Effects of Prolonged and Timing-Specific Exposure to Neighborhood Disadvantage during Childhood and Adolescence on Health and Health Inequalities in Early Adulthood

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Abstract

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Mounting evidence that purely individual-based explanations fail to fully explain persistent disparities in morbidity and mortality has contributed to a surge in research investigating how residential contexts further shape the health of individual residents. Until recently, however, the majority of scholarship in this area measured neighborhood characteristics only once or over just a short window of observation, conflating persons who were recently exposed with those who have – and in the case of many communities of color, are more likely to have – experienced repeated or prolonged residential adversity. Such a conceptualization is inconsistent with most theories of neighborhood effects, which tend to specify mechanisms that imply sustained or age-specific exposures, as well as with a developmental or life course perspective in which experiences earlier in life are posited to have formative and enduring impacts on future outcomes, even when controlling for more contemporaneous determinants. Using data from the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID) merged with census data on respondents' neighborhoods, this dissertation comprises three interrelated studies examining the effects of prolonged and timing-specific exposures to neighborhood disadvantage throughout childhood and adolescence on health and health inequalities later in life, including self-rated health status, obesity incidence, and early smoking initiation. Neighborhood

disadvantage is characterized regularly throughout the child and adolescent life course using a composite index based on the spatial clustering of poverty, unemployment, female-headed households, public assistance receipt, and educational and occupational marginalization.[1] The assumption is that such collective conditions of neighborhoods are concomitant to social and structural resources and opportunities (or the lack thereof) that can, alone or in conjunction with individual- and household-level risk and protective factors, be health promoting or health compromising. Analyses employ marginal structural models with inverse probability of treatment (and censoring) weights to adjust for selection bias without "controlling away" the effects of neighborhood exposures that operate indirectly though the same individual-level covariates that are associated with mobility into and out of different neighborhood contexts. Results indicate that prolonged exposure to neighborhood disadvantage throughout childhood and adolescence is strikingly more common among nonwhite, predominantly African American, respondents and is associated in turn with significantly greater odds of experiencing worse self-rated health as well as obesity in early adulthood. Moreover, contrary to family-level poverty in which experiences during early childhood have been shown to be particularly influential, these analyses suggest that exposure to neighborhood-level deprivation during adolescence may be more detrimental to young adult health than exposure that occurs earlier in life. Nonetheless, the findings for early smoking initiation highlight the potential for differential impacts of neighborhood poverty across different racial/ethnic groups. Overall, however, this dissertation adds support to the growing body of literature suggesting that place-based, developmentally-appropriate, and ongoing investments in the social, economic, institutional, and physical structures of under-resourced communities and communities of color can have long-term benefits for population health and health equity that extend over the life course.

[1] For the early smoking initiation outcome, only the poverty rate of the census tract is used to characterize neighborhood disadvantage.

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Chapter 1. INTRODUCTION

"What is truly American is not so much the individual but neighborhood inequality."

- Robert J. Sampson, *Great American City* (2012)

1.1 HEALTH IS LOCAL

The University of Washington is located in King County, Washington in which life expectancy at birth is 82 years, three years more than the national average (79 years) and only a few months less than the average in the ten longest-lived countries in the world (83 years) [1]. On some of the most common indicators of chronic disease, health behavior, and socioeconomic determinants of morbidity and mortality, King County also ranks among the healthiest in the state, the nation, and the world [2, 3]. In this light, King County looks like an ideal place for health and wellness. When viewed through a more discerning lens, however, these overall averages mask dramatic within-county inequalities. For instance, average life expectancy ranges from 74 years in the bottom ten percent of King County census tracts to 87 years in the top ten percent (Figure 1.1) [2]. This means that a child born today in one of the worst performing census tracts in King County is only expected to live, on average, as long as someone who was born over 60 years ago in one of the world's ten longest-lived countries. On the other end of the spectrum but often only a short drive or a modest bicycle ride away, King County's best performing census tracts boast an average life expectancy that is unlikely to be achieved in the ten longest-lived countries for another 40 years [4]. As former Seattle-King County Director of Public Health, Dr. David Fleming, recently remarked in testimony prepared for the Robert Wood Johnson Foundation Commission to Build a Healthier America:

> *"In fact, there is substantially more variation by census tract within King County than there is by county within the U.S. At the pace of best performing nations, it would take 100 years for the census tract with the lowest life expectancy in King County to catch up to where the highest ones already are today"* [4].

Such small area inequalities in life expectancy, as well as other indicators of health, are not unique to King County, but have been documented within regions across the United States (U.S.) – from San Joaquin Valley, California and the District of Columbia to New Orleans, Louisiana, in which just a few miles can mean a 25-year difference in longevity (Figure 1.2) [5]. Health is thus geographically patterned, and local (more often than

county or state) jurisdictions have become an increasingly crucial dimension across which this patterning occurs. But it is not just health. When rates of what are generally considered social determinants of poor health, including poverty, joblessness, environmental hazards and the like, are mapped, it turns out that these things tend to cluster in specific and highly local neighborhoods as well. Overlay these facets of concentrated disadvantage on a map of poor health (virtually any way you measure it), and they overlap almost exactly. The nature and nuances of this association between the geography of disadvantage and the geography of poor health have motivated a substantial body of so-called neighborhood effects research. This dissertation focuses in particular on the temporal dimensions of neighborhood-health effects, examining how the duration and timing of exposure to neighborhood disadvantage throughout the child and adolescent life course affect health outcomes later in life. This first chapter offers a broad introduction to some of the key themes in the neighborhood effects literature, including ongoing tensions, current theorizing, and extant evidence, as well as a brief overview of the three empirical questions around which the remainder of this dissertation is structured.

1.2 People and Places

> *"Economics is the study of how people make choices, whereas sociology studies how people often have no choices to make at all."*
>
> - James Duesenberry (1960)

Despite growing consensus among social scientists and the public at large that where someone lives matters for their health, this association is theoretically and methodologically complex. Two broad and interrelated tensions, in particular, persist in the literature and continue to influence the extent to which health promotion interventions are directed largely at the characteristics of people as opposed to the characteristics of places [6].

1.2.1 Selection Bias

The first issue relates to selection bias. It is often noted that among the biggest thorns in the side of the neighborhood effects literature – and often the first concern to be raised when such research is presented to academic and non-academic audiences alike – is the role of residential preference in accounting for small area differences in health (or any number of other outcomes from crime and delinquency to education and economic self-sufficiency) [7]. In the simplest of terms, people are not randomly allocated to reside in different types of neighborhoods. They make choices about where to live – whether to move or stay – according to their resources, preferences, and changing life circumstances. For example, lower-income individuals may decide

to live in more disadvantaged neighborhoods because housing is cheaper and more abundant, perhaps their friends or members of their extended family already live there, or any number of other personal inclinations and beliefs associated with particular places. If these same people also tend to be in worse health, such individually-driven choices about where to live no doubt have a hand in producing the geographical patterning of health described above (more on this in the next section).

That said, it is also clear that the choices people make – in fact, the set of choices that are available to be made – are fundamentally shaped not only by who they are, but also by broader contextual phenomena. Prior to the 1970s, legally sanctioned discrimination in housing policies and practices, including restrictive covenants, redlining, and the placement of public housing, ensured that certain neighborhoods were off-limits to most members of nonwhite and immigrant groups [8, 9]. Today, implicit biases and in-group favoritism (as opposed to the more overt forms of out-group hostility that were institutionalized prior to the civil rights laws of the 1960s and 1970s) [10] continue to constrain the residential "choices" of minority group members via documented racial-spatial inequalities in interest rates, subprime loans, and foreclosures [11, 12]. Moreover, neighborhoods are not static in character (although, on the whole, they tend to maintain their relative position in the spatial hierarchy [13, 14]). A family may select into a particular neighborhood because the schools are better resourced or the streets are better maintained, but over time these conditions on which a neighborhood was chosen may change due to the mobility decisions of *other* residents, as in the case of white or middle-class flight from racially integrating and/or inner-city neighborhoods [15, 16]. Such instances in which the neighborhood an individual "selects" is either not a formal option or becomes something other than what was initially selected, illustrate the inherent limitations of explanations for the geographical patterning of health that are based solely on individually-driven preferences (or "choice") [6, 14].

1.2.2 Composition or Context

The second tension stems from the first. Taking seriously the notion that residential choice plays at least some (although by no means the only nor a completely autonomous) role in the geographical patterning of health nonetheless raises concerns about the extent to which health is affected by the characteristics of people ("composition") versus the characteristics of places ("context"). Impoverished areas tend to house more impoverished people, and vice versa in the case of more affluent areas. Moreover, an abundance of research documents a socioeconomic gradient in health such that individuals of progressively higher education, occupation, and income tend to live increasingly longer, healthier, and happier lives [17-20]. Does residing in

a particular type of neighborhood, then, exert an effect on one's health that is independent of the well-known consequences of simply being a richer or poorer individual? Questions such as this have directed the majority of research on neighborhood-health effects over the last quarter century, facilitated in no small part by the advent of multilevel modelling techniques. In general, these studies aim to establish both whether there is any explanatory role for place (usually place of residence) after taking the characteristics of individual residents into account, and how much of the observed geographical variation in health can be explained by different types of places [21].

Findings from this work have been mixed. In the early to mid-1990s, several studies – most of which relied on British datasets – concluded that place mattered little for health over and above individual, or compositional, factors. For example, it was suggested that the major determinants of geographical variation in smoking and drinking behavior [22] and psychiatric morbidity [23] in the United Kingdom were individual-level factors, while the role for contextual effects was limited. An examination of the relationship between social deprivation in electoral wards and premature death in England and Wales reached a similar conclusion such that excess mortality associated with residence in areas designated as deprived was said to be "wholly explained by the concentration in those areas of people with adverse personal or household socioeconomic factors" [24]. By the turn of the century, however, most research using larger samples and a wider array of geographical areas tended to show contextual effects on various health outcomes over and above the influence of individual-level characteristics [25, 26]. Nevertheless, the magnitude of these contextual effects on health has often been modest in comparison to compositional effects, leading some scholars to maintain that where someone lives may matter for their health, but who they are matters a whole lot more [27]. It has also been argued, however, that such a qualified (and ultimately, individually-oriented) conclusion may stem from the tendency in many studies to assume, at least implicitly, that compositional and contextual effects have competing rather than spatially and temporally interrelated influences [6, 21, 28].

1.3 Either/Or Versus Both/And

In estimating neighborhood effects on health, the two interrelated tensions described above are often viewed as statistical problems to be controlled away and not as phenomena to be confronted explicitly [13]. To summarize, these issues stem from the basic notion that the demographic and socioeconomic characteristics of individuals influence their residential choices as well as their health. Thus the most common approach in

the neighborhood effects literature has been to address concerns about both selection and the "competing" impact of compositional as opposed to contextual characteristics by estimating models that simply control for individual-level variables. In this way, the roles of residential choice and compositional characteristics in helping to shape health are construed as if they are unrelated to the effects of neighborhoods on health. Such an either/or conceptualization may at first seem advantageous for allocating resources and directing health practice and policy efforts – in other words, for determining whether to focus on people or the places in which they live. However, as Kawachi and Berkman [6] assert, "very likely the correct answer is: both [people and places]".

By most accounts, the relationship between individual characteristics and residential choice is reciprocal. That is, residential mobility decisions are a function of individual demographic and socioeconomic factors, particularly race, income, education, and home ownership [13], but these individual-level factors are also affected by the neighborhoods in which people "choose" to live. For example, a considerable body of prior scholarship argues that living in a neighborhood characterized by concentrated poverty and endemic joblessness will isolate individuals from networks of institutions and employed people, making it more difficult for them to obtain information about or make connections that lead to job and other income-generating opportunities [29-32], which may in turn affect mobility decisions (as well as health). Moreover, as alluded to briefly above, changes in the racial composition of a neighborhood have been shown to differentially impact residential preference and individual moving behaviors: white and, to a lesser extent, Latino residents are more likely to move from neighborhoods where African Americans and Latinos have a growing presence [33], which serves to further concentrate advantage and power derived from a racialized social hierarchy (and associated opportunities for residential mobility and health promotion) within predominantly white neighborhoods [8, 9]. Simply controlling for individual-level variables in an attempt to isolate the effects of neighborhoods on health therefore distorts the reality that individual demographic and socioeconomic factors are in fact intervening variables on the pathways between neighborhoods and health, not merely "confounders" as they are so often construed [21, 34]. Such statistical over-adjustment for the indirect ways through which neighborhood effects transpire likely contributes to the mixed results and relatively modest contextual effect estimates often seen in prior research.

1.4 WHY PLACE MATTERS

One way in which scholars have attempted to move beyond the artificial people or places dichotomy is through a more explicit theoretical and empirical focus on the mechanisms linking neighborhoods and health – in other words, explanations for *why* neighborhoods might matter that incorporate both people and places. Sampson and colleagues [7] refer to this as the "process turn" in the broader neighborhood effects literature. Accordingly, insights from various disciplines have been adapted and combined to reveal a fairly consistent and widely accepted set of social and structural processes (or mechanisms) [7, 35-38] that stem from the collective conditions of places to affect individual outcomes, including the more proximal behavioral and biological precursors of disability, disease, and death. These "processes of place" can be very broadly classified under four interrelated rubrics: those based on (1) social isolation, (2) social disorganization, (3) institutional resources, and (4) the physical environment.

1.4.1 Social Isolation

The social isolation concept was broadly introduced in Wilson's seminal book, *The Truly Disadvantaged*, in which he contends that macroeconomic changes related to the 1970s-era deindustrialization of central cities resulted in the social isolation of low-income African American residents from middle- and upper-income families whose presence in the same urban neighborhoods had previously served to validate mainstream sources of influence as well as helped to attract and sustain the basic institutions of the area (e.g., schools, childcare centers, churches, credit unions, grocery stores, and recreational centers) [31, 39]. According to Wilson, the shift from a manufacturing to a service economy drastically reduced the number of steady, working-class jobs on which a substantial share of African American families had relied since the Great Migration of southern blacks to northern cities. Poverty, joblessness, and an array of associated social problems increased dramatically as a result of these changes, concentrating disadvantage not only in space but also, because of racial residential segregation, within predominately African American areas [8]. Middle- and upper-income black families, however, were better able to leverage recent civil rights gains to move out of the deteriorating central cities to which African Americans of virtually all income levels had been historically relegated [40]. Although the economic stability and locational attainment of these middle- and upper-income groups was and continues to be far more tenuous than that of their white counterparts [41-43], the more general notion that residents of disadvantaged, predominantly nonwhite neighborhoods tend to be isolated from social groups, organizations, and institutions that connect people to the labor market and other advantaged (or salutary)

resources remains a prominent mechanism through which neighborhood effects are posited to transpire, especially when it comes to the collective socialization of children and youth.

Whereas conventional conceptualizations of social isolation assume that young people in disadvantaged neighborhoods are exposed and subsequently subscribe to distinct worldviews (what some have called "ghetto-specific" cultural repertoires) that are qualitatively different from those advanced in middle- and upper-income neighborhoods, there is strong empirical support for considerable heterogeneity within disadvantaged areas, including both mainstream and alternative ideals regarding work, education, marriage, and childrearing [44-47]. Nonetheless, Harding [48] argues that the socialization of children and youth in disadvantaged neighborhoods is disproportionately likely to include formative interactions with older peers who tend to be unemployed, not in school, and involved in the underground economy (and therefore more likely to be available and visible in the neighborhood). These individuals are largely absent from or make up a relatively small minority of the residents in more advantaged neighborhoods where socialization typically involves adult (rather than older peer) role models who have achieved a degree of social and economic stability through mainstream channels. In disadvantaged neighborhoods, therefore, health compromising norms and behaviors associated with establishing and maintaining a tough or street-wise persona (e.g., school dropout, crime, violence, drug and alcohol abuse, and risky sexual activity) may not only be particularly salient and respected, but also a necessary adaptation to lived experience. In addition, research on chronic stress suggests that the profound distrust of others, perpetually heightened sense of vigilance, and predominant focus on the here and now that often comes with such a street-wise orientation may also have a more direct adverse impact on various physiological systems that influence susceptibility to and the progression of disease as well as on aging and allostatic load [49-51].

1.4.2 Social Disorganization

The social disorganization concept was originally defined as "the inability of a community to realize the common values of its residents and maintain effective social controls" [52]. Its conceptual emphasis on the importance of strong friendship, kinship, and acquaintanceship networks facilitated its alignment with (perhaps even its reinvention as) social capital theory. That is, neighborhoods lacking social capital, typically construed as a resource activated through dense social ties among residents, were said to be less able to realize common values and maintain the informal social controls that promote positive development, health, and well-being [53]. Over time, however, various scholars challenged the notions that disadvantaged neighborhoods are

inherently disorganized, that thick ties among neighbors are always prosocial, and that successful social control requires strong network connections. Building on the social isolation paradigm, Wilson [29] argues, for example, that although residents of disadvantaged neighborhoods may be tightly interconnected, such ties are extremely insular and detached from the networks that actually connect people to jobs and other health promoting resources. Pattillo [54] and others [55-57] further document how dense social ties can foster the growth of drug- and gang-related networks that, although they may provide an unconventional form of organization and social control in disadvantaged neighborhoods, they may simultaneously inhibit efforts to address crime and other health compromising norms and behaviors. Furthermore, and perhaps most critically, research expanding on Granovetter's [58] "weak ties" thesis has shown that thick ties among neighbors may not only be unnecessary for fostering effective social control, but also unrealistic in contemporary neighborhoods in which there are simply too many people to know (or to want to know) intimately [13].

In recognition of such critiques, Sampson and colleagues [13, 59, 60] have since advanced an adapted theory of collective efficacy. It accepts the basic ideas of the conventional social disorganization paradigm in that social control remains both a shared characteristic of places (not people) and a crucial source of variation across neighborhoods in individual outcomes. However, it relaxes the need for close-knit relationships: "repeated interactions, observations of interactions, and an awareness of *potential* interactions that could be invoked all establish shared norms (a sense of 'we') beyond the strong ties among friends and kin" [13]. Differences in collective efficacy – that is, differences in mutual trust as well as shared expectations and capacities for prosocial action – are most often associated with variations across neighborhoods in rates of crime and violence as residents in areas with low levels of collective efficacy are less likely to feel empowered to intervene and to enforce salutary norms and behaviors [60]. Violence, in particular, not only threatens the health and well-being of those individuals directly involved, but it may also act as a stressor for other residents who witness such incidents or who live in fear of violent victimization. For example, prior research has linked lower levels of collective efficacy and social control in disadvantaged neighborhoods to perceptions of danger and subsequent reductions in outdoor play and other forms of both recreational and non-recreational physical activity, as well as with related increases in overweight/obesity and other stress-related chronic conditions such as depression, asthma, and cardiovascular disease [61, 62]. Moreover, various studies have found a more general regulatory effect of collective efficacy on health compromising behaviors, including illicit substance use, tobacco and alcohol (mis)use, child and elder neglect and abuse, intimate partner violence,

and early sexual activity [63, 64]. At the institutional level, collective efficacy may also influence residents' willingness and capacity to mobilize and extract resources, for example, to protest the closure of a local hospital or clinic, to advocate for passage of a local ordinance to restrict smoking in public places, or to use zoning restrictions to prevent the incursion of toxic waste, fast food, or alcohol outlets in their neighborhood.

1.4.3 Institutional Resources

Neighborhood resource theories contend that disadvantaged neighborhoods lack important institutional assets, such as quality schools, daycare centers, grocery stores, pharmacies, adequate police and fire protection, sanitation services, and recreational opportunities, which can negatively affect health [36, 65]. High-quality, accessible, and culturally-sensitive medical institutions are an obvious determinant of health outcomes; however, persistent challenges to care exist for residents of disadvantaged neighborhoods and neighborhoods of color, including transportation difficulties, insensitive or culturally inappropriate treatment, long waiting room times, inadequately stocked pharmacies, and a lack of multilingual staff [65, 66]. Prior research further documents that supermarkets have sharply declined in low-income, predominantly nonwhite neighborhoods, forcing many residents (given additional constraints on transportation) to rely on convenience and other small stores with more limited, calorie-dense, and often higher priced food selections [67, 68]. Disadvantaged neighborhoods are also more likely to include a disproportionate share of fast food and alcohol outlets and associated advertising, which have been associated with poor diet, excessive alcohol consumption, and related harms, including chronic disease and intentional and unintentional injuries [69, 70]. Moreover, many high-poverty and racially segregated neighborhoods do not have enough fire stations or police walking the streets, which can lead not only to more fires and crime, but may also make residents more reluctant to venture outside for exercise and more susceptible to stress-related conditions such as heart disease, high blood pressure, and infant mortality [65]. Finally, the quality of educational institutions may be directly linked to neighborhood socioeconomic conditions because public school funding is often geographically determined. Thus the schools in disadvantaged neighborhoods tend to have more dilapidated buildings, fewer qualified teachers, more limited curricula, little serious academic counseling, fewer connections with colleges and employers, and higher levels of teen pregnancy, which may lead in turn to peer pressure against academic achievement and in support of crime, substance use, and other health compromising norms and behaviors [71]. The availability of extracurricular activities and services associated with school quality, such as daycare centers and recreational programs, may also delay or prevent the initiation of health compromising behaviors among children and youth.

1.4.4 Physical Environment

Lastly, environmental theories of neighborhood effects focus on the physical condition, or "built environment", of disadvantaged neighborhoods, including streets, housing, businesses, schools, parks, and patterns of regional design, growth, and change [36, 72]. Research has shown that high-poverty neighborhoods and neighborhoods of color are disproportionately located near major freeways, ports, and other industrial sources of diesel and air pollution [73], which may trigger asthma attacks or other respiratory problems, especially among children and other vulnerable populations. Disadvantaged neighborhoods are also more likely to contain dilapidated housing, which can affect health through exposure to indoor allergens (e.g., dust and mold), toxins (e.g., lead paint), and structural hazards (e.g., dilapidated housing or abandoned buildings), leading in turn to greater risk of disease, developmental impairments, and injuries, as well as higher rates of school or work absences [65]. Substandard schools and workplaces may also constitute similar health hazards. Moreover, the presence (or absence) of sidewalks and safe, well-maintained streets and parks with adequate lighting can influence health via opportunities for health-promoting activities such as exercise and social interaction. Finally, patterns of suburban sprawl and systematic disinvestment in central cities play a critical role in health and health inequalities by not only limiting access to economic opportunity, but also by contributing to visual cues of neighborhood disorder (e.g., abandoned buildings, brownfields, graffiti, persistently broken windows), which have been shown in turn to affect the neighborhood social processes discussed above, such as social control and collective action [74].

1.5 WHEN PLACE MATTERS

The social and structural processes outlined above all suggest that neighborhood effects on health may depend on when and for how long someone was exposed [75]. Yet the majority of extant research in this area has relied on cross-sectional data in which neighborhood-health effects are assumed, at least implicitly, to be instantaneous and equivalent across the life course. To the extent that these effects are more or less consequential during specific stages in the life course or accumulate over time, previous studies that use static models and point-in-time measurements may substantially underestimate the effects of timing-specific and long-term exposures to neighborhood conditions that occur earlier in life, particularly during childhood and adolescence.

1.5.1 Timing of Exposure

Prior research has shown that early childhood exposure to poverty-related stressors can have lasting impacts on adult physical and mental health, net of future income, suggesting that early childhood (often defined as birth through age 5) is a developmental period that may be especially sensitive to the effects of poverty-related adversity [76-81]. However, such inferences are generally based on measures of income that are assessed at the family- or household-level rather than at the neighborhood-level. Whether the effects of neighborhood disadvantage are similarly sensitive to the developmental timing of exposure remains largely underexplored. Nevertheless, extrapolating from the research on family poverty implies that the effects of neighborhood disadvantage during early childhood are likely to be more indirect (i.e., mediated by the family) compared to later stages of development. For example, cross-sectional research has shown that high-threat and resource-poor neighborhoods, operationalized by high poverty, unemployment, female-headed households, racial segregation, abandoned housing, and population loss, may engender unsupportive and harsh parenting and distraction and withdrawal of affection, leading in turn to higher rates of child maltreatment [82, 83] with lasting consequences for health, including heightened immune responses that are known risk factors for the development of cardiovascular disease, diabetes, asthma, and chronic lung disease in adulthood [84, 85]. In addition, physically dangerous neighborhoods or those that lack high-quality childcare, libraries and children's programs may more directly impact future outcomes by restricting opportunities for early childhood development, including peer interaction and learning-related play, during a critical period of early childhood biopsychosocial development [86, 87]. Finally, as discussed above, neighborhoods are characterized by differential likelihoods of exposure to environmental toxins, including dust, mold or lead paint from dilapidated housing, and diesel or air pollution from proximity to major freeways, ports, and hazardous waste sites [73]. Such exposures may be particularly damaging during earlier stages of the life course when neurological and physiological systems (e.g., brain or lungs) are still developing and therefore more sensitive to environmental stimuli [76, 81, 84].

On the other hand, however, there are compelling reasons to believe that neighborhood disadvantage will matter more for future health if exposure occurs during the adolescent (rather than the early childhood) phase of the life course. In particular, adolescence is marked by an increasing need for autonomy and expanding social interactions [88]. Neighborhoods, in turn, are thought to be one of the primary contexts for adolescents' out-of-home time. They provide not only the physical space in which youth frequently operate, but also the

social space in which a wide array of interactions occur [89, 90]. These physical conditions and social experiences not only structure opportunities to make healthy choices but also shape norms, values, attitudes, knowledge, and behavioral tendencies related to health and health care [91]. For example, previous research has shown that dietary choices are influenced by the availability of food stores (e.g., supermarkets vs. convenience or corner stores) and other food service providers (e.g., fast food or alcohol outlets), and that low-wealth and predominantly black neighborhoods tend to have less access to healthy food options [25, 65, 92-94]. As adolescents are increasingly able, allowed, and required to manage some or all of their own meals, the neighborhood food environment may become particularly influential for establishing life-long eating behaviors. A similar phenomenon may also occur for physical activity such that the presence and safety of neighborhood parks and recreation settings during the increasingly independent and active periods of the adolescent life course may have lasting implications for the level and intensity of exercise as well as related conditions such as obesity and cardiovascular disease in later life [95]. Moreover, if parents are suffering higher rates of family, residential or financial stress, it is likely that other adults in the neighborhood are as well. The socializing impact of this collective reality may influence developing belief systems, including lowered expectations and self-efficacy, reduced goals and planning, and awareness of fewer resources and opportunities, especially among adolescents [82] who may experience worse mental health or who may turn instead to illicit or other health-compromising behaviors such as violence, substance use, or risky sexual activity.

1.5.2 Duration of Exposure

In addition to, and perhaps more important than, the timing of neighborhood exposures is their persistence or duration. Prolonged residence in disadvantaged or advantaged neighborhoods may be thought of as a source of increasing inequality that clusters and compounds over time through processes of cumulative disadvantage and cumulative advantage [96, 97]. On the one hand, Merton [98] argues that individuals in advantaged social circumstances are able to reinforce their position through exposure to better employment opportunities and accruing higher incomes (and related health benefits) over the course of their lives, while simultaneously reducing their exposure to risk factors that may compromise health [96]. Such processes are typically observed in the case of health behaviors wherein people who live in more affluent neighborhoods, with more prevalent grocery stores, recreational facilities and aesthetic features, as well as fewer reports of crime and social or physical disorder, tend to eat better and exercise more [99]. On the other hand, persistent exposure to disadvantaged neighborhoods exposes residents to stressful conditions (e.g., residential

instability, disorder, crime and violence) and physical hazards (e.g., noise, traffic, pollution and other toxins) that accumulate and may reinforce health compromising behaviors over time through local norms about fitness, diet, tolerance for substance use, and views about when and where to seek health care [96].

In addition to shaping such health behaviors, research also suggests that persistent residence in disadvantaged neighborhoods may directly impact the regulation of physiological systems engaged in the body's response to stress. Such work adds complexity to more traditional notions that required an existing genetic predisposition by showing that neighborhood disadvantage, as a source of stress, may itself increase biological vulnerability to disease, especially when compounded over time [100]. In particular, the stress response generally involves the immune, endocrine, and/or cardiovascular systems [101]. In the immune system, the adrenal gland produces hormones that suppress the activity of B and T lymphocytes, thereby preventing the body from destroying or neutralizing foreign substances, such as bacteria and viruses, and increasing vulnerability to disease [102]. Moreover, two endocrine response systems are thought to be particularly reactive to stress-eliciting exposures: the hypothalamic-pituitary-adrenocortical axis (HPA) and the sympathetic-adrenal-medullary (SAM) system [101]. Cortisol, the primary initiator of HPA activation in humans, regulates a broad range of physiological processes, including anti-inflammatory responses, metabolism of carbohydrates, fats and proteins, and gluconeogenesis. Similarly, catecholamines, which are released in response to SAM activation, work in concert with the autonomic nervous system to exert regulatory effects on the cardiovascular, pulmonary, hepatic, skeletal muscle, and immune systems [101]. Chronic (prolonged or repeated) activation of the HPA and SAM systems resulting, for example, from neighborhood-related stressors is considered particularly toxic because it is the most likely to result in long-term or permanent changes in the physiological, emotional, and behavioral responses that influence susceptibility to and the progression of disease [101, 103, 104].

