 2005; Squires and Kubrin 2006) or when less credit is available to existing or potential residents on the basis of their race and/or ethnicity (Ladd 1998; Turner and Skidmore 1999). While redlining operates at the institutional and neighborhood levels (Medoff and Sklar 1994), the implications of credit risks affect the ability of (potential) residents and investors to access credit throughout the urban landscape as a function of race and place. Such categorization converts the abstract idea of race into a concrete social fact: black people are not creditworthy. Therein, race is naturalized.

The end result of the mesolevel reification of race is structural discrimination through racial domination, whereby inequality is etched into the rules, norms, and logics of institutions in ways that further disadvantage racially marginalized people and privilege racially dominant people. An application of such a perspective to the dual mortgage market infers that racial bias in the distribution and quality of mortgage-related capital is also implicated in the

formation of unhealthy neighborhood environments and the ghettoization of racially marginalized people. Because reification processes occur in the everyday interactions of institutions, political economic processes have the elasticity to embed racism deep into the makings of society. Although other scholars have identified the importance of structural inequality for well-being (Massey 2004; Peterson and Krivo 2010), my conceptual framework privileges political economic forces in the construction of racial health disparities.

Privileging the Mesolevel

Scholars have long asserted that racism is etched into self and society through mechanisms that reify race (Bonilla-Silva 1997; Feagin 2006; Memmi 2000; Miles 1989). Moreover, health disparities researchers have long advocated for critical imaginations of race in studies of minority health that acknowledge the intertwined nature of race, economics, and politics (Diez Roux 2001; King 1996; Krieger et al. 1993; LaVeist 1993; Williams 1997). A sociopolitical definition of race recognizes the centrality of reification and institutions in concretizing race. Race reification begins with the transformation of racist ideologies (i.e., ideologies of worth based on racial classification schemes) into tractable sources of racial inequality. Tractability is codified in institutional practices, rules, and norms.

As ideologies beget actions, racist ideologies beget racist actions (i.e., behaviors that reflect racist ideologies of worth). I suggest that sources of racial inequality instigated by the actions of institutional gatekeepers are the upstream roots of racial health disparities. Racist actions, in and of themselves, produce stratified social outcomes. Racist actions also generate and sustain stratified social systems. Stratified social systems, kinetically, produce racial inequality. I identify direct and indirect pathways through which race, the social fact, materializes in a society as people act upon norms and values justified by racism. I argue that institutional (in)actions represent an unobserved source of heterogeneity in racial health disparities research and in studies of the health consequences of residential segregation.

Upstream reification can be theorized and operationalized at the mesolevel, because social scripts of inferiority based on phenotypic traits are codified in institutional arrangements that link racially stigmatized individuals to environments that bear the fate of racial stigma. Some mesolevel institutional arrangements are formulized in the political economy through processes of traditional (Berkovec et al. 1994; Lang and Nakamura 1993; Tootell 1996) and reverse redlining (Brescia 2009; Fisher 2009; Rheingold, Fitzpatrick, and Hofeld 2000). Others take on a more informal nature. For instance, Farley and colleagues (1994) show that negative racial stereotypes drive whites' lower ratings of neighborhood quality once blacks are present and ultimately sustain and reinforce patterns of residential segregation. Even such informal forces serve to stratify demand for housing in racialized neighborhoods (Emerson, Chai, and Yancey 2001; Farley, Fielding, and Krysan 1997; Krysan and Farley 2002; Krysan et al. 2009), thereby contributing to spatial and racial inequalities in lending practices. Research on implicit and unconscious racism and stereotyping (Blanton and Jaccard 2008; Quillian 2006, 2008) suggests that racial bias serves to reinforce the separation of ethnoracial groups, even in the absence of explicit attitudes endorsing the myth of racial inferiority (Hofmann et al. 2008). Implicit and unconscious endorsements of racial inferiority, then, also inform how institutional gatekeepers control the supply of mortgages and respond to racially contingent demands for housing.

Through the lens of home mortgages, I link the mesolevel political economy of race and racism to (ethno)racial health disparities via channels that operate through geographically-specific likelihoods and relative risks of receiving (quality) home mortgages. The provision of home mortgages is a mechanism of segregation by which institutionalized financing flows systematically into and out of neighborhoods according to the ethnoracial and economic characteristics of mortgage applicants and neighborhoods (Saegert, Fields, and Libman 2011). Racial inequalities in lending, then, result in ethnoracial inequalities in economic power (e.g., wealth, earnings) that become indelibly wedded with the multilevel disadvantages of segregation (Bradford 1979; Massey and Denton 1993). This study is distinct in its specificity when elaborating the pathways that link political economic structures to racial health disparities.

THE POLITICAL ECONOMY OF RACIAL HEALTH DISPARITIES

Areal inequalities in the (dual) mortgage market are an "upstream" apparatus, or risk factor, whereby racism structuralizes race by creating institutional conditions that foster racial inequality. The eventual consequences of these relationships are racial health disparities, which, in chainlike fashion, represent the reification of race

vis-à-vis institutional policies, processes, and practices. Extant understandings of the consequences of the political economy of race primarily have been applied to understanding institutional foundations manifested at the macrolevel (Bonilla-Silva 2015; LaVeist 1992; Omi and Winant 1994). This holds true for studies of the (dual) mortgage market. For example, empirical studies link inequalities in the cost of mortgage financing at the metropolitan area to levels of racial residential segregation in urban areas across the United States (Rugh and Massey 2010). Moreover, between-neighborhood city-specific studies have focused exclusively on access to mortgage financing, a perspective advocated by early structural race theorists (Carmichael and Hamilton 1967). These studies provide mixed evidence of the health effects of mesolevel political economies.

Mortgage Market Access

Researchers, frame the health effects of racial inequalities in access to mortgage financing within structural race theory (Gee 2002; Mendez, Hogan, and Culhane 2012). Yet, some studies indicate that residential redlining, via areal inequalities in the relative risk of access to mortgage financing between (ethno)racial groups, may be protective against illness. For instance, Gee (2002) found that the health of Chinese Americans is better if they live in neighborhoods where Asians are denied loans more than whites. In other words, *steering* Asians *into* certain areas (e.g., ethnic enclaves) may be harmful to health net of ethnoracial segregation, neighborhood socioeconomic status, and perceived discrimination. This study suggests that the actions of institutional gatekeepers that disproportionately place Asians into community together are the ultimate culprits of the negative health effects of residential segregation.

Although Mendez, Hogan, and Culhane (2011) did find that black-white differences in loan denials are linked to the density of blacks in a neighborhood and the isolation of blacks from whites, Mendez, Hogan, and Culhane (2013) find that living in redlined neighborhoods is linked to a lower risk for preterm birth for all women, especially black women. Moreover, Mendez and colleagues (2012) found that living in neighborhoods where blacks are denied loans more than whites is linked to poor health for white residents but not for black and Latino residents. Altogether, these researchers found relationships that do not meet the expectations of structural race theories.

Although these studies did not assess the mediatory effects of political economic mechanisms, the documented racial variation in the effect of redlined neighborhoods on health (Mendez, Culhane, and Hogan 2012, 2013) would suggest that there is some attenuation of racial differences in health once political economic mechanisms are considered. Furthermore, research suggests that between-neighborhood disinvestment on the part of lenders is a mechanism by which institutional racism leads to residential redlining (Oliver and Shapiro 1995; Squires 1994). As such, to truly capture the full effect of redlining, geographical disparities in loan denial should also be considered.

Dual Mortgage Market

Still, a broader conception of the institutional conditions undergirding segregation is needed. Other forms of lending inequalities, such as the ghettoization of Federal Housing Administration (FHA) loans or subprime loan concentration, may be important in contemporary U.S. society. Inclusionary processes of discrimination in the dual mortgage market vis-à-vis inclusionary processes of regulation discrimination provide a fitting instance of such institutional conditions for multiple reasons.

First, the dual mortgage market (Apgar and Calder 2005; Immergluck 1999) generates racial inequality in the distribution of nonsustainable capital (e.g., predatory, subprime, less regulated loans) because minorities face a higher risk for being provided credit products that are of poor quality upon gaining *access* to the mortgage market (Immergluck 1999; Jackson 1985). The health effects of inclusionary processes of regulation discrimination, dictated by loan characteristics in both the primary and secondary markets, have not been considered. Figure 2 charts the various decisions that can be made about a submitted application that is reviewed by an underwriter. These decisions contribute to two types of bias that can occur: exclusionary processes of access discrimination (inequalities in the ability to get an application funded in the primary mortgage market) and inclusionary processes of regulation discrimination (inequalities in the federal oversight of the applications that are funded in the primary or secondary markets).

Access discrimination is demonstrated in Figure 2 by the shaded box enclosed with dashed lines. The decision to deny a loan indicates access discrimination may be in operation. If a submitted

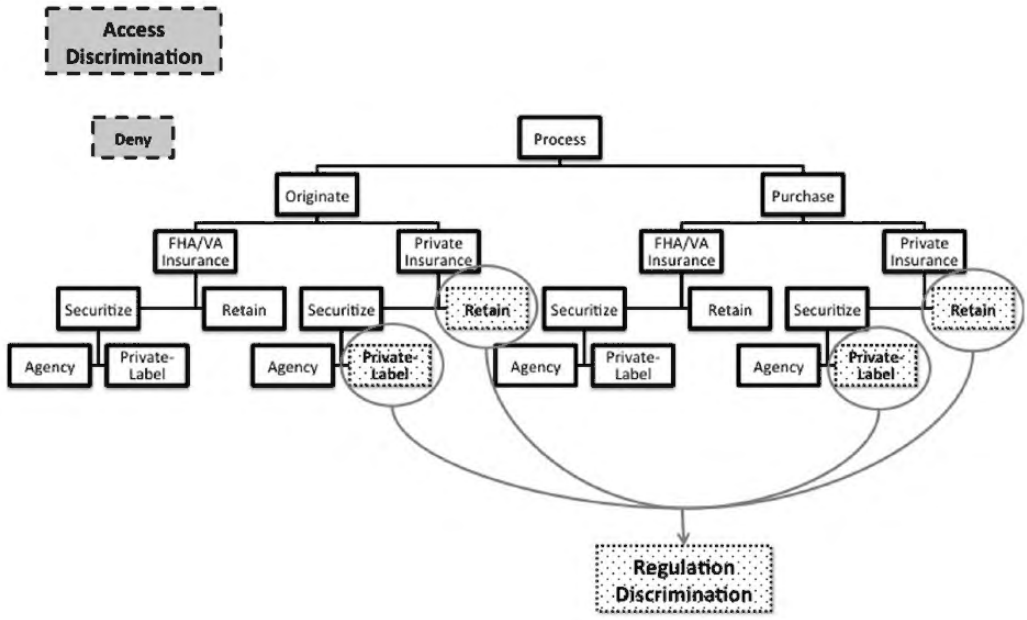

Figure 2. Dual Mortgage Market Flow: Transactions at the Application Level in the Home Mortgage Disclosure Act Database.

