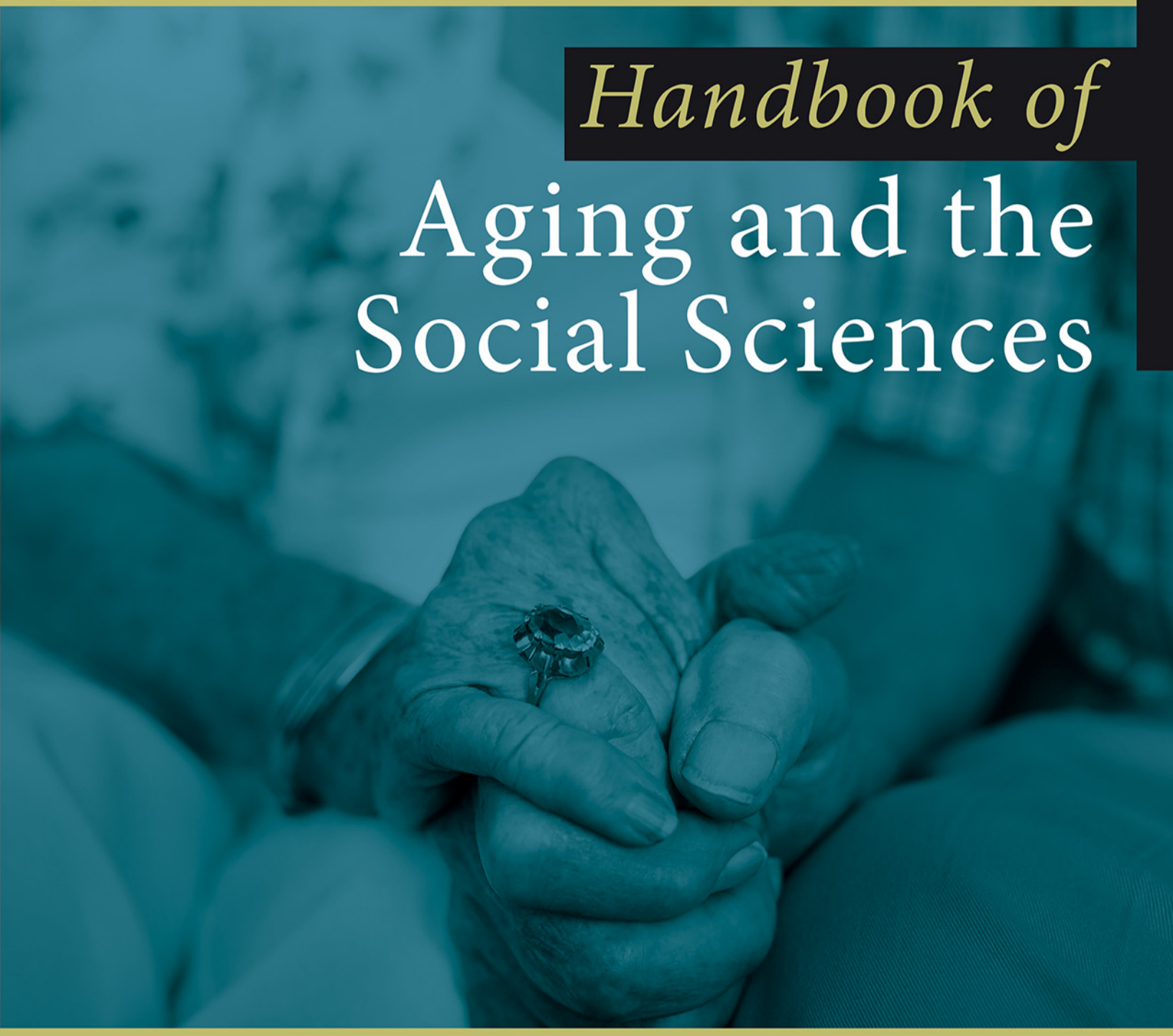
Ninth Edition

# *Handbook of* Aging and the Social Sciences



Edited by
**Kenneth F. Ferraro**
**Deborah Carr**

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# *Handbook of* Aging and the Social Sciences

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The ***Handbook of Aging and the Social Sciences 9e*** provides a comprehensive synthesis and review of research in the social sciences of aging. The handbook includes both foundational, classic themes of aging research, and emerging topics. Geographic, socioeconomic, cultural, and generational factors shape the context of the aging process, including the risks humans face and the resources available to them. The handbook identifies factors influencing optimal aging by discussing how biology and behavior may jointly influence outcomes.

The volume is organized in four parts: scientific approaches and methods; structural constraints and adaptation; social institutions; and aging and social intervention. New topics include sexuality, technologies of aging, and genetics. New content in core topics focuses on innovative perspectives bolstered by cutting-edge data and methods. Examples include coverage of the socioeconomic gradient in health and generational differences in political participation.

**Key Features:**

- Reviews and synthesizes relevant research from multiple disciplines
- Covers demography, economics, political science, psychology, sociology, and more
- Identifies factors influencing healthy aging
- Includes new chapters and new coverage in core chapters
- Serves as a reference, an upper level textbook, and as a guide for needed research

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CHAPTER

# 9

# Early origins of racial health disparities: human capital policy is health policy

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OUTLINE



## Introduction

Two strands of research—(1) racial health disparities and (2) early-life origins of adult disease—are typically studied separately. But they are in fact parallel problems that must be considered together in order to make significant progress on either question from both a "research-to-impact" perspective and in service of the development of transformative policy and practice. The first of these research areas has devoted significant attention to the impacts of segregation and role of neighborhood quality. The second has employed a life course perspective and human capital theoretical framework to provide central insights. At the intersection of these two prominent highways of research lies the path to new discoveries to develop effective interventions wherein prevention is the best cure.

Blacks have been shown to experience earlier onset of illness, have more aggressive progression of disease, and suffer poorer survival rates across various conditions (Johnson, 2018; Williams, 2012). In particular, black men have the lowest average life expectancy, and among the most significant contributors are chronic diseases which are amenable to primary and secondary prevention. Significant racial health disparities exist at all ages, as black men have a 70% higher risk of developing heart failure than white men, while black women are 3.4 times more likely to die from pregnancy or childbirth-related complications than white women (National Center for Health Statistics, 2017). A voluminous literature has documented these and other racial health disparities, but few studies have cleanly identified the causal sources that explain their large magnitude or earlier life origins. Moreover, these disparities persist even though the stubbornly

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wide gap in life expectancy between whites and blacks has declined by 50% since 1990 (from 7 years to 3.5 years), and is now at its lowest level ever. This apparent paradox has received scant attention but life course analysis is key to understanding it.

Most studies of older adults or aging populations neglect analyses that account for the influence of early-life and childhood factors on current health status and chronic disease. Data limitations have often been a driving factor of why there has been considerably less attention in analyses of aging populations at earlier stages of the life course. However, recent research contributions using longitudinal studies of younger cohorts for aging research are uncovering important new insights pertinent to understanding sources of current racial health disparities. Prominent examples include analyses of data from the Panel Study of Income Dynamics (PSID), the Health and Retirement Survey (HRS), the National Longitudinal Study of Adolescent to Adult Health (Add-Health), and showing a significant portion of racial health disparities in adulthood are rooted in childhood conditions (Johnson, 2018).

In the last decade we have seen an explosion of research across multiple disciplines on the nonmedical, socioeconomic determinants of health (Adler et al., 2017). Exploration of potential effects of structural racism is an extremely active research area in this field, which is a rapidly expanding literature in numerous directions (Williams, Lawrence, & Davis, 2019). The reason for enduring interest into the sources of racial health disparities stems in part from their large magnitude at every age, persistence across generations, and evolution over the life course, which requires insights from multidisciplinary perspectives. There has been a resurgence of interest in segregation, and resultant differences in childhood neighborhood quality, as a fundamental cause of health disparities. This has accompanied the related large body of research on the consequences of high and rising income inequality, which has been experienced increasingly since 1980 in the United States. Part of what ignites this work is the fact that while more highly educated, affluent individuals experience better health and live longer, socioeconomic status alone does not account for racial health disparities (Braveman, Cubbin, Egerter, Williams, & Pamuk, 2010). In fact, college-educated blacks are in worse health on average than non-Hispanic whites who did not graduate from high school. That is, college-educated individuals can expect to live about 10 years longer than their less educated counterparts on average, but the health benefit of educational attainment for minorities appears to be lower and the black−white gap in life expectancy is greater at higher levels of education (Braveman et al., 2010).