The increased "wear and tear", so to speak, induced by persistent stressful exposures that overwork and dysregulate the physiological pathways normally used for adaptation to threat may also be associated with an acceleration of the normal aging process, or "weathering", as well as with the breakdown of physiological steady state, a phenomenon referred to as "allostatic load" [84, 105, 106]. For example, recent research examining leukocyte telomere length (LTL), essentially the protective tips of chromosomes, as a marker of immune system aging and cumulative life stress suggests that compared to whites, blacks have either shorter telomeres or a higher rate of telomere shortening, which may occur in response to heightened replication

associated with disproportionate and prolonged exposure to disadvantaged neighborhoods across the life course [100]. Conversely, neighborhood advantages, such as social cohesion, collective efficacy, informal social control as well as better access to transportation, childcare, jobs and other institutional resources, may limit, counteract or buffer the impacts of psychosocial and physical stress, aiding in the development of stress response patterns characterized by less frequent activation and quicker return to normal functioning [107]. Finch and colleagues [108], for example, have shown that concentrated neighborhood advantage, operationalized in terms of neighborhood educational composition, is associated with lower (healthier) levels of allostatic load, and that this association is especially true for those individuals who already have higher levels of education.

1.6 Methodological Considerations

In addition to a more explicit theoretical and empirical focus on the mechanisms linking neighborhoods and health, recent scholarship has attempted to better model this relationship using statistical methods that not only move beyond the artificial people or places dichotomy but also incorporate the temporal complexities associated with dynamic processes of residential selection and the interrelationships between compositional and contextual effects on health across time. Whereas an experimental study design in which individuals are randomly assigned to reside in more or less disadvantaged neighborhoods over the course of their lives would be considered the ideal scenario for addressing selection bias and related concerns about confounding by individual (or compositional) characteristics, such studies are rare (for various ethical, economic, and logistical reasons). Moving to Opportunity (MTO), a five-site housing mobility experiment sponsored in 1994 by the US Department of Housing and Urban Development (HUD), was a notable exception. MTO assigned low-income families living in concentrated poverty (40 percent or greater) to one of three experimental conditions. In the first, families were given a housing voucher (as well as counseling and housing assistance) and required to move to a neighborhood with less than 10 percent poverty. In the second, families were also given a housing voucher, but no restrictions were imposed on where they could move. The third condition was a control in which no housing vouchers or other assistance was offered. Results from the MTO experiment have been mixed. To be sure, significant positive effects of moving to a more affluent neighborhood have been reported for adult mental health, young female education, physical and mental health of female adolescents, and risky behavior among young girls. For other outcomes such as cognitive achievement and economic self-sufficiency,

however, no statistically significant effects have been found, while adverse effects of moving to a more affluent area have been documented for physical health and delinquency among adolescent boys [13]. Among the various explanations offered for such mixed findings is the notion that many of the families who participated in the MTO experiment had lived in impoverished areas for considerable periods of time prior to the start of the intervention and may therefore have already experienced the timing-specific and/or cumulative effects of neighborhood disadvantage [13].

Nevertheless, the ability to address selection-related biases through random assignment was a noteworthy breakthrough in the neighborhood effects literature and identifying other techniques for improving causal inference remains a central aim in the field. Given the challenges associated with conducting social experiments similar to MTO, various more recent studies have instead employed observational panel data and marginal structural models with inverse probability of treatment (IPT) weighting to mimic such an experimental design [36, 109-111]. Typically, studies that rely on observational data simply control for the individual-level demographic and socioeconomic factors that characterize the residents of different types of neighborhoods (as opposed to equating otherwise dissimilar individuals through random assignment as in experimental designs). Unfortunately, there are at least two fundamental limitations to such a conventional regression approach. First, values on these individual-level characteristics may change over time – that is, they are time-dependent or time-varying (e.g., when a change in employment or marital status results in more or less income). Second, as discussed previously, such individual-level demographic and socioeconomic factors may operate not only as confounders of the neighborhood-health relationship, but also as mediators (e.g., when neighborhood conditions affect job prospects and other income-generating opportunities which in turn influence health [32]). Controlling for these characteristics (or, more likely, the average of all the values on each of these characteristics over the study period) would remove from the model the mediated (but still relevant) pathways through which neighborhood effects may transpire.

Marginal structural models using IPT weights are a means of incorporating the indirect effects of neighborhood disadvantage on health while still adjusting for possible confounding (as a function of residential selection) by time-varying individual-level characteristics. In essence, the IPT weights are used to produce a pseudo-population in which random assignment of exposure to neighborhood disadvantage is simulated and to which the marginal structural model can be subsequently fit. In short, this pseudo-population is generated by using what is known (or observed) about an individual's (or in the case of children and adolescents, about

their household head's) demographics as well as income, work hours, employment, marital and homeownership statuses, and level of neighborhood disadvantage in prior years to predict their level of exposure to neighborhood disadvantage in subsequent years. Each individual is then weighted according to the inverse of this predicted probability. In this way, proportionally more (or less) weight can be given to those individuals whose prior time-varying covariates are underrepresented (or overrepresented) in the neighborhood disadvantage quintile to which they are actually exposed at each year. That is, the IPT weights ensure that the values of all observed individual-level factors are balanced in expectation across the various levels of neighborhood disadvantage at each year. Figure 1.3, produced by Wodtke and colleagues [36], illustrates this process for a simple two-wave dataset. It displays the relationships among neighborhood disadvantage, time-varying individual-level factors, and an outcome of interest (such as health), as well as how such relationships change as a function of the IPT weights. In the pseudo-population generated using the IPT weights, shown on the right-hand side of the figure, individual-level characteristics (L_k) no longer predict exposure to neighborhood disadvantage (A_k), mimicking a situation in which exposure to neighborhood disadvantage is randomized across time. Overall, then, marginal structural models using IPT weights account for selection into different types of neighborhoods while also allowing compositional effects to coexist with contextual effects on future health.

1.7 Research Overview

1.7.1 Data Source

The following three empirical chapters build on the theoretical and methodological advances discussed above to examine the consequences of prolonged and timing-specific exposure to neighborhood disadvantage throughout childhood and adolescence on future (young adult) health outcomes, as well as racial disparities therein. Individual- and household-level data for all three studies are drawn from the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID). The PSID is a large, longitudinal survey of U.S. residents and their families conducted every year between 1968 and 1997 and every two years thereafter. The original sample included approximately 5,000 families and 18,000 individuals; however, because the descendants of original PSID families continue to be interviewed once they form their own independent households, the sample has grown to more than 9,000 families and 22,000 individuals in 2011. In addition to information on health risk and protective factors, such as employment, income, education, marriage, social program participation, and

homeownership, the PSID also records the residential location of individual respondents at each interview. This makes it possible to merge the PSID dataset with census data on the characteristics of respondents' neighborhoods and to track both the duration and timing of exposure throughout the entire child and adolescent life course.

Consistent with most prior research in this area, census tracts are used to approximate neighborhood boundaries. As described elsewhere, there are well-known limitations to operationalizing neighborhoods (and the measurement of characteristics within them) in terms of just a single, predefined administrative unit [112, 113]. There is, however, broad consensus that census data at the tract level not only provide convenient access to considerable information over extensive time periods, but they also serve as a reasonable proxy for, or are at least highly correlated with, the "true" causally relevant definition of a neighborhood [14, 26, 114, 115]. Census data in 1970, 1980, 1990, 2000, and for the third empirical chapter, 2010 come from the Neighborhood Change Database (NCDB) in which information at each decade has been normalized to the most recent tract boundaries and can therefore be compared without having to adjust for potential changes in boundary definitions over time. Linear interpolation is used to impute values in intercensal years. The first two studies measure neighborhood disadvantage using a composite index of seven census tract items: poverty, unemployment, female-headed households, public assistance receipt, educational attainment (high and low), and occupational prestige. Individual respondents are assigned a score on this index for every year throughout childhood and adolescence. To facilitate the implementation of the statistical approach described above, these scores are divided into quintiles ranging from the least (level 1) to the most (level 5) disadvantaged neighborhoods based on the distribution of all tract-year observations from 1970 to 2000. The third study focuses on the effects of just neighborhood poverty, measured as a three-level ordinal variable based on the poverty rate of the census tract in which respondents resided for every year during their youth.

1.7.2 Chapter 2

The first empirical chapter (Chapter 2) focuses on the duration of exposure to neighborhood disadvantage, examining the effects of prolonged residence in more impoverished areas from birth through age 17 on the probability of reporting an incidence of fair or poor (as opposed to excellent, very good, or good) health in early adulthood (ages 18-30), as well as the extent to which group differences in exposure duration help to explain racial disparities therein. The chapter begins by reviewing patterns of racial residential segregation and associated inequalities in social and structural resources and opportunities between predominantly white and

nonwhite neighborhoods. It goes on to reiterate the theoretical mechanisms through which chronic (prolonged or repeated) exposure to neighborhood disadvantage is thought to affect the more proximal behavioral and biological precursors of poor health. Following this discussion, compositional and contextual data on 1,757 respondents are used to fit a marginal structural logistic regression model in which the parameters are estimated using IPT weights, as described above. Findings reveal that exposure to neighborhood disadvantage throughout childhood and adolescence is considerably more common and more persistent among nonwhite than white respondents and is associated, in turn, with significantly greater odds of experiencing an incidence of fair or poor health at least once between ages 18 and 30. The chapter concludes, therefore, that the separate and unequal neighborhood environments in which white versus nonwhite children and youth tend to live, learn, and grow may play a critical role in producing and perpetuating racial disparities in self-rated health status in early adulthood (and beyond).

1.7.3 Chapter 3

The second empirical chapter (Chapter 3) examines the effects of both duration and timing of exposure to neighborhood disadvantage during childhood and adolescence. The health outcome of interest is the probability of being obese at least once between ages 18 and 30. Accordingly, the chapter begins with a review of neighborhood social and structural processes that are more specific to obesity incidence, including those associated with diet and physical activity related norms and behaviors. The duration component of the investigation is similar to the previous chapter in that exposure to neighborhood disadvantage is assessed continuously from birth through age 17. Findings for obesity incidence reiterate those for self-rated health: prolonged exposure to neighborhood disadvantage throughout childhood and adolescence is more common among nonwhites than whites and is associated with significantly greater odds of worse health in the future (in this case, of being obese at least once in early adulthood). For the timing component of the investigation, the child and adolescent life course is divided into three stages: early childhood (ages 0-5), late childhood (ages 6-11), and adolescence (ages 12-17). Consistent with the hypothesis that the neighborhood environment becomes an increasingly salient influence as children grow older and more independent, a series of marginal structural logistic regression models with IPT weights reveal that exposure to neighborhood disadvantage during adolescence is more consequential for future (young adult) obesity incidence than exposures that occur earlier in childhood. Thus, although the chapter reiterates the importance of durable place-based investments in children and youth that last across the life course, it suggests that efforts targeting adolescents (as opposed

to younger children) may be particularly successful in addressing the health-related consequences of exposure to neighborhood disadvantage.

1.7.4 Chapter 4

The third empirical chapter (Chapter 4) shifts attention to health behavior, focusing on the age of smoking initiation among children and youth who are exposed to varying levels of neighborhood poverty from age four onwards. As described in more detail in the chapter, smoking is a health-risk behavior that has been associated in primarily cross-sectional research with neighborhood social and structural factors, such as collective socialization, collective efficacy, retail availability, and tobacco advertising, as well as with chronic health conditions such as cardiovascular and respiratory disease, stroke, and lung cancer. Of particular concern in this chapter is the fact that tobacco use that is initiated at younger ages is more likely to result in habit-forming behavior that subsequently accumulates over longer periods of time and increases risk for disease. Similar to the previous empirical chapters, IPT weights are used to create a pseudo-population to which race-specific discrete-time logit models are fit to estimate the risk of initiating smoking by age 25 as a function of the duration of exposure to low, moderate-, and high-poverty neighborhoods. Findings indicate that more prolonged exposure to high (>20%) as opposed to low (<10%) poverty neighborhoods is associated with an increased risk of smoking onset by age 25. Although consistent with prior literature, this effect is only observed among white and not nonwhite respondents. The chapter, therefore, underscores the importance of the neighborhood environment across the life course while simultaneously highlighting the potential for differential impacts across different health outcomes and different racial/ethnic groups.

1.8 CONCLUSION

The final chapter of this dissertation summarizes the theoretical and policy relevance of the findings from the previous three empirical chapters. Study limitations, remaining gaps in the literature, and suggestions for future research examining neighborhood-health effects over the life course are also discussed including, for example, incorporating biomarkers to better assess emerging disease, examining intergenerational effects of prolonged and/or timing-specific exposure to neighborhood disadvantage, as well as assessing the health-related consequences of additional neighborhood stressors such as racial-spatial patterns of incarceration and prisoner reentry. Overall, this dissertation contributes to better understanding the temporal dimensions of neighborhood effects on health and adds support to the growing body of literature suggesting that durable,

place-based investments in the social, economic, institutional, and physical structures of disadvantaged neighborhoods can have long-term benefits for population health and health equity that extend from birth through adulthood.

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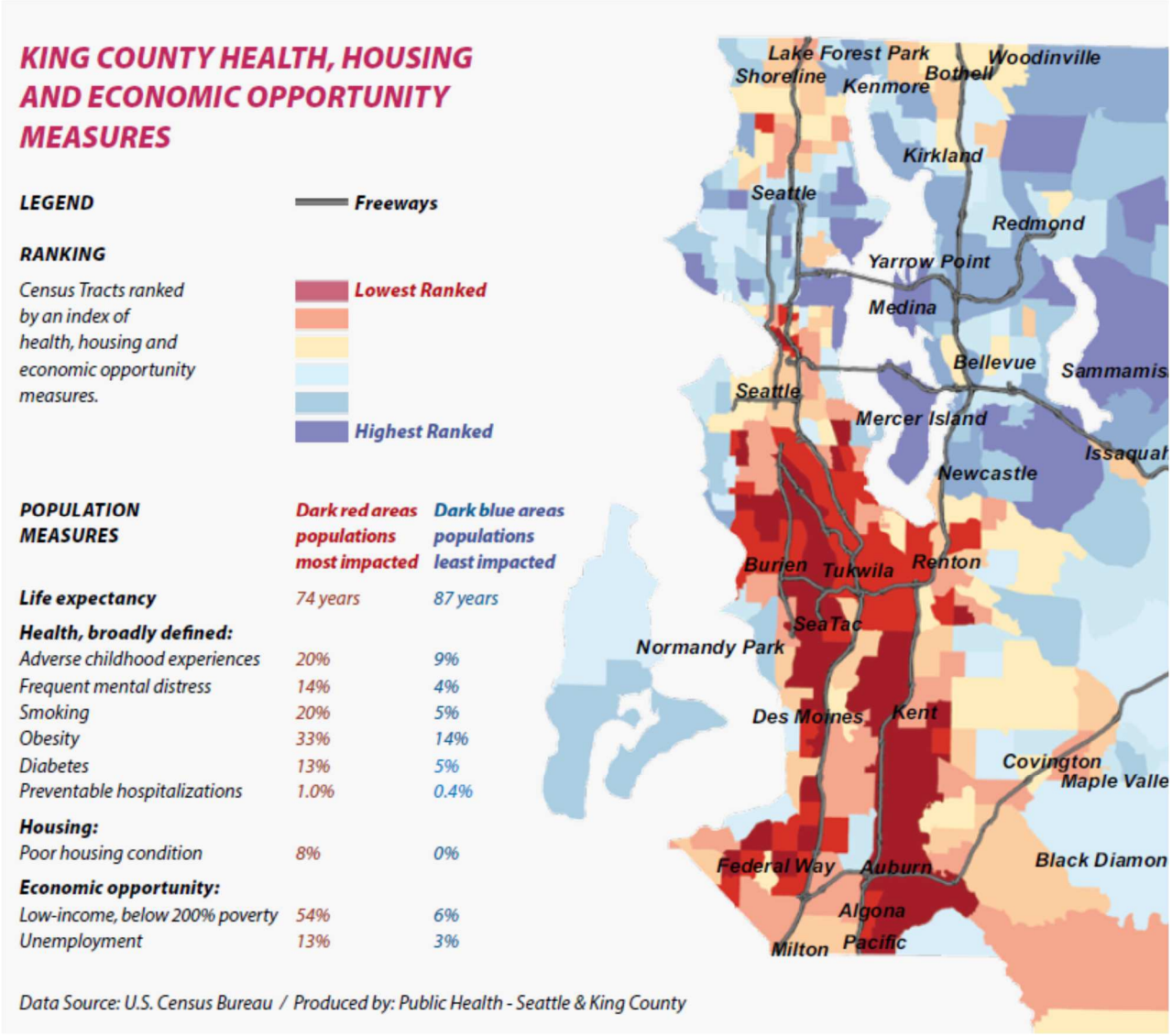

Figure 1.1. Geographical patterning of health, housing, and economic opportunity measures in King County, Washington [2].

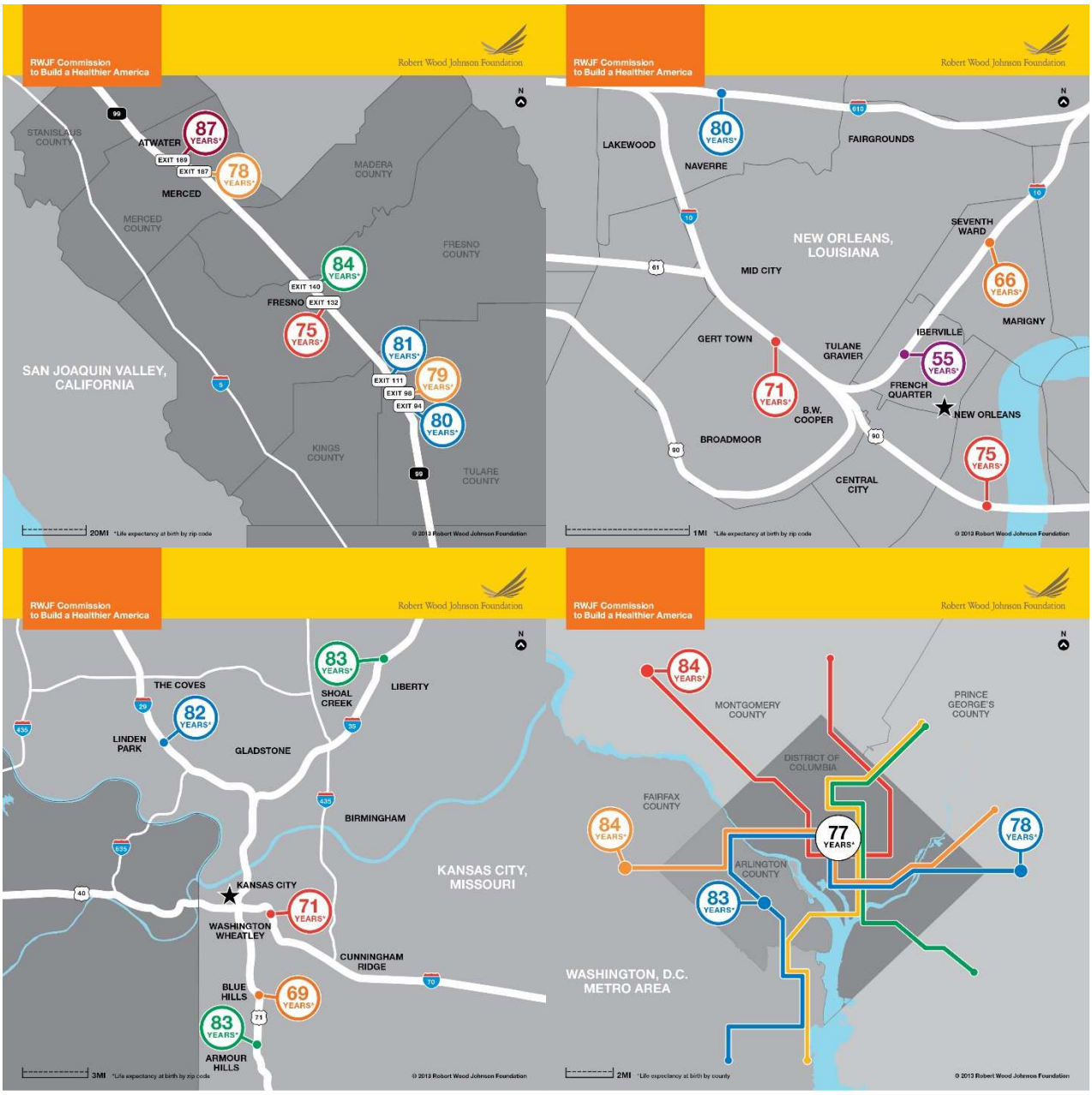

Figure 1.2. Small area inequalities in life expectancy by region [5].

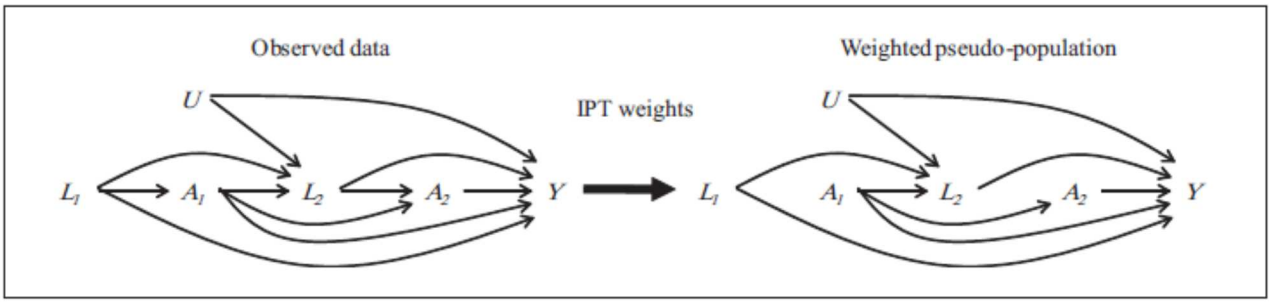

Figure 1.3. Causal graph for the effect of weighting by the inverse probability of treatment (IPT); A_k = neighborhood context, L_k = observed time-varying confounders, U = unobserved factors, Y = outcome [36].

Chapter 2. CUMULATIVE EFFECT OF GROWING UP IN SEPARATE AND UNEQUAL NEIGHBORHOODS ON RACIAL DISPARITIES IN SELF-RATED HEALTH IN EARLY ADULTHOOD

2.1 INTRODUCTION

Mounting evidence that purely individual-based explanations fail to fully explain persistent disparities in morbidity and mortality has contributed to a surge in research investigating how residential conditions further shape the health of individual residents [1]. Neighborhood and community-level characteristics, particularly concentrated poverty and other forms of socioeconomic disadvantage, have been associated with premature death [2, 3], poor self-rated health [3-6], depression and other mental health problems [7-10], health-risk behaviors such as alcohol abuse, tobacco use, poor diet, and physical inactivity [11-14], and related chronic conditions such as obesity, diabetes, cardiovascular disease, and certain cancers [15-19], net of individual-level factors. In addition, numerous studies have documented that exposures to such adverse neighborhood conditions are unequally distributed both in the population and across the life course. African Americans, in particular, are not only more likely than statistically comparable whites to ever reside in areas characterized by high levels of disadvantage but to do so for repeated or prolonged periods of time [20-32]. These findings point to the importance in neighborhood effects research of considering both if and for how long exposures occur, especially when assessing their contribution to racial inequality.

Until recently, however, the majority of scholarship in this area measured neighborhood characteristics only once or over just a short window of observation, conflating persons who were recently exposed with those who have (and in the case of some groups, are more likely to have) experienced more sustained residential adversity. Such a conceptualization is inconsistent with most theories of neighborhood effects, which tend to specify mechanisms that imply persistent exposure [27, 28, 33-36], as well as with a developmental or life course perspective in which experiences earlier in life are posited to have formative and enduring impacts on future outcomes, even when controlling for more contemporaneous determinants [37, 38]. Among the relatively small but growing number of neighborhood-level studies that have employed longitudinal designs, few measure residential conditions regularly across both childhood and adolescence and even fewer examine subsequent effects on adult health.

Using data from the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID) merged with census data on respondents' neighborhoods, this study estimates a marginal structural logistic regression model with inverse probability of treatment and censoring weights to examine: (1) whether cumulative exposure to varying levels of neighborhood disadvantage from birth through age 17 affects self-rated health in early adulthood (ages 18-30); and (2) the extent to which variation in such exposure helps to explain racial disparities therein. To capture both continuity and change in residential conditions throughout childhood and adolescence – children can move in and out of different neighborhoods or remain in areas that may or may not change around them – levels of neighborhood disadvantage are characterized once per year, every year during this time. In addition, by employing a statistical method that accounts for selection bias without controlling away neighborhood effects that may operate indirectly through the same individual-level covariates that have been associated with residential mobility, this study provides a more comprehensive assessment of the impacts on young adult health of exposure to varying levels of neighborhood disadvantage that accrues across the entire child and adolescent life course.

2.2 THEORETICAL FRAMEWORK

To theorize the ways in which the accumulation of neighborhood exposures from birth through age 17 impacts self-rated health in early adulthood, this study draws on Gee and Payne-Sturges' [39] stress-exposure-disease framework and incorporates a life course perspective. In the adapted conceptual model depicted in Figure 2.1, race is a fundamental determinant of the level of neighborhood disadvantage into which a child is born as well as their (or their family's) likelihood of remaining in or moving out of similar residential environments over the next 18 years. Neighborhood disadvantage, in turn, is operationalized as the spatial clustering of poverty, unemployment, female-headed households, public assistance receipt, and educational and occupational marginalization [33, 34, 40-42]. The assumption is that such composite neighborhood characteristics are concomitant to social and structural resources and opportunities (or the lack thereof) that can directly, and in conjunction with individual- and household-level risk and protective factors, be salutogenic (health promoting) or pathogenic via their effects on more proximal behavioral and physiological determinants of health [43, 44]. A life course perspective embeds in this model the importance of time. Residing in a disadvantaged neighborhood for a particular moment (or even several moments) in time can be hazardous, but it is less likely to dramatically impact one's long-term health if surrounded by years of otherwise salutogenic

residential exposures (homicide is a notable exception). On the other hand, experiencing those same or similar adverse conditions repeatedly over time is more likely to have a compounding influence on future health [45]. The various components of this model are discussed more fully below, beginning with an overview of the fundamental relationship between race, residence, and inequality.

2.2.1 *Race and Residence in Separate and Unequal Spaces*

Residential segregation is central to understanding the racial-spatial patterning of health in the United States (US). Discriminatory housing policies and practices, including restrictive covenants, redlining, residential steering, and differentials in interest rates, subprime loans, and foreclosures, helped produce and continue to perpetuate the distinctive separation of whites and nonwhites in residential space [30, 46]. Although true for Latino and Asian Americans, such residential segregation from whites is particularly pronounced for African Americans. For example, in the average metropolitan area in 1970 (the start of data collection for this study and the post-WWII peak of black/white segregation), nearly four out of every five African American (or white) residents would have had to move to a different census tract in order to achieve an "even" residential pattern – that is, one in which every neighborhood replicated the black/white composition of the wider city [47]. Although black/white residential segregation has declined slowly but steadily since then, it remains at levels that are still considered "very high" (requiring at least 3 of every 5 black or white residents to move to achieve evenness) [47].

Of particular consequence is the inequality that comes with such separation. Residential segregation concentrates advantage and power derived from a racialized social hierarchy within predominantly white neighborhoods while disadvantage and marginalization in the form of poverty, joblessness, low-wage jobs, and the like are disproportionately concentrated in predominantly nonwhite neighborhoods [23, 30, 46]. Given enduring structural constraints on mobility coupled with race-specific preferences for and aversions to "in-group" and "out-group" neighbors [48], inter-neighborhood migration does little to alter such residential patterns. In fact, most moves occur (if at all) between areas of similar racial and thus socioeconomic composition [29, 49, 50]. In turn, even relatively affluent black, Latino, and Asian Americans are more likely than the average white to be born into and remain in areas characterized by comparatively high levels of deprivation [23]. Such a durable racial divide in the geography of disadvantage is thus at the heart of why neighborhoods are so fundamental to understanding racial inequality generally and racial health disparities more specifically [30, 51]. That is to say, even if neighborhood disadvantage exerts a similar effect on health

across racial groups (and there is little evidence to suggest otherwise), the greater likelihood for people of color to reside in resource-poor neighborhoods (especially over long periods of time) implies that the adverse health effects of neighborhood disadvantage will be more concentrated among these groups.

2.2.2 *Neighborhood-Level Social and Structural Processes*

Under conditions of racial residential segregation, salutogenic and pathogenic social and structural processes are dispersed unevenly across race and space. As mentioned above, this racial-spatial differentiation reflects broader inequalities in the distribution of social and structural resources and opportunities as well as greater investment (or disinvestment) in those areas in which more privileged white (or marginalized nonwhite) groups predominate. Thus, it is not just the genetic composition or unhealthy choices of individual residents, per se, that govern racial-spatial inequalities in health but rather the collective aspects of neighborhoods that facilitate (or constrain) access to society's goods and resources, such as employment, housing, schools, social services, and beneficial social connections, that can be used in different ways and in various situations to promote health, avoid risks, and buffer the short- and long-term consequences of injury or disease if they occur [52, 53]. Children who grow up in more disadvantaged neighborhoods are therefore exposed to a fundamentally different constellation of concomitant social and structural processes through which neighborhood-health effects have been theorized to transpire.