Note. Dashed lines and gray-shaded area indicate loan classifications that reflect access, or inclusionary processes of, discrimination. Dotted lines and dotted area indicate loan classification that reflect regulation, or inclusionary processes of, discrimination. Agency = government-sponsored enterprise; FHA = Federal Housing Administration; Private = traditional or conventional; Private-Label = nonagency conduits; VA = Veterans Administration.

loan is not denied, then it is processed. Processed loans, demonstrated in the transparent boxes enclosed with a solid line, have many different qualities, as dictated by (1) whether the institution reporting on the loan originated the loan or purchased the loan from an originating institution, (2) the type of mortgage insurance the applicant elected for the proposed loan, (3) whether the originating or purchasing institution decides to retain the loan in the financial institution's profile or securitize the loan with another institution (i.e., pool together various types of contractual debts and sell the related cash flow to third-party investors as securities), and (4) whether the institution that securitizes the loan is sponsored by the federal government (e.g., Fannie Mae or Freddie Mac) or not (i.e., a private-label entity).

All financial institutions are regulated by the federal government to some extent; however, if a loan incurs mortgage insurance from a federal agency (e.g., FHA, Veterans Administration [VA]) or is securitized by a government-sponsored enterprise (i.e., "agency"), then that loan demands additional federal scrutiny. As such, loans can "travel" pathways to regulation discrimination, which reflects the lesser extent to which institutions are regulated by the federal government. The loan characteristics that result from these pathways are circled with a shade of gray. Together, these four loan types capture regulation discrimination, which is demonstrated with a dotted box enclosed with short dashes in Figure 2. Although access discrimination largely reflects entry into homeownership and the capacity to collect housing-related wealth, regulation discrimination reflects different levels of assets used to secure a loan, with more assets needed to secure a less regulated (i.e., "private") loan in the form of larger down payments and higher originating fees.

Second, during the 1990s housing boom, a two-tiered loan system existed among funded loans, with minorities being less likely to receive prime interest rates and conventional loans than whites (Bond and Williams 2007; Squires and Kubrin 2006). Barriers to highly regulated institutional resources, such as mortgage credit from big banks, raise the cost of lending in ways that make it difficult for those who own homes to maintain or improve their homes, for those who leave their homes to sell them, and for people who want homes

to afford them (Squires, Friedman, and Saidat 2002; Squires et al. 2004). The difficulties ethnoracial minorities face eventually increase reliance on dubious financial institutions, such as subprime and payday lenders. Research indicates minorities are more likely than whites to be exposed to such fringe lenders because they are more likely to respond to public signposts and mail advertisements to enter the real estate market (Krysan 2008).

Third, mortgage lenders are more likely to sell unconventional (i.e., more regulated) loans to minorities (Bradford 1979; Canner, Gabriel, and Woolley 1991; Galster and Hill 1992). Unconventional loans are insured by the federal government (e.g., the FHA, the VA, and the Rural Development Services), whereas conventional loans are insured by all other types of banking and credit institutions (Gotham 2000; Hoyt 1972). FHA loans are the easiest type of mortgage credit to qualify for, because such loans have the most flexible loan requirement guidelines of all mortgage loans that require less than a 5 percent down payment (Hoyt 1972). FHA loan guidelines allow less than stellar credit histories, lower or absent credit scores, less cash in the bank (i.e., reserves) after closing, and higher debt-to-income ratios to secure financing. FHA loans are also less costly (e.g., lower interest rates, lower down payments) than conventional loans, especially for mortgage applicants with problematic credit histories. The versatility and cost-effectiveness of FHA loans make them attractive for all mortgage applicants and the *only* option for many mortgage applicants. Banks consider FHA loans to be less risky investments than loans backed by private companies because the government guarantees the mortgage insurance. The selling of unconventional loans to mortgage applicants, then, might be considered a form of risk aversion by credit institutions. Yet, the health effects of (less) government regulation of loans have not been considered.

Critique of Extant Research

Institutional processes are an important component of conceptualizing racial health disparities (King 1996; Krieger et al. 1993; LaVeist 1992, 1993; Williams 1997). As a macrolevel stressor (Holmes and Rahe 1967; Pearlin et al. 2005; Thoits 1995), structural racism elevates stress levels among racially marginalized people through its linkages to racial residential segregation and other manifestations of institutional racism, including economic inequality, poor access to quality housing, and maltreatment in medicine and science (Darity 2003; Feagin and McKinney 2003; Gee and Ford 2011; powell 2007; Williams 1985). As an opportunity structure, structural racism creates uneven access to the goods and services of society through institutional norms, rules, and roles that favor whites and disfavor ethnoracial minorities (Bonilla-Silva 1997, 2001; Feagin 2000, 2006). Such goods and services of society, if had, could be used to avoid illness, protect health, and recover from stress (Phelan and Link 2015; Williams 1990).

Researchers assert that racial residential segregation plays a causal role in racial health disparities (Ellen, Cutler, and Dickens 2000). Particularly, Williams and Collins (2001) indicate that racial residential segregation links multiple forms of inequality by producing racial differences in household socioeconomic status and neighborhood differences in (1) socioeconomic status (e.g., neighborhood poverty or wealth), (2) environmental stressors (e.g., exposure to violence or presence of disorder), (3) health-related social processes (e.g., social cohesion, social networks), and (4) organizational resources (e.g., access to neighborhood organizations, safe built environments). But where in these models are the institutional actors that create segregation? Because discrimination shapes the neighborhoods in which people live and the organizations upon which people rely (Diez Roux 2001), political economic processes undergirding segregation are important to highlight as "upstream" mechanisms of population health inequalities by race.

The "contract" between the dominant and the oppressed (Mills 1997, 2000), then, is the fundamental starting point for understanding the constrained agency of individuals marked indelibly by racial stigma. As subjects of the U.S. empire state built on foundations of white supremacy (Feagin 2006, 2013; Jung 2015), racially marginalized populations (un)wittingly respond to "racial domination," a type of symbolic, political, social, and economic power situated in the hands of whites that manifests in both institutional and interpersonal forms of racism directed at people of color (Desmond and Emirbayer 2009:344–45). Similar to treating black political empowerment as a metropolitan-level predictor of infant mortality rates for African Americans (LaVeist 1992, 1993), an examination of the dual mortgage market focuses attention to institutional conditions that shape racial health disparities. It does so, though, by focusing on a political economic entity that informs the very fact that black political representation will even solidify in a metropolitan area via the voting proclivities of blacks.

Phelan and Link (2015) suggest that racism is a "fundamental cause" of racial health disparities because (1) racism instigates racial differences in socioeconomic status; (2) socioeconomic status is linked to health inequalities in multiple ways through varying intermediate mechanisms; and (3) racism, in its multiple levels, exerts associations with health independent of socioeconomic status. Yet, from their perspective, political economic institutions are just one way through which racism creates links between racial group status and health. I argue that the (in)actions of institutional gatekeepers give rise to uneven access to the goods and services of society and, thus, themselves are worthy subjects of inquiry.

My focus on dual mortgage market allows a specific instance of institutions as "rivers": the backdrop upon which the strive for health unfolds (Stewart 2008). Even proponents of the fundamental cause thesis note that it is not segregation, per se, that is causative but rather the racist ideologies that generate segregation (Williams, Sternthal, and Wright 2009). Thus, racial segregation is not itself institutional racism, but rather segregation is an outcome, or manifestation, of institutional racism. Why not then spell out the connections between health disparities and institutional (in)actions rooted in racism?

THE RACISM-RACE REIFICATION PROCESS

Using a synergistic conceptual tool called the racism-race reification process, or R^3p ("R-cubed-p"), in this essay I reimagine the central role played by mesolevel indicators of the dual mortgage market in fostering and perpetuating racial health disparities via "local racial formations" (Bonilla-Silva 2015:82). Centralizing mesolevel political economies highlights the power dynamics between institutions and consumers as the pivot point through which ghettoization occurs. Institutional (in)actions cluster racially marginalized people into ethnoracially segregated places that produce detrimental risk factors. R^3p situates racial health disparities as an inevitable product of racial and geographical inequalities in the actions and inactions of institutional gatekeepers. R^3p identifies the links between institutional (in)actions and more immediate determinants of health, such as racial residential segregation and household socioeconomic status.

Studies of the health consequences of "neighborhood effects" (Diez Roux 2001; Leventhal and Brooks-Gunn 2000) provide analytical and theoretical leverage to study the illness consequences of living in a system of racial domination by highlighting the efficacious role of community processes in shaping quality of life. At a more general level than neighborhood effects theory, structural race theories suggest that upstream forms of racism shape life chances through the etching of an ideology of race into the institutions of society (Bonilla-Silva 1997, 2001; Carmichael and Hamilton 1967; Feagin 2006; Jung, Costa Vargas, and Bonilla-Silva 2011; Zuberi 2001). Thus, this essay represents a merging of two theoretical traditions in service of each other; the result is a critically informed neighborhood effects approach that privileges institutional artifacts of race and racism.

