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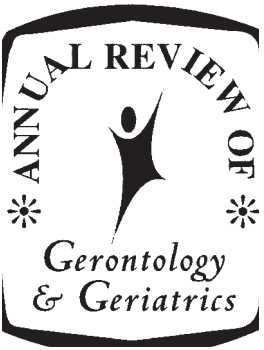

Annual Review of Gerontology and Geriatrics

Life-Course Perspectives on Late-Life Health Inequalities

VOLUME 29, 2009

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Introduction

Increased longevity and reduced fertility are producing a unique occurrence in many countries around the world. The coming aging society is resulting from the reshaping of the age distribution such that there soon will be more individuals over the age of 65 than there are under 15 years of age (Rowe & Berkman, 2009). This is a significant achievement of the 20th and early 21st centuries. It is evidence that we have conquered (or nearly conquered) many challenges, especially in advanced industrial nations. These challenges include: death by childbirth and infectious diseases, better control of chronic illnesses, and reduced rates of infant mortality. The changes have generated many new challenges for individuals and governments (Robinson, Novelli, Pearson, & Norris, 2007). Also significant in the changing age demographics in the United States is the increase in the numbers and types of ethnic and racial minority groups because of the same population fertility and longevity forces, as well as significant immigration (Angel & Hogan, 2004). Individuals in many of these race–ethnic groups have distinct life-span histories and life course experiences that unfortunately are often associated with early disadvantages and health inequalities in later life (Jackson & Govia, 2009).

Considering social, economic, health, and psychological disparities among social groups over the life course demands that we consider how such disparities may arise, how they are maintained, and how they are reproduced over generations. Most theoretical formulations are either silent about the sources of such disparities or are static in their notions of fundamental social causes. In this book we conceptualize disparities as a complex, dynamic function of intergenerational positioning, historical time, period events, and cohort differences. While this conceptualization makes more complex the notion of fundamental social causes, we think that it is appropriate and necessary. It will aid us to understand at any given point in time and space and individual age, how group memberships shape socioeconomic circumstances, life chances, and health statuses.

People arrive at specific points in their life course at particular ages, in particular spaces, with particular liabilities because of a complex web of

genetics, epigenetic influences, parental opportunities, as well as their family, friends, and institutional memberships, and their own unique experiences over their lives. More importantly, because group memberships produce very complex, interweaving experiences dictated by genetic endowments, historical family experiences and opportunities, environmental affordances as well as individual and group life experiences, any individual sharing a particular social attribute (gender, race, ethnicity, etc.) may become enmeshed with the life experiences of others who may share their race, ethnicity, gender, or geographical location. Thus, the individual intergenerational and life-course experiences become entangled with those of other, nonfamilial members of their social groups for whom their contact may only be imagined over vast geographical (and historical) distances (Anderson, 1989). For example, the particular lives of religious Jews is not only a function of their current daily lives, the lives of their contemporary and intergenerational families, but also a rich historical understanding of the embedded nature of the group experiences over an extended historical period encompassing not only the 20th-century holocaust but also Egyptian bondage in 300 BC.

What is becoming clear and is well represented in the contributions to this volume is that individual, intergenerational familial, and social group exposures intersect with historical time, cohort experiences, and individual aging. These intersections produce contemporary social, economic, psychological, and health disparities at different points in the individual life span when the race, ethnic, or socioeconomic group membership(s) of one person is compared to the group membership(s) of another. The chapters in this volume examine the complex web of genetic and epigenetic influences, congenital, and early life circumstances, life exposures, and life experiences that eventuate in late-life inequalities among groups and individuals by social, economic, and race and ethnic statuses.

ORIENTATION TO CHAPTERS IN THIS VOLUME

Section I: Frameworks for Understanding Late-Life Health Disparities

The first section of the book addresses frameworks and approaches to understanding lifetime influences on late-life inequalities in health. In the first chapter Fuller-Iglesias, Smith, and Antonucci make several important points about the crucial role that life-span and life-course considerations play in understanding health differentials among social and economic groups in late life. Drawing a distinction between intraindividual development in the individual focused life-span theories of development and aging, and the more

macroconsiderations of social pathways inherent in the life-course conception of development and aging, they argue for the need for a blending of both conceptual perspectives in understanding the nature of disparities and inequalities in later life.

In the second chapter Glymour, Ertel, and Berkman explore the nature of life-course epidemiology. They examine how core findings from a life-course epidemiology perspective can better inform an understanding of health inequalities in late life, and view this as an important first step in thinking about the most promising points to intervene in order to eliminate disparities in later life. The core premise of life-course epidemiology is that health in late life reflects a lifetime of past exposures, yet the authors feel that this perspective has not been fully integrated into the design and implementation of health interventions or preventive health policies. They note that the life-course perspective can inform several fundamental considerations in developing interventions, for example, when the risk factor or exposure is amenable to modification; when the disease process begins; and the most sensitive time points to interrupt its progression. They note a number of interesting causal etiologic models that may possibly be observed over the life course and examine the implications of each in relation to social-economic position and its effects on cardiovascular diseases and cognitive health in later life. They focus their conclusions on the implications of a number of potential policy choices that can be made in late life and how these may influence the types and timing of interventions that may be most effective in the elimination of health disparities.

The third and final chapter in this section by Whitfield, Bromell, Bennett, and Edwards focuses on the "biobehavioral" research approach as one that characterizes a broad area of research encompassing biological underpinnings of behavior or behavioral implications for biological phenotypes. They note that this broad conceptualization reflects the interest in providing a deeper level of understanding for how a complex phenomenon like aging can be understood in an ecologically valid, multivariate fashion. They describe how the biobehavioral approach is now a widely accepted framework that can be used to improve understanding of age related changes in health and disease in late life. In the first part of the chapter they provide an introduction to the concept, as well as some examples of this new and unique field of inquiry in the areas of stress, cognition, and physical health morbidities. They conclude by noting that the biobehavioral definition and approach describes a useful interdisciplinary approach to understanding health and particularly health disparities. Finally, they view the biobehavioral approach as an integral framework for educating future gerontologists in understanding the inter- and multidisciplinary foci needed to comprehend the complexity of age-related

changes, especially health disparities among socioeconomic and race–ethnic groups in later life.

Section II: Examples of Specific Health Morbidity Inequalities in Late Life

Building on the theoretical, epidemiological, and biobehavioral perspectives in the first section, the chapters in the second section present specific examples of late-life disparities and their potential sources. In chapter 4 Mezuk focuses on cardiometabolic disorders that include cardiovascular disease (CVD), for example, heart disease, such as atherosclerosis or hardening of the arteries, which can lead to angina pectoris or a heart attack and stroke, and Type 2 diabetes mellitus, an endocrine disorder. These cardiometabolic problems have multifactorial, but related, causal origins. Mezuk notes that many potentially modifiable individual-level risk factors for these conditions have been identified, including *health behaviors* such as smoking and physical inactivity and *psychosocial factors* such as mental health and social support. However, substantial group inequalities in the burden of cardiometabolic conditions exist. In order to understand determinants of health for the population, it is important to identify risk factors at multiple spheres beyond the individual, including at the level of the workplace, the neighborhood, and political structures.

Picking up the theme of the chapters in the first section, Mezuk presents data that reveal at younger ages little difference between mortality rates comparing across African American and non-Hispanic White male and females. However, across age these mortality rates begin to diverge, and by age 65 the annual mortality rate from cardiometabolic conditions for White males is nearly 60% lower than that for African American males (866.5 deaths per 100,000 versus 1485.6 deaths per 100,000). Mezuk points out that these mortality estimates reflect a combination of disease incidence, disease severity, and access to treatment, all affected by differences in socioeconomic position and race–ethnicity. These population trends demonstrate the need for a life-course approach to understanding disparities in cardiometabolic disorders, because while these inequalities generally emerge at younger ages, they clearly accelerate over the life course. Similar to Whitfield and colleagues in Chapter 3, Mezuk notes that the proximal and distal sources of these inequalities are not well understood. Mezuk reviews relevant literature on the proximal causes of these conditions and discuss how these and other contextual factors influence the observed life-course inequalities in cardiovascular disease and diabetes over the life span. Finally, Mezuk focuses on how observed differences by chronological time, age, gender, race–ethnicity, and geography can further the understanding of the emergence of these disparities over the life

course, and similar to the general points made by Glynmour, Ertel, and Berkman (chapter 2), inform interventions that are relevant and responsive to the needs of particular subgroups facing unequal health burdens.

Chapter 5 by Taylor focuses on hypertension, one of the cardiometabolic chronic conditions defined by Mezuk in chapter 4. Taylor notes that it is a chronic health condition that has become a common diagnosis among Americans, particularly African Americans. Hypertension is defined as repeated blood pressure readings that are consistently at or above 140/90 for adults and at the 95th percentile or greater for age and height among children. Taylor reviews research showing that the development of hypertension is influenced by both genetics and lifestyle behaviors. Although several race–ethnic and lower socioeconomic groups are at increased risk for hypertension, African Americans have been shown to have earlier onset and greater severity of hypertension. Thus, Taylor's chapter focuses mainly on this particular group. She notes that lifestyle behaviors that include lack of physical activity, high fat and sodium food consumption, and resulting obesity all are negative habits that develop early in childhood and have long-lasting effects on health throughout the life span. The diagnosis of hypertension and obesity has been steadily rising among both African American adults and children. Using the biobehavioral framework outlined by Whitfield and colleagues in chapter 3, Taylor suggests that hypertension is a multifaceted chronic disease that is approaching epidemic proportions among African Americans. The bulk of Taylor's chapter discusses the prevalence of hypertension and obesity, environmental lifestyle risks, genetic risks, gene-environment interactions, related comorbidities, and research trends for reducing this disparate condition across the life span. Taylor concludes, consistent with the recommendations by Glymour, Ertel, and Berkman in chapter 2 that research that begins with children are the keys to developing interventions for early detection and intervention for reducing hypertension inequalities among African Americans in late life. Positive lifestyle behaviors instilled early in childhood can help to prevent obesity and resulting hypertension over the life course. Although the causal factors for hypertension may be particularly affected by epigenetic influences, Taylor notes that the genetic heredity of disease is nonmodifiable and that environmental lifestyle behaviors (diet, physical activity, etc.) can influence phenotypic expression of disease and severity of chronic health outcomes. Similar to Whitfield et al. in chapter 3, Taylor concludes that more research on gene-environment interactions for hypertension is needed to provide improved recommendations for disease prevention and management over the life span. Similar to Glymour, Ertel, and Berkman (chapter 2), she notes that gene-environment research on hypertension should include

large longitudinal studies of healthy children of hypertensive parents in order to better understand these interactions.

Section III: Disparities in Cognition and Health in Late Life

Section III focuses on cognition and dementia differences among groups in late life as well as the life-course and life-span influences on both cognition and the influence of cognition and dementia on other chronic health conditions. In chapter 6 Lichtenberg continues the theme in the Taylor chapter and focuses on African Americans, while noting as do all the chapters in the first and second sections preceding, that race–ethnicity and socioeconomic status group differences encompass a wide-ranging number of different groups in the United States. As did Mezuk (chapter 4), Lichtenberg notes that data have been more plentiful on disparities due to race in this area and he draws on these data in his illustrative presentation. Lichtenberg notes that African Americans comprise an important group in the aging community. Not only does this group experience higher rates of dementia than other race–ethnic groups in late life, but they also experience a greater number of vascular risk factors known to enhance the behavioral expression of dementia, such as diabetes, high cholesterol, and hypertension (Mezuk, chapter 4). In the not too distant past, African Americans were legally barred from educational opportunities, and large numbers of African Americans are currently receiving segregated and inferior education. African Americans historically have also had less access to quality medicine. Even today, studies indicate that African Americans may not always be treated as aggressively for chronic conditions, and may not be diagnosed with a primary progressive dementia as early as non-African Americans. As the Baby Boomers age and the older population becomes increasingly ethnically and racially diverse, understanding factors affecting diagnosis and treatment of this group of older adults becomes essential. Lichtenberg, using African Americans as an example, focuses on understanding the factors contributing to increased rates of dementia in older African Americans as well as best practices in clinical assessment of cognition and cognitive decline over the life span. As with all the preceding chapters, the evidence reviewed by Lichtenberg in chapter 6, clearly implicates the important role of early experience on late-life outcomes of dementia among older samples of African Americans who were relatively deprived at earlier periods of their individual and group life course. Building on the biobehavioral and life-course epidemiological frameworks in the first section, Lichtenberg notes that the optimal point of intervention may be early in the life span in order to

aggressively address the observed age-related declines and inequalities found among different socioeconomic and race–ethnic groups in late life.

Building on the observed inequalities in dementia in chapter 6, Ryan and Smith in chapter 7 examine the consequences of these declines and disparities on chronic health conditions. Their chapter focuses on the significant contribution to health inequalities over the life course of cognitive ability and functioning. They consider research on inequalities in longevity as one indicator of the outcome of health inequalities among subgroups within a population. Their chapter also points to the significant role of cognition in healthy lifestyles and health inequalities noting the positive moderate associations between cognitive functioning and socioeconomic status. They highlight the possible important role of selection since often measures of individual differences in general cognitive ability are used to screen for entry into educational institutions and occupations early in life, but that income and wealth in late adulthood are less highly correlated with cognitive ability. In fact, Ryan and Smith report that within homogenous samples of high cognitive functioning persons there are significant differences in personal wealth, income, and occupational achievement. In chapter 7 Ryan and Smith review research showing a strong positive relationship between cognition and longevity. Building on Lichtenberg in chapter 6, they note that the significant long-term influence of cognitive functioning has been found in studies that have linked childhood mental performance with later survival in old age. Similar to Taylor (chapter 5) they note potential genetic explanations for the association between cognition and health with later survival. Using illustrative data from the Health and Retirement Study (HRS) they discuss how these various approaches and findings merge.

Finally, the second part of chapter 7 reviews the theories proposed to explain the association between cognition and longevity. As with Glymour, Ertel, and Berkman (chapter 2) and Mezuk (chapter 4), explanations range from the contribution of cognition to disparities in environmental experiences, health behaviors, lifestyle preferences, and material resources, underlying genetic influences associated with cognition and health, and similar to Whitfield et al. (chapter 3) and Taylor (chapter 5) complicated genetic-environmental dynamics over the life course. Ryan and Smith note the complicated causal models in health disparities that might fit the observed life-course data (Glymour, Ertel, & Berkman, chapter 2) and conclude, consistent with the biobehavioral framework proposed by Whitfield et al. (chapter 3), that the effect of inherited physiological and intelligence factors on later longevity may help to explain the aggregate social group inequalities over the life course. They note that inequalities in material, psychological, and social resources associated with

socioeconomic (and race–ethnic) disparities may interact with genetic predispositions and epigenetic gene expressions to create individual differences over the life span.

Section IV: Functional Limitations and Responses to Stress in Late Life

Section IV builds on the theoretical frameworks and epidemiological data that demonstrate inequalities in chronic conditions to discuss functional limitations, stress, and coping differences among groups in late life. In chapter 8, Kershaw and colleagues explore potential physiological mechanisms associated with positive coping behaviors and strategies, including religiosity–spirituality, mastery, physical activity, meditation, and social support. Research articles that examine the relationship between physiological responses to chronic stress, assessed primarily by differences in the diurnal cortisol pattern in naturalistic settings and positive coping behaviors are considered in this chapter. In reviewing the available literature, the authors conclude that across different studies the findings are simple, but there is some evidence suggesting beneficial physiological effects of positive coping behaviors. The authors suggest that more research is needed to better understand whether or not positive coping behaviors can work effectively to insulate, buffer, or moderate the effects of chronic stressors on disease outcomes and chronic health conditions, especially through the HPA axis and related hormonal pathways. The review in chapter 8 suggests that intrinsic religious faith may help regulate chronic stress via the HPA axis, but there is insufficient evidence at this time to conclude that mastery is an effective coping strategy, and while social support shows some promising results, the findings are not definitive. Intense physical activity and long-term transcendental meditation both show promise. In general, Kershaw and colleagues note that understanding the effectiveness of potential stress reducing strategies have important implications for disentangling the epidemiology of chronic diseases that are more prevalent among low SES and certain race and ethnic groups (e.g., Glymour, Ertel, & Berkman, chapter 2 and Whitfield et al., chapter 3). Kershaw et al. review a variety of coping strategies that may help reduce the body's response to chronic stressors. Some of these are linked to having negative health consequences and others linked to more positive health outcomes. They conclude that more research needs to be conducted to establish positive coping behaviors as viable choices for alleviating the physiological response to stressful events among different social and economic groups,

In chapter 9 McIlvane builds on the general coping review of Kershaw et al. in chapter 8 to examine a specific, though multifactorial, chronic condition,

arthritis. She suggests that there is a dearth of research on the reasons for disparities among African Americans and Latinos with arthritis. She notes that this is unexpected given the prevalence of arthritis over the life span and its demonstrated influence on functioning and quality of life. As do all other chapters in the volume, McIlvane implicates a combination of factors that may contribute to disparities in arthritis, including socioeconomic status, access to health care, chronic stress, illness perceptions, and mistrust of the health care system. Just as we noted earlier, the aging of the baby boomers and the increasing racial–ethnic diversity of our older population, will increase the importance of arthritis as a significant public health issue. Unfortunately, her review reveals scant research on arthritis among African Americans and even less among Latinos.

McIlvane argues the importance of studying arthritis inequalities based upon racial–ethnic disparities in arthritis prevalence, symptoms, and treatment. She also considers the little understood potential contributors to arthritis health disparities including barriers to access to care, mistrust of medical system, and differences in illness perceptions. Finally, she considers the role of protective factors, notably religion–faith, coping, and family support that may be beneficial particularly to race–ethnic minority group members. McIlvane concludes that there is consistent evidence for racial–ethnic differences in arthritis symptoms and under utilization of effective treatments. Health disparities and especially their correlates, however, have received little attention thus far in arthritis research. She suggests that in order to understand the observed inequalities in late life more research needs to focus on the unique contexts and cultural traditions–values of racial–ethnic minorities. This will enable a fuller understanding of their experiences living with arthritis and other stressors (e.g., discrimination, financial strain) as well as their experiences interacting with the health care system.

Section V: Contextual Influences on Health Disparities in Late Life

It is clear that a major objective of this volume is to illuminate that context is important in understanding observed differences in the incidence and prevalence of health differences among social and economic groups in late life. Context is considered broadly and includes genetic and epigenetic influences, early life experiences, generational membership and family influences, ones race–ethnic background and cohort and period influences, as well as the individual and group life-course and life-span trajectories that occur as individuals and cohorts age.

The chapters in the fifth section address the role of specific contextual influences using illustrations of individual differences (e.g., Ryan & Smith, chapter 7), work, and national origin on disparities in late life. Gerstorf and colleagues in chapter 10 begin at the lowest context and focus on late life well-being and death to illustrate the ways that between-person's disparities contribute to observed inequalities. More broadly, Gerstorf and colleagues are interested in how this line of work informs and contributes to refinement in extant theories of well-being in late life, and specifically conceptual notions that link mortality as a major force underlying developmental change in the last years of life. They argue succinctly that the changes in well-being that occur late in life provide a helpful lens for exploring between-person disparities and the correlates of these disparities. Their review of the empirical evidence suggests that end-of-life declines in well-being and psychological health may be a normative experience, noting that these declines are more a function of closeness to death than age itself. At the same time, not all individuals experience their last years alike, and while end-of-life decline may be normative, it should not be considered inevitable. Similar to Whitfield et al. (chapter 3) they suggest that there is tremendous variability in late-life patterns of change as people age. For many people well-being declines sharply near death, whereas others maintain their well-being into their last years. Gerstorf et al. also examine these between-person disparities in late life as well-being outcomes change, noting some of the key factors that contribute to inequalities. These factors encompass key predictors of mortality and well-being, including age at death, gender, education, and disability. Finally, following the conclusions of authors in prior chapters, they suggest that these factors may not only reside at the individual level, but may also be found at the community and society. They suggest some of the possible mechanisms linking macrocontextual factors to individual outcomes and inequalities in individual well-being changes in late life (see particularly Glymour, Ertel, & Berkman, chapter 2; Whitfield et al., chapter 3; and Ryan & Smith, chapter 7).

In Chapter 11 Brown takes a conceptual step back to consider work and the workplace as an important source of material and social disparities that contribute to well-being postretirement in later life. Her chapter focuses on the complex relationship between work, retirement, race, and health disparities among aging African Americans to illustrate the role of race–ethnicity more broadly, but also more sharply in delineating how early life-course work opportunities and experiences influence the nature of changed roles such as retirement (see Lichtenberg, chapter 6). This chapter utilizes a life-course perspective to consider how preretirement experiences, including health and job statuses, affect transitioning out of the labor force for these older Americans.

She demonstrates how life-course theorizing and research (see Fuller-Iglesias et al., chapter 1) facilitates the examination of continuity between pre- and postretirement experiences. Brown concludes that understanding the health consequences of labor entry, experiences, and exits is important in guiding policy to assist the growing aging population. She suggests that illness and disease limits productivity of adults during their most productive working years, affecting the entire society. Excessive health problems among any and all groups are associated with increases in the use of social services, decreased accumulation of postretirement benefits, and places undue strain on the health care and retirement systems.

The final chapter in this section uses control beliefs and disabilities as an example to illustrate how a cross-national comparison can illuminate the nature of inequalities in the United States. Clarke and Smith focus on differences in the United Kingdom and the United States to make this point. They note that a major reason for comparing health across nations is an attempt to understand how differences in macrosocial and economic contexts shape individual health and well-being. Much of this research has been undertaken at a population level to examine the underlying association between social inequality and health outcomes such as average life expectancy and the prevalence of preventative illness (e.g., cardiovascular disease, disability). Attention has been given to country differences in terms of the absolute levels of social inequality (especially the extent of poverty) and the magnitude of relative inequality (e.g., the differential gap between rich and poor). There are also country differences linked to gross national product and the allocation of finances to health services. Beyond economic and political mechanisms, contemporary researchers also consider the roles of cultural and psychosocial factors at community and individual levels.

Clarke and Smith note that cultural differences in beliefs, health-related behaviors, and social cohesion have been highlighted as important factors involved in health disparities, both within and between countries, and as modifiers of the association between social inequality and health outcomes. They also focus on cross-national differences in belief systems, specifically beliefs about personal control found between the older populations in the United Kingdom and the United States and the moderating role of control beliefs in the social inequality–health outcome association. They use national data from the United States and England to illustrate the socioeconomic gradient in health across the two countries, and show how it varies according to sense of control. Clarke and Smith conclude that cross-national differences in these relationships are partly a function of differences in the lifetime social construction of a sense of control. They note that cultural influences across

the life course play an important role in shaping late-life health inequalities in different sociopolitical contexts.

Section VI: Approaches to Public Policies to Address Health Disparities in Late Life

We end this volume on a public policy theme. Just as Clarke and Smith (chapter 12) highlight the importance of macropolitical, economic, and social factors interacting with group and individual differences, this final chapter in the volume focuses on public policy proscriptions that might help to address observed inequalities in health in late life. Miles begins from a perspective that assumes the multideterminant view of health and health disparities illustrated repeatedly in the prior five sections of the book. Miles notes that public health data consistently show subpopulations that experience higher than average rates of disease and mortality. These groups include racial and ethnic minorities, persons with less than a high school education, persons clustered in specific geographic regions, and members of households that are supported by low wage jobs. She lists several factors that may contribute to these rates, but focuses specifically on barriers in access to health care, a major incentive driving the legislative efforts toward health care reform now occurring in the 111th Congress.

A central theme of Miles's chapter is that health disparities researchers have focused almost exclusively on studies that document differences (witness studies), illuminate possible correlates and causal factors (etiology studies), or measure the effectiveness of a particular intervention on individuals (treatment effectiveness studies). She believes that a focus on a perspective that includes both the individual and the system of health care can accomplish the objectives of these three types of studies (i.e., witness, etiology, and treatment effectiveness, respectively) while adding an assessment of the impact of systemic factors. In order to illustrate this change in focus Miles considers the problem of health disparities in the Medicare population noting that current policy research is focused exclusively on Medicare spending and system financing. She suggests that dual eligible beneficiaries—persons simultaneously enrolled in Medicare and Medicaid—form a cohort of the nation's most vulnerable older adults and asks the question of what would the health disparities research framework represent for older adults who are dual eligible? Using this model of dual eligibility Miles argues in the reminder of the chapter for a public health research agenda on disparities and inequalities that incorporates policies directed toward the health care delivery infrastructure. She believes that current health care reform efforts present an opportunity for

researchers to prospectively study health disparities and related policies and to work collaboratively with federal, state, and local governments to make changes at the intersections of health care reform and legislation to address more comprehensively the elimination of health inequalities.

CONCLUSION

Collectively, the chapters in this volume illustrate the complexities of studying health inequalities in later life. Though selective, since no one volume can address the entire landscape of this difficult topic, they provide a broad sampling of recent thinking and research on this important set of issues. Our authors speak in many voices. Their range of focus moves from genetics to geography, from one minority group to several. Reflecting the current acceptable practice in scientific writing (see the American Psychological Association Publication Manual), our authors use the terms African American and Black interchangeably. We believe these multiple voices enrich the dialogue and encourage discussion within, between, and across topics. The chapters in the first section address the theoretical perspectives on a life-course/life-span perspective (Fuller-Iglesias et al.), as well as two meta approaches to empirical research that grow from this perspective, life-course epidemiology (Glymour, Ertel, & Berkman) and biobehavioral (Whitfield et al.) frameworks. These perspectives emphasize the intersection among genetic, environmental, and social group influences from conception to death that produce individual trajectories and risks for morbidity and mortality at different points in the individual life course. The chapters in the second section represent examples of specific diseases, cardiometabolic illnesses (Mezuk) and hypertension (Taylor) for which research has implicated both a biobehavioral genetic and epidemiological perspectives on etiology and process over the life course.

The chapters in the third section represent examples of the important role of cognition in health disparities. They illustrate both the etiology and correlates of poor cognition in different race–ethnic groups (Lichtenberg), and the manner in which low cognition may be related to economic and social group differentials in health and longevity (Ryan & Smith). The first chapter in this section (Kershaw et al.) picks up on this theme and addresses how positive coping behaviors may be related to the biobehavioral indicators described in chapter 3, representing disparities in underlying biological responses to risk exposure, disease onset and progression, and chronic health disorders. The second chapter (McIlvane) in the fourth section addresses functional limitations due to chronic disorders, using arthritis as an example. Arthritis,

which afflicts a large portion of the population in late life, and is thought to be much more prevalent among African Americans and Latinos. The author (McIlvane) addresses ways of coping with arthritis that may be culturally and age graded.

The chapters in the fifth section address the range from individual to large organizational and macrocontextual factors that may influence observed disparities over the life course, eventuating in late-life inequalities. They illustrate influences of individual differences (Gerstorf et al.), to the work place (Brown), to differences that may represent national cultural and political system differences (Clarke & Smith). The final section concludes with a focus on the need to consider the ways in which organizational structures, and especially the nature and organization of health care delivery systems, may intersect with the genetic, individual, social, economic, and group social influences on life-course development risks for poor health in late life. Miles suggests that the current emphasis on health care reform may provide a unique point in history to bring about systematic changes. Such changes may address deficiencies in systems of health care that contribute to observed economic and social disparities over the life course that result in unequal prevalence rates of chronic disease and early mortality.

In this volume we have focused on the age, gender, socioeconomic, cultural, and race–ethnic graded influences on life-course development that culminate in unequal burdens of health morbidity and mortality for some groups late in life when compared to others. This examination also reveals that studying the stark disparities and health inequalities among groups illustrates the appropriateness and utility of the basic life-course framework, encompassing the intersection of genetic, socialization, cumulative life experiences, and risk exposures on the fundamental nature of life-span development.

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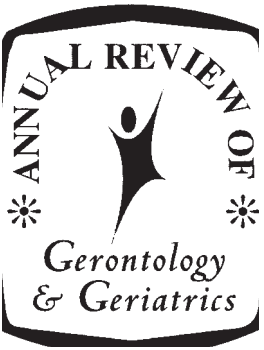

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SECTION I

FRAMEWORKS FOR UNDERSTANDING LATE-LIFE HEALTH DISPARITIES

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CHAPTER 1

Theories of Aging From a Life-Course and Life-Span Perspective

An Overview

Heather Fuller-Iglesias, Jacqui Smith, and Toni C. Antonucci

Life-course and life-span theoretical perspectives have important implications for gerontological studies. Both frameworks address patterns of change over time that inform our understanding of the process of aging. In considering health inequalities in later life, both life-course and life-span perspectives play an integral role in determining micro- and macrolevel influences on health and well-being in late life. In this chapter we consider the theoretical underpinnings of a life-long approach to health disparities, major themes in the life-course and life-span perspectives on aging, and cumulative inequalities and disparities on well-being and health.

Theoretically, there is now a broad consensus across many disciplines in the social (sociology, psychology, philosophy, economics, and demography) and medical sciences (biology, genetics, medicine, public health, and epidemiology) that in order to understand the phenomena of old age and aging processes it is important to take a life-span developmental and life-course perspective. Economists sometimes call this a life-cycle perspective. For some research disciplines, this idea has a long history, in others the history spans several decades, and for a few it remains counterintuitive. Historically, phases (e.g., childhood and old age) and experiences (e.g.,

education and health) of the life course were considered somewhat independently, because few believed that the normative events of early life had consequences for old age.

There are many similarities between the two perspectives, the most obvious one being that both advocate taking a long-term, multilevel, contextual, and dynamic view of aging. *Life-span* theories draw attention to the length of the life of an *individual* and to the idea that processes and trajectories of development and aging are lifelong (Lerner, 2002). *Life-course* theories, in contrast, differentiate between subgroups in society and focus on the *social pathways* that define the sequence of events, transitions, roles, and experiences in the lives of individuals (e.g., Alwin & Wray, 2005; Settersen, 2007). While life-span researchers are interested primarily in understanding microlevel (endogenous) processes within the aging individual (e.g., the aging brain and mind), life-course researchers typically analyze the macrolevel (exogenous) processes that characterize the influence of groups, organizations, and institutions on the individuals within them.

Despite these differences in topical emphases and levels of measurement, in general, there is much concordance between the life-span and life-course perspectives. Together these perspectives present a comprehensive theoretical framework of human development over time. Recent advances in both suggest that there is considerable value in integrating the premises and conceptualizations of both theories and expanding them to form a truly multidisciplinary framework.

The life-span and life-course perspectives each, by moving beyond the study of development as isolated and age specific, recognize that all age periods (including old age) are dynamic and cumulative. To understand the human experience it must be viewed across time. Research should examine both micro- and macro-influences, consider gains as well as losses, and identify factors of risk and resilience. These frameworks can be seen as integral not to preventing aging, but rather to promoting optimal development at all ages, including old age. In sum, because aging is a lifelong process and old age is the result of a lifetime of experiences, it is essential to study old age within a life-span and life-course perspective. Increasingly researchers in many of the disciplines involved in gerontology are adopting this general position. We believe that only by adopting. This perspective can we fully understand the health inequalities that are evident in later life.

The next section of this chapter outlines important major themes from life-span and life-course perspectives of aging and describes how they are integral to understanding the aging society.

MAJOR THEMES IN THE LIFE-COURSE AND LIFE-SPAN PERSPECTIVES ON AGING

It can be argued that the primary distinction between life-span and life-course theories is the disciplinary lens that logically guides a divergence; on the weight placed on comprehending individual versus social contextual processes. The central themes that are listed in Table 1.1 highlight a few of these disciplinary differences. Presenting such a tabular distinction harbors the dangerous connotation that life-course and life-span researchers occupy opposing camps, a viewpoint that we especially do not intend to convey. Instead, our intention in this section is to provide a didactic overview of a select illustrative set of salient themes that guide the choice of specific topics and methods among researchers in different fields. As illustrative examples, they can not reflect the eclectic, conceptual state of contemporary research on aging and the many models of biopsychosocial co-construction of aging. Life-span researchers, for example, increasingly utilize study designs that examine the role of social context, subgroup variation, and cohort in the examination of psychological outcomes (Heckhausen, 1999). Similarly, life-course researchers have moved toward the incorporation and examination of individual-level processes in their models and analyses (e.g., Berkman, Glass, Brissette, & Seeman, 2000; Mayer, 2003). Both perspectives are fundamentally important for gaining a fuller understanding of health inequalities in later life.

THEMES IN LIFE-COURSE RESEARCH

Life-course theory takes a macrolevel, group, or societal view of developmental influences. It is a theoretical framework for addressing the temporal sequence of age-graded roles and expectations that are embedded in social institutions

TABLE 1.1
A Selection of Major Themes to Illustrate the Life-Course and Life-Span Perspectives

Major Themes	
Life Course Perspective	Life Span Perspective
Age, cohort, & historical effects	Individual differences
Accumulation of (in)equalities	Adaptivity and plasticity
Life Course & Life Span Perspectives	
Linked lives	
Differential trajectories and pathways of aging	

and history (Elder, Johnson, & Crosnoe, 2003; Mortimer & Shanahan, 2003). The life-course perspective makes a unique contribution in understanding, at the population or group level, the roles of *age stratification, cohort and historical period effects,* and the *accumulation of (in)equalities over time*.

Age, Cohort, and Historical Effects

Normative age structuring and age stratification are ideas in the forefront of life-course theories and research. In general, these concepts describe the fact that most societies use chronological age formally and informally to structure the experiences, roles, and statuses of individuals (Dannefer & Uhlenberg, 1999; Riley, 1987). In a formal way, social institutions define, segment, and construct individual lives in the interconnected domains of education, family, and work over the life course (Mayer & Tuma, 1990). Because some social institutions (e.g., education, work, retirement) are typically created for specific age groups, chronological age also stratifies the ways that opportunities are, or sometimes are not, made available to individuals in a population. At an informal level, individuals and subgroups create beliefs and attitudes about age categories (e.g., young, middle age, old), aging, and the kinds of behavior appropriate to individuals of different ages.

Kohli (1986) proposed that the life course itself has become increasingly institutionalized. He considers the normative age-graded structural patterning of education, work, and retirement as evidence that the life course has become a modern social institution. This institutionalization of the life course suggests that there is a standard life pattern that is guided by rules about how individuals structure their lives that are upheld by an infrastructure of social and legal norms and organizational systems (i.e., education: Kohli, 2007; Kohli & Meyer, 1986). Some are more advantaged by the institutionalization of these life-course patterns while others are not. The three central features of the institutionalization of the life course are *chronologization* (the saliency of age and time), *institutionalization* (ways in which the life course is structured by organizations, institutions, and the state), and *standardization* (the resulting normativity of life-course patterns). In effect, the societal context influences and implements a standard life course. Each of these features contributes to inequalities and different pathways across the life course.

The principle of *time and place* reflects the notion that the life course of individuals is embedded and shaped by the historical times and places they experience over their lifetime (Elder et al., 2003). The primary emphasis of the principle of time and place is the importance of *historical effects* on human development. Major historical events have the power to not only transform societies, but also have great impact on development at the generational and individual levels. A prime example of how time and place have significance

is the effect of the Great Depression (Elder, 1974). Individuals living in the United States were all affected by this major historical stressor. However, a *cohort effect*—differentiation of the lives of different birth cohorts as a result of historical change—also occurred as a function of the differential timing of the Great Depression in the lives of children, adolescents, and young adults; some cohorts were more vulnerable than others. Similarly, African Americans were differentially affected by the Civil Rights Movement depending upon their age and cohort. Young people had new opportunities opened to them such as access to education, while old people were not able to take advantage of this new access (Jackson, 1993). The preceding examples illustrate how time and place (i.e., birth year and historical context) can result in differing outcomes between cohorts. A *period effect* occurs when a historical event has a similar effect on multiple birth cohorts, such as 9/11 and Hurricane Katrina.

In contrast to the principle of time and place that focuses on historical influences on development and aging in different generations, the principle of timing in lives focuses on time in relation to individual trajectories. This principle highlights the fact that the effects of life transitions, events, and behavioral patterns on human development vary according to the timing in a person's life (Elder & Shanahan, 2005). In effect, whether the timing of a transition or life event is normative or nonnormative directly affects the individual's developmental outcomes. Elder describes four concepts that are related to timing in lives (Elder et al., 2003): (1) Social pathways that refer to patterns of education, career, family, and living arrangements that are socially ascribed and followed by individuals and groups within a society; (2) Trajectories that refer specifically to the sequences of roles and experiences that make up an individual's development throughout the life course; (3) Exit transitions, changes in state or role, which refers specifically to times in the developmental trajectory when the individual makes a normative change; and, (4) Transitions that generally reflect on time role changes, such as becoming a parent during the childbearing years or becoming a widow in old age. These turning points can involve substantial changes in the direction of an individual's life and generally reflect nonnormative or unexpected life events. An example of such a turning point is deployment to a war combat zone or becoming a widow in early adulthood. Each of these concepts represents the temporal nature of the life course and points to the importance of considering the differential impact of inequalities.

Accumulation of Equalities and Inequalities

Status disparities are fundamental aspects of virtually all societies and social groups. Status is structured and ascribed on many dimensions, for example, by wealth, knowledge, competence, physical strength, health, group membership

(e.g., ethnic group, immigrant, sex, age), and perceived contribution to a group. A person's position in the social stratification system is the most consistent predictor of his or her behavior, attitudes, and life chances. Some authors describe this as an individual's life course capital (e.g., O'Rand, 2003). One consequence of the social and historical structuring of the life course is that it is associated with a hierarchy of opportunities and exposure to constraints and risks for individuals and groups within a population. At a single point in time, this contributes to observations of heterogeneity in resources, such as wealth, education, and health. Some individuals have greater access to resources and more opportunities than others. Because of their birth cohort and position in the life course, some individuals benefit from technological advances or periods of economic growth whereas others do not. Furthermore, personal histories of differential opportunity and exposure are transferred from one generation to the next in the contexts of families and social groups.

Additionally, disadvantages may accrue over the life course and have a cumulative effect on outcomes late in the life, such as health and longevity (Glymour, Ertel, & Berkman, 2009). Social disadvantage is likely to be related to stress exposure. Cumulative disadvantage is the term used to refer to disadvantages that sum over the life course and predict worse health in old age. Much of the research on cumulative disadvantage focuses on socioeconomic status, race, ethnicity, and gender (House, Latz, & Herd, 2005; Marmot, 2006; O'Rand, 2003; Smith & Kington, 1997). Older adults with lower education levels experience worse health sooner than their higher SES counterparts (Marmot, 2006; Willson, Shuey, & Elder, 2007). Moreover, Shuey and Willson (2008) found a cumulative disadvantage for Blacks in that they did not experience the same health benefits from advanced education that Whites did. House and colleagues (2005) suggest that understanding social disparities in health is a key problem for life-course researchers and is essential to understanding health and illness in old age.

Race and ethnicity are important group level factors to address in aging research as they encompass biological, environmental, historical, cultural, and social influences. Within the United States, ethnic and racial minorities show relatively poorer status in old age as compared to their majority White counterparts (Jackson, Brown, & Antonucci, 2004). Of note is the fact that based upon current estimates of mortality and life expectancies, older minority populations are growing rapidly and will continue to do so (Jackson & Sellers, 2001). Some data indicate a crossover effect in that some minority populations of advanced ages, for example Blacks (e.g., Gibson & Jackson, 1992), may be more robust in comparison to Whites, perhaps reflecting different aging processes and selection over time for hardier individuals (Manton, Patrick, &

Johnson, 1987). However, at every point earlier in the individual life span most members of racial and ethnic minority groups are at greater mortality and morbidity risk than Whites (Jackson & Sellers, 2001).

A life-course perspective illuminates the fact that current and aging cohorts of underrepresented race–ethnic minorities have been exposed to conditions that will profoundly influence their social, psychological, and health statuses from childhood to adulthood and older ages in the years and decades to come (Baltes, 1997; Barresi, 1987). Indeed, historically gerontologists have described the double (race, age) and triple (race, age, and gender) jeopardy experienced by some groups.

THEMES IN LIFE-SPAN RESEARCH

The life-span perspective complements the life-course perspective with a greater focus at the individual level. The life-span approach as defined by Baltes (1997; Baltes & Goulet, 1970; Baltes, Reese, & Nesselroade, 1988) consists of a family of proposals that attempts to describe the patterning of growth, stability, and change in behavior throughout the entire life course. The emphasis is on understanding the general principles of development and change at all ages, interindividual differences in change trajectories, and the modifiability (plasticity) of development and aging. This three-component emphasis on questions about development and aging (communalities, interindividual differences, and intraindividual plasticity) is a core feature that distinguishes the life-span approach.

Because of the complexity and plasticity of the conditions shaping the course of human development, the general approach of life-span theorists has always been (a) to highlight the *pluralistic* and *dynamic* nature of contextual influences on individual change, and (b) to consider individual development itself as a changing phenomenon. With regard to the first issue, life-span theorists are in agreement with life-course theorists. Thinking about the changing individual interacting with a changing world is more unique to the life-span perspective.

Individual Differences in Functioning

Many life-span researchers emerge from the research tradition of exploring individual differences in social relationships (e.g., social support), cognitive ability (e.g., intelligence), and disposition (e.g., personality), and the intricate ways that these individual characteristics shape the life-span development of the person. Further, they highlight the importance of examining individual differences in intraindividual change. The focus on individual differences

serves to highlight the heterogeneity of functioning within age and social groups. Indeed, in some instances there is greater variation within groups than between groups. Whereas much research on health inequalities focuses on group differences in pathological versus nonpathological groups, a life span perspective points to the importance of individual differences within each of these categories. We often neglect to examine the range of normal healthy functioning or the potential to optimize health. In addition, within pathological functioning there are also well-known individual differences in the range and progression of disease (e.g., in dementia, diabetes, and cardiovascular disease).

Adaptivity, Plasticity and Resilience

Life-span development is adaptation by definition. Adaptation includes an evolving balance of *gains and losses* across the life course. An important adaptive task across the life span is the allocation of resources to address functions of growth, maintenance, and regulation of loss (Baltes, Lindenberger, & Staudinger, 2005). This dynamic between gains and losses is moderated by the interaction between biological and cultural resources and strategies of self-regulation (e.g., life management strategies of selection, optimization, and compensation). Baltes has noted that while the role of culture increases with age, current cultural infrastructure lacks the appropriate support mechanisms to promote successful development into late life. As individuals age, they move from using their capacities for growth to using their capacities for maintaining the skills that they have acquired, as well as the prevention of loss of those skills or specific efforts to recover things that have been lost. However, lifelong development encompasses not only advances in adaptivity but also presents challenges and even losses of adaptive capacity across the life span. Individual differences influence the capacity to adapt to their life circumstances. In addition, people in different social groups may be exposed to and be required to adapt to different life circumstances.

Plasticity refers to within-person variability (Gollin, 1981; Lerner, 1984; 2002). Such variability is an indication of the individual's potential for different levels of functioning or development. Physiological systems that adjust our heart rate depending on energy expenditure and temperature sensitivity in relation to environment changes exemplify this notion (for other examples see Bornstein & Suess, 1998). In some psychological domains (e.g., self and personality) the capacity to adapt to a changing environment implies resilience, another exemplification of intraindividual variability (flexibility; e.g., Eizenman, Nesselroade, Featherman, & Rowe, 1997; Nesselroade, 1991). In other domains (e.g., cognitive functioning), interest lies in determining an

individual's potential (or reserve capacity) to learn new things or increase the level or speed of performance. The concept of plasticity (and possible changes in the range of plasticity across the life span) underlies questions about modification principles associated with optimizing behavior and development. Rowe and Kahn (1987) have argued that successful aging "is a generalized capacity to respond with resilience to challenges from one's mind, body, and environment." Because old age is a stage of increased psychosocial stressors, such as the death of loved ones, the onset of physical health declines, and increased chronic and disabling diseases, it may be the case that certain forms of adversity become more normative in old age. Old age presents more challenges than other stages of the life span; plasticity and resilience in late life addresses the ability to compensate for age-related losses. It is likely that plasticity and resilience are influenced by and influence accumulated life inequalities.

LIFE-COURSE AND LIFE-SPAN THEMES

Though life-course and life-span research have developed across distinct trajectories, there are research topics in which these two theoretical frameworks have overlapped. Both life-course and life-span perspectives make important contributions to the understanding of interdependencies or links between the lives of individuals within society and differential pathways and trajectories of aging. *Linked Lives* and *differential pathways and trajectories of aging* are examined within the context of individual and group differences.

Linked Lives

The principle of linked lives refers to the interconnectedness of individuals as they develop and adapt. Human lives are lived interdependently and sociohistorical influences are expressed through a network of shared relationships and exchanges of social support (Antonucci, 2001; Elder, 1998; Smith & Christakis 2008). According to life-course theory the social context affects individuals both directly and indirectly. This indirect route is by way of social relations and interconnectedness between individuals. Sociohistorical influences may be experienced by one family member but indirectly affect other family members. For example, women who fought for women's rights during the Feminist Movement may transmit influences of this historical event to their children, despite the fact that their children did not experience the sociohistorical event firsthand. One family member's tragedy or triumph (e.g., terminal illness, job promotion) fundamentally influences other members of the family. Similarly, living in a family with one child or 10 children, or with a developmentally

disabled child, influences all members of that family. Empirical investigations of the effects of linked lives are relatively recent, but reflect increasing sophistication in methodological and analytical strategies, such as dyadic dependent statistical models (Roberts, Smith, Jackson, & Edmonds, 2009).

The convoy model of social relations (Antonucci, 1985; Kahn & Antonucci, 1980) offers a life-course perspective on the role of linked lives in development and aging. The convoy model is grounded within a life-span perspective recognizing that these relations are both individual and cumulative and that they reflect a lifetime of experiences and exchanges. Family and intergenerational relations are conceptualized as longitudinal in nature, shaped by personal (e.g., age, gender, personality) and situational (e.g., role expectations, resources, demands) characteristics. One can think of personal characteristics as representative of the life-span perspective's focus on the individual and situational characteristics as representative of the life-course perspective's focus on the broader context. Both influence the structure and exchange of social support that, in turn, can buffer the effects of stress and influence health and well-being. The positive support families provide accumulates over time, can help an individual feel competent to cope with stress, succeed in achieving multiple goals, and face the challenges of life. For the aging process, the convoy model highlights the influential nature of the interconnectedness of individuals. It is not only individual factors or the broader contextual factors that predict aging, but also the direct interactions and reciprocal influence between individuals in families with linked life trajectories. These linked lives can be positive or negative promoting optimal aging or accumulating inequalities.

Differential Pathways and Trajectories of Aging

Both life-course and life-span researchers use longitudinal data to examine hypotheses about different patterns of change over time associated with the effects of biogenetic, life history, social, and personal factors. While researchers from a life-course perspective typically examine *subgroup differences in social pathways of aging*, those from a life-span perspective typically focus on *individual differences in trajectories of intraindividual change*. This distinction is an overgeneralization: in fact, proponents from both perspectives use the terms trajectories and pathways. Nevertheless, it serves to highlight the reasons for different questions, statistical methods and selection of predictors of age-related change. The examples described below illustrate this point.

When life-course researchers consider trajectories of aging, they embed the trajectories in social pathways defined by social institutions (e.g., subgroups with different levels of education or socioeconomic status) and relationships that provide social support (e.g., family). Life-course trajectories are charted by

linking a sequence of events, states, or transitions across successive years (e.g., the states of employment, marriage, disability, cognitive status, and health). Questions about cohort differences in patterns of trajectories (e.g., trends in disability or transition to retirement) are prevalent in life-course research.

Life-span researchers, on the other hand, generally consider trajectories at the level of the individual and model developmental changes in behavior and functioning over time. The subsequent categorization of trajectory types might be based on a standard measure of individual differences (e.g., intelligence, personality) or functional status at baseline. Frequently, researchers characterize trajectories post hoc regarding whether they reflect *pathological, normative, or successful patterns of aging* (e.g., Rowe & Kahn, 1987; Smith & Baltes, 1997). Life-span researchers are also particularly interested in determining whether trajectories of change in a functional domain are age- and/or death-related (*terminal decline*) and whether different types of trajectories (e.g., stability vs. change) are observed in different life phases (e.g., the young old vs. oldest old). For example, theories of terminal decline suggest that multiple domains of psychological functioning show incremental negative trajectories that are larger and qualitatively different from normal age-related changes in behavior, especially during a window of 5 to 7 years before death in very old age. Birren and Cunningham (1985; Berg, 1996) suggested that terminal decline may represent the emergence of a *cascade of structural change*. Examination of this phenomenon is important because it opens a window on different emergent mechanisms that may underlie change in function (e.g., ones not necessarily related to early-life factors.)

The two perspectives are also characterized by different sets of questions about change linked to aging. Whereas the life-course perspective poses questions about levels of *subgroup disparities* in trajectories of aging (e.g., the social gradient of health or mortality), the life-span perspective asks questions about the *distribution of interindividual differences* in the level, rate (slope), and profile of developmental change trajectories.

Both perspectives ask whether the range of disparities and distribution of functioning observed in a sample expands or contracts across the life course. The various positions on these questions reflect different lenses on the *forces of heterogeneity across the life course*. One position argues that early adult life is a phase when within-cohort heterogeneity and social pathways expand greatly and that the consequences of this expansion are maintained for the rest of life. Young adulthood, for example, is a life phase that stimulates the initiation of a wide range of life-course trajectories in terms of education, work, family, and health behaviors (e.g., Elder, 1998). Gender is also associated with different lifelong careers and life conditions in old age (Moen, 1996; Smith & Baltes, 1998).

Humans are very susceptible to influence in their early years, but with age are thought to become increasingly stable in important respects. Furthermore, the social and cultural opportunities for change linked to the life course generally decline with age.

A second position on the distribution of social disparities and interindividual differences suggests that heterogeneity may increase with age (e.g., Dannefer, 1988). The combined effects of an individual's unique experiences over more years should magnify differences between them. Genetically based differences would also have had more time to be expressed and to contribute to divergence. Moreover, older adults, somewhat freer from social constraints, might be more likely to select their own course of action.

Counter to these two positions, however, are proposals regarding reduced heterogeneity with age, especially among the oldest old. Selective mortality contributes to a corresponding reduction in observed heterogeneity, at least for those factors and aspects of functioning that predict mortality (Vaupel et al., 1998). We turn next to a consideration of a selection of contemporary evidence informed by these perspectives.

CUMULATIVE EQUALITIES AND DISPARITIES ON WELL-BEING AND HEALTH

Effects of Social Class, Racial, and Ethnic Minority Aging

Numerous studies over the past few decades have indicated that individuals of lower socioeconomic status have worse health than individuals of higher socioeconomic status (e.g., Adler, Boyce, Chesney, Folkman, & Syme, 1993; Marmot, Shipley, & Rose, 1984; Preston & Taubman, 1994). Findings show evidence of this effect of socioeconomic status (SES) at all ages of the life course, and interestingly enough suggest that there are age-graded group differences in that individuals of low SES are more likely to have health problems at younger ages than individuals of higher SES (House et al., 1994). This research suggests that inequalities in health status by SES vary systematically across the life course. This life-course variation is particularly salient in old age; House and colleagues (2005) have shown that the compression of morbidity is specific to higher SES groups while lower SES groups are more likely to experience multiple health problems earlier in old age.

Though it was originally suggested that these health disparities were due to a lack of access to proper medical care, investigations have indicated that the causes are much more complicated and significantly influenced by the individual's resources and other environmental factors (Adler et al., 1993;

Marmot et al., 1984; Pappas, Queen, Hadden, & Fisher, 1993). Furthermore, this health disparity is not merely a case of the "haves" versus the "have-nots" but rather there is a social gradient effect at all levels of SES and even in the face of universal access to health care and despite access to sufficient resources, increasing social status remains related to better health (Marmot, 2006). Group differences in SES are due to complex contextual effects leading to differing life-course trajectories of health and sharply divergent life expectancies. While there is strong evidence for the social gradient effect in health disparities, the challenge to current and future research is in determining the causes of these inequalities. In effect, the divergence in social capital accrued over the life course has significant implications (Frytak, Harley, & Finch, 2003). The life-course approach to aging suggests that it is imperative to investigate the implications of cumulative lifelong influences on development in old age. Promoting better health in old age will not be as simple as providing lower SES individuals with adequate health care access, but rather necessitates interventions in public, family life, and individual life styles, and is best initiated at earlier rather than later stages of the life course.

It is widely recognized that many ethnic minorities live in a culture of prejudice and discrimination that both influences the psychological resources available to them as well as their physical and mental health (cf. Jackson, Antonucci, & Gibson, 1990). The life-course perspective notes that the effects of discrimination across the life course may differ in form and intensity as a function of birth cohort and timing (Williams & Williams-Morris, 2000). A number of additional factors may contribute to these disparities, ranging from biological dispositions (Baquet & Ringen, 1987) to dietary habits (Hargreaves, Baquet, & Gamshadzahi, 1989), to a failure to receive adequate health care (Jones & Rice, 1987). The specific mechanisms, however, that produce these differential outcomes are less clear (LaVeist, 2000; Williams, 1999; for an exception, see recent work of Jackson, Knight, & Rafferty, 2009.).

Aging research on ethnic minorities also addresses contextual experiences that may be unique to some ethnic groups. Research on aging immigrants indicates that in addition to the challenges faced by ethnic minorities, this group also experiences strains related to the migration experience (such as language barriers, for example; Angel & Angel, 2006). Interestingly enough, researchers have noted an unlikely effect termed the Hispanic Paradox in which the longer Latino immigrants reside in the United States, the worse off their health becomes (Markides & Eschbach, 2005) indicating the importance of examining the interaction of context and time along with the contribution of biology. The utility of a life-course perspective is further noted in considering that because of inadequate access to resources and/or

sociohistorical experiences of war or political unrest in their native countries, many immigrants may bring with them a life-course path that has a significant impact on the aging process.

When considering group differences, it is critical to recognize the role that race, ethnicity, and culture play over the life course and particularly for the aging experience (Jackson & Govia, 2008). Longstanding ethnic diversity, as well as recent immigration patterns, indicates that special attention needs to be paid to these differences. Group level differences related to ethnicity and race are evident in the aging process, and indicate the need for a perspective that integrates biopsychosocial influences across the life course. As society becomes increasingly diverse, a culturally sensitive approach to aging (e.g., health, self, commuting issues) will be even more critical (Jackson, Brown, & Antonucci, 2005).

Effect of Linked Lives

The influence of linked lives on health in old age is impressive and should be considered when addressing health disparities in late life. Specific family relationships have been shown to have differing effects on health and well-being. Married people report better overall physical health, lower levels of depression, better social support, and relationship quality compared to unmarried adults (Murphy, Glaser, & Grundy, 1997; Umberson & Williams, 1999). As a group, married adults are more active, suffer lower rates of chronic illness, and are less likely to engage in risky health behaviors compared to unmarried groups of adults (Lyyra, Törmäkangas, Read, Rantanen, & Berg, 2006; Mendes de Leon, Appels, Otten, & Schouten, 1992). They report lower levels of depression, stress, and loneliness and greater life satisfaction than adults who are not married (Diener, Suh, Lucas, & Smith, 1999; Dykstra & de Jong Gierveld, 2004). Conversely, adults who are divorced and widowed report more mental health issues, including depression, loneliness, and suicidal ideation, compared to married and never-married adults (Dykstra, 1995; Williams, 2003). Among adults who have never married, men experience higher levels of loneliness compared to unmarried women, although no such gender differences emerge among married adults (Dykstra, 1995; Peters & Liefbroer, 1997). However, longitudinal studies of these associations suggest that distress and negative psychological assessments subside or are ameliorated over time (Booth & Amato, 1990).

Intergenerational relationships, especially the parent–child relationship, is one of the most long-term and emotion-laden social ties (Bowlby, 1980; Cairns, 1977; Fingerman, 2001). Gerontologists have suggested that offspring experience both increased feelings of autonomy and closeness toward parents

across adulthood (Fingerman, 2001). Across the life span, the parent–child tie is characterized by a paradox of solidarity and conflict (Fingerman, 2001; Luescher & Pillemer, 1998). Interpersonal problems in this tie appear to be normative (Clarke, Preston, Raksin, & Bengtson, 1999; Fingerman, 2001; Luescher & Pillemer, 1998). Much less is known about how this family tie affects the health of either party. The parent–child relationship may directly influence health in old age on various levels such as induction of stress, caregiving, and access to resources. On some level, health disparities may be a result of the extent of resources and/or support provided through social and family relationships.