Therein is the puzzle: why are there large racial/ethnic disparities in health at middle age and older ages even among those with the same levels of adult socioeconomic status? An essential component of the answer lies in what happens in childhood and the socioeconomic mobility process that occurs in young adulthood when minorities achieve those levels of SES.

The purposes of this paper are first, to discuss the research on the strength of the link between segregation and racial health disparities over the life course in the United States, and second, to provide an evidence-based review of the effectiveness of existing policies and targeted interventions designed to address unequal opportunities that accompany segregated environments in childhood. Programs that ensure more equitable opportunities and hold promise to narrow racial and SES gaps encompass education, public health, and housing policies and the social safety net—as will be argued, human capital policy is health policy. We focus the review primarily on empirical studies with credible research designs that enable causal inferences, along with those that use innovative data and measures to elucidate previously unmeasured processes, with emphasis on those published since 2000. This is important as simple comparisons of the health of residents in different quality neighborhoods, or between program participants and nonparticipants, yield misleading conclusions. The paper unfolds as follows. The second section provides a theoretic framework and outlines conceptual issues underlying the multilevel, developmental influences of racial inequality in health over the life course that connect research in this area with those in the biological and social sciences. The third section lays out the evidence-based argument that human capital policy is health policy and vice versa. This section also summarizes research on the efficacy of human capital and health policy interventions. The chapter concludes with a summary discussion of promising directions for future research.

## Conceptual issues and potential mechanisms

While popular media and news coverage of US health inequality in recent years have highlighted stagnant life expectancy and increasing inequality in mortality, that attention has been focused on trends among middle-aged and older adults, and non-Hispanic whites (e.g., *New York Times*, February 12, 2016). There are vastly different trends in inequality in mortality for children versus adults, and for blacks, which each highlight the importance of a life course perspective to understand the sources of these changes in population health. Inequality in mortality in the United States has declined significantly among children since 1990, and

now approaches the low levels observed in developed countries such as Canada (Currie, 2018). The United States still lags other advanced developed countries in early childhood health particularly in the significantly higher levels of health inequality across racial/ethnic and socioeconomic groups. For example, US infant mortality rate was ranked 32nd among the 34 countries of the OECD in 2010 with black women having the highest maternal mortality rate (Centers for Disease Control and Prevention CDC, 2013), but substantial increases in public investments over the past several decades have significantly improved these childhood conditions with disproportionate beneficial effects experienced for low-income and minority children.

Three important policy reforms played significant roles in this improvement: (1) higher proportions of more recent cohorts have health insurance coverage prenatally and in childhood [due to Medicaid and State Children's Health Insurance Program (SCHIP) expansions]; (2) higher public spending on social programs for children [e.g., Earned Income Tax Credit (EITC), food stamp and related nutrition programs (SNAP), lead abatement programs]; and (3) lower child poverty as demonstrated by trends in the supplemental poverty measure inclusive of taxes and transfers [from 30% in 1985 to 15.6% in 2015 (Fox, Wimer, Garfinkel, Kaushal, & Waldfogel, 2015)]. Before detailing the new collection of evidence on these issues, we first outline the potential mechanisms behind early-life conditions, including segregation and neighborhood quality, that have important life course implications for health.

An important takeaway that motivates the discussion is that there is not a single cause driving trends in health disparities, but multiple specific causes that are differentially affecting racial and socioeconomic demographic subgroups—blacks differently than Hispanics, Asians differently than whites—and that vary substantially in their salience over the life course. This is in part due to the contours of residential segregation and the SES gradient of early-life conditions. This chapter will focus on black–white differences, but will also touch on disparities for other underrepresented minorities to illustrate similarities and differences in how disparities emerge and persist. To integrate the two aforementioned topics into a single conceptual framework, we embed our study of racial health disparities within a life course perspective to examine the evolution of health—an approach adopted by various disciplines including economists (human capital modeling framework), epidemiologists, developmental psychologists, and sociologists. A conceptual framework is outlined of how public investments in early childhood and human capital development may help address the intergenerational transmission of disadvantage, and ameliorate the persistence and reproduction of health inequality across generations.

## Early-life events

The early-life origins of adult disease may begin in the womb. When a fetus receives limited nutrition, its metabolic and physiological makeup fundamentally changes. While the consequences may not be evident at birth, or even in early childhood, they can appear much later in life, manifested as early onset of hypertension, increased risk of cardiovascular disease, stroke, and diabetes. This phenomenon is known as the "fetal origins hypothesis," developed by epidemiologist David Barker (1998). A voluminous empirical literature supports Barker's hypothesis, mostly drawn from the UK (see Barker, 1998 for a review), and I have previously documented the long-run impacts of poor infant health in the United States using the PSID (Johnson & Schoeni, 2011), where black children in the United States are twice as likely to be born low weight.

Along with the lifelong consequences of prenatal nutritional deprivation that are both persistent and latent, with some manifestations not observed until middle age, the medical literature posits stress as an important biosocial mechanism through which adverse early-life health conditions may lead to problematic health later in life. Emerging research has sought to identify whether, and how, early life differences in access to health care and exposures to stressful life conditions "get under the skin." Early-life experiences of toxic stress, even in the womb, may have profound implications for later-life health (Aizer, Stroud, & Buka, 2016; Jaddoe, 2006; Johnson & Schoeni, 2011). Specifically, when a fetus is exposed to excessive amounts of the stress hormone cortisol in utero, there can be a reprogramming of the hypothalamic–pituitary–adrenal axis (HPA) that impairs fetal development (Dunkel-Schetter, 2011). Recent findings in neuroscience highlight prolonged exposure to stress hormones (e.g., cortisol) can suppress the body's immune response and cause greater vulnerability to chronic health conditions (Shonkoff & Garner, 2012). More generally, recent evidence in the developmental origins of adult disease and neuroscience literatures emphasizes the critical period of development from conception to age five as one that is extremely sensitive to stressful environmental conditions, as the speed of growth is more rapid than any other stage of the life course and the nutritional and health care needs are greatest [e.g., see Lynch & Smith (2005) for a review; Heckman (2007), and references therein].

Insights from developmental neuroscience have been a catalyst for the life course approach to health and human capital formation, revealing that in the first 3 years of life, the brain develops at astonishing speed, forming about one million new neural connections *each*

*second* (Harvard Center on the Developing Child, 2009). By the age of three, a child's brain weighs 90% of what it will weigh in adulthood (Dekaban & Sadowsky, 1978). What's most vexing is how much poverty can affect neurologic development, condemning a child to a life of underachievement and elevated health risks before he or she has even learned to walk. A host of studies show that early-life poverty produces higher risks of impaired social and cognitive development as well as greater sensitivity to stress (Shonkoff & Garner, 2012). Nurturing, interpersonal interactions and play-based educational enrichment in the preschool years can release a cascade of neurotransmitter development and synapse growth in the young developing brain.

Recent findings in neuroscience also indicate that developmental health trajectories can be altered more readily during sensitive periods of rapid developmental change than during other periods. Heckman (2007) emphasizes that, "common developmental processes are at work where some cognitive and noncognitive skills and health capabilities at one stage in childhood cross-fertilize the productivity of investment at later stages" (p. 13254). Research evidence from this field increasingly supports the notion that the greatest opportunities to invest in health occur during the first 20 years of life. This suggests a need to shift some of the emphasis on treatment in later stages of disease toward the promotion of earlier, more effective prevention, and an investment-oriented approach to health spending aimed at its most productive uses.

Chronic health conditions, like hypertension, typically grow out of socioeconomic conditions over a lengthy period rather than from circumstances at a single point in time. The life-course perspective taken here emphasizes that health problems early in life could affect health later in life because the problem is chronic, because the health shock damaged health stock making it more susceptible to deterioration later in life, and because the health problem affects socioeconomic outcomes such as education, which in turn influences health later in life (Kuh & Wadsworth, 1993). Poor black children are less likely to escape poverty than poor white children (Bhattacharya & Mazumder, 2011). Johnson (2015) shows that blacks are trapped in high-poverty neighborhoods for a significant share of the life course to a far greater extent than whites. Toxic environmental factors, including elevated exposure to pollution from emissions from manufacturing plants, superfund hazardous waste sites, hazardous water pollutants, and highway traffic are among many elevated risks to which minority children are disproportionately exposed. Stressful neighborhood conditions, due to high-poverty rates, crime, violence, and weaker sources of social support, may lead to increased risk of high blood pressure.