In the coinage of the term *institutional racism*, Carmichael and Hamilton (1967) described institutional manifestations of a racist society as "acts, decisions, or policies which: (a) occur at the community level through the operation of established and respected forces in society, and (b) . . . rely on the active and pervasive operation of anti-black attitudes and practices" (pp. 4–5) Thus, structural racism can be associated with illness through an understanding of the microlevel (e.g., individual, household) consequences of institutional (in)actions codified at the mesolevel. Institutions, basically, are the organizing structure between the microlevel and macrolevel. The implication of this principle is that mesolevel contexts, such as the neighborhood, should have sufficient variation in the mesolevel manifestations of institutional processes, policies, and practices to distinguish population health. The simultaneous application of structural theories of place and race, then, would suggest that institutional processes and conditions that circumscribe and create communities (Immergluck 1999; Jackson 1985; Massey and Denton 1993) instigate and perpetuate racial health disparities.

Theoretical Roots

The perspective of the neighborhood I use begins with postcolonial theories of race and racism that centralize political economic relationships forged around real property. Racial inequality is a function of sociopolitical powers needed to initiate and sustain colonialism and its derivatives via tools such as slavery and segregation. According to W.E.B. Du Bois (1898), "the Negro Problem" is fundamentally one of the symbiotic relationship between economic exploitation and racial

subjugation in colonial and postcolonial societies. *The Philadelphia Negro* (Du Bois 1899) includes detailed examinations of racial inequities in homeownership, housing-related wealth, and rent discrimination. For instance, in Philadelphia's Seventh Ward, where the vast majority of black Philadelphian homeowners lived at the end of the nineteenth century, Du Bois (1899:179) noted that only 8 percent of black families owned property. Moreover, the value of these properties was only 4.5 percent of the value of the property for the entire Seventh Ward, a locale where more than two in five residents were black. Moreover, Du Bois (1899) indicated that black Philadelphians (1) faced lower rates of property ownership and property equity, (2) were afforded use of poorer quality property, (3) spent more of their income on temporary attachments to property than white Philadelphians, and (4) resided in unhealthy living environments.

During the social movements of the mid-twentieth century, postcolonial writings on race more pointedly highlighted the tangled nature of racism and place by delineating the importance of racial constructs to colonial relationships built around the appropriation, distribution, and exchange of property. For instance, in *Black Skin, White Masks*, Frantz Fanon ([1952] 1967) noted that race reification is a symptom of racism serving as a mechanism by which capitalistic visions of society are justified and actualized. In addition to outlining political prescriptions for the colonized, Fanon's ([1952] 1967, [1961] 1963) most influential works highlighted the consequences of economic exploitation for the mental health of both the individual and society, making plausible links between political economies and health outcomes.

Albert Memmi, meanwhile, gave racism an even more central place in the development and propagation of colonialism. In *The Colonizer and the Colonized*, Memmi ([1957] 1965) noted that racism is a function of domination: "All efforts of the colonialist are directed towards maintaining the social immobility [of the colonized], and racism is the surest weapon for this aim" (p. 74). From Memmi's perspective, racism is ingrained in every colonial institution (e.g., America) and establishes the subhumanity of the colonized. So it is not that economic needs shape racism but that racism in and by itself is necessary as a form of colonial power. Colonizers use techniques of terror (e.g., lynchings, policing) to reinforce fear in the mind of the colonized, solidify submission to colonial rule, and quell reactionary uprisings. Population growth among the colonized is viewed favorably, as it facilitates competition among laborers within a fixed resource system that inevitably leads to compromised standards of living. The disappearance of the colonial relationship is not feasible in the mind of the colonized, because the position of both the colonized and the colonizer within the colonial system is fixed, even if assimilation is successfully and thoroughly achieved. Racism is a tool used by colonizing subjects to maintain a status hierarchy that allows the colonizer to reap the rewards of colonialism, including health and quality of life. The disappearance of the colonial subject is both desired (to eliminate the illegitimate roots of the colonizer's newfound power) and impossible (because the new society has no meaning without the presence of the colonized), which further illustrates the integral role that political economic forces have on racial inequality.

Furthermore, postcolonial theorist Aimé Césaire ([1955] 1972) described a racial disposition to the colonial relationship that is fundamental to understanding capitalism itself. According to Césaire's *Discourses on Colonialism*, antiblack racism was deployed in the development of European colonies to dehumanize populations through systematizing conceptions of barbarism and tools of exploitation. Institutional (e.g., forced labor, taxation, policing), social (e.g., intimidation, pressure, theft, rape, degradation), and psychological (contempt, mistrust, arrogance, self-complacency) apparatuses of colonial imposition were used to create an exploitative relationship that is inarguably negative. From Césaire's perspective on postcolonialism, then, racialization, racial classification, and racial subjugation are elements of the colonial relationship that allows the dehumanization of ethnoracial minorities even in the postcolonial empire state context.

America is no longer a colonial state; still, the relationship between ethnoracial minorities and whites is one that first formed under the colonial relationship between the United States and Britain and, contemporarily, befits an internal colony (Blauner 1969, 1972; Carmichael and Hamilton 1967). In the making of the U.S. global empire, slavery and segregation were used as tools to maintain racial domination (James 1994; Thompson 1975). As such, ethnoracially marginalized populations are disinvested imperial subjects of a white supremacist nation-state (Jung et al. 2011; Jung and Kwon 2013; Left Quarter Collective 2009). The basic point to draw from postcolonial theories of race and racism is that the reification of race

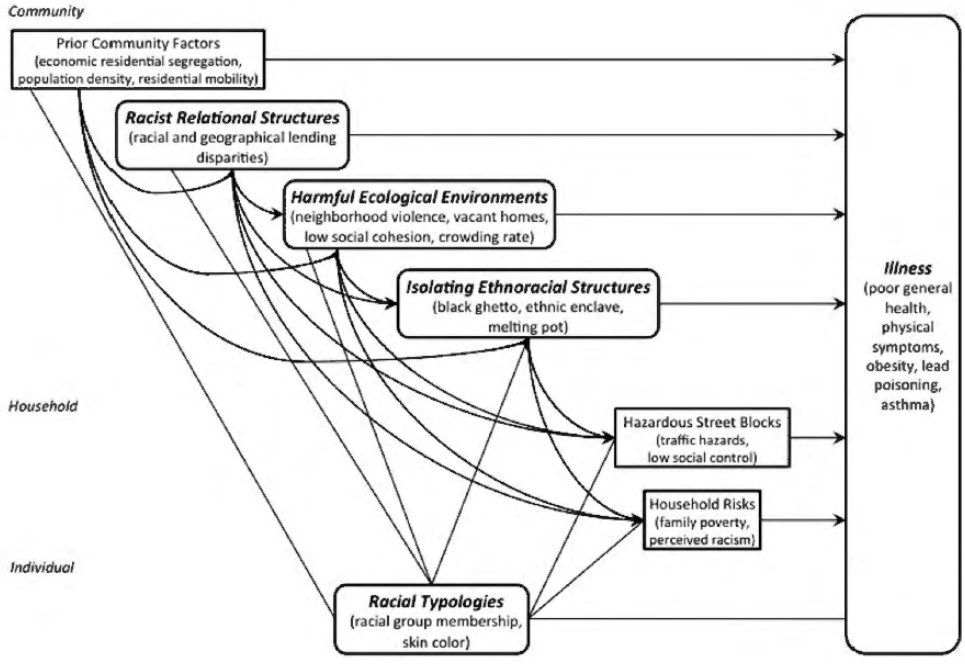

Figure 3. Illness and the Racism-Race Reification Process.

resultant from the political economic exploitation of ethnoracial minorities can have consequences that get inside the body and mind (Brown 2003; Gee and Ford 2011). A political economic perspective on illness is not new to medical sociologists (McKinlay 1975; Navarro and Shi 2001), but it typically is not applied to understanding racial health disparities.

Components

R^3p decomposes the health effects of racial and ethnic residential segregation into three definable systems: racist relational structures, harmful ecological environments, and isolating social structures. These structures buttress the reification of race and concretize racial bias. Racist relational structures are institutionalized sources of treatment bias that develop out of the (in)actions of institutional gatekeepers. Harmful ecological environments are conditions and processes in the neighborhood that cluster together problems related to the social, organizational, and environmental elements of a community. Isolating social structures are the separation of ethnoracially or economically marginalized groups from socially dominant groups in an area. These social forces capture mesolevel manifestations of the reification of race and the systematization of racial bias and race-based stigma. The conceptual model asserts that racial health disparities result from the ways that political economic processes pertinent to racial stratification (racist relational structures) ghettoize racially marginalized people into disadvantaged communities (isolating social structures) and detrimentally shape the communities in which racially marginalized people live (harmful ecological environments).

Political economic institutions create racial inequalities in health by constraining racial minorities to segregated environs that impart both supraindividual and proximal health risks to the individual. Figure 3 presents a graphical depiction of the role R^3p plays in creating racial health disparities. The content in the parentheses of each box is a representative example of each of the key concepts delineated in the graph. Arrowed lines represent directional relationships, while nonarrowed lines reflect associational relationships.

Racist relational structures encapsulate the central component of the reification of race and R^3p. Racist relational structures are institutionalized sources of treatment bias that develop out of the actions and inactions of institutional gatekeepers. The term *relational* is used in the sense of Goldberg's (2009) "relational racisms," whereby ethnoracial prejudice and discrimination are

located in the common relationships between racial classification schemes and racialized conditions that operate through structured interactions of inequality and asymmetrical power relations. Such structures reflect and perpetuate differential treatment on the basis of the racial characteristics of people and places (Gee 2002). They also serve as precursors to the deterioration of the social environment and the spatial isolation of racially marginalized people (Jackson 1985; Massey and Denton 1993). Racist relational structures capture the political economic mesolevel of structural race theory.