Health Over the Life Course

The benefits of studying health during old age within a life-course and life-span framework are considerable. Typically health and illness have been studied with a focus on acute illness and onset of a diagnosed illness. Studies often examine the progression of disease prospectively as opposed to the lifelong trajectory leading up to the onset of the disease. Pearlin, Lieberman, Menaghan, and Mullan (1981) proposed a now classic model describing how the experience of stress affects health and well-being. According to their stress model, individuals experience stressors that affect their health, however, moderating factors such as social relations may reduce the stressors and mediating factors, such as social support, may reduce the interaction between the stressor and health. For aging individuals then, health status is determined by the ability to mediate or moderate the relation between stressors and health. For example, stressors may be mediated by psychological and social resources that reduce the effect of the stressor on health. A life-course perspective is central in examining this link between stressors and health because it has been found that chronic stressors are more likely than acute stressors to have significant effects on health (George, 2003; Krause, 2000). The cumulative impact of prolonged chronic stress exposure and the physiological response may cause declines in overall health status over time (Robles & Kiecolt-Glaser, 2003). Older adults not only have a longer life course in which to experience chronic stress, but are also more likely to have chronic conditions that are exacerbated by stress, perhaps creating greater vulnerability to stress with age (House et al., 2005).

Because the persistence of hardships over the life course can result in cumulative damage to health and well-being, and chronic stress over the life course has significant repercussions for health in old age (Kahn & Pearlin, 2006), it is essential to examine health and aging from a life-course perspective. George (2003) suggests the importance of using the life-course approach to study risk factor trajectories and their influence on health. This approach is

not yet common, but has promising possibilities for understanding health in late life. For example, Barrett (1999) found that women who remarried after a divorce had higher depression levels than women who were continuously married, indicating the significance of not just marital status, but marital history in predicting depression. The value of longitudinal research in examining health and well-being in old age is critical. Life-course research on health now emphasizes the importance of considering historical and cohort effects. For example, the current cohort of older adults experiences relatively high rates of lung cancer, a finding that, when considered absent of the historical context, could lead researchers to interpret this as part of the aging process. In reality, however, historical context has an important influence as the current cohort of older adults came of age in the decades before the ramifications of smoking was fully understood; high rates of lung cancer are actually the result of a high rate of long-term smoking for that cohort.

Due to their living longer, older adults are more likely to have chronic diseases and experience multimorbidities of diseases (Crimmins, 2004). Health in old age is sometimes determined by rates of mortality, but more recent trends in research have focused on morbidity of disease, disability, and frailty (Fried, Ferrucci, Darer, Williamson, & Anderson, 2004). Comorbidity refers to the multiple concurrent health problems that many older adults face. Because the risk of multimorbidity increases with age, it is important to delineate the various pathways into multimorbidity and frailty in old age, especially because there is an asynchrony in the onsets of the various diseases (Yancik et al., 2007). Threats to health clearly increase over the life course and, to some extent, are related to individual lifelong experiences. They are likely to be cumulative in that risk increases with age and earlier ill-health is likely to contribute to later ill-health, and the risk is more likely to be multiplicative than additive. Evidence suggests that events, experiences, and relationships can either help offset or exacerbate vulnerabilities.

CONCLUSION

The current state of the art in research on aging requires a merger between life-course and life-span perspectives and a joint consideration of the themes in Table 1.1. While there is still much to learn about aging, much more is now known as a result of research designed from a life-span and life-course perspective than was known when these approaches were originally proposed in the mid-20th century. At the individual level, for example, we understand that biological, social, and cognitive development and change is not only or always cumulative and linear. It can also be nonlinear, dynamic, progressive and regressive, adaptive and maladaptive, usual and unusual. Initially, the

scientific field rather naively believed that once set upon a path, development unfolded in a manner that was essentially predetermined. Similarly, while life-course theorists understood that institutions, events, and historical periods influence the individual's development, not much thought was given to how these experiences would influence the developing individual, cumulatively or interactively. The life-span and life-course perspectives were in different disciplines and researchers from the two camps rarely communicated with the other. As this book attests, this separation is a luxury we can no longer afford or justify.

As knowledge accumulates, it is increasingly evident that a combination of life-span and life-course perspectives will be most informative to the exploration health inequalities in later life. As the following chapters indicate, this approach provides a broad, clear lens on those factors influencing aging and health inequalities.

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CHAPTER 2

What Can Life-Course Epidemiology Tell Us About Health Inequalities in Old Age?

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The core premise of life-course epidemiology—that current health reflects a lifetime of past exposures—is now firmly established. The life-course perspective can inform several fundamental considerations in intervention development, for example, when the risk factor or exposure is amenable to modification; when the disease process begins; and the most sensitive time points to interrupt its progression. Although results from this body of research have not yet been fully integrated into the design and implementation of health interventions or policies, the next generation of life-course research can potentially provide valuable guidance for strategies to improve population health.

The goal of our chapter is to describe alternative etiologic models frequently considered in life-course epidemiology and briefly review some methodologic approaches and challenges to distinguishing these alternative models. By etiologic, we mean to focus on the *causal exposures* that contribute to the development of disease. We are particularly interested in the way that socioeconomic position (SEP) at various stages of the life course impacts cardiovascular disease (CVD) and cognitive health in old age. We conclude by summarizing progress and highlighting important research gaps.

GENERAL CONCEPTS AND METHODS IN LIFE-COURSE EPIDEMIOLOGY

Alternative Models of the Etiologic Period Linking Exposures and Health

Life-course epidemiology research distinguishes among alternative etiologic models describing how the potential for a toxic exposure to cause disease depends on the timing of exposure to the toxic factor (Power & Hertzman, 1997; Wadsworth, 1997). Etiologic timing models have divergent implications for optimal moments for health interventions. Figure 2.1a–e show causal diagrams reflecting five alternative conceptual models of links between exposure timing and the etiology of disease. In these figures, an arrow from one variable to another represents the assumption that the first variable affects the second; if there is no arrow between two variables, this represents the assumption that those variables do not directly affect one another.

The five models are:

Immediate effect models typically assume very short etiological periods: once the risk factor is removed, risk declines, and possibly returns to the baseline (unexposed) level. For example, in Figure 2.1a SEP in midlife immediately affects cardiovascular risk and SEP in childhood or early adulthood is irrelevant. This is implicit in much conventional epidemiology, when there is no explicit life-course framework. Study designs such as the case crossover, in which each individual serves as his or her own control, are often premised on immediate risk models (Maclure & Mittleman, 2000).

Social trajectory models apply to risk factors that follow a "sticky" trajectory, in which previous exposure strongly influences subsequent exposure (Figure 2.1b). This describes many if not most social exposures. For SEP, it is well established that education influences income, and that income in early adulthood influences income later in working life, and so on. In Figure 2.1b, we show a model in which the effects of early life SEP are entirely mediated by a succession of harmful social exposures; it is only the final exposure to SEP that has physiological consequences in this causal structure. Early deprivation may not cause immediate physical harm, but rather put one "at risk of risk." For example, poverty early in life may reduce educational access, thereby increasing the likelihood of adult poverty, which may increase CVD risk in late life. Such models may incorporate trajectory "elbow" or "lever" periods—moments when the direction of future social experiences can be more easily changed, either for better or worse. Examples of crucial turning points could be first years in school, the transition from school to work, or

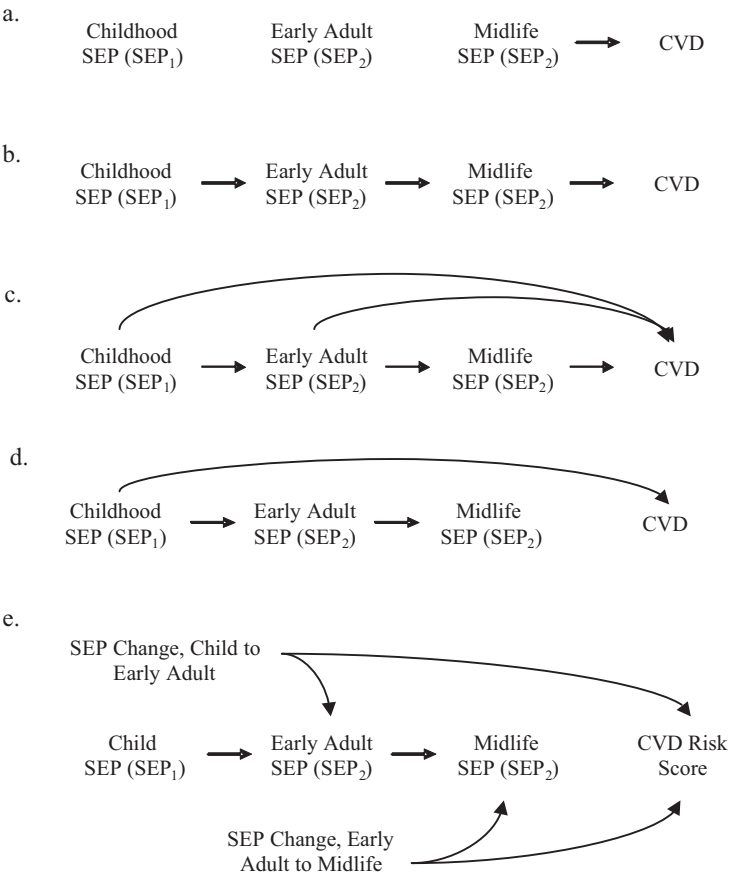

FIGURE 2.1 Causal diagrams for alternative etiologic timing models linking life-course SEP and CVD. a. Immediate effects of SEP; b. Immediate effects model combined with social trajectory of SEP; c. Cumulative effects combined with social trajectory of SEP; d. Early sensitive period combined with social trajectory of SEP; e. Physiologic effects of trajectory–change in SEP.

the period following widowhood or divorce. The important feature of social trajectory models is that the earlier exposure must induce subsequent adverse exposures in order to harm later health; interrupting the cascade of social disadvantage at any point can interrupt the health consequences of social disadvantage.

Cumulative biological models assume that each period of risk factor exposure wreaks physiologic harm that increases risk of later developing illness,

even if the individual is subsequently removed from exposure (Figure 2.1c). Toxic exposures, such as lead or pesticides, may have cumulative biological effects on risk of cognitive impairment. Critical events altering an individual's underlying biological vulnerability may be embedded within cumulative processes (e.g., first birth may change breast cancer risk, menopause may alter heart disease risk).

Sensitive period or *latency* models apply if there is a window of time during which exposure to the risk factor has a special influence and alters risk for development of disease much later in life. Effects may not manifest for years after exposure is removed. In a latency model, exposures encountered *after* the critical window closes do not influence risk. In Figure 2.1d, early childhood SEP influences adult CVD, but early adult and midlife SEP have no effects in and of themselves. Latency models typically focus on early life experiences that do not have a cumulative effect but are difficult to remediate later in life (e.g., epigenetic marks; Meaney, Aitken, Bodnoff, Iny, & Sapolsky, 1985; Meaney et al., 1996) or changes in early growth patterns (Barker, 1990; Barker & Lackland, 2003; Fox, Levitt, & Nelson, in press).

Models that hypothesize *physiological effects of trajectory* are distinct from models that merely note the existence of a trajectory. In this last type of model, it is the magnitude and direction of change in SEP itself—rather than the level of SEP at any given point in time—that influences CVD risk (Figure 2.1e).

Three Difficulties With Conventional Analytic Models

Although it is relatively easy to make theoretical distinctions between these models, doing so with empirical studies is often difficult due to problems we call sticky exposure trajectories, nebulous measures, and multiple pathways (Glymour, 2007). Because SEP follows a sticky trajectory, measures at different points in the life course are highly correlated. Furthermore, SEP is a multifaceted construct that is rarely measured comprehensively and reliably. For example, we frequently measure education and only one characterization of occupation (e.g., manual–nonmanual), ignoring countless SEP dimensions such as educational quality, occupational prestige, workplace autonomy, earnings, and wealth. Measurement is probably especially inadequate for childhood SEP. Finally, many social exposures influence health through multiple distinct pathways: The same exposure may affect an outcome via several separate, independent pathways, each of which is best described by a different etiologic timing model. For example, there may be a sensitive prenatal or infancy period

of rapid vascular growth, during which disadvantaged SEP and accompanying malnourishment results in permanent changes to vascular architecture, which predispose to adult CVD (Barker & Lackland, 2003). Decades later in life, disadvantaged SEP may exert an immediate effect on cardiac risk, by increasing exposure to short-term acute stressors triggering autonomic dysregulation and arrhythmias (Samuels, 2007). Such a combination of independent pathways, influenced by distinct periods of life, will appear to be consistent with a cumulative effects model. A related problem arises when the health outcome is a broad measure (e.g., self-rated health or disability), influenced by many distinct pathological conditions. The exposure may influence many different but related health outcomes that become bundled into self-rated health or disability.

Few studies have done an adequate job testing alternative etiologic models against one another, because many empirical associations would be predicted by any of the etiologic models. For example, note that in all of the models shown in Figures 2.1a–e, early adult SEP (SEP_2) would be expected to predict CVD, even though it is only causally relevant in models 2.1b and 2.1c, and only has a direct effect, independent of later SEP, in model 2.1c. One of the most common analyses used to test life-course models is to measure SEP at two time points and test whether both predict independently of one another (i.e., does childhood SEP predict after adjusting for some measure of adult SEP). This is often misleading. If model 2.1b is the true causal model but early adult and midlife SEP are not comprehensively measured, then childhood SEP may predict CVD even after adjusting for early adult and midlife measures (incorrectly suggesting that the correct etiologic model is 2.1c or 2.1d). Conversely, if childhood SEP is very poorly measured, but midlife SEP is very well measured, then midlife SEP might predict CVD better than childhood SEP even if childhood SEP is the only influential exposure.

A second common approach to testing life-course models involves creating a cumulative measure of deprivation by counting the number of periods of low SEP out of a handful of time points assessed and showing that this number predicts more strongly than SEP at any single time point. Unfortunately, in the context of inadequate measurement and sticky social trajectories, the cumulative SEP measure might predict independently of or better than the single time point measure even if SEP is only causally influential at one point in time.

A final popular approach to testing for physiological embedding of social trajectory is to examine whether trajectory (e.g., moved up, moved down, or remained flat) predicts the outcome. However, because trajectory predicts

final social position (i.e., people who went up must have ended high; people who went down must have ended low), these analyses will conflate direct effects of ending position and effects of trajectory.

In summary, the analyses frequently used in life-course epidemiology produce results consistent with any or several of the alternative causal structures shown in Figure 2.1 (Rosvall, Chaix, Lynch, Lindström, & Merlo, 2006). There is a critical need to advance our ability to test these models against each other and identify empirical associations that would falsify one or more of the models even in the context of measurement error and correlated life-course exposures.

Linking Exposure Development Models With Etiologic Timing Models

While the life-course models operate at the juncture of risk factor and disease etiology, they are primarily informed by our understanding of the etiology of the disease or health outcome. Here we suggest that for purposes of intervention, similar emphasis on the etiology and life-course patterns of the *exposure* will greatly enhance our ability to intervene successfully. We must ask, when can social exposures be changed? That is, we must put renewed effort into understanding the dynamics of our risk factors across the life course in order to develop appropriate (and appropriately timed) interventions to alter these risk factors. Integrating knowledge of the etiology of disease with an understanding of the onset and trajectory of the exposures could be invaluable in organizing interventions and social policies to improve health of the aged. This is not primarily the domain of epidemiology or health research in general, but will require drawing on expertise from other disciplines, for example, sociology, psychology, economics, and political science.

To be useful, an understanding of the life-course process linking an exposure and any specific health outcome must be integrated with an understanding the dynamics of the exposure, and we propose the following questions as a framework:

1. At what point in the life course does the exposure begin to exert important health consequences and when in the life course are these effects largest? Is there any point at which removing the risk factor will no longer offer substantial health benefits, because too much physiologic damage has already been incurred?
2. Is there a typical pattern for changes in the exposure across the life course either socially (e.g., retirement or widowhood) or physiologically (e.g., early childhood or in the aftermath of a major health crisis)

defined "elbow" points at which people frequently experience changes in the exposure?

3. How likely is it that socioeconomic conditions can be modified by macropolicies or individual interventions at any given point in the life course?

EFFECTS OF SEP ACROSS THE LIFE-COURSE ON CARDIOVASCULAR OUTCOMES

The socioeconomic gradient in CVD has been a core finding of social epidemiology. It appears with almost every domain of SEP, for disease incidence and mortality, and for almost every specific cardiovascular outcome.

Observational Studies Linking Life-Course SEP to Cardiovascular Disease

Several recent reviews summarize the now large body of evidence that childhood SEP, typically measured as parental education or occupational status, affects CVD risk factors, risk of CVD incidence, and mortality (Galobardes, Lynch, & Smith, 2004; Galobardes, Shaw, Lawlor, Lynch, & Smith, 2006; Pollitt, Rose, & Kaufman, 2005). In addition to the numerous conventional observational epidemiology studies, reductions in all cause mortality from improvements in education have also been shown in a quasiexperimental context (Lleras-Muney, 2005). This research stands in interesting contrast to recent studies suggesting that although the level of SEP predicts CVD risk, changes in SEP in middle and old age predict onset of CVD only for some conditions (Adams, Hurd, McFadden, Merrill, & Ribeiro, 2003) or not at all (Michaud & van Soest, 2008).

In general, in epidemiological studies, the magnitude of the associations between adult SEP indicators and CVD outcomes are larger than the associations with childhood SEP. Frequently the relationships between childhood SEP and adult CVD are small or nonexistent in models adjusted for adult SEP (Power, Hypponen, & Smith, 2005; Singh-Manoux, Ferrie, Chandola, & Marmot, 2004). A recent systematic review suggested that evidence is most consistent with a cumulative effects, model-linking life-course SEP and CVD, including a modest direct effect of childhood SEP (Pollitt et al., 2005).

There are several explanations for the apparent dwarfing of childhood effects by adult SEP observed in the epidemiologic studies: (a) the effects of childhood SEP on adult CVD may be primarily mediated by adult SEP; (b) measurement of childhood SEP may be so inadequate compared to measurement of adult SEP that childhood SEP measures lose significance; (c) adult

SEP may be a common effect of childhood SEP and other unmeasured causes of CVD, and adjusting for adult SEP induces collider bias obscuring the direct effects of childhood SEP (Cole & Hernán, 2002).

Evidence from research in social science and medicine suggests that the first explanation—no direct effects of childhood SEP on cardiovascular risk—is unlikely. Social conditions in early life appear to induce physiological changes that persist into adulthood and would change cardiovascular risk. For example, Miller and Chen found that parental home ownership at ages 2 to 3 predicted expression of two genes regulating inflammation in a group of female adolescents when these girls were aged 13 to 19 (Miller & Chen, 2007). Retrospectively reported family SES also predicted amygdala activation in response to threatening facial stimuli, independently of perception of own social standing (Gianaros et al., 2008).

These studies suggest that childhood SEP likely does have effects that are not entirely mediated by adult social conditions. This is consistent with extensive evidence suggesting that physical characteristics at birth predict adult cardiovascular risk (Godfrey & Barker, 2007), childhood health predicts adult earnings and health (Haas, 2007; Hayward & Gorman, 2004), physiological differences in cardiovascular risk are evident even in childhood (Berenson et al., 1992), and that childhood differences track into adulthood (Bao, Srinivasan, Wattigney, & Berenson, 1994; Davis, Dawson, Riley, & Lauer, 2001; Lauer & Clarke, 1989). Overall, the evidence is most consistent with the hypothesis that early life factors may have a disproportionate influence because they both cause physiological changes and set in motion a trajectory of social and behavioral exposures that persists into old age, but socioeconomic deprivation across the life course has a cumulative effect on CVD risk.

Intervention Studies Linking SEP or Cognitive Enrichment to Cardiovascular Disease

The best tests of the etiologic hypotheses linking SEP at different life-course moments to either CVD or cognitive aging will come from intervention studies. Unfortunately, few randomized trials assess the consequences of changes in SEP on adult health or CVD. The majority of SEP interventions have focused on short-term medical service outcomes (e.g., negative income tax studies), effects on children's health (e.g., Progresa, and the South African pension plan), or adult mental health (Moving to Opportunity). Overall results from these studies are fairly encouraging.

Cash payouts given to very poor families in Mexico's Oportunidades (formerly Progresa) program apparently improved various measures of health

in children, including anthropometric indicators, anemia risk, and cognitive measures. Definitive evidence on the health effects of Oportunidades has not yet emerged (Fernald, Gertler, & Neufeld, 2008; Lagarde, Haines, & Palmer, 2007). Furthermore, attributing the Progresa effects to SEP is difficult because the intervention not only provided poor families with extra money but also increased attendance at school and health visits.

The South African pension plan has been used as an exogenous source of financial payouts, because most South African Blacks did not anticipate receiving the pension. Pension receipt appears to improve both the self-reported health of the pensioner and in households that pool money, also the self-reported health of all other coresident adults and growth indicators of children. In households that do not pool money, the pensioner is actually often healthier than nonpensioner adults (Case, 2001). We do not have any direct measures of how the pension affects cardiovascular health or cognitive outcomes however.

The Social Security notch study provides evidence from an interrupted time series–quasiexperimental design that extra earnings for the elderly increased overall mortality, but the study identified this effect off a single discontinuity in Social Security payment amounts (Snyder & Evans, 2002). A number of other policies that introduced drastic changes in SEP (e.g., the GI bill, unemployment benefits, numerous other changes in the Social Security benefits structure) and might be considered to provide quasiexperiments for the effects of SEP have not been evaluated with respect to long-term evolution of chronic diseases such as CVD.

In summary, we have multiple pieces of evidence from across the life course, but few trajectory studies to help us understand the entire process. We know that early childhood interventions improve child health, and that child health predicts subsequent cardiovascular health. We do not yet have intervention results directly demonstrating effects of changes in adult SEP on adult CVD risk or of changes in childhood SEP predicting adult onset CVD.

EFFECTS OF SEP ACROSS THE LIFE-COURSE ON COGNITIVE OUTCOMES

Observational epidemiologic studies have linked aspects of socioeconomic position across the life-course, from infancy and early childhood, through primary education years, and adulthood, to an array of old age cognitive outcomes. It is worth noting that the most relevant time periods and the magnitude of effects of social exposures may differ for specific cognitive skills (e.g., attention, episodic memory, or executive functioning), however, the evidence

at this point is far too preliminary to guide us in understanding what types of skills are most influenced by SEP.

Evidence That Childhood Conditions Affect Old Age Cognition

Several studies suggest that parental SEP predicts old age cognitive outcomes. This evidence arises from direct associations between parental SEP measures and old age cognitive outcomes; associations between anthropometric measures thought to reflect nutritional deprivation and cognitive aging; and associations between parental SEP and child IQ.

In a sample of Finnish men, father's occupation and mother's education were correlated with cognitive function at ages 58 and 64 (Kaplan & Lynch, 2001). Moceri used United States census records to show that area of residence at birth and number of children in the family were associated with risk of AD (Moceri et al., 2001).

Parental SEP may predict children's cognitive outcomes because of genetic factors that influenced parents' attained social position (e.g., via IQ) and their children's cognitive outcomes. This cannot be ruled out based on current evidence. Alternatively, parental SEP may influence complexity and cognitive demands of early childhood environments, and, quite separately, parental SEP may influence cognitive development via material conditions. Nutrition is among the best elaborated pathway potentially linking childhood SEP to cognitive impairments. Substantial research has focused on the relation between cognitive aging and growth measures. Measures of growth, such as height or cranial size, are thought to remain stable in adulthood and reflect childhood nutritional status at specific developmental periods. Several independent studies have found an association between measures of brain or head size and late-life cognitive function (Graves et al., 1996; Katzman et al., 1988; Schofield, Logroscino, Andrews, Albert, & Stern, 1997). Efforts to identify a specific sensitive period for nutritional deprivation in humans have been limited but suggestive (Gale, O'Callaghan, Godfrey, Law, & Martyn, 2004). Some studies, however, find no association between measures of head size and subsequent dementia or cognitive impairment (Edland et al., 2002; Jenkins, Fox, Rossor, Harvey, & Rossor, 2000). Some of the null results have arisen from relatively prosperous populations in which cranial size may not reflect nutritional deprivation. A number of other growth measures have been linked to cognitive aging or dementia, including birth weight, height, arm span, and leg length (Abbott et al., 1998; Huang et al., 2008; Richards, Hardy, Kuh, & Wadsworth, 2001).

SEP may also influence parental behaviors such as breastfeeding (although the relationship between SEP and breastfeeding may differ across various cultural contexts). Research on breastfeeding also provides an indirect source of evidence for special cognitive plasticity in early childhood, because the effect sizes are remarkably large. Many observational studies have shown that breastfeeding benefits child IQ, and a recent randomized trial confirmed these findings (Kramer et al., 2008). Especially interesting is the finding that this association appears to be modified (perhaps even reversed) by a genotype that controls fatty acid metabolism (Caspi et al., 2007).

Evidence for Direct Effects of Schooling on Old Age Cognitive Outcomes

To the extent that studies have tried to disentangle specific components of SEP, education often appears more important than adult SEP indicators such as income or occupation in predicting cognitive outcomes (Karp et al., 2004). Education strongly predicts improved cognitive function (Brayne & Calloway, 1990) and reduced risk of dementia (Fratiglioni & Wang, 2007). Education may be so powerful because it influences both material pathways via effects on earnings and also fosters encounters with cognitively demanding environments.

Not all studies find a protective effect of education, however, (Brayne & Calloway, 1990; Hébert et al., 2000; Munoz, Ganapathy, Eliasziw, & Hachinski, 2000), and many existing studies have methodological limitations (Glymour, Weuve, Berkman, Kawachi, & Robins, 2005). A number of studies show that various measures of cognitive function earlier in life (e.g., IQ) predict old age dementia, (Butler, Ashford, & Snowdon, 1996; Plassman et al., 1995; Whalley et al., 2000) and this has been used as an argument that perhaps the apparent effects of schooling on old age outcomes is due to confounding by IQ. The epidemiologic evidence is particularly uncertain when considering the effect of education on cognitive change among the elderly (rather than the effect of education on cognitive level achieved prior to old age; Alley, Suthers, & Crimmins, 2007; Christensen et al., 2001; Glymour et al., 2005; Van Dijk, Van Gerven, Van Boxtel, Van der Elst, & Jolles, 2008; Wilson et al., 2009).

The finding that education appears more important than income or occupation should also be accepted with caution because confounding bias potentially introduced by early life IQ would presumably be stronger for education than for any other indicator of SEP. The best observational designs to rule this out, that is, longitudinal change models with cognitive score (instead of dementia) as the outcomes, have not been implemented sufficiently frequently to be confident that education is truly stronger. In a

quasiexperimental design, changes in state compulsory schooling laws during the early 20th century predicted improved memory test scores decades later for children constrained by longer mandatory schooling periods (Glymour, Kawachi, Jencks, & Berkman, 2008). This suggests that at a very minimum schooling improves level of cognitive function, which would be expected to delay onset of clinical impairments.

Some studies (e.g., the Baltimore Epidemiologic Catchment Area cohort) suggest that the relationship with cognition is stronger at lower levels of education (Lyketsos, Chen, & Anthony, 1999), but other studies show graded education effects in both high and low average education populations (Ofstedal, Zimmer, & Lin, 1999). A related question is whether there is a continuous education effect or particular thresholds that are important, perhaps due to credentialing effects.

Evidence for Continued Neuroplasticity in Adulthood

Although there is tremendous emphasis on the special importance of early childhood for cognitive development, it is also manifest that humans maintain neurologic plasticity throughout their lives. Not only do humans continue to learn, acquire new memories, and master new skills throughout adulthood, adult brains can remodel in response to environmental demands or insults. The extent and biological basis of adult neuroplasticity is an area of intense and expanding research. It now seems that dramatic changes, underwritten by both synaptic–network remodeling and neurogenesis, can be achieved in the adult brain.

Much of the most compelling research on the biological plausibility of neurologic changes in response to environmental complexity comes from animal research. For example, recent evidence indicates that spatial memory deficits induced by focal neuronal injury in adult mice can be offset by environmental enrichment. In this model, the mice actually recovered memories that had been lost due to the neuronal injury. The recovery was associated with synaptic growth apparently prompted by chromatin changes (Fischer, Sananbenesi, Wang, Dobbin, & Tsai, 2007). In humans, neuroplasticity after cortical injury such as stroke has received greater focus in the context of motor recovery rather than cognitive recovery (Cramer, 1999; Nelles, Jentzen, Jueptner, Müller, & Diener, 2001).

Other studies suggest extensive use-dependent plasticity. For example, Maguire and colleagues found that the posterior hippocampus—an area of the brain responsible for spatial representation and thus navigation—is larger in London taxi drivers compared to controls (Maguire et al., 2000). Taxi drivers performed worse than bus drivers (who follow fixed routes and thus face

fewer navigation challenges) on various visuospatial tests (Maguire, Woollett, & Spiers, 2006). Studies that incorporate a longitudinal design following interventions are especially compelling. Juggling training induced localized gray matter enlargements, and subsequent neglect of juggling was associated with loss of the new volume (Draganski et al., 2004). Adults who receive cochlear implants have been shown to recruit new brain areas (outside the typical language processing regions) to make sense of sounds (Giraud, Price, Graham, & Frackowiak, 2001). Among blind individuals who learn Braille, areas of the brain typically devoted to visual interpretation are recruited to assist with tactile information processing (Hamilton & Pascual-Leone, 1998). More directly related to the exposures and outcomes we focus on here, there is also some evidence drawn from humans that schooling-related skills, such as literacy acquisition, influence the functional architecture of the brain (Petersson, Reis, & Ingvar, 2001).

Epidemiologic evidence also suggests that leisure activities or complexity of activities protects against cognitive decline or dementia in humans (Friedland et al., 2001; Scarmeas, Levy, Tang, Manly, & Stern, 2001; Schooler & Mulatu, 2001), but the direction of causation is unclear. The association may occur because cognitive stimulation protects against decline or because undiagnosed neuropathological processses reduce activity levels. In what might be considered an effort to mirror the concept of environmental enrichment used in animal studies in the context of humans, measures of life space have been used to predict cognitive decline (Croweet al., 2008). This is a promising area, but these associations may be primarily driven by physical mobility, because current measures of life-space capture where the individual has actually traveled (only in one room, in multiple rooms of his/her dwelling, outside the dwelling to yards or porches, outside the house but in the neighborhood, etc.).

Because of the strong influence of early life educational experiences on subsequent cognitive exposures (via for example, occupational stratification, social norms about leisure time, financial conditions), observed associations between education and cognitive changes in old age provide incomplete information about whether and how the timing of exposures matter. There is biological evidence for neural plasticity in early life, but adult exposures may also affect reserve and resilience.

Effects of Adult Occupation and Income on Cognitive Outcomes

Evidence that neuroplasticity continues into adulthood can also be derived from links between adult SEP measures and cognitive outcomes. Both occupational characteristics (Kröger, et al., 2008; Lindesay, Briggs, & Murphy,

1989) and income have been demonstrated to predict cognitive outcomes in the elderly. Occupation is especially interesting because of the possibility that occupational characteristics define environmental complexity and cognitive challenges for most adults. A handful of studies suggest complex links between occupational characteristics and subsequent cognitive outcomes (Kröger et al., 2008; Smyth et al., 2004; Stern et al., 1995). For example, Kröger found that occupations with high complexity of interactions with people predicted reduced dementia risk. Interactions with people were more important than other types of occupational demands, such as complexity of work with objects or ideas. Although these studies included other indicators of SES besides occupational characteristics (e.g., education), they cannot conclusively rule out confounding by parental SES, educational quality, or IQ.

Finally, we note that studies linking SEP to cardiovascular risk provide very strong evidence that SEP will also influence cognitive aging. Vascular disease is now recognized as a key component of cognitive aging; the line between Alzheimer's disease and vascular dementia is increasingly blurred, although the precise etiologic processes remain unclear. Thus, even when research does not include cognitive function or impairments as explicit outcomes, findings that show SEP influences diabetes, hypertension, and other important vascular risk factors suggest that SEP will in turn influence cognitive aging.

Intervention Studies Linking SEP or Cognitive Enrichment to Adult Cognitive Outcomes

Although a number of early studies suggested cognitive interventions were quite beneficial for older adults, the study designs were not always ideal and the outcomes primarily focused on skills very similar to those specifically trained in the intervention (Thompson & Foth, 2005; Willis & Schaie, 1986). Subsequent efforts focusing on rigorously randomized studies have produced mixed results. Among the most promising is the Experience Corps project (Fried et al., 2004), The Advanced Cognitive Training for Independent and Vital Elderly (ACTIVE) was another major trial in which elderly people were randomized to a cognitive training intervention. This trial certainly succeeded in improving the trained cognitive skills, and these improvements persisted for years after completion of the training. However, transfer to other cognitive domains and to maintenance of daily function were not observed in initial follow-ups but were found for self-reported functioning in subsequent reports (Ball et al., 2002; Willis et al., 2006; Wolinsky et al., 2006). Because it was not possible for participants to be unaware of their intervention status, self-reported outcomes are not ideal. It is now uncertain if the specificity of the results to the trained skills means that the gains were not important, or if these skills will ultimately

protect participants against onset of impairments that prevent independent living.

Finally, a number of studies have examined the effects of cognitive training on patients with mild cognitive impairments. These studies have several design flaws, and few have been well-conducted RCTs, but they suggest that even among a group of elderly with already manifest deficits, cognitive training may be beneficial (Belleville, 2007; Rozzini et al., 2007).

In summary, childhood cognitive skills influence adult cognitive level and thus predict dementia risk; adult cognitive engagement or activity predicts cognitive level, and cognitive training can be implemented for at least localized changes in cognitive skills. This suggests a model in which childhood factors determine cognitive level or reserve, and thus can increase or decrease dementia risk through processes unrelated to the pathologic process of dementia. Cognitive engagement may reduce pathological processes or improve resilience and compensation, but the current evidence for this is inconclusive. Finally, material conditions determined by adult SEP probably affect the development of some aspects of neuropathology, for example, vascular health.

STABILITY OF SEP ACROSS THE LIFE COURSE

Understanding typical trajectories of SEP can help inform policy and interventions. For example, life-course periods or ages characterized by extensive natural variability in SEP are likely to provide promising opportunities for planned interventions. Capitalizing on naturally occurring moments of transition (e.g., high school graduation) or instability may increase the likelihood of success of planned interventions. We now turn our attention to exploring the life course of social processes rather than disease processes.

Life Course Trajectories of Income

Parental SEP strongly influences the SEP of children: recent U.S. data indicate that for every 10% change in the earnings of the father, a 6% change in the earnings of the son was anticipated. With this level of stability, it would average five to six generations for descendants of a family at the poverty level to be within 5% of the national average earnings level (Haskins, Isaacs, & Sawhill, 2008; Mazumder, 2005). There is also substantial intergenerational transmission of neighborhood average SEP, so children raised in highly impoverished neighborhoods are themselves very likely to live in similarly impoverished neighborhoods in adulthood (Sharkey, 2008).

In the conventional income trajectory, workers begin with low earnings, which peak in middle age and decline slightly into old age (Willis, 1986). Relative position at each point in the life course is more stable compared to cohort members than compared to other age groups. However, despite the overall stability, even within relatively brief follow-up periods, there is substantial variability in income. Over a 10-year follow-up using the British Household Panel Survey, only about a quarter of respondents followed a "flat" income trajectory that is, remaining within +/–15 percentile points of their average position (Rigg & Sefton, 2006). Another quarter followed a "flat with blips" (one or two blips of +/–15 percentile points) trajectory. Major life events that occurred during the follow-up period, such as retirement, partnership formation, or birth of children, predicted divergent income trajectories. Marriage dissolution was especially relevant for White women and was associated with substantial declines in income (Rigg & Sefton, 2006; Willson, 2003). In summary, despite the correlations in income levels at various moments in the life course, trajectories of change or instability are quite common. These changes appear to be associated with major life events, and the importance of specific events differs by race and gender categories.

Changes in Education

Although education is often considered largely completed by the time of early adulthood, participation in adult education programs is remarkably common and has increased in recent decades (Kim, Hagedorn, Williamson, & Chapman, 2004). In the National Household Education Surveys conducted in 2000–2001 and 2004–2005, 54% of all adults ages 16 to 64 reported participating in some sort of formal learning activity in the previous 12 months (Kienzl, 2008). These were most frequently work-related (33%) or personal development (23%) courses. There was a strong social gradient: adults who previously obtained more education were more likely to continue participation in adult education. For example, only 7% of individuals without a high school diploma enrolled in a work-related training course, while 54% of individuals with college degrees did so. Although these participation rates may not always translate into new credentials, they demonstrate that adults are involved in numerous cognitively demanding learning contexts even after leaving formal schooling.

WHAT INTERVENTIONS OR POLICIES HAVE SUCCESSFULLY CHANGED SEP?

In addition to understanding naturally occurring trajectories, evidence regarding the timing of interventions can be drawn from the successes and failures of

previous intervention efforts. In some cases these interventions, for example, educational training programs, were implemented without specific regard for health consequences, but they are nonetheless relevant to planning interventions to improve health. A number of major interventions have attempted to improve individual or family SEP for its own sake, without attention to possible health benefits.

Childhood Interventions to Change Education or Labor Market Opportunities

Initiatives to improve childhood educational opportunities range from clearly defined intervention programs serving individual children or families to larger school or community reforms (e.g., Harlem Children's Zone) to macrolevel federal schooling policies (e.g., integration). Most such interventions are not evaluated or even, in the case of social policies, implemented in a manner that would ever permit rigorous evaluations. However, the handful of interventions that have been thoughtfully evaluated suggest that children benefit substantially from enhancements in educational access and resources and that those benefits persist for decades. A cluster of well-known early childhood interventions—including the Chicago Child–Parent Center and Expansion Program (CPC; ages 3 through 9; Reynolds, 2000). The Abecedarian Project (serving high-risk children from birth to age 8; Campbell, Ramey, Pungello, Sparling, & Miller-Johnson, 2002), and High/Scope Perry Preschool (serving children ages 3 and up; Schweinhart et al., 2005)—have produced significant benefits in the lives of the children who received the interventions. These benefits include (depending on the study), higher lifetime earnings, greater employment stability, lower risk of criminal involvement, or higher educational attainment. The primary mediators are unclear: SEP benefits may be due to better social skills, more motivation, or higher cognitive functioning, all elements that are important in succeeding in educational and workplace settings.

A more recent generation of childhood interventions include more comprehensive changes in family and community conditions. The Mexican poverty program Progresa (now "Oportunidades"), initiated in 1998, provides grants to poor families if their children (grades 3–9) attend 85% of school days. This program has proven successful at improving a range of child physical and cognitive measures (Fernald et al., 2008).

From these and many other studies, we know that we can intervene in childhood to enhance educational outcomes via either individual level interventions or changes in social policies. Additionally, some interventions have also favorably affected employment and income later in the child's life. The

success of early childhood interventions suggests that interventions aimed at changing SEP outcomes are perhaps most effective when enacted early in life.

Adult and Family-Based Interventions to Change SEP

One avenue for enhancing income and occupational status in adults are job training programs and income or tax policies. Similar to educational interventions, there are countless programs that could be considered job training programs, ranging from skills training offered by employers to federally funded programs to promote work as an alternative to welfare. Overall, results from trainings or interventions that attempt to improve adult or family SEP have been disappointing. LaLonde reported that in general, public sector-sponsored training programs for the economically disadvantaged could modestly raise the income of adult women, but results for adult men and youth showed either no effect or even a negative effect on postprogram income (1997). A meta-analysis of government-funded training programs for economically disadvantaged individuals operating from 1964 through 1998 showed the same results as LaLonde's review: earnings effects were largest (though still modest) for adult women, effects were small and mixed for men, and largely absent for youth (Greenberg, Michalopoulos, & Robins, 2003).

The Moving to Opportunity for Fair Housing Demonstration (MTO), which randomized families to receive vouchers to move to low poverty neighborhoods, also showed somewhat disappointing results with heterogeneous treatment effects. Adult women and adolescent females experienced mental health benefits and obesity reductions. These findings did not extend to adolescent males, who appeared to suffer from the new neighborhood. Qualitative research suggests that for females, reduced fear of sexual harassment and pressure was an important contributor to mental health improvements (Popkin, Leventhal, & Weismann, 2008). On the other hand, intervention participants showed no labor market benefits for adult (e.g., employment or earnings), or sustained differences in reading and math scores for children (Kling, Liebman, & Katz, 2007; Sanbonmatsu, Kling, Duncan, & Brooks-Gunn, 2006). The MTO illustrates that many important consequences of social interventions may not be measured in standard evaluations and that treatment effects probably vary substantially across demographic groups.

In summary, interventions to enhance job skills and employment have not been particularly effective. Contrasting these outcomes with the early childhood interventions has led some to argue that current subsidies for schooling and job training for teenagers and adults are too high (Heckman, 2000). However, the assessments of various programs are not standardized and it is possible that some of the most important benefits (e.g., health, reductions

in criminal activities) of the adult interventions were not included in many evaluations. Although there is good evidence that investments in young children *do* pay off, it need not imply that investments in adolescents and adults do *not* pay off.

Social Policies to Change SEP

While previous sections have focused on individual interventions, broad-based social policies offer perhaps the best avenue for improving outcomes for a large portion of the population. Education is certainly amenable to legal policy changes. In the United States, early 20th century increases in state mandatory schooling laws or regulations on child labor laws apparently substantially increased schooling (Lleras-Muney, 2002). Mandatory schooling changes in Britain, Northern Ireland, and Sweden were also effective (Oreopoulos, 2006). In Sweden, a careful evaluation of these changes suggested that the increases in schooling translated into improved earnings for high ability children whose fathers had manual occupations (Meghir & Palme, 2005). As discussed earlier, United States changes in educational policies have been linked to improved cognitive test scores (Glymour et al., 2008) and reduced mortality rates (Lleras-Muney, 2005) for birth cohorts affected.

There are many government programs and policies designed to increase income among the poor and near-poor. These programs are typically targeted at families, thus they affect adults and children simultaneously. Welfare and other transfer programs directly increase income. The Earned Income Tax Credit (EITC) offers incentives for employment (Ellwood, 2000) and increases income through an annual tax credit. Temporary Assistance for Needy Families (TANF) is a cash assistance program. Other government programs provide noncash benefits such as food, housing, and health care, which, by reducing household expenditures, has a positive effect on money available for household needs. These programs can increase income and lift families out of poverty. Plotnick estimates a 38% reduction in the child poverty rate in 1995 due to such government programs (1997).

Poverty reduction policies aimed at direct distributions, such as AFDC, EITC, and Social Security have generally been successful at remediating poverty at several points in the life course; however, there have been few evaluations of these policies' health consequences. The health consequences of financial receipts may differ based on how the money is delivered (i.e., lump sums versus slow payouts) and anticipated money versus unanticipated lotteries (Strully, Rehkopf, & Xuan, 2008). This important possibility has received little attention in epidemiologic research.

PRIORITY AREAS FOR FUTURE RESEARCH

This review highlights important gaps in the current literature on life-course epidemiology. Current research in life-course epidemiology is hobbled by two methodologic difficulties: inadequate measurement and few applications of randomized or quasirandomized trials. Improved measures of childhood SEP—with greater specificity of the exposure and the timing—is a key domain for future progress in life-course epidemiology.

Some of the most important sources of evidence of the effects of social conditions on health emerge from social interventions implemented with nonhealth related goals. These include interventions to change socioeconomic conditions and labor market outcomes in which health was either minimally addressed or entirely omitted. These interventions could be reexamined to assess their health consequences.

There are also important substantive challenges that must be addressed in life-course research. Our understanding of strategies for modifying neurologic structure and functioning in adults is also a troubling gap in the current research. This suggests the importance of implementing cognitive trials for adults, potentially with small samples to permit testing a broader range of interventions and using quasirandomized designs such as lagged entry.

Although heterogeneous treatment effects are very plausible—current exposures may have different consequences for people with different life-course histories—evidence to date is limited. Persistent and unexplained inequalities (e.g., excess stroke rates in the southeastern United States and elevated chronic disease rates in the United States compared to European countries) may reflect the much harsher consequences of current risk factors on a background of heightened vulnerability incurred in childhood.

CONCLUSIONS AND IMPLICATIONS

A primary implication of life course epidemiology is that what we do now for young children can payoff for elderly people decades in the future, and for many such investments the payoffs will be greater if investments are made in early childhood. We are now very likely reaping the health benefits of major social investments made in earlier decades of the 20th century, when we put high school within reach of the majority of adolescents, implemented much stronger social safety nets for working adults, and improved occupational conditions (Crimmins & Saito, 2001). The evidence in support of early childhood investments is large (Knudsen, Heckman, Cameron, & Shonkoff, 2006). Although this calls into question the justification for a policy environment in which infants and children are the demographic groups most likely to live in

poverty, it does not imply that investments in older people are useless. The evidence suggests that social or educational interventions in adulthood are both feasible and can have important health benefits.

With respect to the etiology of CVD, a cumulative model probably best describes the translation of social disadvantage into biological risk. There are likely several etiologic pathways through which SEP influences cardiovascular health; behavioral pathways may follow etiologic models that are distinct from those for stress-related pathways. This suggests there are many potential avenues for intervention and interventions. Even late-life interventions may have benefits, if they target the right pathways.

The etiology of cognitive impairment may follow a similar model but at this point it is difficult to establish. We believe the evidence is most consistent with the idea that neuroplasticity is greatest in childhood (Knudsen et al., 2006), but cognitive activities and training alter the brain beneficially well into old age. Such training is likely to offset the harm induced by age-related diseases and ultimately reduce disability. Brains can learn new skills and recover and compensate from injuries. Even adult brains respond to environmental conditions and cognitive challenges. Current understanding of adult neuroplasticity is limited, with greater depth in a few isolated topics (e.g., motor skills). As a result, the best approaches to intervention are not obvious. The systems are complex and interlocking and it is difficult to predict consequences of interventions. It seems clear that we can change specific cognitive skills, and these specific changes may justify the intervention even if they do not translate well to other skills.

Though social trajectories show significant stickiness, there is sufficient instability to indicate that changes can be made. Even in the absence of organized interventions, people move up and down the socioeconomic ladder, suggesting that organized interventions or social policies should be able to change exposures. The socioeconomic position seems to be especially dynamic around certain major life events. These moments set the stage for future trajectories, and we might ideally focus on such moments in intervention or policies designed to protect SEP.

We must consider not only the benefits of good policies, but also the potential deleterious effects of social interventions and policies. Many social policies may have negative consequences for financial security or cognitive environments of the elderly. Given the evidence on environmental cognitive demands and cognitive development, removing environmental complexity and educational opportunities from the daily lives of the elderly is likely to have negative consequences for their cognitive health. Retirement policies, housing policies, and the organization and targeting of public education

systems should be considered in light of these concerns. Do these institutions serve elderly people or do they foster isolation, environmental constraints and simplification, and loss of social roles for the elderly? Elderly who have had low SEP throughout their lives may be the most vulnerable. Because of this, many policies serve to maintain and stabilize lifelong trajectories of disadvantage: individuals deprived schooling in childhood also have limited access to workplace training or environmental stimulation, and ultimately are most likely to be isolated from cognitive challenges and engagement in old age.

Much health research literature emphasizes the consequences of small, manipulatable exposures. In efforts to translate life-course epidemiology findings to improve population health, however, individual level interventions are likely to be only a small component of a successful response. The broad-sweeping social changes in the past century demonstrate that contextual factors can entirely reshape the population distribution of socioeconomic conditions, including financial and educational opportunities. For example, key social trends reflected in current elderly cohorts include dramatic differences in the levels and content of education, changes in the nature of work and the demographics of the work force, and changes in experiences of poverty and the gender patterns of financial insecurity. These changes are likely to have consequences that manifest over the coming decades in many domains of population health.

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CHAPTER 3

Biobehavioral Aspects on Late-Life Morbidities

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Advances in the study of aging have benefited from the ability to acquire reliable indicators of biological functioning. Increases in the efficiency and subsequent reductions in the cost of processing bioassays have made available biomarker data to researchers interested in not only the biology of aging but also behavioral characteristics of late-life developmental processes. These new affordable tools, when used properly, offer the capability to disentangle the multiple, complicated contributors to health and disease. These tools are also promoting multi- and transdisciplinary investigations that require revised nomenclature to describe the research being undertaken. The term "biobehavioral" is quickly becoming a popular manner of characterizing this broad area of research encompassing biological underpinnings of behavior or behavioral implications for biological phenotypes. This broad conceptualization reflects the interest in providing a deeper level of understanding of how a complex phenomenon like aging can be understood in an ecologically valid, multivariate fashion. Biobehavioral inquiries are now a widely accepted approach used to improve understanding of age-related changes in health and disease in late life. In this chapter we provide an introduction, as well as some examples of this new and unique field of inquiry in the areas of stress, cognition, and physical health morbidities.

This new conceptualization is akin to Chaos theory, which postulates that behavior of dynamical systems may appear to be random. What is clearer

now than ever before is that systems influence other systems in complicated and sometimes unforeseen fashions. Among most scientists, there is general consensus that aging is a complex process or set of processes, involving many causes and subsequent consequences (McClearn, 1997). These multiple inputs and outcomes for both behavioral and biological systems produce variability that may also change with age (McClearn, 1997). The move from reductionistic approaches to studying elements in complex systems to examinations that encompass more holistic approaches to understanding multifaceted relationships should provide improved clarity about how biology and behavior work in concert to affect health statuses. Fundamentally, we are suggesting that a biobehavioral perspective is the most advantageous approach to date for understanding challenges involving the study of late-life morbidities (e.g., cardiovascular diseases, diabetes, arthritis, COPD, etc.) and moreover, to understanding how disparities across racial–ethnic as well as socioeconomic groups arise. Exploring these relationships is needed to ultimately establish public health intervention programs and reduce health disparities for all.

An important element in biobehavioral investigations is how the variables of interest vary by and change with age. Age is inextricably linked to health and the incidence of most indicators of chronic conditions and health problems increase with age. Whether increasing age offers the potential for exposure to environmental contributions to risk factors (e.g., additive effects of stress on blood pressure; Matthews et al., 2004), or that increasing age works to modify age-associated health conditions (vascular thickening and stiffness; Marin, 1995; Scuteri et al., 2004), it is clear that age impacts health. Still unclear is how risk factors, health conditions, and the use of health care interact with age during adult development, particularly for minorities who have shortened life spans due largely to early and more severe rates and courses of chronic health conditions (Arias, 2004, 2007). In addition, it is still unclear if the interrelationship among these variables change as a function of how race and ethnic minorities perceive health and health maintenance; and whether these perceptions change with age and experiences with health problems. While the precipitating factors underlying health disparities are undoubtedly social, genetic, physiological, biological, and behavioral factors complicate understanding how these disparities occur and are modified with advancing age.

An excellent example of the interaction of genetic, physiological, and psychosocial factors that serve as underpinnings to, and sources of variance in, aging is recent work on samples of patients with the genetic disorder sickle cell disease (SCD). Over the past 10 years there has been a relative explosion of studies that document psychosocial factors that influence SCD outcomes

(Edwards et al., 2005, 2006; Harrison et al., 2005; McDougald et al., 2009; O'Connell-Edwards et al., 2009; Pells et al., 2005, 2007). More specifically, these groundbreaking studies have demonstrated that a range of biobehavioral predictor variables account for variance in clinical outcomes like pain, depression, hostility, health care utilization, and coping. Recent biobehavioral explorations have discovered, for example, a complicated set of biological and psychosocial factors that contribute to an aging threshold across which individuals with and without illness are likely to retain physical and psychological functioning well into their later years (Feliu et al., under review). Patients with SCD who survive without significant illness past the age of 35 are likely to live past the age of 50 without significant morbidities. This has been interpreted as one of multiple instances where a biobehavioral model is most efficient in explaining variance in clinical outcomes among a complicated patient population.

There is ample information suggesting that differences in environmental factors between ethnic groups account for variance in disparities in health status (Macera, Armstead, & Anderson, 2000). Much of the previous research has focused on the behaviors and social structures that produce differences in health and disease across ethnic groups. Recent research on the multifactorial nature of risks for disease processes that demonstrate genetic and environmental influences are both important, is perhaps our best indicator that science must avoid a reductionistic view (see Whitfield, Brandon, & Wiggins, 2002). Sickle cell is just one example of a genetically determined disease but modified by the environment. The role of genetics in the origin of racial health disparities is receiving growing attention but has been susceptible to considerable misinterpretation (Whitfield, 2005). The misinterpretation of genetically informative samples should not deter this line of inquiry. Instead, scientifically sound studies examining biobehavioral relationships, such as studying the health of minority families with older family members (Burton & Whitfield, 2006), will provide an excellent avenue for advancing the science of aging particularly as it relates to health disparities. In this chapter, we are not offering genetic factors as a primary causal operator in health disparities, but we do believe that they are involved in a myriad of risk factors for disease that evince as health disparities. The following examples bring to bear not only genetics but also physiology and behavior in this discussion of biobehavioral perspectives on morbidity.

PERCEIVED STRESS AND HEALTH

Deaths associated with cardiovascular disease arise from a myriad of risk factors that include but are not limited to: elevated blood pressure, cigarette

smoking, hypercholesterolimia, excess body weight, sedentary lifestyle, and diabetes, all of which are influenced to varying degrees by behavioral factors (e.g., Winkleby, Kraemer, Ahn, & Varady, 1998). Many of the chronic conditions that account for mortality and morbidity differentials experienced by African Americans can be explained in part by behavioral factors (Winkleby et al., 1998). Chronic stress is one of the primary behavioral culprits that significantly contribute to the health disparities experienced by African Americans, particularly those related to cardiovascular mortality. For example, heart disease continues to be the leading cause of death in the United States (e.g., Peters, Kochanek, & Murphy, 1998) and is a major contributor to the gap in life expectancy (U.S. DHHS, 1995a, 1995b). Trends suggest that while heart disease is decreasing among White men, it may be increasing in African American men (Hames & Greenlund, 1996). Similarly, African Americans experience higher age-adjusted morbidity and mortality rates than Whites not only for coronary heart disease but also stroke (NHLBI, 1985). Previous research indicates that perceived stress is associated with increased blood pressure, which is a major risk factor for heart disease (Suter, Maire, Holtz, & Vetter, 1997).

There are a few studies that have examined the impact of more global measures of stress on health. For instance, Suter and colleagues (1997) examined the relationship between self-perceived stress and blood pressure in a sample of 1666 Swiss volunteers. Measures included self-rated health, family health history, overall stress, BMI, and two blood pressure measures. The results of the regression analysis indicated that age, measures of body fat, self-rated health, and perceived stress were significant predictors of systolic blood pressure (Suter et al., 1997). It is interesting to note that perceived stress was inversely related to systolic blood pressure. In addition, those with high normal blood pressure (SBP of 130–139 or DBP of 85–89) had significantly lower stress scores than normotensive participants (Suter et al., 1997).

In a more recent study, Heslop and colleagues (2001) examined the relationship between measures of perceived stress and coronary risk factors. This study focused on the impact of socioeconomic position in determining the impact of stress on risk factors (Heslop et al., 2001). The analysis was performed on secondary data collected from a sample of 6,832 men and women from various working classes in Britain. Coronary risk factor measures included blood pressure, plasma cholesterol level, BMI, FEV, physical activity level, alcohol consumption, and smoking. Perceived stress was measured by the Reeder stress inventory, a five item Likert-like scale designed to measure global daily perceived strain (Heslop et al., 2001). The results of the study show an inverse relationship of perceived stress with diastolic blood

pressure, BMI, FEV, physical activity. Perceived stress scores, however, were significantly and positively associated with plasma cholesterol, smoking, and alcohol consumption (Heslop et al., 2001).

The literature on the relationship between psychosocial stress and blood pressure is somewhat variable and underdeveloped (Suter et al., 1997). Although there is evidence that specific environmentally mediated psychosocial factors and stressors such as education and racism are associated with blood pressure elevations among African Americans (e.g., Wang, Chen, & Edwards, 2006), data concerning the relationship between perceived stress and blood pressure or cardiovascular risk among African Americans are limited (Adams, Aubert, & Clark, 1999). One exception is the work of Dressler (1990) who found that age, BMI, lifestyle incongruity, and chronic stressors were significant predictors of blood pressure. There were also significant interactive effects for age, lifestyle incongruity, and chronic stressors for blood pressure (Dressler, 1990). For instance, in the total sample, lifestyle incongruity and chronic stressors were significant predictors of systolic and diastolic blood pressure for older (40–55 years), but not younger (25–39 years) participants. Hypertensive status also showed an interactive effect with age, chronic stressors, and lifestyle incongruity. For younger hypertensives, compared to normotensives, higher blood pressure was associated with lower socioeconomic status and fewer chronic stressors. However, for older participants lifestyle incongruity was associated with higher blood pressure values regardless of hypertensive status (Dressler, 1990).