What are these neighborhood-level processes and how do they exert their effects on health? In essence, why would neighborhoods (in addition to family- or individual-level characteristics) matter for individual well-being? Over at least the last two decades, researchers from across the social and health sciences have identified a fairly consistent set of potential neighborhood-level processes thought to be responsible for the neighborhood-health linkage [1, 34, 36, 54, 55]. These explanations can be very broadly categorized within two domains – social and structural – although there is often considerable intersection. Within the social domain, explanations linking neighborhood disadvantage to individual health are generally derived from two interrelated paradigms: social isolation and social disorganization. The social isolation paradigm is grounded in Wilson's [56] seminal thesis in *The Truly Disadvantaged*. In it he contends that macroeconomic changes related to the deindustrialization of central cities resulted in the spatial and thus social isolation of low-income residents from middle and upper income families whose presence in the same neighborhoods had previously served to validate "mainstream" sources of influence as well as helped to attract and sustain the basic institutions of the area (e.g., schools, childcare centers, churches, credit unions, grocery stores, and

recreational centers) [57, 58]. In an extension of Wilson's work, Massey and Denton [46] maintain that racial residential segregation further ensured that such effects were disproportionately absorbed by and magnified in nonwhite, predominantly African American, neighborhoods.

Applied more specifically to health, social isolation has been posited to engender differences across neighborhoods in collective tolerance for and intergenerational transmission of potentially health compromising norms and behaviors (or what Jencks and Mayer [36] describe in terms of "collective socialization") [59]. As Anderson [60] documents in his ethnographic account of street violence in Philadelphia, some of these health compromising orientations may be adaptive for more immediate survival, such as establishing and maintaining a tough or street-wise persona or involvement in the informal or illicit economies to compensate for unmet need. On the other hand, the profound distrust of others, perpetually heightened sense of vigilance, and predominant focus on the "here and now" often associated with such activities, along with corresponding violent and other risky behaviors, can have short- and long-term consequences for health [59, 61, 62]. In addition, Small's [58] work documenting the significance of neighborhood institutions as "resource brokers" connecting residents to health-relevant information and services highlights how social isolation may not only reduce access to conventional role models but because middle and upper income families help attract and sustain basic (or anchoring) institutions, it may also limit the access of residents of impoverished neighborhoods to potentially salutogenic organizational connections and services (e.g., job opportunities, preventive health care, childcare centers, and early learning services).

The social disorganization paradigm, like social isolation, has evolved over time. Whereas previous iterations of the theory emphasized the importance of dense, intimate, and strong neighborhood ties (e.g., through friends or family) and the lack of conventional organization in disadvantaged neighborhoods, more recent accounts recognize the influence of "weak ties" [63] as well as the coexistence of both organization and disorganization in the same impoverished spaces [50]. The key constructs in these newer conceptualizations are "collective efficacy", or residents' perceived ability to mobilize and to undertake collectively desired actions, and "informal social control", a collective characteristic of neighborhoods wherein members of the community feel empowered to enforce shared norms [64]. Such processes do not require close-knit relationships. Rather, as Sampson [42] argues, "repeated interactions, observations of interactions, and an awareness of *potential* interactions that could be invoked all establish shared norms (a sense of 'we') beyond the strong ties among friends and kin" (p. 153). For example, the effects of widespread trust, neighborhood attachment, and

readiness to intervene for the common good on factors such as stress, fear, self-respect, and self-efficacy can benefit residents' health as well as parenting regardless of individual-level network ties [59]. Within such contexts, parents are more likely to know the parents of their children's friends, share information about children's behavior, monitor spontaneous play groups among children, and feel empowered to step in and enforce salutary behavioral norms. Moreover, at the institutional level, collective efficacy influences residents' willingness and capacity to mobilize and extract resources, for example, to protest the closure of a local hospital or clinic, to advocate for passage of a local ordinance to restrict smoking in public places, or to use zoning restrictions to prevent the incursion of toxic waste, fast food, or alcohol outlets in their neighborhood.

Explanations within the second, or structural, domain tend to focus on the content and quality of the institutional and physical environments and their impacts on health-related opportunities and choice sets. As already alluded to with respect to social isolation, disadvantaged neighborhoods are disproportionately underserved by high-quality and affordable housing, schools, daycare centers, grocery stores, pharmacies, police and fire personnel, sanitation services, public transit options, and recreational spaces. The quality of educational institutions, for example, is often directly linked to neighborhood socioeconomic conditions because public school funding is often geographically determined. Inequalities in school quality and subsequent inequalities in educational attainment, as well as occupational and income opportunities, in turn, are fundamental determinants of disparities in health and health care [52]. The availability of extracurricular activities and services associated with school quality, such as recreational programs and daycare centers, may also delay or prevent the initiation of health compromising behaviors among children and youth.

Disadvantaged neighborhoods are also more likely to encompass a disproportionate share of fast food and alcohol outlets as well as major freeways, ports, and other industrial sources of noise, diesel, and air pollution [65-67]. Such characteristics affect exposure to health risks (e.g., toxins in the air, water or soil; lead paint in dilapidated housing; unsafe working conditions) as well as the availability of and access to health-relevant institutional resources (e.g., culturally appropriate treatment options; employer-provided health care; adequately stocked pharmacies; safe, well-lit parks or green space; access to fresh fruits and vegetables) which can shape life-long norms and behaviors related to health and health care [12, 66, 68, 69]. Moreover, structural disinvestment in racially segregated, low-income neighborhoods may contribute to visual cues of neighborhood disorder (e.g., abandoned buildings, brownfields, graffiti, persistently broken windows), which

have been shown in turn to affect the neighborhood social processes discussed above, such as collective efficacy and informal social control [70].

2.2.3 *Individual-Level Behavioral and Biological Determinants of Health*

The constellation of neighborhood-level social and structural processes discussed above are further posited to "get under the skin" to affect the more proximal, individual-level determinants of health in at least two primary ways. First, they can impact health *indirectly* through their effects on an individual's health-related behaviors and coping strategies. For example, rates of violence, injury, substance abuse, risky sexual activity, and poor diet are more prevalent in disadvantaged neighborhoods where healthier alternatives are more difficult to access and maintain or otherwise ill-suited to the lived experiences of residents. Second, prior research further suggests that persistent residence in disadvantaged neighborhoods can *directly* impact the regulation of physiological systems engaged in an individual body's stress response systems. Such work adds complexity to more traditional notions that required an existing genetic predisposition by showing that neighborhood disadvantage, as a source of stress, may itself increase biological vulnerability to disease, especially when compounded over time [71]. In the immune system, for example, the adrenal gland produces hormones in response to stress that suppress the activity of B and T lymphocytes, thereby preventing the body from destroying or neutralizing foreign substances, such as bacteria and viruses, and increasing vulnerability to disease [72]. Moreover, two endocrine response systems are thought to be particularly reactive to stressful exposures: the hypothalamic-pituitary-adrenocortical axis (HPA) and the sympathetic-adrenal-medullary (SAM) system [73]. Cortisol, the primary initiator of HPA activation in humans, regulates a broad range of physiological processes, including anti-inflammatory responses, metabolism of carbohydrates, fats and proteins, and gluconeogenesis. Similarly, catecholamines, which are released in response to SAM activation, work in concert with the autonomic nervous system to exert regulatory effects on the cardiovascular, pulmonary, hepatic, skeletal muscle, and immune systems [73]. Chronic (prolonged or repeated) activation of the HPA and SAM systems, resulting, for example, from stressful neighborhood conditions, is considered particularly toxic because it is the most likely to result in long-term or permanent changes in the physiological, emotional, and behavioral responses that influence susceptibility to and the progression of disease [73-75].

The increased "wear and tear", so to speak, induced by persistent stressful exposures that overwork and dysregulate the physiological pathways normally used for adaptation to threat may also be associated with an acceleration of the normal aging process, or "weathering", as well as with the breakdown of physiological

steady state, a phenomenon referred to as “allostatic load” [76-78]. For example, recent research examining leukocyte telomere length (LTL), essentially the protective tips of chromosomes, as a marker of immune system aging and cumulative life stress suggests that compared to whites, African Americans have either shorter telomeres or a higher rate of telomere shortening, which may arguably occur in response to heightened replication associated with disproportionate and prolonged exposure to disadvantaged neighborhoods across the life course [71]. Conversely, neighborhood advantage may limit, counteract, or buffer the impacts of psychosocial and physical stress, aiding in the development of stress response patterns characterized by less frequent activation and quicker return to normal functioning [79]. Finch and colleagues [80], for example, have shown that concentrated neighborhood advantage is associated with lower (i.e., healthier) levels of allostatic load and that this association is especially strong for those individuals who already have higher levels of education.

Neighborhood disadvantage may have an additional direct effect on health via changes to the epigenome. The epigenome, which means “above” the genome, consists of chemical compounds that modify or mark the genome in ways that tell it what to do, where to do it, and when to do it; essentially turning gene expression off or on [81]. The epigenome is innately plastic and can be programmed or reprogrammed by chemical tags linked to socio-environmental experiences [71]. Although most epigenetic changes are probably harmless, researchers have discovered that certain changes may trigger or increase the severity of various cancers, diabetes, autoimmune diseases, and mental illnesses, as well as affect the uterine environment [71, 82]. Although epigenetic research is still in its infancy and few if any studies have examined the particular role of neighborhood disadvantage, recent work on childhood maltreatment (e.g., abuse and neglect) and the expression of genes critical to the stress response [83, 84] have led some scholars to posit that presumed genetic differences in various disease states may in fact be due to epigenetic differences that arise in response to chronic, stress-inducing contextual conditions experienced over the life course [71].

2.2.4 *Existing Research on Neighborhoods and (Self-Rated) Health*

As mentioned at the outset, the vast majority of extant research on neighborhoods and health has relied on more easily available, cross-sectional datasets. Findings from this body of work are mixed. A substantial number of studies have documented significant effects of neighborhood disadvantage, over and above individual-level factors, on various health outcomes that are linked to self-rated health (such as health behaviors, prevalence of chronic conditions, and mental health) [85-96]. Several studies, however, have found

significant effects of neighborhood affluence but not neighborhood disadvantage [41, 59, 97]. Moreover, a few studies have found no significant effects of neighborhood disadvantage on health after controlling for individual-level socioeconomic status, attributing contextual effects instead to the concentration of poor people in such areas [98-100]. One reason for these latter findings, as well as for the often modest neighborhood-level (relative to individual-level) coefficients in those studies in which an association with health is observed, may be the inability to account for the duration of exposure to various neighborhood environments over time.

The small but growing number of studies in which individuals' residential environments have been assessed longitudinally show a more consistent and often larger effect on health. For example, in a pioneering study investigating the influence of current and prior neighborhoods on mental health using panel data that collected information across three waves, Wheaton and Clark [101] find that childhood neighborhood has a lagged and cumulative effect on both externalizing (e.g., bullying, aggression) and internalizing (e.g., fear, anxiety, low self-worth) problems in early adulthood. As the authors describe, their findings underscore the notion that "all of these effects require time, and that shorter time frames may be ineffective in detecting the effect of early neighborhood" (p. 701). Johnson and colleagues [102] have further examined the relationship between neighborhood characteristics in young adulthood (ages 20-30) and self-rated health in mid-to-late life (ages 35 and older) using data on individuals who were followed for up to 38 years. Results suggested that young adult neighborhood characteristics, particularly concentrated poverty, account for up to 20 percent of the variation in health status in mid-to-late life, even in the presence of a reasonably large amount of unobserved confounding due to individual- and household-level factors. Moreover, Clarke and colleagues [45] have investigated changes in health status using a cumulative measure of exposure to neighborhood disadvantage, affluence, and ethnic/immigrant concentration among adults aged 25 and older over 15 years and four waves of observation. Their findings documented a significant cumulative effect of neighborhood disadvantage (but not affluence or ethnic/immigrant concentration) on changes in health status, such that increased duration of exposure to neighborhoods characterized by greater disadvantage was associated with 20 and 40 percent higher odds of functional decline and death, respectively, net of individual-level socio-demographic factors. Together, these results emphasize the potentially critical role not only of residential exposures at some earlier point in time, but of the combined history or accumulation of those past exposures for more precisely capturing neighborhood effects on health.

The current study grows this emerging literature in at least two important ways. First, it explores the young adult health consequences of past residential exposures when neighborhood characteristics are measured regularly throughout the entire child and adolescent life course, from birth through age 17, rather than at only a few discrete points during this time (as in Wheaton and Clarke [101]) or at already more advanced ages (as in Johnson et al. [102] and Clarke et al. [45]). Second, this study addresses the potential for bias due to the differential selection of individuals into or out of certain neighborhoods using statistical methods that do not "control away" the indirect effects of neighborhoods which may operate through time-varying individual- and household-level factors, such as employment status or household income, as is the case in conventional regression-based approaches. To be sure, research focusing on outcomes other than health, particularly in the realms of education and criminology, has been at the forefront of this so-called "temporal turn" in the literature on neighborhood effects, and several studies have already engaged with such issues [see 33, 103, 104-106]. This study builds on such research to help clarify the effect of cumulative exposure to neighborhood disadvantage throughout childhood and adolescence on self-rated health in early adulthood using a marginal structural model with inverse probability of treatment and censoring weights.

2.3 Data and Methods

Data for this study are derived from the Panel Study of Income Dynamics (PSID), produced and distributed by the University of Michigan's Institute for Social Research and funded by the National Institutes of Health (NIH) and the National Science Foundation (NSF). The PSID is a large, nationally representative survey of United States (US) residents and their families conducted annually between 1968 and 1997 and every two years thereafter. Respondents from original family units are said to have the PSID "gene" and are followed over their entire lives, regardless of where they live. All individuals born to or adopted by somebody with the PSID gene acquire it themselves and are followed as well. As a result, when children in PSID families grow older and form their own independent households, they continue to be interviewed. With low attrition and high success in following young adults as they form their own families [107], the PSID sample grew from roughly 5,000 families and 18,000 individuals in 1968 to more than 9,000 families and 22,000 individuals in 2011.

There are several reasons the PSID is uniquely suited to examining the cumulative effects of neighborhood disadvantage on health and health inequalities. First, the dataset contains an oversample of low-income families, as well as a wealth of information on a variety of potential individual- and household-level health-risk

and protective factors, including employment, income, education, marriage, social program participation, and housing characteristics. Second, the residential location of individual respondents at each interview can be linked to their corresponding census tract identifiers using the PSID's supplemental, restricted-use Geospatial Match Files. These identifiers make it possible to characterize neighborhood disadvantage for prolonged intervals as well as to account for the potential mobility of PSID respondents into and out of different neighborhoods. Third, the longitudinal design of the PSID makes it possible to adjust for the temporal sequencing and compounding influence of both individual and residential conditions across time.

2.3.1 *Sample Selection*

To ensure respondents have data that can span from birth through early adulthood, the analytic sample for this study consists of the 4,523 individuals born into PSID family units between 1970 and 1980.[2] These individuals (or their families) are surveyed annually from birth until the last available wave of PSID data in 2011 or until they are lost to follow-up. Given the focus on exposure to adverse neighborhood conditions throughout the entire child and adolescent life course, respondents were dropped if they were not continuously present in the survey for every year from ages zero to 17 (N=4,523-1,881=2,642). Of those, an additional 885 individuals did not respond to any questions about general health status in early adulthood and were excluded from analyses.[3] The final sample includes 1,757 individuals: 634 are classified as nonwhite, of which the vast majority (94%) are African American, and 1,123 are classified as white. Adjustment for potential nonrandom attrition using censoring weights will be discussed at a later point.

2.3.2 *Dependent Variable*

The dependent variable is self-rated health. It has been shown to have high predictive validity for mortality, independent of other medical, behavioral, and psychosocial risk factors, and to reliably predict future morbidity, health care utilization, and hospitalization [88, 108-113]. Whereas other common indicators of health status, such as functional decline or chronic disease diagnoses, tend to be more relevant at older ages, prior research shows that because people perceive their health in terms of not only the presence or absence of illness, but

[2] Sample members were asked for their year of birth at multiple survey waves. To address potential discrepancies in individual responses across waves, year of birth was determined as the modal response. For the few cases in which respondents never answered the year of birth question but did respond to question(s) about their age, age was used to determine their year of birth (i.e., interview year minus age at interview). Again, if this approach produced discrepancies across waves, the modal response was used. There were only 110 of 73,251 (<1%) respondents in the total PSID sample for whom year of birth could not be determined.

[3] Fifty-eight percent of respondents were either lost to follow-up between birth and age 17 or did not respond to any self-rated health questions in adulthood. Although considerable, this is comparable to other studies using the PSID to assess cumulative neighborhood effects over such an extended period of time (see Wodtke et al. 2011). Moreover, bias from potential nonrandom sample attrition will be minimized via censoring weights.

also their ability to do what they need and want as well as general feelings of well-being, vitality, strength, and endurance, self-rated health is one of the few measures that can capture broad differences in health status across populations at both older and younger ages [114-116]. Self-rated health has been assessed in the PSID at every wave since 1984 using the question, *"Would you say your health in general is excellent, very good, good, fair, or poor?"* Consistent with prior research, responses are dichotomized such that 0 = "excellent, very good, or good" health and 1 = "fair or poor" health. Given respondents' relatively young age and thus lower likelihood of ill-health, as well as this study's focus on the incidence of poor health in early adulthood, the primary outcome of interest is *any* self-report of fair or poor (as opposed to excellent, very good, or good) health between ages 18 and 30.

2.3.3 *Main Independent Variable*

Similar to most prior research in this area, census tracts are used to approximate neighborhood boundaries. Although there are limitations to this operational definition [117], and plenty of arguments for and against competing units of spatial analysis (from cities, counties, metropolitan areas, and states to census blocks, street corners, and even individual housing units), there is broad consensus that census data at the tract level not only provide convenient access to considerable information over extensive time periods but also, for most practical purposes, serve as a reasonable proxy for, or are at least highly correlated with, the "true" causally relevant definition of a neighborhood [1, 118-120]. Information on census tracts in 1970, 1980, 1990, and 2000 comes from the Neighborhood Change Database (NCDB) in which data for all four decades has been normalized to 2000 tract boundaries and can therefore be compared across years without having to adjust for potential changes in boundary definitions over time. Data for intercensal years are imputed using linear interpolation.[4]

Recognizing that neighborhoods are multidimensional and that no single indicator can adequately approximate the various ways in which neighborhood effects transpire, this study characterizes neighborhood disadvantage in terms of seven census tract items thought to engender the neighborhood-level social and structural processes described previously: (1) proportion of residents below the poverty line; (2) proportion of residents (age 16 and older) in the civilian labor force and unemployed; (3) proportion of households with public

[4] If decennial census tract data in 1970 and/or 1980 was missing (likely due to as yet untracted areas) and linear interpolation was impossible, data from 1980, 1990, and/or 2000 was used to extrapolate values for up to five years prior to the most recent value (e.g., if data in 1970 was missing, the linear interpolation from 1980 to 1990 was extended to 1975).

assistance income; (4) proportion of households with children that are female-headed; (5) proportion of residents (age 25 and older) with less than a high school diploma; (6) proportion of residents (age 25 and older) with a bachelors or graduate/professional degree; and (7) proportion of residents (age 16 and older) employed in managerial or professional/technical occupations. Following Wodtke and colleagues [33] and others [121-124], principal components analysis is used to transform these seven items into a composite index of neighborhood disadvantage reflected by the first principal component. Each item's loading on this index is used to weight its contribution to a neighborhood disadvantage score which is calculated for every census tract at every year.[5] The resulting scores are then divided into quintiles ranging from the least (level 1) to the most (level 5) disadvantaged neighborhoods based on the distribution of all tract-year observations between 1970 and 2000. This information is merged with individual-level data on sample members in the PSID such that each respondent ends up with 18 different measurements of exposure to neighborhood disadvantage, one for every year from birth to age 17. Respondents' cumulative exposure to neighborhood disadvantage throughout childhood and adolescence is calculated in two ways: (1) the average of the 17 neighborhood disadvantage quintiles to which they were exposed from ages one to 17;[6] and (2) the proportion of time spent in the most disadvantaged neighborhood quintile from ages one to 17. As described below, neighborhood disadvantage at birth is not included in either measure but rather as part of a vector of baseline covariates.

2.3.4 *Covariates*

A large number of individual- and household-level variables are included in analyses as either time-invariant (baseline or constant) or time-varying. Time-invariant covariates include race (1=nonwhite; 0=white), gender (1=female; 0=male), birthweight (1=less than 88 ounces; 0=88 ounces or more), the mother's age at birth, the mother's marital status at birth (1=unmarried; 0=married), and the household head's educational

[5] Although principal components analysis forms as many independent linear combinations as there are variables (seven, in this case), only the first principal component, which accounts for the largest possible proportion of the total variability in the component measures (about 63 percent, in this case), was retained. Item loadings on this first principal component ranged from 0.341 for the proportion of female-headed households to 0.413 for the proportion of households receiving public assistance income. See Appendix A for more details.

[6] Because the same average value can be achieved for very different patterns of exposure to neighborhood disadvantage (e.g., consistently moderate levels may produce the same average value as a mixture of high and low), a preliminary latent class growth analysis was also performed to examine exposure trajectories throughout childhood and adolescence. Results showed that, in general, children and youth in this study do not experience much upward or downward mobility in the level of neighborhood disadvantage to which they are exposed between ages zero and 17 (more details available upon request). These findings suggest that an average measure of cumulative exposure to neighborhood disadvantage may therefore be a reasonable reflection of the experience of children and youth in this study.

attainment at birth (1=less than high school; 2=high school graduate; 3=at least some college)[7], all of which have been associated in previous studies with both neighborhood residence and individual health-risk and protective factors. Time-varying covariates, measured at each wave between ages zero and 17, include the household head's marital status (1=unmarried; 0=married), employment status (1=unemployed; 0=employed), and work hours, as well as family size, homeownership (1=does not own home; 0=owns home), transfer income receipt, including public assistance (1=yes; 0=no), and total household income, standardized using the Consumer Price Index (CPI-U) to 1985 dollars.

2.3.5 *Statistical Analysis*

To investigate the effects on self-rated health of cumulative exposure to different levels of neighborhood disadvantage, this study specifies a marginal structural logistic regression model in which the parameters are estimated using inverse probability of treatment weights (IPTW). Marginal structural models and IPTW estimators were introduced near the turn of the century and have since gained the most traction in the epidemiological literature, particularly in the analysis of clinical trial data [125-128], although applications in the social sciences generally and to neighborhood effects more specifically are mounting [see 33, 106, 129]. The rationale behind the approach has to do with the failure of conventional regression models to take proper account of time-varying covariates that may be simultaneously confounders for the effects of future exposures and mediators for the effects of past exposures on future outcomes. For instance, a family's income directly influences both the type of neighborhood they can afford to live in and the health and health care of its members. If a researcher does not control for family income (among other confounding covariates) when modeling the effects of neighborhood characteristics on health, she may overstate (or even spuriously induce) the neighborhood-health relationship. On the other hand, if she controls for family income (among other mediating covariates), she removes from the final estimate the indirect (but still relevant) effects of residential conditions that operate on health through family income (e.g., when neighborhood conditions affect job prospects and other income-generating opportunities which in turn influence health [see 130]). In longitudinal studies, this basic dilemma is compounded across time: family income, measured at any one wave, is a function of past income and past neighborhood conditions as well as a determinant of future income, future neighborhood conditions, and future health. Controlling for family income and the other time-varying covariates

[7] The household head's educational attainment is treated as time-invariant and measured at respondents' year of birth because the PSID does not measure parental education at regular intervals. If data at year of birth was missing, the most recent subsequent measurement was used, which for the vast majority of respondents was within 3 years of birth.

all 18 times they are measured in this study would not only be cumbersome but would wipe out the various indirect pathways through which neighborhood effects can transpire. Not controlling for such factors, however, can amount to what many critics view as among the biggest thorns in the side of neighborhood effects research: "selection bias".

Marginal structural models using the IPTW estimators are a means of incorporating the indirect effects of neighborhood disadvantage on health while still adjusting for possible confounding (that is, the differential selection into and out of certain neighborhoods) by time-varying individual- and household-level covariates. This method proceeds in two steps. First, a series of so-called treatment weights are generated from ordinal logistic regression models for each respondent at each age based on the inverse of the probability that they are exposed to their actual quintile of neighborhood disadvantage (as opposed to the other four levels of disadvantage), conditional on their neighborhood conditions in the prior year, baseline or time-invariant covariates (including neighborhood conditions at year of birth), and both prior year and concurrent time-varying covariates (as well as interactions between prior year and concurrent measures of both marital status and employment status to account for possible effects of a recent divorce or job loss on mobility into or out of neighborhoods characterized by different levels of disadvantage). In order to obtain narrower confidence intervals around the neighborhood effect estimate, these weights are stabilized by multiplying each one by the same probability as was used to generate it above, except the time-varying covariates are excluded from the ordinal logistic regression models. The series of 17 stabilized treatment weights for each respondent at each age are then multiplied together to produce a single, summary weight reflecting the probability of exposure to that respondent's actual *sequence* of neighborhood disadvantage quintiles throughout all of childhood and adolescence.

More intuitively, this weighting process creates a "pseudo-population" in which randomization across the different levels of neighborhood disadvantage is simulated at each year by giving proportionally more (or less) weight to those respondents whose prior time-varying covariates are underrepresented (or overrepresented) in the neighborhood disadvantage quintile to which they are actually exposed at that age. In this way, exposure to different levels of neighborhood disadvantage at each year is independent of respondents' prior time-varying covariates, thereby avoiding the problem of differential selection based on such variables while still allowing them both to be affected by prior neighborhood conditions and to affect future covariates and future health status (just not future neighborhood conditions).

In the second step, the marginal structural logistic regression model estimating the effects of cumulative exposure to neighborhood disadvantage throughout childhood and adolescence on the probability of self-rated fair or poor health in early adulthood is fit to the "pseudo-population" generated via the stabilized treatment weights just described. Controlling for time-varying covariates is no longer necessary since their confounding effects on neighborhood selection have already been accounted for through the weighting process. However, because baseline or time-invariant covariates are included in the logistic regression models used to calculate both the numerator and the denominator of the stabilized weights, they must still be controlled in the marginal structural model.

2.3.6 *Missing Data and Sample Attrition*

Missing data on all independent variables are multiply imputed using the two-fold fully conditional specification algorithm in STATA v.13.1 [131]. The two-fold fully conditional specification algorithm was specifically designed to impute missing data in longitudinal studies. Whereas conventional multiple imputation methods ignore the longitudinal and dynamic structure of the data and are difficult to implement in large databases with many respondents and long periods of follow-up, the two-fold approach imputes missing values using chained equations at each time point conditional on information at the same time point and user-specified adjacent time points [131]. This study employs concurrent information plus or minus the two years adjacent to any missing values for the imputation. Due to computational constraints, missing data on the seven census tract items used to characterize neighborhood disadvantage are not imputed separately. Rather, only the summary scores for neighborhood disadvantage generated through PCA based on those seven items are imputed. These scores are then divided into quintiles using pre-established cut-points from the distribution of all (nonmissing) tract-year observations in the NCDB.

As discussed previously, a considerable number of respondents were either lost to follow-up between birth and age 17 or did not respond to any self-rated health questions in early adulthood. This was not unexpected given the length of observation and stringent requirement that sample members be present at every wave of data collection from birth through age 17 plus at least one wave in early adulthood. To minimize the effects of biasing attrition – that is, loss to follow-up that is selectively related to self-rated health – stabilized censoring weights are generated in the same manner as the stabilized treatment weights described above, except now the weights model the probability of remaining in the study for each respondent at each age, conditional on the same covariates as before (e.g., race, sex, household income, household head's employment and marital

statuses, and prior exposure to neighborhood disadvantage). Again, the numerator of the stabilized censoring weight excludes time-varying covariates. In this way, loss to follow-up at each survey wave (like exposure to neighborhood disadvantage) is essentially independent of respondents' prior time-varying characteristics. A summary censoring weight is generated by multiplying the age-specific weights for each respondent. The "pseudo-population" to which the final marginal structural model is fit, as described above, is actually constructed based on the product of the stabilized treatment weights and the stabilized censoring weights for each respondent (not just the treatment weights).

2.4 RESULTS

2.4.1 *Sample Characteristics*

Tables 2.1 and 2.2 display summary statistics for the time-invariant and time-varying characteristics (measured at three distinct points in childhood, mid-childhood, and adolescence), respectively, among respondents not lost to follow-up before age 18 and who answered at least one self-rated health question in early adulthood. A comparison between nonwhite, largely African American, and white respondents reveals a pattern of notable differences. First and foremost, nonwhite young adults were nearly two times more likely than their white counterparts to report fair or poor health at least once in early adulthood (27% vs. 15%), a finding consistent with prior studies documenting a persistent racial disparity in self-rated health. As children, nonwhite respondents were also more likely to be born to an unmarried mother and into a household in which the head had less than a high school education. Moreover, they were nearly twice as likely as whites to be born low birthweight (less than 5.5 pounds), a risk factor for ongoing health problems ranging from cognitive delays to respiratory disorders.

