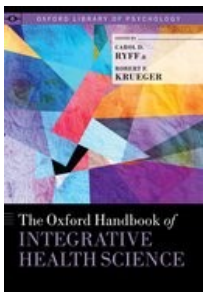

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CHAPTER

33 Disparities in Health Between Black and White Americans: Current Knowledge and Directions for Future Research

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Abstract

Conceptual frameworks for racial/ethnic health disparities are abundant, but many have received insufficient empirical attention. As a result, there are substantial gaps in scientific knowledge and a range of untested hypotheses. Particularly lacking is specificity in behavioral and biological mechanisms for such disparities and their underlying social determinants. Alongside lack of political will and public investment, insufficient clarity in mechanisms has stymied efforts to address racial health disparities. Capitalizing on emergent findings from the Midlife in the United States (MIDUS) study and other longitudinal studies of aging, this chapter evaluates research on health disparities between black and white US adults. Attention is given to candidate behavioral and biological mechanisms as precursors to group differences in morbidity and mortality and to environmental and sociocultural factors that may underlie these mechanisms. Future research topics are discussed, emphasizing those that offer promise with respect to illuminating practical solutions to racial/ethnic health disparities.

Keywords: racial/ethnic health disparities, MIDUS, black adults, white adults, morbidity, mortality, environmental factors, sociocultural factors, discrimination, early life adversity

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Introduction

Healthy People 2020, a key federal initiative that serves to set the priorities of US public health departments and organizations, describes the reduction of health disparities as one of its four overarching goals (https://www.healthypeople.gov). One striking domain of disparities, documented across a wide range of health outcomes over recent decades, is the differences in health and longevity that exist between black/African American (AA) and white/European American (EA) adults in the United States (Adler & Rehkopf, 2008; Cummings & Jackson, 2008; Williams & Mohammed, 2009). Progress in reducing the black–white health gap demands a comprehensive understanding of biological, behavioral, psychosocial, and contextual influences that are involved, as well as an integrative perspective on the various domains of influence.

In this chapter, we evaluate the status of research on health disparities between black and white American adults and suggest directions for future work. We start by summarizing existing research on black–white disparities in health indicators and health outcomes across the life span, with a focus on measures that are relevant to the Midlife in the United States (MIDUS) study. Next, we consider adult context and experience mediators with a focus on three domains: experiences of discrimination, neighborhood environments, and socioeconomic status (SES). We then bring in childhood context and experiences, namely early life adversity (ELA), before turning our attention to health behaviors and then ending with a distilled set of
p. 458 recommendations for future research. ↳

Core Questions and Guiding Conceptual Framework

Figure 33.1 provides a guiding conceptual heuristic that serves to orient and contextualize the various sections of this chapter. The left side of the figure illustrates that current institutionalized racism (such as within the criminal justice system), the historical legacy of more overt systemic racism (such as slavery, Jim Crow laws, and exclusionary housing and lending policies), as well as direct interpersonal experiences of discrimination (such as maltreatment and prejudicial attitudes) all combine to shape large race differences in the context and experiences during childhood and across the life span. In addition, ubiquitous messaging around problems and deficits within the black community and antiblack bias can manifest as internalized racism among AA individuals. As a result of these various layers of past and present racism and discrimination, race in America is consistently associated with income and wealth, neighborhood of residence, and exposure to adversities (e.g., community violence). As depicted in the center of the model, racism and discrimination affect childhood context and experiences, as well as adult contexts and experiences, including education, work environment, and neighborhood context. Childhood and adult contexts and experiences then combine to influence health behaviors (sleep and physical activity), health indicators (markers of physiologic dysregulation), and ultimately health outcomes (morbidity and mortality). Moreover, the behavioral and biological mechanisms for race differences in health outcomes are depicted with boxes on the right side of the figure and will also be an important consideration. We note that the conceptual model described herein is not exhaustive, and that further research and theoretical work are needed to elucidate the role of affective, psychological, and cognitive processes in the associations discussed in this chapter (for one relevant review, see Dressler, Oths, & Gravlee, 2005).

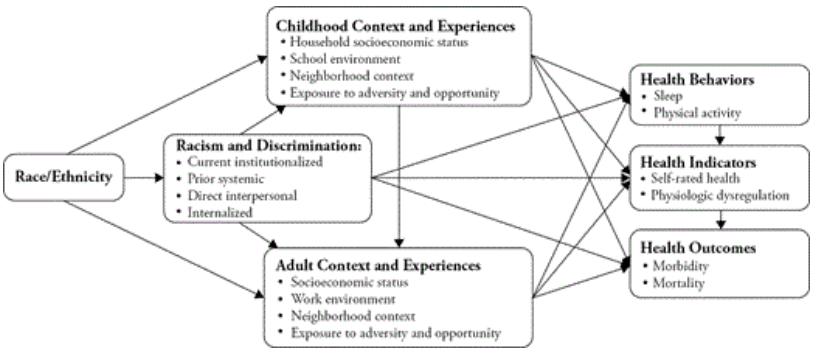

Figure 33.1 Conceptual model of mechanisms for racial health disparities.

There are several core questions that motivate this chapter. Which health indicators and outcomes evidence disparities? When in the life span do specific group differences arise? What are the behavioral and biological mechanisms? And, what are the social and contextual determinants of these mechanisms? Although a significant amount of research has been conducted on these topics, the state of current knowledge suggests that most of these questions remain largely unanswered.

Extant Findings on Group Differences in Health Outcomes and Indicators

We begin by summarizing prior research on racial disparities in health, focused on areas of inquiry that relate to key health measures in MIDUS and that point to needed future research directions. We underscore that this section is not an exhaustive review of the larger black–white health disparities literature. Where available, we give emphasis to longitudinal studies in summarizing what is known about racial differences in health outcomes.

Our primary focus in this section is on race differences in health outcomes that disadvantage AAs. However, it should be noted that there are domains wherein AA adults have more favorable health profiles than EAs (e.g., cholesterol, cardiac vagal control; Finkelstein, Khavjou, Mobley, Haney, & Will, 2004; L. K. Hill et al., 2015). Future ↳ research needs also to attend to the possible role of these biomarkers as protective in the morbidity risk of AAs and to examine racial differences in age trends to illuminate whether AAs maintain their advantage across the life course (for an example, see Fuller-Rowell et al., 2013).

