

LERNER, R.M., Easterbrooks, M.A., Mistry, J., &
WEINER, I.B. (2013). *Developmental Psychology*

CHAPTER 17

Disease, Health, and Aging in the First Decade of the 21st Century

ILENE C. SIEGLER, HAYDEN B. BOSWORTH, ADAM DAVEY, AND MERRILL F. ELIAS

DEMOGRAPHIC AND PUBLIC HEALTH CONSIDERATIONS 439
INCREMENTAL FINDINGS FROM REVIEW CHAPTERS 440
UNDERSTANDING RISK AND PREDICTIONS OF COGNITIVE AND FUNCTIONAL DECLINES 441
MEDICAL SELF-MANAGEMENT AND NEW MODELS OF CARE 443

END OF LIFE ISSUES 443
PREDICTIONS FOR THE FUTURE AND THE ROLE OF DEVELOPMENTAL PSYCHOLOGY 445
CONCLUSIONS 445
REFERENCES 446

In the 10 years or so since the last version of this chapter, there has been steady progress in research on developmental health psychology. Areas that were described in our previous chapter (Siegler, Bosworth, & Poon, 2003, Chapter 17) that are still applicable remain as written and are updated where necessary. Most of the ongoing research in the psychology of adult development and aging has been incremental, with the emphasis on qualitative changes in the lifecycle most prominent at the start of

adulthood (see, e.g., Arnett, 2010; Arnett & Tanner, 2006). Research in psychosomatic medicine started taking aging and the life course seriously, with a special issue (Ory & Chesney, 2002) published after the previous chapter was written. We argue that the major changes in understanding disease and health among the elderly over the past decade come from increased methodological sophistication about how to model and deal with time and from the role of the Web in making data and international collaboration commonplace; the latter improvement has resulted in a new paradigm for research in the future that previously was only common in the study of centenarians (e.g. Robine, Vaupel, Jeune & Allard, 1997). This new paradigm is just getting organized in psychology of aging (e.g., Hofer & Piccinin, 2009) but is in full operation in the studies of Alzheimer's disease (AD) modeled on the collaborative study, with multiple centers pooling data with public-private partnerships and international

Dr. Siegler's work on this chapter was supported by NIH Grant #R01 HL55356 from the National Heart Lung and Blood Institute (NHLBI) and cofunding by the National Institute on Aging (NIA): P01 HL36587, Grant #IRG-08-89565 from the Alzheimer's Association and the Duke Behavioral Medicine Research Center.

Dr. Elias' work on this chapter was supported by research grants 1R01-HL67358 and 1R01-HL081290 from the NHLBI and The National Institutes of Health, to the University of Maine. The content is solely the responsibility of the author and does not represent the official views of the NHLBI.

Dr. Bosworth's work on this chapter was supported by a NHLBI grant (R01 HL070713), a VA Health Services Research and Development grant (20-034), an Established Investigator Award from the American Heart Association, and a VA Career Scientist Award(08-027) to the third author. The views expressed in this manuscript are those of the authors and do not necessarily represent the views of the Department of Veterans Affairs.

Dr. Davey's work on this chapter has been supported by grants from the National Institute of Aging R01 AG13180, the National Cancer Institute R21CA158877, and from the U.S. Department of Agriculture PENR-2008-05011, PENR-2010-04643.

We would like to thank Shirley Austin for her work on coordinating this chapter.

We wish to acknowledge the editorial work by Ms. Danielle Briggeman, The University of Maine.

collaboration (see Weiner et al., 2010). These advances have the potential to improve the worth of psychology of adult development and aging to public health (Lahey, 2009).

The goals of developmental psychology and the place of adult development and aging and the study of health have changed only in incremental ways. Thus, most of our previous introduction is still relevant. Developmental psychology's goal is to describe, predict, and understand the changes that come with age. Developmental psychology is a multidisciplinary field within psychology—a field that cuts across all of the standard areas of psychology. We adopt the conventional terminology for changes, which implies variations in longitudinal measurements on the same persons over time; this is the core of the psychology of aging. We reserve the term *age differences* for cross-sectional age comparisons at a single point in time; such comparisons are the province of experimental aging research or a psychology of the aged. Understanding the variance accounted for by particular health problems or disease statuses requires both approaches.