The author interpreted the results as supportive of the notion that increased lifestyle incongruity and concomitant stress result in increased blood pressure values for older participants (Dressler, 1990). Furthermore, the results also revealed that age is an important factor affecting the relationship between indicators of stress and blood pressure among African Americans. While the results reveal an increase in blood pressure associated with higher levels of lifestyle incongruity, the study failed to explicitly address perceptions of stress in the sample. In addition, the study implicitly assessed the stress associated with occupation and income, which are relatively specific stressors. In another study, Adams and colleagues examined the relationship between a global measure of perceived stress, John Henryism, education, and blood pressure among African Americans. The results of the analyses indicated that perceived stress was negatively associated with systolic blood pressure in this population (Adams et al., 1999).

A study by LaViest, Sellers, and Neighbors (2001), hypothesized that an external attributional orientation (which we refer to as system blaming) as opposed to an internal attributional orientation (which we refer to as

self-blaming) would be related to greater survival (as measured at 13-year survival interval) when individuals are exposed to racism. LaViest and his colleagues found that African Americans with a system-blaming orientation who reported experiencing racism were more likely to survive the 13-year follow-up period, compared to self-blamers who did not perceive themselves to have been exposed to racism. Their findings suggest that the attribution of negative events to external factors, such as systemic societal racism, rather than to individual characteristics, may be adaptive and protective of health status.

In summary, the results of these studies indicate a definite association between blood pressure and measures of stress. Interestingly, several of the studies cited above show an inverse relationship between these factors, with lower reported levels of stress being associated with increased blood pressure values. However, these studies also have several limitations. For example, most of these studies have no or very few African American participants, with the exception of Adams and colleagues (1999) and Dressler (1990). As a result, there is still little known about the relationship between measures of perceived stress and blood pressure in this population. Blood pressure as a dependent measure is a useful indicator to provide a noninvasive way to assess cardiovascular functioning. It is also clear that context puts behavior in perspective and has significant impact on the results of examinations of stress and health. The impact of stress on health has many examples. Here, we provide just a few of the more obvious and studied intersections including diabetes, blood pressure, and memory.

STRESS AND DIABETES

Distress and stress have been found to impact cardiovascular functioning as well as insulin resistance. A social gradient in diabetes risk has been well documented (e.g., McNeil et al., 2007; Steenland, Henley, & Thun, 2002). Chronic stress has been shown to be one of the central factors underlying this gradient (Björntorp, Holm, & Rosmond, 1999; McNeil et al., 2007; Mooy, de Vries, Grootenhuis, Bouter, & Heine, 2000). Stress is a potential contributor to chronic hyperglycemia in diabetes and has long been shown to have major effects on metabolic activity. The stimulation of various hormones can occur with stress and produce elevated blood glucose levels. Although this is of adaptive importance in a healthy organism (Surwit, Schneider, & Feinglos, 1992), in diabetes, as a result of the relative or absolute lack of insulin, stress-induced increases in glucose cannot be metabolized properly. Furthermore, regulation of these stress hormones may be abnormal in diabetes.

Björntorp posed a theory on the biological plausibility of this stress–diabetes connection. This theory suggests that defeatist or helplessness response to perceived stress leads to an activation of the hypothalamo-pituitary-adrenal axis (Björntorp, 1991, 1997). This creates a cascading effect that produces endocrine abnormalities, including high cortisol and low sex steroid levels that antagonize the actions of insulin. In addition, this hormonal imbalance causes visceral adiposity, which plays an important role in diabetes and cardiovascular disease by contributing to the development of insulin resistance (Mooy et al., 2000). Mooy and colleagues also found that stressful events were weakly positively associated with waist-to-hip ratio (WHR); a well-known factor in CVD risk and associated with measurements of BMI.

There is growing evidence that at least part of the link between these psychosocial risk factors and CVD and diabetes may operate through the metabolic syndrome (Vitaliano, 2002), a clustering of several CVD risk factors including: (a) abdominal obesity, (b) hypertriglyceridemia, (c) low high-density lipoprotein (HDL) cholesterol, (d) hypertension, and (e) hyperglycemia. In the elderly population, psychosocial risk factors such as stress are associated with a higher prevalence of the metabolic syndrome (Vogelzangs et al., 2007). This work suggests that exceptional survivors may be better at managing psychosocial risk factors such as stress, which in turn improves the metabolic profile.

STRESS AND MEMORY

Stress, anxiety, physical exertion, pain, and trauma have the potential to activate the hypothalamic pituitary adrenal (HPA) axis. Cortisol, a neurohormone that assists recovery following stressful periods, is released shortly after exposure to stress (e.g., Berdanier, 1987). The HPA axis activation and cortisol are often accompanied by activation of other neurohormonal systems. Thus, physical and psychological stressors have the potential to activate the neurohormonal system through neuronal mechanisms involving the HPA axis (American Medical Association, 1995).

Cortisol is secreted from the zona fasciculata and reticularis of the adrenal cortex and its serum levels are controlled by a negative feedback loop directly to the hypothalamus and anterior pituitary gland. As serum cortisol levels rise, adrenocorticotropic hormone (ACTH) production is inhibited in the pituitary gland and signals are sent to decrease hypothalamic corticotropin releasing hormone (CRH). As ACTH and subsequent cortisol levels rise in response to stress or pain, opioid peptides, including beta-endorphin, are also secreted both centrally and peripherally to counterbalance the initial neurohormonal responses (Vigorito & Ayres, 1987).

Several neurohormonal systems appear activated in conjunction with increased HPA axis activity. This cascade of neurohormonal processes in response to stress can alter memory and cognitive functioning (see Figure 3.1). Edwards, Carlson, McCubbin, & Schmitt (1995) investigated the effect of pain as a stressor on physiological functioning as well as on word list recall and recognition. Consistent with other research findings (Eysenck, 1976), they found that subjects' recognition performance was negatively affected by the administration of the pain stressor. Specifically, following a painful stimulus, subject's recognition performances decreased. They also found that heart rate and blood pressure increased proportionally following the administration of the painful stimulus. Although it could be argued that autonomic activation interfered with memory performance by occupying attention in a limited capacity system (Mandler, 1982), Edwards and colleagues (1995) argued against simple distraction effects.

The investigators employed a signal detection analysis that provided a measure of item discriminability during a fixed observation interval (Swets,

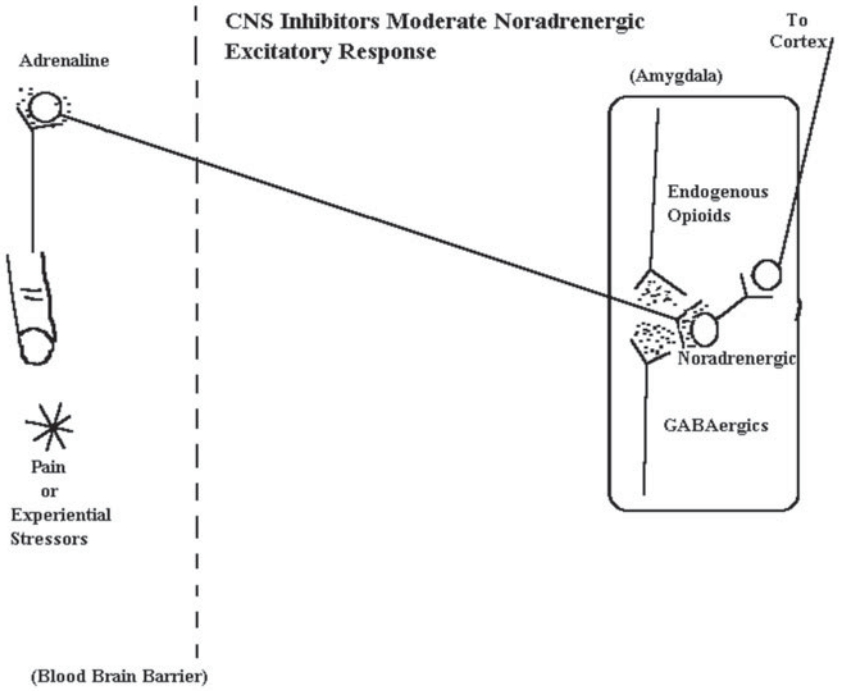

FIGURE 3.1 Peripheral and CNS system for cognition and BP.

1964). Results from this analyses revealed that subjects did not differentiate actual stimuli from cognitive distractors during a given time; they interpreted these data as exemplifying a meaningful difference in the amount of memory processing exhibited by subjects when stress was present. They also found an inverse relationship between the level of arousal and both recall and recognition performance. As subjects' blood pressures increased, recall and recognition performances decreased. They concluded as others had before them (e.g., Beese and Morley, 1993) that pain as a specific stressor negatively effected memory functioning by altering mental processing capacity.

Neurological structures also interact with neurohormones to potentially impact memory functioning. For example, the amygdala and surrounding limbic system structures mediate the acquisition and retention of information (Introini-Collison, Miyazaki, & McGaugh, 1991; Pinheiro & Wright, 1991). Amygdaloid lesions have been found to block the effects of gamma aminobutyric acid (GABA), cholinergic, and opioidergic influences on memory processes, suggesting that these neurohormones interact at amygdaloid sites (Dalmaz, Introini-Collison, & McGaugh, 1993; McGaugh et al., 1993). Other evidence indicates that the perirhinal cortex, hippocampal formations, and other related limbic system structures contribute significantly to memory functioning (Alvarez, Zola-Morgan, & Squire, 1995; Murray, 1992). From these observations, many researchers have concluded that the amygdala, hippocampus, and surrounding limbic system structures appear to be the site of many neuroendocrine interactions involved in the physiological, affective, and cognitive response to stressors (Murray, 1992). While these interactions are continually being better understood, the impact and/or relationship with advancing age are still not clear, nor are the possible differences across ethnic groups. The changes in memory for African Americans in later life have been examined (Whitfield et al., 2000), but how these structures may change and interact with neurohormones as a function of an aging brain and in the context of disproportional levels of stress is not clear.

HYPERTENSION AND COGNITION

There are several lines of research (studies examining self-rated health, cardiovascular disease, hypertension, and mortality) that suggest there is an important relationship between cognition and health. One chronic disease that appears to play a central role in lower cognitive functioning is hypertension (HTN). Research indicates that increases in BP and chronicity–duration of HTN are associated with lower levels of cognitive performance on several measures, particularly those assessing memory (e.g., Elias, D'Agostino,

Elias, Wolf, 1995). There have been inconsistencies with regard to the various neuropsychological domains most affected by high BP; for instance, some studies find relationships between memory or attention and BP, while others do not (Waldstein & Katzel, 2001). In addition, studies have found various patterns of neuropsychological and cognitive deficits, such that relative effects of BP on neuropsychological domains are difficult to discern because the effects of HTN on cognition are exacerbated by other factors, including ApoE4 allele status, obesity, and hyperglycemia (Waldstein & Katzel, 2001).

There are several possible explanations for the influence of HTN on cognitive decline. One explanation posits that chronic hypertension can cause structural changes of the cerebral vessel (i.e., thickening of vessel walls), increased vascular resistance, and impairment of cerebral autoregulation followed by a significant drop in BP. As a result, there is a lack of oxygen to the brain, which produces infarcts or white matter lesions and subsequent cognitive decline (e.g., van Dijk et al., 2004).

The prevalence of hypertension among African Americans is one of the highest in the world with a 2:1 prevalence ratio to European Americans (JNCIV, 1997; Whitfield, Weidner, Clark, & Anderson, 2002). However, few studies have investigated the association between BP and cognitive impairment among African Americans. The relative importance of an identifiable association between BP and cognitive decline in African Americans is often illustrated by differential rates of cognitive impairment in African Americans as compared to European Americans. For example, African Americans 71 to 80 years of age have been found to show mild cognitive impairment at twice the rate of European Americans and moderate to severe impairment at ages 76 to 85 and older was nearly three times higher in African Americans (Callahan, Hendrie, & Tierney, 1995). Investigations of health in elderly African Americans indicate cognitive functioning is a variable of significant importance in understanding aging. Investigating the contribution of specific chronic conditions as the link between cognition and physical health indices is an important step toward understanding cognitive aging in African Americans (Whitfield & Willis, 1998).

Given the greater prevalence, severity, and earlier onset of hypertension in African Americans and the role of hypertension as a predisposing factor for several conditions that frequently lead to mortality, cognitive declines associated with hypertension should exist and may be related to the higher rates of premature mortality observed in this population. Previous research in a small sample of African Americans has found that the greater the number of antihypertensive medications and the higher the diastolic BP, the poorer executive functioning and delayed verbal memory. In a recent larger study drawn from

a population-based sample of African American twins, there were significant relationships found between systolic BP and measures of perceptual speed, immediate memory, and cognitive impairment (Whitfield et al., 2008).

In summary, previous studies have shown that individuals with high BP perform more poorly on tasks involving memory, executive cognitive functions, and processing speed. However, virtually all previous studies have been conducted in non-Hispanic White samples, and the BP threshold associated with impaired cognitive function in African Americans is currently unknown. Epidemiologic studies have consistently shown that African Americans have a higher incidence of hypertension-related mortality, including a higher prevalence of end-stage renal disease at comparable ages. It is possible that cognitive impairment appears at lower BP levels in African Americans due to earlier (at younger ages) and more severe experience of these conditions. In addition, there is growing evidence of low availability and utilization of health care and previous findings of high chronic illness rates (hypertension, diabetes mellitus, and coronary heart disease) in this population (Ferraro & Farmer, 1996; Marquis & Long, 1996). These conditions may be principal contributors for increased susceptibility to the development of vascular dementia and hypothesized accelerated age-normative declines in cognition among African Americans (Whitfield, 2004).

PHYSICAL ACTIVITY AND HEALTH

Despite its widely recognized health benefits, only a third of U.S. adults meet national physical activity guidelines (Bennett & Wolin, 2006). Rates of physical inactivity are more pronounced among Blacks who, relative to Whites, consistently report lower levels of physical activity (particularly during leisure time) and are less likely to meet national physical activity guidelines. The determinants of physical activity among African Americans are multilevel in nature, likely emerging from individual, social, and environmental sources.

Among the many unanswered questions about physical activity among African Americans and other groups is whether it contributes to longevity (Singer, 2008). Previous research suggests that physical activity does contribute to important indicators of health status, such as reduced obesity (Flegal, Carroll, Ogden, & Johnson, 2002; Mokdad et al., 2003) and lower blood pressure (Chase, Sui, Lee, & Blair, 2009). Since these risk factors are key predictors of mortality, it stands to reason that physical activity is a key to maintaining longevity. We have even speculated that differentials in physical activity may be somehow related to gender differentials in mortality.

Emerging evidence suggests that genetic factors may also influence physical activity behaviors (e.g., Carlsson, Andersson, Lichtenstein, Michaelsson & Ahlbom, 2006; Joosen, Gielen, Vlietinck, & Westerterp, 2005). However, the precise magnitude of genetic variability in physical activity remains elusive (Frederiksen & Christensen, 2003). For example, using pooled data from seven European twin studies (13,676 MZ and 23,375 twin pairs), Stubbe and colleagues (2006) found that the median heritability of physical activity participation was 62% (with a range of 48%–71%). Little support was found for shared environmental influence on physical activity in the sample. In contrast, however, others have demonstrated that adopting a more restrictive physical activity measure could attenuate estimates of physical activity heritability (Eriksson, Rasmussen, & Tynelius, 2006). Still others have suggested that the environmental affects on physical activity might vary by key sociodemographic characteristics, thus reducing the observable genetic contribution (Lauderdale et al., 1997). Indeed, despite the growing evidence base, we are unaware of any studies that have been conducted among African American populations. This is an important omission, given African American's low physical activity prevalence rates and disproportionate exposure to environmental barriers to physical activity. If genes or shared environment impact physical activity in exceptional survivors, it would be evinced by similarities within families. This is a clear opportunity to elucidate contributing factors to health disparities.

CONCLUSIONS

In this chapter we have outlined a definition of the term "biobehavioral" a useful, but we believe often ill-defined concept and its relationship to health morbidity differentials among social groups in later life. As can be seen from our examples, it describes a useful interdisciplinary approach to understanding health and particularly health disparities among race–ethnic and socioeconomic groups. How researchers capitalize on the novel tools and approaches to understanding health and health disparities is limited only by scientific creativity and the ability to ask the right questions. Asking the right questions in part arise from proper training. With the unprecedented ability to access information within and across scientific disciplinary boundaries, it is imperative that beginning scientists be trained with greater breadth than ever before. Previously, programs such as biopsychology provided students with interdisciplinary training. Now, programs exist that are designed to provide breadth of training across not only disciplinary boundaries like biology and psychology, but also approaches to inquiries like epidemiology, experimental psychology,

and qualitative and mixed methods analyses. Building interdisciplinary (true molding of two or more disciplines) and multidisciplinary (bridging or crossing two or more fields of inquiry, but not necessary integrating) educational approaches to the study of health and aging and the outcomes of the intersection of each in the study of race–ethnic minority groups requires both breadth and depth in multiple disciplines. Striking the appropriate balance is a challenge and currently there is no single best model.

Differences in language–terminology are some of the most pressing challenges in biobehavioral research. We need investigators who are bilingual or multilingual in their knowledge of scientific terms (Choi & Pak, 2007). There is also the need for incentives for working beyond traditional boundaries and finding creative approaches and flexibility in members of research teams. The future looks very bright for these types of new programs. Historically, gerontology training programs emphasized interdisciplinarity in their curricula. The advances in assessing and employing biomarkers in the behavioral aspects of health science may make the training of gerontologists interested in addressing complex age-related phenotypes over the life course more appropriate than ever.

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SECTION II

EXAMPLES OF SPECIFIC HEALTH MORBIDITY INEQUALITIES IN LATE LIFE

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CHAPTER 4

Epidemiology of Diabetes and Cardiovascular Disease

The Emergence of Health Disparities Over the Life Span

Briana Mezuk

Cardiovascular disease (CVD) describes a group of health conditions that affect all parts of the cardiovascular system, including heart disease (atherosclerosis or hardening of the arteries, which can lead to angina pectoris or a heart attack) and stroke. Type 2 diabetes mellitus is an endocrine disorder characterized by hyperglycemia (high blood sugar) caused by insufficient production and/or action of insulin, a hormone produced by the pancreas that influences energy metabolism. Together these conditions constitute *cardiometabolic* disorders, a class of health problems with multifactorial, but related, causal origins. Many potentially modifiable individual-level risk factors for these conditions have been identified, including *health behaviors* such as smoking and physical inactivity, and *psychosocial factors* such as mental health and social support. However, it is clear that even if these health behaviors and psychosocial factors could be addressed, substantial inequalities in the burden of cardiometabolic conditions would still exist. Identifying risk factors at multiple spheres beyond the individual, including at the level of the workplace, the neighborhood, and political structures is necessary to understand determinants of health for the population, not just determinants of health for individuals that comprise the population.

The motivation for including a chapter on cardiometabolic conditions in this volume focused on health inequalities over the life span is exemplified by Figure 4.1. In the youngest age group, there is little difference between mortality rates comparing across African American and non-Hispanic White male and females. However, as age increases these lines begin to diverge, and by age 65 the annual mortality rate from cardiometabolic conditions for White males is nearly 60% lower than that of African American males (866.5 deaths per 100,000 versus 1485.6 deaths per 100,000). These mortality estimates reflect a combination of disease incidence, disease severity (particularly at the point of first identification by a clinician), and access to treatment, all of which in the United States are patterned by socioeconomic position and race–ethnicity. This figure also demonstrates the need for a developmental approach to understanding disparities in cardiometabolic disorders because while these inequalities generally emerge at younger ages, they clearly accelerate over the life course.

This chapter will focus on how patterning by chronological time, age, gender, race–ethnicity, and geography can further the understanding of the emergence of these disparities over the life course and inform the development

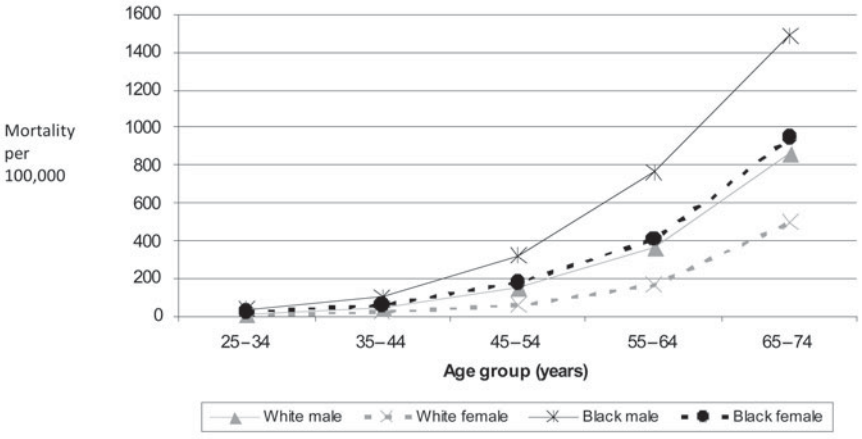

FIGURE 4.1 Disparities in mortality from cardiometabolic conditions per 100,000 persons, United States 2005.

Note. Cardiometabolic conditions include cardiovascular disease (ICD-10 100-19, Q20–Q28) and diabetes (E10-E14) in 2005. Data derived from the Centers for Disease Control and Prevention, National Center for Health Statistics, Data Warehouse LCWK5. (Available at http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/lcwk5_10.htm)

of interventions that are relevant and responsive to the needs of particular subgroups. The first section of the chapter will highlight specific patterns of inequalities in epidemiology of cardiometabolic disorders, particularly cases for which the sources of these inequalities are not well understood. The second section will briefly review what is known about the proximal causes of these conditions and discuss how these factors interact with the inequalities discussed earlier. Finally, the third section will describe how contextual factors drive the development of social disparities in cardiovascular disease and diabetes over the life span.

PATTERNING OF CARDIOMETABOLIC DISORDERS OVER THE LIFE SPAN

Chronological Time

It is now unequivocal that the increase in cardiovascular disease morbidity and mortality in Westernized countries from 1900 to the 1960s represented a true epidemic—that is, these conditions were occurring at a higher rate than in the past. Overall mortality from CVD peaked in 1968 in the United States and has declined for the past 50 years (National Institutes of Health, 2008). Much of this decline in mortality is attributable to decreased prevalence of smoking, dietary changes, and improved medical treatments. One notable exception to the overall trend in declining CVD mortality has been the rising incidence and mortality from heart failure over the past 30 years (National Institutes of Health, 2008).

The past 20 years has seen a significant increase in the prevalence of obesity and, as shown by Figure 4.2, a parallel increase in the incidence of type 2 diabetes (Fox et al., 2006). Diabetes is also a strong risk factor for cardiovascular disease morbidity and mortality (Centers for Disease Control and Prevention, 2008a). Thus, while some CVD risk factors have become less common over the past 50 years (e.g., smoking), others have continued to emerge (e.g., obesity and diabetes).

Although the mortality from cardiovascular disease has stabilized and declined from the mid-20th century peak, there is growing evidence that socioeconomic and racial–ethnic inequalities in the incidence and mortality from these conditions are growing. The rise in the incidence of type 2 diabetes has been particularly strong among racial–ethnic minority groups. Some poor health behaviors, particularly physical inactivity, have become more concentrated in socially disadvantaged groups (Harper & Lynch, 2007). Thus, while the overall rates of cardiovascular disease mortality in the United States have declined since the 1960s, there is evidence that socially patterned disparities in the burden

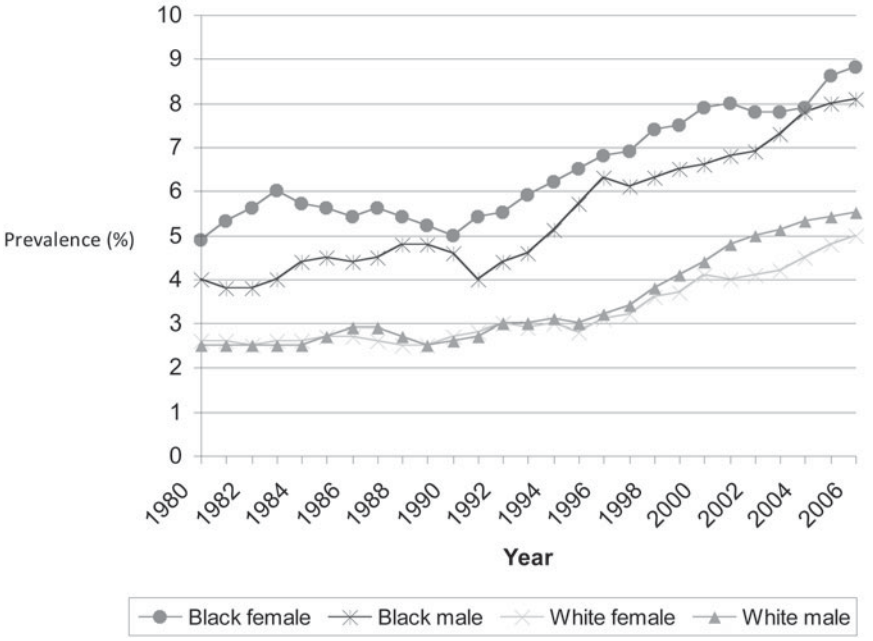

FIGURE 4.2 Age-adjusted prevalence of diabetes by race and sex, United States 1980–2006.

Note. Age-adjusted prevalence of diabetes stratified by race and sex in the United States from 1980 to 2006. Data derived from the Centers for Disease Control and Prevention, Diabetes Data and Trends. (Available at http://apps.nccd.cdc.gov/DDTSTRS/default.aspx).

of cardiovascular morbidity and mortality have widened (Singh & Siahpush, 2006). These findings also emphasize the point that the factors driving morbidity and mortality from diseases in the population overall may not be the same factors that are influencing the distribution of those conditions within the population.

Age

Cardiometabolic conditions are generally first detected by health care providers in middle to late adulthood, however it is believed that the pathogenic processes that underlie these conditions begin decades earlier. For example, studies have shown that several physiologic risk factors, including HDL and LDL cholesterol, blood pressure, and body mass index assessed in

adolescence are predictive of CVD risk in adulthood, even after accounting for concurrent risk factors (Raitakari et al., 2003). Blood pressure in childhood tracks with that in adulthood (Chen & Wang, 2008), suggesting that disparities in hypertension begin early in the life course. Finally, there is a growing body of research suggesting that exposures in utero, particularly low birth weight, are predictive of developing cardiometabolic disorders decades later, suggesting a type of "fetal programming" of metabolism that persists into adulthood (Barker, Osmond, Forsén, Kajantie, & Eriksson, 2005).

Whether age of onset of these disorders varies by sex and race–ethnicity depends on the particular condition being studied. The incidence of cardiovascular disease at all ages is higher for men relative to women, and the average age of a first heart attack is 65 for men and 70 for women (American Heart Association, 2009). The incidence of heart failure (reduced ability of the heart to pump) increases with age, but heart failure before age 50 is remarkably more common among African Americans relative to Whites (Bibbins-Domingo et al., 2009). Average age of diabetes onset is earlier for African Americans and Hispanics relative to non-Hispanic Whites (Centers for Disease Control and Prevention, 2008a). Age of diabetes onset influences the risk of functional impairment due to complications (e.g., peripheral nerve pain, blindness, amputation, kidney problems) because the risk of developing complications increases with longer duration (Hillier & Pedula, 2003). However, the symptoms of diabetes are nonspecific, and therefore many individuals are not clinically identified *until* they develop a complication, and these individuals are more likely to be racial–ethnic minorities and/or socioeconomically disadvantaged (Carter, Pugh, & Monterrosa, 1996).

Gender

While cardiovascular disease is the leading cause of death for both sexes, women of all racial–ethnic groups have lower incidence of these conditions relative to men until old age (generally after age 75). It is unclear what confers women this protection, however sex hormones (e.g., estrogen and testosterone) are thought to play a role because the risk of CVD for women begins to substantially increase during the menopausal transition (Colditz et al., 1987). Some cardiometabolic risk factors are sex-specific, for example, pregnancy. Events during pregnancy, such as older age at pregnancy, preterm birth, and obstetric complications (e.g., preeclampsia), appear to persistently influence risk of cardiovascular events in later life (Catov et al., 2007). There is relatively little variation in the prevalence of diagnosed type 2 diabetes by

sex overall, however within racial–ethnic groups there are gender differences. For non-Hispanic Whites, women have a marginally higher prevalence relative to men (6.1% to 5.8%), while for African American men have higher prevalence than women (14.9% to 13.1%, respectively), and for Mexican American women have substantially higher prevalence than men (14.2% to 11.3%, respectively; Centers for Disease Control and Prevention, 2008a). The inconsistency of the sex difference in diabetes risk is surprising given that one of the strongest risk factors for developing this condition, gestational diabetes, only occurs in women.

Race–Ethnicity

Cardiovascular disease and many of its behavioral risk factors are disproportionally common among racial–ethnic minorities, although a full tabulation of these inequalities is beyond the scope of this chapter. However, two cardiometabolic conditions that have become increasingly prevalent over the past 30 years—heart failure and type 2 diabetes—are discussed here in some detail. African Americans have significantly higher rates of heart failure relative to non-Hispanic Whites (i.e., incidence of 9.1 per 1,000 for African American men versus 6.0 per 1,000 for White men; American Heart Association, 2009; Yancy, 2005). Heart failure among African Americans tends to onset much earlier than among Whites (Bibbins-Domingo et al., 2009), and the case-fatality rate of this condition is substantially greater (Yancy, 2005). African Americans, Mexican Americans, and Native Americans have higher incidence and prevalence of type 2 diabetes relative to non-Hispanic Whites (Centers for Disease Control and Prevention, 2008a). The prevalence of undiagnosed diabetes has fallen over the past 20 years, particularly among racial–ethnic minorities, likely a result of increased awareness and screening (Smith, 2007). Likelihood of having detected diabetes is influenced by many factors, including educational attainment, insurance status, and access to care (Zhang et al., 2008). Some diabetes complications, such as end-stage renal disease, are over twice as common among African Americans relative to Whites, whereas others (e.g., retinopathy), have little variability by racial–ethnic group (Centers for Disease Control and Prevention, 2008a).

No discussion of disparities in health across groups identified by race–ethnicity would be complete without incorporating issues of socioeconomic status (SES). Socioeconomic status, whether indicated by educational attainment, personal income, household income, wealth, or occupation, covaries with race–ethnicity and health status in the United States (LaVeist, 2005). African Americans in particular are overrepresented in lower socioeconomic strata. While there is a well-defined gradient between higher socioeconomic

status and lower prevalence of cardiometabolic disorders and associated risk factors in the population as a whole (Kaplan & Keil, 1993), this pattern is not as consistent within racial–ethnic groups (Figures 4.3a–b). Also, different indicators of SES (e.g., income and education) may not show the same patterning with regard to CVD prevalence *within* racial–ethnic strata. The so-called Hispanic paradox (e.g., favorable mortality rates for Hispanics relative to Whites despite less favorable relative socioeconomic position) is perhaps the most widely known exemplar of this disaggregation of race, ethnicity, and socioeconomic status in the United States (Turra & Goldman, 2007). As these social groups become more heterogeneous (i.e., due to immigration), these patterns may change. There is also suggestive evidence that the relative contribution of socioeconomic status and race–ethnicity to inequalities in risk of cardiometabolic conditions may change over the life course as indicators of SES (e.g., income, wealth, education, etc.) become more or less relevant to health.

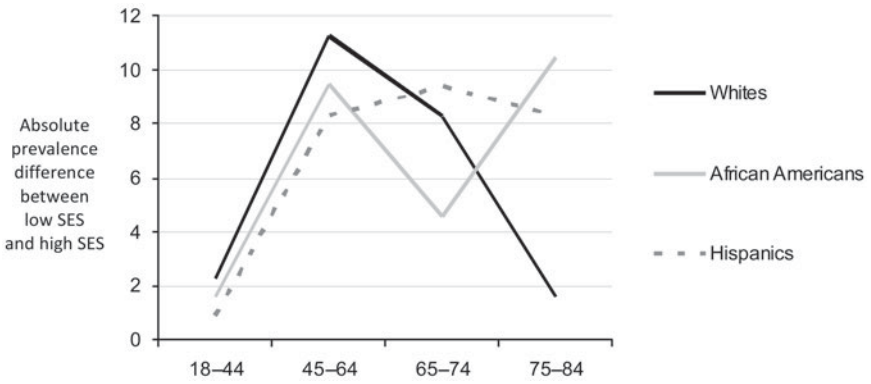

FIGURE 4.3A Absolute difference in prevalence of heart disease comparing low and high socioeconomic status groups over the life span by race–ethnicity.

Note. Absolute difference in prevalence of heart disease by socioeconomic status (Low SES–High SES) over the life span. A value of zero indicates no difference in prevalence between high and low SES levels of each racial–ethnic group. For Whites, the prevalence difference between low and high SES peaks in midlife (age 45–64) and then there is a trend toward a reduction in SES inequalities. In contrast, Hispanics have the smallest SES differential early in life, but this difference rises substantially in midlife and remains stable through older ages. For Blacks, the pattern is much more variable, with SES differences starting low, rising in midlife, falling in early later life, and then rising again in the oldest age group. Notably, Whites have the highest SES-differential in early life, but in the oldest age group they have the lowest. Data derived from the 2005 National Health Interview Survey.

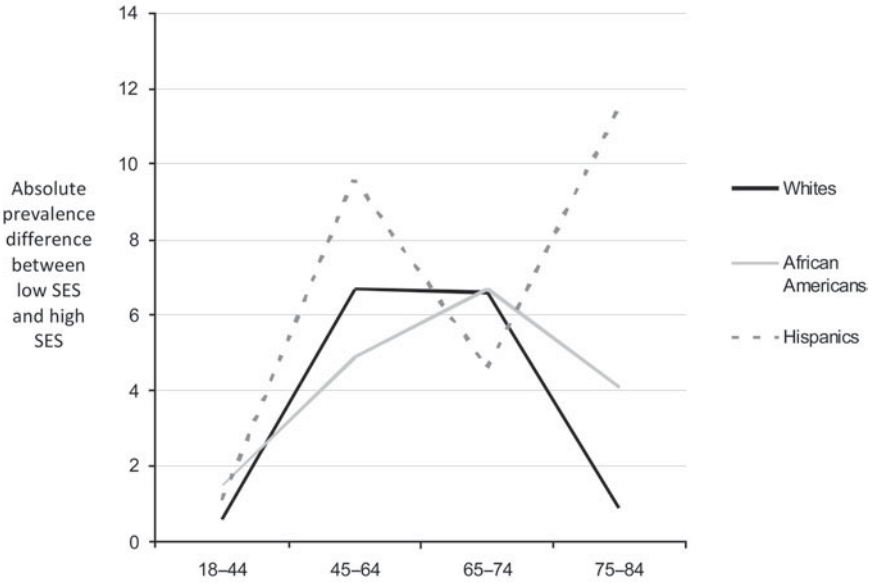

FIGURE 4.3B Absolute difference in prevalence of diabetes comparing low and high socioeconomic status groups over the life span by race–ethnicity.

Note. Absolute difference in prevalence of diabetes by socioeconomic status (Low SES–High SES) over the life span. A value of zero indicates no difference in prevalence between high and low SES levels of each racial–ethnic group. For Whites, the prevalence difference between low and high SES peaks in midlife (age 45–64) and then there is a trend toward a reduction in SES inequalities, consistent with what is observed for heart disease. Hispanics have the most variable pattern with peaks in both the 45–64 and 75–84 year age groups, in contrast to what is observed for heart disease. For Blacks, the SES-differential peaks in later life (65–74 years) and then falls slightly in the oldest age group. Notably, all groups have very similar SES-differentials early in life, but in the oldest age group there is a 10-point difference in difference in these differentials. Data derived from the 2005 National Health Interview Survey.

Geography

It is well established that the prevalence of cardiometabolic disorders varies by region in the United States. For example, the "stroke belt," which includes most of the Southeast plus Indiana, was first documented in 1940 and continues to persist (Cushman et al., 2008). Individuals who were born in this region have excess risk of stroke in adulthood even if they later migrate to a different part of the country, but those who were born elsewhere and moved into this region in adulthood do not have excess risk (Glymour, Avendaño, & Berkman, 2007), indicating that whatever environmental or behavioral exposures

lead to this higher rate are experienced or adopted early in the life course. The rising prevalence of obesity in the United States is also occurring in a nonuniform manner, with states in the Southeast and Midwest regions reaching a population prevalence of obesity ≥25% before states in the Southwest and Northeast regions (Centers for Disease Control and Prevention, 2008b). How geographic variation is translated into risk of cardiometabolic conditions (e.g., via cultural behavioral norms or state policies concerning issues such as taxation or education) is not yet clear.

PROXIMAL RISK FACTORS FOR CARDIOMETABOLIC DISORDERS

Although cardiovascular disease and diabetes have different physiological and clinical characteristics and treatments, they share many of the same risk factors and thus from an epidemiologic perspective can be discussed and understood as different, but related, phenotypic expressions of common exposures. For the purposes of this chapter, *proximal* risk factors are distinguished from *distal* factors in that the former are characterized by both robust evidence from etiologic observational studies and have biological causal mechanisms that have been fairly well characterized, generally at the individual level. Common proximal risk factors for cardiometabolic disorders fall into three categories: (1) physiologic abnormalities, (2) health behaviors, and (3) psychosocial characteristics. The first two sets of factors, physiologic indicators and health behaviors, constitute traditional risk factors for cardiometabolic disorders (i.e., smoking, high cholesterol). Psychosocial characteristics, such as mental health and social support, are generally not identified by organizations like the American Heart Association as being traditional etiologic risk factors for cardiometabolic conditions (although some, such as depression, are now recognized as influencing prognosis; Lichtman et al., 2008).

In contrast, *distal* risk factors are those for which there is fairly consistent, but not unequivocal, evidence from etiologic observational studies or only limited understanding of the biological causal mechanisms linking them to cardiometabolic outcomes. Distal risk factors generally refer to characteristics of the environmental context or ecology in which individuals live, and thus analysis of the influence of these constructs on health involves linking across multiple levels of the environment (e.g., individuals nested within families, families nested within neighborhoods, neighborhoods nested within geographic regions). These factors appear to have independent effects on risk of cardiometabolic conditions even after accounting for the proximal, individual-level risk factors, as discussed in the next section.

Physiology

Three categories of physiologic abnormalities influence cardiometabolic risk: (1) circulating levels of lipids and cholesterol, (2) inflammation, and (3) blood pressure. Levels of total cholesterol ≥200 mg/dL are considered elevated and using this cutoff approximately 45% of U.S. adults have high cholesterol (American Heart Association, 2009). However, the prevalence of high cholesterol varies by sex, race–ethnicity, and socioeconomic status (Mensah, Mokdad, Ford, Greenlund, & Croft, 2005). The C-reactive protein (CRP) is an acute-phase inflammatory marker that is influenced by degree of obesity (particularly centralized-obesity—having an "apple" shape) and is an indicator of systemic, low-grade inflammation. Recent evidence suggests that elevated CRP (>3 mg/L) is an independent risk factor for CVD events (Ridker, Hennekens, Buring, & Rifai, 2000) and development of type 2 diabetes (Lee et al., 2009). The CRP is higher among non-Hispanic Whites with lower educational attainment (Loucks et al., 2006) although it is unclear if this pattern holds for other racial–ethnic groups. Other inflammatory markers, including fibrinogen and interleukin-6, are also hypothesized to play a role in the pathogenesis of cardiometabolic conditions (Mensah et al., 2005; Papanicolaou, Wilder, Manolagas, & Chrousos, 1998). Blood pressure increases in response to stress (both psychological and physical, such as bouts of exercise), but sustained high blood pressure is harmful to the artery endothelium and kidney function. High blood pressure (systolic ≥140 mmHg and/or diastolic ≥90 mmHg) is both considered a form of cardiovascular disease itself and a risk factor for subsequent heart disease and stroke. The prevalence of hypertension in the United States has increased among all groups since the early 1990s and is patterned by race–ethnicity, with African Americans having the higher prevalence than most other groups (i.e., 41.4% versus 28.1% for non-Hispanic Whites; American Heart Association, 2009). While awareness and treatment of hypertension are high among African Americans, adequate control of high blood pressure remains relatively poor, likely influenced by educational attainment and health literacy (Hertz, Unger, Cornell, & Saunders, 2005).

Health Behaviors

Several health behaviors, including smoking tobacco, diets high in fat, cholesterol, and simple carbohydrates, physical inactivity, and being overweight or obese, are associated with risk of developing cardiovascular disease and type 2 diabetes. The prevalence of these poor health behaviors is patterned by age, sex, race–ethnicity, and socioeconomic status and has

changed over time. For example, in 1965 approximately 50% of U.S. adults smoked cigarettes, while currently only 24% do, a drop largely attributed to public health efforts (National Institutes of Health, 2008). In contrast, the prevalence of being overweight or obese has increased substantially in the past 25 years, and is particularly high among African American and Mexican American women (Centers for Disease Control and Prevention, 2008b). Obesity is also more common among lower socioeconomic strata (Mensah et al., 2005), which may be related to the relative lack of availability of fresh fruits and vegetables in socially disadvantaged neighborhoods (Rundle et al., 2009). The prevalence of engaging in these four key preventative health behaviors—not smoking, being physically active, eating five or more fruits or vegetables per day, and maintaining a healthy weight—is remarkably low. In 2000, only 3% of U.S. adults were engaging in all these behaviors, and as expected the likelihood of engaging in these behaviors was patterned by sex, race–ethnicity, education, and income (Reeves, 2005).

Psychosocial Characteristics

The notion that aspects of the psychological and social environment can influence health has been professed for centuries and the subject of empirical investigation for many decades. Three psychosocial characteristics—depression, personality traits, and social support and social isolation—have been extensively studied as predictors of cardiometabolic disease risk and mortality. Depression is a common disorder (lifetime prevalence: 16%), and incidence of this condition peaks in early to middle adulthood—one or two decades before clinical onset of cardiometabolic disorders (Kessler, Merikangas, & Wang, 2007). Numerous epidemiologic studies have demonstrated that depression is associated with risk of a wide range of cardiovascular events, including mortality (Van der Kooy et al., 2007). Depression is also associated with increased risk of developing type 2 diabetes (Mezuk, Eaton, Albrecht, & Golden, 2008), as well as elevated risk of developing diabetes complications (de Groot, Anderson, Freedland, Clouse, & Lustman, 2001). There are several pathways, both behavioral and physiological, that may mediate the association between depression and these conditions. Depression is associated with poor health behaviors that increase risk of cardiometabolic disorders, as described above (Grippo & Johnson, 2009). Depression is also associated with physiological abnormalities, including activation of the hypothalamic-pituitary-adrenal (HPA) axis, sympathoadrenal system, and centralized adiposity, which can induce insulin resistance and contribute to cardiovascular disease risk (Grippo & Johnson, 2009). Despite these findings, there is no compelling evidence that treating depression,

either with antidepressants or cognitive behavioral therapy, is associated with reduced risk of developing cardiometabolic conditions or reduced cardiovascular disease mortality (Berkman et al., 2003).

There is a growing body of evidence that personality influences risk of physical illness, likely by affecting the ways individuals perceive, appraise, and respond to their environment (Smith & MacKenzie, 2006). There is fairly consistent evidence that cynical hostility, a trait characterized by having a mistrustful, suspicious attitude toward events and others, is associated with subclinical cardiovascular disease (Everson-Rose et al., 2006), coronary heart disease (Chida & Steptoe, 2009), and cardiovascular mortality (Everson et al., 1997). There has been a relative dearth of studies on diabetes risk, although there is suggestive evidence that hostility may influence insulin resistance and glucose metabolism (Zhang et al., 2006). Other aspects of personality, including Type D personality, characterized by high emotional negativity combined with inhibited emotional expression, have also been associated with risk of cardiovascular disease (Sher, 2005).

Poor social integration and social isolation are associated with increased all-cause mortality, particularly from CVD and diabetes (Berkman & Syme, 1979). It is unclear whether this relationship is due to a *direct* influence of support on physiology, or if social support simply *buffers* the effect of negative experiences (e.g., exposure to chronic stress) on health (Cohen & Wills, 1985). The influence of social support on cardiometabolic morbidity and mortality may vary by sex and race–ethnicity. Many aspects of the receipt and provision of social support are gendered (i.e., women are more likely to experience the loss of a spouse), and changes in social relations over the life course likewise may differ for men and women (Ajrouch, Blandon, & Antonucci, 2005). Other facets of social life that are correlated with cardiovascular mortality, such as religiosity, also covary with gender and race–ethnicity (Levin & Chatters, 1998). Finally, the relationship between social support and cardiovascular health may vary over the life course as stressors, social roles, and social networks change.

Limitations of Proximal Risk Factor Epidemiology

The proximal risk factors discussed above explain a substantial amount of the variance in individual risk for cardiometabolic disease. However, research has shown that they do not, in and of themselves, completely explain the patterning of these conditions across groups within a given population, much less across populations. While these behaviors and psychosocial characteristics are disproportionately prevalent in lower socioeconomic strata (Harper & Lynch,

2007), even after accounting for this overrepresentation of risk factors socioeconomic disadvantage is still significantly associated with cardiometabolic morbidity and mortality (Kaplan & Keil, 1993; Lantz et al., 1998). Finally, it is likely that the determinants of health inequalities in the population are due to contextual or environmental factors that in turn *determine the patterning* of these more proximal risk factors (e.g., the notion of a fundamental cause; Link & Phelan, 1995).

DETERMINANTS OF HEALTH DISPARITIES OVER THE LIFE SPAN

Three contextual factors have been the subject of extensive empirical investigation as determinants of disparities in cardiometabolic disease: (1) neighborhood environments, (2) work characteristics and settings, and (3) social stressors. The relative influence of these contextual factors may change over the life course as individual characteristics change. For example, neighborhood characteristics, such as the proximity to grocery stores, may exert a stronger influence on health later in life as individuals develop functional limitations (i.e., stop driving due to declining health) than earlier in the life span.

Neighborhood Environment

In the United States, African American and non-Hispanic Whites are generally exposed to very different neighborhood contexts due to residential segregation and differentials in socioeconomic status, and in this way local environments may contribute to racial–ethnic disparities in health (Williams & Jackson, 2005). There are four established ways in which the local environment influences cardiometabolic disease risk: (1) hazards in the physical environment (e.g., air pollution), (2) characteristics of the built environment that influence behavior (e.g., presence or absence of sidewalks), (3) availability of commercial products (e.g., fresh fruits and vegetables, liquor stores), and (4) availability of health care services (e.g., pharmacies, outpatient clinics). Both acute and chronic exposure to particulate matter air pollution has been associated with cardiovascular disease morbidity and mortality (Brook, 2007). Characteristics of the built environment, such as resources that promote physical activity and greater availability of healthy food choices, are associated with lower prevalence of insulin resistance (Auchincloss, Diez Roux, Brown, Erdmann, & Bertoni, 2008). Differences in neighborhood context contribute between one-fifth to one-third of the difference in hypertension prevalence between African Americans and Whites (Thorpe, Brandon, &

LaVeist, 2008). Poor, minority-populated neighborhoods have higher levels of tobacco and alcohol advertising than suburban neighborhoods (LaVeist, Thorpe, Mance, & Jackson, 2007; LaVeist & Wallace, 2000). Finally, the health care services available in low income areas are less accessible and, in general, operated by lower-qualified providers (Williams & Jackson, 2005).

Work Environment

There is a large literature suggesting that exposure to work-related stressors, particularly job strain characterized by high demands and low decision latitude (i.e., limited control over the type or pace of work), is associated with blood pressure and increased risk of cardiovascular disease, stroke, and diabetes (Schnall, Langsbergis, & Baker, 1994). However, the majority of these studies have been cross-sectional and the evidence from prospective reports is mixed. It has been hypothesized that one source of the inconsistency from prospective reports is the reliance on samples of relatively young adults (generally <60 years old), which may be more resilient to the influence of job strain on blood pressure. There is evidence that the relationship between work-related stressors and hypertension risk strengthens with increased accumulation of exposure (Guimont et al., 2006), and that the association between work-related stressors and hypertension is most pronounced in lower socioeconomic strata (Wege et al., 2008). More recently, shift work has been identified as a risk factor for cardiometabolic disorders. Shift work has been associated with subclinical markers of atherosclerosis (Puttonen et al., 2009) and has been implicated in risk of diabetes (Kroenke et al., 2007). Experiences of discrimination, including in the workplace, have also been associated with risk of hypertension and cardiometabolic conditions (Nazroo, 2003). Discrimination also influences ability to obtain employment, and unemployment is associated with poor health behaviors that increase risk of cardiometabolic disorders (Khlat, Sermet, & Le Pape, 2004).

Chronic Social Stress

Social stress includes both *psychosocial and physical demands* that tax or exceed available resources, as well as *structural conditions* that place constraints on opportunities, activities, and individual choice (Wheaton, 1999). These social environmental stressors may influence cardiovascular risk directly through accumulated physiologic insults as hypothesized by such models as allostatic load (McEwen, 1998). For example, studies in both animals and humans have demonstrated that repeated or persistent exposure to stress,

whether physical (e.g., restraint) or psychosocial (e.g., lower or marginalized social status), is associated with the dysregulation of neuroendocrine feedback loops governing the hypothalamic-pituitary-adrenal (HPA) axis (Crimmins, Kim, & Seeman, 2009). The HPA axis stimulates production of proinflammatory cytokines such as interleukin-6 (IL-6), which in turn stimulate C-reactive protein (CRP) (Papanicolaou et al., 1998). Repeated activation of the HPA axis, as in the case of chronically stressful living conditions, can lead to overstimulation and stunted negative-feedback regulation, subsequently dysregulating production of IL-6 and CRP. Chronically elevated CRP can be a consequence of this biological cascade, and as discussed above high levels of CRP have been associated with increased risk of cardiovascular disease and diabetes.

In addition, the ways in which individuals and groups cope with stressors influences health. The availability of resources (e.g., economic, material, personal, social, and cultural), both determines the threshold at which an event or social structure *becomes* a stressor (that is, when a demand exceeds the ability to respond) and the manner in which that stressor is addressed. These resources vary with racial–ethnic background, culture, and socioeconomic status, and change over the life course (Elder, George, & Shanahan, 1996). What is defined as an adaptive coping measure (i.e., problem solving versus emotional) is influenced by situational factors. Many behaviors that influence CVD risk, such as smoking, excessive alcohol use, and overconsumption of high fat foods, are differentially used as coping strategies by socially disadvantaged groups (Krueger & Chang, 2008). The preferential use of coping behaviors that increase cardiometabolic risk (i.e., smoking) rather than those that decrease this risk (i.e., physical activity) is encouraged by structural factors such as the local environment. In this way the emergence of disparities in cardiometabolic disorders through the intersection of stressful circumstances and coping strategies involves links across both individual and environmental characteristics.

CONCLUSION

Heart disease, stroke, and diabetes are the first, third, and sixth leading causes of death in the United States, respectively. While these conditions are among the major sources of mortality for all adults in industrialized nations, the burden is disproportionately high among socially disadvantaged groups. Disparities in the risk of developing diabetes and cardiovascular disease are present early in life and grow over the life span. Contextual factors influence the emergence of disparities in these conditions over the life course, although

the mechanisms by which these factors "get under the skin" remains unclear. Future research should focus on identifying the pathways linking environmental exposure to health risks, keeping in mind a life course framework, which acknowledges that contemporaneous factors may have less to do with current health than those from earlier in development. This approach also emphasizes the need to invest in prevention for cardiometabolic conditions at all ages, as many of the processes (both behavioral and physiological) that produce these disorders in later life begin decades earlier.

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CHAPTER 5

Genetic Influences on Disparities in Hypertension and Obesity in Late Life

Jacquelyn Y. Taylor

Hypertension is a chronic health condition that has become a common diagnosis among Americans, particularly African Americans. Hypertension is defined as repeated blood pressure readings that are consistently at or above 140/90 for adults and at the 95th percentile or greater for age and height among children (Chobanian et al., 2003; National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents, 2004). Research has shown that hypertension development can be influenced by both genetics and lifestyle behaviors (Taylor, Maddox, & Wu, 2009). Although a person's genetic predisposition may not be changed, their lifestyle behaviors may interact with genes to influence when, to what degree, and the severity of onset of disease (Taylor, Sun, Chu, Mosley, & Kardia, 2008). African Americans have been shown to have earlier onset and greater severity of hypertension (Rosamond et al., 2007). Lifestyle behaviors that include lack of physical activity, high fat and sodium food consumption, and resulting obesity all are negative habits that develop as early as childhood and have long-lasting effects on health throughout the life span. The diagnosis of hypertension and obesity has been steadily rising among both African American adults and children (Rosamond et al., 2007; Taylor, in press). Because hypertension is a multifaceted chronic disease that is approaching epidemic proportions among African Americans, this chapter will discuss the prevalence of hypertension and obesity, environmental lifestyle risks, genetic risks,

gene-environment interactions, related comorbidities, and research trends for reducing this disparate condition across the life span.

PREVALENCE OF HYPERTENSION AMONG AFRICAN AMERICANS

According to the Centers for Disease Control and Prevention, the percentage of African American adults diagnosed with hypertension was 37% and 42% among women, respectively, (CDC, Health United States-Table 70, 2007). Between NHANES III and NHANES 1999–2004, the age-standardized prevalence rate of hypertension increased in all racial–ethnic-by-gender subgroups, with a marked 14% increase in African American women (Cutler et al., 2008). African American women have the highest prevalence of hypertension of all demographic groups in the United States (American Heart Association [AHA], 2007; Nesbitt & Victor, 2004). African Americans are at higher risk for developing hypertension, have greater severity of health outcomes, and are diagnosed earlier than other ethnic groups in the United States. African Americans also experience a higher percentage of hypertension-related morbidity at an earlier age (Sile et al., 2007). In 2004, 41% of the 31,608 women who died from hypertension-related diseases were African American; in contrast, 15% were White women (AHA). Hypertension-related diseases include stroke, heart attack, and other cardiovascular compromise.

Because hypertension does not have visible symptoms, many people go undiagnosed until the occurrence of a cardiac event (e.g., heart attack, stroke, etc.). An estimated 16% of adults nationwide meet the clinical criteria for hypertension, but are undiagnosed, with African Americans comprising 25% of these undiagnosed cases (Diaz, Mainous, Koopman, & Geesey, 2004; Graham et al., 2006). The American Heart Association (2007; Rosamond et al., 2007) has indicated that cardiovascular disease is the primary cause of death among African Americans, with nearly 5 out of 10 African Americans developing cardiovascular disease.

Cardiovascular changes that lead to hypertension have become commonplace among African Americans, with more than 40% of African American women diagnosed with hypertension (Glover, Greenlund, Ayala, & Croft, 2005). These changes often begin in childhood. Obesity has been suggested as a leading cause of hypertension, with increases in the number of overweight and obese children associated with increases in the number of children diagnosed with essential hypertension (CDC, 2007; Din-Dzietham, Liu, Bielo, & Shamsa, 2007; Litwin et al., 2007). Between 1988 and 2002, the percentage of children with prehypertension increased from 8% to 10%, with an

increase diagnosis of hypertension rising from 3% to 4% (Din-Dzietham et al., 2007). This translates into approximately 410,150 additional children diagnosed with pre- or essential hypertension across the nation. These statistics signify an important public health concern as hypertensive children may become hypertensive adults who can develop cardiovascular disease and other comorbidities at younger ages.

ENVIRONMENTAL (LIFESTYLE) RISKS FOR HYPERTENSION

Although genetic markers have been identified as precursors for high blood pressure, effects of environmental factors on the phenotypic expression of these genes must be considered. Lifestyle behaviors can prevent or enhance expression of dormant genetic predispositions. In some cases, African Americans practice negative lifestyle behaviors (e.g., consuming foods with high caloric content that can lead to obesity and cardiovascular problems; Rosamond et al., 2007). Maintaining optimal weight is a health index of diet/physical activity/lifestyle choices that may affect gene expression of high blood pressure. Body mass index (BMI) is an applicable predictor of adiposity with a BMI at or greater than 25 considered overweight, and BMI 30 or greater indicative of obesity. The disease burden of being overweight and obese has been associated with chronic health conditions, such as hypertension, type 2 diabetes mellitus, gallbladder disease, and osteoarthritis among both overweight and obese men and women (Rosamond et al., 2007). In adults, hypertension was the most common overweight and obesity related health condition. In addition to hypertension, associated risk factors for cardiovascular disease observed in African American women include being overweight (80%) and obese (51%; Rosamond et al., 2007).