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Life Expectancy and Mortality

Many studies have documented greater physical health declines and increased morbidity risk in young and middle adulthood among AAs as compared to EAs. These differences are implicated in substantial racial disparities in life expectancy. In particular, based on national vital records for 2013, AAs had a life expectancy that was 3.6 years less than that of EAs (Kochanek, Arias, & Anderson, 2015). Large longitudinal studies have also found greater mortality risk among AA than EA adults. Using National Health Interview Survey-Linked Mortality Files, Hummer and Chinn (2011) found that AA adults had 36% higher mortality than EAs during the 2- to 9-year follow-up period. Similarly, following a sample of middle-aged adults (51–61 years) over a 6-year period, Sudano and Baker (2006) reported that AAs had 82% higher mortality than EAs. When examined by age, black–white differences in mortality risk are larger at earlier stages of the life span (Hummer & Chinn, 2011; Şahin & Heiland, 2017).

Nonetheless, the black–white life expectancy gap has seen a reduction over the years (Harper, Lynch, Burris, & Davey Smith, 2007; Harper, Rushani, & Kaufman, 2012). For example, the racial gap has decreased

from 5.2 years in 1999 to 3.0 years in 2013 among females and from 6.8 years in 1999 to 4.4 years in 2013 among males, largely a result of smaller disparities in mortality from heart disease, cancer, HIV, and unintentional injuries (Kochanek et al., 2015).

Self-Rated Health

Using nationally representative longitudinal data, several prior studies have demonstrated a widening gap in self-rated health between AAs and EAs during adulthood. For example, using five waves of the Panel Study of Income Dynamics data (stretching from 1984–2001; *n* = 5,527), Shuey and Willson (2008) examined race differences over a 17-year period and found that AAs had worse self-rated health at baseline than EAs and that the gap increased over time, even after adjusting for socioeconomic indicators (Shuey & Willson, 2008). Similarly, Kim and Miech (2009) used data from three waves of the Americans' Changing Lives (ACL) study over 8 years (*n* = 3,497) to show that racial disparities in functional health widen across the adult years. The most pronounced gaps emerged in functional health through middle adulthood (Kim & Miech, 2009). Beyond midlife and into old age, longitudinal evidence suggests that the magnitude of racial disparities in self-rated health may then begin to stabilize around age 75 and slightly narrow thereafter, even after accounting for attrition and mortality selection bias (Kim & Miech, 2009; Liang et al., 2010).

With respect to historical time trends, Cummings and Jackson used cross-sectional data from the General Social Survey (*n* = 27,650) to examine the racial gap in self-rated health from 1974 to 2004. Results indicated that the black–white health gap narrowed between 1974 and 1984 for women only; otherwise, it remained relatively steady over the 30-year period (Cummings & Jackson, 2008). Using data from the National Health Interview Survey 1972 to 2009 waves (*n* = 1,304,201), Beck, Finch, Lin, Hummer, and Masters (2014) also showed that disparities in self-rated health have persisted across the last few decades.

Cardiovascular Disease

Racial disparities in ischemic heart disease and stroke have been documented and contribute to mortality differences between AA and EA adults (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005; Wong, Shapiro, Boscardin, & Ettner, 2002). One notable study utilized National Health and Nutrition Examination Survey (NHANES) data from 1999 to 2006 to examine black–white differences in prevalence rates for cardiovascular disease (CVD; heart failure, stroke, or myocardial infarction) as a function of age (Jolly, Vittinghoff, Chattopadhyay, & Bibbins-Domingo, 2010). Jolly et al. (2010) found that CVD prevalence was nearly two-fold greater among AAs than EAs aged 35–44, and that the relative risk decreased with each decade of age. Stroke incidence rates among AAs have also been found to be double that of EA adults, a disparity that is attenuated, but still significant, when adjusting for common stroke risk factors (Rosamond et al., 1999). The largest difference in stroke-related mortality between AAs and EAs is generally found in middle adulthood, with a convergence in adults aged 70–80 (Howard, 2013; Rosamond et al., 1999).

p. 460 Hypertension (HTN) is of great interest in the study of racial health disparities because of ↳ its prevalence, early onset, and possible role in contributing to black–white disparities in premature mortality (Wong et al., 2002). However, limited evidence is available on changes in blood pressure between blacks and whites across the life span. Racial differences in blood pressure and HTN prevalence are well documented in cross-sectional studies of adults (Krieger & Sidney, 1996; Piccolo, Yang, Bliwise, Yaggi, & Araujo, 2013; Redmond, Baer, & Hicks, 2011), yet the evidence during childhood and adolescence is mixed, with nearly two thirds of studies (33 of 56) finding elevated blood pressure among AA youth compared to EAs (Harshfield, 2011).

Using longitudinal data, our own and other research has shown that racial disparities in blood pressure emerge early, beginning in childhood or adolescence (Fuller-Rowell, Curtis, Klebanov, Brooks-Gunn, & Evans, 2017; X. Wang et al., 2006). Related research has also indicated that racial disparities in blood

pressure increase with age into young adulthood (Alpert & Fox, 1993). Focusing on a sample aged 15 to 65 (NHANES data; 1999–2002), Geronimus, Bound, Keene, and Hicken, 2007 (2007) examined race differences in HTN as a function of age. Although data were cross-sectional, results indicated that the relative risk of HTN increased from 1.7 to 3.1 between ages 15 and 65, and that racial disparities were larger among women, growing substantially between approximately 35 to 50 years (Geronimus et al., 2007). Longitudinal evidence has generally been consistent. Using two waves of data from the Coronary Artery Risk Development in Young Adults (CARDIA) study (aged between 33 and 45 at baseline), Knutson et al. (2009) found widening of blood pressure disparities between AAs and EAs (Knutson et al., 2009). Piccolo et al. (2013) examined racial disparities in 5-year incidence of HTN among Boston area residents aged 30–79 years and found that AAs had a two-fold higher incidence than EAs. Similarly, Pereira et al. (1999) found that 6-year follow-up HTN incidence rates were nearly twice as high among AAs (29% in AA women, 26% in AA men, 15% in EA women, and 17% in EA men; 45–64 years at baseline). Given the large health and economic burden associated with HTN, research is needed on the developmental periods during which blood pressure diverges between AAs and EAs and associated causes.

Diabetes

Type 2 diabetes has an approximately two-fold higher prevalence rate among AAs than EAs, a disparity that contributes disproportionately to premature mortality and other health conditions among AAs (Wong et al., 2002). Utilizing data from the Alameda County study (individuals aged 17–94 in 1965), Maty, Everson-Rose, Haan, Raghunathan, and Kaplan (2005) found Type 2 diabetes incidence rates over a 34-year period that were nearly double among AAs as compared to EAs. Furthermore, focusing on race differences in incident diabetes over 9 years of in-person and 17 years of telephone follow-up (ages 45–64 years at baseline), analysis of data from the ARIC (Atherosclerosis Risk in Communities) study found that AAs had greater rates of incident diabetes than EAs across the years of midlife and old age (Chatterjee et al., 2014). Across the 9-year follow-up, a separate analysis of ARIC data indicated that incidence rates were 2.4 times higher in AA women and 1.5 times higher in AA men than their EA counterparts (Brancati, Kao, Folsom, Watson, & Szklo, 2000). Studies using data from the Multi-Ethnic Study of Atherosclerosis (MESA; 45–84 years old in 2000–2002) have also found black–white disparities in diabetes incidence (Christine et al., 2015).