Racist relational structures are explicit measures of institutional racism (Gee 2002; Mendez et al., 2011, 2013), whereby institutional racism as used in R^3p refers to "differential access to the goods, services, and opportunities of society by race… codified in our institutions of custom, practice, and law" (Jones 2000:1212). It exists "when the ideology of racial exploitation gives rise to normative prescriptions designed to prevent the subordinate racial group from equal participation in associations or procedures that are stable, organized, and systemized" (Wilson 1976:34). There is considerable variation in how researchers conceptualize institutional racism. Some scholars suggest that institutional racism is indicated by the presence of health-damaging entities in an area, for example, alcohol-serving businesses, smoking advertisements on billboards, and fast food restaurants (Kwate 2008). Other scholars view racial residential segregation as a form of institutional racism (Schulz et al. 2002; Williams and Collins 2001). This essay clarifies the disparate approaches to understanding the health consequences of institutional racism by distinguishing among the health consequences of institutionalized forms of (1) racial bias (racist relational structures), (2) material deprivation (harmful ecological environments), and (3) residential status (isolating social structures). The term *poor neighborhood quality* is used interchangeably with *harmful ecological environments*, and the term *racial/ethnic/class segregation* is used interchangeably with *isolating social structures*. R^3p establishes how racist relational structures, a new term developed here to highlight the interactional processes of structural racism, dictate health-related aspects of neighborhood quality and residential segregation.

Harmful ecological environments are shaped by the prior presence of racist relational structures, according to R^3p. This temporal role is suggested by neighborhood effects theories of health that provide a conceptually distinct space for racial discrimination (Diez Roux 2011). Harmful ecological environments are conditions and processes in the neighborhood that cluster together problems related to the social (e.g., low levels of trust), organizational (e.g., absence of neighborhood institutions), and environmental (e.g., presence of waste sites) elements of a community. Neighborhood effects studies use the term *ecological* to characterize aspects of the environment that reflect the relationship between humans and their physical and social locales. Neighborhood conditions and processes may be located proximally (e.g., the street block) or more distally (e.g., the census tract).

Harmful ecological environments capture institutionalized forms of resource deprivation across a variety of ecological arrangements that dictate the quality of a mesolevel context. The resources may encapsulate social, material, and organizational forms of capital (Galster 2012). Examples include geographical disparities in exposure to violence, vacant housing, and public parks. The presence of poor neighborhood quality in a community increases residents' exposure to illness-producing stimuli and decreases their access to resources that can be used to maintain and better health. Harmful ecological environments capture the social and material components of structural race theory that manifest as community arrangements.

Isolating social structures are social contexts that geographically separate one or more marginalized groups from more dominant groups. They are shaped by the prior presence of racist relational structures, according to R^3p. The most studied isolating social structures have been those of race, ethnicity, and class (Acevedo-Garcia 2000; Lieberson 1963; Massey and Denton 1993). The concentration of racial groups within an area is considered to be a precursor to the concentration of economic disadvantage in an area (Massey and Denton 1993). However, the concentration of ethnic groups in an area is not so intimately tied to economic disadvantage. Within the R^3p conceptual model, neighborhood socioeconomic status is considered a type of isolating economic, or class, structure. To concentrate more clearly on the health effects of racial and ethnic residential segregation, or "isolating ethnoracial structures," isolating economic structures are considered to be temporally consistent and independent of the health effects of isolating ethnoracial structures and other components of R^3p. Isolating social structures capture the compositional components of structural race theory. Distinguishing economic composition from ethnoracial composition

presents both substantive and methodological challenges that neighborhood effects studies have already acknowledged (Diez Roux 2001, 2011), especially in hypersegregated cities such as Chicago (Leventhal and Brooks-Gunn 2000; Mendenhall, DeLuca, and Duncan 2006). Analytically, such challenges can be overcome through strategic modeling as done hereafter.

Racial typologies, racist relational structures, harmful ecological environments, and isolating social structures create conditions of racism that racialize the biological capacities of a population that otherwise would not be differentiable. This reality reflects the concept of "structured racialization" (powell 2007). Each factor is assumed to have direct negative effects on health. The three structural components of R^3p (racist relational structures, harmful ecological environments, and isolating social structures) are mediators of the health effects of racial typologies. Racial typologies are the microlevel component of R^3p. Detailing each subsystem of R^3p facilitates an examination of whether residential segregation is a mediator of the health effects of power system dynamics and poor neighborhood quality (Diez Roux 2011).

Assumptions

Altogether, power system dynamics shape the negative health effects of microlevel racial typologies and mesolevel neighborhood quality and residential segregation. Direct mechanisms in Figure 3 indicate that the structural arrangements of R^3p have immediate effects on health, independent of racial differences in socioeconomic status, access to health care, perceived racism, and other correlates of health measured at the individual household and street block levels. Examples of such factors are provided in the "Controls," "Household Risks," and "Hazardous Street Block" boxes in Figure 3, respectively. Indirect pathways in Figure 3 indicate that racist relational structures generate mesolevel harmful ecological environments and isolating social structures that have negative health effects. I do not assume that there is a feedback loop between the racial group status of the people whose health status is observed and racist relational structures. Thus, racist relational structures are a clustering mechanism that ties disadvantaged people (e.g., racial minorities) to disadvantaged places (e.g., harmful communities): they expose people to different areas according to their racial typology. Lines without arrows indicate this clustering phenomenon.

Still, implicit in the understanding of power system dynamics used in this conceptual framework is the assumption that the racial group status of homebuyers affects institutional practices (Ladd 1998). This assumption is reflected in the choice of social arrangements used to measure racist relational structures. The racial group status of mortgage applicants influences how institutional gatekeepers choose to spatially distribute the goods and services of the mortgage market. Also, the racial group composition of the area (an imputed spatial status) shapes the (in)actions of institutional gatekeepers. As such, no directional relationship is assumed between racist relational structures and racial typologies.

The upstream reality of segregation and racial health disparities is implicitly assumed, but empirically unobserved, in most studies of racial health disparities (Cooper and David 1986; Kramer and Hogue 2009; Williams 1999). R^3p suggests that the reification of race can be codified in a three-step process. First, mesolevel disparities in access to the resources of the mortgage market are a function of the racial group memberships of current neighborhood residents, which themselves are a function of prior ethnoracial, economic, and environmental conditions of the neighborhood. At this initial stage, the reification of race is most evident if lender practices are a function of the ethnoracial composition of the neighborhoods for which mortgage credit is sought. Such race reification is measured using areal indicators of institutional (in) actions.

Second, mesolevel inequalities in access to the resources of the mortgage lending market are a function of who wants to move to a neighborhood. At this subsequent stage, the reification of race is most evident if there is a relationship between the ethnoracial characteristics of loan applicants and lender practices. Such race reification is measured using areal indicators of racial disparities in institutional (in)actions. Third, racist relational structures pattern areal inequalities in health. This race reification is measured by situating precedent political economies as a function of health independent of ethnoracial segregation. At this last stage, race reification occurs as the effects of the political economy of race and racism are converted into a social fact: health.

Implications

The mesolevel reification of race is the central dynamic modeled in this framework: a dynamic

that has not been conceptualized heretofore in racial health disparities research. The central thesis proposed by R^3p is that the health consequences of racial residential segregation and poor neighborhood quality in part represent the direct and indirect health effects of the prior presence of institutionalized treatment bias by gatekeepers of wealth-producing materials. That is, the health effects of isolating social structures and harmful ecological environments are an artifact of the ways that racially biased power system dynamics historically have clustered racially marginalized people into racially marginalized places. Positive health effects of racial/ethnic residential segregation, meanwhile, can be explained by the absence or suppression of racially biased institutional practices.

Focusing on the relationships between political economies and segregation as a demonstration of the utility of R^3p, five hypotheses are considered:

Hypothesis 1: Political economic mechanisms of the dual mortgage market are linked to higher and increasing levels of ethnoracial residential segregation.

Hypothesis 2: Political economic mechanisms of the dual mortgage market are detrimental to health.

Hypothesis 3: Racial health disparities are partially attenuated by political economic mechanisms of the dual mortgage market.

Hypothesis 4: The illness effects of political economic mechanisms of the dual mortgage market are partially attenuated by neighborhood quality.

Hypothesis 5: The illness effects of political economic mechanisms of the dual mortgage market are partially attenuated by ethnoracial residential segregation.

DATA AND METHODS

Data

A multilevel data set is compiled using data from three sources: (1) the Project on Human Development in Chicago Neighborhoods (PHDCN), (2) the 1994 Home Mortgage Disclosure Act (HMDA), and (3) the Neighborhood Change Database (NCDB). The primary unit of analysis is individuals nested in "neighborhood clusters," collections of census tracts delineated in socially meaningful ways (Sampson 2012). Mesolevel data describing community contexts are derived from aggregating mortgage applications filed in calendar year 1994 using data from the HMDA (Federal Reserve System 2013), from aggregating census tracts to the neighborhood cluster level and scoring 1990 and 2000 U.S. census data according to geographical boundaries as defined in 2000 using data from the NCDB (GeoLytics 2003), and from compiling ecometric data (Raudenbush and Sampson 1999) from the 1995 PHDCN Community Survey used in prior studies (Sampson 2012) and provided by the PHDCN principal investigators.

Measures

Two types of outcomes are evaluated at two different levels of measurement. First, at the mesolevel unit of analysis (n_j = 273), isolating ethnoracial structures in 2000 are analyzed as a function of racist relational structures as measured in 1994, prior socioeconomic characteristics of the community as measured in 1990, and prior isolating ethnoracial structures as measured in 1990. To measure racist relational structures, four axes of racial bias are evaluated: areal inequality in access to the mortgage market (neighborhood credit refusal, where higher values indicate less access to the mortgage market), racial disparities in access to the mortgage market across areas (racialized credit refusal), areal inequality in the federal oversight of originated loans (neighborhood credit privateness, where higher values indicate less federal oversight), and racial disparities in the federal oversight of originated loans (racialized credit privateness). These measures capture inclusionary and exclusionary dimensions of racist relational structures via both areal inequality and area-varying racial inequality. Prior characteristics of the community considered are the density of affluent families, the density of homeowners, median home values, population density, and the density of households that had moved within the past five years (residential mobility). Isolating ethnoracial structures are measured as the percentage of blacks among the total population in a neighborhood cluster (black concentration), a z score capturing the correlation between the percentage of Latinos in a neighborhood cluster and the percentage of immigrants in a neighborhood cluster (Latino/immigrant concentration), and Simpson's entropy index (ethnoracial diversity). Appendix A provides descriptive statistics for neighborhood-level measures.