Throughout childhood and adolescence, nonwhite respondents remained in households that were consistently more disadvantaged than white respondents with respect to the head's marital and employment statuses, as well as income and hours worked per week, which were often close to half those of white household heads when respondents were ages one through 17. The single most striking difference, however, was the inequality in exposure to neighborhood disadvantage highlighted at the top of Table 2.2. At age one, for example, 75 percent of nonwhite children resided in the most disadvantaged neighborhood quintile compared to just 17 percent of whites. At the other end of the spectrum, less than two percent of nonwhite versus 14 percent of white one-year-olds resided in neighborhoods characterized by the least disadvantage. This same general pattern of racial-spatial inequality persisted across the entire child and adolescent life

course, with 60 percent of nonwhites compared to 11 percent of whites residing in the most disadvantaged neighborhood quintile at age 17, while just five percent of nonwhites versus 21 percent of whites resided in the least disadvantaged quintile.

2.4.2 *Cumulative Exposure to Neighborhood Disadvantage*

Table 2.3 quantifies more explicitly racial differences in the duration of exposure to neighborhood disadvantage for nonwhite versus white children and youth from ages one through 17, documenting dramatic racial inequalities in both the average neighborhood disadvantage quintile to which children and youth were persistently exposed as well as the proportion of time they resided in the most disadvantaged neighborhood quintile. The top section presents summary statistics by race for the first measure of cumulative exposure to neighborhood disadvantage throughout childhood and adolescence. It reflects the sum of each neighborhood disadvantage quintile to which children were exposed from ages one through 17 divided by the total number of years exposure was assessed – in other words, the average level of neighborhood disadvantage to which children were exposed before age 18. Higher values indicate more persistent exposure to neighborhood disadvantage. Consistent with but more pronounced than the racially disparate pattern of point-in-time measures of neighborhood disadvantage displayed in Table 2.2, less than one percent of nonwhite respondents were exposed to the least disadvantaged neighborhoods, on average, throughout childhood and adolescence whereas nearly two-thirds (63%) grew up, on average, in the most disadvantaged neighborhood quintile. On the other hand, most white respondents spent their youth in neighborhoods that were, on average, in the mid-range of the disadvantage distribution. Only eight percent resided in the most disadvantaged neighborhood quintile, on average, while 12 percent experienced only the least disadvantaged neighborhoods before age 18.[8]

The bottom section of Table 2.3 summarizes the second measure of cumulative neighborhood disadvantage, the proportion of time respondents spent in the most disadvantaged neighborhood quintile from ages one through 17. This measure better captures variability in sustained exposure to the most extreme forms of neighborhood adversity. Again, dramatic racial disparities emerge that are more pronounced than at any single point in time. For instance, the average nonwhite respondent spent an overwhelming 69 percent of their youth in the most disadvantaged neighborhood quintile compared to 14 percent among the average white

[8] The measure of average neighborhood disadvantage is categorized and displayed as an ordinal variable in Table 2.3 for illustrative purposes only. It is treated as a continuous variable in subsequent analyses.

respondent. This translates to approximately 12 versus just two out of 17 years lived in neighborhoods characterized by the most disadvantage for nonwhite versus white respondents, respectively. Overall, these findings reiterate the persistence of racial residential segregation and prior research documenting the extremely "divergent social worlds" [30] and thus health-relevant resources and opportunities (or the lack thereof) to which nonwhites, particularly African Americans, and whites are exposed not only at any one time but cumulatively over the course of childhood and adolescence.

2.4.3 *Weights*

Because race clearly played such a defining role in determining the level of neighborhood disadvantage to which respondents were exposed, it is important to carefully consider how to proceed with analyses estimating the effects of neighborhood disadvantage on racial health disparities. For instance, one of the assumptions needed in order to build a case for causality from observational data is positivity (a.k.a. "overlap"). In this study, both nonwhites and whites must therefore have a non-zero probability of being exposed to every level of neighborhood disadvantage, including having a sufficient number of nonwhite respondents exposed to the least disadvantaged neighborhoods and white respondents to the most disadvantaged areas (i.e., their neighborhood exposure distributions need to overlap). In the case of marginal structural models using inverse probability of treatment (and censoring) weights, which rely in part on observing the neighborhood counterfactuals for each racial group in order to adjust for neighborhood selection processes, violations of the positivity assumption can result in weights that are unstable and sensitive to the presence of rare racial-spatial combinations (resulting in extremely large weights and thus disproportionately influential outlier observations) [132].

A common solution to this general problem is to stratify analyses by race. However, this approach limits comparability of neighborhood effect estimates across race-specific models and hampers estimation of the role of neighborhoods in producing and perpetuating disparities in health by race, a central aim of this study [133]. Fortunately, there were non-zero probabilities of exposure to all levels of neighborhood disadvantage for both nonwhite and white respondents at every time point throughout childhood and adolescence (results not shown).[9] Moreover, as displayed in the summary statistics in Table 2.4, the stabilized inverse probability

[9] In actuality, positivity assumes a non-zero probability of exposure to every level of neighborhood disadvantage for all *combinations* of covariates (not just race in isolation). As more covariates are included, data become sparse and the likelihood of non-positivity naturally increases. However, such zero probabilities are more likely to be random rather than structural in nature.

of treatment weights, censoring weights, and to produce the final weight, the product of the two, have means close to one and small standard deviations (without truncation at the 1st and 99th percentiles) for the combined (non-race-stratified) sample, suggesting that the presence of rare racial-spatial combinations did not exert overt influence on the results.

2.4.4 *Cumulative Neighborhood Effects on Self-Rated Health Disparities*

Table 2.5 shows results of unadjusted (Model 1), partially-adjusted (Models 2 and 3), and marginal structural model (Model 4) estimates of the relationships among race, neighborhood disadvantage throughout childhood and adolescence, and self-rated health in early adulthood. The first model merely quantifies the statistically significant disparity in the probability of reporting fair or poor health at least once in early adulthood among nonwhite versus white respondents, without considering prior neighborhood exposures or individual- and household-level covariates. The unadjusted logit estimates indicate that compared to whites, nonwhite respondents had over two times higher odds of experiencing fair or poor health at least once between ages 18 and 30 (exp(0.75)=2.12). Model 2 introduces a variable for cumulative exposure to neighborhood disadvantage, measured as the average of neighborhood disadvantage quintiles experienced by respondents every year from ages one through 17. Results based on the second measure of cumulative neighborhood disadvantage, the proportion of time spent in the most disadvantaged quintile, are substantively similar and so only those employing the average measure are discussed further.[10] Notably, including this cumulative measure of neighborhood disadvantage, which was significantly associated with worse health ($p<0.001$), rendered the coefficient for race statistically non-significant ($p=0.133$), indicating that race was no longer associated with self-rated health once neighborhood disadvantage was included in the model. As indicated by the interaction term added in Model 3, race also does not appear to moderate the association between cumulative neighborhood disadvantage and self-rated health ($p=0.961$ for the interaction term).[11] On the surface, these findings suggest not only that prior neighborhood conditions matter for future health but that dramatic differences in the average level of neighborhood disadvantage to which nonwhite and white respondents are exposed during childhood and adolescence may account for a considerable share of the racial disparity in self-rated health in early adulthood.

[10] Conventional regression and marginal structural model results based on the proportion of time respondents spent in the most disadvantaged quintile are available from the author upon request.
[11] The interaction term remained non-significant even when the variable for cumulative neighborhood disadvantage was centered on the first, second, third, fourth, and fifth quintile values.

Although instructive, the effect on young adult health of cumulative exposure to neighborhood disadvantage just described is likely confounded by individual- and household-level characteristics that vary across childhood and adolescence in conjunction, to a certain extent, with variations in neighborhood conditions. As discussed previously, characteristics such as household income, family size, homeownership, and the household head's marital and employment statuses at any one time may impact both the level of neighborhood disadvantage to which children and youth are exposed (i.e., neighborhood selection) as well as their health in early adulthood. These same characteristics may not only confound, but also mediate the basic neighborhood-health effect, such as when neighborhood conditions influence employment prospects and other income-generating opportunities among parents which may in turn have an effect on the health and health care of their children.

To account for the potential confounding and mediating roles of such time-varying characteristics on the relationship between cumulative neighborhood disadvantage and self-rated health, Model 4 presents estimates from a marginal structural logistic regression analysis using stabilized inverse probability of treatment and censoring weights while also controlling for baseline (or time-invariant) characteristics. These estimates indicate that residing in more disadvantaged neighborhoods throughout childhood and adolescence is associated with a statistically significant increase in the probability of reporting fair or poor health at least once between ages 18 and 30. More specifically, each unit increase in cumulative neighborhood disadvantage from ages one through 17 is related to a 22 percent increase in the odds of experiencing an incidence of fair or poor health in early adulthood ($\exp(0.20)=1.22$). By extension, respondents who were persistently exposed to neighborhoods in the most disadvantaged quintile from ages one through 17 (a majority of nonwhite respondents) had over two times higher odds of reporting fair or poor health at least once between ages 18 and 30 compared to (predominantly white) respondents who resided, on average, in the least disadvantaged neighborhood quintile ($\exp((5-1)\text{x}0.20)=2.23$).

Figure 2.2 presents a visual representation of the effects of such neighborhood inequality on health. It depicts predicted probabilities and their 95 percent confidence intervals of reporting fair or poor health in early adulthood by the average level of neighborhood disadvantage to which respondents were exposed as children and youth. All other baseline covariates are set to their sample means. The graph displays how the probability of reporting an incidence of fair or poor health in early adulthood would be expected to increase if respondents had been exposed, on average, to more disadvantaged neighborhoods throughout childhood and adolescence

(or vice versa). Estimates are graphed separately for nonwhites and whites for illustrative purposes only. However, because racial differences in the neighborhood-health effect were not statistically significant, only results for the combined (non-race-stratified) sample are described below. As the figure indicates, if respondents had been persistently exposed to the least disadvantaged (1st) quintile of neighborhoods from ages one through 17, only about one in 10 (13%) would have reported an incidence of fair or poor health in early adulthood. Conversely, if the same respondents had been exposed, on average, to the fourth or fifth quintiles of neighborhood disadvantage as children and youth, approximately twice as many – that is, one in five (21%) or one in four (24%), respectively – would have reported an incidence of fair or poor health between ages 18 and 30. Given the links between self-rated health and concurrent and future physical and mental health care need and utilization as well as comorbid conditions, such an increased incidence of fair/poor health among relatively young adults may represent a substantial added burden for this country's most impoverished, under-resourced, and racially segregated communities as well as an important feedback mechanism through which neighborhood inequality is perpetuated.

2.5 Discussion and Conclusions

This study examined the effect of growing up in neighborhoods characterized by varying levels of disadvantage on self-rated health in early adulthood utilizing the 1970 to 2011 waves of the PSID merged with census data on respondents' neighborhoods. Consistent with previous research, findings show that neighborhood disadvantage, defined by the spatial clustering of poverty, unemployment, female-headed households, public assistance receipt, and educational and occupational marginalization, is inversely associated with self-rated health. More notably, this study is among the first to document that more sustained exposure to neighborhood disadvantage throughout the entire child and adolescent life course has negative implications that can manifest in early adulthood as fair or poor health. Whereas the majority of prior studies in this area characterize the residential environment only once or over just a short window of observation, the estimates presented in this study are based on yearly measurements of respondents' neighborhoods from birth through age 17 as well as statistical methods that account for dynamic individual- and household-level factors known to be predictive of future health but also related to the sorting of families into and out of neighborhoods over time. Such estimates suggest more specifically that the effect of residing, on average, in neighborhood disadvantage quintile *q* versus *q*-1 (a less disadvantaged neighborhood) from ages one through

17 is associated with a 27 percent increase in the odds of reporting fair or poor health at least once between ages 18 and 30. This means that respondents who are continuously exposed to the most disadvantaged neighborhood quintile have 27 percent greater odds of reporting fair or poor health in early adulthood compared to those who grow up, on average, in the fourth quintile, 62 percent greater odds compared to those in the third quintile, about two times greater odds than those in the second quintile, and over 2.5 times greater odds than those who were continuously exposed to the least disadvantaged neighborhood quintile.

While these results indicate that prolonged exposure to neighborhood disadvantage during childhood and adolescence may be universally deleterious to young adult health, the totality of the findings in this study further suggest that the adverse effects of neighborhood disadvantage are likely to be more concentrated among African American children and youth. In keeping with other work on racial residential segregation and neighborhood stratification, this study found that exposure to neighborhood disadvantage is strikingly more common, both at any point in time as well as cumulatively from birth through age 17, among African American compared to white children and youth. African American children and youth are not only more likely than their white counterparts to be born into neighborhoods characterized by higher levels of disadvantage and thus fewer social and structural resources and opportunities, but to remain in similar types of health-compromising residential environments for the entirety of their pre-adult years. Simple regression analyses further show that the nearly two-fold disparity between African American and white young adults in the crude incidence of fair or poor health is reduced by about 75 percent and the coefficient for race is no longer statistically significant once a measure of cumulative exposure to neighborhood disadvantage during childhood and adolescence is included in the model (results not shown). The coefficient for cumulative neighborhood disadvantage remains statistically significant in all models, even when adjusting for a host of time-invariant and time-varying individual- and household-level factors in the final marginal structural model. This suggests that the separate and unequal neighborhood environments in which African American versus white children tend to live, learn, and grow may play a critical role in producing and perpetuating black/white disparities in self-rated health in early adulthood (and beyond).

Although this study uses panel data and unique statistical methods to address some of the most common challenges in neighborhood effects research (namely, reverse causation and selection bias), the results should be considered in the context of several remaining limitations. First, the absence of completely “tracted” land in 1970 and 1980 resulted in missing census data on neighborhood characteristics and necessitated imputation,

which may have led to some misclassification of neighborhood disadvantage, especially in nonmetropolitan areas. However, the two-fold multiple imputation algorithm used to model this information was based on an extensive set of individual- and household-level factors known to be strongly related to the neighborhood disadvantage characteristics assessed in this study as well as census data at adjacent time periods. Since the entire U.S. was tracted by 1990, studies in the near future will be able to examine a similar length of the life course, from birth through age 30, without having to include decades in which information on census tracts may be missing by default. Second, as with most longitudinal designs, sample attrition in this study is relatively high. However, the censoring weights used in conjunction with the treatment weights to generate the "pseudo-population" to which the final marginal structural model is fit, up (or down) weight those individuals who are more (or less) likely to drop out of the sample at each wave, effectively randomizing sample attrition across survey waves. Third, although there appears to be sufficient overlap in the race-specific distributions of cumulative exposure to neighborhood disadvantage to generate stable inverse probability of treatment weights and thus to satisfy the positivity assumption on which the final analysis is based, it would be ideal (although perhaps not realistic) to find a sample in which neighborhood stratification by race is less pronounced in order to further disentangle the mechanisms behind the relationships among race, place, and health. Fourth, given the historical timing of this study, the sample is limited to African American and white respondents. Future research examining health disparities as a function of cumulative neighborhood effects among Asian and Latino populations is encouraged. Finally, this study did not assess the mechanisms thought to help explain how neighborhood-health effects transpire but rather the broader social and structural environments thought to engender such processes. Longitudinal research able to more explicitly measure these potential mechanisms, including, for example, network ties, collective efficacy, institutional resources, and environmental toxins, would be enlightening. Absent such measures, which may be particularly difficult to find in or attach to existing longitudinal datasets, future research could examine the adult health effects of timing of exposure to neighborhood disadvantage in order to shed more light on some of these same processes. For example, if the magnitude of the neighborhood effect is larger during early childhood compared to late childhood or adolescence, then institutional resources such as high-quality childcare or social network ties that help lessen the challenges associated with having young children may be the primary drivers of the neighborhood-health effect. On the other hand, if adolescence emerges as a sensitive period, then

employment or internship opportunities, collective socialization, and informal social control may be the more relevant mechanisms.

Nonetheless, findings from the present study add to the growing body of evidence suggesting that place-based, developmentally-appropriate, and ongoing investments in the social, economic, institutional, and infrastructural aspects of under-resourced neighborhoods and neighborhoods of color can have benefits for health and health equity that extend across the life course. On the one hand, a considerable body of prior scholarship has detailed the so-called “long arm” of early-life individual- and family-level disadvantage on health in older ages, including the effects of family poverty in childhood [134] and early-life adversity and toxic stress [77, 135, 136] on later-life physical and mental well-being and mortality. On the other hand, a related but often separate body of extensive research has further shown that neighborhood-level disadvantage is associated with health, at least in the cross-section. This study brings these two literatures into more explicit dialogue, empirically documenting the importance of earlier-life neighborhood circumstances for self-rated health in early adulthood. That is, adverse individual and familial experiences during childhood and adolescence matter for future health, but so do related social and structural adversities in the broader neighborhood environment. Health-related policies aimed at changing individual behaviors are likely of limited utility when the collective characteristics of neighborhoods, including concentrated poverty, joblessness, crime and violence, residential instability, and socioeconomic disinvestment, make such choices more difficult, if not impossible, to envision the benefits of or to enact and sustain.

Moreover, in order to cultivate a more healthful and thus productive cadre of adult citizens, policies must strive to generate durable, place-based investments in children and youth, a so-called “continuum of resources and support” [29], which last across the life course. Whereas prior neighborhood effects research based on cross-sectional data has often concluded with similar sentiments, the implementation of associated health practice and policy changes has generally been hampered by challenges to causal inference. Using longitudinal data and a statistical method that attempts to model the full data distribution (i.e., estimating for each respondent their level of self-rated health under each possible level of neighborhood disadvantage rather than just their actual observed levels), this study provides among the strongest evidence to date for the consequences of prolonged exposure to neighborhood disadvantage throughout childhood and adolescence on future (young adult) health, especially among African American children and youth for whom such exposures tend to be more common and more persistent.

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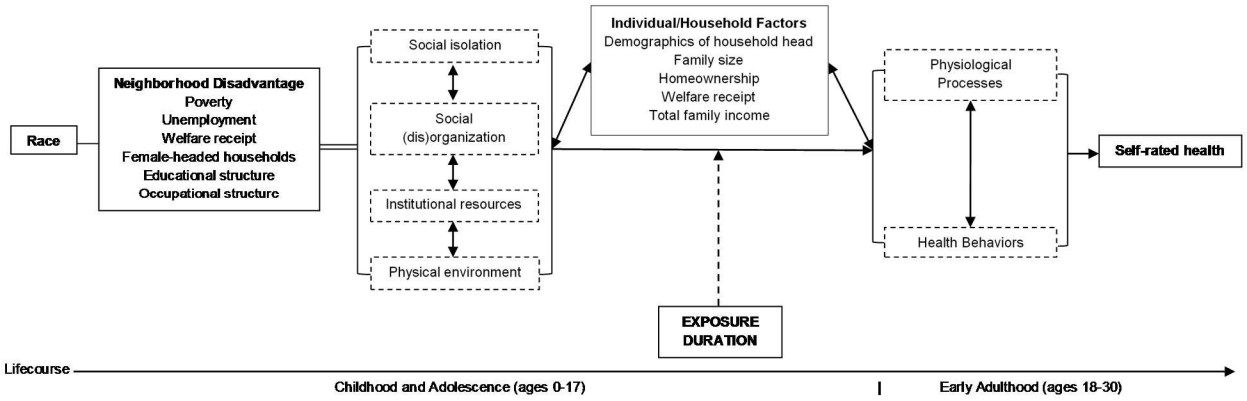

Figure 2.1. Conceptual model of the effects of exposure to neighborhood disadvantage throughout childhood and adolescence on self-rated health in early adulthood

Table 2.1. Time-invariant sample characteristics

	Nonwhite (n=634)	White (n=1123)
Self-rated health in early adulthood, percent		
Fair/poor health at least once between ages 18-30	26.66	14.69
Excellent/very good/good health	73.34	85.31
Gender, percent		
Female	56.78	52.09
Male	43.22	47.91
Birthweight, percent		
Less than 88 ounces	10.73	5.97
88 ounces or more	89.27	94.03
Mother's marital status at birth, percent		
Unmarried	46.69	6.86
Married	53.31	93.14
Household head's educational attainment at birth, percent		
Less than high school	55.21	19.15
High school graduate	31.86	37.93
At least some college	12.93	42.92
Mother's age at birth, mean (SD)	22.67 (5.17)	25.45 (4.95)

Note: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one self-rated health question in early adulthood (first of 10 imputation datasets).

Table 2.2. Time-varying sample characteristics

	Nonwhite (n=634)			White (n=1123)		
	Age 1	Age 10	Age 17	Age 1	Age 10	Age 17
Neighborhood disadvantage quintile, percent						
1st quintile (least disadvantaged)	2.05	3.47	5.52	14.43	18.97	21.73
2nd quintile	2.84	5.52	8.04	20.75	18.79	21.73
3rd quintile	6.15	6.78	9.78	22.97	25.73	25.65
4th quintile	15.14	16.72	17.67	26.27	24.13	21.10
5th quintile (most disadvantaged)	73.82	67.51	58.99	15.58	12.38	9.80
Household head's marital status, percent						
Unmarried	34.07	43.38	49.53	4.99	11.40	17.10
Married	65.93	56.62	50.47	95.01	88.60	82.90
Household head's employment status, percent						
Unemployed	28.86	33.60	29.81	7.93	10.15	9.17
Employed	71.14	66.40	70.19	92.07	89.85	90.83
Public assistance (AFDC) receipt, percent						
Received AFDC	19.40	26.34	14.98	3.65	4.01	1.69
Did not receive AFDC	80.60	73.66	85.02	96.35	95.99	98.31
Homeownership, percent						
Does not own home	64.98	57.26	49.05	38.74	23.06	19.95
Owns home	35.02	42.74	50.95	61.26	76.94	80.05
Household income in (1985) $1,000s, mean (SD)	15.41 (11.01)	16.53 (13.83)	18.06 (15.77)	29.01 (16.85)	25.69 (24.64)	29.87 (31.82)
Household head's work hours/week, mean (SD)	27.92 (17.99)	27.73 (19.90)	29.59 (20.44)	42.10 (15.02)	41.51 (15.39)	41.12 (15.75)
Family size, mean (SD)	4.96 (2.64)	4.74 (1.67)	4.54 (1.62)	3.91 (1.18)	4.49 (1.04)	4.09 (1.15)

Note: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one self-rated health question in early adulthood (first of 10 imputation datasets)

Table 2.3. Cumulative exposure to neighborhood disadvantage from ages one through 17

	Nonwhite (n=634)	White (n=1123)
Average neighborhood disadvantage quintile, percent		
1.0 to 1.4 (least disadvantaged)	1.10	12.56
1.5 to 2.4	3.31	22.62
2.5 to 3.4	8.36	32.06
3.5 to 4.4	26.34	26.18
4.5 to 5.0 (most disadvantaged)	60.88	6.59
Proportion time in most disadvantaged quintile		
Mean percent	67.06	12.89
Mean years	11.4	2.19

Note: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one self-rated health question in early adulthood (first of 10 imputation datasets).

Table 2.4. Stabilized treatment, censoring, and final weights (n=1,757)

Weight	Mean	SD	Min	1%	99%	Max
Stabilized treatment weight (TW)	1.02	0.49	0.08	0.35	2.56	12.52
Stabilized censoring weight (CW)	1.04	0.43	0.33	0.52	2.80	7.22
TW x CW	1.05	0.65	0.11	0.31	3.64	9.41

Table 2.5. Effects of cumulative exposure to NH disadvantage from ages 0-17 on self-rated fair/poor health in early adulthood, n=1,757 (log odds ratios)

	Model 1			Model 2			Model 3			Model 4		
	Coef.	SE		Coef.	SE		Coef.	SE		Coef.	SE	
Average NH disadvantage				0.38	0.07	***	0.38	0.08	***	0.20	0.10	**
Race (white)												
Nonwhite	0.75	0.12	***	0.23	0.15		0.26	0.68		0.34	0.20	*
NH disadvantage X race							-0.01	0.16				
Gender (male)												
Female										0.29	0.15	*
Birthweight (88 ounces or more)												
Less than 88 ounces										-0.56	0.29	*
Mother's marital status at birth (married)												
Unmarried										-0.02	0.29	
HH's education at birth (less than HS)												
High school graduate										-0.49	0.18	***
At least some college										-0.66	0.25	***
Mother's age at birth										0.03	0.02	*
NH disadvantage at birth										0.08	0.09	
HH's marital status at birth (married)												
Unmarried										0.33	0.31	
HH's employment status at birth (employed)												
Unemployed										0.13	0.26	
Homeownership at birth (owns home)												
Does not own home										0.17	0.16	
Family size at birth										-0.04	0.04	
Public assistance (AFDC) receipt at birth (no AFDC)												
Received AFDC										0.04	0.30	
Household income at birth										0.21	0.13	
HH's work hours per week at birth										0.00	0.01	
Year born (1970-72)												
1973-1975										-0.14	0.21	
1976-1978										0.06	0.20	
1979-1980										0.11	0.23	

Notes: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one self-rated health question in early adulthood; Coefficients are combined estimates from 10 multiple imputation datasets; NH=Neighborhood; HH=Household head; *p<0.10; **p<0.05; ***p<0.01

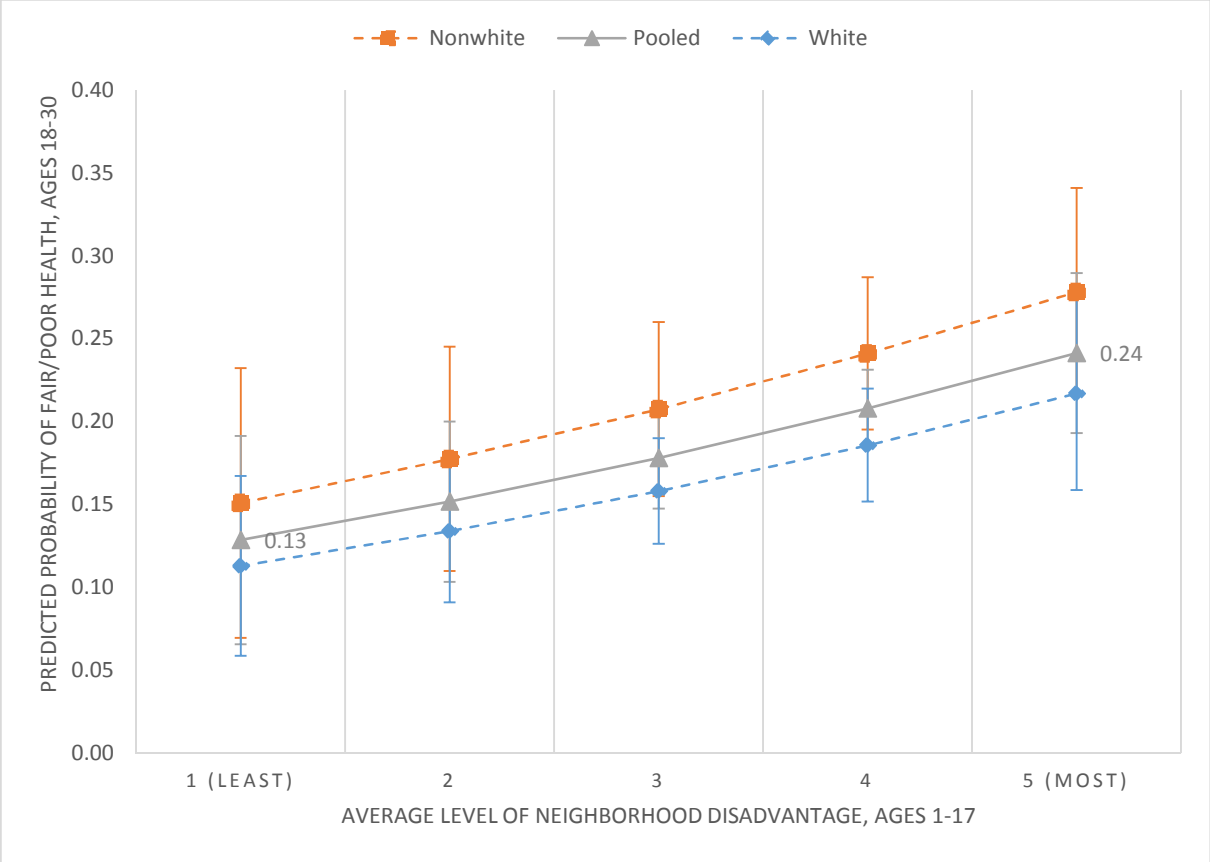

Figure 2.2. Predicted probability of self-rated fair or poor health in early adulthood by average level of neighborhood disadvantage experienced during childhood and adolescence

APPENDIX 2.A

Table 2.6. Component weights and correlations from principal components analysis of seven census tract items used to generate a composite index of neighborhood disadvantage, 1970-2000

Variable	1st PC Weight
Percent poverty	0.403
Percent unemployed	0.374
Percent receiving public assistance	0.413
Percent female-headed households	0.341
Percent without high school diploma	0.385
Percent with bachelors/graduate degree	-0.352
Percent in managerial/professional occupations	-0.372
Component variance	4.402
Proportion total variance explained	0.629

Chapter 3. THE WEIGHT OF A NEIGHBORHOOD: TEMPORAL EFFECTS OF CHILD AND ADOLESCENT EXPOSURE TO NEIGHBORHOOD DISADVANTAGE ON YOUNG ADULT OBESITY

3.1 INTRODUCTION

There is significant variation among neighborhoods in rates of obesity, defined in adulthood as having a body mass index (BMI) greater than or equal to 30 kg/m^2. Prior evidence suggests that residents of under-resourced neighborhoods characterized by the relative absence of healthy food stores, a preponderance of fast food and alcohol outlets, and systemic constraints on physical activity and social interaction tend to have higher BMI [1-9]. In addition, numerous studies have documented that exposure to neighborhood disadvantage is unequally distributed both in the population and across the life course. African Americans, in particular, are not only more likely than statistically comparable whites to ever reside in areas characterized by high levels of social and structural adversity, but are also more likely to do so for repeated or prolonged periods of time [10-22]. These findings point to the importance in neighborhood effects research of characterizing if, as well as when and for how long, residential exposures occur, especially when assessing their contribution to racial disparities in overweight/obesity and other health-related outcomes. Until recently, however, the majority of scholarship in this area measured neighborhood characteristics only once or over just a short window of observation, conflating persons who were recently exposed with those who have – and in the case of some groups, are more likely to have – experienced more sustained residential adversity [23]. Such a conceptualization is inconsistent with most theories of neighborhood effects, which tend to specify mechanisms that are sensitive to the duration and timing of exposure [17, 18, 24-27], as well as with a life course perspective in which experiences earlier in life are posited to have formative and enduring impacts on future outcomes, even when controlling for more contemporaneous determinants [28, 29].