Obesity is a major risk factor in development of high blood pressure and is a preventable cause of death (second to smoking) among Americans. Obesity affects blood pressure by increasing the activity of the Renin/Angiotension System (RAS) that stimulates sympathetic nerve activity that increases constriction of afferent arterioles to the kidney. This condition can lead to increases in aldosterone (i.e., smaller amounts of sodium load to the kidney), activates the RAS, and increases sodium reabsorption capacity. The RAS regulates blood volume and influences blood pressure at the kidney level. Further enhanced RAS activity can lead to glomerulosclerosis that changes sympathetic tone and results in decreased elasticity of blood vessels. Lack of elasticity prevents blood vessels from dilating and causes them to become rigid, resulting in increased systolic blood pressure. The resulting rigidity is a

reason that many patients present with greater variability in systolic pressure in comparison with diastolic pressure readings. Although genetic factors that influence interindividual variation in BMI have been identified, an environmental component to BMI that contributes to high blood pressure remains to be explored. The BMI entails partial genetic makeup, but also represents an internal metabolic and physiological environment that plays a key role in the development of high blood pressure (Taylor et al., 2008).

In a study of age-standardized prevalence rates of hypertension by ethnicity and BMI for men from NHANES III and NHANES 1999–2004, an increase in BMI accounted for significant increases in hypertension among men (Cutler et al., 2008). After adjusting for BMI among women in the sample, the prevalence of hypertension increased, indicating that these increases could have been attributable to factors other than BMI (such as genetic influences; Cutler et al., 2008). A meta-analysis of 21 cohort studies found that weight effects on blood pressure accounted for approximately 45% of the risk for target organ damage of coronary heart disease independent of other risk factors (Bogers et al., 2007).

Obesity has been linked with salt sensitivity (impaired ability of the kidney to excrete sodium, which leads to an increase in blood pressure that promotes natriuresis to correct the sodium balance, which then results in hypertension) and hypertension among African Americans (Flack et al., 2002; Franco, Sanchez-Lozada, Bautista, Johnson, & Rodriguez-Iturbe, 2008; Taylor & Wu, 2009). Weight plays a role in blood pressure response to sodium intake, as well as excretion, especially for women who typically are more salt sensitive than men (Flack, 2003; Flack et al., 2002). Salt sensitivity has been associated with blood vessel constriction, which can lead to increases in blood pressure. Because obesity and salt intake are contributing factors to hypertension, the Dietary Approaches to Stop Hypertension (DASH) diet has been used as a successful treatment in lowering African American patients' blood pressure readings. The DASH diet, which includes fruits, vegetables, fiber, and low-fat dairy foods, reduced fats and sodium, increased potassium, as well as reasonable intakes of protein, has been found to reduce high blood pressure in Africans Americans (Douglas et al., 2003).

Obesity can lead to a cluster of symptoms, termed metabolic syndrome, that are risk factors for hypertension and resulting cardiovascular disease (AHA, 2005). In adults, these criteria consist of elevated triglycerides, BP, and fasting glucose; reduced high-density lipoprotein cholesterol (HDLC); and a larger waist circumference. The AHA maintains that individuals who meet at least three of the criteria are considered to have metabolic syndrome. Researchers involved in the Bogalusa Heart Study (Chen, Srinivasan, Li, Xu, & Berenson,

2007) modified these criteria to be applicable to children, using homeostasis model assessment of insulin resistance, mean arterial pressure, BMI, and triglyceride:HDLC ratio as the components. Following children into adulthood, Chen and colleagues found that obesity is a better determinate of metabolic syndrome than is insulin resistance. This finding is significant since 50% of obese children meet the criteria for a diagnosis of metabolic syndrome and are likely to become adults with metabolic syndrome and the associated morbidities. The prevalence of metabolic syndrome is 57% greater among African American women than among African American men (Ford, Giles, & Dietz, 2002).

Adipose tissue has been recognized to produce increased levels of circulating acute-phase proteins (proteins that increase or decrease based on inflammation) and adipokines (signaling proteins) in obese individuals that lead to low-grade inflammation, which is linked to insulin resistance and metabolic syndrome (Trayhurn & Wood, 2004). Adipokines associated with inflammation include tumor necrotizing factor-alpha, interleukin-1beta, interleukin-6, interleukin-8, interleukin-10, transforming growth factor-beta, and nerve factor, while the associated acute-phase proteins include plasminogen activator inhibitor-1, haptoglobin, and serum amyloid A. Adipose tissue surrounding vessels acts in two ways: first, the inflammation process results in macrophages and T-cells migrating from the adipose tissue into the vessels, damaging them; second, adipokines affect endothelial function by modulating nitric oxide and superoxide release, resulting in hypertension and insulin resistance (Guzik, Mangalat, & Korbut, 2006).

GENETIC PRECURSORS FOR HYPERTENSION AMONG AFRICAN AMERICANS

Although hypertension has been widely recognized as a polygenic trait, many family studies have investigated the hypothesis that high blood pressure may result from simple monogenic Mendelian aberrations, typically associated with dysregulation of renal sodium management. Genetic explanations for longitudinal changes in blood pressure have shown that mutations in *WnK4* and *WnK1* also may control blood pressure at the kidney level and can act as a regulator for NaCl and K+ balance in the body leading to hypertension (Lalioti et al., 2006). Perusse, Moll, and Sing (1991) suggested that allelic variation in a single gene may be associated with steeper increases in systolic blood pressure with age. Familial studies have also revealed that first-degree relatives of hypertensive individuals are 50% more likely to have high blood pressure than normotensive individuals.

Boerwinkle (2003) asserted that the presence of single gene polymorphisms on chromosome 2 increased the risk of African Americans developing high blood pressure, while Levy and colleagues (2000) suggested that genetic loci on chromosome 17 in a population-based study significantly influences blood pressure.

Hypertension is likely to result from complex interactions of many genes and multiple environmental factors (Boerwinkle, 2003). One study identified markers on chromosome 1 and 8 that have significant genetic interactions between obesity and high blood pressure in a small sample of African Americans (Kotchen et al., 2002). Another study that examined the 825T allele of the *GNB3* gene among Germans, Chinese, and Black Africans found that this allele was significantly associated with high blood pressure, although this association occurred only in individuals who were predominately overweight or obese. This finding indicated that high blood pressure may have been more a result of Westernized environmental lifestyle influences than genetic. Although obesity has been related to increases in blood pressure, genetic markers for obesity are not necessarily related to genetic risks for high blood pressure among African Americans (Onions et al., 1998). Conversely, Telgmann and colleagues (2007) determined that certain *SAH* alleles on chromosome 16 were significantly related to BMI in White hypertensives, but the mechanism of this obesity-related high blood pressure is unknown. Studies that examined gene-environment interactions of BMI and high blood pressure investigated different polymorphisms or genetic regions of interest making it difficult to reach definitive conclusions about whether the findings represent true or false positive associations. For example, the HyperGEN study (Gu et al., 2005) found a significant interaction between BMI and promoter polymorphisms in the angiotensinogen gene (AGT) that affects blood pressure in Whites and African Americans. In contrast, the Framingham Heart Study (Daley et al., 2003) found an association between polymorphisms in the chromosome 2p region based on BMI and gender. A study by Wessel et al. (2007) claimed that the level of fat in the body influences c-reactive protein and inflammation, signaling polymorphisms at the catecholaminergic/β-adrenergic pathway loci, thereby increasing blood pressure. Significant associations were found among BMI, blood pressure, and TGF-beta 1 in eastern Europeans and Gln27Glu & Thr164Ile polymorphisms in predominately biracial participants in Brazil (Pereira et al., 2003). A study based in the United States found no evidence of interaction between polymorphisms in a G-protein and BMI influencing high blood pressure among African Americans (Poston et al., 2002).

GENE-ENVIRONMENT INTERACTIONS FOR HYPERTENSION

Research has been inconclusive in determining roles of specific polymorphisms in hypertension (Lohmueller et al., 2006). African American children, especially males, generally have higher blood pressure even when not obese. Several genotypes have been associated with hypertension. A study of White and African American children examining the T235 allele of the angiotensinogen gene found that angiotensinogen levels were higher and the T235 allele more common in African American children (Bloem, Manatunga, Tewksbury, & Pratt, 1995). Four genes significantly related to renal salt absorption (CLCNKA, CLCNKB, BSND, NEDD4L) have indicated differences in phenotypes between populations (Sile et al., 2008). All genes, except BSND, differed in hypertension expression by 50% between African Americans and Whites (Sile et al., 2007). Some researchers (Baker et al., 2007) have demonstrated that the C-532T polymorphism of the angiotensinogen gene has a significant effect on arterial stiffness without necessarily resulting in higher blood pressure, while other researchers' meta-analysis (Sethi, Nordestgaard, & Tybjaerg-Hansen, 2003) concluded that the same polymorphism is indicative of hypertension risks, although all study participants were White.

Previous research has found that African American children with hypertensive mothers are more likely to develop hypertension at an earlier age (Strauss & Knight, 1999; Taylor, Chambers, Funnel, & Wu, 2008). Few studies have focused on the early gene environment risk for high blood pressure among untreated children and their hypertensive parents (Taylor, in press). However, a few studies have shown significant interactions between environmental lifestyle behaviors and single nucleotide polymorphisms on chromosome 2 resulting in increased blood pressure among African Americans beginning as early as childhood (Hunt et al., 2006; Taylor et al., 2009). Such studies provide new insights into combined effects of environmental and genetic risk factors that can contribute to high blood pressure among African Americans early in life. These risks can lead to hypertension health disparities across the life span.

COMORBIDITIES ASSOCIATED WITH GENETIC AND ENVIRONMENTAL RISKS FOR HYPERTENSION (TARGET ORGAN DAMAGE)

Among African American adults with hypertension, a significant association has been found between obesity and hypertension and the development of target organ damage, such as chronic kidney disease (CKD). In addition,

African Americans with target organ kidney damage are more likely than Whites to develop hypertension-end stage renal disease (ESRD), also called hypertensive nephropathy (HN; Kaperonis & Bakris, 2003).

Strong, continuous associations of blood pressure level with risks of target organ damage to the brain, heart, and kidneys make high blood pressure a major risk factor for stroke, heart attack, and kidney failure. In the United States, health care costs attributable to high blood pressure and associated target organ complications exceed $250 billion per year (Rosamond et al., 2007). Areas of target organ damage pronounced in hypertensive African Americans include cardiac: coronary artery disease, left ventricular hypertrophy; brain: stroke; renal: end-stage renal disease (ESRD). Hypertension-related target-organ complications are the consequences of chronic ischemia resulting from atherosclerosis of the conduit arteries delivering blood to the organ and arteriolosclerosis of the small muscular resistance arteries within the target organ.

African Americans may have a genetically based susceptibility for hypertension induced left ventricular hypertrophy and cardiac volume overload (Arnett et al., 2001). Stroke occurs in African Americans at a younger age and they also experience greater severity of morbidity and higher mortality rates for intracerebral hemorrhage and cerebral infarction. Compared with Whites of the same age, African Americans were at significantly increased risk for cerebral infarction and had higher postcerebral infarction mortality (Bian, Oddone, Samsa, Lipscomb, & Matchar, 2003). In addition, African American adults have twice the risk of subarachnoid hemorrhage and 2.3 times the risk of intracerebral hemorrhage when compared with Whites, thus contributing to an increased risk for stroke related vascular dementia.

The incidence of end-stage renal disease (ESRD) is 3.6 times higher in African Americans than in non-African Americans and hypertension induced nephrosclerois is more prevalent in African Americans compared to Whites thus attributing hypertension as the second leading cause of ESRD in African Americans (Kaperonis & Bakris, 2003). In addition, African Americans with essential hypertension have an increased susceptibility to glomerular hyperfiltration in response to salt intake leading to progressive renal injury–damage due to a greater lifetime exposure to sustained blood pressure elevation (Arnett et al., 2001).

CURRENT AND FUTURE TRENDS FOR HYPERTENSION GENETICS RESEARCH

With the advancement and decreasing cost of scientific analysis of genetic material, many researchers have moved from conducting linkage analysis on a few specified single nucleotide polymorphisms (SNPs) to running

genome-wide association analysis. Genome-wide association analysis using 500,000 and 1 million SNP chips can be conducted at a reasonable cost and yields results on a larger number of genetic markers. Many long-standing NIH-funded studies on hypertension genetics including but not limited to GENOA, Framingham Heart Study, HyperGen, and the Jackson Heart Study are currently conducting genome-wide association analyses on large epidemiological samples of Whites, African Americans, and Hispanics. Many epidemiological studies of hypertension have been limited to adult populations, but some have been expanded to include multigenerational pedigrees that incorporate children. These genome-wide association studies will provide information regarding genetic markers for hypertension across the entire genome and across the life span. Identifying hypertension markers will provide researchers and clinicians valuable information in determining appropriate interventions for those diagnosed with hypertension.

In addition to better diagnostic and treatment plans for those with hypertension, genome-wide association studies will provide the biological basis for gene-environment interaction studies. The NIH has recommended an increased focus on the interconnectedness of individual and group level characteristics that comprise both biological and nonbiological determinants of health to address health disparities and improve the population's health. Despite this intellectually rigorous stance, the majority of the nation's health research resources in the past has been directed toward biomedical research endeavors and has failed to consider environmental factors as well as the interaction between the two that may be contributing to health disparities, especially among African Americans.

Gene-environment interaction studies will be able to incorporate psychosocial and environmental measures that will help determine to what degree genetic, environmental, and psychological factors contribute or protect populations from hypertension. These studies will bridge the gap between biological (genetic) and nonbiological (environmental) risks and the links between these risks may result in better personalized health care for those diagnosed with hypertension. Health providers who care for patients with hypertension should integrate both biological and nonbiological environmental (lifestyle) factors to create a holistic plan of care. If results of gene-environment studies are integrated into practice, identification of African Americans' risk for hypertension can be made, with early individualized disease management implemented. Combining information regarding a patient's environment (something they can modify, nonbiological) and genetic make-up (who they are, biological) could lead to greater empowerment to manage and monitor their risk for hypertension. These types of studies can provide information for

evidence-based practice that can be used by health providers to develop effective interventions to prevent, manage, and treat hypertension among African Americans, thereby reducing health disparities.

CONCLUSION

Research that begins with children is the key to developing interventions for early detection and intervention for reducing hypertension inequalities among African Americans later in adulthood. Positive lifestyle behaviors instilled early in childhood can help to prevent obesity and resulting hypertension in adulthood. Although genetic heredity of disease is nonmodifiable, environmental lifestyle behaviors (diet, physical activity) can influence phenotypic expression of disease and severity of health outcomes. More research on gene-environment interactions for hypertension is needed to provide improved recommendations for disease prevention and management. Hypertension gene-environment research should lend its focus to large epidemiological longitudinal studies of healthy children of hypertensive parents in order to better understand these interactions. Genome-wide studies of hypertension gene-environment interactions among samples of children could provide the framework for the individualized preventative health care required to eliminate this health disparity early in life for the security of a healthy childhood, adulthood, and beyond.

ENDNOTE

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SECTION III

DISPARITIES IN COGNITION AND HEALTH IN LATE LIFE

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CHAPTER 6

Disparities in Dementia in Later Life Among African Americans

The Role of Early Life Experiences and Limited Opportunities

Peter A. Lichtenberg

African Americans comprise an important group in the aging community. Not only does this group experience higher rates of dementia, but they are also treated for a greater number of vascular risk factors known to enhance the behavioral expression of dementia, such as diabetes, high cholesterol, and hypertension. In the not too distant past, African Americans were legally barred from educational opportunities, and large numbers of African Americans are currently receiving continued segregated and inferior education. African Americans historically have also had less access to quality medicine. Even today, studies indicate that African Americans may not always be treated as aggressively for chronic conditions, and may not be diagnosed with a primary progressive dementia as early as non-African Americans. As the Baby Boomers age and the older population becomes increasingly ethnically and racially diverse, understanding factors affecting diagnosis and treatment of this group of older adults becomes essential. This chapter will focus on understanding the factors contributing to increased rates of dementia in older African Americans as well as best practices in clinical assessment of cognition and cognitive decline.

African Americans have consistently been found to suffer from higher rates of dementia and comorbid disability than non-Hispanic Whites (Gurland

et al., 1999; Moody-Ayers, Mehta, Lindquist, Sands, & Covinsky, 2005; Tang et al., 2001). Tang and colleagues reported that while the incidence of dementia was higher in Blacks than in non-Hispanic Whites, the presence of the APOE-4 allele was actually greater in Whites than Blacks. Moody-Ayers and colleagues (2005) reported that cognition was the single factor accounting for increased functional decline in Blacks as compared to Whites. Green et al., (2002) reported that in African American families first degree relatives (and spouses) of a person with dementia were 1.6 times more likely to suffer dementia by age 85 as compared to non-Hispanic Whites. This difference in risk existed despite the lack of a differential pattern for those with APOE-4 allele (genetic risk marker). Why do older African Americans suffer from dementia more frequently than do whites? The present chapter will examine this question and provide a guide to cognitive assessment for blacks within a life-course perspective, a focus on assessment techniques, and ways to enhance the clinical validity of assessments.

A recent examination of African Americans treatment experiences indicated that they were treated less often with antidementia medication despite suffering higher rates of dementia. Zuckerman and colleagues (2008) compared Blacks and Whites using 2001–2003 Medicare Current Beneficiary Survey data. Over 1,100 beneficiaries were examined. Claims and self-report data were used to examine dementia rates and dementia treatment. The mean age of the claimants was 80 years with one quarter of the sample receiving an antidementia drug. Whites more often used antidementia medication, and even when income and education were controlled racial disparities continued to exist.

Griffith and Griffith (2008) examined the role of genetics and other health-related factors in dementia prevalence in African Americans in their review. They used cross-cultural data to counter some of the current emphasis on genetics and Alzheimer's disease. While Blacks in the United States have higher rates of dementia than do Whites, rates of dementia for Nigerians were lower than were rates for U.S. Blacks and resembled rates for U.S. Whites. These cross-national findings thus refute genetics as the explanation for why African Americans have higher rates of dementia than non-Hispanic Whites. One explanation for increased dementia in Blacks focuses on increased metabolic problems in Blacks and the role of vascular risk in greater rates of Alzheimer's disease. The increasing linkages between chronic diseases such as hypertension, diabetes, and high cholesterol and Alzheimer's disease supports the idea that in America, Blacks suffer from higher rates of dementia due to having higher rates of vascular risk factors (Griffith & Griffith, 2008).

LIFE COURSE AND COGNITIVE FUNCTIONING

A life-course framework points toward understanding how early factors influence late-life expression of disease. Education, environmental exposures, social support, nutritional health, and a host of other factors have been cited as being important in affecting late-life expression of cognition. Barker (2007) reviews evidence for what has been termed the "Barker Hypothesis." Epidemiological studies in Britain not only found higher rates of coronary disease in certain poorer counties in Britain, but also reduced fetal growth has been linked to adult onset coronary disease. In examining death records from 1968–1978, Barker (2007) noted that the low mortality from coronary disease in wealthier areas contrasted with the higher mortality from coronary artery disease in poorer areas. His hypothesis, that malnutrition in utero and during infancy permanently changes the body's structure, physiology, metabolism, and is linked to higher rates of heart disease in adult life, has been supported by several studies. Finally, Barker concluded that while prevention efforts in adulthood have value, their value may be limited in those individuals who were challenged nutritionally in infancy and early childhood.

Crimmins, Kim, and Seeman (2009) examined poverty and biological risk and their findings fit nicely within the Barker hypothesis. Their data were collected from two National Health and Nutrition Examination Survey (NHANES) samples linked to the National Death Index. The NHANES data revealed that poor people in each decade of life (1920s through 1970s) had higher levels of biological risk than people who are not poor in the corresponding ages, thus supporting the notion of premature aging in those who endure poverty. Below age 70 poor individuals were more likely to die at 2–4 times the rate of the nonpoor. Similar to Barker's conclusions about adulthood lifestyle behaviors, Crimmins and colleagues found that age, poverty, and biological risk are little affected by health behaviors (e.g., smoking, alcohol use, over or underweight). Although African Americans had higher mortality rates, there were minimal effects for race over and beyond the effects for poverty. The median age for survival was dramatic with poor men having high biological risk factors reaching the 50% survival rate for their entire cohort at age 53 and nonpoor women without biological risk factors reaching the 50% survival rate at age 84.

Zhang, Gu, and Hayward (2008) examined how early influences affected late-life cognitive functioning. This study focused retrospectively on early life socioeconomic status, ages birth to 14 years. Questions were asked of elders whether they went to bed hungry, what their participation in education was, and whether they lived in a poorer rural area. In this sample schooling was

concentrated on the lower ends, since by far the majority of the sample did not enter middle school education. Overall the results found significant relationships between early childhood hunger, education, and area of birth and late-life cognitive functioning, although there are limits to the study's methodology. For example, they used cutoff scores, which are known to be problematic, on the Mini Mental State Exam MMSE (Chinese version) to assess impairment. The scale, however, can be used to reflect a measure of cognitive functioning, rather than whether decline had occurred. The authors concluded that early life nutrition and health affects late-life cognition.

The Nun Studies, the world's largest brain donation program, provided the first strong connections between early life experience and late-life Alzheimer's disease or confirmed dementia. The participants were members of the School Sisters of Notre Dame religious congregation who were born before 1917. Two thirds of those eligible (678) agreed to participate. Snowdon et al. (1996) published a study investigating linguistic ability in early life and cognition and Alzheimer's disease in late life. In this study, a subset of 93 sisters' autobiographies, were examined. The autobiographies were written just prior to taking their vows (average age 22) and related to cognitive functioning on average 58 years later. Idea density, that is, the number and complexity of the ideas generated in their autobiography, was rated by a linguist. The linguist did not know whether the autobiography was written by someone who in later life developed Alzheimer's disease and thus was blinded to the cognitive function of the participant. Idea density was significantly related to years of education ($r = .27$, $p < .05$). Cognitive function as measured by the MMSE was significantly related to idea density, as were tests of memory, verbal fluency, and confrontation naming and constructional praxis. Of the 93 sisters in the study, 24 had brain autopsy findings. Ten sisters were classified as low-idea density and 14 as high-idea density. Those with low-idea density had significantly higher percentages with temporal lobe neurofibrillary tangles (particularly in the one portion of the hippocampus most associated with memory loss), and frontal lobe tangles. Low-idea density was confirmed in 90% (9 of 10) of the confirmed Alzheimer's disease group compared to 13% of those without Alzheimer's disease. Because of the common adult environmental (and to a large degree, educational) conditions, the findings clearly implicate the role of early life experience on late-life expression of Alzheimer's disease.

Despite the consistent findings from group data across the studies reviewed above, individual differences remain a significant factor in understanding brain aging, and cognitive function in African Americans. Snowdon et al. (1997) reviewed the details of the life of Sister Mary who lived to age 101. She originally had 8 years of formal education, although served as a teacher

to seventh and eighth grade children. At age 41 she finished her requirements for her high school diploma. Sister Mary taught full time until age 77 and part time until age 84. She was 99 years old when the Nun Study began. She scored better on the MMSE as well as memory and fluency tests than any other nun who died within the first 2 years of the study. Her MMSE score was a 27, well within the normal range of functioning for any age or educational level. On autopsy she was found to have a low brain weight, but relatively fewer plaques and tangles in her hippocampus and neocortex than the other deceased nuns and no infarcts. Thus despite some neuropathology consistent with Alzheimer's disease, Sister Mary died without clinical symptoms of dementia.

A second major contribution of the Nun Studies was the finding that dementia was more often expressed in individuals whose brains exhibited both a lacunar infarct and neurofibrillary tangles (Snowdon et al., 1997). This particular study examined the brains of 102 deceased Sisters. Of those, 61 met the neuropathological criteria for Alzheimer's disease. Participants from that group of 61, who had lacunar infarcts had significantly higher prevalence of dementia as compared to those without infarcts. Of the 15 participants with lacunar infarcts, 14 suffered from dementia whereas only 21 of 37 of those without brain infarcts suffered from dementia. This study heightened awareness of the role of vascular factors in Alzheimer's disease. Kuo and Lipsitz (2004) further confirmed this in their review of cerebral white matter changes and geriatric syndromes. Clear associations between white matter change and executive dysfunction were noted, and they recommended aggressive treatment of vascular risk factors in early and midlife. A clear link exists between higher vascular risk factors in African Americans (and poorer treatment for these risk factors) and dementia. Gurland et al. (1999) and Tang et al. (1998) noted that African Americans have higher risk for diabetes, high blood pressure, high cholesterol, and higher rates of stroke.

AGING, COGNITION, AND AFRICAN AMERICANS

My own research in clinical urban samples underscores some of the population findings indicated above. Our 5-year project in the 1990s led to the study of over 1,000 consecutive admissions of patients aged 60–103 years to a geriatric rehabilitation unit for older adults in Detroit. The 1,000 patient sample consisted of 67% African Americans and 33% White (Latino, Arab-American, and Asian patients were eliminated due to small numbers). Two thirds of the sample were women.

Hanks and Lichtenberg (1996) investigated the comorbidities for a variety of physical, functional, psychological, and social variables by dividing the patients into four groups by decades: ages 60–69 (n = 131), 70–79 (n = 363), 80–89 (n = 263), and 90–103 (n = 55). The racial makeup of the groups was no different across the different ages, but not surprisingly, the oldest decades were made up of significantly more women than in the youngest decade. Principal diagnoses also differed for the age groups. Stroke was a much more common primary diagnosis among the younger groups, whereas hip fractures from falls were more common in the older groups. Mean education ranged from 10.55 years for the youngest group to 8.56 years in the oldest group, but these differences were not statistically significant.

Comorbid physical disease was measured by Charlson, Pompei, Ales, and MacKenzie's (1987) index of comorbidity. This measure was later validated in our sample. Consistent with Crimmins and colleagues' study of poverty and aging, the younger patients exhibited significantly worse comorbid illness than did the oldest group. The younger adult patients actually suffered from more diseases, and from more serious comorbid diseases than did the oldest patients. This sample of younger patients represented an extremely ill sample relative to the base rates of cognitive problems in community samples of comparable ages.

Measures of psychological functioning revealed that while the base rate of significant cognitive dysfunction in the community was 15% for those ages 60–79 (Evans et al., 1989), the prevalence of cognitive dysfunction in our sample for these ages was 40%. In our sample the prevalence of depression was 35% compared to community estimates of 15% (Blazer, Hughes, & George, 1987). Finally, whereas alcohol abuse was estimated to be present in less than 5% of the community samples, it was present in 19% of our sample ages 60–79 years. Clearly then, the younger patients, ages 60–79 years, represent a significantly impaired population and a population of inseparable physical and mental health disease. The oldest group, while more representative of the community population ages 80–103, also demonstrated significant comorbid physical and mental health problems. Cognitive dysfunction was present in 60% of our older patients, compared to base rate estimates of 45%. Depression and alcohol abuse were present at twice the rate in our sample (27% and 8%, respectively) as compared to base rate estimates.

The groups may represent different populations. It is likely that patients in the younger group (ages 60–79) do not survive into the older group. The patients from the older group are only now finding themselves in health care settings, and were vigorous in their younger years. The oldest group

is made up of those who survive into older age and then become disabled, while the younger group is likely to experience mortality before many of its members reach age 80 and beyond.

COGNITIVE SCREENING AND AFRICAN AMERICANS

Cognitive screening, although useful, is filled with many potential pitfalls. Two recent national studies illustrate these issues. Langa et al. (2008), using the telephone screening of cognitive status (TICS) compared a 1993 cohort of 70 and older with a 2002 cohort. He found that when using the standard cutoff of the TICS, which despite higher rates of diabetes, heart disease, and psychiatric problems, cognitive impairment consistent with Alzheimer's disease decreased from 12.2% to 8.7% in just one decade. The mean educational level, however, was significantly greater for the 2002 cohort by nearly a full grade. Thus while it is not at all controversial that there were differences in cognitive test scores, interpretations about cognitive impairment suggests declines in cognitive functioning. Since individuals were likely to have started at different cognitive levels, as indicated by their different levels of educational attainment, it might be misleading to conclude that levels of impairment were declining. Indeed, using a subset of the exact same data set, Plassman and colleagues (2008) reported that levels of dementia plus those with cognitive impairment without dementia were likely underestimated. Plassman et al. (2008) used a more comprehensive assessment approach, with both extensive neuropsychological testing and neurological exams, and found three times the rate of cognitive impairment as compared to Langa's suggesting that there may not be a decrease in cognitive decline in older adults. Given the improvement of assessment techniques, we may actually be seeing an increase in the proportion of people experiencing some cognitive decline (not all of which meets the criteria for dementia).

Age and education typically account for significant variance in cognitive screening measures. The Mini Mental State Exam is the most often used cognitive screening test in the world. The 30-point test has items on orientation, registration, calculation, recall, language, and copying. Crum, Anthony, Bassett, and Folstein (1993) examined age and education differences in mean MMSE scores. Those with 0–4 years of formal education had a mean score of 22 for ages 65–69 and 19 for those ages 85 and over. The standard deviation was also greater for the oldest group indicating that scores are more variable in the oldest old. For those who completed 9–12 years of education, however, a mean score of 28 was found for those ages 65–69 while those over 85 years

had a mean score of 26 (once again the standard deviation was greater for the older old group).

The diagnostic difficulties that exist when using a basic screening test as the only cognitive measure when assessing cognition in African American older adults was demonstrated in Mast, Fitzgerald, Steinberg, MacNeill, and Lichtenberg (2001) comparison of clinical utility of the MMSE and the Fuld Object Memory Evaluation for a group of adults being evaluated for dementia in an Alzheimer's center satellite. The Fuld OME was developed to evaluate different component abilities of memory functioning (e.g., storage and retrieval) in the elderly. The OME uses a procedure that attempts to limit the effects of hearing and vision impairments upon test performance. It forces the individual being evaluated to cognitively process the information to be remembered by tactually recognizing the object, visually recognizing the object, naming it, and then, if still unable to identify the object, hearing the name of it (Fuld, 1980). After ensuring the patient has cognitively processed the stimuli to be remembered, they are asked to recall the objects after a distraction period on each of five trials. After each trial, patients are selectively reminded of items not recalled. The Fuld OME produces scores on four indices: storage, retrieval, repeated retrieval, and ineffective reminders. Two are indices of retrieval skills (i.e., retrieval and repeated retrieval) whereas two are indices of storage (i.e., storage and ineffective reminders). This study involved 137 consecutively evaluated older African Americans. The MMSE was actually considered by the clinicians in the diagnostic process whereas the Fuld scores were kept blinded. Overall, there were significantly different scores in both the MMSE and Fuld when comparing the normal versus the dementia group. There were however, significant differences in the clinical utility of the two measures. The MMSE was significantly poorer in its sensitivity and positive and negative predictive power than was the Fuld. Further analysis indicated that while the MMSE was significantly correlated with education ($r = .32$, $p < .01$), the Fuld was not ($r = .04$, $p > .05$). The finding that the relationship of education and educational quality affects cognitive test score interpretations in African Americans has been found by several investigators (Lichtenberg, 1998; Lucas et al., 2005; Manly, 2005).

The following study underscores the importance of understanding the limits of generalizability of normative data sets. Unverzagt et al. (1996) published the first normative data on African Americans who were given the CERAD battery (Consortium to Establish a Registry of Alzheimer's disease) and who resided in an urban area. Completing a mean of 9 years of education, 83 subjects demonstrated reduced scores on the CERAD Boston Naming and

memory tests as compared to previous normative samples made up of more highly educated, white older adults. Fillenbaum, Heyman, Huber, Ganguli, and Unverzagt (2001) followed up on this line of inquiry by comparing normative data on Blacks and Whites in North Carolina to the original Unverzagt data. This study found that when sex, age, and years of education were controlled, there were no racial differences. In contrast to these studies, years of education has not always accounted for the differences in cognitive scores in normal Blacks and Whites. The quality of education, experienced quite differently by Blacks and Whites, has accounted for even more differences between the groups than simply counting years of education.

Racial differences, even when accounting for years of reported education, have been found in several other samples of older adults including the MacArthur Studies on Successful Aging, The Columbia studies, and the ACTIVE study (Manly et al., 1999; Morgan, Marsiske, & Whitfield, 2008; Whitfield et al., 2000). Literacy differences and the differential quality (as opposed to only quantity) of education accounts for these differences. Manly (2005) reviewed the advantages and disadvantages of using separate normative data for African Americans versus White samples. Simply put the use of African American norms, while clinically more accurate and sensitive, gives the impression that differences are due to race, while the true differences in normative data may be confounded by socioeconomic status, quality of education, and health in early life.

One unique example of this latter observation was our research on the Boston Naming Test (Lichtenberg, 1998). The Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983) is an established clinical tool for assessing naming deficits associated with a variety of neuropathological conditions. Originally published with 85 items (Kaplan, Goodglass, & Weintraub, 1978), the current 60-item version is a commonly used psychometric test in assessing some aspects of language functioning. Patients are presented with pictures of objects that they are then asked to name. If they cannot name the object patients are given a clue (e.g., an ocean animal for octopus). If they still cannot name the object they fail the item.

Our initial research indicated that African Americans scored significantly lower on the Boston Naming Test than did Whites. Instead of using a reading test as a measure of educational quality, we directly measured the items and their relationship to literacy. We investigated the relationship of how prominent (easy to hard) the items on the test were found in written language. Thus, we deconstructed the Boston Naming Test on the basis of literacy. Based on the percentage correct in the overall sample, items were grouped into three categories of 20 items each: ranging from relatively high correct percentages

(i.e., 95–100%) to relatively low ones (4–40%). A total score of correct responses was summed for each group and African American and White subjects were compared. Scores having modest difficulty were the ones that best separated the groups, with Whites identifying more correct responses than did African Americans. This grouping of 20 items included such stimuli to be named as octopus, pretzel, snail, canoe, igloo. In Table 6.1 the items are separated into the three groups (easiest to hardest). As can be seen in the table, there are some discrepancies between the ease of items and the order in which they are presented. For example, items 13, 19, 46, and 49 were found to be of modest difficulty. Two of these items are in the first third of the test, while the other two are in the last third.

Deconstructing race found that literacy level was directly responsible for the differences found between African Americans and Whites on the Boston Naming Test.

Clinical Implications of Normative Studies

Lichtenberg (1998) presented the Normative Studies Research Project test battery that consisted of tests that had normative data based on urban African American elders. The battery consisted of tests of reading, attention, memory, language, visual spatial, and executive functioning tests. Reading levels were also obtained through the Wide Range Achievement Test-Revised (WRAT-R). Reading levels for African Americans were on average, 3 years below their reported number of years of education. This also indicates the

TABLE 6.1
Deconstructing the Boston Naming Test

Item Listing of Boston Naming Test
Easy
BNT1+BNT2+BNT3+BNT4+BNT05+BNT06+BNT07+BNT08+BNT09+BNT10+ BNT11+BNT12+BNT14+BNT15+BNT16+BNT17+BNT18+BNT20+BNT21+BNT28.
Moderate
BNT13+BNT19+BNT22+BNT23+BNT25+BNT26+BNT27+BNT30+BNT31+ BNT32+BNT33+BNT34+BNT35+BNT36+BNT37+BNT38+BNT40+BNT46 BNT47+BNT49.
Hard
BNT24+BNT29+BNT39+BNT41+BNT42+BNT43+BNT44+BNT45+BNT48

importance of assessing the quality as well as the quantity of education (i.e., reported years of education). Extensive normative data can now be found for samples of African Americans through the Mayo Older African American Normative Studies (Lucas et al., 2005) and many other sources (see Spreen and Strauss, 1998).

It is imperative to remember that neuropsychological test scores alone are not sufficient to diagnose dementia. Rather, the essential process is to incorporate test scores with a patient's history and to match the patient's age and education to the normative data available. Then utilize three questions to guide clinical interpretation of test data. (1) Is there evidence of cognitive deficits? (2) What are the cognitive strengths and weaknesses? (3) What practical recommendations can you make? Below I present two case studies to illustrate this approach.

Case 1

Ms. P. was a 79-year-old, African American, widowed, retired beauty home operator whose knee pain worsened over several years to the point of being unable to ambulate. Ms. P. was admitted for a right knee replacement. Other medical problems included anemia. Prior to her rehabilitation admission, Ms. P. received Meals on Wheels, had friends who performed most of her Instrumental Activities of Daily Living (IADLs), but did take care of her own Activities of Daily Living (ADLs). Ms. P. did continue to drive on occasion. She completed 13 years of school. Her friend of 50 years reported to me that Ms. P. was often becoming confused, with decreased memory and concentration. A neuropsychological evaluation was conducted.

Is there Evidence of Cerebral Dysfunction in Ms. P?

To assess this question the results of the current cognitive testing must be compared either to past testing, or more commonly, to a normative score. As highlighted above, normative data for Whites cannot simply be applied to Blacks due to differences in levels of literacy and quality of education. In this case comprehensive analysis of neuropsychological test score results were consistent with a primary progressive dementia such as Alzheimer's disease. While her reading score indicates premorbid cognitive functioning levels to have been in the average range, her scores on other tests, particularly tests of memory and naming, are significantly impaired.

Dementia Rating Scale: 114/144
Logical Memory I: 6/32
Logical Memory II: 0/32

Fuld Storage: 20/50
Fuld Retrieval: 11/50
BNT: 24/60
WRAT-R Reading (scaled score): 88
MAE Aural Comprehension: 17/18
VFD: 30/32
VOT: 15/30

What Are Her Cognitive Strengths and Weaknesses?

The second step in a cognitive evaluation includes an analysis of strengths and weaknesses. Indeed the test results indicate that her attentional skills are relatively well preserved in early dementia, as evidenced by her score on the visual matching. Aural comprehension is unimpaired, but confrontation naming is reduced. Scores on both memory tests are severely reduced, with the Fuld scores being some of the lowest scores that we have seen.

What Practical Recommendations Can Be Made?

Cognitive test results should be applied to recommendations about functioning, the need for caregiving and the impact of cognitive functioning on advanced activities of daily living. Due to the severity of Ms. P.'s memory problems it was recommended that she receive 24-hour supervision. Her dear friend took Ms. P. into her home, but after 6 months Ms. P. had declined further (she was seen after a fall brought her back into the rehabilitation unit) and was transferred to a nursing home. In the interim period she had received a thorough workup to rule out reversible causes of her cognitive decline and been diagnosed with Probable Alzheimer's Disease.

Case 2

Ms. MT. was a 75-year-old, African American, widowed, retired school teacher who had a left hip replacement due to long-term degenerative joint disease. Other medical problems included hypertension and hypothyroidism. For the past 4 years she used a cane at home to ambulate, but Ms. MT. was independent in all of her ADLs and IADLs, including cooking. She retired 14 years prior to her surgery and had enjoyed the freedom of retirement and traveling. Ms. MT. was referred for an evaluation because she lived alone. She had completed 18 years of education.

Dementia Rating Scale (total): 143/144
Logical Memory I: 28/32

Logical Memory II: 24/32
Boston Naming Test: 58/60
Visual Form Discrimination: 32/32
Visual Organization Test: 25/30
WRAT-R (scaled score): 110 (high average)

Is There Evidence of Cerebral Impairment?

Ms. MF.'s case is an example of successful aging. Her test scores indicate strong cognitive functioning across the board. There are no test scores indicative of any cognitive decline. This case emphasizes the point that clinical applications of cognitive testing are often not directly related to broader cognitive research on race and dementia. Many older African Americans are functioning well and exposure to these older adults is invaluable, if for no other reason than to curb any ageist attitudes or biases.

Summary

In this chapter, the current understanding of factors contributing to dementia in older African Americans was discussed. Past research indicates that African Americans are at higher risk for incipient dementia, as well as the vascular factors that can enhance behavioral presentation of dementia. The increased prevalence of dementia in African Americans is not explained by genetic factors, and this chapter discusses potential factors contributing to these findings, as well as discussing issues of clinical utility. Research discussed highlights the potential for in utero factors setting the stage for onset of dementia via vascular factors later in life, as well as early life experiences including malnutrition. Furthermore, educational attainment, social support, and perceived support from the health care community may influence diagnosis, treatment, and ultimate control of chronic vascular risk factors, including diabetes, hypertension, and high cholesterol, which may in turn enhance behavioral expression of dementia in African Americans. Finally, this chapter discussed the importance of appropriate utilization of standardized assessment in this unique population, which includes accurate establishment expected baseline abilities through "hold" tasks such as word recognition or vocabulary, comprehensive integration of assessment results with medical and psychosocial information and finally tying recommendations to test results and psychosocial demands.

The evidence reviewed in this chapter clearly implicates an important role of early experience on late-life outcomes of dementia among older samples

of African Americans who were relatively deprived at earlier periods of their individual and group life course.

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CHAPTER 7

Cognition and Health Disparities Over the Life Course

Longevity as an Example

Lindsay H. Ryan and Jacqui Smith

Life-course models of late-life health outcomes focus on the role of biological, social, and environmental factors and events that shape the lives and the aging trajectories of individuals and subgroups within populations (e.g., Barker, 1997, 2003; Ferraro, 2006; Ferraro & Shippee, 2009; House, 2002; House, Lepkowski, Kinney, & Mero, 1994; Kuh & Ben-Shlomo, 1997; Marmot, 2005; O'Rand, 2006). Research associated with these models examines hypotheses about (a) critical age periods of exposure to risk or advantage in relation to later illness and mortality; (b) the accumulation of disadvantage or advantage and whether this affects health trajectories; (c) the potential to modify the effects of early-life disadvantage in midlife or later; and (d) mechanisms underlying social determinants of health inequalities (e.g., stress: see Chapter 2 for a review). To date, much research deals with the life-course influences of membership in social group categories defined, for example, by socioeconomic status (SES: e.g., parental SES, individual education, occupation), gender, race and ethnicity, and birth cohort. There are also seminal longitudinal studies about the population and cohort-specific impact of historical events and social upheaval on health in old age (e.g., Diewald, Goedicke, & Mayer, 2006; Elder, 1994; Vaupel, Carey, & Christensen, 2003).

Life-span models (see Chapter 1 for a review) focus somewhat less on understanding the macrolevel social determinants of late-life outcomes and

inequalities in health and more on understanding the role of individual characteristics (e.g., cognitive ability and functioning, personality, self-related beliefs, social relationships). These individual characteristics develop and change over the course of an individual's life. They are dynamically coconstructed in interactions with biological, social, and environmental factors and contribute to substantial differences not only between social groups but also within those groups.

This chapter focuses on the significant contribution to health inequalities over the life course of one individual characteristic, namely cognitive ability and functioning. In particular, we review research on the associations between cognition and inequalities in longevity, as one indicator of the outcome of health inequalities within a population. We consider life-span cognitive functioning in a broad, primarily nonpathological sense as it is measured by tests of intelligence and performance on tasks that assess different aspects of memory, processing speed, reasoning, and knowledge. Other chapters in this book consider inequalities in pathological decline and dementia.

Reviews about health behaviors across the life-course point to the significant contribution of cognitive status and knowledge to accounting for variation within a population (Burgess, Powell, Griffin, & Partin, 2009; Deary, 2005; Gottfredson, 2004). There are also positive moderate associations between cognitive functioning and socioeconomic status. Scores on intelligence tests (which measure individual differences in general cognitive ability), for example, are sometimes used as entry screens for educational institutions and occupations. Income and wealth in adulthood, however, are less highly correlated with cognitive ability. Within homogenous samples of high cognitive functioning persons, there are significant differences in personal wealth, income, and occupational achievement (e.g., Friedman et al., 1995; Schaie, 2005; Terman & Oden, 1959).

There are two main parts to the present chapter. We begin with a review of research that shows a strong positive relationship between cognition and longevity (Andersen et al., 2002; Deary, Whiteman, Starr, Whalley, & Fox, 2004; Johnson, Deary, McGue, & Christensen, 2009; Korten et al., 1999; Samuelsson et al., 1997; Small & Bäckman, 1997). Researchers take different approaches to examine this association. Some studies focus on cognitive ability and change in cognitive functioning in older adults. Other studies document the significant long-term influence of cognitive functioning, for example, by linking childhood mental performance with later survival in old age. Another literature examines potential genetic explanations for the association between cognition and health with later survival. We consider each of these research approaches in turn, report illustrative data from the Health and Retirement

Study (HRS), and discuss how these various approaches fit together. The second part of the chapter outlines some of the myriad of theories proposed to explain the association between cognition and longevity and the difficulties in disentangling effects. Explanations range from the contribution of cognition to disparities in environmental experiences, health behaviors, lifestyle preferences, and material resources (Adler et al., 1994; Adler & Snibbe, 2003; Aldwin, Spiro, Park, Birren, & Schaire, 2006; Schooler, Mulatu, & Oates, 2004), underlying genetic influences associated with cognition and health (Atzmon, Roncon, Rabizadeh, & Barzilai, 2005; Gottfredson, 2004; Gottfredson & Deary, 2004), and complicated genetic-environmental dynamics over the life course (Dickens & Flynn, 2001; Johnson et al., 2009).

The three parts to Figure 7.1 illustrates our integrative attempt to summarize various scenarios about the life-span and life-course associations among individual differences in cognition, health, and longevity. The scenarios in Part A and Part B suggest that variability in early-life cognitive status (high, average, and low-level functioning) translates into longevity inequalities. Whereas Part A suggests that individual variation in cognition is maintained over the life course, Part B reflects the idea that advantages or risks associated with cognitive functioning accumulate over the life course and contribute to a magnification of individual differences in longevity. The relative rank order of individuals over the life course remains the same in these two scenarios. The scenario portrayed in Part C, however, reflects the idea that personal experiences and exogenous events in adulthood and old age may modify life trajectories and longevity. Some life-threatening illnesses and their treatments, for example, which are not known to have strong SES associations, could alter trajectories of longevity.

RESEARCH ON LATE-LIFE COGNITION AND LONGEVITY–MORTALITY RISK

Studies of older adults find that higher level and slower rates of cognitive decline are predictive of survival and longevity (e.g., Alwin, McCammon, Wray, & Rodgers, 2008; Anstey, Luszcz, Giles, & Andrews, 2001; Anstey, Mack, & von Sanden, 2006; Bosworth, Schaie, & Willis, 1999; Deeg, et al., 1990; Ghisletta, McArdle, & Lindenberger, 2006; Korten et al., 1999). This trend is highly replicable, as we illustrate in Figure 7.2 using data from the oldest cohort in the Health and Retirement Study (HRS), a nationally representative longitudinal study of middle-aged and older adults in the United States (Juster & Suzman, 1995). To address the current issue of cognition and survival, we selected individuals who first participated in the HRS-AHEAD study in 1993 and were

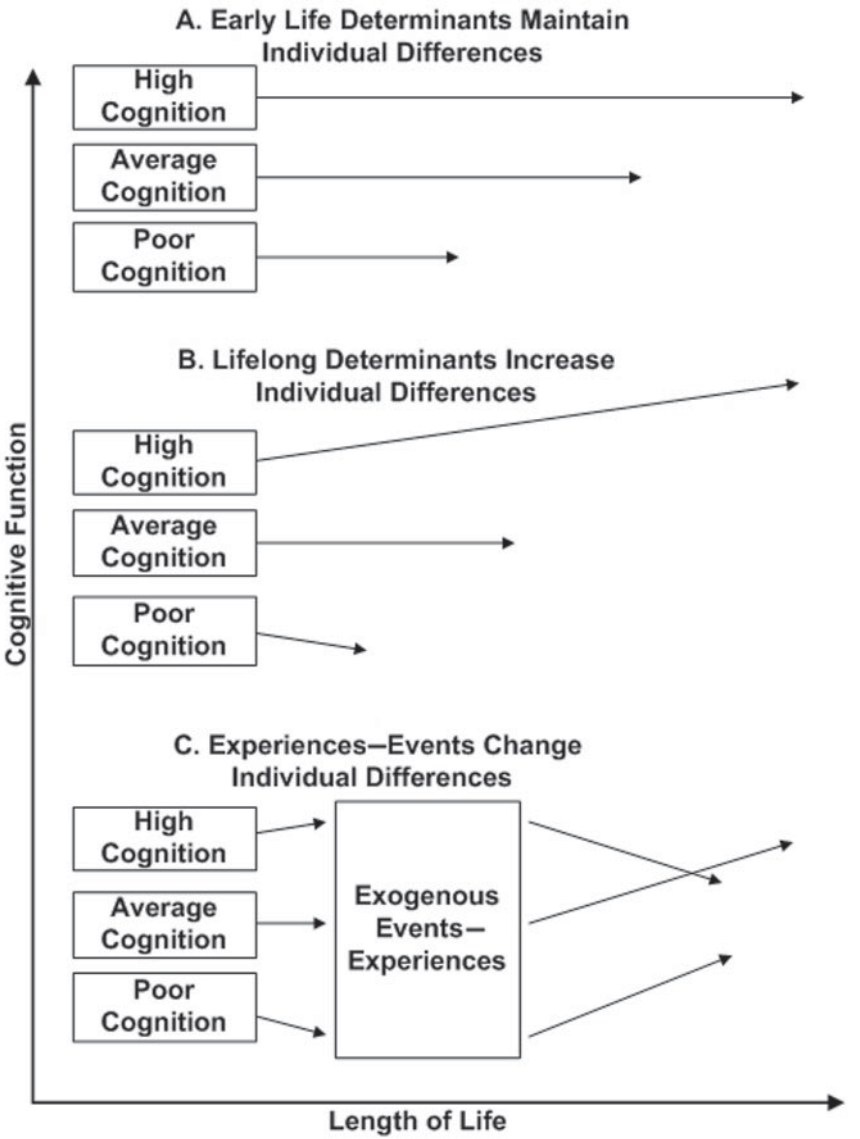

FIGURE 7.1 Three life-span models of cognition-health disparities: Individual differences in longevity.

at least 70 years old (born in or before 1923; *N* = 7,986). Vital statistics on participant survival were retrieved from the National Death Index and verified by the National Center for Health Statistics and by HRS staff. To examine differential survival rates from 1993 to 2006 by baseline cognitive performance, a score of verbal memory was created by summing the immediate and delayed recall of 10 words administered as part of the core HRS assessment in 1993 (Mean summed word recall = 7; *SD* = 4; Range = 0–20: refer also to McArdle, Fisher, & Kadlec, 2007). Three cognitive status groups were created from the baseline memory score; high memory individuals were at least one standard deviation above mean words recalled (*n* = 1,063), poor memory individuals were at least one standard deviation below the mean number of words recalled (*n* = 2,112), and all other individuals were in the average memory group (*n* = 4,811). Figure 7.2 provides the percentage of individuals who remained alive at each wave from 1993 to 2006 by cognitive status group, illustrating the clear pattern of greater survival among the individuals with high memory functioning at baseline. In addition, while Figure 7.2 illustrates the pattern of survival by memory status for the entire sample, the same trend is replicated when survival by memory is plotted by gender and age.

Furthermore, the extant research has indicated that cognitive performance is associated with mortality risk over and above many established health correlates. For example, Korten and colleagues (1999) found that poor

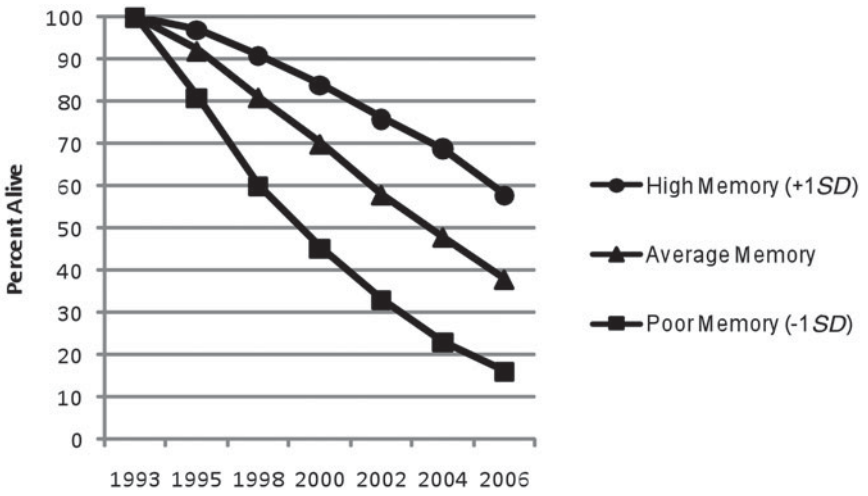

FIGURE 7.2 Percentage of HRS-AHEAD participants surviving from 1993 through 2006 by baseline memory performance (*N* = 7,986).

cognitive performance in a sample of adults aged 70 and above was a significant predictor of mortality, over and above the effects of self-rated health, activities of daily living, number of illnesses, and systolic blood pressure. Additionally, the inclusion of both physical health and cognitive predictors negated the effect of age on mortality risk, suggesting that a portion of the variance in mortality risk may be unique to cognitive functioning over and above age. As support for the ubiquitous association between cognition in late life and longevity, there is evidence that in addition to predicting survival after controlling for physical health, cognitive functioning is also associated with mortality in samples diagnosed with specific illnesses. Anstey and colleagues (2006) reviewed the extant clinical research for three specific chronic diseases (stroke, coronary heart disease, cancer) with available data on cognition and mortality. The findings indicated that individuals with diminished cognitive function who were diagnosed with stroke or cancer had higher mortality risk, while the association between cognition and mortality for samples diagnosed with coronary heart disease was mixed. Finally, several studies have found that the rate of cognitive decline is often a better predictor of mortality risk than baseline cognitive performance (Anstey et al., 2001; Berg et al., 1996; Bosworth & Schaie, 1999; Deeg et al., 1990; Sliwinski et al., 2006; Small & Bäckman, 1997), such that individuals who are experiencing faster rates of cognitive decline regardless of their baseline performance have a higher mortality risk. This pattern is also supported by studies that have found that after accounting for time to death, age effects for memory decline are significantly reduced (Thorvaldsson, Hofer, & Johansson, 2006). Thus, declines in cognitive performance may be more strongly associated with impending death than age per se.

There is also evidence that cognition is a predictor of later survival in samples of the oldest-old. Using data from the Swedish Centenarian Study, Samuelsson et al. (1997) examined predictors of survival in a sample of older adults 100+ years old over a period of 6 years. The authors found that while the strongest predictors of survival were body mass composition (BMI and lean weight) and marital status, cognitive performance was an important direct indicator for later survival. Interestingly, health, as measured by blood pressure and activities of daily living (ADLs), was not a direct predictor of survival. Instead, the effect of health operated through its association with cognitive performance. Specifically, higher blood pressure was associated with higher cognitive function in the sample of centenarians, which was then positively associated with length of survival. This finding illustrates the complexities of examining cognition and health relative to longevity, such that it is difficult to tease apart the causal directions between the effects of health and cognition for survival.

The presence of cognitive impairment in the oldest old has also been identified as a significant mortality risk. Using a national sample of Danes born in 1905 who survived to 1998, Andersen et al. (2002) examined the association between cognitive impairment assessed from the Mini Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975) with survival over 2 years. As expected, the risk for mortality grew with the severity of cognitive impairment. The authors also found that no single component of cognitive performance was more important for survival, suggesting that generalized cognitive function is most critical for survival in the oldest old, which has been substantiated in several other studies (Anstey et al., 2001; Maier & Smith, 1999).

Furthermore, one's level of cognitive performance is important relative to other health domains, such as the functional limitations that are associated with an adult's ability to live independently (Lawton & Brody, 1969). This association is illustrated in Figure 7.3 using a subsample of cross-sectional data from the 2006 wave of the HRS. A random split-half of the entire 2006 sample were eligible to receive an enhanced face-to-face interview that included detailed physical assessments and biomarkers, as well as a leave-behind psychosocial questionnaire that was later returned by mail. Using this subsample (N = 8,601), we created four age groups ranging from middle to late adulthood: (1) mean age 55 (range = 52–59), (2) mean age 65 (range = 60–69), (3) mean age 75 (range = 70–79), and (4) age 80+ (range = 80–104). Memory status groups were created in the same way as described for Figure 7.2, including poor memory (–1 *SD*; n = 2,280), average memory (n = 5,315), and high memory (+1 *SD*; n = 970). Memory status and age groups were compared cross-sectionally relative to the total number of functional limitations participants reported in 2006. Functional limitations were assessed in relation to mobility, large muscle functioning, fine motor skills, gross motor skills, and the ability to perform activities of daily living (ADLs) and instrumental activities of daily living (IADLs). Participants were asked whether or not they have any difficulty with a series of activities due to a health problem, including activities such as running or jogging a mile, walking one block, climbing one flight of stairs, picking up a dime, shopping for groceries, dressing, and bathing.

Figure 7.3 illustrates two main effects. First, as expected, the number of functional limitations is higher with older age. Second, and more interestingly, the number of limitations is inversely associated with memory status for all age groups. This pattern supports the literature that finds associations between cognitive performance and IADLs in older adults, such that individuals who have higher cognitive functioning are typically better able to perform activities necessary for daily living (Lawton & Brody, 1969). Perhaps an individual's ability to solve problems, reason through health challenges,

and keep track of complex health maintenance programs is instrumental in maintaining important functional abilities across adulthood. And while this is not directly associated with longevity, it is also necessary to consider quality of life in late adulthood so that the advances in research not only help to keep individuals living longer, but also to insure that those extra years of life are as happy and healthy as possible.