Second, through a multilevel research design, illness is analyzed as a function of individual, household, and street block characteristics measured at the microlevel and neighborhood cluster characteristics, defined above, measured at the mesolevel. Two

dichotomous illness measures, each reported by the primary caregiver of the youth subject, are considered: poor or fair general health and lead poisoning. The PHDCN Health Screen is used to ascertain each of the illness indicators. Poor or fair general health is ascertained through the question "Would you say in general [NAME]'s health is: 1) excellent, 2) very good, 3) good, 4) fair, or 5) poor?" Responses to the question are dichotomized such that 1 = "fair" or "poor" and 0 = "excellent," "very good," or "good." Lead poisoning is ascertained through the question, "Has [NAME] EVER had . . . Lead poisoning?" Responses to the question are dichotomized such that 1 = "yes" and 0 = "no."

For each individual participating in wave 2 of the Longitudinal Cohort Study with nonmissing data on microlevel covariates (n_i = 3,333), information about the gender (female = 1, male = 0), age at time of the interview, and insurance status since wave 1 of the study is included in the analysis. Moreover, for every primary caregiver providing responses for youth in wave 2 of the Longitudinal Cohort Study, information about the household (highest degree of education in household at wave 1, family income [natural logarithm] at wave 1, homeownership status at wave 1, residential mobility status since wave 1, family structure at wave 2, and primary caregiver perceptions of racism at wave 1) is provided. To account for the effects of research design, covariates indicating the biological mother status of primary caregiver at wave 2, the presence of a consistent primary caregiver between wave 1 and wave 2, and a youth's inclusion in a multisubject household are included as well. For every street block included in wave 2 of the Longitudinal Cohort Study, measures of traffic hazards and ambient hazards (noise pollution; air pollution, garbage, litter, and/or trash on the street block; and poor condition of the streets and/or houses on the street) are included. Appendix B provides descriptive statistics for microlevel characteristics.

In addition to the measures of racist relational structures, isolating ethnoracial structures, and prior community characteristics captured in the mesolevel analysis, the multilevel analysis also considers the independent health effects of harmful ecological environments using three indicators—concentrated disadvantage, broken windows, and low collective efficacy—derived from a principal-components analysis of eight mesolevel measures of neighborhood quality: a neighborhood-level ecometric scale of violence exposure, a neighborhood-level ecometric scale of perceptions of neighborhood decline, the proportion of occupied homes built before 1940 in 2000, the proportion of all housing units that are abandoned in 2000 violence exposure, a neighborhood-level ecometric scale of social cohesion, a neighborhood-level ecometric scale of neighborhood-based activism, the proportion of the population five years old or less in age, and the crowding rate, which is the average number of persons per occupied homes in a neighborhood. Appendix A provides descriptive statistics for the three components used in multilevel analysis, while Appendix C provides descriptive statistics and rotated factor loadings for the three components.

Methods of Analysis

Two preliminary sets of analyses are conducted to test the viability of R^3p as a useful mesolevel framework to understand the relationship between neighborhood effects and racial disparities in health. Using ordinary least squares regression, the first set of analyses examines whether levels of or changes in the three indicators of ethnoracial residential segregation are associated with the political economy of the mortgage market with and without holding constant neighborhood socioeconomic status. Using multilevel generalized linear models for binary outcomes, the second set of analyses examines whether racial disparities in health are a function of the household, street block, and/or community components of R^3p.

RESULTS

Mesolevel Analysis

The results of the first set of analyses evaluate Hypothesis 1, that local political economies are linked to ethnoracial residential segregation. They suggest that, unsurprisingly, barriers to mortgage market inclusion are linked to higher concentrations of blacks in a neighborhood. Model 1 in Table 1 indicates that the proportion of blacks in a neighborhood is greater in neighborhoods where the rate of loan denials is high (neighborhood credit refusal). This association is not an artifact of economic segregation or population dynamics (Model 2). Although Model 3 does not suggest that loan denial rates are associated with increases in black concentration rates, it does suggest that the link between black concentration and areal inequality in access to the real estate market is likely an artifact of loan denial rates being higher in areas with large concentrations of blacks at an earlier time point. This "legacy" link represents the reification of race. Meanwhile, neighborhood credit refusal is associated with lower and decreasing rates of ethnic concentration and ethnoracial diversity.

Table 1. Ordinary Least Squares Regression Predicting the Impact of Racist Relational Structures on Isolating Ethnoracial Structures with and without Level 2 Controls.

	Black Concentration			Ethnic Concentration			Ethnoracial Diversity		
Variable	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Racist relational structures									
Neighborhood credit refusal	1.160***	.950***	.030	−.530***	−.680***	−.290***	−.730***	−.430***	−.230***
	(.100)	(.100)	(.030)	(.120)	(.110)	(.070)	(.110)	(.090)	(.060)
Racialized credit refusal	−.170+	−.030	−.010	−.030	−.070	−.010	.250*	.040	.010
	(.090)	(.090)	(.020)	(.120)	(.100)	(.060)	(.110)	(.090)	(.050)
Neighborhood credit privateness	−.070	−.240*	−.020	−.410***	−.080	−.190**	.090	.150	.190***
	(.090)	(.090)	(.020)	(.120)	(.110)	(.060)	(.110)	(.090)	(.050)
Racialized credit privateness	.300**	.270**	−.010	.000	.040	−.010	−.420***	−.380***	−.180***
	(.100)	(.090)	(.020)	(.120)	(.100)	(.060)	(.110)	(.090)	(.050)
Community characteristics									
Family affluence rate, 1990		.050	.030		−.360***	−.100+		−.030	.010
		(.080)	(.020)		(.100)	(.060)		(.080)	(.050)
Homeownership rate, 1990		−.400***	.090***		.390***	.220***		.310***	.040
		(.090)	(.020)		(.100)	(.060)		(.080)	(.050)
Median home values, 1990		.030	−.080***		−.250**	−.050		.080	.100*
		(.070)	(.020)		(.080)	(.050)		(.070)	(.040)
Population density, 1990		−.120*	−.020		.180**	−.040		.040	.000
		(.050)	(.010)		(.060)	(.040)		(.050)	(.030)
Residential mobility, 1990		−.460***	.110***		.440***	.190***		.680***	−.090+
		(.070)	(.020)		(.080)	(.050)		(.070)	(.050)
Prior isolating ethnoracial structures									
1990 black concentration			1.020***						
			(.020)						
1990 ethnic concentration						.800***			
						(.030)			
1990 ethnoracial diversity									.880***
									(.040)
Constant	−.610***	−.470***	.010	.480***	.400***	.250***	.400***	.310**	.100+
	(.100)	(.090)	(.020)	(.130)	(.110)	(.060)	(.120)	(.090)	(.050)
n	273	273	273	273	273	273	273	273	273
Log likelihood	−311	−279	115	−371	−326	−173	−348	−275	−126
Adjusted R^2	.420	.530	.970	.090	.340	.780	.240	.550	.850
F	49.410***	35.160***	1,009.820***	8.110***	16.350***	99.640***	22.210***	37.220***	150.600***
Stata BIC	651	614	−168	771	709	407	724	606	314

Note. Standard errors are in parentheses. BIC = Bayesian information criterion.
+$p < .100$. *$p < .050$. **$p < .010$. ***$p < .001$.

Table 1, uniquely so, also suggests that discrimination in inclusionary processes shapes ethnoracial residential segregation in ways that are distinct from discrimination in exclusionary processes. Inclusionary processes of discrimination are captured using measures of loan privateness. Specifically, black concentration is higher in areas where minorities are more likely than the average homebuyer to be given loans that are less regulated by the federal government (racialized credit privateness). Again, this association is independent of economic segregation and population dynamics (Model 2). The association represents a higher proportion of minorities originating less regulated loans in areas that are populated with high levels of blacks at an earlier time point (Model 3), also a race reification "legacy" link. Meanwhile, ethnoracial diversity is lower in areas where minorities are more likely to be given less regulated loans than the average homebuyer (Ethnoracial Diversity: Model 1). In fact, areas where racialized credit privateness is higher have declining levels of ethnoracial diversity between 1990 and 2000: these areas become more segregated.

Areal inequality in inclusionary processes also presents distinct links to ethnoracial residential segregation. For instance, ethnic concentration is lower and declines in areas where loans are less regulated. Meanwhile, ethnoracial diversity is higher in areas where loans are less regulated. The reverse association, therefore, is present. Ethnic concentration increases in areas where loans are unregulated, while ethnoracial diversity decreases in such areas. Overall, Table 1 provides support for Hypothesis 1: local political economies are linked to higher and increasing levels of ethnoracial residential segregation.

Multilevel Analysis

The results of the second set of analyses suggest that local political economies matter for understanding racial disparities in health. Table 2 includes four models: the first model identifies black-white and Latino-white differences in health, adjusted for age, gender, and health care access (Model 1); the second model examines hypotheses 2 and 3 by including covariates characterizing the dual mortgage market (Model 2); the third model examines Hypothesis 4 by further including covariates for poor neighborhood quality (Model 3); and the fourth model examines Hypothesis 5 by further including covariates for ethnoracial residential segregation (Model 4).

Model 1 in Table 2 indicates that blacks are more than 2 times more likely to have their general health described as "poor" or "fair" than whites and nearly 19 times more likely to ever be diagnosed with lead poisoning. Similarly, Latino youth are more than 4 times more likely to have their general health described as "poor" or "fair" than Whites and more than 10 times more likely to ever be diagnosed with lead poisoning. These patterns suggest that there are dimensions of health that demonstrate large discrepancies by race.

Holding constant racial differences in health and individual correlates of illness, Model 2 suggests that local political economies shape the prevalence of lead poisoning in a neighborhood, which provides qualified support for Hypothesis 2. Although local political economies do not appear to be linked to caregiver-reported general health, the prevalence of lead poisoning is 56 percent lower in neighborhoods inundated with less regulated loans, which means that the prevalence of lead poisoning is 2.27 higher in neighborhoods inundated with more regulated loans (e.g., FHA loans, loans securitized by Fannie Mae or Freddie Mac). Together, these findings suggest that the effects of local political economies are illness specific. Hypothesis 2 is partially supported.