Using the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID) merged with census data on respondents' neighborhoods, this study estimates marginal structural models with inverse probability of treatment and censoring weights to investigate the effects of duration and timing of exposure to varying levels of neighborhood disadvantage from birth through age 17 on obesity incidence in early adulthood (ages 18-30), as well as racial disparities therein. To capture both continuity and change in residential exposures throughout childhood and adolescence – children and youth can move in and out of different neighborhoods or remain in

areas that may or may not change around them – levels of neighborhood disadvantage are characterized once per year, every year during this time. In addition, by employing a statistical method that accounts for selection bias without controlling away neighborhood effects that may operate indirectly through the same individual-level covariates that have been associated with residential mobility, this study provides a more comprehensive assessment of the effects on young adult BMI of exposures to neighborhood disadvantage that occur during different stages of development and accrue across the entire child and adolescent life course.

3.2 BACKGROUND

The increased prevalence and associated health consequences of overweight and obesity in the United States (U.S.) have been called among the most burdensome public health issues facing the nation today [30]. According to recent age-adjusted estimates, more than two-thirds (68.5%) of U.S. adults aged 20 years and older are overweight and over one-third (34.9%) are obese [31]. Prior research further suggests that the causes and mechanisms of adult overweight and obesity may be initiated during childhood and adolescence, and that there is a strong tendency for obesity to persist from earlier to later stages of the life course [32-35]. While interventions targeting individual dietary and exercise habits retain popular appeal, without regard to factors in the broader social and structural environment, such efforts have been shown to be largely ineffective at achieving sustained behavioral change [36, 37]. There are at least two important themes that emerge from the above findings. First, given the persistence of childhood overweight and obesity across the life course, waiting until adulthood to address such concerns may make change particularly difficult. Second, as the Institute of Medicine has concluded more generally, “it is unreasonable to expect that people will change their behavior easily when so many forces in the social, cultural, and physical environment conspire against such change” [38]. However, even as the evidence points to the importance of both timing and context for effectively preventing and reducing the health-related consequences of overweight and obesity, the majority of extant research in this area has been cross-sectional, focused on adulthood, and generally conducted in small, non-generalizable geographic areas [39, 40]. This makes it particularly difficult to account for the evolving and interrelated nature of individual and neighborhood characteristics over the life course, as well as for the movement of individuals who already have physically active lifestyles and healthy diets into neighborhoods with characteristics that support such behaviors (i.e., selection bias). As a result, the development and implementation of associated health promotion interventions have too often been hampered by challenges to

causal inference and most large-scale efforts continue to target the characteristics of people rather than the characteristics of places.

This study adds to the small but growing body of longitudinal neighborhood-level obesity research [39-42]. With longitudinal data it is possible to incorporate individual and neighborhood characteristics as dynamic rather than static influences on health across various stages of the life course. Moreover, this study employs a statistical approach that explicitly accounts for such time-varying phenomena, allowing individual-level characteristics to moderate the relationship between neighborhoods and health while also adjusting for potential confounding due to selection bias at each wave of data collection. These findings, therefore, provide among the strongest evidence to date for the adverse effects on young adult BMI of more prolonged exposure to neighborhood disadvantage throughout the child and adolescent life course as well as for the sensitivity of such effects to the developmental timing of exposure.

3.2.1 *Mechanisms Linking Neighborhood Disadvantage and Obesity*

Neighborhood disadvantage is posited to influence the more proximal behavioral and biological determinants of BMI through various interrelated social and structural processes. Prominent among these are (1) lack of access to institutional resources conducive to healthy eating and physical activity, such as affordable, high-quality food stores and parks or green space; (2) low levels of collective efficacy and informal social control which may increase fear and discourage outdoor play as well as other forms of both recreational and non-recreational physical activity; and (3) chronic neighborhood stressors, such as concentrated poverty and endemic joblessness, which may lead to unhealthy eating (e.g., as a coping strategy) or affect body composition more directly through physiological stress processes [43, 44]. Within such a framework, choices about where to live as well as what to eat and whether or not to exercise are nonetheless framed as functions of individual- and household-level characteristics, such as age, sex, and socioeconomic status. However, these individual- and household-level characteristics are further conceptualized as operating within particular neighborhood contexts characterized by the racial-spatial clustering of salutogenic or obesogenic social and structural processes that fundamentally shape individual behavior and biological risk.

Prior research documents, for example, that supermarkets have sharply declined in low-income, predominantly nonwhite neighborhoods, forcing many residents (given additional constraints on transportation) to rely on convenience and other small stores with more limited, calorie-dense, and often higher priced food selections [45, 46]. Several researchers have also linked neighborhood disadvantage and disorder to

perceptions of danger, restrictions on outdoor play, and subsequent increases in body mass among children and adolescents [40]. Other work has similarly found that access to a safe rather than any play space is the more relevant determinant of physical activity among children and youth in high-poverty neighborhoods [47, 48]. In addition, an emerging body of scholarship further suggests that stressful conditions in one's neighborhood may affect BMI more directly by eliciting a two-stage stress response (the "fight or flight" response) in which the body is first supplied with energy through a surge of glucose and the breakdown and release of fatty acids. In stage two, cortisol is released into the bloodstream to convert food into stored fats and to induce hunger as a means of compensating for the body's prior energy loss [40]. Chronic (prolonged or repeated) activation of this two-stage response is considered particularly harmful because it is the most likely to result in long-term or permanent changes in the physiological as well as emotional and behavioral responses that influence the risk of obesity, particularly central obesity [40, 49-51].

3.2.2 *Duration and Timing of Neighborhood Exposures*

Despite the predominance of cross-sectional studies in which neighborhood effects on BMI are assumed, at least implicitly, to be instantaneous and equivalent across the life course, most research acknowledges that long-term and cumulative effects are possible, if not more probable. For example, neighborhood gardens and local parks or sports fields have been shown to encourage healthier eating and more physical activity among children and youth [46, 52-54]. However, more sustained exposure periods may be necessary for young people to learn the skills associated with and to internalize preferences for healthier food and more active lifestyles that can then be sustained throughout the life course [39]. Similarly, exposure to disadvantaged neighborhoods for extended periods of time is posited to have a greater influence on the development of overweight and obesity if neighborhood pathways involve processes of collective socialization related, for example, to the quality of the built environment or the availability of institutional resources. That is, children and youth with only short-term exposure to neighborhoods lacking ready access to fresh, healthy, and affordable food may be able to overcome temporary nutritional deficits if they reside in so-called "food oases" otherwise. On the other hand, poor eating and exercise patterns associated with the absence of healthy food stores, a multitude of fast food outlets, and structural constraints on physical activity and social interaction will likely compound over more prolonged exposure periods [40, 55, 56].

In addition to cumulative effects, prior research suggests that adolescence may be a sensitive period for the development of overweight and obesity [40, 41, 57, 58] as well as for the effects of neighborhood

characteristics on health-related outcomes more generally [59]. Adolescence is a time of rapid growth and physiological change. It is also marked by an increasing need for autonomy and expanding social interactions. The residential neighborhood, in turn, often constitutes both the physical and social space in which youth spend a large part of their increasing out-of-home time [60]. These physical conditions and social experiences not only structure opportunities to make healthy choices but also shape norms, values, attitudes, knowledge, and behavioral tendencies related to food and exercise [61]. For example, as adolescents are increasingly able, allowed, and even required to manage some or all of their own meals, the neighborhood food environment may become particularly influential for establishing life-long eating behaviors. A similar phenomenon may also occur for physical activity such that the presence and safety of neighborhood parks and recreational settings during the increasingly independent and active periods of the adolescent life course may have lasting implications for the level and intensity of exercise in adulthood [62].

While there is an emerging body of longitudinal research in this area that has greatly enhanced our confidence in and understanding of the relationship between neighborhood conditions and BMI, additional work is needed to address important outstanding issues. For example, several recent studies have examined neighborhood effects on BMI trajectories from childhood through either adolescence or young adulthood [40, 42]. These studies generally find that children and youth who reside in more disadvantaged neighborhoods not only have higher BMI at baseline, but they also gain body mass at a faster rate over time compared to their counterparts in more advantaged areas. These results have helped define the development of obesity as an age-graded process sensitive to the influences of neighborhood conditions, over and above those of individual- and household-level characteristics. However, they are also subject to complex and dynamic sources of bias that may conceivably be used to undermine their implications for place-based public health policy and practice efforts. In particular, while respondents' heights and weights are assessed at several points over the life course, the majority of prior studies measure neighborhood characteristics along with individual- and household-level control variables at only one time point, usually at the start of data collection or respondents' year of birth. This practice implicitly ignores how such factors and the relationships among them change over time, resulting in neighborhood effect estimates that do not adequately account for residential selection and are largely insensitive to differences in the duration and timing of neighborhood exposures.

3.2.3 *Objectives*

Accordingly, the central aim of this study is to investigate the temporal dimensions of the neighborhood-obesity relationship from birth through early adulthood with a particular emphasis on whether racial differences in patterns of exposure to neighborhood disadvantage help to explain racial disparities in obesity incidence. This includes the following three more specific objectives:

(1) Describe overall and race-specific patterns of exposure to varying levels of neighborhood disadvantage throughout the entire child and adolescent life course as well as during distinct developmental stages;

(2) Estimate the impact of more prolonged exposure to neighborhood disadvantage throughout childhood and adolescence on obesity incidence in young adulthood; and

(3) Compare the effects on young adult obesity of exposure to neighborhood disadvantage that occurs during distinct developmental stages in early childhood, late childhood, and adolescence.

3.3 DATA AND METHODS

Data for this study are drawn from the Panel Study of Income Dynamics (PSID), produced and distributed by the University of Michigan's Institute for Social Research and funded by the National Institutes of Health (NIH) and the National Science Foundation (NSF). The PSID is a large, longitudinal survey of U.S. residents and their families conducted annually between 1968 and 1997 and every two years thereafter. The PSID has several strengths that make it particularly well-suited to the objectives of this study. First, the dataset contains an oversample of low-income families, as well as a wealth of information on a variety of potential individual- and household-level health-risk and -protective factors, including employment, income, education, marriage, social program participation, and housing characteristics. Second, the residential location of individual respondents at each interview can be linked to their corresponding census tract identifiers using the PSID's supplemental, restricted-use Geospatial Match Files. These identifiers make it possible to characterize neighborhood disadvantage for prolonged intervals, as well as to account for the potential mobility of PSID respondents into and out of different neighborhoods. Third, the longitudinal design of the PSID makes it possible to adjust for the temporal sequencing and compounding influence of both individual, household, and neighborhood conditions across time.

3.3.1 *Sample Selection*

The analytic sample for this study consists of the 4,523 individuals born into PSID family units between 1970 and 1980.[12] Restricting the sample in this manner ensures that respondents are old enough by 2011 to have been included in the survey from birth through at least age 30. Given the focus on the duration and timing of exposure to adverse neighborhood conditions throughout the entire child and adolescent life course, respondents are dropped if they are not continuously present for every year from ages zero to 17 or if they do not respond to any questions about height and weight in young adulthood. The final sample includes 1,522 individuals: 524 are classified as nonwhite, of which over 95 percent are African American, and 998 are classified as white. Although a considerable share of the original sample are excluded from the final analyses, these respondents contribute information to the study until they are lost to follow-up. Adjustment for potential nonrandom attrition using censoring weights will be discussed below.

3.3.2 *Dependent Variable*

Obesity is determined using self-reported weight and height to calculate BMI, the ratio of weight in kilograms (kg) to height in meters squared (m^2). BMI is a reliable and widely accepted measure of excess fatty tissue at the population level, and for most people, it is highly correlated with direct measures of body fat such as underwater weighing and dual energy x-ray absorptiometry (DXA) [63-66]. Weight and height have been assessed in the PSID at every wave since 1999 using the questions, *"How much do you weigh?"* and *"How tall are you?"* Consistent with Centers for Disease Control and Prevention (CDC) standards and prior research, obesity is defined as having a BMI greater than or equal to 30 kg/m^2. Given this study's focus on the incidence of obesity in young adulthood, the primary outcome of interest is *any* report of weight and height amounting to a BMI greater than or equal to 30 kg/m^2 between ages 18 and 30.

3.3.3 *Main Independent Variable*

Similar to most prior research in this area, census tracts are used to approximate neighborhood boundaries. Although there are well-known limitations to this operational definition [67], there is broad consensus that census data at the tract level not only provide convenient access to considerable information over extensive time periods, but they also serve as a reasonable proxy for, or are at least highly correlated

[12] Sample members were asked for their year of birth at multiple survey waves. To address potential discrepancies in individual responses across waves, year of birth was determined as the modal response. For the few cases in which respondents never answered the year of birth question but did respond to question(s) about their age, age was used to determine their year of birth (i.e., interview year minus age at interview). Again, if this approach produced discrepancies across waves, the modal response was used. There were only 110 of 73,251 (<1%) respondents in the total PSID sample for whom year of birth could not be determined via the methods described above.

with, the "true" causally relevant definition of a neighborhood [6, 68-70]. Information on census tracts during the years in which respondents are ages zero to 17, including 1970, 1980, 1990, and 2000, comes from the Neighborhood Change Database (NCDB) in which data for all four decades has been normalized to 2000 tract boundaries and can therefore be compared across years without having to adjust for potential changes in boundary definitions over time. Data for intercensal years are imputed using linear interpolation.[13]

Neighborhood disadvantage is operationalized in terms of seven census tract items thought to engender the neighborhood-level social and structural processes that have been posited to influence the more proximal determinants of obesity. These items include: (1) proportion of residents below the poverty line; (2) proportion of residents (age 16 and older) in the civilian labor force and unemployed; (3) proportion of households with public assistance income; (4) proportion of households with children that are female-headed; (5) proportion of residents (age 25 and older) with less than a high school diploma; (6) proportion of residents (age 25 and older) with a bachelors or graduate/professional degree; and (7) proportion of residents (age 16 and older) employed in managerial or professional/technical occupations. Following Wodtke and colleagues [24] and others [71-74], principal components analysis is used to transform these seven items into a composite index reflected by the first principal component.[14] Each item's loading on this component is used to weight its contribution to a neighborhood disadvantage score which is calculated for every census tract at every year. To facilitate the implementation of the analysis below, the resulting scores are then divided into quintiles ranging from the least (level 1) to the most (level 5) disadvantaged neighborhoods based on the distribution of all tract-year scores between 1970 and 2000. This information is merged with individual-level data on sample members in the PSID such that each respondent ends up with 18 different measurements of the level of neighborhood disadvantage to which they were exposed, one for every year from birth through age 17.

For the duration component of this investigation, a cumulative measure of exposure to neighborhood disadvantage throughout childhood and adolescence is calculated as the average of the 17 neighborhood

[13] If decennial census tract data in 1970 and/or 1980 was missing (likely due to as yet untracted areas) and linear interpolation was impossible, data from 1980, 1990, and/or 2000 was used to extrapolate values for up to five years prior to the most recent value (e.g., if data in 1970 was missing, the linear interpolation from 1980 to 1990 was extended to 1975).

[14] Although principal components analysis forms as many independent linear combinations as there are variables, only the first principal component, which accounts for the largest possible proportion of the total variability in the component measures (about 63 percent, in this case), was retained. Item loadings on this first principal component ranged from 0.341 for the proportion of female-headed households to 0.413 for the proportion of households receiving public assistance income. See Appendix 2.A for more details.

disadvantage quintiles experienced by respondents from ages one through 17.[15] Likewise, for the timing component of the study, three separate measures of timing-specific exposure to neighborhood disadvantage during early childhood, late childhood, and adolescence are calculated as the average of neighborhood disadvantage quintiles experienced by respondents from ages one to five, from ages six to 11, and from ages 12 to 17, respectively. As described below, neighborhood disadvantage at birth is not included in these calculations but rather as part of a vector of baseline covariates.

3.3.4 *Covariates*

A large number of individual- and household-level variables are included in analyses as either time-invariant (constant or measured only at birth) or time-varying. Time-invariant covariates include a respondent's race (1=nonwhite; 0=white), gender (1=female; 0=male), birthweight (1=less than 88 ounces; 0=88 ounces or more), mother's age at birth, mother's marital status at birth (1=unmarried; 0=married), and household head's educational attainment at birth (1=less than high school; 2=high school graduate; 3=at least some college)[16], all of which have been associated in previous studies with both neighborhood residence and individual health-risk and protective factors. Time-varying covariates, measured for every respondent at each wave between ages zero and 17, include household head's marital status (1=unmarried; 0=married), employment status (1=unemployed; 0=employed), and work hours, as well as family size, homeownership (1=does not own home; 0=owns home), public assistance receipt (1=yes; 0=no), and total household income, standardized using the Consumer Price Index (CPI-U) to 1985 dollars.

3.3.5 *Statistical Analysis*

This study employs marginal structural models (MSMs) in which the parameters are estimated using inverse probability of treatment (IPT) weights. This approach and its utility for longitudinal neighborhood effects research have been described in detail elsewhere [24, 75-78]. In short, it attempts to mimic an experimental design using observational data. More specifically, because respondents in this study were not in fact randomly assigned to reside in a particular quintile of neighborhood disadvantage at each year of follow-up, it is possible

[15] Because the same average value can be achieved for very different patterns of exposure to neighborhood disadvantage (e.g., consistently moderate levels may produce the same average value as a mixture of high and low), a preliminary latent class growth analysis was also performed to examine exposure trajectories throughout childhood and adolescence. Results showed that, in general, children and youth in this study do not experience much upward or downward mobility in the level of neighborhood disadvantage to which they are exposed between ages zero and 17 (more details available upon request). These findings suggest that an average measure of cumulative exposure to neighborhood disadvantage may therefore be a reasonable reflection of the experience of children and youth in this study.

[16] The household head's educational attainment is treated as time-invariant and measured at respondents' year of birth because the PSID does not measure parental education at regular intervals. If data at year of birth was missing, the most recent subsequent measurement was used, which for the vast majority of respondents was within 3 years of birth.

that what may seem like a neighborhood effect is actually due to the individual- and household-level characteristics of the residents who are more (or less) likely to live in different neighborhoods. Simply controlling for such confounding variables, however, raises a number of concerns in the context of longitudinal studies. First, values on these variables may change over time – that is, they are time-dependent or time-varying. Second, such individual- and household-level covariates may operate not only as confounders of the neighborhood-obesity relationship, but also as mediators (e.g., when neighborhood conditions affect job prospects and other resources which in turn influence opportunities to eat better and move more [79]). Controlling for these characteristics (or, more likely, the average of all the values on each of these characteristics over the follow-up period) would remove from the model the mediated (but still relevant) pathway through which neighborhood effects may transpire.

The MSM approach with IPT estimators uses weighting to produce a pseudo-population in which random assignment of exposure to neighborhood disadvantage is simulated. More specifically, ordinal logistic regression based on all person-year observations is used to estimate each respondent's probability of being exposed to each of the neighborhood disadvantage quintiles (contrary to fact for four of the five quintiles) at each year, conditional on their neighborhood conditions in the prior year, baseline or time-invariant covariates (including neighborhood conditions at year of birth), and both prior year and concurrent time-varying covariates.[17] Respondents are then assigned a series of IPT weights based on the inverse (or reciprocal) of the probability that they were exposed to their actual (observed) neighborhood disadvantage quintile at each year of follow-up. In this way, proportionally more (or less) weight is given to those respondents whose prior time-varying covariates are underrepresented (or overrepresented) in the neighborhood disadvantage quintile to which they were actually exposed at each year. That is, the IPT weights ensure that the values of all covariates included in their construction are balanced in expectation across the five levels of neighborhood disadvantage at each year.

In practice, IPT weights can be highly variable. To increase efficiency and obtain narrower confidence intervals around the subsequent neighborhood effect estimate, the IPT weights for each respondent at each year are stabilized by multiplying each one by the same probability as was used to generate it, except only

[17] Interactions between prior year and concurrent measures of both marital status and employment status are also included to account for possible effects of a recent divorce or job loss on mobility into or out of neighborhoods characterized by different levels of disadvantage.

baseline covariates and prior year neighborhood conditions are included as regressors (i.e., time-varying covariates are excluded). In other words, these reduced-form estimates of the likelihood of exposure to one's actual neighborhood disadvantage quintile at each year become the numerators for the original IPT weights described above. The stabilized IPT weights for each respondent at each year are then multiplied together to generate a single value representing each respondent's overall contribution to the pseudo-population. In such a pseudo-population, obesity incidence in young adulthood can be regressed on either cumulative or timing-specific measures of exposure to neighborhood disadvantage using a conventional logistic regression model that does not include the time-varying covariates as controls.[18] The impact of these covariates as potential confounders is already accounted for through the weighting process, while their role as potential mediators remains intact. As others have described, this final model is termed "marginal" because it models the marginal (rather than the joint) distribution of potential outcomes and "structural" because its coefficients represent causal effects assuming no unmeasured confounding in the estimation of the stabilized IPT weights [77].

3.3.6 *Missing Data and Sample Attrition*

Missing data on all independent variables are multiply imputed using the two-fold fully conditional specification algorithm in STATA v.13.1 [80]. The two-fold fully conditional specification algorithm was designed to impute missing data in longitudinal studies. Whereas conventional multiple imputation methods ignore the longitudinal and dynamic structure of panel data and are difficult to implement in large databases with many respondents and long periods of follow-up, the two-fold approach imputes missing values using chained equations at each time point conditional on information at the same time point and user-specified adjacent time points [80]. This study employs concurrent information plus or minus the two years adjacent to any missing values for the imputation. Due to computational constraints, missing data on the seven census tract items used to characterize neighborhood disadvantage are not imputed separately. Rather, only the summary scores for neighborhood disadvantage generated through PCA based on those seven items are imputed. These scores are then divided into quintiles using the pre-established cut-points from the distribution of all (nonmissing) tract-year observations in the NCDB.

As discussed previously, a considerable number of respondents were either lost to follow-up between birth and age 17 or did not respond to any questions about height and weight in early adulthood. This is not

[18] Because baseline or time-invariant covariates are included in the logistic regression models used to calculate both the numerator and the denominator of the stabilized IPTWs, they must still be included as control variables in the final marginal structural models.

unexpected given the length of observation and stringent requirement that respondents be present at every wave of data collection from birth through age 17 plus at least one wave in early adulthood. To minimize the effects of biasing attrition – that is, loss to follow-up that is selectively related to obesity incidence – stabilized censoring weights for each respondent at each year are generated in the same manner as the stabilized IPT weights described above, except now the weights model the probability of remaining in the study for each respondent at each year, conditional on the same covariates as before. In this way, loss to follow-up at each year (like exposure to neighborhood disadvantage) is essentially independent of respondents' prior time-varying covariates. An overall censoring weight is generated by multiplying the weights for each respondent at each year. The pseudo-population to which the final marginal structural model is fit, as described above, is actually constructed based on the product of the final stabilized IPT weight and stabilized censoring weight for each respondent (not just the IPT weight).

3.4 RESULTS

3.4.1 *Sample Characteristics*

Tables 3.1 and 3.2 present descriptive statistics for the time-invariant and time-varying sample characteristics, respectively. Given notable racial differences across most variables of interest, results are presented for the total sample as well as separately for nonwhite and white respondents. In total, approximately two-thirds of sample members were white[19] and slightly more than half were female (Table 3.1). The vast majority (80%) were born to a married mother who was, on average, 25 years of age, and only seven percent were born low birthweight. Educational attainment among household heads at the time of respondents' birth was split fairly evenly across the three categories, although high school graduates constituted a slightly higher percentage (compared to those with less than a high school diploma and those who had attended at least some college). As young adults, over 30 percent of respondents had been obese at least once. These overall statistics, however, mask considerable racial differences. For instance, nonwhite respondents were disproportionately likely to be female, to be born to an unmarried mother, and to arrive in a household in which the head had less than a high school education. They were also nearly twice as likely as whites to be born low birthweight, a global indicator of health status that may affect future BMI. Finally, nonwhite young adults were over 1.5 times more likely than their white counterparts to experience an incidence of obesity in early adulthood.

[19] The nonwhite category consists of 95 percent African American-identified respondents.

Table 3.2 reports summary statistics for time-varying characteristics at ages three, nine, and 15, representing the mid-points of each of the three developmental stages assessed in this study. For the total sample, the overall trend appears positive for child development and health. For example, comparing age 15 to age three, more respondents lived in owner-occupied households in which the head was married and employed and in which income (standardized to 1985 dollars) had increased slightly and public assistance receipt had decreased. Residence in the most disadvantaged neighborhoods also decreased among the average respondent between age three and age 15. Nonetheless, dramatic racial differences persist across the child and adolescent life course. From ages three through 15, nonwhite respondents remained in households that were consistently more disadvantaged than white respondents with respect to the head's marital and employment statuses, as well as income and hours worked per week. The single most striking difference, however, was the inequality in exposure to neighborhood disadvantage highlighted in the top section of Table 3.2. At age nine, for example, 70 percent of nonwhite children resided in the most disadvantaged neighborhood quintile compared to just 12 percent of whites. At the other end of the spectrum, only three percent of nonwhite versus 20 percent of white nine-year-olds resided in neighborhoods characterized by the least disadvantage. This same general pattern of racial-spatial inequality is also apparent at age three and age 15.

3.4.2 *Temporal Patterns of Exposure to Neighborhood Disadvantage*

Although the descriptive results above provide some indication of the duration and timing of exposure to neighborhood disadvantage during childhood and adolescence (as well as racial disparities therein), Figure 3.1 displays such patterns more explicitly. As described above, cumulative and timing-specific measures of exposure to neighborhood disadvantage were calculated by summing the neighborhood disadvantage quintiles to which each respondent was exposed throughout childhood and adolescence and during each of the three developmental stages, respectively, and then dividing by the total number of times exposure was assessed – essentially creating average measures of exposure for each respondent that can take on any value between one and five. Higher values indicate more prolonged exposure to neighborhood disadvantage. The differently colored bars in Figure 3.1 display the means of these temporal measures of exposure for the total sample (dark gray) and for the nonwhite (medium gray) and white (light gray) subgroups during the entire child and

adolescent life course, as well as separately for early childhood (ages 1-5), late childhood (ages 6-11), and adolescence (ages 12-17).

Because the PSID contains an oversample of low-income households, it is not surprising that the average level of neighborhood disadvantage to which respondents, irrespective of race, are exposed throughout their entire childhood and adolescence is 3.40 – that is, respondents tend to be exposed to neighborhoods that fall, on average, between the third and fourth quintiles of the disadvantage distribution. This value falls in the same general range during each of the three developmental stages, although it is significantly smaller during adolescence (ages 12-17; mean=3.26) – that is, respondents are exposed, on average, to slightly better quality neighborhoods as they get older. Not surprisingly, however, when the total sample is stratified by race, patterns of exposure to neighborhood disadvantage look strikingly different for nonwhite versus white respondents. Among nonwhite respondents, the mean level of exposure to neighborhood disadvantage from ages one through 17 is 4.40 compared to just 2.88 among white respondents, suggesting that while a considerable share of nonwhite respondents reside in the nation's most disadvantaged neighborhoods, their white counterparts tend to experience neighborhoods characterized by significantly less deprivation. Again, the same general pattern of inequality in exposure to neighborhood disadvantage emerges during all three developmental stages for nonwhite versus white respondents.

3.4.3 *Cumulative and Timing-Specific Neighborhood Effects on Obesity*

Table 3.3 displays coefficients from an unadjusted logistic regression model (Model 1) examining the effect of race on obesity incidence, as well as from marginal structural models using stabilized IPT and censoring weights to estimate the effects of duration (Model 2) and timing (Models 3-6) of exposure to neighborhood disadvantage. The first model merely quantifies the statistically significant disparity in the probability of being obese at least once between ages 18 and 30 among nonwhite versus white respondents, without considering prior neighborhood exposures or individual- and household-level covariates. The unadjusted logit estimate indicates that nonwhite respondents have nearly two times greater odds of being obese at least once in early adulthood compared to white respondents (exp(0.68)=1.98).