This range of literature indicates a similar pattern, such that older adults who have higher cognitive functioning and slower rates of decline tend to live longer. However, it is important to address the mechanisms that underlie interindividual variability in level and rate of change in cognitive ability among older adults. In other words, while it's clear that cognitive performance in late life is a critical component of later survival, are there additional underlying factors that affect both cognitive performance and survival? One possibility is the occurrence of terminal decline in aging populations. Terminal decline, the well-documented cognitive decline preceding death (Alwin et al., 2008; Berg et al., 1996; Johansson & Berg, 1989; Sliwinski, et al., 2006; Small & Bäckman, 1997), is an important confounder that needs to be addressed. It is entirely possible that the association between cognitive performance and mortality risk in older adults is driven by those participants in the studies who have begun trajectories of terminal decline. Their trajectories may be

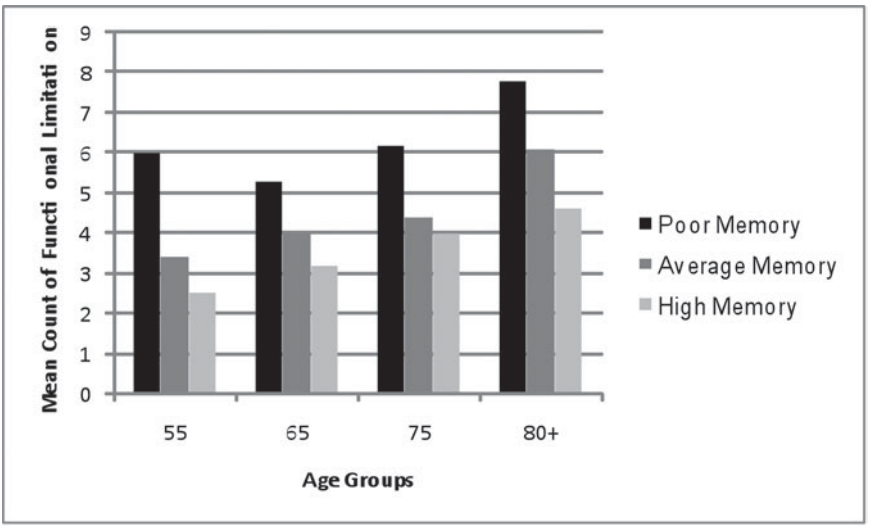

FIGURE 7.3 Mean number of functional limitations across age groups by memory status (N = 8,601).

the result of underlying biological changes that are the true mechanism of impending death. This is particularly relevant to the findings that the rate of cognitive decline in older adults is a stronger predictor of survival than one's level of ability (Anstey et al., 2001; Bosworth et al., 1999; Bosworth & Schaie, 1999; Deeg et al., 1990).

It has been suggested that the rate of cognitive decline in the terminal decline phase is a biomarker associated with mortality, and that other brain-based functions illustrate similar patterns of decline (Baltes & Lindenberger, 1997; Lindenberger & Ghisletta, 2009) due to underlying physiological changes. To investigate the association between cognitive change and another brain-based ability, we examined verbal memory change and balance in the HRS. In this example, we considered individuals' balance, a physical measure assessed in the enhanced face-to-face sample of the 2006 HRS, which is linked to functioning in the brain and mortality (Guralnik et al., 1994; Laukkanen, Heikkinen, & Kauppinen, 1995). The HRS protocol uses three balance tests, including tandem, semitandem, and side-by-side. All participants who were able to stand for 60 seconds without assistance were asked to participate in the balance tests. Those individuals were first given the semitandem test and asked to hold the position for 10 seconds. Those able to hold the position for the full time (96%) were then asked to do the more challenging full-tandem standing test for 30 seconds. Alternately, those who could not hold the semitandem position for 10 seconds were asked to do the easier side-by-side test. For the purposes of the present example, individuals were categorized into two balance groups: (1) the weak balance group included individuals who could not hold the semitandem position for 10 seconds, and (2) the strong balance group included individuals who successfully completed the semitandem position. To assess the rate of recent cognitive change consistent with studies of terminal decline, 4-year change scores in verbal memory were calculated such that negative scores indicate decline over 4 years. Thus, participants were included who partook in the 2002 and 2006 core HRS telephone battery with complete delayed and immediate word recall tests for both waves (with a summed total range of 0–20). To evaluate the possible effect of terminal decline, only older adults (age range 65–104 in 2006) were selected in this example.

Figure 7.4 illustrates several key points that support extant research. First, relative to the older age groups, the 65 to 69-year-old group experienced less decline in verbal memory, which is consistent with findings that reliable, population-level cognitive decline only begins in the sixth decade (Finkel, Reynolds, McArdle, Gatz, & Pedersen, 2003). Second, support was found for the theory that underlying brain changes may cause both cognitive

decline and mortality risk in older adults. Specifically, Figure 7.4 illustrates that, particularly for the 70- to 74-year-old age group, individuals with poor balance in 2006 had greater mean declines in memory performance over the previous 4 years. The 4-year change in balance could not be assessed in the present example, but it is possible that the association with memory change and balance would be even greater had this been feasible. However, a recent study by Lindenberger and Ghisletta (2009) indicates that while cognitive and sensory processes may be affected by a shared underlying factor associated with physiological change, the association is moderate. Specifically, the relationship between cognitive and sensory change was significant but not large, suggesting just a portion of shared variance that may be attributable to underlying physiological change in both domains. Due to the potential confounding association with changing brain physiology that may be affecting multiple facets of aging (such as balance), cognitive performance, and survival in older adults, researchers need to consider ways to evaluate the association between cognition and longevity that can control for associated age-related physical changes. An innovative approach to this problem is the consideration of better estimates of baseline ability prior to late life that avoid confounds with terminal decline and mortality risk.

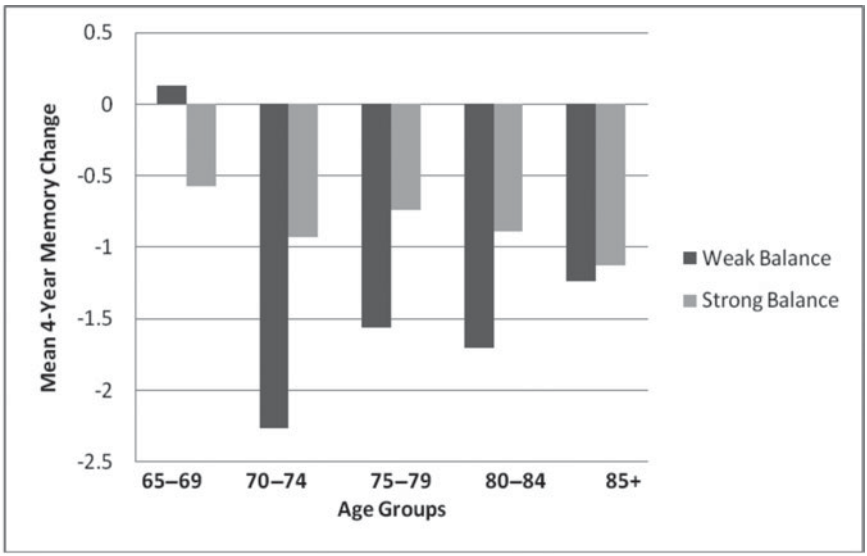

FIGURE 7.4 Mean 4-year memory change (word recall) by balance ability and age group (N = 3,604).

RESEARCH ON EARLY-LIFE COGNITION, HEALTH, AND LONGEVITY

To address the problems of ascertaining accurate baseline cognitive ability and avoiding misleading findings due to unmeasured factors associated with both interindividual variation in cognitive function in older adults and longevity (i.e., terminal decline), several studies have utilized well-documented nationwide childhood intelligence testing and followed up individuals in late life. This has produced compelling evidence that childhood mental ability is predictive of survival into late adulthood (Deary, Batty, Pattie, & Gale, 2008; Deary et al., 2004; Whalley & Deary, 2001). Much of this research has come from the nationally mandated Scottish Mental Surveys in 1932 and 1947 of all 11-year-old school children in Scotland (see Deary et al., 2004 for more detail). Specific analyses reported in a series of papers have indicated that childhood intelligence at age 11 is positively associated with survival at age 76 (Whalley & Deary, 2001), is negatively associated with mortality risk due to lung and stomach cancers, and accounts for later survival after accounting for other identified factors such as personality (Deary et al., 2008). These findings are critical in understanding the larger picture of how cognition and longevity are related. Specifically, they strengthen the argument that cognitive ability across the life course is an important factor associated with individuals' survival.

Beyond predicting survival however, childhood intelligence has been associated with cognitive performance in older adults, identified as an independent factor associated with mortality risk. Several studies have found that lower cognitive ability in childhood is associated with steeper cognitive decline and greater variation in cognitive performance in late life (Bourne, Fox, Deary, & Whalley, 2007; Richards, Shipley, Fuhrer, & Wadsworth, 2004; Snowdon et al., 1996). Adding a layer of complexity, a recent study by Luciano et al. (2009) using the Lothian Birth Cohort 1936 found that while childhood intelligence is positively associated with processing speed in older adults, the effect was attenuated for e4 apolipoprotein E (APOE) carriers, suggesting moderation by genetic effects.

The primary advantage of examining childhood intelligence scores with survival and longevity in older adults is that effects related to baseline cognitive ability carry greater validity, and the confounds inherent in aging research such as underlying physiological changes that may affect both cognition and longevity can be avoided. However, as recognized by Batty, Deary, Schoon, and Gale, (2007), childhood intelligence is often measured in children who have been in school for several years, so the effects of the environment and experiences in the earliest years on intelligence testing in school are not being accounted

for, leaving the possibility that differential experiences and resources available to individuals in infancy and early childhood may be having an unmeasured impact.

The field of research on cognition, health, and longevity is still developing, but there are clear indications that cognitive performance at all stages of the life course is important for longevity. Longitudinal research on contemporary cohorts of older samples provides ample evidence that both baseline performance and the rate of cognitive change are often better predictors of mortality risk than health and age in late life (Korten et al., 1999; Samuelsson et al., 1997; Thorvaldsson et al., 2006). This is complemented by research examining the long-term effects of childhood intelligence, which provides evidence that high mental ability early in life is protective for survival in older adults. A variety of theories have been used to understand these associations, two of which we will consider below.

THEORETICAL PERSPECTIVES ON COGNITION-LONGEVITY ASSOCIATIONS

The obvious and important question is *why* cognitive performance across the life course is associated with disparities in survival. What processes underlie this relationship, for example? A variety of disciplines and researchers have investigated this question, often resulting in seemingly conflicting and controversial conclusions. Two primary perspectives will be considered: (1) societal inequity theory and (2) inherited genetic influences, each supported to varying degrees in the literature to date.

Social Inequities Create Similar Patterns of Disparity in Cognition and Health

There is an abundance of findings that social position, or socioeconomic status (SES), is related to health over the life course (Bassuk, Berkman, & Amick, 2002; Bengtsson & Mineau, 2009; Marmot & Smith, 1997). Further, SES seems to be important to health outcomes at all ranges, not just for individuals living in poverty (Adler et al., 1999). Low SES has been associated with increased risk for hazardous environments over the life course (i.e., prenatal, neighborhood safety, work; Adler et al., 1999; Adler & Snibbe, 2003), less access to healthy nutrition and health care, poor health behaviors, prevalence of chronic disease (Adler et al., 1999), and is often considered a chronic environmental stressor (Goodman, Adler, Wainryb, Smetana, & Turiel, 2008; Goodman, McEwen, Dolan, Schafer-Kalkhoff, & Adler, 2005), which may pose health risks through allostatic load (Adler & Snibbe, 2003; McEwen,

1998). Importantly, many of the factors linked to SES are also associated with cognitive performance and change in old age (e.g., Wilson et al., 2009). For example, individuals of higher SES are more likely to participate in healthier behaviors such as physical activity (Adler & Snibbe, 2003), which is not only important for health but also for cognitive function, including the maintenance of perceptual–processing speed, memory, and brain physiology. In a review of the literature, Churchill and colleagues (2002) found many examples of the relationship between physical exercise with cognitive outcomes and brain physiology. For example, there is evidence that high levels of physical fitness are related to improved maintenance of the frontal lobe and hippocampal regions compared to individuals with poor physical fitness (Lambert, Fernandez, & Frick, 2005). Further, the frontal lobe region, specifically the prefrontal cortex, has been associated with memory function and age-related declines in cognitive performance (Raz, 2000). Thus, SES is positively associated with behavior linked to both health and cognitive outcomes, suggesting that one's social position may exert influences to both health and cognitive function over the life course. A question that still needs to be addressed is why SES is related to behavior. Whether it is due to differential environmental stressors, social learning, access to different knowledge sources, cultural norms, or some unknown factor needs to be investigated further.

It is also important to consider the effect of education on health and its overlap with cognitive ability. Life expectancy has been steadily increasing, though the effect has been greater among individuals from higher SES, resulting in even larger longevity disparities over time (Goodman et al., 2008). This effect has been found in studies using education as a proxy for SES, but not in those that use income. Goodman et al. (2008) suggest that education might provide greater mental resiliency for dealing with stressors, while income is more important for material resources that may not have as strong an impact on dealing with life stressors. Further, education is strongly and positively associated with both level and rate of change in cognitive function (Anstey & Christensen, 2000; Katzman, 1993), is important for cognitive performance at all ages throughout adulthood (Farmer, Kittner, Rae, Barko, & Regier, 1995), and is associated with stimulating, healthier environments over time. However, the association between education and cognition is complex because there is also evidence that cognitive ability is predictive of educational attainment. For example, an individual with high cognitive functioning in childhood is more likely to achieve higher educational attainment and to have prolonged longevity (Deary et al., 2004).

Finally, because cognitive performance in late life is predictive of later survival, the literature suggests that educational activities may indirectly

benefit longevity by buffering cognitive decline through the development of cognitive reserve (Scarmeas, Albert, Manly, & Stern, 2006; Stern, 2002). Cognitive reserve refers to an individual's capacity for cognitive tasks that benefits from having higher intelligence early in life, and can be built up over the life course through stimulating mental activity. It is comparable to physical fitness, in that the more mental exercise one participates in over his or her life, the greater his or her mental functioning will be in late life. It is theorized that individuals with a large cognitive reserve are able to delay normative and pathological cognitive declines in late life. Research has found that the experiential influence of cognitive stimulation via education and environments across adulthood associated with educational attainment (e.g., complex work) attenuate age-related cognitive declines, even when one's brain physiology would predict major cognitive deficits (Brunner, 2005; Bunce & Macready, 2005; Churchill et al., 2002; Gribbin, Schaie, & Parham, 1980; Lovden, Ghisletta, & Lindenberger, 2005; Raz, 2000; Schooler et al., 2004; Shadlen et al., 2005; Stern, 2002). Thus, the development of cognitive reserve through a mentally active, engaged lifestyle is one process by which education may exert an influence on cognitive ability and health in adulthood. This results in SES-graded disparities in both cognition and longevity, in that SES is often defined by one's educational level.

Further, education may assist in the development and maintenance of cognitive reserve by having a direct influence on brain activity (Greenough, Larson, & Withers, 1985), by promoting access to cognitively stimulating environments (e.g., work environments), or by attenuating the effects of neurological decline (Shadlen et al., 2005). Shadlen and colleagues (2005) examined the influence of education on the relationship between apolipoprotein E (APOE) e4 allele on cognitive decline. The authors measured components of attention, memory, language, spatial ability, and executive function. The results indicated that individuals with only one APOE e4 allele did not display significantly different rates of cognitive decline compared to individuals without the allele. However, participants with two APOE e4 alleles experienced greater cognitive decline compared to the rest of the sample. Importantly, the magnitude of decline for individuals with two APOE e4 alleles was attenuated with increased years of education. Although educational attainment did not prevent cognitive decline, it was associated with slowed rates of decline. The influence of education remained after controlling for participant age and health indicators including diabetes and vascular disease. Applying the theory of cognitive reserve, individuals who attained more education were able to maintain a higher level of cognitive function in later life, potentially through activities and experiences associated with high educational attainment,

resulting in diminished rates of cognitive decline than their genetic predisposition would predict. Therefore, individuals who are genetically predisposed to experience severe cognitive decline were able to lessen the decline with education, an important component of SES. This process implies advantages for individuals with access to better education compared to those with fewer opportunities to achieve educational goals, which may also add to the cognitive associated disparities in health over time.

If social position in early life is associated with educational attainment (Deary et al., 2004) that predicts the experience of cognitively stimulating environments and experiences, the early differences in SES may magnify cognitive differences over time leading to later disparities in cognitive performance and longevity in late life. This is the scenario illustrated in part B of Figure 7.1. For example, educational attainment is associated with the level of occupational complexity one is likely to experience. Schooler, Mulatu, and Oates (1999, 2004) have found that cognitively complex, self-directed work experiences in adulthood are predictive of superior intellectual functioning even after retirement, which has been identified as a critical indicator of mortality risk (Schooler et al., 1999, 2004; Yu, Ryan, Schaie, Willis, & Kolanowski, in press). Thus, access to higher educational levels is often predictive of mentally challenging environments across the life course, thereby exacerbating disparities across the SES spectrum for cognition and health. However, an important caveat is the significant association of childhood intelligence on educational attainment, regardless of SES. Because children of high intelligence are more likely to attain higher educational pursuits, general intelligence needs to be considered as a possible resilience factor for individuals with few material or social resources in childhood.

However, a study by Van Dijk and colleagues (2008) has questioned the effect of education on cognitive performance in older adults. The authors examined a range of cognitive and neuropsychological assessments in a sample of 49 to 81-year-olds over a period of 6 years. Results indicated that although educational attainment was positively associated with baseline cognitive functioning, there was no evidence that individuals with higher education were protected against cognitive decline over time. The theory of cognitive reserve suggests that individuals with more years of education might delay the *onset* of normative cognitive decline and specifically pathological decline. In accord with this proposal, it is possible that in very old age, compared to those with less education, individuals with higher education show steeper rates of decline even though their reserve capacity allowed them to maintain adequate cognitive function for an extended period of time. Moreover, some tests may simply be more sensitive to assessing loss of functioning in a broad range

of normal performance. Van Dijk et al. (2008) considered these possibilities and thus examined three-way interactions with age, education, and timing on cognitive decline. However, they found no support for the delayed-onset of cognitive decline hypothesis. This study, which calls into question the impact of education on cognitive reserve, effectively highlights the inherent complexities involved in investigating aging, cognition, and longevity. In addition, the longitudinal findings of Van Dijk et al. (2008) could be confounded with unknown variations in distance from death among their participants, as discussed earlier in this chapter.

Genetic Influences on the Relationship Between Cognition and Longevity

While social inequities associated with SES across the life course is a viable argument for disparities in longevity associated with cognition, there is also significant evidence that genetic influences may underlie the association between cognition and survival in late life. Extant research has found evidence that several genes are associated with risk for cognitive decline and mortality (Perls & Harman, 2002). The prime example is the apolipoprotein E (APOE) e4 allele, which has been linked to Alzheimer's disease, a terminal illness defined by pathologic cognitive decline. It is also likely that, as opposed to health risks, certain genetic profiles are beneficial for both longevity and cognitive maintenance. For example, the APOE e2 allele is more prevalent in centenarians than in the general population (Perls & Harman, 2002), and healthier lipid profiles associated with a cholesteryl ester transfer protein (CETP) gene variant are also associated with extreme longevity and cognitive performance (Atzmon et al., 2005; Rushton, 2004). Thus, while the social inequity perspective outlines environmental and experiential pathways for developing disparities in longevity associated with cognitive performance, research in genetics indicates several potential inherited risk–protection factors for both cognitive performance and health. Further, as discussed earlier, the Shadlen et al. (2005) study provides evidence that the environmental and genetic influences are not independent, but rather operate in tandem as part of a larger system to influence both cognition and longevity.

A major critique of the argument that social inequities explain the association between cognition and longevity is that the availability of health care does not seem to diminish the disparities. The social inequity perspective would predict that if universal health care was available to individuals at all SES levels, the disparities in health and longevity would narrow. However, as Gottfredson (2004) has summarized, the research shows that in countries

where health care was made available to all individuals, the health disparity has actually grown. In these cases, individuals showed improved health; however, there seems to be an interaction with SES on the impact of available health care, such that those in the highest levels benefited more than individuals in lower social positions. Thus, instead of equalizing health discrepancies, the disparity associated with SES on health was actually exacerbated. Gottfredson (2004) and Gottfredson and Deary (2004) postulated that this seemingly counterintuitive pattern is due to the effect of general intelligence.

General intelligence, often referred to as "g" (Spearman, 1904, 1927), is a heritable trait (Bouchard, 1998; Plomin, DeFries, McClearn, & McGuffin, 2001) that Gottfredson (2004) argues influences a range of domains related to survival. It may explain why although individuals of low SES have shown improved health with societal introductions of universal health care, the SES-based disparities in health inequities have grown. One's generalized intelligence may act as a buffer, where the differences in an individual's longevity by social status may be driven by the underlying influence of intelligence. Cognitive ability and functioning in early life is an identified predictor of socioeconomic conditions across adulthood (Kuh et al., 2009). Additionally, Gottfredson (2004) argues that one's underlying general intelligence influences the effectiveness of understanding and utilizing health literacy information and affects one's ability to handle the complexities of health maintenance. For example, there is clear evidence that in older adults, one's cognitive function is positively associated with medical adherence (Insel, Morrow, Brewer, & Figueredo, 2006). Most older individuals take a variety of prescription and over-the-counter drugs, which creates highly complex medical adherence tasks. Prescription doses are often required at various times in the day, at differing frequencies per day, and with differences in how they are taken (e.g., with or without food). This results in a highly complex daily task for older adults, who benefit from higher cognitive performance in accurately adhering to the complicated medical tasks.

In reviewing the literature, Gottfredson (2004) also noted that regardless of equal accessibility across SES levels, those with lower education and income levels do not seek out and take advantage of preventative health care as often as individuals with higher education and income. Therefore, the advances in publicly available preventative care are dependent on individuals' behavior, and it is possible that the SES differences in this behavioral disparity are associated with underlying differences in general intelligence. For example, preventative health care is typically promoted through health literacy information. It is possible that individuals with lower intellectual abilities are not able to take advantage of health literacy information as easily as those with higher

intellectual function, resulting in disparate patterns of preventative health behaviors. Thus, Gottfredson (2004) concludes that individuals with higher general intelligence gain more benefits from health care and health literacy information and are better able to problem solve when dealing with complex health problems such as comorbid disease etiologies, compared to their lower intelligence counterparts. Intelligence may then act as a critical component of resiliency for individuals with few social and material resources.

Evidence in the aging literature suggesting that global cognitive performance is more strongly associated with survival than domain specific abilities (Andersen et al., 2002; Anstey et al., 2001; Maier & Smith, 1999) may reflect underlying cognitive reserve associated with general intelligence. This view is supported by the Scottish Mental Survey research by Deary and colleagues that finds an association between childhood intelligence and later survival. Bourne, Fox, Deary, and Whalley (2007) found evidence that childhood IQ is positively associated with late-life variation in cognitive functioning, such that children with high IQ tend to have slower rates of cognitive decline as older adults. This suggests that individuals with high general abilities in early life have better cognitive trajectories across the life course, which is then known to be important for mortality risk and longevity. This finding fits with Gottfredson's (2004) argument that general intelligence plays an important role in health and longevity across the life course, whether it be due to the effect of intelligence on the type of environments and experiences one has across adulthood, or as a resilience factor for individuals dealing with complicated health protocols.

The extant research in genetics provides several possibilities that explain the association between cognition and longevity. It is likely that a combination of genetic influences on health and intelligence play a role in one's cognitive performance across the life course as well as to life expectancy. However, it seems possible that the genetic influences on physiological outcomes, such as on one's lipid profiles or risk for specific diseases such as Alzheimer's disease, may play a more important role for premature death and extreme longevity. Alternately, inherited general intelligence may be an important risk factor (at low levels) or protective factor (at high levels) that plays an integral role for interindividual differences in cognitive trajectories and longevity within the normative range. While to our knowledge the differential genetic effects of physiology and intelligence on cognition and longevity trajectories have not been examined directly, some support for this hypothesis exists. Using data from the Berlin Aging Study, Maier and Smith (1999) examined a variety of psychosocial factors relative to mortality risk. The results indicated that perceptual speed was negatively associated with mortality risk for the entire sample (age range 70–103 years). However, when the sample was divided into

an old age group (70–84) and oldest-old group (85+), the effect of perceptual speed was not a significant predictor of mortality for the oldest participants. This fits with the hypothesis that individuals at extreme ends of the longevity spectrum are not as affected by cognition compared to individuals within typical life expectancies. Alternately, it is possible that the genetic effects are additive, such that individuals who survive beyond age 85 may be genetically advantaged relative to both intelligence and physiology, whereas those who survive to typical ages have genetic profiles comprised of a combination of risk and resilience, and those who die prematurely have genetic profiles of risk associated with both domains. These hypotheses need to be addressed in future research.

An important critique of the genetic impact of intelligence on longevity is that much of the support is from studies that examine mean trends at the aggregate level and do not necessarily account for individual differences. And while genetics are clearly an important consideration for the association with cognition and disparities in longevity, other evidence illustrates the effects of environmental experiences over time, as well as gene-environment interactions. The Shadlen et al. (2005) study discussed earlier is an excellent example. While the results showed a direct association between genetic risks for cognitive decline among APOE e4 allele carriers, the effect was moderated by the environmental influence of education. Therefore, genetic predispositions for cognitive performance and longevity may feed into one's risk and/or resiliency, but does not account for all the variance in the association between cognition and longevity.

In addition, Dickens and Flynn (2001) argue that traditional heritability underestimates the effect of the environment, which when measured appropriately can explain the curious pattern of generational gains in IQ. Dickens and Flynn (2001) suggest that the gains in IQ over time are the result of complicated environmental, and gene-environment effects that are frequently missed in heritability estimates. One gene-environment interaction of particular relevance to Gottfredson's (2004) and Gottfredson and Deary (2004) intelligence hypothesis is a multiplier effect. Dickens and Flynn (2001) describe multiplier effects as a situation when one's inherent genetic predisposition is paired with a matching environment. Using the intelligence example, this would occur when an individual of high IQ is exposed to environments rich in mental stimulation or when an individual of low IQ experiences low complexity, minimally stimulating environments. The multiplier effect predicts that the paired genetic-environmental influences will exacerbate the original differences in IQ, resulting in a combined genetic-environment influence stronger than a simple additive effect. When considering the known

association between cognition and longevity, the possibility of multiplier effects that will exacerbate one's intelligence-associated risk or resiliency on longevity may help to explain the individual differences in the association. Similarly, Ferraro and Shippee (2009) argue that a combination of genetic and experiential influences over the life course create cumulative inequalities, placing a strong focus on family history on the development of later disparities.

The theory of cumulative inequalities fits nicely with genetic-environmental multiplier effects, where the pairing of inherited and experiential influences magnifies inequalities across a range of domains over time. With this in mind, it seems reasonable to conclude that the effect of inherited physiological and intelligence factors on later longevity may explain the aggregate trends, while the social inequities in material and social resources associated with SES may operate to moderate one's genetic predispositions and to create individual differences over time.

OUTLOOK

One agenda for this chapter was to attempt to build a bridge between individual-level research on cognitive aging and population research on health disparities. Although life-course and life-span perspectives encourage this integration (see Chapter 1), in practice such attempts are rare at least with regard to nonpathological cognitive functioning. As more longitudinal studies follow panels of heterogeneous populations over many years, researchers will be in a better position to decompose the multiple factors underlying the associations between cognition, health, and longevity in late life. Future work may determine if any of the three conceptual scenarios illustrated in Figure 7.1 is the best representation and also examine the life-course effects of cohort improvements in average levels of early-life education and preventive health systems. The contemporary data on disparities in late-life health and longevity is primarily derived from cohorts born prior to 1930. It will be several decades before robust comparative data from subsequent birth cohorts are available. By highlighting the need for multidisciplinary investigations in the study of health disparities that incorporate macrolevel societal influences and individual-level factors such as cognitive function, we hope to promote a research direction that will enhance the field of disparities research.

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SECTION IV

FUNCTIONAL LIMITATIONS AND RESPONSES TO STRESS IN LATE LIFE

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CHAPTER 8

Chronic Stress and the Role of Coping Behaviors in Health Inequalities

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Growing evidence suggests that inequalities in chronic health morbidities and mortality among race and ethnic groups are larger in middle age and later stages of the life course than earlier (e.g., Hayward, Crimmins, Miles, & Yang, 2000). The purpose of this chapter is to explore physiological profiles that may be associated with a range of coping behaviors and strategies to address chronically stressful environments used by different race–ethnic groups, including religiosity–spirituality, mastery, physical activity, meditation, and social support. Research examining the relationship between positive coping behaviors and physiological responses to chronic stress, assessed primarily as differences in the diurnal cortisol pattern in naturalistic settings, by age and race–ethnic group are reviewed.

This review is set against the backdrop of a puzzling epidemiological pattern that reveals that while African Americans have poorer physical health and mortality outcomes than non-Hispanic Whites, for almost all mental disorders they have lower rates (Jackson & Knight, 2006). Lower SES and certain at-risk race–ethnic groups have higher rates of coronary heart disease (Luepker et al., 1993), chronic kidney disease (Merkin et al., 2007), and diabetes (Rabi et al., 2006). There is no simple explanation for why these disparities exist, but it is evident that several risk factors contribute to these

inequalities (Gilman, Abrams, & Buka, 2003; Steptoe & Marmot, 2002; Williams, 1990). We hypothesize that when individuals are confronted with the chronically stressful living conditions that accompany poverty (e.g., lack of safety and basic comforts) they will engage in behaviors that help to alleviate the resulting symptoms of stress. For instance, alcohol consumption may help to quiet the physical feelings of anxiety as well as interrupt intrusive and/or burdensome cognition, such as worry, rumination, or feelings of failure. The same behaviors that help to alleviate stress in the short-term, however, may silently erode physical health in the long-term. Whereas unhealthy behaviors reduce immediate symptoms of stress, while possibly also altering the biological cascade leading from stress to mental disorders, they ultimately interact with poor living conditions to contribute to the development of chronic and degenerative physical health disorders (Jackson, Knight, & Rafferty, 2009).

Recent research has generally focused on the short- and long-term psychophysiological consequences of unhealthy coping behaviors. Little is known about the health affects of healthy lifestyles and behaviors under chronically stressful situations. The objective of this review is to systematically examine existing evidence regarding the association between positive coping strategies and chronic stress, primarily assessed as cortisol secretions, over the life course. The mental health protective role of risky health behaviors that are likely to serve as strategies for coping with chronic stress has previously been reviewed elsewhere, therefore, is not fully addressed in this chapter (Jackson & Knight, 2006). In this review, we focus on a wide range of positive coping behaviors that have received substantial consideration in the health literature: religiosity–spirituality, mastery, physical activity, meditation, and social support.

CHRONIC STRESS AND THE HPA AXIS

Chronic physical and psychological stressors are risk factors for an array of chronic conditions, including coronary heart disease, metabolic disorders, and fibromyalgia (Bierhaus, Humpert, & Nawroth, 2006; Holmes, Krantz, Rogers, Gottdiener, & Contrada, 2006; Van Houdenhove & Luyten, 2006). The hypothalamic-pituitary-adrenal (HPA) axis coordinates the physiological response to stressors through a complex brain–body feedback loop of hormone release. This system evolved to mobilize critical energy necessary for responding to physical threats, but responds identically to psychological stress. Specifically, stress exposure stimulates a cascade of hormone releases beginning in the brain and moving downstream to the periphery: corticotropin-releasing factor (CRH) is released from the hypothalamus, adrenocorticotropin hormone

(ACTH) from the pituitary gland, and cortisol from the adrenal gland. These secretions induce increased respiration and cardiac output, redirecting blood flow to increase perfusion and provide energy to the brain, heart, and muscles, while also minimizing the body's parasympathetic or restorative functions, such as digestion, growth, and healing.

The stress response system ensures that the body is well equipped to handle acute stressors (e.g., running from an attacker). Once the threat is gone, hormone secretion is reduced, respiration and cardiac output slow down, and blood flow returns to normal, restoring homeostasis in the body. The body is not, however, well suited to handle chronic stressors, such as stressful life events, as in the death of a loved one, or daily struggles and hassles, including stressful work environments, marital problems, or discrimination. Exposure to chronic stress results in repeated and prolonged activation of the HPA axis. Long-term and chronic hyperactivation of the HPA axis has been linked to decreased well-being and psychopathology, such as melancholic depression, anorexia nervosa, and obsessive-compulsive disorder (Tsigos & Chrousos, 2002).

There is an extensive literature showing that coping methods are largely dictated by the nature of the problem, with changeable or controllable situations eliciting more problem-focused coping behaviors and unchangeable or uncontrollable situations eliciting more emotion-focused coping efforts (Master et al., 2009; Taylor & Stanton, 2007). Uncontrollability is a defining feature of impoverished circumstances, suggesting that emotion-focused coping strategies should be more adaptive and effective among disadvantaged groups. Consistent with this idea, individuals may engage in coping behaviors that either mute the HPA response itself or, at least, mask the symptoms of a hyperactivated HPA axis. Extensive laboratory and field research has been conducted to better understand this process (Ebner, Wotjak, Landgraf, & Engelmann, 2005; Koh, Choe, Song, & Lee, 2006; Koolhaas et al., 1999; Tull, Sheu, Butler, & Cornelious, 2005; Walter et al., 2006).

Studies have most commonly used the hormone cortisol as a relatively objective measure of physiologic stress. There are several reasons why cortisol is effective as a stress biomarker. Serum levels can be accurately estimated from saliva and urine, making the collection of cortisol data effective and minimally invasive (Kirschbaum & Hellhammer, 1994). In fact, salivary cortisol is frequently collected by research participants themselves. Various types of cortisol measurements are used in the literature, including early morning peak, mean diurnal levels collected over multiple observation points, diurnal slope, and cortisol reactivity and recovery. Though the clinical significance of particular levels of cortisol are not well understood, we know that cortisol

follows a very specific diurnal pattern, beginning with a peak just before waking followed by a steep drop by late afternoon through early evening to almost undetectable levels by the end of the day. This well documented diurnal pattern facilitates comparison and synthesis across studies, even when the particular type of cortisol measure differs.

RELIGIOSITY–SPIRITUALITY AND CHRONIC STRESS

While several studies have evaluated the association between positive health outcomes and religiosity (Fallot, 2007; Krause, 2006; Mofidi et al., 2006; Williams & Sternthal, 2007), there is less research that explicitly examines the relationship between religion and the physiological response to chronic stress. Findings are mixed and existing evidence demonstrates that the association between religiosity and cortisol specifically is dependent on how both religiosity and cortisol are measured.

For example, a recent study examined the association between religiosity–spirituality and diurnal salivary cortisol rhythms in women with fibromyalgia (Dedert et al., 2004), a chronic rheumatic disorder believed to flatten the diurnal cortisol rhythm (Gupta & Silman, 2004). There were statistically significant correlations found between diurnal cortisol slope and each of the religiosity and spirituality categories measured: nonorganizational, organizational, intrinsic religiosity, and spirituality. In each case, higher religiosity–spirituality was associated with a steeper negative diurnal cortisol slope, suggesting a more normal cortisol rhythm. There were no significant correlations between these measures and diurnal mean cortisol. After controlling for age, use of antidepressants, and use of oral contraceptives, only nonorganizational and intrinsic religiosity were significantly associated with smaller diurnal cortisol slope. Furthermore, after controlling for social support, only intrinsic religiosity remained significant.

Another study (Ironson et al., 2002) examined cortisol as a biological mechanism linking spirituality–religiosity and long-term survival in people with HIV/AIDS. Researchers found a significant correlation between the Ironson-Woods Spirituality/Religiousness Index and urinary cortisol concentration. This composite measure of spirituality–religiousness consists of four subscales: sense of peace, faith in God, religious behavior, and compassionate view of others. Of the four, only sense of peace and faith in God were significantly correlated with total urinary cortisol concentration.

Three more recent studies, all based on urinary cortisol levels, found no direct association of religiosity to cortisol. A study of older Taiwanese adults

found no significant associations between religious beliefs, religious practices, or religious service attendance and urinary cortisol output (Yeager et al., 2006). Another study of older U.S. adults had similar findings in their assessment of the relationship between religious service attendance and high urinary cortisol levels (defined as the upper quartile of the sample; Maselko, Kubzansky, Kawachi, Seeman, & Berkman, 2007). A path analysis of the association between spirituality–religiosity, measured by the Ironson-Woods Spirituality/Religiousness Index, and urinary cortisol in HIV-positive individuals, found no significant direct link to cortisol output, but a significant indirect association through individuals' perceptions of positive benefits found by living with HIV/AIDS emerged (Carrico et al., 2006). Specifically, spirituality–religiosity was significantly associated with higher positive benefits or life changes experienced due to living with HIV infection, and perceived benefits was in turn significantly associated with lower cortisol output.

These findings provide some evidence that certain types of religiosity–spirituality are effective means of reducing the physiologic effects of stress as measured by cortisol levels. More intrinsic beliefs, like having strong faith, may have effects on the physiological stress response, while attending religious services do not have the same effects. Further work must be done in this area to determine whether attitudinal and behavioral aspects or spirituality and religiosity are effective coping strategies and whether the effectiveness of these strategies differs among social groups over the life course. One hypothesis is that spirituality–religiosity would be particularly effective during points in the life course when high levels of social support are needed, but fewer sources are available, such as the early child-rearing years and late life.

Consistent with this role by age regulation, use of religiosity–spirituality as a coping resource tends to vary over the life course. Research dating back to the early 1980s has consistently shown a decline in religious service attendance and religious affiliation in young adulthood (Uecker, Regnerus, & Vaaler, 2007; Willits & Crider, 1989). A more recent longitudinal study found that 69% of young adults attended religious services less frequently than they did as adolescents, and 20% reported a decline in the importance of religion in their lives. Not attending college, frequency of sexual activity, frequency of alcohol consumption, and marijuana use were all significant predictors of these declines. Factors such as marriage and child rearing are linked with a resurgence of religious service attendance in middle adulthood (Ingersoll-Dayton, Krause, & Morgan, 2002; Uecker et al., 2007). In older adulthood, while mobility limitations and other factors may limit religious service attendance, some research suggests an increase in nonorganizational religious

participation and a deeper intrinsic faith among this older group (Ingersoll-Dayton et al., 2002; Levin & Chatters, 2008).

MASTERY AND CHRONIC STRESS

Mastery is the extent to which people believe they themselves are in control of their lives (Pearlin, Menaghan, Lieberman, & Mullan, 1981). In one study, researchers examined correlations between psychosocial factors and salivary cortisol levels in a sample of married women with breast cancer and healthy married female controls (Vedhara, Tuinstra, Miles, Sanderman, & Ranchor, 2006). Among the control subjects, researchers found that a greater sense of mastery was significantly correlated with lower total cortisol secretion. Among the breast cancer patients, a higher sense of mastery was significantly correlated with a lower early morning cortisol peak but not with total cortisol secretion. An important implication of this finding is that diurnal cortisol patterns may differ as a function of disease state.

A second study, using data from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, moved beyond simple correlation analysis and examined whether mastery is a conceptual link between measures of SES and several measures of salivary cortisol levels (33). Socioeconomic status was measured separately as income and education. After controlling for sex, race, age, body mass index, wake-up time, and time between sample collection time and awakening, researchers found a significant partial correlation between diurnal cortisol slope and mastery. Multiple mediation analysis, however, showed that mastery was not an independent mediator after accounting for other mediators, including smoking and social network diversity.

There is limited evidence that feelings of high mastery can effectively regulate cortisol output. More studies are needed to better understand whether or not mastery can reduce the body's response to chronic stress. Studies of other measures of personal control may also help to better elucidate the role of this type of coping behavior in altering the stress response.

Both cross-sectional and longitudinal studies have generally shown that the utilization of personal control as a coping resource decreases with age (Cairney, Corna, Wade, & Streiner, 2007). This is believed to be due to a combination of increased exposure to negative life events, increased health problems, and more frequent contact with the health care sector (Rodin, 1986). Thus, in older age, a sense of personal control may no longer be a viable coping resource for many people. This may be particularly true for those who were exposed to more stressors and hardships early in life. Pearlin and colleagues found that life-course mastery, a feeling of having been in control

over one's life trajectory, predicted current mastery in older adults (Pearlin, Nguyen, Schieman, & Milkie, 2007). Life-course mastery was significantly lower in those of lower socioeconomic status and those who experienced more stressors and hardships. Therefore, among older adults who feel they have less control over their current situations, those who are less affluent or who experienced more hardships will be even less likely to effectively use mastery as a coping resource than their more advantaged counterparts.

PHYSICAL ACTIVITY AND CHRONIC STRESS

Although physical activity is itself a physiological stressor and therefore stimulates activation of the HPA axis (Emery, Kiecolt-Glaser, Glaser, Malarkey, & Frid, 2005; W. J. Kraemer et al., 1989; Petraglia et al., 1988), there are two major routes through which exercise is believed to reduce the physiological response to chronic stress. Some studies have suggested that exercise counteracts the effects of stress by altering the release of serotonin and norepinephrine (Dishman et al., 2006). This is known as the monoamine hypothesis, and there is some empirical support for this theory in the literature. For example, one study showed that middle distance interval training in rats results in *increased* serotonin and norepinephrine levels (Morgan, 1985). Low levels of these two neurotransmitters are associated with depression, so it is hypothesized that exercise increases brain levels of one or both, thereby reducing the negative mental health consequences of prolonged stress. Some have suggested, however, that this explanation may be an oversimplification of the role of exercise in preventing or reducing the causal pathways to depression (Paluska & Schwenk, 2000).

More recent research suggests that physical activity alleviates the physiological response to stress by stimulating the release of β-endorphin and β-lipotropin (Kelso, Herbert, Gwazdauskas, Goss, & Hess, 1984; Mastorakos & Pavlatou, 2005). Beta-endorphins are released at the same time as ACTH in response to stress, and they have been shown to produce a euphoric state and reduce pain. Release of these chemicals may make physical activity a successful coping strategy, despite its activation of the HPA axis. Findings have not been consistent, however, and it appears that β-endorphin secretion may be related to duration and intensity of exercise. In one study, plasma β-endorphin and corticotrophin-releasing hormone (CRH) levels increased significantly before and immediately after elite runners participated in a 15 kilometer run (Harte, Eifert, & Smith, 1995). Conversely, another study found no significant change in plasma β-endorphin secretion among trained

and untrained subjects after subjecting them to a 30 minute run at 80% of their maximum heart rate (Kraemer, Blair, Kraemer, & Castracane, 1989). Other studies have found that β-epinephrine levels increased significantly after exercise only at high intensity levels (>90% of maximal oxygen consumption; Petraglia et al., 1988; Rahkila, Hakala, Alen, Salminen, & Laatikainen, 1988), though one group found that even within this intensity level there is evidence of variation in the response of β-epinephrine depending on the percentage of maximal leg power exerted (R. R. Kraemer et al., 1989).

Both the monoamine hypothesis and research on the relationship between exercise and the release of β-endorphins indicate that participation in intense levels of physical activity is likely an effective coping strategy for alleviating the physiological response to chronic stress, though the exact mechanisms remain unclear. Further work is needed to understand the potential effects of other types of exercise, like walking or participation in recreational sports, on the stress response pathway.

Moderate and vigorous physical activity steadily declines over the life course in both men and women (Hawkins et al., 2009; Jones et al., 1998; U.S. Department of Health and Human Services & Centers for Disease Control and Prevention, 2005). This may be due to a variety of factors including time constraints, decreased interest, injuries preventing participation in preferred activities, and mobility limitations. This suggests that even if exercise is effective, it is not generally the main coping resource of choice throughout an individual's life course. As with all coping strategies, use of exercise may differ by race–ethnicity, socioeconomic status and interactively with these statuses at different points in the life course.

MEDITATION AND CHRONIC STRESS

Meditation is another behavior that may reduce stress and be associated with positive health outcomes. Two studies have examined the health effects of Mindfulness-Based Stress-Reduction (MBSR), a complementary and alternative medicine practice developed by John Kabat-Zinn that combines principles of mindfulness meditation, breathing exercises, and yoga. In a study of women with cardiovascular disease, half of the subjects were randomly selected to participate in an 8-week MBSR program while the others served as controls (Robert-McComb, Tacon, Randolph, & Caldera, 2004). Resting morning fasting serum cortisol levels were lower among the MBSR participants versus the controls after the 8-week intervention, but the difference was not statistically significant. Researchers felt this may be due to their small sample size, or the fact that an 8-week program was not long enough for participants to receive

the full protective benefits of MBSR. A study of breast and prostate cancer patients enrolled in an 8-week MBSR intervention found that while mean cortisol levels did decrease, neither mean daily cortisol levels nor diurnal cortisol slopes differed pre- and postintervention (Carlson, Speca, Patel, & Goodey, 2004).

Three other studies have focused on the Transcendental Meditation (TM) program, a technique typically practiced for 20 minutes twice a day to reduce stress. Each of the studies compared a healthy group who had been practicing TM for an average of 7 years or more, to a nonmeditating control group. Researchers in one study found no difference in morning or evening plasma cortisol levels in the TM group and the control group (Infante et al., 1998). A study that measured morning and overnight urinary cortisol excretion found that levels were lower in the TM group compared with the control (Walton, Pugh, Gelderloos, & Macrae, 1995). Among women in the TM group, researchers found that cortisol levels were inversely correlated with the number of months they had been practicing. Another study compared the salivary diurnal cortisol rhythm in a TM group and a control group after the administration of oral glucose, which served as a metabolic stressor (Walton et al., 2004). The cortisol levels after a bolus dose of glucose was significantly higher in the control group, suggesting that regular TM practice may reduce the impact of metabolic stressors on the HPA axis. These two groups did not differ significantly on demographic or lifestyle variables (e.g., age, income, smoking), further strengthening the validity of these results.

These studies suggest that meditation may reduce the harmful effects of stress on the HPA axis. The finding that results were not significant for participants of 8-week MBSR interventions, but were significant for regular TM practitioners, suggests that meditation may only work as a buffer after long-term, consistent use. Future studies are needed to rule out the possibility that some other difference in coping behaviors, such as cigarette smoking or alcohol consumption, exist between the TM and MBSR groups that could be confounding the relationship between meditation and cortisol.

It is not clear how meditation practice varies or is differentially effective over the life course by all social and economic groups. The utilization of TM as a coping resource most likely begins in adulthood, but it can also be practiced by children. It has been shown to benefit physical and mental health in all age groups (Anderson, Liu, & Kryscio, 2008; Barnes, Davis, Murzynowski, & Treiber, 2004; Eppley & Abrams, 1989). However, the high cost of TM training may be prohibitive for those of lower socioeconomic status.

SOCIAL SUPPORT AND CHRONIC STRESS

Research on social support is part of a vast literature assessing the association between personal relationships, broader social networks, and health and well-being. Social support includes resources such as advice, service, assistance, and positive affect (Sherbourne & Stewart, 1991). The range of specific theoretical foci of past research include the causes and consequences of quality and quantity of relationships, the differential benefits of perceived versus received support within networks, and assessments of whether support operates as a direct effect or as a buffering process.

Though several studies have examined the relationship between social support and chronic stress, the findings are mixed (Uchino 2006; Uchino, Cacioppo, & Kiecolt-Glaser, 1996). There is some evidence that a larger social network is significantly associated with lower diurnal cortisol slope and urinary cortisol output (Cohen et al., 2006; Seeman, Berkman, Blazer, & Rowe, 1994; Yeager et al., 2006). A study on the size and quality of instrumental and emotional support, however, found no association between size of support network and mean salivary cortisol levels, though they did find that lower quality instrumental support was associated with higher mean cortisol (Kim & Knight, 2008).

Findings are mixed for studies of emotional support and cortisol output as well. One study using several measures of urinary cortisol found no association with emotional support (Cohen et al., 2006), while another found a significant inverse association (Seeman et al., 1994). Another study exploring the relationship between social support, as measured by the MOS Social Support Survey, and salivary cortisol levels measured quarterly over the course of a year did not find significant cross-sectional correlations between social support and mean log morning or evening cortisol levels (Rosal, King, Ma, & Reed, 2004). They did, however, find a significant inverse correlation with basal morning cortisol levels after adjusting for self-reported stress levels. Longitudinal analyses of these quarterly cortisol measurements also revealed the same inverse relationship, in which participants in the upper tertile of perceived support had significantly lower cortisol levels than those in the lower two tertiles. A study of high and low levels of social support at work and its relationship with cortisol release found that mean daytime salivary cortisol levels were *lower* in the low work-support group in comparison with the high work-support group and that mean evening cortisol levels were generally lower or the same for the low versus high work-support group (Evans & Steptoe, 2001).

Several studies have explored the direct role of social support in the physiological response to chronic stress in women specifically. In a study of the effects of marital and parental status on stress levels in working women, researchers found no association between perceived support and salivary cortisol levels (Luecken et al., 1997). Researchers studying the relationship between social support and salivary cortisol in women with metastatic breast cancer found that mean diurnal cortisol was significantly, inversely correlated with perceived support, belonging support, and tangible support, but not self-esteem support or size of social network (Turner-Cobb, Sephton, Koopman, Blake-Mortimer, & Spiegel, 2000). Diurnal cortisol slope, on the other hand, was not significantly correlated with any of the support measures. A study of the correlation between perceived support and HPA axis functioning in pregnant women found a significant inverse correlation between social support and plasma cortisol levels (Wadhwa, Dunkel-Schetter, Chicz-DeMet, Porto, & Sandman, 1996). A study of 5-day postpartum mothers, however, found no association between social support and cortisol levels measured from breast milk (Groer, Humenick, & Hill, 1994).

Other research on social support has explored the buffering hypothesis, which suggests that the effect of social support is not necessarily direct, but rather is most evident by moderating the effect of stressful conditions or the stress process. Studies suggest that social support may buffer the physiological stress response through the oxytocin-induced suppression of the HPA axis (Knox & Uvnas-Moberg, 1998). Oxytocin is a neurohypophysial peptide hormone known to play a role in mating, parturition, lactation, and maternal behavior. Lactation studies have shown that shortly after breastfeeding, mothers have increased oxytocin levels and decreased cortisol levels in response to stressors compared with those who have not recently breastfed (DeVries, Glasper, & Detillion, 2003).

This theory is weakened by the fact that it remains unclear whether or not oxytocin actually suppresses the HPA response. A laboratory study of couples engaging in instructed conflict discussion after inhaling either oxytocin or placebo found that those who inhaled oxytocin had significantly lower salivary cortisol levels than those who were given the placebo (Ditzen et al., 2009). Conversely, a study that examined the role of social support and intranasally injected oxytocin in the cortisol-mediated stress response found that salivary cortisol levels increased the least in response to a laboratory stressor among those who had both social support from a friend and had inhaled oxytocin (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). A study of oxytocin and cortisol response to an acute stressor found that those with higher plasma

levels of oxytocin, both before and after exposure to the stressor, had higher salivary cortisol levels (Taylor et al., 2006).

Whether through direct or indirect pathways, as in moderating the release of oxytocin, there is weak evidence to suggest that social support is a coping strategy that leads to a muting of cortisol release in response to chronic stress. Further work is needed to better elucidate the aspects of social support that are more important as strategies to cope with chronic stressors (e.g., a large social network, or higher quality support received regardless of quantity). In addition, more research is needed to develop a better understanding of the physiological mechanisms through which social support may exert its mental and physical health benefits.

The size of social networks and the frequency of contact with network members vary with age. In general, older adults tend to have smaller social networks and have less frequent contact with members than younger adults, though this varies depending on the way social networks are measured (Ajrouch, 2001; Mardsen, 1987; Morgan, 1988). This may be because of growing time constraints as well as an increasing awareness of the limited amount of time they have left to live. Research suggests that this awareness may lead older adults to build deeper relationships with a small group of friends they are emotionally close with in favor of maintaining a large network (Krause, 2005). These changes in the nature of social support in later life may influence its utilization and effectiveness as a coping resource over the life course among different socioeconomic and race–ethnic groups (Carstensen, 1993), but more work among diverse groups at different points in the life course are needed.

CONCLUSIONS

This review of the available literature found that positive coping behaviors appear to have limited effects on brain–body mechanisms shown to be associated with less frequent or muted activation of the HPA axis and related hormonal responses. Across different studies the findings were not consistent, but there is weak evidence suggesting some beneficial physiological effects. More research is needed to better understand whether positive coping strategies play a significant role in alleviating the body's response to chronic psychological stress, particularly in different race–ethnic and socioeconomically disadvantaged populations, and what the ultimate outcomes may be for health differentials in later life. Future research is needed to better understand whether or not positive coping behaviors can work effectively to insulate, buffer, or moderate the effects of chronic stressors on disease outcomes and

chronic health conditions, especially through the HPA axis and related hormonal pathways. Our review suggests that intrinsic religious faith may help regulate chronic stress via the HPA axis, but there is insufficient evidence at this time to conclude that mastery is an effective coping strategy. Intense physical activity and long-term transcendental meditation both show promise, and while social support shows some promising results, the findings are quite inconsistent.

Ultimately, it is critical to consider the realistic viability of various coping behaviors. Socioeconomic status and the nature of the social environment, especially in large urban areas, simultaneously help to afford greater opportunities for engaging in negative coping behaviors (e.g., smoking, drinking alcohol, and drug use) and fewer opportunities for relying on positive coping alternatives as discussed in this chapter. Several studies report that lower SES individuals have a more limited set of coping resources at their disposal compared to those with more income or education (McEwen, 1998; McLeod & Kessler, 1990; Mirowsky & Ross, 1986; Myers, Lindenthal, & Pepper, 1975; Pearlin & Schooler, 1978). This suggests that some coping choices may not be readily available to poor populations, particularly those living in urban centers. Limited access to exercise and public recreation facilities, and perceived poor neighborhood safety have been linked to reduced physical activity levels and increased obesity in lower income neighborhoods (Powell, Slater, Chaloupka, & Harper, 2006; Wilson, Kirtland, Ainsworth, & Addy, 2004). Lower levels of education, unskilled and semiskilled versus professional occupations, and lower levels of income, have all been associated with lower levels of emotional support, less contact with friends, and lower frequency of disclosing problems (Mickelson & Kubzansky, 2003; Turner & Marino, 1994; Williams, 1990).

At the same time, people of low SES and traditionally disadvantaged ethnic minority populations are more likely to smoke, use alcohol, and be obese (Gilman et al., 2003; Robert & Reither, 2004). They are more likely to live in close proximity to liquor stores (LaVeist & Wallace, 2000). They are also more likely to have limited access to healthy foods, putting them at greater risk for obesity and obesity-related diseases (Moore & Diez Roux, 2006; Robert & Reither, 2004). This suggests that the environments in which the urban poor live may facilitate certain negative coping mechanisms that are effective at relieving psychological symptoms of stress over more positive behaviors that may be less accessible to these populations, especially in middle and older ages (Jackson & Knight, 2006).

Comprehending the effectiveness of potential stress reducing strategies has important implications for understanding the epidemiology of chronic

diseases that are more prevalent among low SES and certain race and ethnic groups. A variety of coping strategies may help reduce the body's response to chronic stressors; some linked to those having negative health consequences and others linked to more neutral or positive health outcomes. It is clear that more research needs to be conducted to understand the role of positive coping behaviors in alleviating the physiological responses to stressful events among different social and economic groups, and at different ages and at different points in the life course.

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CHAPTER 9

Arthritis and Health Inequalities in Blacks and Latinos

Jessica M. McIlvane

Arthritis is one of the most common chronic conditions and a leading cause of disability in the United States affecting individuals across the life span from childhood through old age (Arthritis Foundation, 1999; Centers for Disease Control and Prevention [CDC], 2009a). Racial–ethnic disparities in arthritis symptoms and access to treatment in adulthood and older age are well documented. Blacks and Latinos with arthritis are at an increased risk for more severe arthritis-related symptoms such as severe pain (CDC, 2005) and activity–functional limitations (Abraído-Lanza, White, Armbrister, & Link, 2006; CDC, 2005; Hootman, Sniezek, & Helmick, 2002; Kington, & Smith, 1997) and are less likely to have joint replacement surgery (CDC, 2009b; Dunlop, Song, Manheim, & Chang, 2003; Escalante, Espinosa-Morales, del Rincón, Arroyo, & Older, 2000). Moreover, there is evidence for poorer quality of life in Blacks (Ibrahim, Burant, Siminoff, Stoller, & Kwoh, 2002) and more distress in Latinos (Escalante, del Rincón, & Mulrow, 2000) with arthritis compared to Whites with arthritis.