Obesity and Adiposity

Black–white disparities in body mass index (BMI) have been documented among females throughout the life span, but disparities among males are not as clearly evident (Y. Wang & Beydoun, 2007). Using NHANES 2011–2012 data, among adolescents (12–19 years) rates of obesity were 18.3 and 20.9 for EA males and EA females, respectively, and 21.4 and 22.7 for AA males and AA females, respectively. By middle adulthood (40–59 years), these same rates for males and females, respectively, were 41.1 and 36.3 for EAs and 38.2 and 58.6 for AAs, indicating a large disparity between black and white females (Ogden, Carroll, Kit, & Flegal, 2014). In the National Heart, Lung, and Blood Institute (NHLBI) Growth and Health Study, black–white disparities in BMI among females were present at age 10 and doubled during adolescence (Tomiyama, Puterman, Epel, Rehkopf, & Laraia, 2013). Longitudinal evidence and age trends among nationally representative samples also suggest that these disparities continue to increase into young adulthood, with a more than 20-point gap in obesity between AA and EA females found by approximately the third decade of life (Gordon-Larsen, The, & Adair, 2010; Y. Wang & Beydoun, 2007).

p. 461 Despite evidence that large racial disparities in BMI exist among females, studies have cast doubts ↳ on the relevance of these disparities for subsequent morbidity and mortality risk. In particular, BMI and other anthropometric markers were found to be associated with mortality only for EA adults, not AAs (waist circumference was the solo significant risk marker across racial groups) (Katzmarzyk et al., 2013).

Furthermore, other studies have shown racial disparities in diabetes and HTN to be larger at lower BMI values (Bell, Adair, & Popkin, 2002; Lipton, Uao, Cao, Cooper, & McGee, 1993; Resnick, Valsania, Halter, & Lin, 1998). Possible reasons for differential effects of BMI by race include that body fat is greater among EAs than AAs at equivalent BMI values (Deurenberg, Yap, & Van Staveren, 1998), and differences in body fat distribution (i.e., less visceral adiposity) may advantage blacks (Carroll et al., 2008). Investigations of black–white disparities in adiposity using MIDUS data can address these inconsistencies by utilizing accurate body scan measures of percentage body fat and fat-free muscle mass to better understand the racial gap in adiposity and its consequences.

Inflammation

Several studies have found higher concentrations of circulating pro-inflammatory markers among AAs than EAs. In MIDUS, higher levels of C-reactive protein (CRP) among AAs than EAs has been documented (Slopen et al., 2010). Convergent findings are evident in the Dallas Heart Study (Khera et al., 2005), the Health and Retirement Study (Mitchell & Aneshensel, 2016), and the North Texas Heart Study (Uchino et al., 2016). MIDUS researchers have also reported higher levels of interleukin 6, fibrinogen, E-selectin, and other markers of pro-inflammatory physiology among AAs (Boylan, Lewis, Coe, & Ryff, 2015; Slopen et al., 2010; Stepanikova, Bateman, & Oates, 2017). Overall, the evidence for race differences in low-grade systemic inflammation is substantial (Fuller-Rowell, Curtis, Doan, & Coe, 2015; Stepanikova et al., 2017). However, few studies have considered accelerated aging processes in relation to inflammation; that is, the degree to which racial disparities in inflammation increase across the adult years.

Beginning to address this research gap, Ferraro and Kim (2014) examined changes in CRP over a 5-year period in a sample of older adults (age 57–91 at baseline), finding higher CRP levels among AAs than EAs at both waves, but not a significant increase in the magnitude of the disparity across the 5-year period. However, when changes in fibrinogen were examined over a 15-year period (baseline age between 23 and 35) with CARDIA data, substantial increases in the magnitude of racial disparities were evident across this period, and they persisted after adjusting for a wide range of sociodemographic factors and health behaviors (Fuller-Rowell et al., 2015).

Neuroendocrine Stress Hormones

Cortisol and catecholamines are considered primary stress mediators of physiologic dysregulation and thus are important potential mechanisms for racial disparities in morbidity (McEwen & Seeman, 1999). However, current evidence suggests no clear black–white disparity in catecholamines. Specifically, in a sample of Chicago adults, AAs had higher epinephrine and norepinephrine levels than EAs, but these differences were not significant after adjusting for creatinine and fat-free mass (Masi, Rickett, Hawkley, & Cacioppo, 2004). Other investigations have also found no race difference in creatinine-adjusted catecholamine levels among adults (Castro-Diehl et al., 2014; Janicki-Deverts et al., 2007; Tomfohr, Pung, & Dimsdale, 2016).

Diurnal cortisol profiles have been linked to prior stress exposure and are predictive of subsequent morbidity and mortality (Kumari, Shipley, Stafford, & Kivimaki, 2011; Matthews, Schwartz, Cohen, & Seeman, 2006). Healthy cortisol profiles are characterized by relatively high waking levels, followed by an awakening response (an increase), declines across the day, and lower levels in the evening. Focusing on 50 AAs and a demographically matched comparison group of EAs from the MIDUS National Study of Daily Experiences, Fuller-Rowell, Doan, et al. (2012) examined racial disparities in diurnal cortisol profiles. Findings indicated that AAs have a flatter diurnal cortisol slope across the day than EAs. Similar race differences in patterns of diurnal cortisol have been found in other midlife samples, including in the CARDIA (Cohen et al., 2006) and MESA (Hajat et al., 2010) studies. Limited research has examined race differences in diurnal cortisol patterns longitudinally: In a small sample of Chicago adults aged 50 to 67 followed over a 5-

year period, DeSantis, Adam, Hawkley, Kudielka, and Cacioppo (2015) found that the magnitude of racial disparities in diurnal cortisol patterns were consistent over four waves.