Model 2 adds a measure of cumulative exposure to neighborhood disadvantage and uses IPT and censoring weights to account for the potential confounding and mediating roles of time-varying individual- and household-level covariates while also controlling for time-invariant characteristics. In this second model, race is no longer a statistically significant predictor of obesity incidence. Instead, the statistically significant

coefficient for cumulative exposure to neighborhood disadvantage suggests that racial disparities in young adult obesity may be due in part to racial differences in neighborhood exposures throughout childhood and adolescence. More specifically, each unit (or quintile) increase in cumulative exposure to neighborhood disadvantage from ages one through 17 is related to a 36 percent increase in the odds of obesity in early adulthood (exp(0.31)=1.36). By extension, respondents who were persistently exposed to neighborhoods in the most disadvantaged quintile from ages one through 17 (a majority of nonwhite respondents) had nearly 3.5 times higher odds of being obese at least once between ages 18 and 30 compared to (predominantly white) respondents who resided, on average, in the least disadvantaged neighborhood quintile (exp((5-1)x0.29)=3.46). These estimates exceed those produced using more conventional regression methods that condition on both time-invariant covariates as well as all time-varying covariates averaged over ages one to 17 (Appendix 3.A), suggesting that prior research based on such techniques may over-control the indirect (or mediated) pathways through which prolonged neighborhood exposure impacts future BMI and racial disparities therein.

Models 3 through 5 examine the effects of timing of exposure to neighborhood disadvantage during early childhood, late childhood, and adolescence, respectively, without regard for the other two developmental stages. There is no statistically significant association between young adult obesity and the average neighborhood disadvantage quintile to which respondents are exposed during early childhood (ages 1-5; Model 3), while race remains a significant (although considerably attenuated) predictor. Exposures that occur during both late childhood (Model 4) and adolescence (Model 5), however, each have independent effects on future BMI. In particular, each unit (or quintile) increase in the average level of exposure to neighborhood disadvantage during late childhood (ages 6-11) and adolescence (ages 12-17) is related to a statistically significant 23 percent ($p<0.05$) and 34 percent ($p<0.01$) increase, respectively, in the odds of being obese at least once between ages 18 and 30. When all three developmental stages are included in the model simultaneously (Model 6), though, only the adolescent time period from ages 12 through 17 remains statistically significant ($p<0.05$), suggesting that exposure to neighborhood disadvantage during adolescence has a more consequential effect on obesity incidence in young adulthood than exposures that occur during early or late childhood.

To further explore this relationship and the disappearance of the late childhood effect from Models 5 to 6, additional analyses using different developmental cut-points were performed. These analyses revealed that

the independent effect of late childhood exposure to neighborhood disadvantage likely reflected the role of exposure to neighborhood disadvantage among 10- and 11-year-olds, not six- through nine-year-olds. In other words, the point in adolescence at which exposure to neighborhood disadvantage begins to matter for future (young adult) BMI appears to be age 10 rather than age 12. More specifically, each unit (or quintile) increase in the average neighborhood disadvantage quintile to which adolescents are exposed from ages 10 through 17 is associated with a 42 percent increase in the odds of obesity in early adulthood (exp(0.35)=1.42), controlling for the effects of exposures that occur earlier in childhood (ages 1-9). The sensitivity of the neighborhood-obesity effect to this adolescent stage of the life course can be juxtaposed with other research showing that family- (as opposed to neighborhood-) level deprivation in early childhood has a more influential effect on future health, including obesity incidence, than exposures that occur during later developmental stages [81]. This is not surprising, however, when considering the particular salience of the family versus the neighborhood during younger versus older periods of the pre-adult life course [82].

Figure 3.2 provides a visual representation of the temporal dimensions of the effects of exposure to neighborhood disadvantage on obesity. It depicts predicted probabilities (and their 95 percent confidence intervals) of being obese at least once between ages 18 and 30 by the average level of neighborhood disadvantage to which respondents were exposed as children and youth. All other baseline covariates are set to their sample means. The graph displays how the probability of having a BMI greater than or equal to 30 kg/m^2 at least once in early adulthood would be expected to increase if respondents had been exposed, on average, to more disadvantaged neighborhoods throughout the entire child and adolescent life course (ages 1-17; orange line) and during just adolescence (ages 10-17), controlling for prior childhood exposures (blue line). As the figure indicates, if respondents had been persistently exposed to the least disadvantaged (1^{st}) quintile of neighborhoods from ages one through 17, about 17 percent would have had an incidence of obesity in early adulthood. Conversely, if the same respondents had been exposed, on average, to the fourth or fifth quintiles of neighborhood disadvantage as children and youth, approximately twice as many – that is, 34 percent and 41 percent, respectively – would have had an incidence of obesity between ages 18 and 30. Such numbers are nearly identical for exposure occurring during the adolescent stage of the life course. Given the links between obesity and concurrent and future physical and mental health care need and utilization as well as comorbid conditions, such an increased incidence of obesity among relatively young adults may represent

a substantial added burden for this country's most under-resourced communities, as well as an important feedback mechanism through which neighborhood inequality is perpetuated.

3.5 Discussion and Conclusions

This study examined the effects of growing up in neighborhoods characterized by varying levels of disadvantage on obesity incidence in early adulthood utilizing the 1970 to 2011 waves of the PSID merged with census data on respondents' neighborhoods. Consistent with previous research, findings show that neighborhood disadvantage, defined by the spatial clustering of poverty, unemployment, female-headed households, public assistance receipt, and educational and occupational marginalization, is associated with higher BMI. More notably, this study is among the first to document that more prolonged exposure to neighborhood disadvantage throughout the entire child and adolescent life course increases the risk of obesity in early adulthood, and that exposure to neighborhood disadvantage during adolescence may be more consequential than exposures that occur during earlier stages of development. Whereas prior studies in this area characterize the residential environment only once or over just a short window of observation, the estimates presented here are based on yearly measurements of respondents' neighborhoods from birth through age 17 as well as statistical methods that account for dynamic individual- and household-level factors known to be predictive of future BMI but also related to the sorting of families into and out of neighborhoods over time.

Such estimates suggest more specifically that the effect of residing, on average, in neighborhood disadvantage quintile *q* versus *q*-1 (a less disadvantaged neighborhood) from ages one through 17 is associated with a 36 percent increase in the odds of being obese at least once between ages 18 and 30. This means that respondents who are continuously exposed to the most disadvantaged neighborhood quintile have 36 percent greater odds of being obese in early adulthood compared to those who grow up, on average, in the fourth quintile, 86 percent greater odds compared to those in the third quintile, over 2.5 times greater odds than those in the second quintile, and nearly 3.5 times greater odds than those who were continuously exposed to the least disadvantaged (first) neighborhood quintile. In addition, the adolescent (compared to early and late childhood) stage of the life course emerged as a sensitive period for neighborhood effects on the incidence of future (young adult) obesity, with each unit (or quintile) increase in the average neighborhood disadvantage quintile to which respondents were exposed between ages 10 and 17 associated with a 42 percent increase

in the odds of being obese at least once between ages 18 and 30, controlling for exposures at ages 9 and younger.

While these results indicate that both the duration and timing of exposure to neighborhood disadvantage during childhood and adolescence have universal effects on the incidence of young adult obesity, a more nuanced interpretation suggests that the adverse effects of neighborhood disadvantage are likely to be more concentrated among nonwhite versus white children and youth. In keeping with other work on racial residential segregation and neighborhood stratification, this study found that exposure to neighborhood disadvantage is strikingly more common, both cumulatively from birth through age 17 as well as during each of three developmental stages therein, among nonwhite, predominantly African American, children and youth. Nonwhite children and youth were not only more likely than their white counterparts to be born into neighborhoods characterized by higher levels of disadvantage and thus fewer social and structural resources for healthy eating and physical activity, but they were also more likely to remain in similar types of health-compromising residential environments for the entirety of their pre-adult years. Simple regression analyses further show that the nearly two-fold disparity between nonwhite and white young adults in the crude incidence of young adult obesity is reduced by about 60 percent once measures of either cumulative or adolescent exposure to neighborhood disadvantage are included in the models (results available upon request). The coefficients for cumulative neighborhood disadvantage and for neighborhood disadvantage during adolescence remain statistically significant even when adjusting for a host of time-invariant and time-varying individual- and household-level characteristics using marginal structural models. This suggests that the separate and unequal neighborhood environments in which nonwhite versus white children tend to live, learn, and grow may play a critical role in producing and perpetuating racial disparities in obesity in early adulthood (and beyond).

Although this study uses panel data and unique statistical methods to address some of the most common challenges in neighborhood effects research (namely, reverse causation and selection bias), the results should be considered in the context of several remaining limitations. First, although there appears to be sufficient overlap in the race-specific distributions of cumulative and timing-specific exposure to neighborhood disadvantage to generate stable IPT weights and thus to satisfy the positivity assumption on which the analyses are based, it would be ideal to find a sample in which neighborhood stratification by race is less pronounced in order to further disentangle the mechanisms behind the relationships among race, place, and

BMI. Second, given the historical timing of this study, the sample is limited to predominantly African American and white respondents. Future research examining disparities in obesity as a function of cumulative and timing-specific neighborhood effects among Asian and Latino populations is encouraged. Finally, this study did not assess the specific mechanisms thought to help explain how neighborhood-obesity effects transpire but rather the broader neighborhood conditions thought to engender such processes. Longitudinal research able to more explicitly measure these potential mechanisms including, for example, continuity and change in supermarket and fast food outlet density, perceptions of safety, collective efficacy, and institutional resources, would be enlightening.

Nonetheless, findings from the present study add to the growing body of evidence suggesting that place-based, developmentally-appropriate, and ongoing investments in the social, economic, institutional, and infrastructural aspects of under-resourced neighborhoods and neighborhoods of color can help reduce obesity and obesity-related illnesses that extend across the life course (e.g., heart disease, stroke, type 2 diabetes, and certain cancers). On the one hand, a considerable body of prior scholarship has detailed the so-called "long arm" of early-life individual- and family-level disadvantage on health in older ages, including the effects of family poverty in childhood [82] and early-life adversity and toxic stress [83-85] on obesity-related physical and mental well-being and mortality later in life [86, 87]. On the other hand, a related but often separate body of extensive research has further shown that neighborhood-level disadvantage is associated with BMI, although most of this work relies on cross-sectional data [8, 88]. This study brings these two literatures into more explicit dialogue, empirically documenting the importance of both duration and timing of child and adolescent exposure to neighborhood disadvantage for obesity incidence in early adulthood. It supports and extends similar work by the author showing that children and youth who experience more prolonged exposure to neighborhood disadvantage are also more likely to report fair or poor (as opposed to excellent, very good, or good) self-rated health as young adults (*working paper*). Whereas this earlier study examined only the duration of exposure to neighborhood disadvantage and assessed a subjective measure of health status, the current study includes the effects of both duration and timing of exposure as well as a more objective (although still self-reported) health indicator: the ratio of weight to height, or BMI. Together, the findings from these studies indicate that adverse individual and familial experiences during childhood and adolescence matter for neighborhood selection and future BMI, but they also demonstrate the crucial role of related social and structural adversities in the broader neighborhood environment. That is, policies aimed at changing individual

diet and physical activity behaviors are likely of limited utility when the collective characteristics of neighborhoods, including concentrated poverty, joblessness, crime and violence, residential instability, and socioeconomic disinvestment, make such choices more difficult, if not impossible, to envision the benefits of or to enact and sustain.

Moreover, in order to cultivate a more healthful and thus productive cadre of adult citizens, policies must strive to generate durable place-based investments in children and youth, "a continuum of resources and support" [19], that last across the life course. Nonetheless, the results in this study suggest that given practical and economic constraints on such initiatives, neighborhood-based efforts targeting the food and physical activity structures and behaviors of adolescents (as opposed to younger children) may be particularly successful in curbing the rising tide of obesity and obesity-related conditions among adults in the U.S. Whereas prior neighborhood effects research based on cross-sectional data may have concluded with similar general sentiments, the implementation of associated health practice and policy changes has often been hampered by challenges to causal inference. Using longitudinal data and a statistical method that attempts to model the full data distribution (i.e., estimating for each respondent their probability of obesity under each possible level of neighborhood disadvantage rather than just their actual observed levels), this study provides among the strongest evidence for the consequences of both sustained and adolescent exposures to neighborhood disadvantage on future (young adult) obesity, especially among nonwhite, predominantly African American, children and youth for whom such exposures tend to be more common and more persistent. .

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Table 3.1. Time-invariant sample characteristics

	Total	Nonwhite	White
	(n=1,522)	(n=524)	(n=998)
Ever obese in early adulthood, percent			
BMI ≥ 30 kg/m^2	31.01	40.84	25.85
BMI < 30 kg/m^2	68.99	59.16	74.15
Gender, percent			
Female	53.75	59.35	50.8
Male	46.25	40.65	49.2
Birthweight, percent			
Less than 88 ounces	7.03	10.11	5.41
88 ounces or more	92.97	89.89	94.59
Mother's marital status at birth, percent			
Unmarried	20.37	47.71	6.01
Married	79.63	52.29	93.99
Household head's educational attainment at birth, percent			
Less than high school	28.52	52.86	15.73
High school graduate	36.86	33.59	38.58
At least some college	34.63	13.55	45.69
Mother's age at birth, mean (SD)	24.55 (5.06)	22.71 (5.06)	25.51 (4.80)

Note: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one set of questions about height and weight in early adulthood (first of 10 imputation datasets).

Table 3.2. Time-varying sample characteristics

	Total (n=1,522)			Nonwhite (n=524)			White (n=998)		
	Age 3	Age 9	Age 15	Age 3	Age 9	Age 15	Age 3	Age 9	Age 15
Neighborhood disadvantage quintile, percent									
1st quintile (least disadvantaged)	11.63	13.99	17.28	2.48	3.05	5.15	16.43	19.74	23.65
2nd quintile	14.13	13.67	16.16	3.24	4.77	6.68	19.84	18.34	21.14
3rd quintile	17.67	18.73	19.97	7.25	5.92	8.78	23.15	25.45	25.85
4th quintile	22.93	21.48	20.17	16.41	16.41	18.70	26.35	24.15	20.94
5th quintile (most disadvantaged)	33.64	32.13	26.41	70.61	69.85	60.69	14.23	12.32	8.42
Household head's marital status, percent									
Unmarried	16.89	22.08	25.89	36.01	43.13	48.47	6.81	11.02	14.03
Married	83.11	77.92	74.11	63.93	56.87	51.53	93.19	88.98	85.97
Household head's employment status, percent									
Unemployed	16.29	16.43	15.18	31.68	32.06	29.20	8.22	8.22	7.82
Employed	83.71	83.57	84.82	68.32	67.94	70.80	91.78	91.78	92.18
Public assistance (AFDC) receipt, percent									
Received AFDC	10.38	11.37	7.03	22.90	26.53	17.75	3.81	3.41	1.40
Did not receive AFDC	89.62	88.63	92.97	77.10	73.47	82.25	96.19	96.59	98.60
Homeownership, percent									
Does not own home	43.36	35.55	30.09	66.79	60.31	51.15	31.06	22.55	19.04
Owns home	56.64	64.45	69.91	33.21	39.69	48.85	68.94	77.45	80.96
Household income in (1985) $1,000s, mean (SD)	25.62 (16.74)	22.53 (22.54)	25.88 (28.79)	15.29 (10.92)	16.33 (13.59)	17.90 (15.51)	21.45 (17.43)	25.79 (25.45)	30.07 (32.98)
Household head's work hours/week, mean (SD)	37.44 (17.32)	36.69 (18.15)	37.59 (17.57)	28.67 (18.23)	27.56 (19.71)	29.17 (19.62)	42.04 (14.89)	41.48 (15.24)	42.02 (14.57)
Family size, mean (SD)	4.27 (1.58)	4.55 (1.30)	4.40 (1.25)	4.69 (2.22)	4.68 (1.72)	4.60 (1.50)	4.06 (1.03)	4.47 (1.01)	4.30 (1.09)

Note: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one set of questions about height and weight in early adulthood (first of 10 imputation datasets).

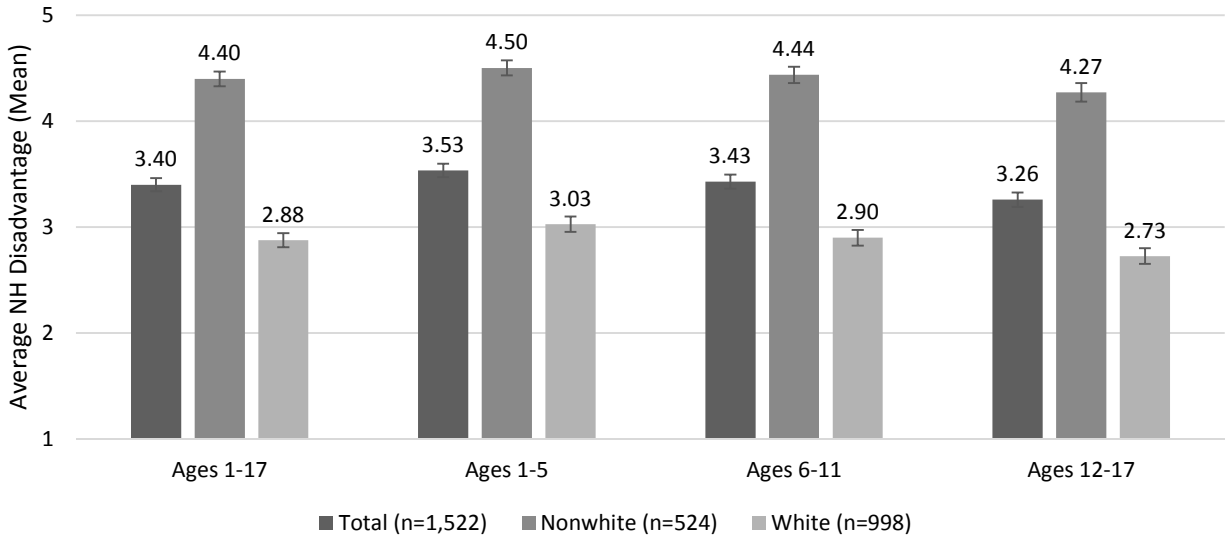

Figure 3.1. Mean level of cumulative (ages 1-17) and timing-specific (ages 1-5, 6-11 and 12-17) exposure to neighborhood disadvantage for all respondents and for the nonwhite and white subsamples; NH=Neighborhood

Table 3.3. Effects of duration and timing of exposure to neighborhood disadvantage from ages 0-17 on obesity incidence in early adulthood (n=1,522)

	Race-Only	Duration	Timing			
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Average NH disadvantage quintile						
Ages 1 through 17		0.31 ***				
Ages 1 through 5 (early childhood)			0.11			-0.06
Ages 6 through 11 (late childhood)				0.21 **		-0.01
Ages 12 through 17 (adolescence)					0.29 ***	0.32 ***
Race (white)						
Nonwhite	0.68 ***	0.22	0.37 **	0.28	0.19	0.20
Gender (male)						
Female		0.13	0.12	0.13	0.11	0.11
Birthweight (88 ounces or more)						
Less than 88 ounces		-0.10	-0.06	-0.09	-0.11	-0.11
Mother's marital status at birth (married)						
Unmarried		0.00	0.03	0.02	-0.02	-0.02
HH's education at birth (less than HS)						
High school graduate		-0.13	-0.17	-0.13	-0.12	-0.12
At least some college		-0.56 **	-0.67 ***	-0.59 ***	-0.56 *	-0.57 ***
Mother's age at birth		0.00	0.00	0.00	0.00	0.00
NH disadvantage at birth		-0.07	-0.02	-0.03	-0.03	0.00
HH's marital status at birth (married)						
Unmarried		0.16	0.12	0.14	0.18	0.18
HH's employment status at birth (employed)						
Unemployed		0.07	0.07	0.07	0.06	0.05
Homeownership at birth (owns home)						
Does not own home		-0.04	0.00	-0.03	-0.05	-0.04
Family size at birth		-0.06	-0.06	-0.06	-0.06	-0.06
Public assistance (AFDC) receipt at birth (no AFDC)						
Received AFDC		-0.60 *	-0.56 *	-0.59 *	-0.63 **	-0.63 **
Household income at birth		0.02	-0.02	0.01	0.03	0.03
HH's work hours per week at birth		-0.01	-0.01	-0.01	-0.01	-0.01
Year born (1970-72)						
1973-1975		0.35 *	0.35 *	0.35 *	0.37 *	0.38 *
1976-1978		0.37 *	0.35 *	0.35 *	0.39 **	0.39 **
1979-1980		0.48 **	0.45 **	0.46 **	0.52 **	0.53 **

Notes: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one set of questions about height and weight in early adulthood; Coefficients are combined estimates from 10 multiple imputation datasets; NH=Neighborhood; HH=Household head; *p<0.10; **p<0.05; ***p<0.01

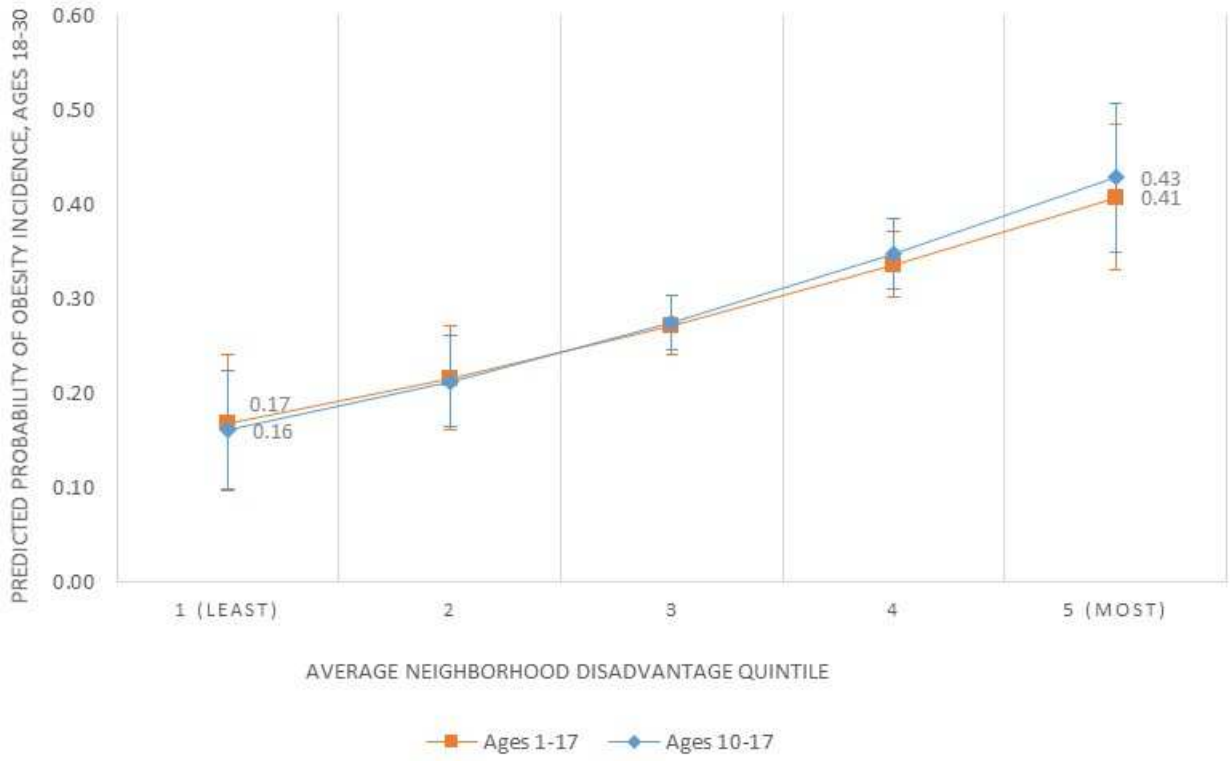

Figure 3.2. Predicted probability of obesity in early adulthood by average level of neighborhood disadvantage experienced throughout childhood and adolescence (ages 1-17) and during adolescence (ages 10-17), controlling for prior exposure

Appendix 3.A

Table 3.4. Regression-adjusted effects of cumulative exposure to neighborhood disadvantage from birth through age 17 on obesity incidence in early adulthood (n=1,522)

Variable	Coef.	
Average NH disadvantage quintile		
Ages 1 through 17	0.25	***
Race (white)		
Nonwhite	0.18	
Gender (male)		
Female	0.19	
Birthweight (88 ounces or more)		
Less than 88 ounces	-0.29	
Mother's marital status at birth (married)		
Unmarried	-0.09	
HH's education at birth (less than HS)		
High school graduate	-0.12	
At least some college	-0.45	**
Mother's age at birth	-0.01	
NH disadvantage at birth	-0.01	
HH's marital status at birth (married)		
Unmarried	0.05	
HH's employment status at birth (employed)		
Unemployed	-0.02	
Homeownership at birth (owns home)		
Does not own home	-0.12	
Family size at birth	-0.03	
Public assistance (AFDC) receipt at birth (no AFDC)		
Received AFDC	-0.25	
Household income at birth	0.03	
HH's work hours per week at birth	-0.01	
Year born (1970-72)		
1973-1975	0.45	***
1976-1978	0.40	*
1979-1980	0.49	***
HH's marital status averaged over ages 1 through 17	0.08	
HH's employment status averaged over ages 1 through 17	-0.35	
Homeownership status averaged over ages 1 through 17	0.29	
Family size averaged over ages 1 through 17	0.02	
Public assistance (AFDC) status averaged over ages 1 through 17	-0.14	
Household income averaged over ages 1 through 17	0.05	
HH's work hous per week averaged over ages 1 through 17	-0.01	

Notes: Statistics reported for respondents not lost to follow-up before age 18 and who answered at least one set of questions about height and weight in early adulthood; Coefficients are combined estimates from 10 multiple imputation datasets; NH=Neighborhood; HH=Household head; *p<0.10; **p<0.05; ***p<0.01

Chapter 4. A DISCRETE-TIME ANALYSIS OF THE EFFECTS OF PROLONGED EXPOSURE TO NEIGHBORHOOD POVERTY ON THE RISK OF EARLY SMOKING INITIATION

4.1 INTRODUCTION

Smoking remains the leading preventable cause of disease, disability, and premature death in the United States (U.S.) [1]. Each year, approximately 443,000 individuals die from smoking-attributable diseases, including cancer, heart disease, and chronic obstructive pulmonary disease, and another 8.6 million people live with a serious illness caused by smoking or exposure to secondhand smoke [2, 3]. Prior research suggests that individuals who initiate smoking at younger ages are at increased risk for future tobacco dependence and continued use as well as for the aforementioned health problems [4-7]. Identifying individual, household, and to a far lesser extent, contextual factors that predict early cigarette use has garnered considerable attention over the last several decades. However, the majority of scholarship in this area has been cross-sectional or conducted over relatively short windows of observation. Few studies have investigated the effects of more prolonged exposure to smoking-related risk factors from childhood through early adulthood. Those that do have tended to focus on individual and household characteristics rather than characteristics of the broader neighborhood environment [8]. Such an approach implicitly ignores how these various characteristics and the relationships among them change over time, resulting in effect estimates that do not adequately account for multiple levels of risk and are largely insensitive to differences in the duration of such exposures.

This study uses the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID) merged with census data on respondents' neighborhoods to estimate a series of race-specific discrete-time logit models for the risk of youth and young adult smoking initiation as a function of neighborhood poverty as well as individual and household characteristics from ages four through 25. Similar to the neighborhood effects literature more broadly, the few longitudinal studies of smoking onset to examine risk factors at multiple levels of influence have been subject to complex and dynamic sources of bias related to the improper treatment of time-varying individual and household characteristics and their role in processes of neighborhood selection. In contrast to more conventional regression-based techniques, this study employs inverse probability of treatment (IPT) weighting to adjust for such methodological difficulties. More

specifically, the IPT weights account for dynamic residential selection processes without controlling away the effects of neighborhood poverty that may operate indirectly through the same individual- and household-level covariates that have been associated with residential mobility (and subsequent smoking initiation). Results from these analyses indicate that long-term residence in an impoverished neighborhood influences smoking behavior above and beyond, as well as in conjunction with, individual and household characteristics, such as age, gender, and household income, among other micro-level social and demographic covariates. In particular, more prolonged exposure to high (>20%) as opposed to low (<10%) poverty neighborhoods is associated with an increased risk of smoking onset by age 25. Although consistent with prior literature, this effect is only observed among white and not nonwhite respondents.