Model 2 also helps assess the validity of Hypothesis 3: that racial disparities in health are partially attenuated upon considering local political economies. There is some support for this assertion. Upon the inclusion of covariates for the dual mortgage market, racial differences in the likelihood of ever having lead poisoning are partially attenuated. Yet racial differences in poor or fair general health persist, most likely related to the nonsignificant effects of local political economies on this dimension of health. Hypothesis 3 is partially supported.

Model 3 evaluates the validity of Hypothesis 4, that the illness effects of local political economies are attenuated by poor neighborhood quality. In fact, the presence of concentrated disadvantage increases the prevalence of poor or fair general health in a neighborhood, and the presence of broken windows and low collective efficacy increases the prevalence of lead poisoning in a neighborhood. However, although measures of harmful ecological environments do exert independent effects on some dimensions of health, there is no evidence that the illness effects of the dual mortgage market are attenuated upon the inclusion of measures for poor neighborhood quality. Hypothesis 4 is not supported.

Model 4 evaluates the validity of Hypothesis 5, that the illness effects of local political economies are

Table 2. Random Intercept Logistic Regression Predicting the Impact of Racial Group Membership, Racist Relational Structures, Harmful Ecological Environments, and Isolating Ethnoracial Structures on Illness Experience, Holding Individual Factors Constant.

	Poor or Fair General Health				Lead Poisoning			
Variable	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Racial group membership								
Black	2.110*	2.080*	1.760	1.490	18.900***	14.280***	12.900***	13.370***
	(2.200)	(2.050)	(1.600)	(.990)	(4.040)	(3.600)	(3.440)	(3.320)
Latino	4.590***	4.570***	3.110***	2.850**	10.240**	8.360**	6.000*	6.050*
	(4.800)	(4.710)	(3.430)	(3.130)	(3.190)	(2.920)	(2.420)	(2.380)
Individual characteristics								
Age at time of interview (centered)	1.020	1.020	1.030*	1.030*	.910***	.910***	.910***	.910***
	(1.730)	(1.750)	(1.960)	(2.010)	(–4.490)	(–4.600)	(–4.520)	(–4.460)
Female	.880	.880	.870	.880	.740	.720+	.720	.720+
	(–.930)	(–.910)	(–1.000)	(–.930)	(–1.630)	(–1.740)	(–1.780)	(–1.720)
Uninsurance spell	1.410*	1.420*	1.360+	1.350+	.980	1.010	.940	.940
	(2.160)	(2.240)	(1.960)	(1.890)	(–.060)	(.040)	(–.250)	(–.250)
Racist relational structures								
Neighborhood credit refusal		1.020	.890	.860		1.240	1.220	1.250
		(.110)	(–.670)	(–.860)		(1.020)	(.920)	(.990)
Racialized credit refusal		1.270	1.280	1.300		1.430+	1.350	1.330
		(1.420)	(1.540)	(1.630)		(1.840)	(1.520)	(1.420)
Neighborhood credit privateness		.810	1.020	1.010		.440***	.410***	.400***
		(–1.260)	(.120)	(.070)		(–3.640)	(–3.650)	(–3.630)
Racialized credit privateness		1.250	1.110	1.130		1.160	1.110	1.160
		(1.340)	(.650)	(.700)		(.750)	(.540)	(.730)
Harmful ecological environments (HEE)								
Concentrated disadvantage: HEE component 1			1.440**	1.430*			.990	1.050
			(3.220)	(2.390)			(–.050)	(.290)
Broken windows: HEE component 2			1.190+	1.190+			1.300*	1.260+
			(1.940)	(1.770)			(2.210)	(1.870)

(continued)

Table 2. (continued)

	Poor or Fair General Health				Lead Poisoning			
Variable	Model 1	Model 2	Model 3	Model 4	Model 1	Model 2	Model 3	Model 4
Low collective efficacy: HEE component 3			1.160+	1.150			1.270*	1.270*
			(1.750)	(1.540)			(2.270)	(2.240)
Isolating ethnoracial structures								
Black concentration				1.340				1.000
				(1.170)				(–.000)
Ethnic concentration				1.130				.970
				(.690)				(–.150)
Ethnoracial diversity				1.210				1.170
				(1.510)				(.960)
n	3,333	3,333	3,333	3,333	3,333	3,333	3,333	3,333
ICC	5.030	3.930	2.770	2.770	4.320	.000	.000	.000
χ^2 (ICC)	5.190*	3.140*	1.750+	1.770+	1.320	.000	.000	.000
Log likelihood	–804	–801	–792	–791	–494	–485	–480	–479
χ^2 (model)	48.630***	54.680***	72.9***90	74.200***	42.690***	57.820***	65.620***	67.550***

Note. Odds ratios are shown, with *z* statistics in parentheses. ICC = interclass correlation coefficient (%).
+$p < .100$. *$p < .050$. **$p < .010$. ***$p < .001$.

attenuated by ethnoracial residential segregation. It appears, however, that local ethnoracial composition is not a mediator of the health effects of the dual mortgage market, as none of the concomitant measures exert an association with health that is independent of local political economies. Supplemental analysis shown in Appendix D indicates that ethnoracial residential segregation does indeed exert effects on health: Model 9 indicates that a standard deviation increase in the concentration of blacks in a neighborhood increases the odds of poor or fair general health by 74 percent, while a standard deviation increase in the concentration of Latinos and immigrants in a neighborhood increases the odds of poor or fair general health by 57 percent. Because these effects are attenuated once poor neighborhood quality measures are considered, it appears that the illness effects of local ethnoracial composition are mediated by neighborhood quality. Hypothesis 5 is not supported: the illness effects of local political economies are independent of local ethnoracial composition.

Table 3 examines whether the illness effects of the mesolevel structures of R^3p are independent of household, street block, and community factors using stepwise regression. Model 5 adds covariates for household risks and resources to Model 4 of Table 2, Model 6 further adds covariates for street block quality, and Model 7 further adds covariates for 1990 community population and economic characteristics.

Model 5 of Table 3 reveals an interesting finding: household characteristics confound the relationship between local political economies and poor or fair general health. Once comparisons are restricted to youth living in similarly affluent households (i.e., household education, household income), it appears that racialized credit refusal is detrimental to health. Living in neighborhoods where minorities are denied loans more than whites increases the odds of poor or fair general health by 39 percent. Household factors do not attenuate the health effects of local political economies; however, they do attenuate the health effects of poor neighborhood quality. They also have a strong effect in reducing racial disparities in lead poisoning. Moreover, street block quality (Model 6) does not attenuate the health effects of local political economies. Yet, Model 7 indicates that community factors in 1990 (i.e., neighborhood home values and family affluence) do attenuate the association between racialized credit refusal and poor or fair general health. Still, there is little change in the substantive association (i.e., the odds ratio). The associations between local political economies and lead poisoning are not attenuated by community factors in 1990.

CONCLUSION

In sum, I argue in this essay that racism underwrites the negative health consequences of racial residential segregation, as codified in the (in) actions of institutional gatekeepers. The preliminary analysis presented heretofore provides some support for this assertion. First, the political economy of mortgage markets is associated with levels of and changes in ethnoracial residential segregation. Second, such mesolevel markets do indeed place individuals at risk for illness. Yet, the effects of the dual mortgage market are illness specific. Third, although racial disparities in illness do not appear to be a direct function of mortgage markets, it appears that they may affect racial disparities in illness indirectly through household mechanisms, such as socioeconomic status. This analysis, however, is preliminary, as negative health effects of ethnoracial segregation were not documented to be independent of poor neighborhood quality.

Several limitations to the analysis exist. First, it is likely that the effects of the political economy of mortgage market are historically and/or developmentally contingent. Second, given the focus on the mortgage market, it is likely that other institutional (in) actions also have consequences for illness, for example, those enacted formally and informally by real estate agents and homeowners. Third, a very limited set of measures of ethnoracial residential segregation are used. Specifically, the concept of isolating ethnoracial structures is measured with two compositional measures, as done in prior studies using the PHDCN (Browning, Leventhal, and Brooks-Gunn 2004; Morenoff 2003), and a measure of diversity, where higher levels of diversity indicate less ethnoracial isolation, while lower levels of diversity indicate more ethnoracial isolation (Reardon and Firebaugh 2002; Theil 1972). However, future studies might benefit from using multilevel data (e.g., block groups nested in census tracts) to create more widely accepted measures of residential segregation that are typically measured at the metropolitan, county, or state level (Charles 2003). Still, the main purpose of this essay is to shift racial health disparities research to focus on the health consequences of institutionalized (in) actions rooted in racist ideologies that serve as a precedent to racial residential segregation. Such insight is buttressed by delineating the specific mechanisms and processes that are operative with regards to the (in)actions of one institution: the mortgage market.

R^3p indicates that institutional (in)actions that reify race heighten exposure to the biological consequences of environmentally mediated racism. In practice, it provides a theoretical toolkit to understand the co-constitutive "racialization of space and organizations"

Table 3. Random Intercept Logistic Regression Predicting the Impact of Racial Group Membership, Racist Relational Structures, Isolating Ethnoracial Structures, and Harmful Ecological Environments on Illness Experience, Holding All Else Constant.