Allostatic Load

p. 462 Aggregate indexes of multisystem physiologic dysregulation such as allostatic load (AL) are also ↳ important to consider in the study of racial health disparities. These measures are thought to index wear and tear on the body resulting from chronic activation of stress response systems. AL measures have been shown to predict subsequent morbidity and mortality (Duru, Harawa, Kermah, & Norris, 2012). Using data for adults aged 18–64 from NHANES IV (1999–2002), Geronimus and coworkers found that AL was higher among black adults than whites (Geronimus, Hicken, Keene, & Bound, 2006). The disparity appeared to grow with age and was largest between 35 and 64 years. Race differences in AL were also larger among women than men. Among adolescents participating in NHANES (1999–2008), a black–white gap in AL was found to already be present by age 12 (Rainisch & Upchurch, 2013).

Few longitudinal studies exist considering race differences in AL over time. One example comes from the Study of Women's Health Across the Nation, in which AL was measured among females aged 42–52 at baseline and 8 years later. Although AA women had higher AL at both waves, the disparity did not grow in size (Upchurch et al., 2015). Future research using longitudinal data across a wider age range is needed to examine race differences in AL accumulation.

Summary of Group Differences in Health Outcomes and Physiologic Dysregulation

In summary, prior research has identified key physical health indicators and diseases that are disproportionately experienced by AAs relative to EAs and lead to consistent racial disparities in life expectancy. Notably, HTN, heart disease, diabetes, and stroke are among the primary contributors to racial disparities in premature mortality, explaining approximately one third of the racial gap between males and more than half of the gap between females (Kochanek, Arias, & Anderson, 2013; Wong et al., 2002). Biomarkers that are likely mechanisms of racial disparities in cardiovascular and metabolic disease, such as circulating levels of pro-inflammatory markers and diurnal cortisol patterns, are also of vital importance to future research on health disparities. In addition, differences between AAs and EAs for both self-rated health and biomarker indicators appear to emerge by adolescence or young adulthood and widen into middle adulthood (Geronimus et al., 2006, 2007; Kim & Miech, 2009; Ogden et al., 2014). Thus, efforts to reduce racial disparities are likely to be optimal if focused on the first half of the life span. Black–white disparities have also been shown to be sex specific, with some outcomes occurring at particularly high rates among black females (e.g., diabetes, obesity), while others have been especially high among black males (e.g., being a victim of gun violence, contracting HIV) (Allgood, Hunt, & Rucker, 2016; Kochanek et al., 2013). Accordingly, contributions to recent progress in reducing the black–white life expectancy gap from 1999 to2013 have also been sex specific, with heart disease being the primary contributor for females and HIV for males (Kochanek et al., 2015). Consideration of sex-specific pathways that lead to racial disparities in physiologic dysregulation and disease risk is needed.

Probing Adult Contexts and Psychological Experiences as Mediators

Many factors, ranging from biological, to psychological, to social, may contribute to race/ethnic differences in health. Although gene expression and epigenetic factors may play a role in racial health disparities (Kuzawa & Sweet, 2009), the current scientific consensus is that purely genetic factors (i.e., the genes themselves) account for very little, if any, of the overall racial disparity in health outcomes at the population level (Kaufman, Dolman, Rushani, & Cooper, 2015; Williams & Sternthal, 2010). Instead, social and historical oppression creates life conditions and opportunities, or lack thereof, that constitute more promising directions for understanding what underlies group differences in health (Williams, Mohammed, Leavell, & Collins, 2010). In particular, past and present racist practices and policies dating back to slavery, Jim Crow segregation laws, housing discrimination, neighborhood segregation, as well as direct experiences of unfair treatment, have translated to profound differences in the social and environmental conditions that exist across racial/ethnic groups (Coates, 2014; Williams & Mohammed, 2009). These conditions—many of which are ongoing—impact the contexts of people's lives and everyday experiences, which in turn shape trajectories of health across the life span (Marmot, 2005; Marmot, Friel, Bell, Houweling, & Taylor, 2008).

Racism has been conceptualized as existing at multiple levels of analysis, including institutionalized practices (past and present), personally mediated experiences, and the internalization of negative messages (Jones, 2000). These layers have shaped the landscape of experiences and opportunities for AAs and thereby set the stage on which health disparities are played out (Brondolo, ↳ Gallo, & Myers, 2009; Krieger, 2014; Williams & Mohammed, 2013). Under the broader umbrella of racism and related sociohistorical processes, four specific aspects of adult experiences that may account for group differences in health are covered in this section: (a) experiences of discrimination, (b) neighborhood characteristics, and (c) SES and the differential health consequences of educational attainment. For each, we discuss current research, with a concluding section at the end of our chapter covering important future research directions relating to black–white health disparities.

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Experiences of Discrimination

Converging evidence from experimental studies of discrimination in employment and housing (Massey & Lundy, 2001; Pager, 2007; Pager & Shepherd, 2008; Pager, Western, & Bonikowski, 2009) as well as from self-reports of interpersonal experiences (Ancis, Sedlacek, & Mohr, 2000; Fuller-Rowell, Curtis, El-Sheikh, et al., 2017; Fuller-Rowell, Doan, et al., 2012) indicate that AAs are exposed to substantially higher levels of unfair treatment than EAs. Experiences of discrimination are therefore one potential explanation for racial health disparities.

Individuals exposed to unfair treatment in lab settings show physiologic responses (Clark, 2000; Harrell, Hall, & Taliaferro, 2003; Pascoe & Smart Richman, 2009), which if repeated over time would be expected to lead to wear and tear on the body and resulting dysregulation of organ systems (Fuller-Rowell, Evans, & Ong, 2012; Mays, Cochran, & Barnes, 2007). A handful of studies have shown that AAs who report higher levels of everyday discrimination have higher levels of CRP (Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010); more coronary artery calcification (Lewis et al., 2006); more visceral fat (Lewis, Kravitz, Janssen, & Powell, 2011); higher blood pressure (Lewis et al., 2009); and greater risk of mortality (Barnes et al., 2008). However, many published studies reported no significant association or a nonlinear association between perceived discrimination and physical health outcomes among AAs (Krieger & Sidney, 1996; Williams & Mohammed, 2008, 2013). Furthermore, relatively few studies have found that perceived discrimination mediates black–white health disparities, despite significant attention to this research area (Fuller-Rowell et al., 2015). Such findings have led scholars to consider alternative conceptualizations of perceived discrimination measures.

Historically, most researchers have conceptualized perceived discrimination as a measure of stress exposure. However, other recent perspectives emphasized that the extent to which individuals report experiences of discrimination also reflects their awareness and interpretation of their social reality. For example, it has been suggested that, for highly stigmatized groups such as AAs, acknowledging the existence of discrimination in daily life may be an important component of a well-adjusted psyche (Cross, 1991; Helms, 1995; Sellers, Smith, Shelton, Rowley, & Chavous, 1998; Spencer & Markstrom-Adams, 1990). Furthermore, evidence suggests that because instances of racial discrimination are likely occurring on a regular basis for members of stigmatized groups, acknowledging the presence of discrimination may be necessary for effective coping and adjustment (Major, Quinton, & McCoy, 2002). Contrastingly, a lack of reported discrimination may indicate avoidance, denial, or suppression, which have been associated with a host of negative health outcomes (Jorgensen & Thibodeau, 2007). These viewpoints suggest that between-person differences in levels of reported discrimination may reflect more than just stress exposure, particularly among highly stigmatized groups.