Little research examines reasons for disparities in Blacks and Latinos with arthritis. This is surprising given that arthritis is so prevalent across the life span and may impact quality of life. It is likely that a combination of factors contribute to arthritis disparities including socioeconomic status (SES), access to health care, chronic stress, illness perceptions, and mistrust of the health care system. With the aging of the baby boomers and the increasing racial–ethnic diversity of our older population, these concerns will increase in importance

as public health issues. Unfortunately, there is a dearth of research focusing on these issues among Blacks, and particularly among Latinos, with arthritis.

This chapter will discuss health inequalities in Blacks and Latinos with arthritis. First, this chapter begins with basic definitions and information about arthritis and discusses why it is important to consider arthritis inequalities. Second, current research on racial–ethnic disparities in arthritis prevalence, symptoms, and treatment will be discussed. Third, although not well understood, potential contributors to arthritis health disparities will be considered (e.g., barriers to access to care, mistrust of medical system, illness perceptions). Fourth, this chapter will consider protective factors (e.g., religion–faith, coping, family support) that may be beneficial particularly to minority group members. Finally, suggestions for future directions will be offered.

WHAT IS ARTHRITIS, AND WHY ARE ARTHRITIS INEQUALITIES IMPORTANT?

While there are over 100 different types of arthritis and rheumatic disease, this chapter will focus primarily on osteoarthritis (OA), the most common type of arthritis that tends to affect people in older age. Racial–ethnic disparities exist in other types of arthritis affecting individuals across the life span, such as lupus and rheumatoid arthritis (RA; see Jordan et al., 2002 for review), but this chapter will primarily review research that examines OA or arthritis in general. Osteoarthritis involves degeneration of cartilage in the joints that leads to the slow progression of joint pain, stiffness, and limitation of motion. The hand, knee, hip, and spine are the joints most commonly affected by OA. Arthritis is associated with increased pain, functional impairment (National Institute of Arthritis and Musculoskeletal and Skin Diseases [NIAMS], 2002), poorer health-related quality of life (Dominick, Ahern, Gold, & Heller, 2004), and higher risk of depression (Dunlop, Lyons, Manheim, Song, & Chang, 2004). Because people with arthritis often have functional limitations, joint pain, and stiffness, it may be challenging to do everyday tasks including household chores, gardening, taking a walk, or picking up a young grandchild. People with arthritis may experience difficulties in multiple domains of life including interpersonal relationships, tasks at work or home, and leisure-related activities (Gignac et al., 2006).

Arthritis involves not only a personal impact on the individual and family but also significant economic costs for society. In 2003, arthritis and other rheumatic conditions accounted for a staggering $128 billion in lost wages and medical costs (CDC, 2007b). These costs are expected to increase

further due to the combination of a rapidly aging population, increased rates of obesity, and physical inactivity (CDC, 2007b). The number of adults with arthritis (age 18+) is projected to increase from 47.8 million in 2005 to 67 million in 2030 (Hootman & Helmick, 2006). In 2030, 61% of arthritis cases will be women (40.9 million) and the percentage of arthritis cases that are older adults will increase from 37.2% in 2005 to 51% of cases in 2030. Thus the projected increases in the number of adults with arthritis are in large part due to high arthritis prevalence rates in older adults.

While the bulk of this chapter will focus on arthritis disparities in older adulthood, arthritis is not just an old person's disease. Arthritis is truly a life-span issue affecting 294,000 children (Sacks, Helmick, Luo, Ilowite, & Bowyer, 2007) and impacting young and middle-aged adults in terms of daily living and work activities (CDC, 2006). The risk of OA does increase with age, with some studies estimating that more than half of all people age 65 and over would show x-ray evidence of OA in at least one joint (NIAMS, 2002). However, OA is not a normal part of aging and it is not inevitable. While there is no cure for arthritis, it is amenable to a variety of treatments including medication, surgery, self-management, and exercise. Early detection and timely treatment of arthritis are key factors to preventing or at least lessoning pain and disability later in life (Arthritis Foundation, 1999; CDC, 2007a). Other preventive factors such as engaging in physical activity–exercise, maintaining a healthy weight, avoiding injury, and avoiding occupational injury due to repetitive joint use are also important. Although the mechanisms by which these factors may lead to arthritis are unclear, these factors may signify important early influences and areas for arthritis prevention across the life span.

Arthritis does affect certain groups disproportionately; arthritis is more prevalent in women and those with low SES, and increases with age (CDC, 2006; Dunlop, Manheim, Song, & Chang, 2001). While the causes of arthritis and reasons for higher prevalence of arthritis in women are unclear, it is possible that factors such as hormones and overweight–obesity play a role. Arthritis is also among the top three chronic conditions in Blacks and Latinos and the leading cause of activity limitations in both groups (CDC, 1996). With the expected increase in arthritis over the next couple of decades and the impact on daily functioning and quality of life of older adults, it will be important to give high priority to arthritis research, prevention efforts, and intervention. This is especially true for older racial–ethnic minorities who experience greater impact of this illness and are surprisingly understudied in terms of psychosocial aspects of arthritis and understanding arthritis health disparities. This chapter will focus on arthritis health disparities primarily in Blacks and Latinos. Throughout the chapter, "Black and African American,"

"Hispanic and Latino," and "White and Caucasian" will be used interchangeably to refer to the three racial–ethnic groups.

RACIAL–ETHNIC DISPARITIES IN ARTHRITIS

There is evidence for racial–ethnic disparities in arthritis prevalence, symptoms, and utilization of treatments. It is likely that a complex combination of factors contributes to these disparities. For instance, African Americans are more likely to have low SES, which is then associated with lower occupational status, less access to health care, less likelihood of having health insurance, and poorer arthritis outcomes. It is possible that these factors compound over a lifetime to impact onset and severity of arthritis (although to my knowledge this complex set of events has not been examined in arthritis research). It is also likely that early experiences play a role in some circumstances. For instance, spending a lifetime working in manual labor–service jobs with repetitive stress and strain on the joints can contribute to the onset and severity of arthritis in later life (Felson et al., 2000; Holte, Tambs, & Bjerkedal, 2000; Rossignol et al., 2005). Similarly, being overweight or obese can also contribute to later onset and severity of arthritis (Bliddal & Christensen, 2006; Felson et al., 2000; Gelber et al., 1999). In both instances, members of racial–ethnic minority groups are likely to be more vulnerable than Whites; they are more likely to hold such occupations (Changing America, 1998) and to be overweight or obese (Wang & Beydoun, 2007). It is difficult to pinpoint the causes of arthritis or of arthritis disparities. Unfortunately, there is a lack of a comprehensive examination of these issues in diverse racial–ethnic groups. Moreover, few longitudinal studies have addressed these issues. Nonetheless, it is well known that disparities exist in several areas related to arthritis.

Arthritis Prevalence Rates. Prevalence rates for arthritis vary with some studies showing higher rates in racial–ethnic minority groups and others showing similar or even lower rates compared to Whites. Recent CDC (2005, 2006) reports using NHIS data show that Blacks and Whites (age 18+) have similar prevalence rates and Latinos have lower rates than Whites. On the other hand, higher arthritis prevalence rates were found in older Blacks and Latinos compared to Whites (age 70+) using AHEAD data (Dunlop et al., 2001). Yet still, other findings from the Health and Retirement Study (HRS) suggest higher rates for Blacks, but not Latinos, compared to Whites (age 51–61; Kington & Smith, 1997). It is not clear why such discrepant findings occur in regards to racial–ethnic differences in arthritis prevalence rates, however Dunlop and colleagues (2001) speculate that comparisons among large national samples varying in age, using different questions for self-assessed arthritis, and other

methodological differences might play a role. It is also possible that Blacks and Latinos are less likely to be diagnosed by a doctor with chronic conditions such as arthritis, which could result in underestimating rates in these two groups (Angel & Angel, 2006). Regardless of mixed findings for racial–ethnic differences in prevalence rates, there is evidence for disparities in the *impact* of arthritis (e.g., pain, disability) and utilization of arthritis health care (e.g., joint replacement surgery) with poorer outcomes for Blacks and Latinos compared to Whites.

Arthritis-Related Symptoms. Various studies demonstrate evidence for a racial–ethnic disparity in the impact of arthritis in terms of pain, functional impairment, and radiographic (x-ray) evidence of arthritis. Blacks and Latinos with arthritis report more severe pain (CDC, 2005), and activity and/or work limitations (Abraído-Lanza et al., 2006; CDC, 2005; Kington & Smith, 1997) than do Whites with arthritis. Other studies show evidence of marked disparities in radiographic evidence of OA in Blacks compared to Whites (Burns, Graney, Lummus, Nichols, & Martindale-Adams, 2007). It is, however, important to note that racial–ethnic differences in symptoms are not found in every study. For example, community- and clinic-based studies have demonstrated similar levels of pain and functional impairment in male veterans (Ang, Ibrahim, Burant, & Kwoh, 2003) and similar levels of pain in middle-aged and older women (McIlvane, 2007) comparing Blacks and Whites with arthritis. Again, discrepant findings might be due to different samples and different methods of assessment across studies. Race differences in perceptions of and reporting of disability may also be a factor. Notably, Burns and colleagues (2007) found that Black and White older adults self-reported similar levels of pain and functional ability, however older Blacks showed more severe radiographic evidence of OA and performed more poorly on a timed walking task.

Some studies also suggest racial–ethnic differences in psychological well-being and quality of life among individuals with arthritis. One study showed that African Americans with arthritis had lower quality of life compared to Whites with arthritis (Ibrahim et al., 2002). There is also evidence for higher levels of depressive symptoms and more psychological distress among Hispanics compared to non-Hispanics with arthritis (RA) and this is especially true for Hispanics who are not fully acculturated to mainstream Anglo culture (Escalante et al., 2000).

Utilization of Treatment. Despite evidence for higher (Dunlop et al., 2001) or equal (CDC, 2005) arthritis prevalence rates in racial–ethnic minority groups, Blacks and Latinos are less likely than Whites to have joint replacement surgery (CDC, 2009b; Dunlop et al., 2003; Escalante et al., 2000; Kane, Wilt,

Suarez-Almazor, & Fu, 2007) and this disparity is not completely explained by SES or access to care factors. Even in the case of presumable equal access (i.e., medical insurance, Medicare coverage), Blacks are hesitant to have surgery, may not trust surgery as a valid option (Figaro, Williams-Russo, & Allegrante, 2004), and have lower rates of total knee replacement (TKR) compared to Whites (Blake et al., 2002).

Another concern is that members of minority groups who need to have surgery may arrive with poorer functioning to begin with and more advanced disease. One study showed that Blacks and Latinos had poorer preoperative statuses in terms of more pain, poorer functioning, and worse quality of life compared to Whites who were about to have hip or knee joint arthroplasty surgery (Lavernia, Lee, Sierra, & Gómez-Marín, 2004). The same poor statuses were found in those who lacked Medicare or private health insurance. In addition to the disparity seen in joint replacement surgery, there is also evidence that racial–ethnic minorities experience delayed onset of receiving arthritis medication (Suarez-Almazor et al., 2007).

POTENTIAL CONTRIBUTORS TO ARTHRITIS HEALTH DISPARITIES

Racial–ethnic disparities in arthritis are complex and not well understood. While research is just beginning to document these disparities, the reasons for the disparities are less clear. In this section, a variety of potential contributors (e.g., SES, barriers to access to care) will be considered that are typically examined in terms of health disparities in general and in other illnesses. It should be noted, however, that research on the causes of arthritis disparities is in its infancy stage.

Socioeconomic Status. In terms of health disparities, race and SES are inextricably linked. Member of racial–ethnic minority groups and individuals with low SES are both more likely to experience poor health and disability. Members of racial–ethnic minority groups are also more likely to have low SES, which makes it difficult to determine whether differences in outcomes are due to race–ethnicity or SES. In some cases, racial–ethnic differences disappear when controlling for SES. Kington and Smith (1997) found that African Americans and Latinos with chronic conditions, including arthritis, had worse physical functioning compared to Whites but that the disparity disappeared when controlling for SES. This would suggest that SES, not race–ethnicity explains the disparity. Yet, studies often show that disparities persist after accounting for SES (Williams, Yu, Jackson, & Anderson, 1997). For

instance, Dunlop et al. (2001) found that the disparity in arthritis prevalence rates between Blacks and Whites was attenuated controlling for SES. However, this was not the case for Latinos (i.e., the disparity was not reduced) and prevalence rates still remained higher in both minority groups compared to Whites suggesting that in this case SES does not completely explain the racial–ethnic disparity.

Other investigators have begun to examine interactive effects of race and SES. Some have argued that the effects of SES should not be controlled away in studies that examine race differences in health (Farmer & Ferraro, 2005; Jordan et al., 2002; McIlvane, 2007). Since SES may impact health differently for African Americans, Latinos, and Whites, it is important to examine not just direct effects of race and SES but also interactive effects. Few studies have attempted to disentangle the effects of race and SES on arthritis symptoms and well-being (McIlvane, 2007). One recent study on arthritis examined interactive effects of race and SES on arthritis-related symptoms, coping, and well-being among African American and White women. Several race × SES interactions emerged showing that African American women with low SES had the highest level of depressive symptoms and were more likely to use maladaptive coping strategies (McIlvane, 2007). African American women with low SES also had more pain and functional impairment although this finding only approached significance. Overall, this study found that low SES (education, occupational status), not race, was related to more pain and functional impairment. This study provides preliminary evidence that it is important to consider both the unique and interactive effects of race–ethnicity and SES in studies on arthritis health disparities. Knowing that SES does not always explain health disparities, it is important to consider other potential contributors. The next sections focus on additional factors that may help explain health disparities in arthritis.

Barriers to Health Care Access. Structural factors may also contribute to disparities in arthritis outcomes and utilization of treatment. In terms of arthritis-related health care, little is known about the barriers facing members of racial–ethnic minority groups. Literature on health care in general shows evidence for barriers to health care access ranging from financial strain, poorer quality health facilities in predominately minority neighborhoods, perceived or actual discrimination, and lack of health insurance (Rooks & Whitfield, 2004; Williams & Braboy Jackson, 2005; Williams et al., 1997). For Latinos, language barriers can also interfere with adequate access to health care (Escalante, 2007; Fiscella, Franks, Doescher, & Saver, 2002). These issues have received little attention specifically in the context of arthritis.

Some arthritis studies find that Blacks are less likely to see a specialist (e.g., rheumatologist) for arthritis care (Katz et al., 1998). However, Mikuls et al. (2003) found that other factors (e.g., gender, urban–rural residence) explained an initially significant race difference in access to specialists (i.e., rheumatologist) but that Whites were still more likely than Blacks to see a primary care physician for arthritis care even after controlling for these other factors. A study of older Blacks and Whites with arthritis hip and knee pain showed that while both groups were just as likely to see an internist or primary care doctor, Whites were more likely to see an orthopedic surgeon for pain and Blacks were more likely to go to clinics and emergency rooms for care (Blake et al., 2002). Recent findings show that while Hispanics are more likely to have chronic joint symptoms, they are also more likely to have never seen a health care provider about their symptoms (CDC, 2003).

One study found few differences in perceptions of access to care between Black and White male VA patients with OA (Lopez, Burant, Siminoff, Kwoh, & Ibrahim, 2005). No significant race differences were found in referrals to specialists such as rheumatologists and orthopedists, or in satisfaction with and confidence in the physician. However, African Americans were more likely to have VA insurance only (i.e., no additional non-VA coverage such as Medigap or private insurance) and there was a trend showing that African Americans were less likely to get referrals to orthopedists in particular.

Chronic Stress. Much evidence suggests that stress plays a role in health disparities in health and well-being (Jackson, 2002; Lantz, House, Mero, & Williams, 2005). As mentioned above, SES alone does not completely explain health disparities and it is possible that chronic stress is another contributor. Yet, few studies on arthritis consider chronic stressors that occur in people's lives in addition to arthritis. Arthritis does not occur in a vacuum and it is likely that older adults, particularly minorities, are living with both arthritis and other chronic stressors. Blacks and Latinos are more likely to experience unique stressors, such as discrimination and financial stress than Whites (Angel, Frisco, Angel, & Chiriboga, 2003; Jackson, 2002; McIlvane, Baker, & Mingo, 2008a; Pérez, Fortuna, & Alegría, 2008). A recent study examined both arthritis-related stress and chronic stress in African American and White women (McIlvane et al., 2008a). African American women with arthritis reported more functional impairment, lower perceived arthritis stress, but more life stressors, financial strain, and everyday discrimination. Moreover, chronic stressors (e.g., discrimination, financial strain) accounted for more variance in depressive symptoms for Blacks compared to Whites, whereas arthritis-related stressors accounted for similar variance in the two groups suggesting that Blacks may be coping with multiple stressors that need to be considered.

Few arthritis studies consider additional chronic stressors, particularly as contributors to health disparities. It will be important for future studies to consider the context in which people from diverse racial–ethnic groups live with chronic illness, including arthritis.

Mistrust of the Medical System. Racial–ethnic differences in perceptions of the health care system (e.g., mistrust) and communications with doctors receive scant attention in arthritis research. Research in other health-related areas demonstrate that African Americans are more likely to report mistrust of the medical system than Whites (Brandon, Isaac, & LaVeist, 2005; LaVeist, Nickerson, & Bowie, 2000). Figaro and colleagues (2004) did find in a qualitative study that Blacks reported fear of doctors and mistrust of the medical community as barriers to having total knee replacement. This author is not aware of any other studies on arthritis that examines mistrust of the medical community or perceptions of discrimination specifically in the health care setting as determinants of arthritis health care and health disparities. To my knowledge, only one study on Blacks and Whites with arthritis considers perceptions of everyday discrimination (not specifically related to health care) in terms of arthritis-related outcomes (McIlvane et al., 2008a); there are no such studies on Latinos (Abraído-Lanza et al., 2006).

It is unclear whether mistrust of the medical system and perceptions of bias play a role in arthritis care and the extent to which people with arthritis seek care and trust the advice of their doctors when it comes to arthritis. Perceptions regarding whether doctors take concerns seriously may serve as barriers to seeking treatment and contribute to health disparities. For the older female minority, these issues may be particularly important in terms of potentially experiencing the combined effects of racism, ageism, and sexism. It is important to understand the experiences and perceptions of older Blacks and Latinos in interacting with health care providers and navigating the health care system while seeking out care for their arthritis. These factors need to be considered in future arthritis research as variables that may affect both adaptation and functioning with arthritis as well as utilization and access to arthritis-related health care.

Cultural Differences in Illness Perceptions. Researchers are beginning to consider racial–ethnic differences in perceptions of arthritis and arthritis health care as contributors to health disparities. While SES and barriers to access to care may play a role in arthritis health disparities, it is also possible that patient perceptions play a role. Cultural beliefs held by Blacks and Latinos may contribute to arthritis health disparities but little is known about older Blacks' and Latinos' perceptions about arthritis and arthritis health care or how such perceptions relate to health disparities. Cultural and psychosocial environments

of patients affect perceptions of disease as well as patient expectations for health care (Ibrahim, Zhang, Mercer, Baughman, & Kwoh, 2004). Moreover, it is essential to consider specific diseases when trying to understand reasons for health disparities because interactions between patients and the health care system depend on the specific disease (Ibrahim, Siminoff, Burant, & Kwoh, 2001). This section will discuss arthritis perceptions of Blacks and Latinos in regards to traditional care, complementary and alternative medicine (CAM), joint replacement surgery, and views on illness and aging.

Given the chronic nature of arthritis and the need to manage pain when traditional treatments (e.g., prescription medications) are not always effective, people with arthritis sometimes use CAMs or folk remedies such as liniment, herbs, dietary supplements, and sometimes even WD-40 (i.e., a product that lubricates metal joints and rusty metal parts on machines, locks, etc.; Katz & Lee, 2007). While many of these remedies are harmless, there is the potential for interactions with prescription medications and this becomes a problem when patients do not tell their doctors about their CAM use. There is also the possibility that people with arthritis use CAMs as a substitute for seeking traditional health care (e.g., less expensive, information comes from more trusted sources such as family–friends). The role played by use of CAMs and negative perceptions of traditional health care in contributing to racial–ethnic health disparities in arthritis is unclear.

Recent studies find high CAM use in diverse samples of older adults with OA with over 80% of African Americans, Hispanics, Asians, and Caucasians reporting CAM use in the past month (Katz & Lee, 2007). About 90% of Hispanic and non-Hispanic Whites with arthritis (OA, RA, fibromyalgia) reported that they had ever used CAM for their arthritis while almost 70% said that they use CAM now (Herman, Allen, Hunt, Prasad, & Brady, 2004). While few studies examine racial–ethnic differences in CAM use in older adults with arthritis, Blacks are shown to be more likely than Whites (Ibrahim et al., 2001) and than Hispanics, Asians, or Caucasians to use complimentary treatments (Katz & Lee, 2007). Another study found no differences in CAM use comparing Hispanics and non-Hispanic Whites and the most commonly used CAMs overall were glucosamine and chondroitin, mind-body therapies, and herbal topical ointments (Herman et al., 2004). Moreover, over one fifth of Hispanic and non-Hispanic Whites reported that they never talked to their doctor about using CAM.

Other studies examine perceptions of effectiveness of CAMs. An early study of self-care practices used by Blacks (primarily with OA) and Hispanics (primarily with fibrositis, which is now called fibromyalgia) from urban, low-income communities found that both groups used nonconventional

treatments and remedies (Bill-Harvey, Rippey, Abeles, & Pfeiffer, 1989). Hispanics rated prayer, heat, and massage as most helpful while Blacks rated prayer, use of appliances (e.g., canes), and heat as most helpful. Interestingly, neither group rated prescribed arthritis medications as being very helpful. One study found that older Blacks and Whites with OA had different perceptions regarding arthritis treatments (Ibrahim et al., 2001). Blacks were more likely to perceive both traditional (e.g., Tylenol, physical therapy) and complementary treatments (e.g., prayer, herbal remedies) for arthritis as effective while at the same time being less likely to perceive joint replacement surgery as effective. It is unclear the extent to which these perceptions of traditional and nontraditional treatments influence arthritis health care seeking behaviors or health disparities.

Recent studies also show that Blacks have low expectations for joint replacement therapy (Figaro, Williams-Russo, & Allegrante, 2005) and report negative perceptions of total knee replacement viewing it as ineffective for themselves or others (Figaro et al., 2004). In a recent study, some expressed fear of being cut, death, doctors, bad outcomes from surgery, and the unknown, as well as distrust of the medical system (Figaro et al., 2004). All participants had medical coverage but many still felt limited access to care. Similarly, there is evidence of lower use of joint replacement among Latinos although the reasons for this disparity are unclear (Escalante et al., 2000). Despite fears about joint replacement surgery, it has been shown to be an efficacious procedure (e.g., Fortin et al., 1999).

The reasons for lower use of joint replacement in racial–ethnic minority groups are complex. For example, the source of information about arthritis and surgery may be an important determinant of perceptions and willingness to undergo surgery. In a sample of older Blacks with OA (Figaro et al., 2005), most (70%) reported learning about OA from their doctor but less than half learned about TKR from their doctor (49%). On the other hand, the majority reported learning about TKR from a neighbor or friend (65%) or family member (29%). Older Whites with hip–knee pain were more likely to know someone who had surgery for hip–knee pain than older Blacks; Blacks were also less likely to report that surgery helped someone they knew although this was not statistically significant (Blake et al., 2002). It is likely that fear, negative perceptions of the medical system, and learning about bad experiences from family and friends may play a role.

While we know something about Blacks and Latinos preferences for traditional and nontraditional treatments, little is known about perceptions of arthritis. It is unclear how older adults perceive arthritis; this is particularly true for older minorities. A study of older Blacks' preferences for arthritis care

demonstrated that many participants believed OA was caused by exposure to cold, inevitable, and a sign of aging (Figaro et al., 2004). They also believed that OA was irremediable. Beliefs that arthritis is a normal part of aging and that nothing can be done about it are common misperceptions held by older adults and health care professionals and may serve as a barrier to seeking treatment (Goodwin, Black, & Satish, 1999).

One study examined perceptions of common medical conditions (arthritis, heart disease, sleep problems) and found racial–ethnic differences in beliefs about these conditions (Goodwin et al., 1999). For example, older Blacks were less likely than older non-Hispanic Whites to view arthritis as a normal part of aging and in open-ended questions both Blacks and Hispanics were more likely than non-Hispanic Whites to list work–environmental exposure as a cause of arthritis. One troubling finding was that regardless of race–ethnicity, participants who believed arthritis (and other conditions) was untreatable were less likely to report having a regular physician and participants who viewed arthritis as a normal part of aging were less likely to have utilized preventive services. This study offers very interesting insights into the beliefs of diverse older adults with arthritis, however, much more work is needed in this area.

WHO PERSEVERES? PROTECTIVE FACTORS ACROSS THE LIFE SPAN

Thus far, this chapter has focused on disparities and potential causes. It is important to also consider cultural values and strengths, which may serve as protective factors. This section will consider the social and personal resources, including coping, religion, and family that may enable people to persevere in the face of adversity.

Coping With Arthritis: Importance of Cultural Values. Literature on coping with arthritis generally suggests that certain strategies such as positive reappraisal and active coping are related to better well-being while other strategies such as self-blame, wishful thinking, passive coping (see Zautra & Manne, 1992 for review) and catastrophization (Keefe et al., 2004) are related to poorer well-being. It is unclear whether these findings hold true for minorities as surprisingly few studies have examined racial–ethnic differences in coping with arthritis. Two arthritis studies suggest that African Americans use more diverting attention (Jones et al., 2008) and praying and hoping (Jones et al., 2008; Jordan, Lumley, & Leisen, 1998) than Whites. Another study on race differences in coping with arthritis found that African American women used more religious coping, wishful thinking, seeking social support, and emotional expression than Whites (McIlvane, 2007).

People with arthritis often report using prayer and religion to manage the pain from their arthritis (Keefe et al., 2001). Religion may serve as an important source of strength and comfort for many individuals dealing with a painful and debilitating chronic illness. Studies examining use of religion in arthritis often find that members of racial–ethnic minority groups are more likely than Whites to rely on faith and prayer. Research shows that Blacks are more likely to use religion–prayer (Ibrahim et al., 2001; Silverman, Musa, Kirsch, & Siminoff, 1999) and that Latinas demonstrate a strong reliance on religion and faith in managing arthritis pain (Abraído-Lanza, Guier, & Revenson, 1996). A focus group study demonstrated that older African Americans with chronic knee and hip pain reported that faith and prayer are important in the care process, are more concerned with quality of care than the race of their physician, and while they do not necessarily expect their physician to share their faith but do expect their physician to respect their religious and cultural beliefs in the care process (Ibrahim et al., 2004). These are important considerations for health care providers working with diverse older adults. It will be necessary to respect beliefs about religion and take these beliefs seriously. Otherwise, they may risk alienating minority patients.

In one of the few studies on Latinos coping with arthritis, Abraído-Lanza et al. (1996) found in a qualitative study that low-income Latina women with arthritis reported engaging in activities (active coping), and religion and prayer as the most common coping strategies. They were also more likely to rely on family members, daughters and spouses in particular, than friends for support suggesting the importance of culture-based norms such as familism in coping with arthritis. They found that religion and faith in God provided both comfort and hope and helped Latina women deal with physical limitations and pain. Latinas also tended to report seeking social support–contact as a means of distraction from pain rather than to gain help with tasks or sympathy.

Little attention has been given to the *effectiveness* of strategies in diverse racial–ethnic groups. Abraído-Lanza, Vásquez, and Echeverría (2004) found that while Latinos with arthritis used high levels of religious coping, it was not significantly related to pain, depression, acceptance of illness, self-efficacy, or psychological well-being. Future research needs to examine the effectiveness of specific coping strategies in Blacks and Latinos with arthritis.

FUTURE DIRECTIONS

This final section will consider future directions in terms of public health efforts, greater inclusion of racial–ethnic minorities in arthritis interventions, and future research directions.

Increasing Public Health Efforts. Arthritis is just beginning to receive much needed attention as a public health issue. Until recently, arthritis did not receive adequate attention probably because it is generally not a life-threatening illness and it is incorrectly perceived as a normal part of aging and untreatable. However, the cost of OA, in terms of quality of life, productivity, functional limitations, and health care dollars is finally being recognized. Several recent initiatives give promise for bringing arthritis to the forefront of public health and prevention efforts. Healthy People 2010 was the first of three 10-year national health plans to include objectives for arthritis (Hootman et al., 2002). National and global initiatives such as the National Arthritis Action Plan (Arthritis Foundation, 1999; CDC, 2009a) and the Bone and Joint Decade (www.boneandjointdecade.org) are making arthritis a public health priority. For instance, the National Arthritis Action Plan involves joint efforts by the Arthritis Foundation, CDC, and a host of other organizations to focus on arthritis surveillance, prevention, and policy issues in an effort to reduce the impact of arthritis in communities around the country. These efforts should continue to reach out to minority and low-income communities, which will further address another main goal of Healthy People 2010, which is to eliminate health disparities.

Greater Inclusion of Members of Racial–Ethnic Minority Groups in Arthritis Intervention Research. The good news is that arthritis is treatable and that there are a variety of arthritis interventions available (e.g., self-management, coping, exercise, education) many of which are offered by the Arthritis Foundation (see Brady, Kruger, Helmick, Callahan, & Boutaugh, 2003 for review). A recent meta-analysis demonstrated that arthritis interventions are generally effective in predominately White samples in terms of reducing pain and disability (Dixon, Keefe, Scipio, Perri, & Abernathy, 2007). Unfortunately, little is known about the effectiveness of arthritis interventions in racial–ethnic minority groups. Inclusion of members of racial–ethnic minority groups in arthritis intervention research has received scant attention.

A recent 10-year review of arthritis interventions (25 randomized controlled trials [RCTs]) found little attention given to racial–ethnic minority groups in terms of inclusion in intervention, examination of race differences in treatment, or special efforts in recruitment, retention, and designing interventions to be culturally appropriate (McIlvane, Baker, Mingo, & Haley, 2008b). In fact, many studies did not report the racial composition of their sample (11 out of 25); only six studies provided detailed information on the racial–ethnic composition of the sample. Most studies included only a small number of minorities. Further, only two reported on the effectiveness of the intervention separately by race and found equal effectiveness, six examined

attrition by race, and none reported systematic efforts to make sure their intervention was culturally sensitive and appropriate. Based on their review, McIlvane and colleagues (2008b) recommend that it will be important to focus more on successfully recruiting and retaining racial–ethnic minorities in arthritis interventions and in designing such interventions to be culturally sensitive and appropriate.

There have been some efforts to include racial–ethnic minorities in arthritis interventions. There is evidence of effectiveness of the Spanish Arthritis Empowerment Program that adapts the well-known Arthritis Self-Help Course for use in Latinos. Wong, Harker, Lau, Shatzel, and Port (2004) found that the program resulted in improved arthritis-related symptoms and self-efficacy, although this was not a RCT. Others have demonstrated effectiveness of arthritis self-management education programs in African Americans (Goeppinger, Armstrong, Schwartz, Ensley, & Brady, 2007). Similarly, the investigators of the ADAPT (Messier et al., 2004) and Fit and Strong (Hughes et al., 2006) arthritis interventions have shown success in recruiting and retaining African Americans. Future studies need to further address the effectiveness of interventions and perceptions of cultural sensitivity for diverse older adults with arthritis.

Suggestions for Future Research. While it is clear that arthritis health disparities exist, researchers are just beginning to document disparities and to investigate reasons for such disparities. Thus, there is a gap in the literature in terms of examining racial–ethnic differences in access to health care, chronic stress, perceptions of arthritis and arthritis health care, and how these factors contribute to health disparities. Research on arthritis health disparities tends to focus on discrete issues (e.g., race differences in joint replacement surgery or in self-care). It will be important as a next step to take a broader, more comprehensive view of the contexts in which people live with arthritis and consider multiple factors that may contribute to health disparities in one study. Likewise, longitudinal studies examining the impact of multiple factors over time will also be necessary. It will be important for researchers to make a concerted effort to recruit older racial–ethnic minorities to more fully examine these issues in older adults with arthritis.

In terms of future research, it seems time to take a step back and conduct more qualitative research to better understand the experiences and perceptions of Blacks and Latinos regarding their arthritis care, barriers to treatment, and experiences with the health care system. Some of the most insightful information discussed in this chapter came from qualitative studies on Blacks and Latinos perceptions of arthritis and experiences in the health care setting (e.g., Abraído-Lanza et al., 1996; Figaro et al., 2004;

Ibrahim et al., 2004). Many of the standard instruments (e.g., coping, social support, illness perceptions) may not capture the experiences of Blacks and Latinos with arthritis (Abraído-Lanza et al., 1996). Conducting mixed or multimethod studies including both quantitative and qualitative components will be instrumental in understanding the experiences of minorities with arthritis and in developing quantitative measures that more fully capture the experiences of older minorities.

CONCLUDING COMMENTS

Despite consistent evidence for racial–ethnic differences in arthritis symptoms and utilization of effective treatments, health disparities have received little attention thus far in arthritis research. Future research needs to focus on the unique contexts and cultural traditions and values of racial–ethnic minorities in order to fully understand their experiences living with arthritis and other stressors (e.g., discrimination, financial strain) as well as their experiences interacting with the health care system. Perceptions about arthritis, the health care system, communications with doctors, and aging need to be taken into consideration. Likewise, protective factors and cultural values in diverse racial–ethnic groups that may buffer the effects of stress and allow them to cope with a painful chronic illness should be included in this area of research. Given the costs associated with arthritis in terms of quality of life, productivity, financial costs, and functional limitations, arthritis health disparities should remain a high priority in research, intervention, and public health efforts. The ultimate goal will be to reduce or eliminate arthritis health disparities and ensure equal access to quality health care for all.

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SECTION V

CONTEXTUAL INFLUENCES ON HEALTH DISPARITIES IN LATE LIFE

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CHAPTER 10

Between-Person Disparities in the Progression of Late-Life Well-Being

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"Most of the people are mildly happy most of the time" (Biswas-Diener, 2009). Even after major negative life events, most people often quickly adapt and return to their characteristic levels (Brickman & Campbell, 1971), and such set points are typically positive rather than neutral or negative (see Diener, Lucas, & Scollon, 2006). In this chapter, we review recent and ongoing endeavors that highlight the utility of focusing on a phase of life during which this positive picture of well-being does not necessarily prevail. Drawing from notions of terminal decline, we argue that the changes in well-being that occur late in life provide a venue for the examination of between-person disparities and the factors that contribute to them. In a first step, we review empirical evidence to suggest that such steep end-of-life declines in well-being and psychological health may indeed be a normative experience, but these declines are more a function of closeness to death than age itself. At the same time, not all individuals experience their last years alike, and while end-of-life decline may be normative, it should not be considered inevitable. There is tremendous variability in late-life patterns of change over time. Many people's well-being drops sharply into death, whereas others maintain their well-being into their last years. In a second step, we characterize how such between-person disparities in late-life well-being progress, and we

highlight some of the key factors that contribute to such inequalities. These factors encompass key predictors of both mortality and well-being, including age (at death), gender, education, and disability. In a third step, we argue that such factors may not only reside at the individual level, but may also be found at the community or society level. In doing so, we review some of the possible mechanisms linking such macrocontextual factors to individual outcomes and inequalities in well-being change late in life. Throughout, we discuss how this line of research informs and contributes to further precision and refinement of widespread theories of well-being as well as conceptual notions implicating mortality as a major force underlying developmental change in the last years of life.

THE PROGRESSION OF LATE-LIFE WELL-BEING: NORMATIVE TRENDS AND BETWEEN-PERSON DISPARITIES

We first summarize several theories of well-being and the myriad of empirical reports to demonstrate that well-being is relatively stable across adulthood and old age. We then review an emerging body of findings suggesting that well-being shows terminal decline at the end of life and discuss implications that arise from these findings for theories of well-being and aging. We finally highlight that the progression of late-life well-being encompasses both alarmingly steep normative proximate-to-death deteriorations and large between-person inequalities therein.

Well-Being Is Stable Across Adulthood and Old Age

Many developmental theories of well-being and self-regulation suggest that well-being remains relatively stable across adulthood and old age. For example, hedonic treadmill models of well-being highlight the role of adaptation processes through which people quickly adapt to changes in life circumstances (Brickman & Campbell, 1971). As a consequence, positive and negative events only have short-term effects, with people returning back to their characteristic initial levels quickly thereafter. Thus, normatively, well-being remains stable over the long-term. Socioemotional selectivity theory (Carstensen, 2006) also provides a framework for normative stability. Here, motivational shifts toward emotional and social goals occurring in conjunction with changes in future time perspective (e.g., how much longer one expects to live) lead to prioritizing the maintenance of well-being. A common theme underlying these and other action-theoretical accounts of development (e.g., Baltes & Baltes, 1990; Brandstädter, 1999; Heckhausen & Schulz, 1995)

is that an objective worsening of life conditions in old age (e.g., increased health constraints or social losses) does, on average, not affect well-being. Well-being is maintained and remains stable.

These conceptual arguments map onto the results of numerous cross-sectional and longitudinal studies finding that various facets of well-being remain relatively stable across the adult life (Argyle, 1999; Carstensen, Pasupathi, Mayr, & Nesselroade, 2000; Costa et al., 1987; Diener et al., 2006; Diener, Suh, Lucas, & Smith, 1999; Haynie, Berg, Johannson, Gatz, & Zarit, 2001; Kunzmann, Little, & Smith, 2000; Mroczek & Kolarz, 1998). After reviewing cross-sectional findings from large-scale probabilistic samples across several nations, Diener and Suh (1998) concluded that "life satisfaction appears to be relatively stable across age cohorts in most societies" (p. 310). Similarly, longitudinal studies evidenced that the emotional well-being facet of positive affect shows stability until age 65 and declines slightly thereafter, and the emotional well-being facet of negative affect remains virtually unchanged into old age (Charles, Reynolds, & Gatz, 2001; Kunzmann, 2008). In sum, although interpreted from different perspectives and implicating different underlying mechanisms, there is general consensus that age-related patterns of well-being over time are characterized by stability. As noted by Biswas-Diener (2009), "Most of the people are mildly happy most of the time."

A Stress-Test Paradigm for Examining Inequalities: Well-Being Declines With Impending Death

Methodologically, pervasive and normative stability does not provide a particularly robust venue for the examination of inequalities. Rather, to achieve a better understanding of the mechanisms leading to differences, we need variance. We need a venue where inequalities are expressed and can be readily observed. For example, in the diagnosis of heart disease, individuals are typically subjected to an exercise stress test wherein their cardiovascular reactivity and regulation is observed as their bodies are pushed toward their physiological limits (e.g., walking or running on a treadmill). Such paradigms have also been used in the examination of differences in cognitive plasticity. For example, in their testing-the-limits paradigm, Kliegl, Smith, and Baltes (1990) pushed individuals to the limits of their mental (learning) capacity in order to better measure and understand the mechanisms contributing to differences or inequalities in cognitive function. The general idea of these experimental paradigms is to produce a situation where interindividual differences stand out in relief and can be more easily observed. Differences in cardiovascular function are not so apparent when individuals are resting or going about their daily lives. However, under stress conditions, differences in functionality become

readily apparent, can be diagnosed and subsequently treated. Following this logic, we propose that stress-test paradigms may also be useful in the study of inequalities in well-being. In contrast to the stability and general happiness usually observed, a natural experiment wherein individuals' adaptive capacities are being pushed to their limits should provide for new opportunities to observe previously often overlooked differences in well-being, diagnose the inequalities, and identify possible mechanisms–treatments.

Studies of late-life well-being and terminal decline suggest that impending death may provide a natural testing-the-limits paradigm for studying inequalities in well-being. Conceptually, developmental changes during adulthood and old age result from primary or normal forms of aging, secondary or pathological aspects of aging, and tertiary or mortality-related processes of aging (Birren & Cunningham, 1985; Busse, 1969). Acknowledging that developmental change at the end of life reflects a combination of these three mechanisms, notions of terminal decline (Kleemeier, 1962) suggests that, as people approach death, mortality-related processes may rise to the forefront and become the primary force underlying late-life changes. The accumulation of mortality-related burdens and systemic dysfunction (e.g., in physical and/or cognitive health) should stress the system and test the limits of individuals' adaptive and regulatory ability and make it increasingly difficult to maintain a sense of well-being. In essence, approaching death serves as an absorbing state that drags individual functioning, including well-being, down. As this occurs, inequalities in (trajectories of) well-being and the underlying mechanisms should become more pronounced. Although providing a pessimistic perspective, mortality-related declines in well-being may offer a unique opportunity to observe and understand the mechanisms that contribute to inequalities in well-being.

Evidence is accumulating that the prevailing happiness and stability picture of well-being does not hold during the last years of a person's life (Gerstorf et al., 2008a; Gerstorf, Ram, Röcke, Lindenberger, & Smith, 2008b; Mrozcek & Spiro, 2005). For example, data from now deceased, 70- to 100-year-old participants in the Berlin Aging Study (BASE) suggest that interindividual differences in within-person changes in well-being were better represented in relation to distance-to-death than in relation to chronological age (Gerstorf et al., 2008b). The variance accounted for (calculated as the pseudo-R^2, after Snijders & Bosker, 1999) increased from less than 8% in the age-related change model to almost 17% in the mortality-related change model. In line with other studies and as illustrated in the upper panel (A) of Figure 10.1, the rate of age-related decline in well-being was relatively minor (–0.33 T-score units per year). In contrast, mortality-related models over distance-to-death revealed

considerably steeper average rates of decline (–0.75 T-score units per year). As can be obtained from the lower panel (B) of Figure 10.1, the end of life indeed appears to provide for a testing-the-limits situation.

Similar results in analyses of decedents from national data sets in Germany, the UK, and the United States gives us some level of confidence in these pronounced late-life deteriorations in well-being with approaching death (Gerstorf et al., in press). For an overview, the middle columns of Table 10.1 contrast the linear rates of well-being change per year over chronological age and distance-to-death separately by study and nation.

Inherent in the conceptual notion of terminal decline is that two phases of late-life change can be distinguished: A preterminal, age-dominated phase of relative stability or minor decline, followed by transitioning into a terminal mortality-dominated phase of precipitous decline (Kleemeier, 1962; Riegel & Riegel, 1972; Siegler, 1975; for overviews, see Bäckman & MacDonald, 2006; Berg, 1996). Despite these conceptual notions having been around for decades, they lack specificity regarding when the onset of terminal decline can prototypically be expected to occur. For example, Birren and Cunningham (1985) proposed that a "cognitive and social slipping" may occur some "months to years" prior to death (Birren & Cunningham, 1985, p. 21). Following pioneering work in the cognitive aging literature (Sliwinski et al., 2006; Wilson, Beck, Bienias, & Bennett, 2007; Wilson, Beckett, Bienias, Evans, & Bennett, 2003), we have applied recent developments in multiphase growth modeling to estimate empirically the prototypical location of this transition. Results from various data sets converged on a time window between 3 and 5 years prior to death after which normative rates of well-being decline steepened by a factor of three or more (for overview, see the right-hand column of Table 10.1). Again, effect sizes for this decline are gloomy. The prototypical individual's life satisfaction (e.g., among decedents in the national German data) declines nearly a full standard deviation over the last 4 years of life.

Conceptual Implications

Several implications arise from these findings for theories of well-being and aging in general. First, contrasting what would be expected by theories of hedonic adaptation (e.g., Brickman & Campbell, 1971), it appears that with approaching death it is increasingly difficult to maintain well-being. Our findings can be interpreted to indicate that mortality-related mechanisms or other progressive processes leading toward death (e.g., deteriorating health) overwhelm the regulatory or motivational mechanisms that usually keep well-being stable, and mortality-related processes become the prime drivers of late-life decline in well-being. One of the key questions in this regard

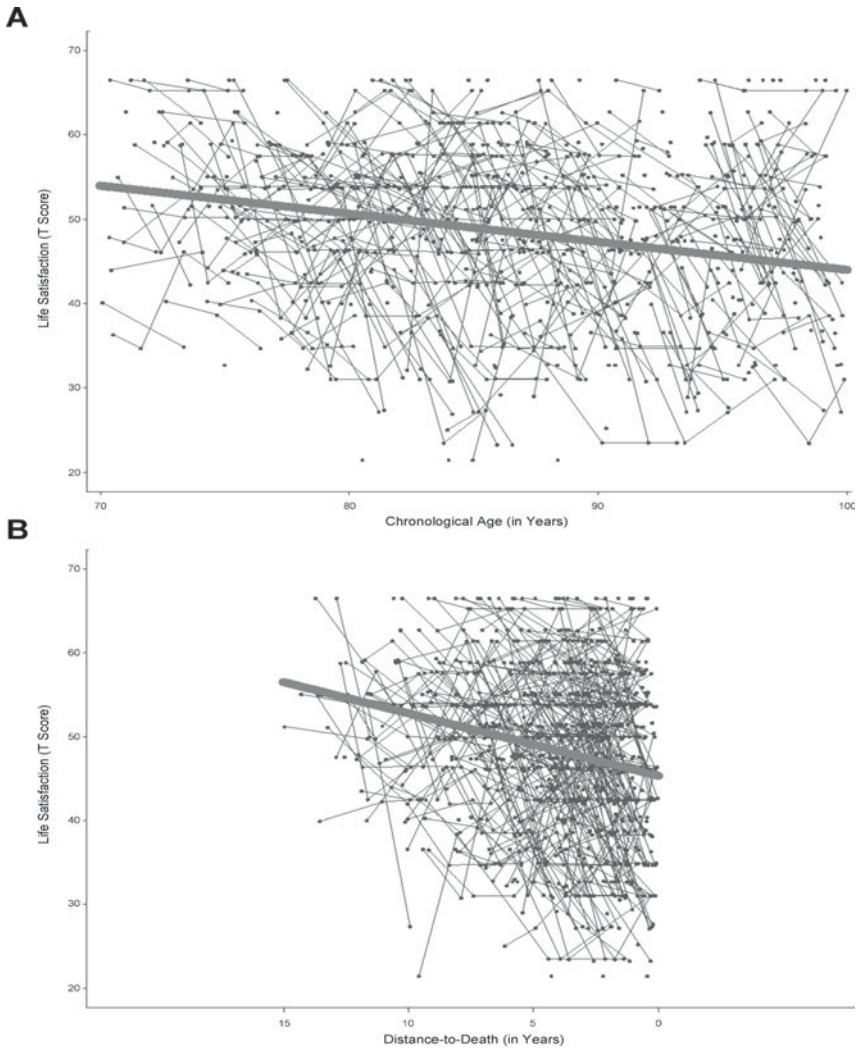

FIGURE 10.1 Contrasting late-life change in well-being over chronological age and distance-to-death.

Note. Data from now deceased, 70- to 100-year-old participants in the Berlin Aging Study (BASE) revealed that interindividual differences in within-person changes in well-being were better represented in relation to distance-to-death (upper panel) than in relation to chronological age (lower panel) and also indicated steeper average rates of decline. Figure reproduced from "Decline in life satisfaction in old age: Longitudinal evidence for links to distance-to-death," by D. Gerstorf, N. Ram, C. Röcke, U. Lindenberger, and J. Smith, 2008b, *Psychology and Aging*, 23, p. 161. Copyright 2008 by (supply copyright holder). Reprinted with permission.

TABLE 10.1 *Linear Rates of Change in Well-Being Indices for Deceased Participants over Age and Distance-to-Death in T-Score Units per Year and Estimated Transition Points to Terminal Decline, Separately by Study and Nation*

	Progression of Late-Life Well-Being		
	Single-Phase Models: Rate of Linear Change Per Year		Multiphase Models: Estimated Transition Point
	Chronological Age	Distance-to-Death	Distance-to-Death
Germany			
Socioeconomic Panel (n = 2,764, age at death: M = 72 years)			
Life satisfaction (cognitive-evaluative well-being)	–0.26[a] (±0.02)	–0.75[a] (±0.03)	4.27 (±0.01)
Berlin Aging Study (n = 414, age at death: M = 92 years)			
Life satisfaction (cognitive-evaluative well-being)	–0.33 (±0.05)	–0.75 (±0.10)	4.00 (±2.50)
Great Britain			
British Household Panel (n = 2,030, age at death: M = 75 years)			
General health questionnaire (affective well-being)	–0.08 (±0.02)	–0.57[a] (±0.05)	4.85 (±0.18)
United States			
Health and Retirement Study (n = 6,195, age at death: M = 80 years)			
Lack of depressive symptoms (affective well-being)	–0.10[a] (±0.01)	–0.56[a] (±0.03)	2.92 (±0.20)

Note. Parameter estimates (standard errors) from growth curve models are reported.
[a]Quadratic component of change reliably different from zero, which indicated some accelerated decline at advanced ages or near death. From "Life Satisfaction Shows Terminal Decline in Old Age," by D. Gerstorf, N. Ram, R. Estabrook, J. Schupp, G. G. Wagner, & U. Lindenberger, 2008, *Developmental Psychology*, *44*, p. 000. Copyright 2008 by (Supply copyright holder). Reprinted with permission. From "Late-Life Decline in Well-Being Across Adulthood in Germany, the UK, and the US," by D. Gerstorf, N. Ram, G. Mayraz, M. Hidajat, U. Lindenberger, G. G. Wagner, et al., (in press), *Psychology and Aging*. From "Decline in Life Satisfaction in Old Age," by D. Gerstorf, N. Ram, C. Röcke, U. Lindenberger, and J. Smith, 2008b, *Psychology and Aging*, *23*, p. 000. Copyright 2008 by (supply copyright holder). Reprinted with permission.

is whether or not well-being is inherently involved in these mortality processes. One line of reasoning has argued that, for example, well-being ratings primarily reflect summary perceptions of what is going on in other domains of functioning that are more directly linked to mortality (Maier & Smith, 1999). Other lines of reasoning have highlighted that well-being itself may (directly or indirectly) be part of the mortality dynamics, either

because of its motivational and behavioral consequences (Levy, Slade, Kunkel, & Kasl, 2002) or because of its physiological effects on cardiovascular and immune functioning (Danner, Snowdon, & Friesen, 2001; Pressman & Cohen, 2005).

Second, the ostensibly normative pattern also provides a rather disconcerting image of late-life psychological health that qualifies notions of successful aging (Rowe & Kahn, 1997; see Baltes, 2006). It is plausible that with limitations in perceived lifetime people indeed get better and better in optimizing their emotion regulation (Carstensen, 2006), but the pervasive nature of impending death may bring a sharp end to the possibilities afforded by such age-related increases in self-regulation.

Third, there exists a "soon to die" segment in the population of highly developed nations for whom a central indicator of quality of life is rapidly deteriorating or who simply report being fairly unsatisfied. For example, individuals in a nationally representative sample from Germany who died older than age 85 reported average levels of well-being that were below the neutral point (see Gerstorf et al., 2008a).

Fourth and finally, empirically localizing the normative onset of transitioning to the terminal phase of life informs future theoretical specifications of when end-of-life decrements can typically be expected to begin and how they may proceed (Birren & Cunningham, 1985). This illustrates that the gained precision in description (through applying methodological advances) requires and calls for new precision in theory. One central question to be addressed in future inquiries relates, of course, to why it is that the normative onset is located in a 3- to 5-year window prior to death rather than, let's say, 2 years prior to death or even in the last year of life.

Between-Person Disparities in Late-Life Well-Being

Although the normative picture of late-life change painted above is one of seemingly inevitable decline in well-being, there are substantial interindividual differences in when and how individuals experience their last years of life. Individuals vastly differ from one another in how much well-being they report as well as in the amount of decline well-being shows with impending death. These are exactly the differences and inequalities we seek to identify.

When. The above reports noted that the prototypical transition into the terminal-decline phase occurs between 3 and 5 years prior to death. However, this represents an average estimate at the population level based on the very strict assumption that the location of the transition point does not vary across individuals. In other words, all persons are assumed to

transition into terminal decline at exactly the same point in time. While likely unrealistic, such an assumption is often required by the limited nature of within-person change data available (e.g., five or six measurement occasions: Gerstorf et al., 2008b; Sliwinski et al., 2006; Wilson et al., 2003, 2007).

Using data from 400 individuals who provided 12 or more data points over 25 years, we were able to relax this assumption and allow for interindividual differences in the timing of the transition to terminal decline (Gerstorf et al., 2008a). As expected, there were considerable between-person differences in where the estimated transition point was located. While individuals, on average, transitioned to the terminal phase at roughly 4 years before death, some individuals entered earlier (e.g., 6 or 8 years prior to death), while others entered later (e.g., 2 years prior), and still others did not show any evidence of entering the terminal phase. Overall, individuals enter the terminal-decline phase at times ranging from 13 years before death to just before death to not at all. These latter individuals have likely not experienced the transition because they have died earlier than expected, presumably of some random cause (e.g., accident; sudden stroke) before they entered terminal decline.

How. Just as individuals differ in the timing of the transition into terminal decline, they also differ in the extent of those declines. All studies have noted significant variability in rates of mortality-related change. For example, among the 400 SOEP participants, the average rate of terminal decline was 3.51 T-score units per year, but some individuals declined substantially more (e.g., 6 T-score units per year), while others declined hardly at all (e.g., 0.5 T-score units per year). Modeling the transition with individual specificity was not possible in other samples, but similar differences in rates of change were noted across studies.

In sum, the extent of interindividual differences in well-being stand out dramatically when the focus is on examining changes in well-being at the end of life. Our modeling efforts and findings suggest that while some segments of society experience great decrements in psychological health as they approach death, other groups of individuals maintain key aspects of quality of life into the very last years. Noting these differences prompts a quest to identify the reasons why such inequalities in late-life well-being arise. Insights into why some persons experience fewer years of decline, or less steep rates of decline prior to death may point to particular pathways for intervention (Baltes & Baltes, 1990; Berkman et al., 1993; Rowe & Kahn, 1997). In the following section, we review theoretical arguments and empirical results regarding key candidate factors that may contribute to such disparities.

DISPARITIES IN THE PROGRESSION OF LATE-LIFE WELL-BEING: THE ROLE OF INDIVIDUAL FACTORS

In the above section, we have identified a venue wherein the mechanisms contributing to changes and differences in well-being may be more easily observed, and we have highlighted the extent of between-person inequalities in the experience of late-life well-being. In this section, we investigate factors underlying these differences. Specifically, we identify some of the specific variables that may contribute to disparities in well-being. Figure 10.2 provides a selective list of prime candidate individual factors that may affect (trajectories of) late-life well-being. In turn, we discuss theoretical notions highlighting the role of each factor and review the available empirical evidence.

Chronological Age

Late-life changes in well-being and the interindividual differences therein appear to be driven primarily by mortality-related change processes and more efficiently charted in relation to impending death (as opposed to age, or time from birth). However, chronological age may still be part of the picture and profoundly influence late-life well-being (see Figure 10.2). People who are dying at relatively younger ages may experience their last years differently from people who are dying at relatively older ages. Conceptual arguments suggest differences that may go in either direction. Riegel and Riegel (1972), for example, have argued that the effects of mortality-related processes are diminished in very old age because people in their 80s or 90s often die from more random causes than relatively younger individuals, let's say in their 70s. A contrasting conceptual perspective suggests that the effects of mortality-related processes are exacerbated in very old age. For example, Baltes and Smith (2003) have argued that the vulnerability, unpredictability, and biocultural constraints that appear in very old age will make the system of self-protective processes associated with the maintenance of well-being become increasingly vulnerable.