A basic contribution of developmental psychology has been the development of methodological advances to improve ways to assess age changes in the aforementioned contexts. Health and disease are prominent factors that influence changes associated with age; thus, this chapter examines the measurement and meanings of health and disease and their contribution to our understanding of the aging process, called developmental health psychology (Siegler, 1989; Siegler et al., 2003, p. 423). The key term in developmental psychology is age—that is, the age at time of measurement; the age at the beginning and at the end of the measurement period; the age at the onset of a disorder; and the age at death—but new statistical methods recognize that the age when the study began is not really the baseline. Baseline measures in longitudinal designs capture a point in time along the age continuum that extends forward and backward.

Developmental psychology sees age as more than just a marker variable or placeholder; age is a dynamic process. A basic contribution of developmental psychology has been the development of methodological advances to improve ways to assess age changes and separate out the times of measurement and cohort effects, all of which get more complex with changes in health status (Schaie, 1977, 2005). However, there is an increasing recognition that more efficient designs do not need to include every measure for every person at every time of measurement, and

indeed that many hypotheses of developmental interest may not even be accessible within traditional complete-data designs (Davey & Salva, 2010).

What were our conclusions 10 years ago, and are they still relevant?

Period effects and changes in technology and treatments are continuing to redefine relationships between aging and disease; however, they are still not applied equally across the socioeconomic status (SES) gradient (cf. Gianaros & Manuck, 2010; Krieger et al., 2008) and have major implications for research and care at the end of life (see Gawande, 2010). The genetics revolution is continuing. Data are accumulating at a dizzying rate; it is too soon to see how this will change our understanding of the aging process. Survey research has moved to include biomarkers and shortened versions of psychological constructs. This has costs as well as benefits. It provides national population data on psychological constructs, but these need to be validated to ensure comparability (see Siegler and Davey, 2012). Finally, there is growing recognition that some studies simply cannot be completed without incomplete data (e.g., accelerated cohort designs in longitudinal studies), and some hypotheses (e.g., effects of repeated testing) cannot be tested in complete data designs.

Further, selection for incomplete data is naturalistic and intimately connected to the structure of the human life span, as considered in greater detail in the following section. Planning a study with incomplete data may represent a cost-effective alternative to collecting complete data on all individuals (e.g., Derks, Dolan, & Boomsma, 2007; Graham, Hofer, & MacKinnon, 1996; Graham, Taylor, Olchowski, & Cumsille, 2006; Helms, 1992; Moerbeek, 2008; van der Sluis, Dolan, Neale, & Posthuma, 2008). In other situations, incomplete designs may be the only practical and/or cost-effective way to plan a study. It may make the difference between collection of data or no study at all (Davey & Salva, 2010; Leon, Demirtas, & Kedeker, 2007). These new methodological developments make synthetic approaches (i.e., those that combine data across multiple samples, measures, cohorts, and/or designs) to estimating the life span possibly more realistic than in the past, but survival of increasingly impaired persons makes a life-span psychology less likely to be a credible model that can be applied universally.

Fears about public policy and demographic transitions have increased as the first Baby Boomers (born 1946–1964) begin to turn 65 in 2011. We will start this chapter with such concerns.

DEMOGRAPHIC AND PUBLIC HEALTH CONSIDERATIONS

Table 17.1 (from Siegler et al., 2003) is still a reasonable description of disability and use of nursing homes until new census data is available, as generally, reports treat “over 85” as a single category. We since learned that cognitive function plays an important role as a mediator between risk factors such as hypertension and disability (M. F. Elias, Dore, Davey, Robbins, & Elias, 2010). Continued research determining how to move the proportion of life spent without disability, which is considered to be about 60% of the remaining life span after age 65 (Kinsella & Gist, 1998), is still needed.