	Poor or Fair General Health			Lead Poisoning		
Variable	Model 5	Model 6	Model 7	Model 5	Model 6	Model 7
Racial group membership						
Black	1.460	1.360	1.250	9.470**	9.340**	9.030**
	(.900)	(.720)	(.530)	(2.830)	(2.800)	(2.750)
Latino	1.620	1.590	1.460	4.410*	4.280+	4.360+
	(1.380)	(1.320)	(1.080)	(1.970)	(1.920)	(1.940)
Individual characteristics	Included	Included	Included	Included	Included	Included
Household characteristics	Included	Included	Included	Included	Included	Included
Street block characteristics		Included	Included		Included	Included
Prior community characteristics			Included			Included
Racist relational structures						
Neighborhood credit refusal	.920	.91v	.850	1.280	1.280	1.220
	(–.500)	(–.610)	(–.970)	(1.080)	(1.090)	(.860)
Racialized credit refusal	1.390*	1.370*	1.340+	1.350	1.340	1.440+
	(2.220)	(2.120)	(1.880)	(1.500)	(1.460)	(1.680)
Neighborhood credit privateness	.880	.850	.930	.370***	.370***	.340***
	(–.760)	(–.950)	(–.380)	(–3.910)	(–3.890)	(–4.000)
Racialized credit privateness	1.250	1.230	1.140	1.200	1.190	1.200
	(1.400)	(1.320)	(.820)	(.880)	(.840)	(.840)
Harmful ecological environments (HEE)						
Concentrated disadvantage: HEE component 1	1.250	1.230	1.110	.990	.970	.920
	(1.580)	(1.460)	(.690)	(–.050)	(–.160)	(–.440)
Broken windows: HEE component 2	1.050	1.000	1.050	1.110	1.080	.900
	(.570)	(–.030)	(.330)	(.830)	(.560)	(–.510)
Low collective efficacy: HEE component 3	1.040	.990	.980	1.160	1.120	.920
	(.450)	(–.080)	(–.160)	(1.350)	(.980)	(–.520)
Isolating ethnoracial structures						
Black concentration	1.110	1.070	1.060	.970	.970	.790
	(.450)	(.300)	(.230)	(–.090)	(–.080)	(–.630)
Ethnic concentration	1.070	1.070	1.000	.970	1.000	.870
	(.430)	(.440)	(.020)	(–.120)	(.010)	(–.540)
Ethnoracial diversity	1.110	1.090	.940	1.180	1.180	.890
	(.820)	(.710)	(–.350)	(1.020)	(1.030)	(–.550)
n	3,333	3,333	3,333	3,333	3,333	3,333
ICC	.190	.070	.000	.000	.000	.000
χ^2 (ICC)	.010	.000	.000	.000	.000	.000
Log likelihood	–749	–746	–742	–466	–464	–462
χ^2 (model)	146.480***	152.320***	158.120***	88.340***	91.000***	94.760***

Note. Odds ratios are shown, with *z* statistics in parentheses. Individual-level, household-level, and street block–level covariates are included but not shown (available in Appendix E). ICC = interclass correlation coefficient (%).

(Bonilla-Silva 2015:80) via quantitative renditions of the racialization of place (Anderson 2015). Similar to Lewis, Diamond, and Forman (2015), it does not view segregation itself as a necessary condition of obstinate racial stratification; rather, institutional (in)actions characterized by the reification of race and sedimentation of racial bias are culpable. Such (in)actions (re) produce ethnoracial segregation.

Altogether, R^3p views proximate risk factors of illness as rooted in upstream risk factors pertinent to ethnoracial residential segregation. From this perspective, the true culprits of racial health disparities are the political, economic, and sociocultural structures that instigate and maintain ethnoracial segregation. It is not the mere clustering of racially similar people that is detrimental to health, but rather it is the political, economic, and sociocultural deprivation that evolves when segregation is manifested, intentionally or unintentionally, as a tool to create barriers to the goods and services of society for racially marginalized people. Because racial segregation persists contemporaneously via kinetic mechanisms, the sedimentation of racial animus in institutional infrastructures largely reflects the embeddedness of the covert, ambiguous, and historical precedent of structured white privilege.

A parallel between R^3p and race-making situations exists (Hirschman 2004; Lewis 2003; Loveman 1999; Thompson 1975), as R^3p offers a constructivist narrative operant through the political economy for how racial distinction in the biological realities of health and death are created out of the structuralization of racism and the reification of race. Instead of arguing that racial ghettos are race-making situations (James 1994), I argue that institutional (in)actions that create the ghetto are the true culprits that "make" race. Racial oppression, according to R^3p, is reframed as the interstitial spaces connecting individual experiences of proximate risk factors to institutional manifestations of upstream structures. As a connective force that is often unobserved in empirical models, the implications of racial oppression are easily confused with the implications of biological correlates and genetic determinants of racial group membership.

By focusing on institutional (in)actions that inform the relationship between segregation and health, several contributions to sociological research are made. First, this essay contributes to research on race/ethnicity by explicating a testable theory of the health consequences of differential treatment by institutional gatekeepers of a major American institution: the mortgage lending industry. Second, this essay contributes to research on neighborhood effects by providing a root-cause interpretation of how neighborhood conditions influence racial differences in health outcomes. Third, this essay contributes to segregation studies by positing a singular reason why the segregation-health link may occur; that is, some segregated neighborhoods are undergirded by racism, whereas other segregated neighborhoods are not. Moreover, this essay provides a policy-conscious model for reducing racial health disparities that works through institutional change.

Appendix A. Descriptive Statistics for Unstandardized Neighborhood-Level Measures (n = 273).

Variable	*M*	*SD*	Minimum	Maximum	Source
Racist relational structures					
Neighborhood credit refusal	.150	.090	.030	.460	1994 HMDA
Racialized credit refusal	1.010	.550	.000	5.480	1994 HMDA
Neighborhood credit privateness	.540	.130	.250	.930	1994 HMDA
Racialized credit privateness	.950	.140	.310	1.430	1994 HMDA
Isolating ethnoracial structures					
Black concentration	.420	.420	.0000	1.000	2000 NCDB
Ethnic concentration	1.860	2.470	.000	10.920	2000 NCDB
Ethnoracial diversity	.600	.390	.020	1.360	2000 NCDB
Harmful ecological environments (HEE)					
Concentrated disadvantage: HEE component 1	.000	1.000	–3.110	2.170	See Appendix C
Broken windows: HEE component 2	.000	1.000	–3.100	2.720	See Appendix C
Low collective efficacy: HEE component 3	.000	1.000	–2.930	2.870	See Appendix C
Prior community characteristics					
Family affluence rate, 1990	.210	.130	.020	.720	1990 NCDB
Homeownership rate, 1990	.390	.230	.010	.920	1990 NCDB
Median home values (in tens of thousands), 1990	90.250	64.220	.000	396.950	1990 NCDB
Residential mobility, 1990	.440	.120	.180	.730	1990 NCDB

Note. HMDA = Home Mortgage Disclosure Act; NCDB = Neighborhood Change Database.

Appendix B. Descriptive Statistics for Illness, Racial Group Membership, and Individual-, Household-, and Street Block–Level Controls.

Variable	*M*	*SD*	Minimum	Maximum	*n*
Illness					
Poor or fair general health	.070		0	1	3,333
Lead poisoning	.040		0	1	3,333
Racial group membership					
Black (*n* = 1,153)	.350		0	1	3,333
Latino (*n* = 1,614)	.480		0	1	3,333
White (*n* = 566)	.170		0	1	3,333
Individual characteristics					
Age at time of interview (centered)	.000	4.970	–8.390	9.020	3,333
Age at time of interview (uncentered)	8.980	4.970	.590	18	3,333
Female	.510	.5	0	1	3,333
Loss of insurance coverage	.200	.4	0	1	3,333
Household characteristics					
Household education					
W1 less than high school (reference)	.150	.360	0	1	2,682
W1 high school without completion	.210	.410	0	1	2,682
W1 high school degree (or equivalent)	.170	.370	0	1	2,682
W1 some more than high school	.340	.470	0	1	2,682
W1 bachelor's degree or more	.130	.340	0	1	2,682
W1 natural log of family income	2.220	.980	0	3.990	2,682
W1 lives in owned home	.400	.490	0	1	2,682
Residential mobility	.330	.470	0	1	2,682
HH family structure					
Biological 2-parent HH (reference)	.500	.5	0	1	2,682
Nonbiological 2-parent HH	.190	.390	0	1	2,682
Single-parent HH	.220	.420	0	1	2,682
Three-generation HH	.080	.280	0	1	2,682
W1 PC perceived racism	–.020	1	–1.170	2.940	2,682
Research design					
Biological Mom PC	.870	.330	0	1	2,682
Same PC in W1 and W2	.950	.230	0	1	2,682
Multisubject HH	.410	.490	0	1	2,682
Street block characteristics					
Traffic hazards	.040	.980	–2.160	2.810	2,682
Ambient hazards scale	.050	1	–1.330	3.810	2,682
Building security visible (ordinal)	.940	.820	0	3	2,682
None have security	.320	.470	0	1	2,682
Some have security	.470	.5	0	1	2,682
At least half have security	.160	.360	0	1	2,682
Most have security	.050	.220	0	1	2,682
Children playing in street (ordinal)	.590	.770	0	2	2,682
No children in street	.590	.490	0	1	2,682
1 or 2 children in street	.240	.420	0	1	2,682
≥3 children in street	.180	.380	0	1	2,682
People observed on street	.700	.460	0	1	2,682
Persons observed: none hostile	.670	.470	0	1	2,682
Persons observed: 1 or 2 hostile	.020	.160	0	1	2,682
Persons observed: ≥3 hostile	.010	.080	0	1	2,682
No people observed on street	.300	.460	0	1	2,682

Note. HH = household; PC = primary caregiver; W1 = wave 1 (1994–1997); W2 = wave 2 (1997–2000).

Appendix C. Descriptive Statistics and Rotated Factor Loadings for Harmful Ecological Environment Components (n = 273).

Variable	*M*	*SD*	Minimum	Maximum	Principal Components 1	2	3	Unique Variance	Source
Neighborhood violence exposure scale, 1995	–2.500	.310	–3.170	–1.510	**.500**	**.680**	–.040	.290	PHDCN CS
Neighborhood decline perceptions scale, 1995	1.970	.170	1.460	2.490	**.760**	–.150	.160	.370	PHDCN CS
Proportion of homes built before 1940, 2000	.440	.170	.030	.810	–.040	**.850**	.090	.260	2000 NCDB
Proportion of homes abandoned, 2000	.850	.160	.000	1.000	–.070	**.550**	.460	.480	2000 NCDB
Absence of social cohesion scale, 1995	–3.340	.260	–3.960	–2.710	.410	.380	**.610**	.310	PHDCN CS
Absence of neighborhood activism scale, 1995	1.930	.470	.400	3.310	.100	–.010	**.890**	.190	PHDCN CS
Proportion of children <5 years old, 2000	.080	.020	.020	.130	**.810**	.200	.230	.260	2000 NCDB
Crowding rate: persons per occupied homes, 2000	2.970	.640	1.400	4.290	**.880**	.070	.030	.220	2000 NCDB

Note. NCDB = Neighborhood Change Database; PHDCN CS = Project on Human Development in Chicago Neighborhoods Community Survey.