The hypothesis that the differential burden of lifetime stress contributes to racial and socioeconomic disparities in health builds on a solid scientific foundation. Prolonged exposure to stress produces elevated risks of a condition known as allostatic load, which refers to the physiological costs of chronic overactivity or underactivity of systems within the body (e.g., the HPA axis or the autonomic nervous system) that fluctuate to meet demands of chronic exposure to environmental stressors (McEwen, 1998). Persistent exposures to disadvantaged neighborhood and family conditions may have a cumulative toll in the form of "weathering" (Geronimus, 1996). Recent findings in neuroscience indicate that early-life risk factors compound over the life course—often-cited examples of the adaptive cost of stress-induced wear and tear ("weathering") include pushing the endocrine system toward diabetes or the cardiovascular system toward coronary artery disease and hypertension (Halfon & Hochstein, 2002). How people are affected and adapt to stressful family circumstances and neighborhood environments may depend, in part, on their access to informal sources of social support. Blacks appear to face more stress than comparable whites (Geronimus, Bound, Waidmann, Colen, & Steffick, 2001), as evidenced in studies that document higher cortisol levels—a hormone that is a marker for stress—among blacks even after accounting for family income (DeSantis et al., 2007).

## Segregation

There is a tendency to wrongly interpret the residual difference in health between blacks and whites after controlling for socioeconomic status measures and health behaviors as arising from genetic causes or individual attributes. Most of the black–white difference in life expectancy stems from racial differences in mortality rates prior to age 65, and is largely rooted in inequality in social, economic, and environmental conditions experienced over the life course. Blacks' higher prevalence of cardiovascular disease-related risk factors accounts for more than half of the racial disparity (Barghaus, Cutler, Fryer, & Glaeser, 2008), with hypertension the leading culprit. Hypertension is a major risk factor for heart disease and stroke, the leading causes of death in the United States. The prevalence of hypertension and diabetes is two to three times higher among blacks relative to whites (Johnson, 2018).

It is also commonplace for analyses of health inequality in various disciplines to consider race, described as a risk factor, without explicitly

considering dimensions of structural racism as a component factor. Moreover, the intergenerational transmission of disadvantage may have biological consequences wherein social inequalities become embodied (Gravlee, 2009). It is important not to reduce biology to genetics (i.e., biological determinism) as it ignores the causal impacts of environmental and SES factors on biology.

A vast portion of the conventional research literature in this area can be summarized as a nested hierarchy of causal influences that gives much more attention to individual- and family-level factors, while minimizing or ignoring contextual factors that give rise to disparities over the life course above and beyond individual-level factors. There is a tendency to focus on individual-level determinants and health care access instead of how those important factors are influenced by and combine with social determinants of health wherein the role of place-based and structural factors are at work. These place-based factors give rise to the conditions of (un)healthy development.

The patterning of cardiovascular disease, diabetes, stroke, asthma, low birth weight, certain cancers, and other specific health conditions along the segregation fault lines of race and poverty demands rigorous research into place-based factors as potential explanations. The primacy of the geography of opportunity has been established in mounting evidence on segregation as a fundamental cause of racial inequalities in health (Alexander & Currie, 2017; Johnson, 2011; Williams & Collins, 2001).

The Center for Disease Control and Prevention (CDC) publishes estimates of life expectancy at the census-tract level and reports life expectancy at the 90th percentile is 83.1 years compared with 73.1 years at the 10th percentile. Among the starkest differences are in the most segregated cities like Chicago, where census tracts a few miles apart can differ in average life expectancies by up to 20 years. Stress associated with living in distressed neighborhoods leads to higher allostatic load and results in adverse health outcomes (Merkin et al., 2009). Minority and low-income individuals experience disproportionate amounts of chronic stress. Biosocial determinants of health interact with cumulative exposure to stressful life conditions to form major risk factors for premature morbidity and mortality (Krieger, 2012). Other correlational studies that hint at the health hazards and stress-inducing environments find blacks, but not whites, in concentrated poverty neighborhoods with high incarceration and crime rates have worse cardiometabolic health—that is, higher rates of hypertension, metabolic syndrome, and dyslipidemia (due to either higher victimization rates and/or greater concerns of unfair treatment from the police) (Topel et al., 2018).

Health behaviors and health care access do not exist in a vacuum but must be carefully contextualized by surrounding neighborhood conditions. Moreover, health-related behaviors that are associated with low SES, such as smoking, alcohol use, and nutritious diets, do not explain differences in health at the population level (Adler et al., 2017). There is too heavy a traditional focus on individual-level recidivism rates without attention to structural changes to the contextual environment that make effective interventions possible and sustainable.

Similarly, racial attitudes (prejudice) and beliefs (stereotypes) are often considered at the individual-level without attention to how group-level biases (implicit and explicit) are formed and expressed through systems and institutions of power that influence policies. When aspects of race are brought into view, an exorbitant focus is placed on interpersonal encounters of discrimination, while often neglecting analyses of the presence and consequences of structural racism. Accordingly, there is mounting evidence that the incidence of direct interpersonal encounters of perceived discrimination (based on self-reports) are significantly associated with worse health outcomes, which have been found to be particularly salient for minorities (Paradies et al., 2015). Yet, interpersonal encounters of discrimination may not represent how and why racism matters most for health. A key mechanism behind the veil of segregation is structural racism, which may influence health and well-being in various ways. For the study of inequality, ambiguity often blurs the definition of structural racism as a scientific construct. Here we refer to structural racism for purposes of the study of inequality as the embedded structures of socioeconomic opportunity and political power. Segregation is embedded in laws, policies, and practices, and its adverse health consequences are most distinctive for African Americans. Middle-class blacks are more segregated than low-income Hispanics and Asians, and the segregation of immigrant groups has never been as high as the current segregation of African Americans (Reardon, Fox, & Townsend, 2015).

It is critical that the study of racial health disparities not be pursued in an ahistorical way, as many of the risk factors have generational policy roots, most prominently segregation (e.g., redlining and modern day forms of exclusionary housing policies). First causes of segregation are efforts to maintain racial hierarchy, produced through the perpetuation of racist ideology that differentially allocates valued societal resources and opportunities and risks to groups defined as inferior. Structural racism can persist in institutions that govern public education, health, and housing policies even in the absence of individuals who are explicitly racially prejudiced (Williams et al., 2019).

Here are three prime examples illustrative of this broader point. First, fiscal federalism—or decentralization—of many of our safety net programs means that programs are administrated by states that are granted substantial latitude in how funds are spent with regard to programmatic features. State policies often act to widen racial inequality and exacerbate racial differences in child poverty rather than ameliorate them. As studies show, the higher the proportion of African Americans in a state, the less likely it is to allocate Temporary Assistance for Needy Families (TANF) and public assistance funds toward the provision of cash assistance and the support of basic expenses, and instead funds have been increasingly diverted to "pro-marriage" initiatives, abortion prevention programs, and abstinence-only sex-education programs (Parolin, 2019). Since 1996 federal welfare reform, the amount of money (in real dollars) spent on direct cash assistance has declined by two-thirds. As a stark example, Arkansas in 2017 spent only 4% of its total TANF budget on cash assistance and instead allocated two-thirds of the budget for the "reduction of out-of-wedlock pregnancies." Studies show that closing the racial differences in states' use of TANF funds would narrow the black–white child poverty gap by 15% (Parolin, 2019). Moreover, race explains a significant degree of the variance in states' spending for low-income families, while a state's wealth, the proportion of single mothers in the state, and which political party controls the legislature explained little of the variance (Parolin, 2019). These have consequences for child health and educational outcomes (Dahl & Lochner, 2012; Hoynes, Schanzenbach, & Almond, 2016).