4.2 THEORETICAL BACKGROUND

Research examining the health effects of neighborhood characteristics, particularly concentrated poverty and other forms of socioeconomic disadvantage, has surged in recent decades. Numerous studies have linked adverse residential conditions to premature death [9, 10], poor self-rated health [10-13], depression and other mental health problems [14-17], health-risk behaviors such as poor diet and physical inactivity [18-21], and related chronic conditions such as obesity, diabetes, cardiovascular disease, and certain cancers [22-26], net of individual- and household-level factors. There is also a sizable body of literature on the relationship between neighborhoods and substance use, including smoking behavior. Early findings from this work suggested that residing in more deprived neighborhoods was associated with an increased risk of tobacco use [27-33]. More recent studies, however, reveal a more nuanced landscape in which the effects of neighborhood characteristics on tobacco use vary across age and racial groups. Among adolescents, for example, Lee and Cubbin [34] find no associations between low neighborhood socioeconomic status or high neighborhood social disorganization and smoking behavior, whereas Ennett and colleagues [35] document higher rates of cigarette use in schools located in neighborhoods perceived as less, rather than more, socially disadvantaged. With respect to adolescents of different racial groups, Nowlin and Colder [36] find that higher levels of neighborhood poverty are associated with increased smoking for white but not black youth [37, 38]. Other scholars have also found that living in a predominantly black neighborhood protects black but not white adolescents from cigarette use, whereas living in a

predominantly white neighborhood is associated with more cigarette use among both black and white youth, especially in more disadvantaged neighborhoods [39].

To a certain extent then, such findings challenge expectations based on the theoretical frameworks commonly used to help explain neighborhood effects on health and health behaviors. The social isolation paradigm, for example, contends that macroeconomic changes related to the 1970s-era deindustrialization of central cities resulted in the systematic separation (often in both physical and social space) of low-income residents from middle- and upper-income families whose presence in the same urban neighborhoods had previously served to validate mainstream norms as well as helped to attract and sustain the basic institutions of the area [40-42]. In terms of its influence on health behaviors such as cigarette smoking, social isolation is often invoked in conjunction with theories of collective socialization and place-based epidemic or contagion processes [43]. That is, the relative isolation of residents (particularly younger residents) of impoverished neighborhoods from adult role models who have achieved a degree of social and economic security through mainstream channels increases the likelihood that the socialization of low-income children and youth will include formative interactions with older peers who tend to be unemployed and not in school (and therefore more likely to be available and visible in the neighborhood), as well as more likely to engage in deviant or health compromising behaviors [44]. In impoverished areas, therefore, cigarette smoking may not only be more prevalent,[20] but also particularly salient and respected, and therefore more likely to spread through collective processes of exposure to and social learning via qualitatively distinct, influential others [45]. Moreover, because middle- and upper-income families help attract and sustain basic (often salutary) institutions, such as churches, grocery stores, and recreational centers, their relative absence means that impoverished neighborhoods are disproportionately likely to be served by convenience and other small stores in which tobacco products are more prominent, more heavily marketed, and more accessible [46, 47].

Similarly, theories of social (dis)organization, or what has been re-conceptualized in terms of social capital and more recently, as collective efficacy and informal social control, suggest that differences across neighborhoods in mutual trust as well as shared expectations and capacities for prosocial action (a

[20] Given the strong inverse relationship between individual socioeconomic position and smoking, it is likely that disadvantaged neighborhoods (containing more disadvantaged residents) have a higher prevalence of smoking than advantaged neighborhoods.

collective sense of ‘we’, so to speak) are associated with variations in rates of crime and other deviant behaviors as residents of more advantaged areas with higher levels of collective efficacy are more likely to feel empowered to intervene and to enforce salutary norms and behaviors [48]. In such neighborhoods, parents are more likely to know the parents of their children’s friends, share information about young people’s behavior, monitor spontaneous hang outs among youth, and feel justified stepping in to actively discourage tobacco use in both private and public spaces and among both known and unknown adolescents. Moreover, at the institutional level, differences in collective efficacy across more or less deprived neighborhoods may influence residents’ willingness and capacity to mobilize and extract resources, for example, to advocate for the passage of a local ordinance to restrict smoking in public places or to use zoning restrictions to prevent tobacco advertising and tobacco outlets in their neighborhoods.

Finally, theories related to the quality of and access to resources in the institutional and physical (or “built”) environments suggest that such characteristics constitute so-called “opportunity structures” which may promote or damage health either directly or indirectly through the possibilities they provide for people to make and maintain healthy choices and live healthy lives [45, 49]. As already mentioned in relation to both social isolation and collective efficacy, neighborhoods vary according to their levels of tobacco advertising and availability, the extent to which local institutions and public places encourage cigarette smoking by permitting it on their premises and selling smoking-related products, or whether they visibly discourage it by prohibiting smoking and having signage to that effect, raising awareness about the hazards of smoking, having signs indicating that they do not sell to minors and individuals as well as organizational practices responsible for enforcing such policies [45, 50]. More socially advantaged neighborhoods tend to have more anti-smoking structural aspects, whereas residents of impoverished neighborhoods are disproportionately likely to encounter institutional and built environments which encourage, or at least do not actively discourage, tobacco use [50].

In theory, therefore, the relationship between neighborhood disadvantage and smoking appears straightforward and deleterious. However, as already discussed, many studies report substantially higher prevalence of substance use, particularly cigarette smoking, among white compared to Hispanic, and especially, African American adolescents despite greater exposure to individual-, household-, and neighborhood-level risk factors, including neighborhood poverty [33, 37, 51, 52]. Explanations for such

racially disparate patterns have been largely speculative, although a few rationales have gained traction in the literature. Central among these are the notions that white youth are more susceptible to pro-smoking peer pressure and smoking-related status attainment than are black youth [53], while black youth are more likely to encounter parental disapproval of smoking than are white youth [54, 55], both of which are posited to mediate the relationship between neighborhood disadvantage and smoking initiation/use. On the whole, however, it remains largely unexplained why cigarette smoking diverges so considerably from other health compromising behaviors for which neighborhood-level risk factors tend to have universally deleterious impacts and to which nonwhite, particularly African American, groups are disproportionately exposed.

However, as mentioned at the outset, largely absent from discussions of neighborhood effects on smoking is the element of time. Not only are nonwhites more likely to live in disadvantaged areas than whites, but prior research documents that the proportion of nonwhites who reside in impoverished neighborhoods for repeated or prolonged periods of time is greater than the proportion who do so at any one point in time [56-58]. Conversely, whites are more likely to experience episodic as opposed to sustained residence in disadvantaged neighborhoods [59]. The majority of research on neighborhoods and smoking, though, has relied on cross-sectional data, which implicitly ignores how the duration of exposure to neighborhood-level risk and protective factors may moderate their effects on smoking onset or race-specific patterns therein. This study focuses explicitly on how more prolonged exposure to varying levels of neighborhood poverty affects the risk of smoking initiation among white and nonwhite, predominantly African American, youth and young adults. Given the potential for point-in-time measures of the neighborhood environment to underestimate exposure among nonwhites but overstate exposure among whites, a central aim of this study was to assess how including a duration-sensitive measure of neighborhood poverty would influence smoking initiation risk within each racial group, and in particular, whether a neighborhood effect might now be evident among nonwhites.

4.3 DATA AND METHODS

Data for this study are drawn from the Panel Study of Income Dynamics (PSID), produced and distributed by the University of Michigan's Institute for Social Research and funded by the National Institutes of Health (NIH) and the National Science Foundation (NSF). The PSID is a large, longitudinal survey of U.S. residents and their families conducted annually between 1968 and 1997 and every two years

thereafter. The PSID has several strengths that make it particularly well-suited to the objectives of this study. First, the dataset contains an oversample of low-income families, as well as a wealth of information on a variety of smoking-related individual- and household-level risk and protective factors, including employment, income, education, marriage, social program participation, and housing characteristics. Second, the residential location of individual respondents at each interview can be linked to their corresponding census tract identifiers using the PSID's supplemental, restricted-use Geospatial Match Files. These identifiers make it possible to characterize neighborhood poverty for prolonged intervals, as well as to account for the potential mobility of PSID respondents into and out of different neighborhoods. Third, the longitudinal design of the PSID makes it possible to adjust for the temporal sequencing and compounding influence of individual, household, and neighborhood conditions across time.

4.3.1 *Sample Selection*

The analytic sample for this study consists of the 2,121 PSID respondents who were born in 1966 or later and who responded to at least one set of questions about smoking status in early adulthood. Among current or former smokers, the age at which they initiated the behavior was also ascertained. These respondents were followed from age four until they first became nonresponse, began smoking, or they reached the administrative end of follow-up, defined here as either age 25 or calendar year 2010. Final analyses are based on 27,488 person-years of observation: 17,303 person-years from respondents classified as white and 10,185 person-years from those classified as nonwhite.

4.3.2 *Dependent Variable*

The dependent variable is whether or not an individual began smoking by age 25. Beginning in 1999, PSID respondents who were classified as either a household head or the spouse ("wife") of a household head were asked, *"Do you smoke cigarettes?"* If they answered no, they were also asked, *"Did you ever smoke cigarettes?"* If they answered no again, they were coded as nonsmokers (0) for the duration of follow-up. However, if they answered yes to either of the above questions, respondents were subsequently asked, *"How old were you when you first smoked cigarettes regularly?"* Responses to this question were recorded in whole-year increments. Because this same question was asked at multiple survey waves (1999, 2001, 2003, 2005, 2007, 2009 and 2011), there may be discrepancies in individual responses across waves. If a respondent reported more than one age at smoking onset, the smallest (youngest) modal response was used in analyses.

4.3.3 *Main Independent Variable*

Similar to most prior research in this area, census tracts are used to approximate neighborhood boundaries. Although there are well-known limitations to this operational definition [60], there is broad consensus that census data at the tract level not only provide convenient access to considerable information over extensive time periods, but they also serve as a reasonable proxy for, or are at least highly correlated with, the "true" causally relevant definition of a neighborhood [61-64]. Information on census tracts in 1970, 1980, 1990, 2000, and 2010 comes from the Neighborhood Change Database (NCDB) in which data for all five decades has been normalized to 2010 tract boundaries and can therefore be compared across years without having to adjust for potential changes in boundary definitions over time. Data for intercensal years are imputed using linear interpolation.

Neighborhood poverty is operationalized as a three-level, time-varying variable based on the poverty rate of the census tract in which a respondent resided at each survey wave. Consistent with the cutoffs used in prior literature [65], tracts with less than a 10 percent poverty rate are coded as "low-poverty neighborhoods (1)"; tracts with poverty rates between 10 and 20 percent are coded as "moderate-poverty neighborhoods (2)"; while tracts with greater than a 20 percent poverty rate are coded as "high-poverty neighborhoods (3)". To assess the duration of time a respondent was exposed to each level of neighborhood poverty, a three-category dummy-coded measure of cumulative (or "duration-weighted" [65, 66]) exposure was generated as the proportion of time that respondents lived in low-, moderate-, and/or high-poverty neighborhoods from age five through the survey wave at which they were censored (i.e., became nonresponse, began smoking, or reached the administrative end of follow-up).[21] For example, if the data are organized in long form (person-years) and a respondent spent ages (years) five and six in low-poverty neighborhoods and age seven in a moderate-poverty neighborhood, they would be coded "1" on the low-poverty dummy and "0" on both the moderate- and high-poverty dummies at age five. The same would be true at age six. However, at age seven, they would be coded "0.667" on the low-poverty dummy, "0.333" on the moderate-poverty dummy, and "0" on the high-poverty dummy to reflect the proportion of time they had resided in each of the different levels of neighborhood poverty by age seven (two of three years in low-poverty and one of three years in moderate-poverty neighborhoods).

[21] Neighborhood poverty at age four is not included in this measure but rather as part of a vector of baseline covariates.

4.3.4 *Covariates*

A large number of individual- and household-level variables are included in analyses as either time-invariant (constant) or time-varying. Time-invariant covariates include a respondent's race (1=nonwhite; 0=white), gender (1=female; 0=male), birthweight (1=less than 88 ounces; 0=88 ounces or more), mother's age at birth, mother's marital status at birth (1=unmarried; 0=married), and household head's educational attainment at age four, the start of follow-up in this study (1=less than high school; 2=high school graduate; 3=at least some college)[22], all of which have been associated in previous studies with both neighborhood residence and individual health-risk and protective factors. Time-varying covariates, measured for every respondent at each wave between ages four and 25, include household head's marital status (1=unmarried; 0=married), employment status (1=unemployed; 0=employed), and work hours, as well as family size, homeownership (1=does not own home; 0=owns home), public assistance receipt (1=yes; 0=no), and total household income, standardized using the Consumer Price Index (CPI-U) to 1985 dollars.

4.3.5 *Statistical Analysis*

This study employs a series of race-specific discrete-time logit models based on all person-year observations in which the parameters are estimated using inverse probability of treatment (IPT) weights. This estimation technique has been described in more detail elsewhere [65]. In brief, the IPT weights are used to mimic random assignment of exposure to different levels of neighborhood poverty based on individual and household characteristics, essentially adjusting for residential selection bias by generating a weighted "pseudo-population" to which the discrete-time logit models are subsequently fit. This process proceeds in two steps. First, ordinal logistic regression is used to estimate each respondent's probability of being exposed to low-, moderate-, and high-poverty neighborhoods (contrary to what is observed for two of the three levels of poverty) at each survey wave (year), conditional on their level of neighborhood poverty in the prior year, baseline or time-invariant covariates (including neighborhood poverty at age four), and both prior and concurrent time-varying individual- and household-level covariates. Respondents are then assigned a series of IPT weights based on the inverse (or reciprocal) of the probability that they were exposed to their actual (observed) level of neighborhood poverty at each wave of follow-up. In this way,

[22] The household head's educational attainment is treated as time-invariant and measured at respondents' year of birth because the PSID does not measure parental education at regular intervals. If data at year of birth was missing, the most recent subsequent measurement was used, which for the vast majority of respondents was within 3 years of birth.

proportionally more (or less) weight is given to those respondents whose prior time-varying individual- and household-level covariates are underrepresented (or overrepresented) in the neighborhood to which they were actually exposed at each wave. That is, the IPT weights ensure that the values of all covariates included in their construction are balanced in expectation across the three levels of neighborhood poverty at each wave.

In practice, IPT weights can be highly variable. To increase efficiency and obtain narrower confidence intervals around the subsequent neighborhood effect estimate, the IPT weights for each respondent at each wave are stabilized by multiplying each one by the same probability as was used to generate it, except only time-invariant covariates and prior year neighborhood conditions are included as regressors (i.e., time-varying covariates are excluded). In other words, these reduced-form estimates of the likelihood of exposure to one's actual level of neighborhood poverty at each wave become the numerators for the original IPT weights described above. The stabilized IPT weights for each respondent at each wave are then multiplied together to generate a single value representing each respondent's overall contribution to the pseudo-population.

In the second step of the analytical process, the IPT-weighted pseudo-population is used to fit a conventional discrete-time logit model for the risk of smoking onset in which the dummy-coded measure of exposure to low-, moderate-, and high-poverty neighborhoods is included as the main explanatory variable, along with a quadratic function of age and the time-invariant covariates.[23] This model does not include the time-varying covariates, however. The impact of these variables as potential confounders is already accounted for through the weighting process, while their role as potential mediators of the neighborhood-smoking relationship remains intact. All models, including those used to generate the stabilized IPT weights, are estimated separately for nonwhite and white respondents because prior research suggests that processes of smoking initiation differ by race [53-55].

4.3.6 *Missing Data*

Missing data on all independent variables are multiply imputed using the two-fold fully conditional specification algorithm in STATA v.13.1 [67]. The two-fold fully conditional specification algorithm was

[23] Because the time-invariant covariates are included in the logistic regression models used to calculate both the numerator and the denominator of the stabilized IPT weights, they must still be included as control variables in the final discrete-time logit models.

designed to impute missing data in longitudinal studies. Whereas conventional multiple imputation methods ignore the longitudinal and dynamic structure of panel data and are difficult to implement in large databases with many respondents and long periods of follow-up, the two-fold approach imputes missing values using chained equations at each time point conditional on information at the same time point and user-specified adjacent time points [67]. This study employs concurrent information plus or minus the one year adjacent to any missing values for the imputation.

4.4 RESULTS

4.4.1 *Sample Characteristics*

Tables 4.1 and 4.2 display descriptive statistics for the time-invariant and time-varying sample characteristics, respectively, among nonwhite and white respondents. As shown, there are considerable racial differences across most variables of interest. For example, nonwhite respondents – approximately 95 percent of whom are African American – are over six times as likely to be born to an unmarried mother, more than twice as likely to be low birthweight, and about three times as likely at age four to live in a household in which the head had less than a high school education compared to white respondents (Table 4.1).

To provide an indication of the temporal patterns of exposure to time-varying individual-, household-, and neighborhood-level risk factors, Table 4.2 reports summary statistics at ages four, 12, and 17 by race. Although the general pattern appears positive over time for both nonwhite and white respondents – that is, the percentage of respondents exposed to smoking-related risk factors decreases between ages four and 17 – striking racial disparities persist across time. For instance, nonwhite respondents at all three ages are nearly four times more likely than whites to live in a household in which the head was unmarried and unemployed. They are also considerably more likely to live in households that received public assistance and less likely to live in an owner-occupied residence compared to white respondents at ages four, 12, as well as 17. Some of the most notable racial differences, however, are in relation to the level of neighborhood poverty to which nonwhite versus white respondents are persistently exposed. At age four, for example, only 11 percent of nonwhite respondents resided in low-poverty neighborhoods compared to nearly two-thirds of white respondents. This disparity is reversed at the less affluent end of the neighborhood poverty spectrum. That is, 64 percent of nonwhite four-year-olds were exposed to high-poverty neighborhoods

compared to just eight percent of white four-year-olds. Although slightly smaller percentages of nonwhite respondents were exposed to high-poverty neighborhoods at age 12 and then age 17 than had been at age four, the racial gap in exposure to neighborhood poverty remained largely intact across time.

Table 4.3 describes the risk of smoking initiation by age and race. Despite being exposed to more disadvantaged individual, household and in particular, neighborhood characteristics throughout childhood and adolescence, the risk of smoking initiation is smaller among nonwhite respondents compared to their white counterparts at almost every age. For example, at age 14, the estimated probability of smoking onset is 0.01 for nonwhites and 0.03 for whites. Smoking risk peaks at age 18 for all respondents with an estimated probability of 0.10 for nonwhites and 0.15 for whites. Overall, 192 nonwhite and 486 white respondents, or 24 percent and 37 percent, respectively, began smoking by age 25 (Figure 4.1). Such findings align with prior research documenting lower prevalence of smoking among nonwhite, predominantly African American, adolescents and young adults. However, as shown more clearly in Figure 4.2, which depicts the probability of smoking onset by age among respondents still at risk, there is some evidence to suggest that nonwhite respondents may begin to smoke at slightly higher rates than whites after about age 20.

4.4.2 *Cumulative Neighborhood Effects on Smoking Initiation*

Table 4.4 presents unadjusted (Model 1), IPT-weighted (Model 2), and conventional regression-adjusted (Model 3) estimates of the risk of smoking initiation by age 25 as a function of the cumulative proportion of time nonwhite and white respondents, respectively, spent in moderate- and high-poverty neighborhoods relative to low-poverty neighborhoods. The unadjusted models, which simply include the neighborhood poverty variable and the quadratic function of age, indicate that the odds of smoking initiation increase as respondents get older, but decelerate during the later years among both nonwhite and white respondents (as indicated by the age and age-squared terms, respectively). With respect to neighborhood poverty, white respondents who experience more prolonged exposure to moderate- versus low-poverty neighborhoods have 33 percent greater odds ($p<0.10$) while those who spend more time in high- versus low-poverty neighborhoods have 166 percent (or more than 2.5 times) greater odds ($p<0.001$) of smoking initiation by age 25, although only the effect of high-poverty neighborhoods reaches statistical significance at greater than the 95 percent confidence level. Among nonwhite respondents, however, there are no

statistically significant effects on smoking initiation of more sustained exposure to either moderate- or high-poverty neighborhoods compared to low-poverty neighborhoods, although the effect of high-poverty neighborhoods is moderately significant at the 90 percent confidence level ($p<0.10$).

The IPT-weighted estimates in Model 2 adjust for selection into neighborhoods characterized by different levels of poverty as well as for covariates measured at birth and age four (start of follow-up). Even after adjusting for such a host of individual and household characteristics, these estimates indicate that among white respondents, the odds of smoking initiation increase by 72 percent ($p<0.05$) with more prolonged exposure to high- as opposed to low-poverty neighborhoods. Among nonwhite, predominantly African American, respondents, the IPT-weighted estimates indicate no statistically significant association between the duration of exposure to more impoverished neighborhoods and smoking initiation. Overall, the findings suggest that more sustained residence in a high- versus low-poverty neighborhood is associated with smoking initiation by age 25, even after accounting for individual- and household-level factors as both mediators and confounders; however, this association is only evident among white and not nonwhite respondents. Duration of exposure to neighborhood poverty, therefore, does not appear to alter the substantive conclusions of prior cross-sectional studies in which the association between neighborhood poverty and smoking behavior is evident in white but not nonwhite youth and young adults. Moreover, when the IPT-weighted estimates are compared to the conventional regression-adjusted estimates in Model 3, which condition on time-invariant and baseline (age four) covariates as well as all time-varying covariates averaged across the follow-up period, the IPT-weighted estimates of the effect of cumulative neighborhood poverty are slightly attenuated for both nonwhite and white respondents. This suggests that respondents' differential selection into more or less impoverished neighborhoods as well as individual- and household-level factors may in fact account for some of the effects on smoking behavior of neighborhood disadvantage that have been reported in prior research.

4.5 Discussion and Conclusions

This study examined the effects of more prolonged exposure to neighborhoods characterized by varying levels of poverty on the risk of smoking initiation by age 25 utilizing the 1970 to 2011 waves of the PSID merged with census data on respondents' neighborhoods. Two primary conclusions emerge from this investigation. First, the overall risk of early smoking initiation is lower among nonwhite compared to white

youth and young adults despite higher rates of exposure to adverse socioeconomic characteristics at the individual, household, and neighborhood levels. Second, residing in a high- compared to a low-poverty neighborhood for repeated or prolonged periods of time is associated with early smoking initiation among white but not nonwhite respondents. More specifically, among whites, more sustained exposure to neighborhoods in which more than 20 percent of residents are in poverty is related to a 72 percent increase in the odds of smoking onset before age 25 compared to more prolonged residence in neighborhoods with less than 10 percent poverty rates. Whereas the majority of prior scholarship on smoking behavior generally, and age at smoking initiation more specifically, has characterized the residential environment at only one time point, the estimates presented here are based on a duration-sensitive measure of respondents' neighborhoods from age four onwards, as well as statistical methods that account for dynamic individual- and household-level factors known to predict future smoking behavior but also related to the sorting of families into and out of neighborhoods over time. As such, they provide among the strongest evidence to date for neighborhood effects on smoking initiation among white youth and young adults, over and above the influence of individual- and household-level risk and protective factors.

However, consistent with prior research on smoking behavior, no statistically significant neighborhood effects were found among nonwhite respondents. Nonwhite respondents who grew up in predominantly high-poverty neighborhoods were no more likely than those who spent most of their youth in low-poverty areas to initiate smoking before age 25. Such an absence of an effect, especially in light of the significant findings among white respondents, is particularly striking given dramatic racial disparities in exposure to neighborhood poverty throughout the early life course. Nonwhite, predominantly African American, youth and young adults at every age were often as much as eight times more likely to reside in high-poverty neighborhoods compared to their white counterparts. Nevertheless, overall smoking rates were consistently lower among nonwhite respondents and age at initiation was unrelated to the poverty rate of the neighborhoods to which they were persistently exposed. Whereas a lack of attention to the temporal aspects of neighborhood exposures has been cited as a possible reason for null findings in the broader neighborhood effects literature [65, 66], quantifying the duration of exposure to neighborhood poverty among nonwhite respondents does not appear to alter the substantive conclusions of smoking-related studies that have relied on cross-sectional data. Future research in this area should more fully investigate

the protective and/or anti-smoking strategies employed by low-income, communities of color that appear to buffer the effects of adverse neighborhood characteristics.

Although this study uses panel data and unique statistical methods to address some of the most common challenges in neighborhood effects research (namely, reverse causation and selection bias), the results should be considered in the context of several remaining limitations. First, given the historical timing of this study, the sample is limited to an aggregate “nonwhite” category (consisting of predominantly African American respondents) and a white category. Future research examining smoking behavior as a function of cumulative neighborhood effects among Asian and Latino populations is encouraged. Second, this study did not assess the psychosocial and behavioral mechanisms thought to differentially influence the race-specific associations observed between neighborhood poverty and smoking initiation. Longitudinal research able to more explicitly measure these potential mechanisms including, for example, peer pressure, parenting practices, and related sociocultural norms and attitudes about smoking in the context of racial-spatial patterns of inequality, would be enlightening. Finally, this study only assessed one aspect of the neighborhood environment: poverty. Although neighborhood poverty is often a good proxy for other aspects of structural disadvantage, such as greater tobacco retail density, future research that more explicitly measures smoking-related characteristics of the residential environment is necessary.

Nonetheless, findings from the present study support prior research indicating that nonwhite, predominantly African American, youth and young adults are less likely than whites to initiate smoking during the early life course. The fact that nearly one in four nonwhite and more than one in three white respondents in this study had started smoking before age 25, however, underscores the continued importance of tobacco prevention policies and programs that target multiple spheres of influence, from individuals and families to the neighborhood environment, although different sets of micro- and macro-level interventions may be more efficacious among different racial/ethnic groups. Viewed in light of earlier work by the author showing that children and youth who experience more prolonged exposure to neighborhood disadvantage are more likely to report worse self-rated health and to be obese as young adults, the findings from this study, although limited to white respondents, further highlight the importance of neighborhood context (and inequalities therein) for health and health behavior across the life course.

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Table 4.1. Time-invariant sample characteristics

	Nonwhite (n=808)	White (n=1,313)
Gender, percent		
Female	50.62	50.27
Male	49.38	49.73
Birthweight, percent		
Less than 88 ounces	10.64	4.80
88 ounces or more	89.36	95.20
Mother's marital status at birth, percent		
Unmarried	50.99	7.77
Married	49.01	92.23
Household head's educational attainment at birth, percent		
Less than high school	42.82	13.71
High school graduate	37.62	31.84
At least some college	19.55	54.46
Mother's age at birth, mean (SD)	23.45 (5.39)	26.11 (5.00)

Note: Statistics reported for the first of five imputation datasets.

Table 4.2. Time-varying sample characteristics

	Nonwhite			White		
	Age 4 (n=808)	Age 12 (n=630)	Age 17 (n=463)	Age 4 (n=1313)	Age 12 (n=1130)	Age 17 (n=827)
Neighborhood poverty, percent						
Low-poverty	11.01	18.41	21.60	64.66	65.84	66.38
Moderate-poverty	24.75	23.17	25.27	27.42	26.99	24.43
High-poverty	64.23	58.41	53.13	7.92	7.17	9.19
Household head's marital status, percent						
Unmarried	40.22	46.98	47.95	8.38	13.54	13.18
Married	59.78	53.02	52.05	91.62	86.46	86.82
Household head's employment status, percent						
Unemployed	31.68	30.00	29.81	8.53	7.26	7.50
Employed	68.32	70.00	70.19	91.47	92.74	92.50
Public assistance (AFDC) receipt, percent						
Received AFDC	24.75	21.43	12.74	4.04	2.65	1.09
Did not receive AFDC	75.25	78.57	87.26	95.96	97.35	98.91
Homeownership, percent						
Does not own home	68.19	55.08	46.00	31.30	17.35	12.82
Owns home	31.81	44.92	54.00	68.70	82.65	87.18
Household income in (1985) $1,000s, mean (SD)	18.11 (14.46)	21.75 (18.15)	24.99 (20.58)	36.34 (32.78)	48.21 (44.44)	63.43 (76.32)
Household head's work hours/week, mean (SD)	28.60 (19.27)	29.07 (19.69)	29.34 (19.72)	42.01 (16.06)	41.98 (14.67)	43.07 (14.66)
Family size, mean (SD)	4.83 (2.25)	4.80 (1.73)	4.59 (1.58)	4.28 (1.09)	4.46 (1.10)	4.16 (1.08)

Note: Statistics reported for uncensored respondents (first of five imputation datasets).