To test these perspectives, we used MIDUS data to examine the influence of perceived discrimination on the diurnal cortisol rhythm of AAs and a comparison group of EAs (Fuller-Rowell, Doan, et al., 2012). Findings indicated that perceived discrimination did not account for disparities in diurnal cortisol across racial groups. Rather the influence of discrimination on the diurnal slope was in opposite directions for each racial group. For EAs, higher levels of discrimination were associated with a flatter (more dysregulated) diurnal slope. This pattern is consistent with the conceptualization of perceived discrimination as a chronic stressor and a risk factor for physiologic dysregulation and poorer physical health (Fuller-Rowell et al., 2011; Fuller-Rowell, Evans, et al., 2012). For AAs, however, perceived discrimination was associated with a steeper (healthier) diurnal slope. Alongside other research in adult samples (Chae, Lincoln, Adler, & Syme, 2010; Chae, Nuru-Jeter, & Adler, 2012; Cunningham et al., 2012), the findings suggested that conceptual and methodological innovations will be necessary to more fully account for the impact of discrimination and unfair treatment on racial health disparities. Possibilities for future research in this area are discussed further in this chapter.

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Neighborhood Environments

Numerous studies have indicated that AAs live in far less advantaged neighborhoods than EAs, with lower average levels of income and education, greater concentrated poverty, poorer schools, fewer opportunities for employment and physical activity, and less access to healthy food (Fuller-Rowell, Curtis, Klebanov, et al., 2017; Osypuk, Galea, McArdle, & Acevedo-Garcia, 2009; Rossen, 2014; Roux et al., 2001; Williams & Collins, 2001; Zenk et al., 2005). The sociohistorical factors underlying race differences in neighborhood quality are well documented (Coates, 2014), as are processes of stigma and racism that have played a central role in creating and sustaining neighborhood segregation (Massey, 2007; Wilson, 2008). Race differences in neighborhood quality remain substantial even after accounting for individual-level socioeconomic variables (Roux et al., 2001; Sharkey, 2014). Illustrating this point, one recent study found that AA families making upward of $100,000 typically live in neighborhoods inhabited by EA families making $30,000 or less (Sharkey, 2014).

Although neighborhood factors generally account for a portion of the association between individual SES and health outcomes, both individual and neighborhood SES have been found to exert independent effects on health and often account for unique variance in racial health disparities (Chen & Paterson, 2006; Fuller-Rowell, Curtis, Klebanov, et al., 2017; Robert, 1998; Roux et al., 2001). However, few studies have examined the role of neighborhood factors as an explanation for black–white disparities in health; fewer still have done so longitudinally or explored mechanisms for neighborhood effects on health disparities. We examined neighborhood disadvantage as an explanation for race differences in blood pressure trajectories of young children (Fuller-Rowell, Curtis, Klebanov, et al., 2017). Results showed that black–white

differences in blood pressure began to emerge and grew across childhood, and that neighborhood disadvantage mediated a portion of these race differences in trajectories. This and other studies suggest that addressing race differences in neighborhood environments will be key to reducing racial health disparities, and that a policy agenda toward this goal is sorely needed (Massey, 2007; Sharkey, 2013).

Socioeconomic Status and the Health Consequences of Educational Attainment

Black-white disparities in education and income are well documented (Williams et al., 2010). Furthermore, large black–white gaps in wealth exist independent of other socioeconomic indicators and have widened since the Great Recession (McKernan, Ratcliffe, Steuerle, & Zhang, 2014). It is therefore not surprising that SES has received considerable attention as an important determinant of group health disparities (Myers, 2009; Williams & Sternthal, 2010). Although many studies have demonstrated that SES indicators account for a portion of group differences in health outcomes, these studies often lack detailed socioeconomic indicators or longitudinal data on SES, health, or both. MIDUS thus affords important opportunities to examine the role of SES in health disparities across the life span.

Overall, the health benefits of educational attainment are well established (Cutler & Lleras-Muney, 2006; Rogers, Hummer, & Everett, 2013). One approach to addressing health disparities is therefore to reduce inequality in levels of educational attainment across racial groups. However, this route to achieving health equity is complicated by the possibility that the health benefits of educational attainment may be less pronounced for AAs than for EAs. In order to advance pathways to upward mobility and life success, it is critically important to understand how and why breakdowns in the link between education and health might occur. Such an understanding will be essential to the elimination of racial health disparities.

A handful of studies have focused on race differences in the health consequences of educational attainment. Focusing on self-rated health (Reagan & Salsberry, 2014; Shuey & Willson, 2008); markers of cardiovascular risk (Fradkin et al., 2015; Fuller-Rowell et al., 2015; Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009; Lewis et al., 2005; Y. Wang & Beydoun, 2007; Williams, 2003); sleep problems (Fuller-Rowell, Curtis, El-Sheikh, et al., 2017); and mortality (Hayward, Hummer, & Sasson, 2015; Montez, Hummer, & Hayward, 2012), these studies provided initial evidence that, compared to EAs, the health benefits of educational attainment may be less pronounced for AAs. In a recent longitudinal analysis, we found that among EAs, higher levels of educational attainment were associated with an attenuated increase in fibrinogen, a thrombotic and pro-inflammatory marker, between ages 30 and 45. However, educational attainment showed no protective benefit for AAs (Fuller-Rowell et al., 2015). Furthermore, a recent study using MIDUS data found that higher levels of educational attainment were associated with higher levels of systemic
p. 465 inflammation among ↳ AAs, particularly among those who scored higher on measures of anger (Boylan et al., 2015). Studies also suggest that the social costs of academic and occupational success may be greater for AAs than for EAs (Fuller-Rowell & Doan, 2010; Smith & Witt, 1993), such that social support increases over time for academically successful EAs, while AAs show no social benefit from academic achievement (Fuller-Rowell & Doan, 2010). Although studies to date have been illuminating, research on this topic is in its infancy.

Probing Intervening Health Behaviors

In this section, we discuss sleep and physical activity as two health behaviors that may play important roles in health disparities. Both represent promising areas for future research on racial disparities using MIDUS data.