Another case study providing suggestive evidence of the way in which racialized perceptions of the beneficiaries of safety net programs affects public support of them can be found in the pattern of state differences in the Affordable Care Act's (ACA) health care reform adoption. Specifically, eight of the eleven states with the largest black populations are among those that elected not to implement ACA's Medicaid expansions. A new study shows this policy choice continues to have grave consequences. Specifically, economists Miller, Altekruse, Johnson, and Wherry (2019) using linked large survey and administrative data, and a difference-in-differences research design to identify causal effects, find about 15,600 people died between 2014 and 2017 as a result of states deciding not to expand Medicaid eligibility through the ACA. They show the policy-induced effects are driven by disease-related deaths and the impacts grow over time. Supporting evidence by Adamson et al. (2019) shows states that adopted Medicaid expansions following ACA passage led to the elimination of racial disparities in the timely receipt of cancer treatments and related care.

The second example is represented by the public divestment from minority communities, which has resulted in a higher preponderance of concentrated poverty neighborhoods. Minority, low-income children are significantly more likely to grow up in neighborhoods near factories that emit high levels of pollution, and live in areas with greater exposure to lead and poor air quality (in utero and in childhood) (Persico, Figlio, & Roth, 2019). Physiological effects of early-life lead exposure can be irreversible (e.g., lead tends to stay in the brain once deposited there). This is compounded by poor housing conditions, housing instability (eviction, foreclosure, and related financial stress), income and food insecurity, nutritional deficits, and material deprivation. Children who reside in predominantly black neighborhoods have lower access to fresh food (known as neighborhoods in food deserts), less access to safe parks and recreational areas, but higher exposure to crime-ridden areas with greater proximity to unhealthy food options and liquor stores (Reardon et al., 2015), all of which may contribute to the child obesity epidemic.

Neighborhood exposures in childhood trigger increased asthma prevalence and other health insults as children develop. For example, black children have double the asthma rates as other children. New studies of historical redlining maps show they are still predictive of current racial health disparities, which highlight both the persistence of racial residential segregation and legacy of past segregation. In particular, Nardone, Thakur, and Balmes (2019) find that people who live in historically redlined neighborhoods today are more than twice as likely as others to go to the emergency room for asthma, a leading cause of child disability in the United States. Historically redlined neighborhoods exhibit much worse air quality as measured by higher amounts of diesel particulate matter in the air, and the evidence shows housing conditions continue to contribute to disparities in the morbidity of asthma.

A third example is the fact that greater racial fragmentation in metropolitan areas results in lower support of public education funding and unequal educational opportunity, as it leads to a greater propensity for inequity in property wealth and other resources across communities. Increased segregation leads to more inequality in spending across districts of the same metropolitan area, with harmful consequences for children in poor minority communities (Johnson, 2019). These patterns may help to explain why the long-term benefits of public human capital investments targeted to improve early life conditions varied for African-Americans who were exposed to different levels of segregation in childhood (Johnson, 2019).

## Implicit biases in health care

Racial segregation may also act to engrain and fester implicit biases among physicians in practice style. The significance of this potential factor is evidenced in the fact that across virtually every therapeutic intervention, ranging from high technology procedures to the most elementary forms of diagnostic and treatment interventions, minorities receive fewer procedures and poorer quality medical care than non-Hispanic whites (Institute of Medicine, Smedley, Stith, & Nelson, 2003; Williams et al., 2019). These differences persist even after differences in health insurance, socioeconomic status, stage and severity of disease, comorbidity, and the type of medical facility are taken into account. Moreover, they persist in contexts such as Medicare and the Veterans Administration Health System, where differences in economic status and insurance coverage are minimized (Institute of Medicine, 2003). But how could well-meaning and highly educated health professionals, working with diverse populations of patients, create a pattern of care that appears to be discriminatory? New evidence suggests implicit bias provides part of the answer. For example, in the case of pregnancy and birth outcomes, black women have the highest maternal mortality rate, and are three to four times more likely to die in child birth, some of which may be due to implicit bias of physicians that leads to differential health care provider sensitivity to pain and risk factors. New quasiexperimental studies on physician–patient race concordance provide evidence that supports hypotheses surrounding these links and sheds additional light on how and why racial diversity matters for health. Alsan, Garrick, and Graziani (2019) employed a randomized experimental design of race of doctors assigned to patients, and found that black men are much more likely to select preventive services, particularly invasive services, once meeting with a black doctor. Black patients were more likely to talk with a black doctor about their health problems and black doctors were more likely to write additional notes about their patients, markers of better patient–doctor communication and trust (and not necessarily reflective of discrimination). Based on their findings, the authors report black doctors could help reduce the black–white cardiovascular mortality gap by 19%.

A prime example of the point that there are generational and historical roots of some contemporary racial health disparities is found in the legacy of the infamous Tuskegee experiment on black distrust of the health care system and explain some of the racial differences in health care utilization patterns. To explore this empirically, Alsan and Wanamaker (2017) used data on medical trust, migration, and health utilization from the National Health Interview Survey and the General Social Survey, as well as morbidity and mortality data from the Centers for Disease Control and Prevention. In order to identify the legacy effects of the Tuskegee experiment, they used a difference-in-difference-in-differences approach, comparing older black men to other demographic groups, before and after the Tuskegee revelation, in varying proximity to the study's victims. They found life expectancy at age 45 for black men declined by 1.5 years in response to the revelation of the egregious medical exploitation, accounting for about 35% of the 1980 life expectancy gap between black and white men. That is a remarkably large health effect—on the order of entirely eliminating obesity among black men. This work illuminates a part of the root of racial disparities in the likelihood to seek preventive medical screenings.

Two dimensions of race to break apart are the biological and cultural—as Gravlee (2009) put it "the phenotype of skin pigmentation and the cultural significance of skin color as a criterion of social classification." Instead of racial–genetic determinism explanations that have been debunked scientifically, recent research has posited that "[the] embodiment of socioeconomic inequality passes through biological systems regulated by genes" (Gravlee, 2009). The intergenerational transmission of disadvantage may have biological consequences wherein social inequalities become embodied.

Suggestive evidence in empirical research testing the embodiment hypothesis of how stress gets under the skin is found in a number of studies. Lauderdale (2006) analyzed administrative data from birth records of all births in California in the 6 months before and after September 2001. They found that women with Arabic names experienced a 34% increase in the likelihood of having a low birth weight infant after 9/11—an increase not found for any other demographic group. Moreover, the effect was most pronounced among infants who were given more ethnically distinctive Arabic names, among whom the risk of low birth weight doubled after the attacks of September 2001, compared to 1 year earlier. These results may hint at a potential consequence of toxic exposure to racism. An alternative mechanism may be increased fears leading to reduced utilization of preventive health services, which may also apply to effects of immigration policies in the case of both documented and undocumented immigrants. For example, a recent study found that a large federal immigration raid was associated with an increase in the likelihood of low birthweight among infants born to Hispanic mothers in that community a year after the raid, which was not observed among non-Hispanic white mothers and communities not subject to the raid (Novak, Geronimus, & Martinez-Cardoso, 2017).

## Connections across policy domains

A different descriptive portrait on trends in health inequality emerge when focusing on younger cohorts versus older cohorts, with life course influences serving as keys to explain divergent patterns by race and SES. This work highlights the role of place and the accompanying effects of housing policies. For premature deaths, black–white racial gaps among men have narrowed substantially because of significant drops in homicide rates resultant from large declines in crime (Sharkey & Friedson, 2019), and improved medical therapies for HIV.

This narrowing of the black–white gap in life expectancy is a relatively recent phenomena of the past decade or so, for example, in 1995 the gap was still 6.9 years at roughly the same level as prevailed in 1955 (while the underlying sources were different). Surprisingly, a significant source of this decline may be related to an oft-asked question in a completely different field—criminal justice—that connects public health with violent crime trends. Specifically, why have violent crime and homicides rates unexpectedly dropped in half since the early 1990s? While there may be many factors that contributed to the declines, an often overlooked explanation is that this is in part a by-product of more effective lead abatement public health policies that have reduced the incidence of early-life lead exposure of minority children in high-poverty neighborhoods.