The empirical evidence regarding age differences in the progression of end-of-life well-being is fairly consistent in suggesting that the oldest old are often at the limits of their adaptive capacity. There is evidence to suggest that the pathways into mortality for very old individuals are portended by relatively stronger well-being decline and/or by spending more years in the terminal periods of decline than individuals dying at earlier ages. For example, in the Berlin Aging Study we found that the rate of mortality-related well-being decline for individuals who died after age 85 was twice as steep than that of individuals who died when between ages 70 to 84 years. Similarly, those dying at older ages exhibited a threefold increase in steepness of decline from the preterminal phase to the terminal phase, relative to a much shallower increase for those dying at younger ages. When looking at differences in the

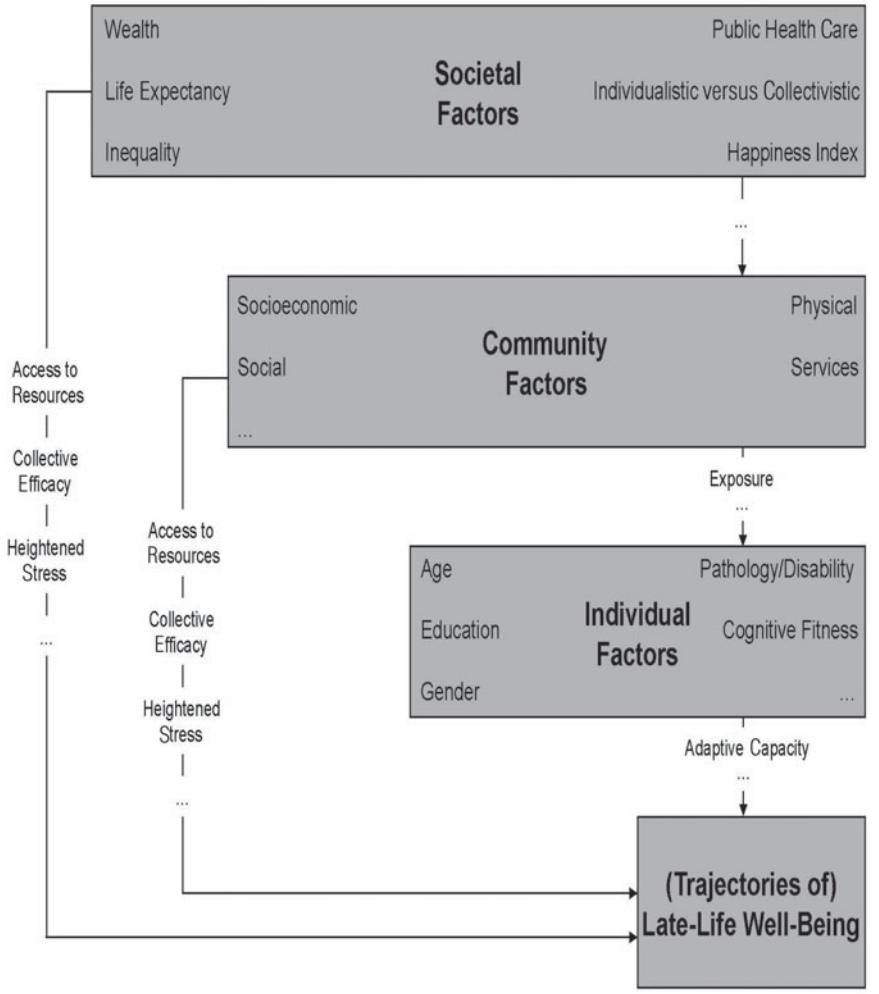

FIGURE 10.2 Graphical representation of possible factors (boxes) and underlying mechanisms (arrows) affecting late-life well-being and its progression toward the end of life.
Note. At the individual level, factors such as sociodemographic characteristics, pathology, disability, cognitive fitness, and so forth may each contribute to between-person disparities in (trajectories of) late-life well-being. One exemplar mechanism by which individual factors may operate on well-being are limitations in one's adaptive capacity. At some higher levels of aggregation, community factors such as socioeconomic, service, social, or physical characteristics may also affect well-being. Possible direct mechanisms include access to resources, collective efficacy, or heightened stress as well as exposure as an exemplar indirect mechanism. At a still higher level of aggregation, societal factors including wealth, life expectancy, or a public health care system may additionally shape the nature and course of late-life well-being. Such national factors may operate either directly or indirectly.

onset of terminal decline, we found that individuals dying at older ages spent more years in the terminal periods of life satisfaction decline than individuals dying at earlier ages. This effect amounted to an estimated additional 7 months being spent in the terminal-decline phase per additional decade lived (Gerstorf et al., 2008a). This overall pattern is in line with the virtual lack of empirical support for the Riegel and Riegel hypothesis in the large body of research accumulated in the cognitive aging literature. If anything, studies suggest the opposite with steeper cognitive decline per additional year of age (for review, see Bäckman & MacDonald, 2006).

By and large, the evidence to date suggests that the effects of terminal decline are more pronounced with greater age at death. A related point to be targeted more specifically in future research is how late-life well-being changes manifest in individuals who die at relatively young ages, lets say in their 50s or 60s. Following the reasoning of increasing limits in adaptive capacity (Baltes & Smith, 2003), one might argue that middle-aged adults, relative to older people, may have a larger pool of resources to draw from and thereby might be better able to ward off the detrimental effects of impending death. There is initial evidence to suggest that late-life declines in well-being are not restricted to individuals who died in old age, but are also found when all adult decedents, no matter their age of death, are examined (Gerstorf et al., in press). If substantiated, this pattern speaks to the pervasive nature of mortality-related processes, but also signals that more in-depth work is necessary to thoroughly examine these and other age-differential questions (e.g., the role of cause of death).

Gender

Gender-linked inequalities have long been acknowledged in the lifespan and gerontological literature (for overview, see Moen, 1996). To start with, various life-course sociological theories contend that women have experienced status and opportunity disadvantages throughout their lifetime and across a variety of different life domains including education, work, social status, family life, and finances (Elder, 1998). It is argued that such disadvantages follow current cohorts of women into old age and continue to reveal lasting negative effects into the last years (cf. Smith & Baltes, 1998). In a similar vein, the objective life circumstances late in life can be expected to be different for men and women. Regarding health, for example, it is well established that women typically experience more debilitating diseases than do men (Crimmins, 2001). Also, demographic trends show that women have higher life expectancies than men and that husbands are prototypically older than their wives. Both demographic factors make for a scenario where women must often deal with the late-life challenges as widows, whereas older men are often still married and

able to draw resources from their partners (see Suzman, Willis, & Manton, 1992; Turner & Troll, 1994). Conjointly, all these factors suggest that one can expect gender differences to persist into and be readily apparent late in life (see Figure 10.2), and women can be expected to be more vulnerable than men.

Empirical evidence addressing such notions is relatively scarce, partly due to several study samples exclusively involving men, but no women. For example, the above conceptual reasoning may be taken to suggest that the steep well-being decline exhibited by men from the Normative Aging Study who died within 12 months after assessment (Mroczek & Spiro, 2005) may not generalize to women. Instead, women's reports of late-life well-being may be even lower or show more abrupt decline. Detailed studies are warranted to pinpoint if and how lower levels of well-being found in some middle-aged and older samples (e.g., more depressive symptoms: Smith & Baltes, 1998) persevere into the last years and to address the extent to which gender may moderate the progression of late-life change.

Education

People in different educational strata or, by implication, socioeconomic strata may experience the last years of life differently. One of the central arguments is that people with higher educational attainment may have more resources to adapt to late-life challenges and thereby are better equipped to maintain well-being later in life (see Figure 10.2). Such resources are thought to generally help individuals achieve their goals and deal with changes in living conditions, and thus encompass a variety of factors ranging from finances to personal characteristics, such as self-regulation strategies. In contrast, one opposing view may suggest that end-of-life declines are so pervasive and all-absorbing that preexisting differences are diminished or even eliminated.

A relatively ubiquitous finding in studies of adult samples is that education and socioeconomic conditions show small, but positive and consistent associations with levels of well-being across adulthood and old age (for reviews, see Argyle, 1999; Diener et al., 1999). It is a fairly open question, however, whether differences between education groups in late-life well-being exist, and if so, whether they reflect the persistence of differences that have already existed since earlier phases of adulthood or whether such differences become minimized or exacerbated at the end of life.

Pathology and Disability

Etiological questions about what underlies precipitous late-life declines in well-being have not yet been thoroughly addressed, but processes of pathology and

disability can be expected to play a major role (see Figure 10.2). The Disablement Process model, for example, implicates disability as a major force underlying developmental change (Verbrugge & Jette, 1994). According to this model, intraindividual resources such as a high levels of well-being may serve as a protective factor in the course of disablement, while low levels of well-being may exacerbate rates of physical decline. Likewise, being multidirectional in nature, this disability-driven perspective suggests that systematic changes in well-being may be driven by the time course over which individuals experience an accumulation and increasing severity of chronic diseases and disability (see Ram, Gerstorf, Fauth, Zarit, & Malmberg, 2009). It is similarly conceivable that the different causes of death in very old age and the conditions associated with the process of dying may account for differential portions of between-person disparities in terminal decline of well-being. More specifically, people who have died from debilitating diseases may be more likely to display steeper well-being declines than people who have died from more sudden causes of death (e.g., stroke or heart attack).

What does the empirical evidence suggest? To begin with, severe disability has been reported to be associated with long-lasting changes in well-being (e.g., lower levels persist over time: Headey, 2008; Lucas, 2007). Future research should specifically target if, for example, terminal decline in well-being is steeper among persons with late-life disability relative to nondisabled older persons. It would also be instrumental to determine the shared and unique contributions of processes involved in accumulating disability and approaching death to late-life changes in well-being. In addition, the cognitive aging literature suggests that terminal decline is relatively independent of cause of death (e.g., Anstey, Mack, & von Sanden, 2006; Small, Fratiglioni, von Strauss, & Bäckman, 2003; but see Wilson et al., 2007), but this pattern may not generalize to terminal decline in well-being. Unfortunately, researchers are often unable to examine such questions with the data at hand because (reliable) information about cause of death is not available. If data were indeed available, substantial insights into the phenomenon could be gained.

Cognitive Fitness

A final individual factor we discuss in this chapter is cognitive fitness (see Figure 10.2). This is important because mortality-related declines in well-being may merely reflect the effects of low levels of cognitive functioning or cognitive decline and thereby essentially parallel the well-established cognitive terminal decline (Bäckman & MacDonald, 2006). In other words, well-being declines may simply be an epiphenomenon of cognitive declines. Another position highlighting the relevance of taking into account cognitive fitness and deteriorations

therein argues for a possible downward cascade of steps involving an initial decline in intellectual functioning, followed by a decline in subjective well-being, and finally death (cf. Maier & Smith, 1999; see also Birren, 1959).

Again, the empirical evidence is very sparse and much more work is needed to fully resolve this open issue. Data from the Berlin Aging Study, however, has provided some level of evidence to suggest that terminal decline in life satisfaction cannot be attributed to cognitive impairments and decline alone (Gerstorf et al., 2008b). More specifically, we examined the role of clinical diagnosis of dementia and perceptual speed at baseline assessment as time-invariant predictors, as well as preclinical dementia as a time-varying predictor of between-person inequalities in level and change of well-being. Results revealed (a) no predictive effects of cognitive functioning for well-being decline, (b) low intercorrelations between cognitive declines and well-being declines, and (c) that the overall pattern of mortality-related well-being decline remained unchanged after cognitive functioning was taken into account. In sum, there was no evidence for a primacy of cognitive terminal decline over mortality-related decline in life satisfaction. However, limitations of statistical power make it difficult to provide definitive answers regarding such cross-domain links (Hertzog, Lindenberger, Ghisletta, & von Oertzen, 2006).

In sum, we have reviewed theoretical accounts and empirical reports that a number of individual factors may play a pivotal role for between-person disparities in the progression of late-life well-being. Our review has been selective in nature and many further factors such as race and ethnicity can be expected to profoundly shape late-life well-being (see Jackson, Antonucci, & Gibson, 1995; Whitfield & McClearn, 2005), but have rarely been targeted in empirical research. Examining these variables separately, or preferably conjointly (see Baltes & Smith, 1997; Garfein & Herzog, 1995), will shed light on why some individuals are capable of maintaining well-being into very late in life, whereas many others' well-being declines steeply.

DISPARITIES IN THE PROGRESSION OF LATE-LIFE WELL-BEING: MOVING BEYOND THE INDIVIDUAL LEVEL

Having identified a number of individual difference variables that may substantially contribute to between-person disparities in end-of-life change in well-being, we now move one step further and consider variables located at the contextual level. Specifically, following developmental contextualist ideas, the characteristics of the communities and societies in which individuals live likely provide insights into if, how, and why between-person disparities in (the progression of)

end-of-life well-being exist. To set up a framework for future inquiries, we first briefly provide a broad conceptual embedding for our proposal and consider theoretical accounts that relate the various service, social, and physical components of the community (listed in Figure 10.2) to key individual-level outcomes, such as (trajectories of) psychological health and well-being. In a second step, we target the societal level and consider conceptual arguments and empirical evidence if and how between-person inequalities in well-being can be accounted for by various national characteristics (also listed in Figure 10.2).

Communities and Between-Person Disparities in Late-Life Well-Being

Life-span psychological and sociological perspectives have long advanced that individuals live in contexts that create both opportunities for, and constraints on, individual developmental pathways (Baltes, 1997; Cairns, Elder, & Costello, 1996; Elder, 1974; Lerner & Kauffman, 1985; Magnusson, 1996; Riley, 1987; Verbrugge & Jette, 1994). One branch of this perspective has focused on the importance of ecological factors, which include views as diverse as social disorganization (Faris & Dunham, 1939), human ecology (Bronfenbrenner, 1979), environmental gerontology (Lawton, 1982), or environmental psychology (Wohlwill, 1970). For example, social disorganization theory (Faris & Dunham, 1939; Sampson, Raudenbush, & Earls, 1997) proposes that characteristics of disadvantaged neighborhoods such as poverty and residential instability result in attenuated institutional strength, limited network interaction, diminished neighborhood attachment, and low levels of informal social control. Such macrolevel factors, in turn, can be expected to be linked to individual-level outcomes such as increased risk for victimization and compromised mental health (e.g., depression, psychiatric disorders). In a related vein, notions of environmental gerontology have long highlighted the critical role of both physical and social environments for the maintenance of functioning into late life (Baltes, 1996; Wahl & Lang, 2004). As a classic example, Powell Lawton's environmental docility hypothesis suggests that such environmental features become increasingly important as personal competences decline (1990).

We acknowledge that the nomenclature and specific definitions differ between the various areas of study, and that important distinctions are often made (e.g., communities vs. neighborhoods) in the various disciplines. In the current chapter, we are primarily focusing on the common feature of these approaches to highlight that characteristics of the broad residential area people are living in are important for individual-level outcomes. We thus decided to primarily use the more generic term community effects, but also use this term interchangeably with neighborhood effects or effects of the residential environment.

Making liberal use of these perspectives, we consider three features of individuals' contexts—service, social, and physical components ranging from more microlevel features of one's residence to more macrolevel characteristics of one's neighborhood, city, or county (for overview see Leventhal & Brooks-Gunn, 2000; Robert, 1999; Sampson, Morenoff, & Gannon-Rowley, 2002; Subramanian, Kubzansky, Berkman, Fay, & Kawachi, 2006; Wahl & Lang, 2004). In the following, we review conceptual accounts linking these contextual-community features (see Figure 10.2) to individual-level outcomes and use select empirical findings for illustration. We note that the distinction between components is primarily of heuristic value and acknowledge that a given community characteristic often cuts across various components.

Service. One set of features highlights the importance of the service environment and particularly the role of institutional resources (e.g., quantity, quality, density, availability, affordability, and accessibility) for promoting a healthy environment and accommodating people's needs (Leventhal & Brooks-Gunn, 2000). For example, wealthy communities may allow for and draw higher quality health services and institutions that directly or indirectly benefit health (Browning & Cagney, 2003). Similarly, individuals who spent the last years of their lives in communities with a dense system of high-quality health care may have better chances of maintaining well-being in the face of increased risks for debilitating diseases as compared with individuals living in communities with poor health care access.

Social. A second set of concepts point to the social environment and to mechanisms that are broadly tied to processes of social cohesion and collective efficacy (see Bandura, 1986; Sampson et al., 1997; Thompson & Krause, 1998). For example, socioeconomically deprived areas are often defined by factors that undermine the development or maintenance of social integration, positive affiliations with others, and a generally supportive and engaging community culture. These factors might manifest as features of the social environment including residential instability, various forms of incivility (e.g., criminal victimization) or high levels of social mistrust. The resulting lack of social ties and support may either have direct implications for well-being and health or indirect effects, either via factors such as ambitions, attitudes, and motivation, or via processes of informal social control such as monitoring, supervision, and the availability of role models (see Berkman, Glass, Brisette, & Seeman, 2000; Cacioppo, Hughes, Waite, Hawkley, & Thisted, 2006; Cohen & Wills, 1985; House, Landis, & Umberson, 1988; Seeman, 2001).

Physical. A third broad category of neighborhood factors with possibly moderating (e.g., deleterious or beneficial) effects on individual health and well-being includes the physical features of environment (Lawton, 1982;

Wahl, 2001). Physical factors that may be of relevance for well-being include noise and pollution, heightened stress and risks for accidents, or inadequate sanitation (see Krause, 1996). In a similar vein, the Disablement Process model (Verbrugge & Jette, 1994) proposes that extra individual factors in the physical or built environment can make a profound difference in whether or not functional limitations result in disability. For example, a woman with limitations in lower body functioning (e.g., being able to walk only a couple of steps consecutively) may still be able to leave her house on a regular basis when she is living on the ground floor, but not when she is living on the sixth floor without an elevator (see also Clarke & George, 2005).

The above conceptual arguments map onto a series of empirical reports documenting that neighborhood-level characteristics such as socioeconomic disadvantages or violence are indeed linked with individual-level physical and psychological health variables reflecting functional health, well-being, and mortality (Argyle, 1999; Balfour & Kaplan, 2002; Kawachi & Berkman, 2003; Krause, 2003; Marmot & Wilkinson, 1999; Sampson et al., 2002; Silver, Mulvey, & Swanson, 2002). However, the great bulk of these studies has focused on outcomes in early phases of life such as childhood or adolescence and often has been cross-sectional in nature. We propose that key insights can be gained from addressing if and how community factors relate to between-person disparities in well-being at the end of life as well as its progression over time (i.e., developmental change).

Data from adult samples provides some preliminary evidence for such a proposal. For example, Shields, Price, and Wooden (2009) reported from an Australian data set that neighborhood effects accounted for less than 3% of interindividual differences in reports of life satisfaction (for similar reports from the United Kingdom, see Propper et al., 2005). Based on tenets of life-span psychology (Baltes, 1997), we would expect that the size of the overall community-level effects would be considerably larger when the last phase of life is targeted and also generalizes to the consideration of change trajectories. More specifically, an ever-growing need for cultural resources (e.g., supportive environments) can be expected in order to compensate for the increasing "biogenetic incompleteness of human life" (Baltes, 1997) as evidenced in, for example, accumulating health constraints. Accordingly, it appears conceivable that individuals late in their lives are particularly susceptible to residential environments, probably because health constraints and functional limitations may necessitate a greater reliance and dependency on municipal resources and services (e.g., Lawton, 1990).

To empirically address such a proposal, two sets of analyses could prove instrumental. In a first step, it would be key to quantify the relative contribution

of community characteristics to late-life disparities in well-being and its progression toward the end of life. Such a finding would be important in and of itself because it provides a rough quantification for the extent to which individual development and end-of-life quality of life is shaped by the structure of opportunities and constraints in one's living environment. This would also help determine how much of what would conventionally be thought to reflect differences between persons in reality is a reflection of differences between the residential areas where people live. In a second step, the objective would be to explore the role that specific service, social, and physical characteristics of the communities may play for between-person heterogeneity in (the progression of) late-life well-being. The above considerations lead us to expect that, for example, individuals living in wealthy communities may report higher late-life well-being and show less pronounced decline relative to individuals living in poorer communities, over and above key individual predictors of well-being. Also, it appears conceivable that the predictive effects of individual factors such as lower education or disability do not necessarily operate similarly across context, but are exacerbated in structurally disadvantaged communities. For example, disabled people in poor communities may show more pronounced decline with approaching death than do disabled people in wealthy communities. Taken together, such results would be consistent with conceptual notions that the contexts in which people are living and dying may impose additional constraints onto the already compromised adaptive capacity at the end of life. More in-depth studies are needed to pinpoint the specific mechanisms by which macrolevel characteristics permeate or get under the skin of individuals (cf. Seeman, 2001).

Societies and Between-Person Disparities in Late-Life Well-Being

A better understanding of between-person inequalities in late-life well-being may also substantially benefit from targeting the role of factors that reside at an even higher level of aggregation, namely nations and societies (see Figure 10.2). A first research approach is to treat samples from different nations that share basic characteristics (e.g., individualistic societies with comparable wealth) as independent replications of one another. If substantively similar findings emerge, this may serve replication and generalization purposes and be taken to indicate the robustness of the phenomenon of interest. For example, we have used long-term longitudinal data of deceased participants in national samples from Germany, the United Kingdom, and the United States to examine the prototypical onset of mortality-related well-being decline. Despite only partial overlap at the measurement level, we found strikingly similar construct-level results: In all three nations, we identified prototypical transition points between

3 and 5 years prior to death, after which normative rates of well-being decline steepened by a factor of three or more (Gerstorf et al., in press).

A second highly valuable research approach is to specifically target the role that communalities and differences between nations may play for between-person disparities in late-life well-being. To start with, national differences have long been reported in average levels of well-being (see Diener et al., 1999). For example, the Gross National Product per capita, an indicator of national wealth, typically shows moderate to strong relations with indicators of well-being across nations (Inglehart, 1990): People living in richer nations also report higher average levels of well-being. Similar relations have been repeatedly documented for other economic and social indicators at the national level, including inflation rate, overall level of economic inequality (e.g., Gini coefficient), unemployment rate, amount of government-provided unemployment benefits, and so forth (see Di Tella, MacCulloch, & Oswald, 2003; Haller & Hadler, 2006; Veenhoven, 1995). Further, as noted in several comparisons of average well-being in individualistic versus collectivistic nations, cultural norms and attitudes also relate to average levels of well-being (e.g., Diener & Suh, 2000). However, the available longitudinal evidence regarding changes in well-being is less conclusive. Some studies report that national changes in economic development and democratization are accompanied by changes in average levels of well-being (e.g., Inglehart, Foa, Peterson, & Welzel, 2008), whereas other studies rather suggest stability (Diener & Oishi, 2000; Easterlin, 2005; Kahneman &Krueger, 2006). Even less is known about if and how societal-level differences map onto individual-level changes in well-being and what such associations look like at the very end of life.

We argue that between-person inequalities in late-life well-being and its progression toward the end of life may in part be shaped by societal-level differences (see Figure 10.2). To start with, it is conceivable that effects of the above-listed key variables like income inequalities follow people into the last years of life. One possible scenario is that between-person disparities in well-being decline are substantially exacerbated in nations with large income inequalities. In addition, national differences in factors that directly or indirectly link to late-life health outcomes are well documented. These variables include, but are not limited to, life expectancies or a public health care system. We would expect between-person inequalities in well-being decline to be diminished in nations with a well-functioning public health care system as opposed to nations that lack such systems.

To conclude, our objective has been to draw from a long history of research in a variety of different disciplines so as to argue that community

and societal factors both represent a very important unit of analysis in understanding between-person disparities in the progression of late-life well-being. The preliminary evidence reviewed generally supports this proposal and offers routes for future inquiry to substantiate and expand these initial findings.

SUMMARY AND OUTLOOK

In sum, we have made three sets of arguments about the study of between-person disparities in late-life well-being. First, ubiquitous reports of a "stability-despite-loss phenomenon" of well-being do not generalize into years of life immediately preceding death. Instead, mean-level representations of the end of life are characterized by a rapid deterioration in well-being. Second, there is vast heterogeneity in how people experience the last years, and biopsychosocial individual difference factors like sociodemographic characteristics, cognitive fitness, pathology, or disability may all provide both unique and shared contributions to such disparities. Third, we have argued that macrocontextual factors such as the social, service, and physical characteristics of the communities and societies people are living and dying in profoundly shape the nature of microlevel processes and thereby contribute to inequalities in the progression of individual late-life well-being. The conceptual reasoning forecasts some of the insights that can be gained by pursuing this line of research, but also underscores the challenges researchers must deal with.

Living a life with dignity and maintaining well-being is a key ingredient of quality of life. The line of research briefly sketched in this chapter promises to shed some light on the patterns of well-being decline in late life, and help us understand between-person differences in such trajectories and the role that factors at the individual-, community-, and societal-level may play. Such knowledge about how, why, and which segments of society may be experiencing decrements in psychological health and well-being shall inform the development of social policy and preventive intervention programs that may eventually alleviate the societal and personal costs of late-life decline and reduce or minimize between-person disparities in the decline of well-being at the end of life.

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AUTHOR NOTES

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CHAPTER 11

Work, Retirement, Race, and Health Disparities

Edna Brown

The number of individuals reaching retirement age and living well beyond is increasing at a rapid pace. The U.S. Census Bureau (2006) predicts that by 2020 over 42 million adults will reach 55–64 and over 54 million adults will be 65 years old or over. Although previously much of the adult life was spent in the workforce, in the last few decades Americans have changed the way work and retirement is approached and organized (Rozen, 2007). According to the latest labor statistics (U.S. Bureau of Labor Statistic [BLS], 2008), changing occupational patterns include increased self-employment, working out of the home, and serving as independent contractors. Thus, much of our current and future workforce is employed in professional and technical jobs. Unfortunately, these statistics also show that the gap between the types of occupations, salaries, and rates of employment for minority groups and White Americans is significant.

Labor discrimination at many levels has existed for many years. Historically, the types of jobs that were available to Black Americans have often been low-skilled, low-paying, transient positions (e.g., laborers, domestic workers; Ferraro & Farmer, 1996; Flippen & Tienda, 2000). Further, many of the available jobs have included those with hazardous working conditions or physically demanding labor associated with increased health risks (Williams, Costa, Odunlami, & Mohammed, 2008; Williams & Jackson, 2005). Yet, even in the face of structural disadvantages (e.g., discrimination) and unequal access to the labor market, Black Americans have long histories of

labor force participation. For example, current labor force trends show that more women of all ethnic backgrounds are entering the workforce. Regardless of marital status, Black American mothers have the highest labor force participation of mothers of any other racial or ethnic group (Johnson & Staples, 2005). In fact, historically, Black American women's employment has been vital to the economic survival of the Black American family. Too often, Black American men have been portrayed as inadequate providers, but studies show that when discriminatory obstacles and barriers are removed and they are allowed fair access to traditional economic opportunities and resources, such as education and jobs, then Black American men are able to successfully fulfill the provider role for their families (McLoyd, Hill, & Dodge, 2005; Taylor, Jackson, & Chatters, 1997; Warner & Hayward, 2006).

This chapter focuses on the complex relationship between work, retirement, race, and health disparities among aging Black Americans. This chapter utilizes a life-course perspective to consider how preretirement experiences, including health and job statuses, affect transitioning out of the labor force for these older Americans. Life-course theorizing (e.g., Baltes, 1987; Carstensen, 1993; Freund & Baltes, 1998) facilitates examining the continuity between pre- and postretirement experiences.

According to Elder (1998), the trajectories and transitions individuals face are influenced by the biological and social pathways encountered across the life span. Thus, the quality of retirement experiences is directly dependent on preretirement health and work histories. Research has consistently shown that Blacks have lower income, education, and job status than others in the population, and are less likely to have pension plans or savings needed for a smooth transition into retirement (Hayward, Friedman, & Chen, 1996). Research has also shown that Blacks have poorer health and higher mortality than other groups in our society (Ferraro & Farmer, 1996; Williams & Jackson, 2005). These preretirement work and health disparities can have negative influences on the quality of the retirement experiences of Black elderly and deleterious effects on their health and well-being in later life. On the other hand, being in poor health and retiring from low-paying, low-status positions might be beneficial to overall well-being. Examining the continuity between work and retirement among Blacks yields a richer, more comprehensive understanding of this process for an American group that is disadvantaged. Life-course development posits that health problems experienced early in life affect later life health status. Health inequities begin early and persist throughout the life course and by normative retirement ages, health status has already been established. A goal of this chapter is to discuss the dynamics of health disparities in the contexts of work and retirement.

THE LINK BETWEEN WORK, RETIREMENT, AND HEALTH DISPARITIES

Defining Retirement

One of the most important transitions for all aging Americans is from active, productive work to nonwork statuses. The transition, however, is a complex process influenced by preretirement resources, status, and opportunities (Kim & Moen, 2002). For example, on one hand, for many older Black Americans in low-skilled, low-status jobs reaching normative retirement ages, there is no real change in the level of income but there may be less uncertainty about earning that income. On the other hand, retiring from low-paying, low-status positions might be beneficial to overall well-being. Whereas for many White American retirees the receipt of Social Security may serve as supplement income to pension benefits and other financial assets, for many Black Americans Social Security represents a steady, reliable sole source of income (Jackson & Sellers, 2001). Thus, retirement with Social Security benefits may improve quality of life and health outcomes during normative retirement ages among Black Americans.

Economic advantages throughout the life course serve as a protector against adverse outcomes later in life (Jackson, 2001a; Williams et al., 2008). Early financial security leads to higher educational attainment and thus better occupational status during adult working years. These socioeconomic factors contribute to greater income and wealth assets, health benefits, and ultimately health outcomes (Kahn & Fazio, 2005). Lack of financial resources, on the other hand, are related to lower educational attainment, lower occupational status, fewer benefits, and negative health outcomes across the life span. These socioeconomic factors are a few of the mechanisms that influence the quality of transition experiences during the retirement years.

Scholars have also argued that individuals' subjective perceptions of work and retirement status are good gauges of the quality of the transition experience (Jackson & Gibson, 1985; Marshall, Clarke, & Ballantyne, 2001). The transition from work to retirement status often remains blurred for older minority workers (Brown, Jackson, & Faison, 2006; Marshall et al., 2001). For example, Jackson and Gibson (1985) and Gibson (1987) have conceptualized the working experiences of older Black Americans along three, interrelated, perceived objective and subjective dimensions: working (20 hours per week or more), retired (not working at all or working less than 20 hours per week and self-identified as retired), and nonretired (reported not working at all or working less than 20 hours per week for reasons other than being retired). Research has shown that each of these subjective statuses, depending upon earlier life experiences, had significantly different

characteristics reflecting financial and health conditions, social and psychological background factors, social statuses, and social attitudes (Brown et al., 2006; Jackson, 2001b). Not atypically, many black elderly continue to work out of economic necessity rather than intrinsic reward (Coleman, 1993).

Defining Disparities in Health and Work

Health disparities refer to those avoidable differences that result from cumulative social disadvantage. Healthy People 2010 refer to health disparities as inequalities that occur across different demographic factors such as gender, race or ethnicity, education, and income among other factors (U.S. Department of Health and Human Services [DHHS], 2000). Adler and Rehkopf (2007) maintain that health disparities are related to both biological and social conditions. They suggest that biological differences in health disparities may be attributable to natural biological differences between men and women. These types of differences occur, for example, in the rates of cancer for certain areas of the body, as in ovarian cancer among women or prostate cancer among men. Adler and Rehkopf (2007) maintain that health disparities that are attributed to social conditions are related to possession of power, access to resources and accordingly, are "avoidable and inherently unjust" (p. 237). One study provides an illustration. In research designed to examine the complexity of race, income, and disparities in health, Williams and Jackson (2005) found racial differences in health at comparable levels of income. They found that throughout adulthood, Black American men and women reported poorer health than their White counterparts. Although education attainment is related to better health, Williams and Jackson (2005) found that among women, Black American college graduates had higher infant mortality rates than White, Hispanic, and Asian and Pacific Islanders who were high school dropouts. This provides further evidence that within group analyses are needed to disentangle the link between race, income, work, and health and that the cycle of racial disparities in health begins early in the life course.

Early life social and health conditions, work, and unemployment are among the various social determinants that influence individual health (Centers for Disease Control and Prevention [CDC], 2005; Williams & Collins, 1995). Retirement period is often associated with downward mobility for those with intermittent employment during working years. Instability in employment is related to higher health and mortality risks than in more stable positions (Lipscomb, Loomis, McDonald, Argue, & Wing, 2006; Pavalko, Elder, & Clipp, 1993; Warner & Hayward, 2006). While poor working conditions and meager benefits contribute to disparities in health, this effect is bidirectional. Individuals with poor health have lower

rates of employment and lower wages than individuals in relatively good health. These work and health connections persist across the adult working life (Bound, Waidmann, Schoenbaum, & Bingenheimer, 2003).

PRERETIREMENT WORK, HEALTH, AND ECONOMIC CONDITIONS

Factors that influence health disparities in older age have origins in earlier life (Miech, Eaton, & Brennan, 2005). Over the years, research has documented that Black Americans across the life course suffer health, social, and economic deprivations in comparison to others in this society; these disadvantages make the transition from working to retirement status more difficult than among other race and ethnic groups (Hayward et al., 1996; Hayward, Friedman, & Chen, 1998; Jackson, 2001a, 2001b; Kahn & Fazio, 2005). On average, Black Americans earn only about 65% of what other Americans earn with similar education and employment positions (McAdoo, 2007; McLoyd et al., 2005). Black households tend to rely on earnings from multiple members. Studies indicate that college educated Black adults are more likely than their White counterparts to experience unemployment (Williams & Collins, 1995). Further, when Black Americans start at the same income levels in the same jobs, their rate of promotion and raises are lower than other employees (McAdoo, 2007; McLoyd et al., 2005). In addition, receiving fewer benefits due to lower paying jobs is also related to poor health outcomes (Williams, Yu, Jackson, & Anderson, 1997).

Work force histories associated with low-skilled jobs, longer periods of unemployment, more temporary and part-time positions are linked to higher mortality, fewer economic resources, and retirement benefits (Marshall et al., 2001; Pavalko et al., 1993). Research has found that compared to White women, Black women are in the workforce longer and are more likely to remain in the workforce during normative retirement ages. Black Americans are vulnerable to involuntary job separation and are likely to drop out of the work force rather than retire, in the traditional sense (Gibson, 1991b). Instability in the workforce prevents an accumulation of benefits and security (Kim & Moen, 2002; Marshall et al., 2001). Further, Black Americans are more likely to be engaged in physically demanding jobs that cause rapid physical functional decline.

Compared to higher socioeconomic status, lower socioeconomic status is associated with higher levels of disease and illness (Williams & Jackson, 2005). Across all income levels, the health of Black Americans of all ages decline faster than White older adults (Ferraro & Farmer, 1996; Williams &

Jackson, 2005). These race differences in health, occupation, and career paths are among the various factors related to racial health disparities (Jackson & Sellers, 2001; Pavalko, Mossakowski, & Hamilton, 2003; Williams & Jackson, 2005). Further, research suggests that the pattern of work histories is related to longevity. Pavalko and colleagues (1993) found that compared to men who remained in stable positions, those who experienced instability in the labor market had a higher mortality risk.

Preretirement Occupation, Working Conditions and Health Disparities

Occupation status is a better gauge of the link between work and health than is a measure of income at any one point in time (Gueorguieva et al., 2009; Williams, 2005). Lipscomb and colleagues (2006) state that few studies have examined the direct link from intermittent work histories, gender, race, and health disparities. They contend, however, that there is ample empirical evidence of the conceptual links between these factors. For example, to examine the impact of occupation on health over time Gueorguieva and colleagues (2009) conducted a longitudinal study of adults in preretirement ages (50–64) at baseline. Over seven waves (12 year period) they found that across a series of occupations occupation-related differences in health outcomes remained after controlling for education, income, and wealth. Participants in lower status jobs rated their health poorer than those with higher status occupations and these health differences persisted over time. Although the magnitude of the differences was reduced when sociodemographic variables were included in the models, the differential effect of types of occupation on health remained. These findings are similar to other studies that examined the link between work-related dynamics and health (Warren, Carayon, & Hoonakker, 2008). Several factors associated with occupation status over the life course, particularly in preretirement ages, are related to self-rated health in retirement. Self-rated health is used in several studies as a reliable, valid measure of health and a significant predictor of functional health decline, morbidity, and mortality (Gueorguieva et al., 2009; Idler & Benyamini, 1997; Warren et al., 2008).

Over the course of one's working life, feelings of control, physical and psychological stressors, benefits and compensation are among the factors related to health outcomes, including physical functioning and mental health (Burgard, Brand, & House, 2007; Gueorguieva et al. 2009). Black workers are more fearful of losing their jobs and finding other work than White workers (Lipscomb et al., 2006). Feeling a low sense of control in the job decision-making process, poor working conditions, demanding manual labor, job insecurity, and receiving little or no benefits or compensation for stressful work is related to

adverse health outcomes (Burgard et al., 2007; Lipscomb et al., 2006; Warren et al., 2008). Minorities are more likely to be employed in jobs with high exposure to hazardous work conditions. Lower skilled jobs are related to higher exposure to environmental hazards and physically demanding work. Further, exposure to toxic materials and chemical and hazardous conditions found in some industries where many minorities find work increases the risk of illness (Lipscomb et al., 2006; Williams & Collins, 1995). Occupational accidents are among the major causes of mortality for Black men. Although White men are more likely to experience a work-related injury, Black men are more likely to die from an accident in the workplace (Braithwaite, 2001). Previous research found that White male workers were more likely to receive accommodations for returning to work after work-related health concerns than are minority workers (Strunin & Boden, 2000). Employers were willing to reduce work hours, reduce work tasks or load when White employees returned to work, while non-White workers were not offered these same accommodations, and in some instances were not allowed to return to work after an injury. These types of discriminatory behaviors and policies in the workplace contribute to exiting and reentering the workforce and ultimately observed health disparities.

In addition to environmental toxins, chronic exposure to stress also contributes to chronic health conditions. Persistent discriminatory practices in the labor force foster perceptions of racism. Perceptions of racism in the workplace and related stress are mechanisms through which poor physical and mental health outcomes occur (Williams & Jackson, 2005). Williams and Jackson, (2005) argue that chronic exposure to stressors such as low control, perceptions of racism, and discrimination in the workforce is linked to altered physiological functioning and health conditions and contribute to elevated risks of disease and health disparities.

More recently there has been a transition from manufacturing to service-oriented jobs resulting in higher rates of unemployment for unskilled minority workers and even less autonomy or control regarding employment options (Flippen & Tienda, 2000). Further, little opportunity exists for retraining minority older workers. Research (e.g., Brown et al., 2006; Kim & Moen, 2002) suggests that minority men and women are more likely to withdraw from the labor force and tend to move from unemployment to nonretired or nonparticipation statuses rather than a formal retirement transition.

Previous research has found that at all levels of education Black and Latino men face economic disparities during both pre- and postretirement periods. Hogan, Kim, and Perrucci (1997) found that the employment income of Black and Latino men averaged 60% of White men employment earnings. During retirement, the racial gap is somewhat reduced due to

Social Security benefits. Black and Latino men average 72% of White men's retirement earnings; compared to earning assets, however, the gap increases to 15% of White men's asset earnings. They concluded that these gaps in income during pre- and postretirement indicate that even among the highly educated minority men, Black and Latino men are unable to accumulate assets that will sustain them in their retirement years. These findings provide additional evidence that Black older adults may have more problems during retirement paying bills, meeting financial obligations, and receiving adequate health care.

THE RETIREMENT YEARS

The retirement transition is a complex process. The various patterns and decisions of leaving and reentering the workforce are complex and relate to education, economic resources, health and well-being, expectations for retirement and preretirement precursors and consequences (Flippen & Tienda, 2000; James & Spiro, 2006; Kim & Moen, 2002; Marshall et al., 2001). According to research studies, by traditional retirement ages the retirement pattern has been set by earlier life health status and disabilities, types of jobs (pensions or retirement plans, medical insurance), and employment (in) consistency (layoffs, displacements, involuntary unemployment; Flippen & Tienda, 2000; Gibson, 1991a).

Kim and Moen (2002) maintain that to understand the complex decision making regarding transitioning into retirement and adjustment, continuity theory and life-course ecological models are useful frameworks for examining underlying mechanisms such as economic and personal resources that affect well-being during the transition period. Economic resources involve the usual financial, educational, and work-related benefits outlined in other research. Kim and Moen, however, include health as a personal resource vital to pre- and postretirement decisions. They argue that health problems possessed when entering into retirement periods are not likely to improve or disappear. Indeed, physical and mental health problems are likely to worsen and progress during postretirement periods, thereby affecting adjustment during that time. Thus, understanding the dynamics of health and retirement in the context of disparities is a useful endeavor.

Similarly, in a longitudinal research investigation to understand health and the pathways to retirement, Flippen & Tienda (2000) found that minorities were more likely to be unemployed and out of the labor force and more likely to become unemployed over time. Similar to other studies (Brown et al., 2006; Gibson, 1991 a,b), they also found that Black Americans were more likely to

withdraw from the labor force rather than officially retire. In these studies, many labor force drop outs did so before they were eligible for Social Security benefits. Factors associated with these processes and status were education levels, previous employment history, and failing health.

In another study designed to assess the effects of instability in the retirement transition on health, Marshall et al. (2001) found that postretirement expectations were in congruence with life satisfaction. Among workers who had retired and returned to work, greater stress was experienced among the retirees who left long-term, stable positions and had to unexpectedly reenter the labor force than for retirees who desired to work after retirement. Retirees who returned to work due to financial difficulties experienced the worse adjustment and psychological distress. Involuntary retirement because of health was associated with the most negative effects on subsequent health and psychological well-being.

In another longitudinal study that examined the impact of work on the psychological health of older Americans, James and Spiro (2006) found that depressive symptoms were related to exiting the work force. Participants who experienced depressive symptoms at any given wave were more likely to be retired by the next wave. They also found that retirement status was related to larger numbers of depressive symptoms in that older adults who retired experienced an increase in depressive symptoms by the next wave. Returning to work was associated with a decrease in depressive symptoms for those who desired to work. Individuals were 56 at wave one and 66 at wave two. Exiting and reentering the labor force was associated with stress but in complex ways.

These findings from longitudinal investigations regarding different paths to retirement provide support for the contention that poor health at retirement is likely to worsen over time causing poor adjustment. But they also confirm the complex bidirectional patterns of the health-retirement process. Research however, confirms that these transitions and adjustments differ somewhat by gender.

Women, Work, and Retirement

Utilizing the Jackson and Gibson (1985) and Gibson (1987) conceptualization of work experiences (working, retired, and nonretired), Brown and colleagues (2006), designed a study to examine three different cohorts of Black older adults at preretirement and retirement periods. The purpose of the study was to understand whether the social and health determinants of subjective retirement status among Black Americans changed or remained the same over

the previous 25 years (1979, 1987, & 2003). They found that the major factors related to perceived retirement status were education, income, marital status, gender, and health. The majority of older Black adults who were labeled as nonretired (not working or working less than 20 hours per week but do not identity themselves as retired) had less than a 9th grade education but none had over an 11th grade education in all three cohorts. The nonretired were also overwhelmingly represented in the lowest income quartile (0 to $14,999), and were mostly widowed and female. The age range of these older black nonretired Americans was 65 to 79 years old. Over the course of 25 years, these findings are discouraging and of some concern. Large percentages of retirement age older Blacks consider themselves nonretired, indicating that they worked less than 20 hours a week but still did not perceive themselves as retired. Their reasons for not working more hours varied from health reasons to unable to find work and involuntary job separation. Their low-income levels also suggest that the few hours worked is not due to having sufficient funds or ample assets that would allow them to work so few hours.

The results of the Brown et al. (2006) study varied by gender and also point to the nonretired in all years as being primarily single females. This result is similar to other research findings. It is well established that on average, women have less stable work histories than men and earn about 77% of men's salaries overall (Flippen & Tienda, 2000; Kim & Moen, 2002; Pavalko et al., 1993). Women, in general, often enter, exit, and reenter the labor force several times over the life course due to family caregiving responsibilities and thereby earn less job security and future retirement benefits than their male counterparts (Flippen & Tienda, 2000). Further, research shows that women are more likely to earn lower income, and have family obligations resulting in early withdrawal from the labor market. This unstable work history means that they approach retirement age with less income or benefits. These work pattern outcomes vary by race. Black women have longer employment histories than White women but have less economic security. The literature also suggests that older Black women are the poorest among the elderly, having the lowest net worth, are widowed or divorced, and having to return to work because of lack of retirement benefits than any other group (Clark, Mungai, Stump, & Wolinsky, 1997; Fernandez, Mutran, Reitzes, & Sudha, 1998; Mutchler, Burr, Massagli, & Pienta, 1999). This pattern differs from the "bridge jobs" in which older adults voluntarily work part-time or become self-employed in order to ease into retirement, especially after retiring from high status positions (Hogan et al., 1997; James & Spiro, 2006; Quinn & Kozy, 1996). For example, in their study of older Black and White women, Zhan and Pandey (2002) found that although older Black women had worked more years and longer hours than

older White women, Black women reported lower Social Security benefits and meager, if any, additional income assets. The authors suggest two possibilities for this finding. Either Black women had participated in lower status, lower paying jobs and thus accumulated less Social Security benefits than White women, or Black women inherited little or no retirement benefits from their late or ex-husbands; of course both circumstances are also possible. The literature does suggest that older Black women, similar to Black men, usually held domestic jobs or other service jobs that required little skill or education, and offered little or no retirement benefits (Coleman, 1993).

In the Brown et al. study (2006), the group of nonretired older adults (65 to 79 years old) also reported more chronic health problems and less satisfaction with their health status than the working or retired groups. The majority were receiving Supplemental Security Income rather than Social Security, suggesting that their lack of employment was most likely due to health disabilities. Because Brown et al. (2006) also examined preretirement periods among older Black adults 50 to 64 years old, they were able to observe that the social and health determinants of the nonretired had begun earlier than traditional retirement age. A large portion of *pre*retirement age adults also reported being retired or nonretired and were also experiencing chronic health problems. The authors speculated that the self-identified retired group had access to health and retirement benefits and were therefore psychologically and financially prepared for retirement. The nonretired group, however had access to fewer benefits and were not psychologically or financially able to officially exit the labor force. Poor functional health may have forced them out of the labor market or caused them to substantially reduce the number of hours spent working. Interestingly, those 65- to 79-year-olds who were retired also reported lower incomes than their working counterparts. This emphasizes that older Black adults' incomes are likely to be from earned income rather than accumulated wealth or financial assets. This might be one reason why a high percentage of Blacks over 70 years old continue to work, despite poor health.

Interestingly, the retired groups consistently reported less financial worry than the working and nonretired groups (Brown et al., 2006). One might wonder whether for older Black elderly receiving Social Security and SSI were more financially beneficial, or at least *as* financially beneficial, as those who were working. These results might indicate that receiving a steady, dependable income, regardless of the amount, relates to better financial well-being for Black elderly. Some older Black Americans may have lower expectations for retirement based on their working experiences in low wage jobs as many of the previously held jobs lacked pension or retirement benefits. Many older

Black Americans recognize that Social Security will be the sole source of income and may look forward to exiting the labor force with a steady income (Gibson, 1991a, 1991b).

As for psychological well-being among Black elderly, the majority of the 65- and 79-year-old adults reported high levels of life satisfaction and happiness. Brown et al. (2006) also found that the nonretired, those who worked less than 20 hours per week but did not consider themselves retired, to express the greatest life dissatisfaction and least happiness, while the retired seemed the most satisfied with their lives and happiest. Other studies also indicate that when assessing self-reported health, Black elderly psychological well-being is comparable or better than White Americans (Kahn & Fazio, 2005). Brown et al. (2006) also found that despite functional health and economic wealth deficits, older Black Americans had relatively high levels of life satisfaction and happiness. These findings are similar to those from past research on Black elderly mental health (e.g., Jackson, Chatters, & Neighbors, 1982; Jackson, Chatters, & Taylor, 1993). Jackson and Gibson (1985) argued that Blacks, being more socialized to adapt to uncertainty and change within their lives, arrive at old age more fortified, more rehearsed, and better able to adapt to its exigencies, despite fewer economic and social resources (Danigelis & McIntosh, 1993; Williams et al., 1997). The key is surviving to reach old age.

Work, Retirement, Health and Mortality. Several research studies indicate that Black Americans have higher mortality rates and poorer health than other groups (Adler & Rehkopf, 2007; Ferraro & Farmer, 1996; Williams & Jackson, 2005). In a longitudinal study designed to examine the double jeopardy to health hypothesis for older Black Americans, Ferarro and Farmer (1996) found that compared to their White counterparts, Blacks of all ages experienced poorer health. Black Americans had poorer health across a 15-year time period. The findings indicated that during the preretirement period, the health of Black adults' ages 50 to 64 years old was worse than White adults of the same age. In addition to poorer health, Black men ages 50 to 64, in particular had the highest mortality rates. A higher percentage among this older Black cohort were deceased than their White counterparts. This startling statistic indicated that fewer Black men actually live to *reach* traditional 65-year-old retirement age.

Thus, the investigators concluded that the double jeopardy hypothesis of health worsening due to being old and being Black was not supported. There is no double jeopardy because a substantial number of Black men have not lived to be old and Black. The same holds true for the triple jeopardy hypothesis of being old, being Black, and being female for older Black women. There

was no support for the triple jeopardy hypothesis of being old and Black and female. Similar to Brown et al. (2006) findings, Ferraro and Farmer (1996) found that Black women also experienced health inequities throughout adulthood. However, for the healthier Black men and women who survived to 65, there was no evidence of deterioration of health relative to their White counterparts. In fact, the health disparities and differences that did exist began in earlier periods. Mortality risks, however, declined among Black older adults once they reached traditional retirement ages of 65 and over. Ferraro and Farmer (1996) concluded that the health inequities found in many studies of older adults begin earlier in adulthood. Gibson (1991b) also explained that morbidity was worse for preretirement age Black Americans. She reported that Black Americans who reach 75 years old have a similar or better life expectancy as White Americans. Brown et al. (2006) also found that during preretirement ages of 50 to 64, older Black women were married but that during retirement ages 65 to 79, older Black women were more likely to be widowed. Compared to Black men, there were significantly more Black females in the 65- to 79-year-old age groups.

CONCLUSIONS AND IMPLICATIONS

Health inequalities begin at birth, decline during childhood, adolescence, and young adulthood then begin to deteriorate more quickly during middle age and beyond (Adler & Rehkopf, 2007; Williams & Jackson, 2005). These statistics suggest a need to focus on middle age as a period to disentangle the complexity of the links between work, retirement, and health disparities (Brown et al. 2006; Ferraro & Farmer, 1996). Researchers (Brand, Warren, Carayon, & Hoonakker, 2007; Burgard et al., 2007) conclude that understanding the health consequences of labor force exits is an important research endeavor that may help guide policy to assist our growing aging population. Lipscomb et al. (2006) and Miech et al. (2005) note that the relationship between health disparities and work involves government policies, historical segregation, and inequalities in the workplace, and their effects on workers' health. According to Williams and Jackson (2005), illness and disease limits productivity of adults during their most productive working years. The effect of which impacts the entire society. Nonproductivity among working age adults limits tax revenues and strains public and private resources. Excessive health problem among any and all groups is associated with increases in the use of social services and places a strain on the health care system. Therefore, it behooves us to reduce and eventually eliminate the observed life-course disparities in health and their social and economic correlates.

Recent studies examining the heterogeneity among Black older adults indicate that some conditions have improved, but for many others stagnation in disparities continues (Brown et al., 2006). The Brown et al. (2006) findings suggest that the educational attainment of Blacks has increased. This increase often is not noted in the literature when studies compare older Black American and White Americans. Research suggests that increased education can translate to better jobs with better benefits for subsequent cohorts of older Blacks during the preretirement years translating into better social, economic, and health outcomes in later life (Brown et al., 2006).

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CHAPTER 12

Cross National Disparities and Disabilities

Philippa Clarke and Jacqui Smith

An emerging body of work compares health across nations with a view to understanding how differences in macrosocial and economic contexts shape individual health and well-being (e.g., Adler, Marmot, McEwen, & Stewart, 1999; Lynch et al., 2004). Much of this research has been undertaken at a population level in the context of efforts to understand factors underlying the association between social inequality and health outcomes such as average life expectancy and the prevalence of preventative illness (e.g., cardiovascular disease, disability). Attention has been given to country differences in terms of the absolute levels of social inequality (especially the extent of poverty) and the magnitude of relative inequality (e.g., differential gap between rich and poor). There are also country differences linked to gross national product and the allocation of finances to health services. Beyond economic and political mechanisms, contemporary researchers also consider the roles of cultural and psychosocial factors at community and individual levels. Cultural differences in beliefs, health-related behaviors, and social cohesion have been highlighted as important factors involved in health disparities, both within and between countries, and as modifiers of the association between social inequality and health outcomes (e.g., Adler & Snibbe, 2003; Berkman, Glass, Brissette, & Seeman, 2000; Marmot, 2006; Marmot et al., 1998; Seeman, Crimmins, Weinstein, Hermalin, & Stoto, 2001).

In this chapter, we focus on an intriguing difference in beliefs about personal control found between the 50+ population in the United Kingdom

and the United States and the moderating role of control beliefs in the social inequality-health outcome association. To provide some background to this, we begin with brief reviews of cultural and life-course differences in health. We then review the work on cultural differences in the sense of control, and the moderating role of control for socioeconomic inequalities in health. We use national data from the United States and England to illustrate the socioeconomic gradient in health across the two countries, and show how it varies according to sense of control. We argue that cross-national differences in these relationships are partly a function of differences in the lifetime social construction of a sense of control (House, 1981; Marmot, 2006; Mirowsky & Ross, 2007). Our aim in this chapter is to highlight the role of culture in shaping health inequalities across adults aging in different sociopolitical contexts.

CULTURAL DIFFERENCES IN HEALTH: INSIGHTS FROM CROSS-NATIONAL RESEARCH

Country differences in population health outcomes, especially life expectancy, are well documented. Contemporary research in the field of aging also tracks trends in the compression of morbidity, especially functional (activity) limitations and disability (e.g., Crimmins & Saito, 2001; Melzer, McWilliams, Brayne, Johnson, & Bond, 2000). Jagger and colleagues (2008), for example, calculated life expectancies and healthy life years (HLY: i.e., remaining years spent free of activity limitations) at 50 years of age by sex for 25 European countries. They found that an average 50-year-old man in the 25 EU countries could expect to live until 67.3 years free of activity limitation, and a woman to 68.1 years. There was a 14.5-year gap between the country with the longest HLY for men at age 50 (23.6 years in Denmark) and the shortest (9.1 years in Estonia). This gap was similar for women who, on average, were estimated to have 1 to 1.5 HLY more than men after age 50. The HLY estimated for the U.K. population over 50 was 19.74 for men and 20.78 for women. Jagger and colleagues (2008) found that there were also substantial differences in the magnitude of inequalities within countries by level of education and income. Using data from the European Social Survey (2002–2004), Bambra and Eikemo (2009) found that across multiple welfare state regimes in Europe, a consistent relationship exists between unemployment and poor self-rated health.

Differences in socioeconomic inequalities in health across countries illustrate the role of macrosociopolitical contexts in shaping health at the individual level. Using 2004 data on adults age 50 and over from the U.S. Health and Retirement Study (HRS), the English Longitudinal Study of

Ageing (ELSA), and the Survey of Health Ageing and Retirement in Europe (SHARE), Avendano and colleagues (2009) found that U.S. adults report more chronic diseases than the English or other Europeans at every wealth level. These differences were greatest at the lowest levels of the socioeconomic hierarchy, but even Americans with higher incomes reported health problems at comparable levels to substantially poorer Europeans (Avendano, Glymour, Banks, & Mackenbach, 2009). Banks and colleagues (2006) also found that gradients in health by socioeconomic position (SEP) are more pronounced in the United States than in the United Kingdom, with the result that health disparities between the two countries are largest at the lower end of the SEP continuum (Banks, Marmot, Oldfield, & Smith, 2006).

Banks and colleagues (2006) used comparable 2002 survey data from the U.S. HRS and U.K. ELSA to examine the possible role of national differences in health behaviors in health disparities. They found that Americans report higher levels of disease than the British at similar ages, and that these differences were not explained by differences in health behaviors across the two populations. Comparable data from the Whitehall II Study (1997–1999) and the Western New York Health Study (1996–2001) suggest that Americans are more likely to be sleep deprived than the British (Stranges et al., 2008), but the relationship between short sleep duration and physical health is similar in England and the United States.

Cross-national differences in health, and especially socioeconomic inequalities in health, are particularly telling, and suggest that the way societies are structured (i.e., with respect to their social safety net and health care systems) can play an important role in shaping health inequalities in populations, particularly as they are exposed to such social structures cumulatively over the life course.

THE LIFE COURSE IN A CULTURAL CONTEXT

Exposure to different macrosociopolitical contexts over the life course is likely to result in notable differences in the health and psychological well-being of populations in later life. The United States and Britain are similar in many ways. Both countries are western, developed nations. Both are considered to be individualistic as opposed to collectivist nations, giving general priority to the individual over the group (Diener, 1996; Diener, Oishi, & Lucas, 2003; Hofstede, 2001; Triandis, 1995). Yet, in the spectrum of welfare state societies, the British social and health care policies are more comprehensive than the U.S. programs. Access to health care in the United Kingdom is universal,

while about 41 million Americans remain uninsured (Adams, Dey, & Vickerie, 2007). Whereas the United States has been described as an "ownership society," in which health care and social security programs fall more heavily under the responsibility of the individual (Robinson, 2005), England is considered a universal welfare state (Bambra & Eikemo, 2009). Comprehensive social protection programs (e.g., universal health care, comprehensive primary care and unemployment protection programs) may buffer the health of individuals over the life course, particularly those lower in the socioeconomic hierarchy (Hanson, 2006; Starfield & Shi, 2002). As a result, older adults across the two countries will have experienced structurally different life chances, which have consequences for health and health disparities in later life.