Sleep Problems

Insufficient or low-quality sleep has been consistently associated with increased disease risk, including diabetes (Knutson, 2010) and heart disease (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011), as well as life expectancy and mortality (Gallicchio & Kalesan, 2009). Evidence also suggests that sleep is a consequential mediator of established associations between social/physical environment factors and health (Benham, 2010; Jackson, Redline, & Emmons, 2015; Knutson, 2010). All told, sleep problems are significant impediments to health and well-being, which in the United States alone carry an economic burden of hundreds of billions of dollars each year (Institute of Medicine [US] Committee on Sleep Medicine and Research, 2006).

Recent reviews revealed that AA adults show consistently poorer sleep than EAs (Grandner, Williams, Knutson, Roberts, & Jean-Louis, 2016; Petrov & Lichstein, 2016; Ruiter, DeCoster, Jacobs, & Lichstein, 2011). In particular, studies indicated fewer total sleep minutes, poorer sleep efficiency, greater onset latency, and worse overall sleep quality among AAs relative to EAs. These differences have been documented using self-report and objective sleep measures, with objective measures generally showing larger racial disparities (Grandner et al., 2016; Petrov & Lichstein, 2016). Further, race differences in sleep are partially accounted for, but endure, after measures of SES are controlled (Grandner et al., 2013, 2016; Whinnery, Jackson, Rattanaumpawan, & Grandner, 2014).

The mechanisms for race differences in sleep are not well understood and analyses considering life course determinants of group differences are sorely needed (Grandner et al., 2016; Laposky, Van Cauter, & Diez-Roux, 2016; Petrov & Lichstein, 2016). Possible candidate mechanisms include discrimination (Fuller-Rowell, Curtis, El-Sheikh, et al., 2017; Slopen, Lewis, & Williams, 2016); chronic stress (De Lange et al., 2009); health behaviors (Strine & Chapman, 2005); and exposure to physical environment toxins (Kordas et al., 2007). Neighborhood contexts represent an additional candidate mechanism, likely to operate through a range of individual-level variables (Bagley, Fuller-Rowell, Saini, Philbrook, & El-Sheikh, 2016; DeSantis et al., 2013; Hale et al., 2013; T. D. Hill, Trinh, Wen, & Hale, 2016).

Using MIDUS data, we recently examined objective census tract measures of neighborhood disadvantage as a mediator of differences in objectively measured sleep between AA and EA adults (Fuller-Rowell et al., 2016). AA adults obtained 36 minutes less sleep and were awake for 13 minutes more during the night than EA participants. Furthermore, neighborhood disadvantage was shown to be associated with waking after sleep onset, a measure of disrupted sleep, and to mediate one fourth of the race difference in this sleep parameter. The race difference in waking after sleep onset was further attenuated by one quarter when adjusting for key health behaviors (BMI, substance use, physical activity, depression) (Fuller-Rowell et al., 2016). One direction for future research is to consider specific mechanisms for neighborhood effects on sleep disparities. Thus, one strength of MIDUS is the census tract–based sampling design of the AA sample in Milwaukee, Wisconsin, that can be paired with the extensive measurement of person-level psychosocial and health factors, thereby allowing for within-group investigation of potential psychosocial and health mediators of the association between neighborhood context and sleep.

Key research has also begun to consider sleep as a mediator of race differences in health outcomes (Knutson et al., 2009; Sherwood et al., 2011). Knutson et al. (2009) found that shorter objectively measured sleep duration among AA adults compared to EAs mediated the race difference in diastolic blood pressure change over 5 years. Sherwood et al. (2011) also found that AAs experienced poorer sleep quality than EAs (based on an index using subjective and objective reports), and that this difference explained nearly half of the
p. 466 increased likelihood among AAs not to experience ↳ decreases in nocturnal blood pressure (i.e., to be nondippers).

Our ongoing research in MIDUS has shown that—using a composite of seven cardiometabolic biomarkers similar to metabolic syndrome—approximately one half of the black–white disparity in cardiometabolic risk is mediated by objective measures of sleep duration and quality (Curtis, Fuller-Rowell, et al., 2017). Importantly, in sex-stratified models, associations between sleep parameters and cardiometabolic risk were found only among females, mediating up to two thirds of the difference between black and white females. We have also shown that objectively measured sleep quality was a mediator of diverging cardiometabolic risk between black and white females over a 10-year period in middle adulthood (Curtis et al., 2017). Given that more than one half of the black–white gap in life expectancy stems from cardiometabolic conditions, reducing differences in sleep is important for broader efforts to reduce racial health disparities.

Physical Activity

Frequent moderate-to-vigorous physical activity is one of the most robust predictors of healthy aging (T. F. Nelson, Gortmaker, Subramanian, Cheung, & Wechsler, 2007; Peel, McClure, & Bartlett, 2005). In addition to consistent associations with mental health and cognitive functioning (Lautenschlager et al., 2008; Paluska & Schwenk, 2000), meeting recommended levels of physical activity has beneficial effects on a host of health indicators, including adiposity (Must & Tybor, 2005); blood pressure (Cornelissen & Smart, 2013); insulin resistance (Ivy, 1997; R. K. Nelson et al., 2013); and inflammation (Abramson & Vaccarino, 2002; Geffken et al., 2001; Tsukui et al., 2000). The health benefits of physical activity are widely known and the related physiologic mechanisms are quite well understood.

Although some population studies have found similar levels of physical activity among AA and EA adults (Ranchod, Roux, Evenson, Sánchez, & Moore, 2013), most studies have found that AAs engage in less moderate or vigorous activity (Tucker, Welk, & Beyler, 2011) and are less likely to meet overall recommended activity levels than EAs (Haskell et al., 2007). Black–white disparities in physical activity generally remain significant after measures of SES are controlled (Parks, Housemann, & Brownson, 2003; Trost, Owen, Bauman, Sallis, & Brown, 2002; Wilcox, Castro, King, Housemann, & Brownson, 2000). Considered across the life span, there is some evidence to suggest that disparities in physical activity between AAs and EAs emerge in early adulthood (Harris, Gordon-Larsen, Chantala, & Udry, 2006; Richmond, Hayward, Gahagan, Field, & Heisler, 2006). Longitudinal evidence, albeit limited, suggests some stability in the magnitude of racial disparities during adulthood (Boone-Heinonen et al., 2011). However, more research is needed to determine the degree to which disparities in physical activity are stable across the adult years or continue to grow.