Lead is a hazardous neurotoxicant with a wide spectrum of potential adverse effects on cognition, health, and behavior, such as decreasing IQ, increasing learning disabilities, behavior problems, and aggressive behavior, as well as adversely affecting cardiovascular functioning and other physiological processes (Aizer & Currie, 2019; Aizer, Currie, Simon, & Vivier, 2018; Reyes, 2015). The growing fetus, infants, and children are most sensitive to low-level exposure, because ingested lead is more likely to be absorbed from the gastrointestinal tract and, conditional on absorption, is more likely to affect the developing nervous system than the mature brain. Geographic concentration of three of the main sources of lead in urban areas include lead in deteriorating paint found in old homes, residual lead in soil at former industrial sites which were often located in central cities, and residual lead in soil near high traffic areas.

The primary empirical challenges in investigating the impacts of early-life lead exposure are threefold: (1) information on blood lead levels are not universally tested; (2) one must distinguish the more general negative effects of growing up in concentrated poverty areas from the negative effects that emanate directly from childhood lead exposure; and (3) such an investigation of longer-term effects requires longitudinal data on children from birth to adolescence to adulthood. Recent research has overcome these challenges by first combining several sources of administrative data on county–year–birth cohort level blood lead measures obtained from the CDC Environmental Public Health Tracking Network system and information on county–year-level public health investments in lead abatement from a state's Department of Public Health lead abatement monitoring system. The research design used in recent work has analyzed the impacts of policy-induced changes in early-life lead exposure risks across successive cohorts from the same childhood counties (Aizer & Currie, 2019; Reyes, 2007).

Using administrative data linking preschool blood lead levels, birth, school, and detention records for 125,000 children born 1990–2004 in Rhode Island, Aizer and Currie (2019) estimate the impact of lead on deviant behaviors and find a one-unit decrease in lead exposure reduced the probability of suspension from school by 6% and detention by 57%. Reyes (2007) used the sharp state-specific reductions in lead exposure during the late 1970s resulting from the removal of lead from gasoline under the Clean Air Act, and finds that the reduction in childhood lead exposure in the late 1970s and early 1980s is responsible for significant declines in violent crime in the 1990s.

Sharkey and Friedson (2019) conducted a simulation suggesting that life expectancy for black men would have been 0.8 years lower if homicide rates had persisted at their peak levels of the early 1990s. They estimated that 17% of the narrowing of the black–white life expectancy gap for men between 1991 and 2014 can be explained by the substantial reductions in homicide rates witnessed over this period.

## HIV/AIDS

Another case that exemplifies surprisingly strong links between criminal justice policy and health lies in the connection between incarceration dynamics and HIV/AIDS infection rates. Individual-level risk factors alone have proven inadequate to explain the substantial geographic heterogeneity in the diffusion patterns of the AIDS epidemic in the United States both between and within racial/ethnic groups. Johnson and Raphael (2009) investigated the connection between incarceration dynamics and AIDS infection rates, with particular emphasis on the black–white AIDS rate disparity. They identified incarceration dynamics as a primary mechanism through which the AIDS epidemic transformed from one impacting almost exclusively young gay men to a disease increasingly transmitted through heterosexual sex that disproportionately afflicts minority women.

Changes in male incarceration rates alter HIV transmission risks within defined sexual relationship markets through a number of channels. In particular, male incarceration lowers the sex ratio (male to female), disrupts the continuity of heterosexual relationships, and increases the exposure of incarcerated men to high-risk sex amid a population with a high prevalence of HIV. All of these factors elevate an individual's or group's AIDS infection risk and disproportionately affect the AIDS infection rates of black women and men.

Using case-level US data spanning 1982–96, we modeled the dynamic relationship between AIDS infection rates and the proportion of men in the age-, state-, and race-matched cohort that are incarcerated. We found strong effects of male incarceration rates on male and female AIDS rates. The dynamic structure of this relationship—that is, the lagged effects of the proportion of males incarcerated—parallels the distribution of the incubation time between HIV infection and the onset of full-blown AIDS, with small effects for early lags and relatively large effects for later lags. Given the sizable racial differentials in incarceration rates at the beginning of the AIDS epidemic and the increases in these differentials thereafter, the results revealed that the lion's share of the racial differentials in AIDS infections rates for both men and women were attributable to racial differences in incarceration trends for that period.

At the peak of the HIV epidemic in the mid-1990s, before the widespread availability of antiretroviral drug treatments, AIDS was among the leading causes of death among young black men and women, with age-adjusted mortality rates of almost 60 per 100,000, nearly six times the prevailing rate today. Currently, there are more than one million people in the United States living with HIV, 42% of whom are black (CDC, 2013). While blacks still comprise the majority of AIDS-related deaths, the large reduction has contributed to the significant narrowing of the black–white life expectancy gap.

## Mental health and substance abuse

Unhealthy behaviors, including addiction, substance abuse, and risky sex, may be better understood as coping mechanisms, adapted responses to a lack of opportunity, adaption to one's socioeconomic environment, hopelessness, and loss of connection to purpose, exacerbated by isolation and disconnections from social networks. This has been recently referred to as "deaths of despair" (Case & Deaton, 2017).

Historically, drug epidemics have disproportionately had devastating impacts on minorities and those in concentrated poverty neighborhoods. But mortality rates for non-Hispanic whites caused by all drugs have more than quadrupled since 2000, and are now 32% higher than for blacks. Among those who died due to opioid overdoses in 2017, 78% were white, and the age-adjusted mortality rate due to opioids for whites was roughly 20 per 100,000. Among nonelderly adults, the opioid epidemic, rapid increases of overdose-related deaths and suicides have all occurred at much higher rates for whites and have occurred disproportionately in more affluent areas.

Studies have documented substantial geographic variation in rates of prescribing opioids, and paradoxically provided evidence that the pattern may be related to racial discrimination in the health care system on the part of physicians. For example, one study using administrative hospital discharge data of pain-related visits to emergency rooms during the years that led up to the epidemic (between 1993 and 2005) found that whites were significantly more likely to obtain an opioid prescription, even among otherwise similar patients with the same reported severity of pain and other characteristics (Pletcher, Kertesz, Kohn, & Gonzales, 2008). Furthermore, physicians were found to be much more likely to stop prescribing opioids for blacks after suspecting or identifying illegal drug use. Thus the case of opioids has an unforeseen twist from the traditional narrative when racial bias is at work—here, physician racial bias may have saved black lives.

The role of access to health care must extend beyond early detection and treatment of physical ailments, to include mental health conditions like depression, which may be stigmatized and therefore be more likely to remain undiagnosed and untreated. Many mental health problems manifest in young adulthood but start in early childhood. Moreover, maternal mental health has important impacts on children. Bridge et al. (2018) report that since 2000 the suicide risk for black boys between the ages of 5 and 11 was two to three times higher than that of white boys, and this troubling trend continues through adolescence (Kann et al., 2018). Exposure to trauma, internalized dismal expectations for the future (including low life expectancy resultant from high homicide rates), and "nothing to lose" attitudes developed in distressed neighborhood environments of hopelessness, and a greater propensity of underdiagnoses of depression for black boys, have been documented as factors (Harris, Duncan, & Boisjoly, 2002).

Recent research emphasizes "soft skills matter for producing hard outcomes," such as educational attainment, health, and labor market outcomes (Almond, Currie, & Duque, 2018). Emerging evidence shows socioemotional learning in childhood (i.e., social and emotional skills, habits, and mindsets) has large impacts on academic and life success, including

health-related behaviors (Heckman & Kautz, 2012; Jackson, 2018). These have implications for healthy development as they may reduce risk factors for suicide and the likelihood of early onset of substance use (alcohol and smoking) and obesity. Mental health like soft skills are more difficult to measure empirically, particularly with large quantitative survey data sets relative to physical health and cognitive test scores, which often causes them to go underappreciated, overlooked, and undiagnosed as part of the problems facing at-risk youth when interventions may be most effective.

We need a better understanding of the mechanisms undergirding how and when segregation exerts its greatest influence on health outcomes. There are multiple mechanisms that can underlie a given empirical pattern. Without identifying precise mechanisms, designing effective interventions to narrow health disparities will be more limited, and will not enable one to determine which investments and interventions would be most cost-effective in addressing unequal opportunity in children's lives and hold the most promise to improve their future life trajectories. Effects may operate through mechanisms that involve socioeconomic influences as well as biological pathways. This is also critical to target interventions in the most effective ways.