Appendix D. Random Intercept Logistic Regression Predicting the Impact of Racial Group Membership, Harmful Ecological Environments, and Isolating Ethnoracial Structures on Illness Experience, Holding Individual Factors Constant.

	Poor or Fair General Health		Lead Poisoning	
Variable	Model 8	Model 9	Model 8	Model 9
Racial group membership				
Black	1.580	1.420	13.480***	17.740***
	(1.340)	(.880)	(3.540)	(3.650)
Latino	2.940**	3.000***	6.330*	8.310**
	(3.280)	(3.300)	(2.490)	(2.780)
Individual characteristics				
Age at time of interview (centered)	1.030+	1.030+	.910***	.910***
	(1.890)	(1.820)	(–4.350)	(–4.430)
Female	.870	.890	.730+	.740
	(–1.010)	(–.870)	(–1.660)	(–1.620)
Uninsurance spell	1.350+	1.380*	.940	.980
	(1.890)	(2.030)	(–.270)	(–.080)
Harmful ecological environments (HEE)				
Concentrated disadvantage: HEE component 1	1.430**		1.180	
	(3.290)		(1.390)	
Broken windows: HEE component 2	1.190+		1.210+	
	(1.940)		(1.660)	
Low collective efficacy: HEE component 3	1.160+		1.290*	
	(1.720)		(2.270)	
Isolating ethnoracial structures				
Black concentration		1.740*		1.150
		(2.530)		(.500)
Ethnic concentration		1.570***		1.200
		(3.360)		(.980)
Ethnoracial diversity		1.260+		1.150
		(1.800)		(.870)
n	3,333	3,333	3,333	3,333
ICC	3.620	4.210	2.010	4.050
χ^2 (ICC)	2.950*	3.910*	.300	1.160
Log likelihood	–794	–797	–489	–493
χ^2 (model)	67.470***	61.430***	51.380***	44.330***

Note. Odds ratios shown, with *z* statistics in parentheses. ICC = interclass correlation coefficient (%).
+$p < .100$. *$p < .050$. **$p < .010$. ***$p < .001$.

Appendix E. Random Intercept Logistic Regression Predicting the Impact of Racial Group Membership, Racist Relational Structures, Isolating Ethnoracial Structures, and Harmful Ecological Environments on Illness Experience, Holding All Else Constant.

	Poor or Fair General Health			Lead Poisoning		
Variable	Model 5	Model 6	Model 7	Model 5	Model 6	Model 7
Racial group membership						
Black	1.460	1.360	1.250	9.470**	9.340**	9.030**
	(.900)	(.720)	(.530)	(2.830)	(2.800)	(2.750)
Latino	1.620	1.590	1.460	4.410*	4.280+	4.360+
	(1.380)	(1.320)	(1.080)	(1.970)	(1.920)	(1.940)
Individual characteristics						
Age at time of interview (centered)	1.020	1.010	1.010	.920***	.920***	.920***
	(1.060)	(.970)	(.930)	(–3.830)	(–3.820)	(–3.850)
Female	.900	.890	.910	.700+	.700+	.700+
	(–.760)	(–.790)	(–.690)	(–1.860)	(–1.900)	(–1.840)
Uninsurance spell	1.250	1.260	1.260	.910	.910	.910
	(1.380)	(1.410)	(1.410)	(–.400)	(–.380)	(–.380)
Household characteristics						
W1 high school without completion	.720+	.720+	.720+	1.510	1.500	1.510
	(–1.780)	(–1.810)	(–1.800)	(1.240)	(1.210)	(1.230)
W1 high school degree (or equivalent)	.340***	.350***	.360***	1.280	1.280	1.310
	(–4.410)	(–4.360)	(–4.270)	(.690)	(.690)	(.750)
W1 some more than high school	.230***	.230***	.240***	.990	1.020	1.020
	(–6.130)	(–6.030)	(–5.980)	(–.030)	(.050)	(.070)
W1 bachelor's degree or more	.110***	.110***	.120***	.360	.370	.380
	(–4.490)	(–4.420)	(–4.290)	(–1.510)	(–1.470)	(–1.430)
W1 natural log of family income	.890	.890	.900	.800*	.810*	.810*
	(–1.370)	(–1.290)	(–1.230)	(–2.080)	(–2.000)	(–1.980)
W1 lives in owned home	.830	.850	.850	.750	.750	.740
	(–1.080)	(–.910)	(–.950)	(–1.240)	(–1.200)	(–1.260)
Residential mobility	.990	1.000	1.000	1.240	1.260	1.210
	(–.080)	(–.000)	(.000)	(1.070)	(1.120)	(.940)
Nonbiological 2-parent HH	.770	.760	.750	1.350	1.310	1.310
	(–1.310)	(–1.400)	(–1.420)	(1.150)	(1.030)	(1.040)

(continued)

Appendix E. (continued)

Variable	Poor or Fair General Health			Lead Poisoning		
	Model 5	Model 6	Model 7	Model 5	Model 6	Model 7
Single-parent HH	.680+	.650+	.660+	.720	.700	.700
	(–1.740)	(–1.920)	(–1.870)	(–1.110)	(–1.190)	(–1.170)
Three-generation HH	.440*	.430*	.440*	.970	.960	.980
	(–2.190)	(–2.210)	(–2.180)	(–.080)	(–.130)	(–.060)
W1 PC perceived racism	1.160*	1.170*	1.160*	1.010	1.010	1.020
	(2.060)	(2.200)	(2.110)	(.070)	(.130)	(.190)
Biological mom PC	.750	.740	.730	.870	.870	.860
	(–1.310)	(–1.400)	(–1.450)	(–.460)	(–.450)	(–.520)
Same PC in W1 and W2	1.260	1.280	1.330	1.080	1.080	1.060
	(.680)	(.720)	(.830)	(.190)	(.170)	(.130)
Multisubject HH	.940	.930	.930	.950	.930	.940
	(–.440)	(–.520)	(–.490)	(–.280)	(–.380)	(–.320)
Street block characteristics						
Traffic hazards		1.120	1.110		.920	.910
		(1.510)	(1.420)		(–.840)	(–.960)
Ambient hazards		1.010	1.010		1.040	1.040
		(.060)	(.090)		(.370)	(.360)
Building security visible (ordinal)		1.090	1.080		.980	.980
		(.940)	(.890)		(–.170)	(–.120)
Children play in public (ordinal)		1.100	1.070		1.040	1.030
		(.930)	(.730)		(.350)	(.230)
People observed on street		1.280	1.310		1.390	1.350
		(1.280)	(1.400)		(1.230)	(1.120)
Prior community characteristics						
Family affluence rate, 1990			.670+			.780
			(–1.690)			(–.780)
Homeownership rate, 1990			1.650*			.970
			(2.040)			(–.110)
Median home values, 1990			1.010			.960
			(.070)			(–.210)
Residential mobility, 1990			1.420*			1.360
			(2.230)			(1.480)

(continued)

Appendix E. (continued)

Variable	Poor or Fair General Health			Lead Poisoning		
	Model 5	Model 6	Model 7	Model 5	Model 6	Model 7
Racist relational structures						
Neighborhood credit refusal	.920	.910	.850	1.280	1.280	1.220
	(–.500)	(–.610)	(–.970)	(1.080)	(1.090)	(.860)
Racialized credit refusal	1.390*	1.370*	1.340+	1.350	1.340	1.440+
	(2.220)	(2.120)	(1.880)	(1.500)	(1.460)	(1.680)
Neighborhood credit privateness	.880	.850	.930	.370***	.370***	.340***
	(–.760)	(–.950)	(–.380)	(–3.910)	(–3.890)	(–4.000)
Racialized credit privateness	1.250	1.230	1.140	1.200	1.190	1.200
	(1.400)	(1.320)	(.820)	(.880)	(.840)	(.840)
Harmful ecological environments (HEE)						
Concentrated disadvantage: HEE component 1	1.250	1.230	1.110	.990	.970	.920
	(1.580)	(1.460)	(.690)	(–.050)	(–.160)	(–.440)
Broken windows: HEE component 2	1.050	1.000	1.050	1.110	1.080	.900
	(.570)	(–.030)	(.330)	(.830)	(.560)	(–.510)
Low collective efficacy: HEE component 3	1.040	.990	.980	1.160	1.120	.920
	(.450)	(–.080)	(–.160)	(1.350)	(.980)	(–.520)
Isolating ethnoracial structures						
Black concentration	1.110	1.070	1.060	.970	.970	.790
	(.450)	(.300)	(.230)	(–.090)	(–.080)	(–.630)
Ethnic concentration	1.070	1.070	1.000	.970	1.000	.870
	(.430)	(.440)	(.020)	(–.120)	(.010)	(–.540)
Ethnoracial diversity	1.110	1.090	.940	1.180	1.180	.890
	(.820)	(.710)	(–.350)	(1.020)	(1.030)	(–.550)
n	3,333	3,333	3,333	3,333	3,333	3,333
ICC	.190	.070	.000	.000	.000	.000
χ^2 (ICC)	.010	.000	.000	.000	.000	.000
Log likelihood	–749	–746	–742	–466	–464	–462
χ^2 (model)	146.480***	152.320***	158.120***	88.340***	91.000***	94.760***

Note. Odds ratios are shown, with *z* statistics in parentheses. HH = household; ICC = interclass correlation coefficient (%); PC = primary caregiver; W1 = wave 1 (1994–1997); W2 = wave 2 (1997–2000).

NOTE

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AUTHOR BIOGRAPHY

Abigail A. Sewell is an Assistant Professor at Emory University in the Department of Sociology and a Vice Provost's Postdoctoral Fellow at the University of Pennsylvania in the Population Studies Center. She focuses on the political economy of racial health disparities, the social psychology of health care disparities, and quantitative approaches to studying racial inequality and supraindividual racism. She received her Ph.D. and M.A. in Sociology from Indiana University and her B.A. summa cum laude in Sociology (Minor in Women's Studies) from the University of Florida.