The social determinants of racial disparities in physical activity are likely to be numerous but are not well understood. Experiences of discrimination have been conceptualized as one possible predictor (McNeill, Kreuter, & Subramanian, 2006). However, little empirical attention has been given to this topic, and the limited evidence suggests that discrimination may not be significantly associated with physical activity (Shelton et al., 2009; Womack et al., 2014). Further research is needed using various measures of discrimination, cross-sectional and longitudinal data, as well as potential moderating roles of age, gender, and SES. Another likely explanation for race differences in physical activity are neighborhood factors. Although a range of studies have found that characteristics of the physical environment, such as the availability of sidewalks, parks, and recreational facilities play an important role in levels of physical activity (Hoehner, Brennan Ramirez, Elliott, Handy, & Brownson, 2005; Humpel, Owen, & Leslie, 2002; Huston, Evenson, Bors, & Gizlice, 2003; Saelens, Sallis, Black, & Chen, 2003; Sallis et al., 2009), few studies have considered the role of neighborhood environments in black–white physical activity disparities. MIDUS data provide important venues to pursue discrimination, neighborhood context, and other psychosocial factors as mechanisms for disparities in physical activity using both cross-sectional and longitudinal data.

Bringing in Childhood Context and Experiences

Prevailing theories hold that frequent activation of stress response systems—particularly during sensitive periods in childhood—influence the maturation and functioning of physiologic systems, especially endocrine, immune, and central nervous (Danese & McEwen, 2012; Miller, Chen, & Parker, 2011). For
p. 467 example, prior studies have shown that ↳ ELA leads to dysregulated hypothalamic–pituitary–adrenal (HPA) axis functioning (e.g., elevated evening cortisol, flatter diurnal slope), elevated circulating pro-inflammatory markers, blunted autonomic responses to stress, and changes in brain volume and function (e.g., reduced prefrontal and hippocampal volume, enlarged amygdala) (Danese & McEwen, 2012; McLaughlin et al., 2015; Miller et al., 2011). Thus, ELA can become embedded into biology in a way that predisposes AA children to chronic diseases through prolonged activation and diminished responsiveness across physiologic systems. Furthermore, ELA-induced impairments in executive functioning, top-down emotion regulation, as well as the adoption of ineffective coping strategies can lead to associated risky health behaviors (Evans, Fuller-Rowell, & Doan, 2012; Lovallo, 2013). The biological and behavioral changes associated with ELA, in turn, have been shown to have a wide impact on physical health. Among adult samples, ELA has been associated with many chronic diseases (e.g., heart disease, cancer, lung disease) and premature mortality (Felitti et al., 1998; Holman et al., 2016; Kelly-Irving et al., 2013; Tamayo, Herder, & Rathmann, 2010).

Race Differences in Childhood Adversity

The ecological context in childhood among AAs, on average, varies substantially from that of their EA peers, resulting in large differences in ELA. AA youth generally experience greater health risks in the form of poverty, single-parent households, segregation, discrimination, and violence exposure. For example, rates of childhood poverty in 2010 were 17% among EAs yet 38.2% among AAs (Macartney, 2011). The influence of the racial poverty gap is substantially worsened by the increased likelihood of AA youth to live in neighborhoods characterized by concentrated poverty and hypersegregation (Lichter, Parisi, & Taquino, 2012; Massey & Denton, 1989). Beyond disparities in economic conditions, the experience of being black in America is nearly synonymous with exposure to discrimination (Seaton, Caldwell, Sellers, & Jackson, 2008; Williams & Mohammed, 2008).

As a result of these diverging ecologies, AA youth experience greater ELA than EAs (Slopen et al., 2010; Slopen, Shonkoff, et al., 2016; Thoits, 2010). One recent study used National Survey of Children's Health data to document black–white disparities in family financial hardship, parental death and incarceration, interparental violence, neighborhood violence, and racial discrimination, which are particularly pronounced among higher income strata (and absent among lower income comparisons) (Slopen, Shonkoff, et al., 2016). Overlooking these divergent contexts when examining reasons for black–white disparities in health in adulthood may therefore be a critical misstep.

Childhood Adversity as Mediator of Race Differences in Health

Given that ELA influences health throughout the life course, the degree to which ELA exposure varies by racial group may help to explain diverging health trajectories for AAs and EAs beginning in youth and extending into adulthood (Braveman & Barclay, 2009). However, little research has considered this question empirically. Our prior research and others' has found support for the hypothesis that ELA is a mediator of the black–white gap in BMI in adolescent and young adult samples (Curtis, Fuller-Rowell, Doan, Zgierska, & Ryff, 2016; Lee & Hicken, 2013). For example, controlling for SES, Curtis et al. (2016) found substantial race differences in exposure to ELA, assessed using a broad 21-item index of adverse events/contexts. Furthermore, after adjusting for sociodemographics and relevant health behaviors, ELA was significantly associated with BMI and accounted for 42% of the racial disparity. Midei, Matthews, Chang, and Bromberger (2013) also reported that childhood physical abuse rates were higher among AA adults than EAs, which in turn predicted incident metabolic syndrome over a 7-year follow-up. In contrast, using data from the CARDIA study, Fuller-Rowell et al. (2015) examined ELA as an explanation for race differences in pro-inflammatory markers across a 15-year period in middle adulthood but did not find support for this hypothesis. However, the ELA measure in CARDIA primarily included items about parenting rather than early life factors known to vary by race (i.e., neighborhood conditions, violence exposure, discrimination).

Furthermore, at least two prior studies have found support for AAs being more vulnerable to the sequelae of ELA (Curtis et al., 2016; Slopen et al., 2010). Our own research found that the association between ELA and BMI was only present among AA young adults and not EAs, such that there was no racial gap in BMI at low levels of ELA but wide disparities at high ELA exposure (Curtis et al., 2016). Utilizing MIDUS Biomarker project data, Slopen et al. (2010) showed that ELA was associated with several pro-inflammatory markers
p. 468 among AAs, but not EA adults. In this ↳ study, ELA was measured with items from the MIDUS 2 survey that assessed school problems, household dysfunction, parent–child relationship quality, and parent-perpetrated verbal and physical assault. One potential reason for race differences in vulnerability to ELA may be the broader societal context that offers AA youth fewer resources while presenting a broad array of stressors. Therefore, future research on racial disparities should consider additional types of ELA known to disproportionately affect AA youth (e.g., neighborhood instability, broader violence exposure; Bures, 2003), as well as comprehensive measures of background socioeconomic resources (MIDUS has broad assessments of parents' socioeconomic position).