There are also important distinctions between how segregation may influence health for black and Hispanic Americans. For example, Lee and Ferraro (2007) find that for Mexican Americans, ethnic enclaves promote health by facilitating the flow of informal health resources and social support. The Hispanic health paradox refers to the pattern in the United States wherein Hispanics appear to have comparable health status as non-Hispanic whites, despite significantly lower socioeconomic status. But upon closer inspection using longitudinal data on country of birth and lived experiences, studies have documented worse health for second-generation Hispanics, and found that middle-aged US-born Mexican Americans and Mexican immigrants who had resided at least 20 years in the United States had similar health as African Americans (Kaestner, Pearson, Keene, & Geronimus, 2009).

## Methodological issues and measurement challenges

Causal evidence on the effects of segregation requires research designs that can distinguish between neighborhood effects and the effects of family characteristics common to people living in disadvantaged neighborhoods. One must disentangle the separate influences of childhood family background, and school quality and neighborhood factors on subsequent health trajectories. There are limitations of using a community-level geographical approach as analyses of geographic areas are particularly vulnerable to bias from selective migration. Residential mobility patterns make it difficult to conduct valid comparisons of health at the county-level (or neighborhood-level) over time. If the most advantaged people are more likely to leave poverty-stricken areas, then the average health in those areas will decline over time even if there was no actual change in any individual's health. In reverse, gentrification may lead a neighborhood to attract more affluent families back to urban communities, but failing to account for these demographic changes can lead to misleading conclusions about the role of the neighborhood conditions themselves.

The most powerful way to address selection is through a randomized trial. But an experimental design where neighborhoods are randomly assigned is rare. A significant exception is the evaluation of the Move to Opportunity (MTO) program, where an experimental design was used to estimate the effects of offering housing assistance that allows individuals to move out of low-income, poor neighborhoods. Evidence from MTO demonstrates that moves to better neighborhoods had beneficial effects on the health of children and adults (Katz, Kling, & Liebman, 2001; Leventhal & Brooks-Gunn, 2001). This evidence is consistent with the claim that neighborhood factors do in fact influence health status, at least in the short-run among poor families. While all of the "treated" children in MTO moved to lower poverty neighborhoods, the new neighborhoods were just as racially segregated as the old ones, and the duration of exposure in these new neighborhoods was limited.

Studies that extend the frontier of knowledge in this area require multilevel longitudinal data with attention to valid measurement and credible research designs to facilitate causal inferences. The increasing use of large-scale linked survey and administrative panel data to track children over time (e.g., from natality files of birth records, mortality and cause of death records; death statistics come from the Vital Statistics mortality data collected for each county, hospital discharge data, combined with Census data) have enabled ways to better address issues of measurement error, selective attrition from surveys, and have contributed greater statistical power to detect effects of interventions due to large sample sizes. This recent data revolution has allowed scholars to conduct policy evaluations by linking current health outcomes in adulthood with past childhood exposures to policies in ways not previously possible. The measurement challenges stem from the multilevel and multidimensional nature of poverty,

inequality, and public investments as well as the child developmental and well-being outcomes that result from them. Single-level and unidimensional metrics are insufficient for tracking the combined impacts of socioeconomic conditions on health over time.

While the adverse health consequences of toxic stress have a strong theoretic and scientific foundation, it remains difficult to directly demonstrate empirically because the most common biometric measures of stress—for example, cortisol—are not available in most health datasets. Lacking direct measures of stress, the most common approach uses proxies for an entire range of stressful life experiences and more subtle exposures that may have been suffered (in some work this has included racially charged, discriminatory encounters), and tests of their association with hypothesized affected health outcomes.

## Human capital policy is health policy (and vice versa)

Learning opportunities and nurturing interpersonal relationships affect how the brain is built in its early developmental stages, and how it evolves over the life course. Studies of neuroplasticity demonstrate that a young developing child's brain exhibits a substantially higher degree of plasticity than adulthood. This also holds true for the immune system with implications for healthy development, as shown in work on the vulnerability and consequences of early-life lead exposure. But a degree of plasticity continues as we age and does not stop at the end of our early childhood years. This malleability drawn from insights in developmental neuroscience have been a catalyst for research on showing there are common forces at work in cognitive and noncognitive development and health formation (Shonkoff & Phillips, 2000).

Research evidence suggests a close connection between cognitive and noncognitive developmental outcomes, physical and mental health status, and both health and educational attainments. These developmental processes are best understood when considered not in isolation but in concert with recognition of dynamic spillover effects and feedback loops that may operate over the life course.

The interlocking problems of poverty require interlocking solutions. That is, every problem (e.g., early-life lead exposure, underresourced schools) intensifies the impact of the others, and all are so tightly interwoven that a reversal of any singular problem can produce a chain reaction with results far distant from the original cause. New lines of research on inequality are motivated by the idea of intersectionality—that different forms of inequality and segregation overlap and compound one another with effects that are both interactive and reinforcing over the life course. I investigated how much the combined effects of early- and later-childhood education and health investments, targeted to poor and minority communities, can narrow health disparities by race and class. Do the combined effects exceed the sum of effects of individual, separate policies?

To this end, I summarize below research findings on three related childhood public policy interventions that have significantly reduced later-life health disparities.

1. The interactive effects of pre-K and K-12 educational investments, using evidence from Head Start and public school spending (via school finance reforms).
2. The long-run impacts of school and hospital desegregation (1960s, 1970s, 1980s).
3. The beneficial effects of childhood public health investments on subsequent health, education, and economic outcomes in adulthood.

Earlier work attempting to answer these research questions has been hampered by the paucity of longitudinal data with this multiplicity of factors, as it requires a systems approach to analyzing inequality across sectors (education, health, housing), and compiling intersectional data bases to empirically track distal outcomes from the original intervention or exposure. An aim of my work on this topic was to fill the research gap by linking data on early childhood through K-12 school resource inputs with data on adult health and SES attainment outcomes. The aim was to improve our understanding of the long-run economic and health returns to access to high- versus low-quality K-12 school systems. To that end, I analyzed the life trajectories of children who were followed from birth to adulthood, using the geocoded PSID (PSID, 1968–2015) linked with multiple other data sources. The results show that policies that aim to increase educational attainment are an important means for improving population health.

## Long-term impacts of preschool investments

Head Start was established in 1964 as part of Lyndon B. Johnson's "War on Poverty," and is a national, federally-funded, early-childhood program with the aim of improving the human capital of poor children. The Head Start curriculum aims to enhance literacy, numeracy, reasoning, problem-solving, and decision-making skills. The program emphasizes healthy nutrition for the family through parent education and a school meal program. Participating children receive development screenings, and programs connect families with medical, dental, and mental health services. In their book, *The*

*Hidden History of Head Start*, Zigler and Styfco argued that "Head Start has never received sufficient credit for improvements in children's physical health." In the first 2 years of Head Start's operation, 98,000 children had eye defects diagnosed and treated, 900,000 dental problems were discovered with an average of five cavities per child, and immunizations were given to 740,000 children who had not previously been vaccinated against polio and to 1 million children who had not previously been vaccinated against measles (Office of Economic Opportunity, 1967).

The availability of preschool varied across communities and time, and we exploit this variation to identify their long-term effects. Johnson and Jackson (2019) employed compelling identification strategies to tease out causal effects. We used children's differential exposure to Head Start spending (at age four) and court-ordered school finance reforms (SFRs) (between the ages 5 through 17), to examine whether the marginal effect of Head Start spending on children's adult outcomes are larger among individuals who were subsequently exposed to SFR-induced K12 spending increases. Results showed positive synergies and complementarities of human capital (public health and education) investments at different childhood stages of development.

The long-run benefits of Head Start were larger when followed by access to better-funded schools, and increases in K12 spending were more efficacious when preceded by Head Start exposure. The results highlight the importance of modeling early and later educational investments jointly. The findings reveal that early childhood investments that are sustained throughout child developmental stages have the power to break the cycle of poverty.

Lumeng et al. (2015) and Lee, Zhai, Han, Brooks-Gunn, and Waldfogel (2013) found that preschool-aged children with an unhealthy weight status who participated in Head Start had significantly lower likelihoods of being obese, overweight, or underweight by kindergarten entry age than comparison children. Related evidence on the long-term effects of targeted high-quality preschool programs for low-income children from the Abecedarian project, finds that by their mid-30s, individuals in the treatment group had lower levels of multiple risk factors for cardiovascular disease as compared with those in the control groups who did not have access to the pre-K program.