The benefits of social protection programs as they operate cumulatively over the life course (and conversely the negative consequences of fewer comprehensive protection programs for late life health) highlight the importance of social structural influences in the life course (O'Rand, 1996). The life course perspective, in all its various manifestations, places explicit emphasis on social and environmental contexts as they shape the course of individual lives and cohorts through historical time (Elder, 1994, 1998; Elder & Johnson, 2003; Ferraro & Shippee, 2009; Ferraro, Shippee & Schafer, 2009; Moen, 2001; O'Rand, 2006; Riley, 1979; Williams & Collins, 1995). Aging is therefore recognized as a dynamic process linking biographical time as it intersects with social and historical contexts. We would therefore expect to see variation in health and psychological indicators across older adults situated in different sociopolitical contexts. However, life-course scholarship (particularly in North America) has tended to emphasize the role of socioeconomic, race–ethnic, gender, and age structures at the relative neglect of other macrolevel social structures, such as the role of the state (Marshall & Mueller, 2003; Mayer, 1988, 1997, 2004; Townsend, 1981; Walker, 1981).

A political economy perspective on aging emphasizes that different cultures experience structurally different life chances that are linked to the moral economy of different nation states (Townsend, 1981; Walker, 1981). Social, political and economic structures within nations are shaped by cultural conceptions of legitimacy, equity or fairness, citizens' rights, and moral contracts between the generations (Walker, 1981, 2006) that shape the nature and extent of social protection programs including health care and unemployment insurance. The life course is socially constructed in light of such economic and moral concerns at the structural level of each society, which has implications for the accrual and development of health and psychological resources of individuals over time, as we argue in more detail below. Our purpose in this chapter is to deliberately focus on the role

of macrolevel social structures by comparing health across two different nations, in order to better understand the consequences of experiencing different cultures over the life course for later life health inequalities.

LIFE-COURSE PSYCHOLOGICAL RESOURCES AS MODERATORS OF HEALTH DISPARITIES

While cross-national differences in health are increasingly studied, a parallel body of research has also been examining national differences in psychological well-being (Diener & Suh, 2000; Inglehart, Foa, Peterson, & Welzel, 2008), including cultural differences in the psychology of control and sense of autonomy. Research has shown that personal control is more prevalent in North American than in Asian countries (Yamaguchi, Gelfand, Ohashi, & Zemba, 2005). Social structural differences in the role of the state may account for these differences if individuals in North America are socialized to rely more on individual compared to collective resources throughout their lives (Mayer, 1986; Townsend, 1981; Walker, 1981).

Personal control, or a perceived lack of external constraints imposed by powerful others or by the role of chance (also called fate control), is associated with better self-reported health in American adults, fewer health problems, and greater longevity (Lachman, 1986; Marmot et al., 1998; Rodin, Timko, & Harris, 1985). Sometimes referred to as the illusion of control (Taylor & Brown, 1988), a high sense of personal control is also considered to be an adaptive resource for Americans in the face of adversity, particularly among those with fewer resources to draw on. If individuals believe they have some degree of control over their lives, they may be more likely to take action in difficult situations regardless of their underlying probability of success (Lachman & Weaver, 1998).

The adaptive potential of perceived control prompted Lachman and Weaver (1998) to examine whether control beliefs moderate the relationship between SEP and health. Using 1995 data from the Midlife in the United States Study (MIDUS), they found that socioeconomic differences in functional limitations were attenuated by a higher sense of control. Control beliefs operated as a buffer on the negative association between social class and health. Mirowski and Ross (2007) have shown that those with lower social status generally report a lower sense of perceived control over their lives but that there are individual differences within all status levels (i.e., some low status individuals do have a high sense of control over their lives). Yet, these analyses have not been replicated across countries, even though documented cultural differences in the sense of control could conceivably have different effects on the socioeconomic gradient in health across nations.

Our purpose in this chapter is to examine differences in the buffering effects of control on socioeconomic inequalities in health in England and the United States. We focus on disability as a socially and psychologically constructed health outcome where the consequences of disease for independent functioning may vary depending on psychological adaptive strategies (World Health Organization, 2001). As such, we expect socioeconomic disparities in disability to vary across national contexts since cultural differences in perceptions of personal control (or differential importance placed on control), are likely to differentially affect the socioeconomic gradient in disability. We extend the work of Lachman and Weaver (1998) by revisiting these associations with a different U.S. data set (one that represents older adults) and also extend this work to another national setting to see how these relationships are replicated in societies that may place less emphasis on the importance of individual control.

HEALTH AND CONTROL ACROSS OLDER ADULTS IN THE UNITED STATES AND ENGLAND

We draw on cross-sectional data from two nationally representative surveys of older adults, one from the United States (the Health and Retirement Study [HRS]) and one from England (the English Longitudinal Study of Ageing [ELSA]). The HRS is an ongoing, nationally representative, longitudinal survey of more than 20,000 community-dwelling Americans age 51 and over conducted biennially since 1992. African Americans, Hispanics, and households in the state of Florida were oversampled and reweighted for analyses as described below. Refresher cohorts have been added to the original sample to maintain national representation of this age group over time. The HRS asks detailed questions about work activity, retirement expectations, and finances, but to date, has collected limited data on psychosocial aspects of well-being. We therefore focus our analyses on a random one-half sample of 7,455 respondents who completed an additional self-administered questionnaire on psychological and social well-being that was left behind following the face-to-face interview in 2006 (Health and Retirement Study, 2006). The response rate among those who were invited to complete the questionnaire was 91%.

For comparison, we use data from the 2006 wave of the English Longitudinal Study of Ageing (ELSA-3), which is a nationally representative survey of approximately 12,000 English adults age 50 and over, conducted biennially since 2002 (Banks, Breeze, Lessof & Nazroo, 2008). A refresher sample was added in wave 3 (2006) in order to maintain representation of the population age 50 and over. Similar to the HRS, data on psychosocial well-being were collected using a self-completed questionnaire that was left behind after the

main interview, with a response rate of 87% (Banks, Breeze, Lessof, & Nazroo, 2008). In order to be comparable with the American data, we restrict our analyses to the 7,601 community-dwelling adults who were age 51 and over in 2006.

The two surveys were designed to be comparable in their measurement. *Disability* was assessed in both surveys with a measure of difficulty performing eight daily life activities (shopping, meal preparation, taking medications, walking 100 yards, using the telephone, managing finances, using a map, and doing housework). A dichotomous indicator of disability was created by contrasting those who reported any difficulty with those who reported no difficulty with any of the eight activities. *Socioeconomic position* (SEP) was assessed with a measure of subjective social status that "reflects the cognitive averaging of standard markers of socioeconomic situation" (Singh-Manoux, Adler, & Marmot, 2003; Demakakos, Nazroo, Breeze, & Marmot, 2008). Using a graphical representation of a ladder, respondents were instructed to rank themselves on the ladder in terms of money, education, and occupation relative to other people in society (Cantril, 1965; MacArthur Scale of Subjective Social Status, 1999). An indicator of lower SEP was created by contrasting those in the lower three quintiles with those in the upper two quintiles of the SEP ladder.

Control was assessed in the self-completion module of both surveys with a single item that had only a slight variation in wording. In HRS the item reads: "What happens in my life is often beyond my control" and in ELSA the item reads: "I feel that what happens in life is often determined by factors beyond my control." In both surveys respondents were asked the degree to which they agreed or disagreed with the statements according to a 6-point scale. A high score indicates a high sense of personal control. A dummy indicator for a low sense of control was created by contrasting those who gave an affirmative response (any indication of agreement) with those who gave a negative response (any indication of disagreement) to the question.

While our primary purpose is to illustrate the relationship between SEP and disability, as well as the moderating effect of control, our models also account for five background variables that could confound the relationships under study, including age, gender, race, marital status, and employment status. Logistic regression was used to examine the relationship between socioeconomic position and the log odds of reporting any disability, controlling for background characteristics. Separate models were conducted for HRS and ELSA and weighted by the appropriate sample weight for each study to account for the sampling design and nonresponse in each survey.

Table 12.1 presents descriptive statistics for both study samples. In general, the two samples are similar except that participants in the ELSA study

TABLE 12.1
Descriptive Statistics: Means (± Standard Deviation) and Percentages for Study Variables

	HRS (*N* = 7,455)	ELSA (*N* = 7,601)
Age		
51–65	41.4%	55.2%
66–80	45.4%	36.0%
80+	13.2%	8.8%
Female	58.4%	54.8%
Race/ethnicity		
White	83.0%	98.2%
Nonwhite	17.0%	1.8%
Marital status:		
Married	64.5%	68.6%
Separated/divorced	12.5%	10.4%
Widowed	20.2%	15.7%
Never married	2.8%	5.3%
Employment status:		
Employed	31.4%	35.0%
Unemployed	1.4%	0.7%
Retired	48.3%	51.3%
Disabled	7.2%	4.6%
Homemaker	11.7%	8.4%
SEP ladder:		
Lower quintile	2.5%	2.7%
Q2	9.7%	15.8%
Q3	31.2%	42.5%
Q4	46.6%	34.4%
Upper quintile	10.0%	4.6%
IADL difficulty:		
Shopping	6.0	7.4
Meal preparation	3.7	3.2
Money management	3.2	1.9
Taking medications	2.7	1.0

TABLE 12.1
Descriptive Statistics: Means (± Standard Deviation) and Percentages for Study Variables (Continued)

	HRS (*N* = 7,455)	ELSA (*N* = 7,601)
Mobility	30.0	10.7
Housework	22.3	13.5
Reading a map	10.3	3.8
Using the telephone	2.6	1.3
Any IADL disability	42.6%	20.8%
Sense of control		
High control	72.1%	21.9%
Low control	27.9%	78.1%
Mean control score	4.5 (±1.6)	2.7 (±1.3)

Note. HRS = Health and Retirement Study 2006; ELSA = English Longitudinal Study of Ageing 2006; SEP = socioeconomic position; IADL = instrumental activities of daily living.
[a]Health and Retirement Study 2006 (*N* = 7,455). [b]English Longitudinal Study of Ageing 2006 (*N* = 7,601)

tend to be younger than those in HRS. In addition, more Americans position themselves higher in the SEP strata than the British. In part, due to the higher proportion of older adults in the U.S. survey, the prevalence of disability is higher among the Americans, particularly with respect to activities related to mobility. On average, older adults in the United States report a higher sense of control than those in the United Kingdom.

In both countries there is a socioeconomic gradient in disability such that those in the lower socioeconomic strata report more difficulty with activities related to independent living while those higher in the socioeconomic distribution are less likely to report difficulty (Figure 12.1). However, across all socioeconomic strata, the British are more likely to be free from disability than the Americans.

In spite of the general similarities in health and sociodemographic characteristics, older adults in the two countries differ markedly in their sense of control (Figure 12.2A). Older Americans overwhelmingly report a high sense of personal control. But in Britain, older adults are more likely to say that some things in life are beyond their control. In both countries there is a socioeconomic gradient in control, where those at the lower end of the socioeconomic ladder report less control while those at the upper end of the ladder report a greater sense of perceived control (Figure 12.2B). However,

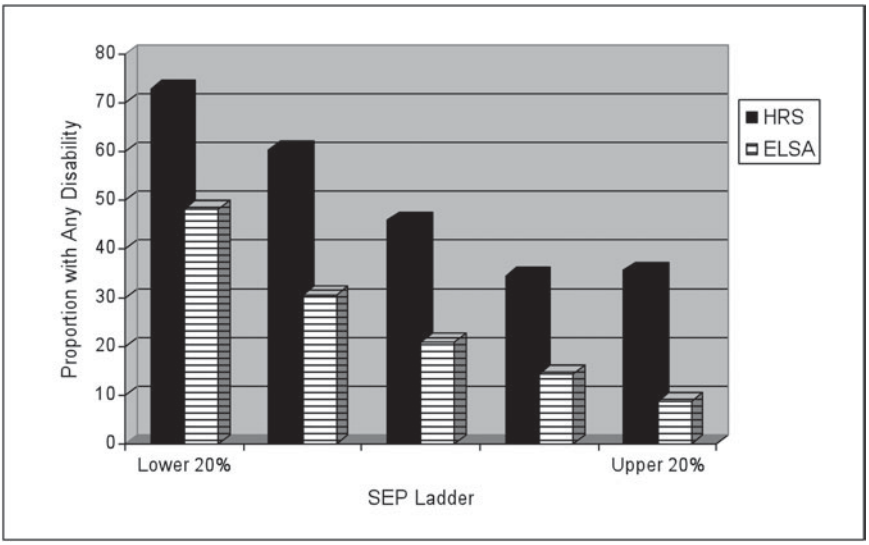


FIGURE 12.1 Socioeconomic gradient in disability across HRS and ELSA.

the gradient in control is less steep in the British compared to the American sample. The difference in mean control score between those in the upper and lower SEP strata is 1.45 in HRS compared to .79 in ELSA. As a consequence, cross-national differences in the sense of control are much greater among those in a higher socioeconomic position than they are among those in the lower socioeconomic strata.

Table 12.2 presents the results from the binary logistic regression analyses for any disability (no disability is the reference group). Separate models are presented for the HRS and ELSA samples. Model A presents the logistic regression coefficients and odds ratios for the independent variables as they relate to any disability. In both countries the odds of disability are higher among women, racial–ethnic minorities, persons who are separated–divorced or widowed, those who are not employed, and those at older ages. Those who never married show no differences in disability compared to the married respondents (both ELSA and HRS). In the British sample, the odds of disability are much higher in the unemployed compared to the employed, but in the U.S. sample there are no differences in disability between the employed and unemployed. Compared to married adults, the odds of disability among those who are separated or divorced are considerably stronger in the ELSA sample than in HRS. Similarly, the odds of reporting disability among those who are retired or homemakers (in comparison to those who are employed) are higher in the United Kingdom than in the United States.

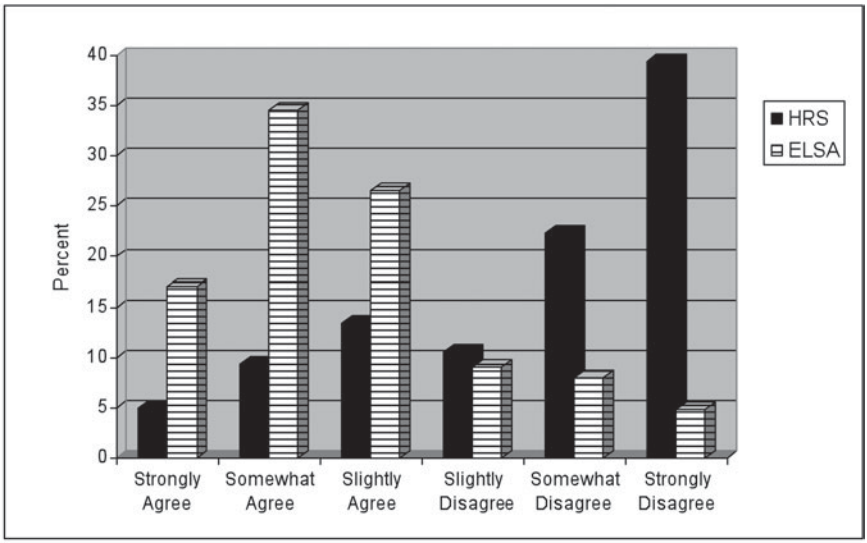


FIGURE 12.2A Control beliefs in HRS and ELSA: "What happens in life is often beyond my control."

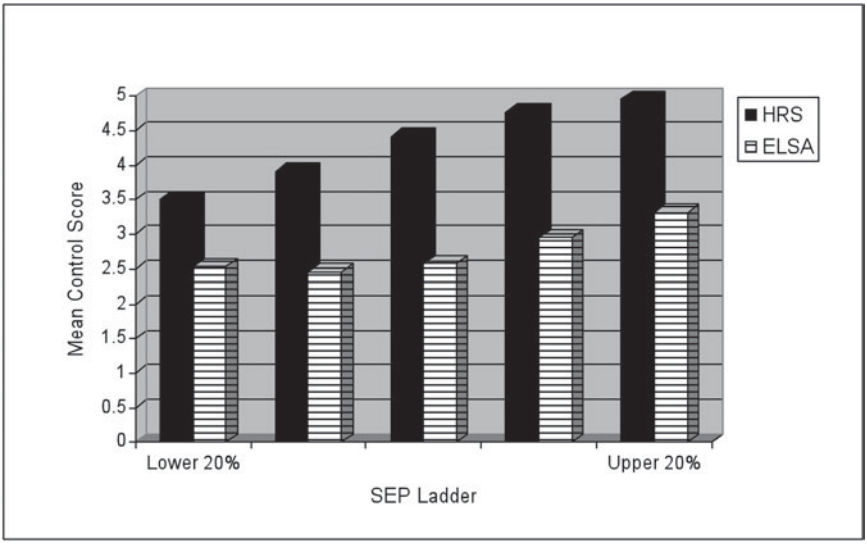


FIGURE 12.2B Mean control score in HRS and ELSA by socioeconomic position (SEP).

TABLE 12.2
Binary Logistic Regression Models for Any Disability: HRS (2006) and ELSA (2006)

	HRS				ELSA			
	Model A		Model B		Model A		Model B	
	Estimate	Odds Ratio	Estimate	Odds Ratio	Estimate	Odds Ratio	Estimate	Odds Ratio
Intercept	–1.919*** (.073)		–2.005*** (.075)		–3.352*** (.104)		–3.603*** (.150)	
Age 66–80[a]	.398*** (.069)	1.489	.394*** (.070)	1.483	.350*** (.087)	1.419	.342*** (.088)	1.407
Age 80+[a]	1.242*** (.102)	3.461	1.186*** (.104)	3.275	1.324*** (.118)	3.757	1.327*** (.120)	3.772
Female[b]	.303*** (.060)	1.354	.312*** (.061)	1.367	.300*** (.070)	1.350	.285*** (.071)	1.329
Nonwhite[c]	.240** (.087)	1.271	.267*** (.079)	1.306	.622** (.216)	1.862	.527* (.224)	1.694
Separated/divorced[d]	.191* (.088)	1.211	.184* (.088)	1.202	.470*** (.106)	1.600	.482*** (.107)	1.619
Widowed[d]	.326*** (.076)	1.385	.302*** (.077)	1.353	.306*** (.088)	1.359	.304*** (.088)	1.355
Never married[d]	.204 (.173)	1.226	.191 (.175)	1.210	.188 (.144)	1.207	.207 (.145)	1.230
Unemployed[e]	.383 (.232)	1.467	.275 (.238)	1.317	1.514*** (.342)	4.545	1.454*** (.354)	4.282

Retired[e]	.756*** (.074)	2.129	.744*** (.075)	2.103	1.242*** (.112)	3.462	1.227*** (.112)	3.412
Homemaker[e]	.889*** (.098)	2.434	.848*** (.100)	2.335	1.234*** (.140)	3.437	1.200*** (.141)	3.321
Disabled[e]	3.128*** (.156)	22.831	3.049*** (.158)	21.101	4.078*** (.163)	59.009	4.041*** (.163)	56.874
Lower SEP[f]	.532*** (.055)	1.703	.397*** (.067)	1.487	.389*** (.070)	1.476	.593*** (.160)	1.809
Low control[g]			.463*** (.087)	1.589			.352* (.142)	1.422
SEP* control			.241* (.123)	1.273			–.251 (.178)	.778

Note. Standard errors are in parentheses under the parameter estimates.
[a]Reference group is age 51–65. [b]Reference group is male. [c]Reference group is White. [d]Reference group is married. [e]Reference group is employed. [g]Reference group is high control. [f]Lower SEP refers to those in the lower three quintiles of the SEP ladder (lower 60%); [g]Reference group is upper SEP (upper 2 quintiles of the SEP ladder).
*$p < .05$. **$p < .01$. ***$p < .001$.

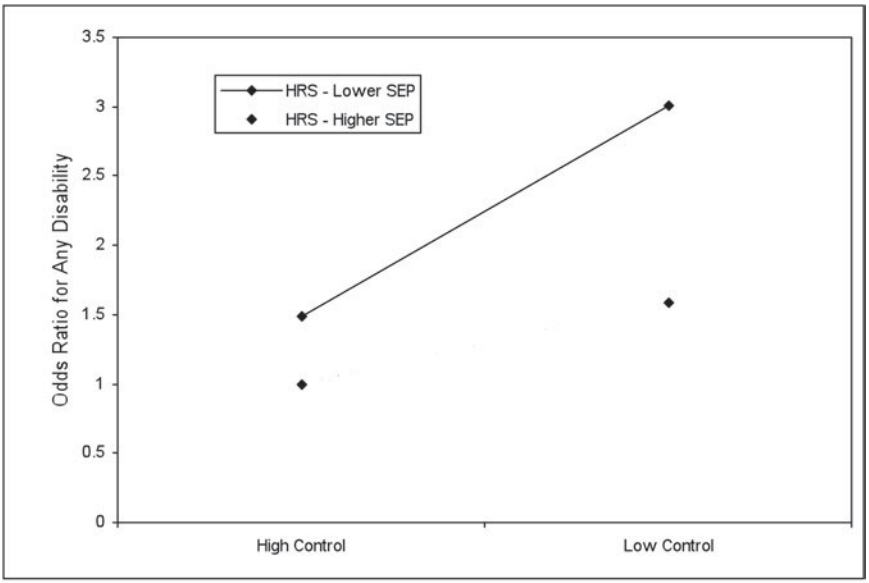
In both countries a lower socioeconomic position is associated with increased odds of disability, but the effect is somewhat stronger in the United States than in England (although not statistically different in a combined analysis with a concatenated dataset). Compared to those in a higher socioeconomic position, the log odds of reporting any disability among lower SEP individuals in the American sample increases by .532, for an adjusted odds ratio of 1.7 (95% confidence interval: 1.53, 1.89). In contrast, the log odds of reporting any disability in the British sample increases by .389 for those in a lower SEP, for an adjusted odds ratio of 1.5 (95% confidence interval: 1.29, 1.69).

Model B in Table 12.2 adds the indicator of low control as well as the interaction term to assess how socioeconomic inequalities in disability vary according to the sense of control. In the British sample the interaction term is not statistically significant. But in the American sample, the effect of socioeconomic position varies according to the sense of control. The predicted odds ratios for both the HRS and ELSA samples are illustrated in Figure 12.3.

In both countries the odds of disability are highest among those in a lower socioeconomic position and in those with a lower sense of control. However, there are notable differences in the results across HRS and ELSA. In the British sample socioeconomic inequalities in disability are no different in those with a high sense of control compared to those with a low sense of control. However, in the American sample socioeconomic inequalities in disability are much greater among those with a lower sense of control. With all other factors held constant, the socioeconomic difference in the log odds ratios for any disability is .241 greater (the regression coefficient for the interaction) among individuals with a low sense of control. Exponentiating this coefficient yields an odds ratio of 1.273, indicating that the socioeconomic inequality in the odds of disability is almost 30% greater among Americans with a lower compared to a higher sense of control.

SUMMARY: SOCIOECONOMIC DISPARITIES IN DISABILITY AND THE MODIFYING EFFECT OF CONTROL

In this chapter we have drawn on the life-course perspective to argue that different social and economic structures across nations translate into different psychological resources as they develop over the course of a lifetime, which have consequences for socioeconomic inequalities in health in later life. Research has repeatedly demonstrated that older Americans are much less healthy than their British counterparts and that gradients in health by socioeconomic position are more pronounced in the United States than in the United Kingdom (Avendano et al., 2009; Banks et al., 2006; Marmot,



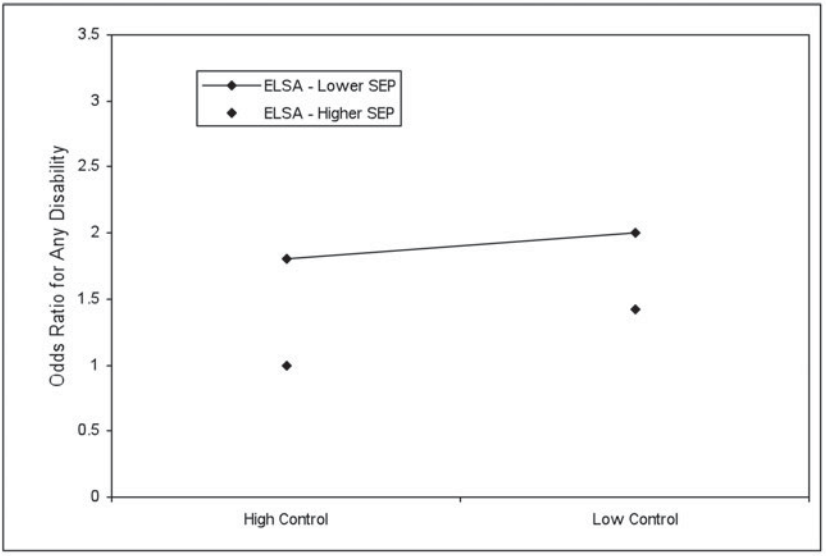


FIGURE 12. 3 Socioeconomic inequalities in disability by the sense of control: HRS versus ELSA.

2006). Our results suggest that over the age of 50, Americans have a stronger sense of personal control than the British, which operates as a psychological resource to reduce socioeconomic inequalities in health. Although on average the British have fewer disabilities than the Americans, the sense of control operates as a leveler, such that the odds of disability are more similar among Americans and British with a higher sense of control.

In both populations the risk of disability is highest among those in a lower socioeconomic position and among those with a lower sense of control. However, cultural differences surrounding the sense of control, as it likely develops and is entrenched in a particular society over the life course, result in cross-national differences in socioeconomic inequalities in disability by the level of control. Similar to the results reported by Lachman and Weaver (1998) we found that in the United States socioeconomic inequalities in disability are attenuated among those with a high sense of control, with the result that socioeconomic inequalities in health are greatest among those with a low sense of control. On the other hand, we found no significant differences in health inequalities among the British according to the sense of control.

The fact that a higher sense of perceived control in the United States reduces socioeconomic inequalities in disability suggests that in the United States, control is a psychological resource that can be used to manage the constraints posed by declines in health and function, particularly among those who are aging with fewer social and economic resources. As argued by Ferraro and colleagues (2009) psychological, as well as social and economic resources, can retard or accelerate disability trajectories in older adults. Although those in lower socioeconomic positions are more likely to believe in the role of fate or powerful others (Gurin & Brim, 1984; Gurin & Gurin, 1970; Lachman, 1985; Levenson, 1981; Mirowsky & Ross, 2007), the sense of control in the U.S. culture is so strong that even among those in the lowest socioeconomic stratum, Americans report a greater sense of control than the British. This psychological resource is therefore operational even at the lowest end of the socioeconomic spectrum, and operates to modify the socioeconomic gradient in disability in the United States.

Our results provide some support and evidence for the role of individual agency within the constraints of macrosocial structures (Marshall, 2005; Walker, 2006). This approach postulates interactionist and potentially dialectical mechanisms whereby the individual's lived experience is meaningfully constrained by social structure, but where the impact of that social structure on human lives can be modified through individual action and social interaction (in this case psychological resources). Glen Elder considers agency to be one of five defining principles of the life course: "Individuals construct their

own life course through the choices and actions they take within the opportunities and constraints of history and social circumstances" (Elder & Johnson, 2003, p. 60). Situated within a comparative context, our findings suggest that older Americans find ways to modify the socioeconomic structuring of disability through the engagement of psychological resources.

Because disability is measured by the ability to perform an activity without difficulty, it has fluidity depending on the social, psychological, and environmental resources that can be accessed to manage disability in a social context (World Health Organization, 2001). Individuals may draw on a variety of different strategies (e.g., substitute activities, change the way they do an activity, use alternate social and physical resources—including assistive technology) to maximize their ability to perform an activity without difficulty. Similar to the concepts of selective optimization with compensation (Baltes & Baltes, 1990) the ability to engage these alternate strategies may well depend on one's sense of control over one's life. The greater sense of perceived control among American adults may operate as a resource to initiate some of these adaptive strategies in the face of declining health and function.

Structural differences in the macrosocioeconomic organization of the American compared to the British society may readily translate into differences in perceptions of, as well as the importance of, control across populations in these two national contexts. Since the culture in the United States tends to emphasize and promote control over one's individual actions and choices, control inevitably becomes a resource that can be used to manage declining health and function and can be particularly salient among those who have fewer social and economic resources upon which to draw. In contrast, the cultural propensity in the United Kingdom to accept that some things in life are beyond your control renders this a more neutral resource, with the result that social inequalities in disability do not vary according to the sense of control.

These findings highlight the role of culture in shaping health inequalities across adults aging in different sociopolitical contexts. Variations in cultural, institutional, and social configurations across nations have the potential to have large impacts on differences in individual health and well-being (Mayer, 1986, 2004). The contribution of cross-national research for understanding and suggesting the causal pathways behind socioeconomic disparities in health holds great promise for future research endeavors as the number of comparable data sets expands across the developed and developing world. There is surprisingly little research on cultural differences in beliefs and values and how these are dynamically linked to health disparities over time. The advent of harmonized cross-national studies of aging, however, may foster future endeavors.

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SECTION VI

APPROACHES TO PUBLIC POLICIES TO ADDRESS HEALTH DISPARITIES IN LATE LIFE

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CHAPTER 13

Health Care Reform and Health Disparities

Reasons for Hope?

Toni P. Miles

Public health data consistently shows populations within the larger American demographic that experience higher than expected rates of disease and death (Murray et al., 2006). These groups include racial and ethnic minorities, persons with less than a high school education, persons clustered in specific geographic regions, and members of households that are supported by low wage jobs. There are a number of factors that contribute to these rates. *Barriers in access to health care* make a substantial contribution to this excess death and disability. Ever increasing numbers of members of the U.S. middle class are experiencing this specific barrier. Indeed, this collective experience is a major political force driving the legislative efforts toward health care reform now occurring in the 111th Congress (2009–2010).

A central theme of this chapter is that health disparities researchers need to shift the frame from *individual experience* of disease to a frame that includes both the *individual and the system of health care*. When the *individual* occupies the entire frame, the main product of health disparities studies can be classified in three ways. There are studies that document the size of the gap between groups. These studies serve a witness function. At the end of the study, there is usually a statement like "Blacks experience greater rates than other groups and more research is needed to identify causal factors." Other studies seek to define causal factors and can be thought of as etiology studies. The summary

statement for this research can be paraphrased as follows "Individual genetic effects may account for higher rates of diabetes." A third variety of studies focuses on a specific factor and attempts to measure its effectiveness on an individual. These studies typically end with a statement like "Our approach improves individual adherence to the treatment among Hispanics." A frame that includes both the *individual and the delivery system* could serve all the functions described—witness, etiology, and treatment effectiveness assessment. The design, however, could estimate the impact of systemic factors. Summary statements for the extended frame would include statements estimating the size of the gap attributable to systemic organization, the systemic etiology of individual differences in outcome, and variation in treatment effectiveness attributable to systemic differences.

To illustrate a starting place for an expanded frame of reference, consider the problem of health disparities in the Medicare population. Current policy research is focused exclusively on Medicare spending and system financing. Is there evidence of health care inequalities in a population that, theoretically, is insured and, therefore, has access to health care? Can the Medicare population provide a starting place to examine the connection between health disparities and the reformation of health care to improve access, lower cost, and improve quality? Medicare is a federal health insurance program for 45 million elderly and disabled Americans. What role does Medicare play in either creating or attenuating health disparities? Medicare helps to pay for hospital and physician visits, prescription drugs, and other health care services. According to the Kaiser Family Foundation, Medicare paid for half of beneficiaries medical expenses in 2005 (www.kff.org, Fact Sheet, May 2009). Dual eligible beneficiaries—persons simultaneously enrolled in Medicare and Medicaid—form a cohort of the nation's most vulnerable older adults. This population is defined by its low income. More than 61% live on less than $10,000 annually (Coughlin, Waidmann, O'Malley-Watts, 2009). Although some members of this cohort have engaged in a spend down process, it is likely that most dual eligible beneficiaries have lived with lifelong socioeconomic disadvantage. What would the health disparities research frame look like for older adults who are dual eligible?

Enter Medicare into a search engine and more than 90% of the research reports retrieved will focus on one of two areas: costs of services or system financing. The frames of reference are factors that drive health care consumption and pricing. Reports that are concerned with financing focus on the stability of the Medicare Trust Fund (Kaiser Family Foundation, Fact Sheet, May 2009). There is no mention of health disparities or economically disadvantaged elders. The Bureau of Economic Analyses created a Web site to

showcase its research to develop new indices for pricing health care (see http://www.bea.gov/national/health_care_satellite_account.htm). Cost control, not population-level access, is the main focus of this site and a recent report released in February 2009 (a new approach to price measures for health care). A discussion of health disparities does not appear in reports that focus on the profitability of a hospital or other health care provider. See the report that U.S. hospitals experienced zero profit margins during the first quarter of 2009 (U.S. Hospital Profits, 2009). There are a few studies that have examined health disparities using the Medicare population as a sample population (Skinner & Zhou, 2006). They propose several strategies for measuring trends in health inequality—expenditures and life expectancy to name a few. The analyses provide compelling evidence that when Medicare beneficiaries are stratified by income, there are striking disparities in expenditures. These differentials in expenditures are significantly correlated with gaps in life expectancy. In other words, low income Medicare beneficiaries have lower per capita expenditures and lower life expectancies.

Regional variation in both disease and Medicare expenditures is another potential starting point for health disparities research. The Dartmouth Atlas Project (DAP) is a claims database. It comes from the Centers for Medicare and Medicaid Services (CMS), the federal agency that collects data for every person and provider using Medicare health insurance. Access to these data is made available for research purposes. (See http://www.dartmouthatlas.org/faq/data.shtm for a full discussion of the sampling frame and other details.) Other data sources used to develop this resource include U.S. Census, the American Hospital Association, American Medical Association, National Center for Health Statistics, and Claritas Incorporated. What does the Dartmouth data tell us? Reports using these data show regional variation in Medicare payments (http://www.dartmouthatlas.org/faq/geogappdx.pdf). Table 13.1 was developed to illustrate the paradoxical relationship between Medicare expenditures and quality care measures. In the table, Hospital Referral Regions (HRR) were ranked from 1 through 306 based on a standardized per capita Medicare expenditure. The final rank is further determined by the expenditures *after* the data were adjusted for population differences in age, gender composition, and racial and ethnic differences. This adjustment removes biases associated with advanced age, gender differences in patterns of illness, and severity of illness within racial and ethnic groups.

One unanswered question in health disparities research involves the influence of Medicare policies and their influence on regional variation in costs. Some areas have persistent disparities in health, more disease, and higher death rates. Medicare policies recognize the added costs associated

TABLE 13.1
Quality Indicators Comparison: Top and Bottom 10 Regions, Before Adjusting Expenditures for Medicare Policies

Hospital Referral Region	Rank	Discharge for Ambulatory Care Sensitive Conditions (Per 1,000 Enrollees)	% Diabetic Enrollees With Eye Exam (Age 65–75)	% Diabetic Enrollee With Hemoglobin A1c Test
United States		**77.3**	**67.8**	**79.8**
Top Ten				
Miami, FL	1	94.4	**68.6**	76.9
McAllen, TX	2	103.1	66.6	74.0
Bronx, NY	3	101.9	**71.9**	68.7
Manhattan, NY	4	136.1	62.9	75.1
Harlingen, TX	5	133.7	64.7	75.9
Los Angeles, CA	6	98.0	65.9	70.6
E. Long Island, NY	7	105.3	65.9	78.7
Dearborn, MI	8	104.4	**64.5**	**80.7**
Newark, NJ	9	130.2	**65.6**	70.7
Chicago, IL	10	**59.8**	**74.7**	66.7
Bottom Ten				
Sioux Falls, SD	297	**75.0**	**72.1**	**84.6**
Salem ,OR	298	**41.8**	**69.4**	**82.5**
Iowa City, IA	299	**66.4**	**69.4**	**85.9**
Grand Junction, CO	300	**46.9**	**68.2**	**83.0**
Appleton, WI	301	**49.9**	**75.6**	**88.4**
Minot, ND	302	**72.6**	**80.4**	**83.3**
Rapid City, SD	303	**61.0**	**71.4**	79.2
Dubuque, IA	304	**64.6**	**81.3**	**91.1**
La Crosse, WI	305	**58.1**	**74.4**	**88.9**
Honolulu, HI	306	**31.2**	**69.3**	**82.7**

Note. Discharge for ambulatory care sensitive conditions: These are hospital discharge rates for illnesses that have been proven to decline when appropriate outpatient care is provided. Goal = Less than U.S. rate; Percentage of diabetic enrollees with eye exam: Eye examines are a USTFPS recommended = *A screening test* to prevent blindness. All persons with diabetes should have a yearly examination. Goal = 100% of all patients;
Percentage of diabetic enrollees with hemoglobin A1c tests: A USTFPS recommended = *A screening test* to monitor long-term control of blood glucose levels. Goal = 100% of all patients; Rank represents adjusted Medicare expenditure compared to all 306 HRR. Hospital Referral Region (HRR) represent health care markets for tertiary care. Each HRR is a grouping that contains at least one hospital that performs major cardiac procedures and neurosurgery. For more details describing the definition of HRR see http://www.dartmouthatlas.org/faq/geogappdx.pdf

with health disparities. A portion of enrollee Medicare expenditures reflect the costs of caring for a higher number of patients who are older and sicker by recognizing Diagnostic Related Group associated payments. Another portion of the cost is determined by policies recognizing that many hospitals provide uncompensated care. The hospitals that care for large numbers of minority elders also serve large numbers of persons without health insurance: the Medicare Disproportionate Share Hospitals (DSH). Since 1981, federal law requires state Medicaid programs to take into account the circumstances of hospitals serving a disproportionate number of low-income patients when setting payment rates for inpatient hospital services. Over the past two decades this requirement has led to the emergence of the Medicaid disproportionate share hospital (DSH) programs in many states. In fiscal year 2005, the federal government paid $8.5 billion in Federal Medicaid DSH payments, which supplement the regular reimbursements hospitals receive for treating Medicaid beneficiaries on an inpatient basis. Some areas have more medical schools than others. Medicare is a major supporter of education. Graduate medical education (GME) is based on a formula that includes salary for physicians engaged in postgraduate training plus costs to accommodate the inefficiencies associated with training in a clinical setting. Inefficiencies include longer hospital stays, increased medical testing, and lower patient volume per physician. There is regional variation in the distribution of GME dollars because almost 50% of all medical residency training slots are located in the northeastern region where many of the top 10 regions in Table 13.1 are located.

Do all the additional dollars contribute to higher quality of care? The DAP Web site also provides data for quality measures commonly used in analyses of policy. The measures shown in Table 13.1 are: discharge for ambulatory care sensitive conditions (per 1,000 enrollees), percentage of diabetic enrollees with eye exam (age 65–75), and percentage of diabetic enrollee with hemoglobin A1c test. *Ambulatory care sensitive conditions* are a technical term describing preventable hospitalizations. The underlying idea is that hospitalizations for conditions such as diabetes and asthma could be reduced if they were properly managed in the Primary Care setting. The other two quality measures illustrate the types of tests that should be routinely done in Primary Care. Blindness is a common complication of uncontrolled diabetes and regular eye examinations is the recommended approach toward prevention. The Hemoglobin A1c test is recommended for monitoring blood sugar levels and provides insight to the physician about stability over a 3-month window. Each of these tests is part of quality care for patients with diabetes. In the table, the top 10 costliest HRRs are less likely to provide quality care when compared to the least costly HRRs. In other words, after

controlling for severity of illness and other patient factors, quality care is not necessarily costly care.

Does Medicare policy contribute to health disparities? Table 13.2 shows that regional variation in expenditures persists once we have removed the costs associated with the severely ill, the uninsured, and the need to support training. It also adjusts for age, sex, and race. There is still a twofold gradient in Medicare expenditures across the United States. The regional variation persists but new areas emerge. The differences in the quality of medical care also persist. The regions with the lowest costs, paradoxically, continue to rate the highest on quality measures. Patients in the lowest cost areas are less likely to be hospitalized for a chronic condition. Persons with diabetes in these areas are more likely to get preventative care. Regular eye exams prevent blindness. Regular HA1c checks to determine long-term control of blood sugars. Control of blood sugar is a key strategy to preventing the complications of diabetes (heart attack, stroke, blindness, amputation, and premature disability). The Dartmouth Atlas data tells us that policies promoting quality care will lower costs for all regions. The Atlas, however, does not account for factors that contribute to underlying poor health in the population.

Health disparities are both social and political issues as well as public health concerns. Health care reform legislation creates a unique opportunity for social scientists to explicitly access changes in the statistical measures of health disparities. The challenge is linking legislative efforts to correct the system with changes in metrics of health. How can we identify beneficial effects of newly enacted reform measures as well as identify unintended consequences? What kinds of study designs will generate answers that have a direct application to political and public health officials as well as driving social science research? The health care reform efforts present both an opportunity to improve and a risk of exacerbating existing health disparities in the United States. Are there cases from our history that can help rapidly identify unintended consequences? Can prior legislative efforts specifically designed to improve health care provide guidance to researchers and public health officials? By extending the analyses of Medicare expenditures beyond the usual focus on cost containment, health disparities researchers can develop a nuanced view of the causes of and cures for health disparities in late life.

HEALTH DISPARITIES, LEGISLATION, AND PUBLIC HEALTH RESEARCH

The primary product of the legislative process is a new law. How would a social scientist connect a newly enacted law to indices of mortality and morbidity?

TABLE 13.2
Quality Indicators Comparison: Top and Bottom 10 regions, After Adjusting Expenditures for Medicare Policies.

Hospital Referral Region	Rank	Discharge for Ambulatory Care Sensitive Conditions (Per 1,000 Enrollees)	% Diabetic Enrollees With Eye Exam (Age 65–75)	% Diabetic Enrollees Tested for Long-Term Control (Age 65–75) (HA1c)
United States		**77.3**	**67.8**	**79.8**
Miami, FL	1	94.4	**68.6**	76.9
McAllen, TX	2	103.1	66.6	74.0
Harlingen, TX	3	133.7	64.7	75.9
Monroe, LA	4	136.1	62.9	69.1
Alexandria, LA	5	133.7	64.7	74.3
Elyria, OH	6	98.0	65.9	77.7
Corpus Christi, TX	7	105.3	65.9	**80.3**
Shreveport, LA	8	104.4	64.5	74.8
Slidell, LA	9	130.2	65.6	73.7
Fort Lauderdale, FL	10	**59.8**	**74.7**	**82.9**
Anchorage, AK	297	**63.1**	54.4	67.3
Rapid City, SD	298	**61.0**	**71.4**	79.2
Medford, OR	299	**54.7**	**70.1**	**84.5**
Yakima, WA	300	**55.3**	**70.1**	**80.7**
Lebanon, NH	301	**58.3**	**76.0**	**85.2**
Grand Junction, CO	302	**46.9**	**68.2**	**83.0**
Eugene, OR	303	**53.4**	**70.9**	**82.3**
Salem, OR	304	**41.8**	**69.4**	**82.5**
La Crosse, WI	305	**58.1**	**74.4**	**88.9**
Honolulu, HI	306	**31.2**	**69.3**	**82.7**

Note. Discharge for ambulatory care sensitive conditions: These are hospital discharge rates for illnesses that have been proven to decline when appropriate outpatient care is provided. Goal = Less than U.S. rate;
Annual percentage of diabetic enrollees with eye exam: Eye examines are a USTFPS recommended = *A screening test* to prevent blindness. All persons with diabetes should have a yearly examination. Goal = 100% of all eligible patients; Percentage of diabetic enrollees with hemoglobin A1c tests: A USTFPS recommended = *A screening test* to monitor long-term control of blood glucose levels. Goal = 100% of all eligible patients; Rank represents adjusted Medicare expenditure compared to all 306 HRR. Hospital Referral Region (HRR) represents health care markets for tertiary care. Each HRR is a grouping that contains at least one hospital that performs major cardiac procedures and neurosurgery. For more details describing the definition of HRR see http://www.dart mouthatlas.org/faq/geogappdx.pdf

Analyses of laws and their impact on society are classically the province of legal scholars and historians. The tools employed by these researchers are not familiar to most of the social science research community. For example, legal scholars rely on court cases and develop their analyses from a case by case review of court rulings. Historians are probably the most fortunate of all scholars. They have the gift of being able to operate with hindsight. Timelines of 50 to 100 years are ideal for historical analyses. Neither legal scholars nor historians routinely utilize the types of statistical tools that social scientists use to measure health disparities. Laws and historical documents do not provide the level of accounting required for quantitative analyses of health disparities used in current social science research. How does the current health care reform create opportunities for more direct measures—allowing social scientists and public health researchers to directly measure the connection between newly enacted legislation and population level trends in morbidity and mortality? Biohistorical analysis is one approach used to link historical events with public health problems occurring over time and geographic distance (Miles & McBride, 1997). This technique linking historical public health data sets with demographic and historical events is still under development.

The remainder of this chapter presents specific research questions that can be used to design studies and facilitate this linkage. These bills illustrate specific opportunities for research. This section is structured as follows:

Law
This is the specific Public Law enacted to directly address a health care concern. For each law cited, the reader can obtain a full copy of the entire bill at http://www.thomas.gov

Issue. Based on a review of committee reports and other legislative source materials, this author summarized congressional intent for each law. The source reports are also available online.

Research. This is a discussion of research questions that have direct application to health disparities in the current era.

HEALTH DISPARITIES LEGISLATION

Public Law 79–725: The Hill-Burton Hospital Survey and Construction Act of 1946

Issue. This law was not enacted, as it is sometimes proposed, to desegregate U.S. hospitals during the era of Jim Crow Medicine (Government Accounting Office Legislative History, PL 79–725, Senate Report No. 674, Volume 3: 1–21, 79th Congress. October 30, 1945, House of Representatives Report

No. 2519, 79th Congress, July 13, 1946, Interstate and Foreign Commerce Committee Report, House of Representatives Report No. 2519, 79th Congress, July 13, 1946.). The Act was designed to provide federal assistance for the planning, construction, and improvement of health care facilities. Recipients of these funds were obligated to provide a minimum dollar amount of uncompensated care to individuals who could not pay for medical care. This minimum dollar was based on a formula included in the legislation that accounted for operating costs. The length of time that the facility was obligated to provide free care was based on the type of monies received. Those facilities receiving loans were obligated until the loan was repaid. Facilities receiving grants were obligated into perpetuity. This Act provided support to build or renovate many of the public hospitals across the United States. As of 2009, there were 217 Hill-Burton obligated facilities nationwide. There are no obligated facilities in Indiana, Nebraska, Nevada, Rhode Island, Utah, and Wyoming. In many areas of the country, the Hill-Burton obligated facilities are outpatient clinics or nursing homes. Very few of the remaining services include hospitals. This lack of full service health care presents a significant barrier for rural communities as well as under resourced urban areas. A complete list of these facilities can be found on the Health and Human Services Web site (http://www.hrsa.gov/hillburton/hillburtonfacilities.htm). For a detailed discussion of the place Hill-Burton obligated facilities occupy in the biomedical history of the United States see Byrd and Clayton (2002). Wailoo (2001) also provides a lively presentation of the process surrounding implementation of Hill-Burton legislation in Memphis, Tennessee during the 1940s and 1950s.

Legislation is not made in a vacuum. It evolves out of a combination of perceived need, envisioned solution, and political will. Just enacting public law is no guarantee that a need will be addressed. Once legislation is made into public law, it can still be stymied by challenges through the court system. The new law takes on a life of its own. In our democracy, a law's effectiveness is still dependent upon a consensus process. If passage is marked by a contentious process, then the process of law-initiated change will require continued expenditures of energy by its supporters. If the passage is the result of a broad consensus, then the process of change will begin with a broad-base of local support. Local concerns, however, will still require attention from proponents of the new law. These concerns carry the potential to distort the original intent of the law—diminishing effective application to the need it was designed to resolve in the first place. When Hill-Burton was enacted, there was a convergence of opinion around the idea that the United States needed to make health care available to a broad group of citizens and that the federal government

could play a role in this process by making grants and loans available to local communities for the construction of facilities.

Research. Current health disparities researchers who use an *individual's experience* to frame their research might be inclined to make the following observation: "Hill-Burton, enacted in 1946, is an historical event. It is irrelevant to the measurement of mortality and morbidity rates in 2009." When health disparities are reframed to include the *individual and Hill-Burton obligated facilities*, new questions and study designs immediately come into view. Across the United States, 217 facilities are currently designated as Hill-Burton obligated. Does their geographic placement contribute significantly to the variation observed in health disparities statistics across areas? Since the 1960s, the number of Hill-Burton facilities has declined. Using time-trend modeling methods, can we detect an association between these declining numbers and changes in infant mortality and maternal mortality? Simple counts are one approach to measuring Hill-Burton facilities. Per capita expenditures in each Hill-Burton facility is another approach to measuring the resources available to these facilities. Historical appropriations data for Hill-Burton facilities is available through congressional records. Over time, does declining appropriations account for significant variation within a community in mortality and morbidity for users and nonusers for Hill-Burton facilities? These simple questions create points of collaboration between legislative initiatives and social science models of health disparities.

Public Law 100–713: Indian Health Care Amendments (1988)

Issue. Federal Indian Health Services is an issue dating back to 1789, when the Department of War was charged by Congress to handle Indian Affairs and care was provided by military doctors. The earliest Indian treaty providing for health services was signed in 1832 with the Winnebago of Wisconsin (7 Stat. 370). Overall, congress made 44 treaty commitments to provide tribes with a physician, a hospital, medicines or vaccine, or some combination of these resources. Many of the treaties contained a delimited time period. The Snyder Act of 1921 provides basic authorization for Indian Health Care without time or dollar limitations. It authorized the Bureau of Indian Affairs to "direct, supervise, and expend such moneys as Congress may from time to time appropriate, for the benefit, care, and assistance of the Indians . . . for relief of distress" (Kappler, CL. 1979) The Indian Health Care Improvement Act, Public Law 94–437 was enacted to provide "the highest possible health status to Indians and to provide existing Indian Health services with all the resources necessary to effect that policy." Some of the specific goals of the

Act were "to increase the number of Indian health professionals, to eliminate deficiencies in health status and resources, to improve health facilities, and to provide health care services for urban Indians." Lack of accessible medical providers is an important issue in the Native American community, because most health care services provided by the Indian Health Service (IHS) are provided in rural areas and on reservations. The IHS service provision is further complicated by the fact that more than 50% of Native Americans live in urban areas. Financing health care for the Native American community is a significant problem, despite the federal government's responsibility to provide health care for American Indians and Alaska Natives from federally recognized tribes (Majette, 2009; Walke, 2009).

The impact of appropriations authorization over time on health disparities among clients of the Indian Health Service is an issue that needs to be explored formally by health disparities researchers. The IHS appropriations are divided into two budget categories—health services and health facilities. The federal government considers the provision of these services a trust responsibility based on federal statues, treaties, court decisions, executive actions, and the Constitution that assigns authority over Indian relations to Congress (Walke, 2009). Congress, however, only has a moral obligation (not a legal one) to provide Indian Health Care. The Supreme Court has rejected the idea that IHS is under any obligation to provide specific health program to Indians (U.S. Statutes at Large).

Research. The population served by the IHS has the highest rates of morbidity and mortality of all populations in the United States (i.e., the greatest burden of health disparities). The availability of longitudinal data on appropriations and health indices creates the opportunity for specific analyses to estimate the effect of appropriations on health disparities after controlling for individual level traits. How much of a decrease in mortality and morbidity could we observe if IHS facilities, services, and personnel were funded at originally proposed levels? What if a comparison is made between the Veterans Administration Health Service and the Indian Health Service? What could we learn about the association between resources and health outcomes?

Public Law 106–525: Minority Health and Health Research and Education Act of 2000

Issue. The law has five titles that specify the creation of a National Institutes of Health based center (Title I); guidelines for Health Disparities by the Agency for Health Care Research and Quality (Title II); a study and report to congress by the National Science Foundation (Title III); a component focused on Health Professions Education (Title IV); and a directive to engage in Public

Awareness and Information Dissemination Campaign (Title V). There is also specific statutory language defining minority individuals.

The National Center on Minority Health and Health Disparities (NCMHD) was established by the passage of the Minority Health and Health Disparities Research and Education Act of 2000. Public Law 106–525, was signed by the President of the United States on November 22, 2000 and Dr. John Ruffin was sworn in as its first director of the NCMHD in 2001. Programs mandated by Congress were implemented to expand the infrastructure of both intramural (NIH) and extramural institutions (colleges and universities) committed to health disparities research and to encourage the recruitment and retention of highly qualified minority and other scientists in the fields of biomedical, clinical, behavioral, and health services research. In 2002, the first National Advisory Council of the NCMHD was convened.

The origins of NCMHD can be traced to 1990 when the Office of Research on Minority Health (ORMH) was established by the NIH Director. Two years later, the Minority Health Initiative (MHI), a centerpiece of the ORMH agenda, was launched and initially funded at $45 million. In 1993 Congress enacted Public Law 103–43, the Health Revitalization Act of 1993 to establish the Office of Research on Minority Health in the Office of the Director, NIH. In 1997, the Advisory Committee on Research on Minority Health was established providing advice to the Director, ORMH, and to the Director, NIH, on research and research training with respect to minority health issues. By 2003, the ORMH issued the first NIH Strategic Research Plan and Budget to Reduce and Ultimately Eliminate Health Disparities. Source: National Center for Minority Health and Health Disparities, http://www.ncmhd.nih.gov).

Research. What impact has this governmental initiative had on statistical measure of health disparity? In other words, how large were the gaps in morbidity and mortality prior to this law's enactment? Has there been a downward trend in key indicators of health over the past 8 years? Trans-NIH cofunded investigator initiated research is the Center's highest priority. Is there evidence that this disease-specific approach to eliminating health disparities is yielding results? All government agencies are dependent upon final appropriations to have the resources necessary to complete their stated mission. What is happening with appropriations for NCMHD? In FY08, appropriations were 9.2% less than requested. This downward trend in appropriations and the overall productivity of this center is worth watching because it reflects the commitment of the United States government to improve the health of its minority populations. As this Center matures will it survive several presidential transitions?

PROSPECTIVE STUDIES AND NEWLY ENACTED LEGISLATION

A confluence of factors has created the *political will* to create new laws that have the potential to diminish health disparities. This section is designed to alert researchers to the potential for prospective studies of two new laws as they are implemented.

Public Law 110–343 §511 and §512: Paul Wellstone and Pete Dominici Mental Health Parity and Addiction Equity Act of 2008 (Toxic Assets Relief Plan or TARP)

Issue. Researchers will find this act embedded within legislation to address the crisis experienced by banks that have subprime interest mortgages. To honor the work of these two legislators, Congress passed an amendment bearing their names to create parity in treatment, payment, and other features of mental illness and placing these services on par with the treatment of physical illnesses. Title V (subtitle B) §512 amends section 712 of the Employment Retirement Income Security Act of 1974. It requires group health insurance plans to provide benefits for mental illness and substance abuse treatment that are comparable to covered treatments for physical illnesses. The legislative language goes into considerable detail describing the timeline for phasing in these provisions and employer requirements for financing.

Research. Provisions of this law will become active beginning in 2010. Do these provisions influence mental illness treatment access for men and women in the workplace and for racial and ethnic groups who are employed in similar positions? In short, what does this legislation do to diminish mental health disparity? Are there unintended consequences for loss of employment among persons who utilize these benefits? Are these consequences differentially experienced by racial and ethnic groups?

Public Law 111–03: The American Revitalization and Reinvestment Act (The Stimulus Package)

Issue. To accurately assess the true extent of health disparities across racial and ethnic groups, the government needs to examine data controlled by the Centers for Medicare and Medicaid. In the Stimulus Package, the following section: §3002(b)(2)(vii): The use of electronic systems to ensure the comprehensive collection of patient demographic data, including at a minimum, race, ethnicity, primary language, and gender information.

Research. This new data element will create the opportunity to measure disparities in morbidity and mortality at multiple levels (by geographic areas,

by zip code, and by health care institution. For the first time, researchers will be able to compare hospitals and clinics within a city to measure accessibility by disadvantaged groups and to detect differences in treatment outcome.

CONCLUSION

Public health research that targets health disparities needs to be reframed to incorporate laws directed toward hospitals, clinics, and other health care infrastructure. There is historical evidence that legislative activity by the U.S. Congress can diminish health disparities. Unintended consequences have also resulted from these legislation efforts. The formal study of these unintended consequences needs to be incorporated into statistical models that estimate the relative contribution of individual and systematic barriers to health. Health care reform presents an opportunity for researchers to prospectively study health disparities and related policies. Health care reform is driven by policy. Policy is defined by research. There is considerable work for researchers in collaboration with federal, state, and local governments to measure the intersection between health care reform, legislation, and the indices of health disparities.

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