Studies Considering Childhood Adversity Alongside Adult Context and Experiences

Although ELA may influence physical health into adulthood through cascading socioeconomic influences (i.e., decreased likelihood of educational attainment and lower earnings) and proliferation of chronic stress (Pavela & Latham, 2015; Pearlin, Menaghan, Lieberman, & Mullan, 1981; Tamayo et al., 2010), a range of studies provides evidence that ELA has effects beyond those associated with adult socioeconomic factors and adult stress exposure (Danese et al., 2009; Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Kelly-Irving et al., 2013; Poulton et al., 2002). One MIDUS study, for example, found socioeconomic disadvantage in childhood and socioeconomic disadvantage at the MIDUS 1 and 2 assessments to be independently associated with AL (Gruenewald et al., 2012). Life course socioeconomic trajectories were also influential, such that individuals with persistent high socioeconomic disadvantage had the highest AL scores, followed by the downwardly mobile, upwardly mobile, and persistent low socioeconomic disadvantage groups. However, analyses were based primarily on EA participants, leaving open many important questions relating to the role of ELA and adult factors for AAs and the role of ELA in racial health disparities.

Along these lines, one area of research indicates that the health consequences of upward mobility may be substantial for AA youth (Brody, Yu, Miller, & Chen, 2016; Chen, Miller, Brody, & Lei, 2014), suggesting that

additional studies considering the interactive effects of early life experiences and adult socioeconomic factors across the life span may be revealing. We believe that careful attention should be given to the various ways in which early life and adult experiences combine to influence health trajectories across the adult years and how such influences vary between AA and EA adults.

Promising Future Directions for Research on Racial Disparities in Health

We have demonstrated in this chapter that black–white disparities in many health outcomes have been well documented. However, the degree to which group differences in specific health indicators widen across the adult years has received considerably less attention. Furthermore, the biological mechanisms that precede disparities in specific outcomes are insufficiently understood. Because MIDUS has extensive assessments of health and physiologic dysregulation, samples with a large age range, and two assessment periods spaced apart by a decade (with biomarker data from both assessments soon to be available), there is great potential for substantial contributions to knowledge of racial health disparities relating to age and sequencing of physiologic dysregulation. For example, cross-lagged analytic models could be used with two waves of MIDUS data to consider how disparities in pairs of health indicators unfold (e.g., interleukin 6 and insulin resistance). Furthermore, applying such an approach and its variants systematically to multiple pairs of health outcomes can lead toward a clearer picture of the biological mechanisms that underlie racial disparities in health.

More detailed analyses considering changes across specific age ranges will also be essential to elucidate the points in the adult life span (e.g., early adulthood, early midlife, etc.) that are most critical for the study of disparities in specific outcomes. For example, with two waves of data, it will be possible to examine the degree to which age moderates changes in health indicators over time. Such analyses will provide important insight into the question of when in the life span disparities in specific health outcomes emerge, which will facilitate focused empirical examination of social determinants during specific developmental periods.

Social and Behavioral Determinants

Many avenues for future research on the social determinants of racial health disparities exist. Here, we highlight those that can be addressed using MIDUS data.

Discrimination

p. 469 An important next step in research on discrimination is to consider latent (or residualized) change ↳ scores of perceived discrimination as predictors of health outcomes and changes in health over time. So doing will move from an emphasis on between-person differences in levels of reported discrimination—which may be confounded with racial identity and other personality factors—and instead emphasize within-person fluctuations over time to capture changes in social stress exposure. Such an approach can be considered across the range of measures of unfair treatment and health outcomes included in MIDUS to attempt to advance research on the pathways through which discrimination may be influencing physiologic dysregulation. Additionally, because racial identity is known to be associated with perceived discrimination (Sellers & Shelton, 2003), another important approach will be to remove variance relating to racial identity (e.g., items in MIDUS on how closely individuals identify with their racial group and prefer to be around those of the same race) from measures of perceived discrimination (e.g., by regressing perceived discrimination on racial identity measures and saving the residuals). Such an approach allows for examination of whether the remaining variance in perceived discrimination may be more reflective of stress exposure and thus more predictive of health outcomes.

Differential Consequences of Educational Attainment

The research described indicates that, with respect to health, AAs do not benefit from higher levels of education to the same degree as EAs. However, studies have not adequately considered psychosocial mediators of education–health associations across racial groups or which behavioral and biological mechanisms account for racial differences in the association between education and health. MIDUS has the potential to contribute to addressing these knowledge gaps. For example, detailed biomarker assessments spanning multiple physiologic systems (Karlamangla et al., 2014; Wiley, Gruenewald, Karlamangla, & Seeman, 2016) will allow for examination of the biological pathways explaining race differences in the health consequences of educational attainment. Furthermore, data from MIDUS 3 can be paired with detailed psychosocial assessments to examine potential mediators of race differences in the physiologic consequences of education using longitudinal data.

Work Environments

Work environments are an understudied yet important topic for moving the science of racial health disparities forward. The MIDUS study affords important opportunities in this domain. In particular, building on prior research, objectively measured characteristics of occupations from occupational databases can be linked to individual occupations, which have been measured in detail in MIDUS and coded based on the 501 census occupation categories (Grzywacz & Butler, 2005; Meyer, Cifuentes, & Warren, 2011). This rich objective information on work environments can be examined alongside detailed self-reports also included in MIDUS to allow for a robust examination of the role of workplace context in racial health disparities.

Health Behaviors

Future research utilizing MIDUS data could advance knowledge on the links between sleep and health by considering both subjective reports (i.e., Pittsburgh Sleep Quality Index) and objective measurements of habitual sleep as well as a broad assessment of physiologic markers. Such research could elucidate potential race differences in the meaning of subjective sleep reports and the differential health consequences of self-reported short sleep duration, findings suggested by prior research (Jackson, Redline, Kawachi, & Hu, 2013; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Pandey et al., 2013). Longitudinal biomarker data will also add critical evidence of the role of sleep as a mediator of widening racial health disparities over adulthood, overcoming existing limitations of cross-sectional studies.

Using the formula developed by Strohacker, Wing, and McCaffery (2013), detailed measures of physical activity in the MIDUS Biomarker project can be standardized into metabolic equivalents (METs) to account for varying intensities (e.g., moderate vs. vigorous) and whether individuals meet recommended physical activity guidelines. Furthermore, in the questionnaire component of the larger MIDUS study, self-report measures of physical activity broken down by context (e.g., leisure time vs. work) are also collected. The MIDUS dataset is therefore well positioned to consider the social determinants of disparities in physical activity and the mediating role of physical activity in the link between psychosocial factors and health indicators.

With two large nationally representative samples of adults and two city-specific samples of AAs, the MIDUS study provides significant opportunities to move the science of racial health disparities forward. Our hope is
p. 470 that by critically reviewing and ↳ providing commentary on the current state of the literature, this chapter will stimulate new and impactful work to understand and address disparities in health between black and white Americans.

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