Table 4.3. Risk of smoking initiation by age and race

	Nonwhite			White		
Age	n	Y=1	P(Y=1)	n	Y=1	P(Y=1)
5	808	0	0	1313	0	0
6	772	0	0	1283	0	0
7	748	0	0	1259	1	0.001
8	726	0	0	1234	0	0
9	703	0	0	1200	5	0.004
10	678	1	0.001	1176	6	0.005
11	651	2	0.003	1148	4	0.003
12	622	8	0.013	1107	23	0.021
13	589	11	0.019	1062	29	0.027
14	559	5	0.009	1019	31	0.03
15	524	20	0.038	946	59	0.062
16	478	31	0.065	839	91	0.108
17	436	27	0.062	775	52	0.067
18	383	37	0.097	664	98	0.148
19	354	13	0.037	609	41	0.067
20	274	9	0.033	424	26	0.061
21	206	11	0.053	254	11	0.043
22	154	7	0.045	175	5	0.029
23	130	3	0.023	125	1	0.008
24	106	2	0.019	110	0	0
25	92	5	0.054	95	3	0.032

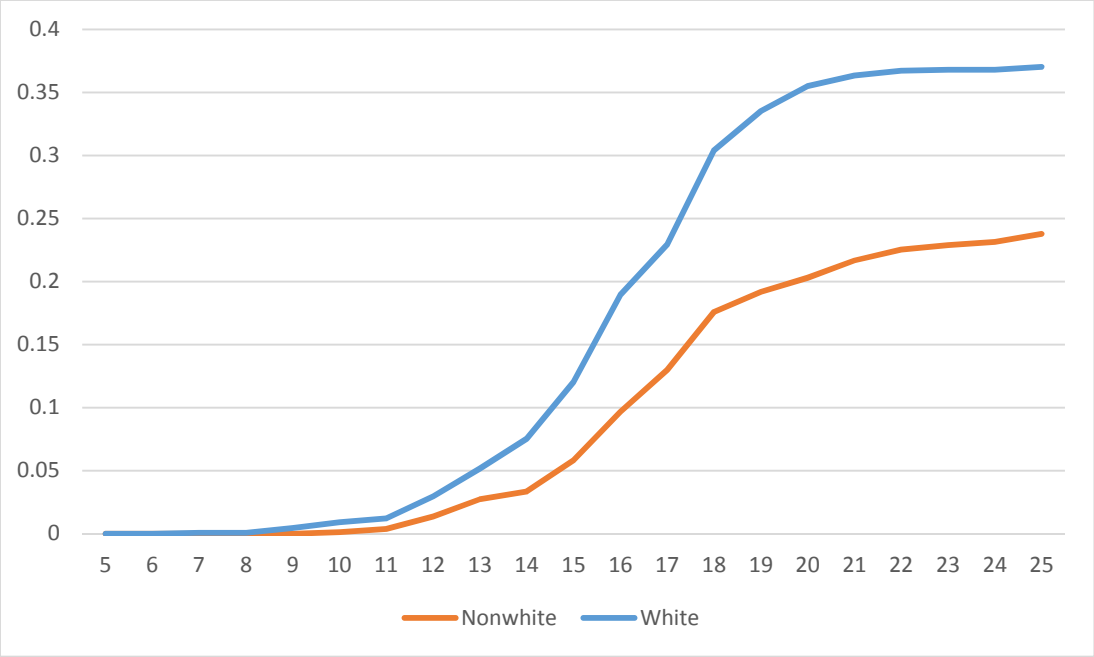

Figure 4.1. Cumulative probability of smoking initiation by age and race

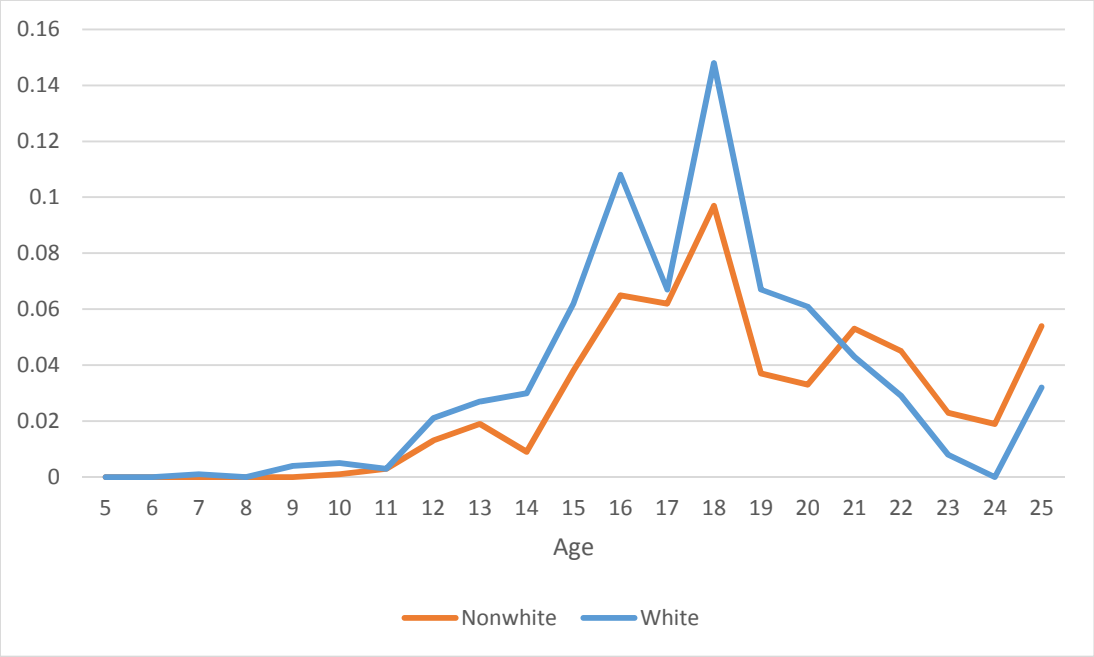

Figure 4.2. Age-specific probability of smoking initiation by race among respondents still at risk

Table 4.4. Discrete-time logit models for the effects of neighborhood poverty on the risk of smoking initiation by age 25

	Nonwhite (n=10,185)									White (n=17,303)								
	Model 1			Model 2			Model 3			Model 1			Model 2			Model 3		
	Unadjusted			IPT-Weighted			Regression			Unadjusted			IPT-Weighted			Regression		
	OR	SE	*p*	OR	SE	*p*	OR	SE	*p*	OR	SE	*p*	OR	SE	*p*	OR	SE	*p*
NH poverty, cumulative proportion (low)																		
Moderate	1.19	0.44		1.05	0.55		1.34	0.58		1.33	0.20	*	1.18	0.26		1.14	0.22	
High	1.59	0.44	*	1.46	0.68		1.51	0.58		2.66	0.58	***	1.72	0.46	**	1.94	0.49	***
Age	5.37	1.09	***	5.55	1.42	***	5.46	1.21	***	7.07	1.07	***	7.64	1.42	***	7.79	1.20	***
Age squared	0.96	0.01	***	0.96	0.01	***	0.96	0.01	***	0.95	0.00	***	0.94	0.01	***	0.94	0.00	***
Gender (male)																		
Female				0.77	0.13		0.68	0.11	*				0.88	0.09		0.87	0.08	
Birthweight (88 ounces or more)																		
Less than 88 ounces				0.81	0.28		0.78	0.24					0.67	0.18		0.66	0.17	
Mother's marital status at birth (married)																		
Unmarried				0.89	0.20		0.91	0.19					0.96	0.27		0.93	0.23	
Mother's age at birth				1.01	0.02		1.01	0.02					0.98	0.01		0.98	0.01	*
HH's education at age 4 (less than HS)																		
High school graduate				0.82	0.18		0.83	0.16					0.57	0.10	***	0.60	0.09	***
At least some college				0.86	0.24		0.87	0.21					0.40	0.07	***	0.44	0.07	***
NH poverty at age 4 (low)																		
Moderate				1.01	0.44		0.90	0.31					0.94	0.17		0.96	0.15	
High				1.15	0.50		1.07	0.36					0.95	0.25		0.89	0.07	
HH's marital status at age 4 (married)																		
Unmarried				1.24	0.28		1.18	0.30					1.17	0.32		1.03	0.26	
HH's employment status at age 4 (employed)																		
Unemployed				0.93	0.23		0.86	0.21					0.69	0.20		0.76	0.17	
Homeownership at age 4 (owns home)																		
Does not own home				1.34	0.29		1.13	0.27					1.11	0.15		1.03	0.15	
Family size at age 4				0.95	0.04		0.92	0.05					1.00	0.06		1.07	0.08	
AFDC receipt at age 4 (no AFDC)																		
Received AFDC				2.19	0.48	***	1.56	0.37	*				1.16	0.49		0.66	0.25	
Household income at age 4				1.08	0.18		1.01	0.14					1.06	0.07		1.01	0.08	
HH's work hours per week at age 4				1.01	0.01		1.01	0.01					0.99	0.00		0.99	0.00	
Year born (1966-1969)																		
1970-1974				0.72	0.25		0.76	0.20					0.94	0.16		0.90	0.14	
1975-1979				1.02	0.31		1.09	0.28					1.38	0.22	**	1.41	0.21	**
1980-1984				1.48	0.42		1.43	0.37					1.20	0.20		1.18	0.18	
1985-1989				1.13	0.41		1.10	0.40					0.67	0.15	*	0.68	0.15	*
1990-1993				0.54	0.62		0.62	0.68					0.38	0.34		0.40	0.30	
HH's marital status, average							0.88	0.27								1.57	0.41	*
HH's employment status, average							4.94	2.84	***							1.46	0.69	
Homeownership, average							1.26	0.38								1.00	0.21	
Family size, average							1.04	0.08								0.86	0.07	*
AFDC receipt, average							1.12	0.45								1.08	0.56	
Household income, average							1.00	0.00								1.00	0.00	
HH's work hours per week, average							1.03	0.01	*							1.00	0.01	

Odds ratios (OR) and standard errors (SE) are combined estimates from five multiple imputation datasets
NH=Neighborhood; HH=Household head
*p<0.10; **p<0.05; ***p<0.01

Chapter 5. CONCLUSION

> *"…we cannot limit our attention to characteristics of individuals and families, to policies targeting individual poverty, or to macro-level forces leading to growing income inequality. We must also consider places. We must consider the various sets of forces that affect neighborhoods and cities and the ways that the trajectories of people and places are connected over time."*
>
> - Patrick Sharkey, *Stuck in Place* (2013)

The introductory chapter of this dissertation began with a basic claim: the geography of disadvantage and the geography of poor health are closely aligned. A considerable body of prior research has linked neighborhood and community-level characteristics, particularly concentrated poverty and other forms of socioeconomic disadvantage, to premature death [1, 2], poor self-rated health [2-5], depression and other mental health problems [6-9], health-risk behaviors such as alcohol abuse, tobacco use, poor diet, and physical inactivity [10-13], and related chronic conditions such as obesity, diabetes, cardiovascular disease, and certain cancers [14-18], net of individual- and household-level factors. Yet conventional paradigms about the individualistic nature of health coupled with (1) a reliance on cross-sectional data and (2) methodological concerns about reverse causation and selection bias continue to inhibit the translation of such research into health-related policies and practices that target the characteristics of *places* in addition to the characteristics of *people*.

The findings presented in this dissertation add to extant scholarship documenting neighborhood effects on individual health and racial disparities therein. However, they move research in this area forward in several key respects. First, data for all three empirical chapters are drawn from the 1970 to 2011 waves of the Panel Study of Income Dynamics (PSID) in which the same respondents are followed from early childhood through adolescence and into young adulthood. Such a longitudinal data structure makes it possible to account for the temporal ordering of variables, virtually guaranteeing that exposure to neighborhood disadvantage precedes the incidence of a health outcome of interest. Second and relatedly, neighborhood characteristics (as well as individual- and household-level factors) are measured at regular intervals throughout the child and adolescent life course, allowing the values on such indicators to vary across time as well as for the creation of duration- and timing-specific measures of exposure to neighborhood disadvantage. Third, analyses employ marginal structural models in which the parameters are estimated using inverse probability of treatment (IPT) weights. This statistical approach has two primary advantages over conventional regression-based techniques: (1) it

uses weighting to simulate random assignment of exposure to neighborhood disadvantage, and (2) in so doing, it allows time-varying individual- and household-level characteristics to act simultaneously as confounders and mediators of the basic neighborhood-health relationship. The associations among individual-, household-, and neighborhood-level characteristics and health are dynamic and interrelated. Embracing this theoretical and methodological complexity in research is essential for advancing evidence-based interventions that create neighborhood environments that support rather than hinder individual health over the life course and across generations.

5.1 MAIN FINDINGS

> *"We need more life course research, which will require longitudinal studies with extensive multilevel information on social and physical context as well as biology and behavior over time."*
>
> - Paula Braveman and Colleen Barclay, *Pediatrics* (2009)

There is a well-known literature detailing the so-called long arm of early-life individual- and family-level disadvantage on health in older ages, including the effects of family poverty in childhood and early-life adversity and toxic stress on later-life physical and mental well-being and mortality [19, 20]. As already discussed, a related but often separate body of research has further shown that neighborhood-level disadvantage is associated with health, although the vast majority of this work has relied on cross-sectional data. An overarching aim of this dissertation was to bring these two literatures into more explicit dialogue by situating the study of neighborhood effects on health in a life course perspective. More specifically, the three empirical chapters conducted for this dissertation are part of a larger research agenda aimed at examining the impact of place and concomitant social and structural processes on population patterns of health and illness across the life course. Central to this objective is the notion that the places in which individuals live, learn, work, and grow over time, particularly residential neighborhoods, function as a fundamental means of social stratification and therefore play a crucial role in the production and perpetuation of inequalities in health.

Consistent with this assertion, the results presented in the three preceding chapters document a prominent and persistent racial-spatial divide along residential lines. Nonwhite, largely African American, respondents are not only more likely than whites to ever reside in neighborhoods characterized by high levels of structural disadvantage and disinvestment, but they are also more likely to be born into and remain in similar types of environments for repeated or prolonged periods of time. More notably, the studies detailed here are among

the first to show that more prolonged exposure to such adverse neighborhood conditions throughout the child and adolescent life course and in particular, from ages 10 to 17, has deleterious impacts on future (young adult) health, including increased risk of self-rated fair or poor health (Chapter 2) and obesity incidence (Chapter 3). Together, these findings indicate not only that the duration and timing of exposure to neighborhood disadvantage matter for health generally, but also that the separate and unequal residential environments in which nonwhite versus white children and youth tend to grow up likely play a critical role in producing and perpetuating racial disparities in health in early adulthood and beyond. Such results underscore the long-term influence of past exposures, including those at the neighborhood level, and further highlight that attending to the residential environments of children and youth is imperative for improving adult health and reducing disparities therein.

The finding in Chapter 4 that more prolonged exposure to high-poverty neighborhoods is associated with early smoking initiation adds support to the general conclusion that neighborhoods are consequential for health, and that such effects may be sensitive to the duration (and timing) of exposure. However, the fact that this result was only observed among white and not nonwhite (predominantly African American) respondents is more difficult to explain, especially in light of dramatic and persistent racial inequalities in the quality of neighborhood resources and opportunities as well as largely consistent adverse neighborhood effects on other health compromising behaviors and conditions. Nonetheless, this is a fairly common finding in the broader tobacco use literature. Smoking prevalence is consistently shown to be lower among African American adolescents than their white counterparts [21], although black/white differences in cigarette use are considerably reduced by adulthood and smoking-attributable morbidity and mortality still tends to be greater among African American than white adults [22]. Thus, although studies often find that the influence of neighborhood context is limited to smoking behavior among white and not African American youth [23-26], it is possible that neighborhood characteristics exert effects on tobacco-related outcomes later in the life course and through different pathways among nonwhite groups. However, the question of why neighborhood poverty is a stronger predictor of smoking initiation for white than nonwhite young people remains to be explored in future research including, for example, investigations into racial differences in susceptibility to peer versus parental influences and in social norms and desirability around smoking versus other licit and illicit health-risk behaviors.

5.2 LIMITATIONS

Although this dissertation uses panel data and unique statistical methods to address some of the most common challenges in neighborhood effects research, there are several remaining limitations worth noting in more detail. First, the absence of completely “tracted” land in 1970 and 1980 resulted in missing census data on neighborhood characteristics and necessitated imputation, which may have led to some misclassification of neighborhood disadvantage, especially in nonmetropolitan areas. However, the two-fold multiple imputation algorithm used to model this information was based on an extensive set of individual- and household-level factors known to be strongly related to the neighborhood disadvantage characteristics assessed in this study as well as census data at adjacent time periods. Since the entire country was tracted by 1990, studies in the near future will be able to examine a similar length of the life course, from birth through early adulthood, without having to include decades in which information on census tracts may be missing by default. Second, as with most longitudinal designs, sample attrition in the first two empirical studies is relatively high. However, the censoring weights used in conjunction with the IPT weights to generate the pseudo-population to which the final marginal structural logistic regression models are fit, up (or down) weight those individuals who are more (or less) likely to drop out of the sample at each wave, effectively randomizing sample attrition across survey waves. Third, given the historical timing of this study, the sample is limited to a cohort of predominantly African American and white respondents. Future research examining health (disparities) as a function of prolonged and timing-specific neighborhood exposures among Asian and Latino populations, as well as across different cohorts, is encouraged.

Fourth, discriminatory housing policies and practices, including restrictive covenants, redlining, residential steering, and differentials in interest rates, subprime loans, and foreclosures, helped produce and continue to perpetuate the distinctive separation of whites and nonwhites in residential space [27, 28]. It would be ideal (although perhaps not realistic) to find a sample in which neighborhood stratification by race is less pronounced in order to further disentangle the mechanisms behind the relationships among race, place, and health. Fifth and relatedly, the studies in this dissertation did not assess the mechanisms thought to explain how neighborhood-health effects transpire but rather the broader social and structural environments thought to engender such processes. Longitudinal research able to more explicitly measure these potential risk (and resilience) mechanisms, including, for example, network ties, collective efficacy, institutional resources, and

environmental toxins, would be enlightening. Finally, the PSID has many unique strengths and its utility for health research has grown considerably over the years. However, the available measures of health are limited to a relatively few self-reported general health status and chronic disease outcomes that are often only assessed among household heads and their spouses. Including a wider range of outcomes, including more objective indicators and biomarkers of health status, that are evaluated throughout the life course may help identify additional levers of intervention, especially among younger populations.

5.3 Implications for Policy and Practice

> *"Public health agencies and organizations will need to work with those who are best positioned to create policies and practices that promote healthy communities and environments and secure the many co-benefits that can be attained through healthy public policy."*
>
> - Adewale Troutman and Georges C. Benjamin, *Health in All Policies* (2013)

The basic premise at the core of this dissertation is that neighborhood social, physical, and economic characteristics have a far greater impact on population patterns of health and well-being than is normally recognized given the predominant focus on individual lifestyle factors and medical care. The policies and practices intended to address health, therefore, cannot be limited to those agencies and institutions directly charged with protecting and improving the public's health. Rather, diverse partners and stakeholders must work together to create communities in which health considerations are institutionalized across the many sectors that influence the places in which individuals and families live, learn, work, and grow, including housing, employment, transportation, business, food, the environment, and education [29]. Doing so requires sustained investments in the basic structures of all communities, but in low-income communities and communities of color in particular. Such a place-based investment approach to addressing neighborhood effects on health is often contrasted with strategies that focus on moving residents out of disadvantaged areas, or what have been termed mobility programs and include the well-known Gautreaux and Moving to Opportunity (MTO) interventions in which residents were given vouchers to move into less impoverished neighborhoods [30]. Evidence on the effectiveness of these mobility strategies has been mixed, although there are compelling reasons to believe that at least part of this inconsistency is due to differences in the durability of such moves. Consistent with the findings presented here on the importance of the duration of neighborhood exposures, the most promising outcomes (health and otherwise) are generally found among those families who were able to make seemingly permanent rather than short-term moves out of disadvantaged areas. Whether affecting such

sustained mobility can be achieved on a scale larger than the few-city Gautreaux or MTO efforts is questionable [30-32], as are the potential implications, through such a predominant emphasis on moving poor people out of poor places, that people rather than places are at the heart of *why* neighborhoods matter for health.

More specifically, if the mechanisms underlying the relationship between neighborhoods and health are collective in addition to individual in nature, as argued throughout this dissertation, then addressing the health consequences of prolonged and timing-specific exposure to neighborhood disadvantage does not necessarily require moving poor families out of such areas. The relationship between poor health and neighborhood disadvantage is not inevitable nor the result of some innate quality of poor people of color but rather, as Patrick Sharkey and others have argued, "because areas composed primarily of poor racial and ethnic minorities have been the object of severe disinvestment and abandonment for most of the past half century" [30]. What would addressing this historical legacy of marginalization look like? What would it mean to improve health through investments in the characteristics of places? First and foremost, the results of this dissertation underscore the need for place-based programs and policies that provide structural support, resources, and opportunities over sustained periods of time, ideally from birth through adulthood. Point-in-time interventions or those in which the effects are likely to fade away quickly are unlikely to overcome the cumulative influence of repeated or prolonged exposure to neighborhood disadvantage.

Among the most well-known examples of such a far-reaching intervention is the Harlem Children's Zone (HCZ) [33], a pipeline of resources and services designed to provide children with a consistent, comprehensive system of supports from birth until college graduation through education, social services, family support, health, and community-building programs. On one level, HCZ primarily provides a charter school education for those children lucky enough to win admission through a lottery. Arguably, however, the success of HCZ is grounded in its place-based approach to transforming an entire neighborhood by saturating nearly 100 Harlem blocks – the entire neighborhood "zone", so to speak – with the ideals, services, and resources that are provided in the charter schools. In this way, the efforts and successes of HCZ are intended to extend beyond just the children and families that attend the schools and participate in the affiliated programs. They are meant to "infect" the entire area with a continuum of resources, opportunities, and supports, from birth onwards, which are crucial for cultivating a more healthful and thus productive cadre of adult citizens. A few of HCZ's programs address health-related issues directly through, for example, the provision of healthy meals, nutrition education, and physical activity classes; however, the vast majority of HCZ services target the broader social determinants of

health – “the conditions in which people are born, grow, live, work, and age, and which are shaped by the distribution of money, power, and resources” [34], such as employment and income, family and household structure, social support and isolation, education, housing, transportation, and social institutions, among others [35]. Preventative action on these nonmedical factors helps people avoid getting sick or injured in the first place as well as helps to buffer the effects of poor health if it does occur.

5.4 FUTURE RESEARCH

In addition to the program and policy implications outlined above, this dissertation sets the stage for a series of future research projects, three of which seem particularly well-suited to more immediate investigation and further discussion here. The first potential project pertains to the intergenerational transmission of prolonged and timing-specific neighborhood effects on health. In particular, does the duration and timing of a mother’s exposure to varying degrees of neighborhood disadvantage throughout her childhood and adolescence affect the health and well-being of her future children, including indicators of cognitive development, behavior, and ongoing health status? This project would utilize data from the Child Development Supplement of the PSID, which includes extensive information on physical health, emotional wellbeing, intellectual achievement, and socialization for a cohort of children who were age zero to 12 in 1997, to more fully explore the notion that neighborhood effects on health depend not only on where someone lives today, but also on where they, as well as their parent(s), lived in the past. The supplemental data on children could then be appended to the more than four decades of geocoded demographic and economic data on their mothers derived from the main PSID interview. To the extent that the neighborhoods in which mothers grow up influence not only their own psychosocial and physiological development, but also the resources and opportunities available to them for education, employment, mobility, health care, and childrearing, it is hypothesized that neighborhood effects will extend well beyond a single generation.

The second potential project aims to explore if and how physiological processes (e.g., endocrine, immune, metabolic, inflammatory, and cardiovascular functions) interact with social and structural processes (e.g., informal social control, collective efficacy, institutional resources, and the content and quality of the built environment) in response to neighborhood-level stressors, such as concentrated poverty and racial residential segregation during adolescence as well as the transition from adolescence to adulthood. This project is inspired by past critiques from prominent scholars that most research on neighborhoods and health is still

attempting to establish *that* rather than *why* context matters. Data could be drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health), which includes both individual- and contextual-level socioeconomic data as well as multiple biomarkers of stress. As such, it comprises one of the only data sources from which to glean empirical insights into the physiological mechanisms through which neighborhood characteristics and concomitant social and structural processes "get under the skin" to affect health across significant portions of the life course. Whereas the empirical portion of this dissertation necessarily relied on subjective indicators of general health status and self-reported measures of height and weight and smoking behavior, using Add Health for this new project would allow for the inclusion of objective physiological markers of emerging disease, which can be difficult to capture in younger adult populations when conscious or clinical manifestations have yet to surface. Including biomarkers in longitudinal neighborhood effects research may thus provide more sensitive indicators of health to help build a more compelling case for place-based policies and practices that address population health and inequalities therein.

Finally and more concretely, in an anticipated collaboration with Drs. Jeffrey Morenoff (University of Michigan) and David Harding (University of California, Berkeley), the third potential project would explore how neighborhood inequalities affect race and gender disparities in substance use, other mental health-related outcomes, and mortality among former prisoners released on parole. Despite renewed interest in integrating former prisoners back into society, only a handful of recent studies have examined the role of residential environments and concomitant social and economic resources in shaping such processes [36, 37]. So far this research has focused on housing and employment related factors. Relatively little is known about the effects of pre- and post-incarceration neighborhood characteristics on substance use and other mental health conditions among former prisoners, which may in turn be important predictors of successful social and economic reintegration and desistance from crime. This project would utilize a unique panel dataset derived from detailed administrative records on a randomly selected sample of former prisoners who were placed on parole in Michigan during 2003, including substance use and residential treatment data along with information on housing, employment, and recidivism from parole agent case notes. Census data on neighborhood socioeconomic characteristics and if applicable, information on causes of death from the National Death Index would also be appended to parolee records. The recidivism and reintegration-related consequences of substance abuse and other mental health problems among former prisoners likely vary by the social and structural characteristics of their pre- and post-incarceration neighborhoods. Documenting such variation has

the potential to aid in the development of place-based policies and practices that help reduce prison cycling as well as affect the broader social and institutional consequences of disproportionately high rates of both incarceration and reentry on low-income communities and communities of color over lifetimes and across generations.

Collectively, these three future research projects can contribute to better understanding and addressing the temporal dimensions of neighborhood effects on population patterns of health and illness. They reflect mounting interest in the biopsychosocial mechanisms that influence the social and spatial patterning of health within and across generations, and are intended to be collaborative and multidisciplinary. While they are consistent with and draw extensively from extant work in sociology, public health and related fields in the social and biomedical sciences, the proposed projects aim to evaluate a number of underexplored facets of the dynamic relationship between neighborhoods and health, including (1) the intergenerational transmission of neighborhood disadvantage, (2) the intersection of upstream causes, downstream circumstances, and the meso-level mechanisms through which neighborhood-health effects transpire, and (3) the ways in which the causes and health-related consequences of incarceration are moderated by neighborhood context. Findings have the potential to add support to the growing body of literature suggesting that durable, place-based investments in the social, economic, institutional, and physical structures of disadvantaged neighborhoods and neighborhoods of color can have long-term benefits for population health and health equity that extend over the life course and across generations.

5.5 Concluding Remarks

This dissertation sits firmly within the large body of research on neighborhoods and health, adding support to the fundamental claim that where someone lives matters for their health. But such a general conclusion, although essential to an expanded understanding of health, is no longer sufficient nor particularly effective for translating research into policy and practice. The three studies conducted for this dissertation (as well as the proposals for future research) are intended to strengthen our confidence in and further our understanding of why and how (as opposed to just if) neighborhoods influence health, focusing in particular on the dimension of time. The omission of such a temporal element from most prior work has severely limited our ability to address two of the prevailing tensions in this literature – namely, selection bias and the extent to which neighborhood-health effects are due to compositional versus contextual factors. Longitudinal studies of neighborhood effects

on health, such as those conducted here, are better able to capture the complexity reflected by the aforementioned tensions. In brief, the demographic and socioeconomic characteristics of individuals influence their residential choices as well as their health. As such, it is possible that what may seem like a neighborhood effect on health is actually due to the individual (or compositional) characteristics of the people who are more (or less) likely to live in different places (i.e., selection bias).

As a result, research on neighborhoods and health has often attempted to isolate the effects of people from the effects of places. Such an "either/or" conceptualization, in conjunction with cross-sectional data limitations, has led to the use of statistical models that simply control for individual-level variables as if people and the places in which they live have competing rather than spatially and temporally interrelated influences. In fact, as the analyses for this dissertation reflect, the relationship between individual characteristics and residential choice is reciprocal. That is, residential mobility decisions are a function of individual demographic and socioeconomic factors, such as race, income, education, and home ownership, but these individual-level factors are also affected by the neighborhoods in which people "choose" to live. Simply controlling for individual- and household-level variables in an attempt to isolate the effects of neighborhoods on health therefore distorts the reality that individual demographic and socioeconomic factors are in fact intervening variables on the pathways between neighborhoods and health, not merely confounders as they are so often construed [2, 38].

This dissertation is grounded instead in a "both/and" approach to the effects of people and the effects of places on health. Individual- and household-level characteristics are considered important mediators of the effect of neighborhoods on health, yet their role as potential confounders cannot be ignored either. The assessment of individual-, household-, and neighborhood-level characteristics repeatedly over time facilitated the use of statistical methods that were able to account for selection bias without controlling away neighborhood effects that may operate indirectly through the same individual- and household-level covariates that have been associated with residential mobility. As such, the findings from this dissertation provide among the strongest evidence to date for the consequences of prolonged exposure to neighborhood disadvantage throughout childhood and adolescence on future (young adult) health outcomes, especially among nonwhite, largely African American, children and youth for whom such exposures tend to be more common and more persistent. The additional finding, highlighted in Chapter 3, that neighborhood effects may be more consequential during the adolescent phase of the life course, however, also helps reiterate that policies and

programs to address health should be comprehensive and target multiple spheres of influence from the micro to the more macro levels depending on the age of the intended recipients and perhaps, their race as well as the health outcome of interest (as suggested by the race-specific results in Chapter 4 on smoking initiation). Nonetheless, the confluence of increasingly sophisticated research on neighborhoods and health with mounting attention to the social determinants of health from both the philanthropic and policy sectors suggests that the window of opportunity for change may be wider than ever before.

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