## Long-run impacts of hospital and school desegregation

Among older middle-age Americans (ages 50–64), the narrowing of the black–white life expectancy gap is due in part to faster declines in mortality from heart disease for blacks than for whites. This may be connected to the reverberating lifelong health consequences of early black cohorts from the South who first gained access to integrated hospitals in utero, at birth, and in early childhood during the 1960s that led to significant improvements in infant health (Almond, Chay, & Greenstone, 2007).

Hospital desegregation was a monumental policy shift that, alongside the rollout of school desegregation, impacted the overall long-term well-being of minority children of the post-Brown era. The desegregation of hospitals in the South began in 1964 when federally-mandated policies began to be enforced. In particular, developments in all three branches of government—judicial, executive, legislative—were influential. First, Hill-Burton Act's "separate but equal" clause was ruled unconstitutional in 1963. Second, Title VI of the Civil Rights Act of 1964 put teeth in enforcement. Third, with the introduction of Medicare in 1965, a hospital had to be racially desegregated in order to be eligible to receive Medicare funding. The staggered timing of hospital desegregation in the South led to differences in the timing of improved access to hospital care for minorities, and resulted in timing differences in the implementation of Medicare in parts of the South that had not desegregated their hospitals prior to 1965.

Using the American Hospital Association's Annual Survey of Hospitals along with the Centers for Medicare Provider of Service data files to identify the precise date in which a Medicare-certified hospital was established in each county of the United States (an accurate marker for hospital desegregation compliance), I find that one quarter of counties in the South—and 75% of counties in the Mississippi Delta—lacked a Medicare-certified hospital by the end of 1966. Almond et al. (2007) used this variation in the timing of hospital desegregation in Mississippi to document substantial declines in blacks' postneonatal infant mortality from diarrhea and pneumonia in counties that had desegregated by February 1969, relative to counties whose hospitals remained segregated through the late 1960s. These are early-life health conditions that require immediate access to adequate hospital care to prevent mortality. They show a 40% reduction in black infant mortality between 1964 and 1972. In the early 1960s, black infant mortality rates were significantly higher in the 21 states, plus the District of Columbia, with de jure segregation than in non-Jim Crow, northern states. By the late 1960s, these differences in black infant mortality rates in the South versus nonSouth had disappeared (Krieger, Chen, Coull, Waterman, & Beckfield, 2013). Furthermore, Chay, Guryan, and Mazumder (2009)

provide evidence that racial convergence in early-life health and hospital access during ages zero to five subsequently led to a significant narrowing of the black–white test score gap for cohorts born during the mid-to-late 1960s.

My prior research findings demonstrate that both school desegregation and hospital desegregation independently contributed to the improvement in adult attainment of black cohorts that transitioned from the era of segregated to desegregated and integrated hospital and school contexts (Johnson, 2019). The findings show healthier children are better learners. We identify impacts of both hospital desegregation and school desegregation that led to significant narrowing of health disparities in adulthood for blacks, which again underscored the importance of considering the interrelationship between early-childhood investments in health and public school spending. When these two types of investments occur together, the combined effect can be substantial, and larger than the sum of the two investments in isolation.

## Long-run effects of child health insurance: evidence from medicaid expansions

Medicaid, first established in 1965, was part of President Lyndon Johnson's "War on Poverty" to create a public health care system to improve the health of the poor. Medicaid's founding architects aimed to play a foundational role in restructuring American health care inequalities by poverty and race. Federal spending on children has increased substantially over the past 25 years—particularly, increased access to health care via expansions of health insurance coverage for poor pregnant women and children, and expanded access to public state- and federal-funded pre-K programs, and public health lead abatement programs, which were each phased in for children across states and local communities at different rates. Medicaid represents the largest increase in the federal spending program for children. By 2001, all poor children through age 17 had access to public health insurance coverage; the fraction of all children covered by public health insurance coverage through age 17 went up from just 15% in 1986 to over 40% by the early 2000s. Currently, Medicaid and the Children's Health Insurance Program (CHIP) provide low-cost health coverage to nearly 44 million children, covering one-half of all low-income children (Centers for Medicare and Medicaid Services).

The significant variation in access to child health insurance coverage across birth cohorts and states over time has been used in research designs as a quasiexperimental approach to isolate its impacts on child health and educational outcomes (Cohodes, Kleiner, Lovenheim, & Grossman, 2016; Goodman-Bacon, 2018; Miller & Wherry, 2019). Results from several recent studies find notable improvements in an array of outcomes caused by access to childhood health insurance, including reductions in mortality and the likelihood of hospitalizations for chronic illness, particularly for black children.

Studies using quasiexperimental methods find that legislative expansions of Medicaid eligibility since 1980 led to large reductions in mortality for infants, children, and adults (Currie & Gruber, 1996; Sommers, Baicker, & Epstein, 2012; Wherry & Meyer, 2015). As Card and Shore-Sheppard (2004) show, the corresponding increases in any insurance coverage are relatively small during this later period, however, leaving considerable uncertainty about the mechanism for these effects. The Oregon Health Insurance Experiment, while providing the strongest research design to tease out health insurance coverage effects, is limited to only short-run health outcomes (Finkelstein et al., 2012). Recent studies find these Medicaid expansions (since 1980) led to improvements in poor children's academic outcomes (Cohodes et al., 2016; Levine & Schanzenbach, 2009). Relating to health, Goodman-Bacon (2018), found that Medicaid's introduction substantially reduced black infant mortality and reduced childhood mortality among black children by 20% in the 1960s and 1970s.

To examine longer-term impacts, I extended Goodman-Bacon's work to show the long-run returns to childhood Medicaid spending. For child cohorts after Medicaid implementation, childhood health care utilization increased—and more rapidly so among children from high-Medicaid-eligibility states. These increases in childhood public health insurance access led to notable reductions in the likelihood of low birth weight, increased educational attainment and the likelihood of graduating from high school, increased adult earnings, reduced the annual incidence of poverty in adulthood, and significantly reduced adult mortality and the annual incidence of health problems (Johnson, 2018).

The health returns to educational investments have received less attention than the traditional focus on short-run test scores and more recently, labor market returns. This is an important omission, given that the return to education in terms of health is about half of the return to education on earnings (Cutler & Lleras-Muney, 2008; Johnson, 2012)—that is, we calculate that the health benefits from education increase the total returns to education by as much as 50%.

Education is the single most significant correlate of health status, the relationship has been documented as far back as data are available, across countries, and

intergenerationally. Richer, better-educated people live longer: those in the top 5% of income have 25% longer life expectancy than those in the bottom 5% in the United States (Deaton, 2003). The age-adjusted mortality rate of high school graduates aged 25 to 64 is more than twice as large as the mortality rate of those with a college education (Xu, Kochanek, Murphy, & Tejada-Vera, 2010). This is corroborated by studies such as Lleras-Muney (2005) and Buckles, Hagemann, Malamud, Morrill, and Wozniak (2013) who both find large and significant effects of increased education on declines in mortality in the United States. In particular, causal evidence indicates that early adult mortality rates are about 2.2 times greater for noncollege graduates than for college graduates. Results from Buckles et al. (2013) imply that a 10 percentage-point increase in college completion rates would reduce early cancer deaths by 17% and early heart disease deaths by 10%—these are leading causes of death in the United States. An additional year of education lowers the probability of dying in the next 10 years by 3.6 percentage points (Lleras-Muney, 2005). Taken together, the returns to education, measured only in terms of earnings increases, substantially underestimate the total societal returns to education.

There has been a recent wave of reforms strategically designed to connect the implementation of health policy and education policy. This is in part motivated by research evidence showing healthier children are better learners (Johnson, 2019), and early detection and treatment of health conditions is therefore key. An example is represented in the movement over the past decade to establish school-based health centers, which aim to expand primary care services to low-income students to address health care access and disparities in health and education. Lovenheim, Reback, and Wedenoja (2016) find that school-based health centers led to significant reductions in the likelihood of teen pregnancy, reducing the birth rate for girls age 15 and younger by 23% and by 8% among girls ages 16–19.

In related work on the health consequences of education policies, I investigate the impact of school physical education (PE) on students' cardiorespiratory fitness, in a joint paper with UC-Berkeley colleagues (Thompson, Johnson, Madsen, & Fuller, Forthcoming). California's education law mandates that elementary students receive 200 minutes of PE every ten days—the equivalent of 20 minutes per day. While this law signals the importance of PE, elementary students are the least likely to receive the mandated PE minutes. Over half of California elementary school districts do not comply with the law. We find students in noncompliant districts are less likely to meet physical fitness standards, and noncompliant districts have a higher proportion of minority and low-income students than compliant districts. We examined whether unequal provision of PE contributes to racial/ethnic and income-related health disparities. Results showed African American children had the lowest levels of cardiorespiratory fitness, on average, at baseline. We found increases in the proportion that are in the Healthy Fitness Zone of cardiorespiratory fitness following the law suits. These results hold true on average for all fifth graders, boys and girls, with larger effects for girls. The largest impacts are found for African American fifth-grade children, who experienced a 3 percentage-point increase in the proportion that are in the Healthy Fitness Zone of cardiorespiratory fitness following the law suits. Our evidence indicates unequal provision of PE contributes to racial disparities in cardiorespiratory fitness for elementary school students, which may portend childhood obesity risks in the future.

## Role of the socioeconomic mobility process

In closing this section of the literature review, we return to the puzzle we set out at the beginning of the paper: why are there large racial/ethnic disparities in health at middle age and older ages, even among those with the same levels of adult socioeconomic status? As we foreshadowed, the answer lies in what happens in childhood and the socioeconomic mobility process that occurs in young adulthood when minorities achieve those levels of SES. The results in Johnson (2018) reveal that racial differences in early life neighborhood conditions and family background characteristics play an important role in explaining racial disparities in hypertension through at least age 50, while contemporaneous socioeconomic factors account for relatively little of the racial disparities in this health condition in adulthood.

Upwardly mobile minorities who beat the odds and escape poverty achieve better economic outcomes, but may experience greater exposure to stress including discrimination and structural racism that requires sustained coping (Gaydosh, Schorpp, Chen, Miller, & Harris, 2018). Patterns of health dynamics and the evolution of health disparities over the life course, particularly salient for minorities and those from lower-income backgrounds, appear broadly consistent with "John Henryism." Particularly among those from lower-income backgrounds, John Henryism suggests that individual characteristics such as perseverance, tenacity, and self-control enable higher socioeconomic attainments but often exact a physiological toll because they result in sustained activation of the stress-response system. This stress-related deterioration and resultant high allostatic load leads to biological wear and tear, accelerated aging, and accumulated risk over the life course (James, Keenan, Strogatz, Browning, & Garrett, 1992).

Metabolic syndrome is a cluster of health conditions that occur together—including hypertension, high blood sugar, abnormal cholesterol, and excess body fat—that increase the risks of heart disease, stroke, and diabetes. Research evidence shows that college degree attainment is associated with lower depression, but among minorities from lower-income backgrounds it is also associated with higher metabolic syndrome (Gaydosh et al., 2018). In stark contrast, among those from socioeconomically advantaged childhood backgrounds, college completion is associated with significantly better mental and physical health.

Levine, Markus, Austin, Chen, and Miller (2019) provide evidence that school climate and childhood experiences among minority students are associated with levels of interleukin, an inflammatory biomarker that contributes to cardiometabolic disease. Furthermore, experiences and perceptions of being treated unfairly are associated with elevated blood pressure and greater inflammatory biomarker levels, indicating higher risk of accelerated aging. Even after accounting for parental socioeconomic status, black adolescents exhibit greater insulin resistance (relative to whites), which increases the risk of diabetes, and they have higher carotid artery intima-media thickness, a marker of preclinical atherosclerosis that portends elevated risks for cardiovascular disease. In contrast, students who attend integrated schools and experienced a greater sense of belonging have lower concentrations of inflammatory biomarkers, over and beyond interpersonal experiences of discrimination. They show that when a school emphasizes the value of diversity and commits to a multicultural curriculum, minority students tend to have better cardiometabolic health—across multiple dimensions including biomarkers of inflammation, insulin resistance and β-cell activity, and metabolic syndrome. This significant association persists after accounting for parental socioeconomic status, class size, school resources, and other observable demographic characteristics. Furthermore, this pattern was not found among whites (suggestive of a race-specific explanation). Results were consistent across multiple biomarkers that have been linked prospectively to cardiovascular disease. While this is certainly a provocative finding, one caveat is that it is a correlational study, not design-based study for causal inference, but it is still noteworthy in this budding and rapidly growing field.

## Conclusion

Four ideas that most people believe, but have been debunked by rigorous longitudinal studies using nationally representative data that chronicle the lived experiences from birth to adulthood: (1) health care is the most significant determinant of health status and health disparities (not nonmedical, socioeconomic determinants of health); (2) segregation is inevitable and not the direct consequence of policy choices; (3) upward mobility outcomes are mostly a function of work ethic; and (4) racial and SES-based health disparities are synonymous.

What these popular misconceptions have in common is a narrow focus on individual-level determinants and health care domain explanations for population health outcomes. These ideas have been disproven by highlighting the effects of structural factors—in particular, the profound effects of neighborhood conditions and school quality on children's life chances, including their health trajectories over the life course. Remaining wedded to these wrong ideas for decades, however, has had extremely detrimental health consequences for low-income and minority children growing up in concentrated poverty neighborhoods. But the conventional view and old consensus has been upended by the big data and causal inference revolution that has brought new compelling evidence to these old debates.

Rugged individualism as an accepted doctrine believed to be the primary determinants of achieving the American Dream is at odds with the empirical realities showing the substantial impacts segregation has in both tilting the intergenerational playing field creating uphill struggle, and contributing to the fact that 42% of poor children remain poor as adults (Johnson, 2018). The strong persistence of health inequality transmitted across generations plays a role in this immobility.

Even among black and white families with similar incomes, white families are much more likely to live in good neighborhoods with high-quality schools, parks, and playgrounds. Reardon et al. (2015) show the typical middle-class black family lives in a lower-income neighborhood than the typical low-income white family. This work implicates racial differences in parental wealth as a chief culprit, and provides further evidence why race and SES are not interchangeable and should not be viewed as such (i.e., they are related, but distinct).

With increasing scientific consensus of the importance of early-life conditions on later-life health and the large body of evidence about the realities of how segregation perpetuates unequal opportunities, identifying effective interventions is critical. The question policy makers most want to know is what policies and programs should be invested in that would yield the greatest return on public investments in childhood. This paper has aimed to provide some answers to those questions. Moreover, these are wise public investments in our future that more than pay for

themselves in the long run. Indeed, the societal returns to pre-K investments far exceed the returns received by the individuals who participate in them. As our work highlights, the societal benefits include monetary savings from the lower educational remediation costs down the road, from the reduced likelihood of public assistance, from the averted costs of crime, and from the reductions in health care costs, as well as the boon received from increased tax revenues from working-age adults who are more productive because of the individual gains they made as youths. When we forgo these critical early-life public investments in disadvantaged children, we pay for it dearly down the road in the form of reduced national and state tax revenues, greater strains on the state and federal budgets, increased crime, poorer health, reduced political participation, and reduced intergenerational mobility (Hendren & Sprung-Keyser, 2019; Johnson, 2019).

Three types of siloing have hampered our progress toward building a more cohesive, coherent healthy equity agenda. First, traditional siloing of research, policy, and practice; second, siloing of research by disciplinary field at the expense of insightful, multidisciplinary and transdisciplinary approaches; and third, siloing of programs according to what aspect of child development is targeted (health vs education, pre-K vs K-12). Moreover, too much of the research on inequality tends to be ahistorical, yet many of the risk factors have generational policy roots, most prominently segregation (e.g., redlining and modern day forms of exclusionary housing policies). Siloed policy design features yield suboptimal impacts and undermine broader, collective, enduring impacts on racial health disparities. There is a tendency toward incremental, fragmented policy solutions, not comprehensive reforms with the transformative power to break the cycle of poverty. There is a difference between a collection of good policies versus a collaboration of policies that are strategically implemented.

The broader takeaway message of this chapter is that health disparities are not immutable—they are not simply a product of genes, but are the consequences of our history and our policies.

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