Life expectancy continues to increase, but the increases are gradual in the United States. Boomers born in 1946 turned 65 in 2011. Thus there is great concern on how this huge and varied cohort will behave in the next 30 years. Life expectancy at 40 is now 43.6 years, so half of all who have reached 40 can expect to live beyond age 83.6 (IRS, 2009). Even Social Security, in the documents it sends yearly, informs us of about possible future increases in life span and has raised the retirement age to 66 for persons born after 1943.

Findings from the Georgia Centenarian Study

Recent findings from the Georgia Centenarian Study (Arnold et al., 2010) can help to fill in some of the details of what increased longevity might bring. First, surviving to 100 happens with chronic disease. Following findings from the New England Centenarian Study (Evert, Lawler, Bogan & Perls, 2003), we found similar pathways to longevity: *Survivors*, who had chronic disease before age 80 (43%), *Delayers*, who developed disease between ages 80 and 98 (36%), and *Escapers*, who developed disease after age 98 (17%); compare these percentages to 38%, 42%, and 19% in the New England Study (see Table 3, page 4 from Arnold et al., 2010). Dementia was present

in 57% of the population; while cardiovascular disease was the most prevalent (80%), diseases with under 10% prevalence included diabetes (9%), kidney disease (7%), neurological disease (6%), and chronic obstructive pulmonary disease (see Table 5, p. 7 from Arnold et al., 2010) in this very small slice of the population. While data from centenarian studies are generally more optimistic than expected, they represent a very small slice of the population. We need to think about how to manage survival of the non-fittest, longevity without cognition or personality, and dependence on caregivers when the care recipient has no family left or never had any offspring (Siegler, 2010). Thus differences between fatal and nonfatal disease becomes increasingly important in the extreme aging. It is also useful to have a full picture of what the extreme aging actually look like (in terms of their cognitive functional health physical capacity, biomarkers and health habits, and chronic disease status) and how this is associated with gender, race, residential living arrangements and educational attainment, both taken separately and modeled together.

Davey and colleagues (2010) provide detailed normative data on the 244 members of the Georgia Centenarian Study who were aged from 98 to 109 at the time of measurement, circa 2005. While an N of 244 seems small, it compares favorably to national data from HRS AHEAD study, with 143 individuals in that age range, and 253 from the National Long Term Care Survey. In general, physical illness profiles provide little independent information after living arrangements and educational attainment are accounted for, which are the two primary variables accounting for race and gender differences. When considering cognitive outcomes, the major source of systematic variation was associated with formal educational attainment, followed by residential status (community or facility). In terms of physical performance measures, the major source of systematic variation was associated with residential status, followed by gender. Readers are invited to look at the variables they know best to get a fuller understanding of the extreme variation in this increasingly important segment of the population found in Davey and colleagues (2010) and described in the following paragraph.

The protocol is composed of three domains of physical health: (1) physical examination and health history; (2) blood chemistry profile, including nutritional assessment; and (3) physical function assessment, including measures of bed mobility, bed-to-chair transfer skills, standing balance, walking, step-up, and chair standing abilities.

TABLE 17.1 What Are Older People Like, and How Old Is Old?

Age Group	% No Disability	% in Nursing Home
65–69	83%	3%
70–74	83%	5%
80–84	62%	10%
85–89	45%	17%
90–94	35%	32%
95–99	20%	42%
100+	18%	48%

Cognition, neuropsychology and mental health were assessed by a variety of instruments. These include: Mental State Examination (MMSE; M. Folstein, Folstein, & McHugh, 1975), Global Deterioration Scale (Reisberg & Ferris, 1982), Severe Impairment Battery (Saxton & Swihart, 1989), Fuld Object Memory Evaluation (FOME; Fuld, 1981), Wechsler Adult Intelligence Scale-III, Similarities subtest, Letter Number Sequencing subtest, and Matrix Reasoning subtest (Wechsler, 1997), Behavioral Dyscontrol Scale (BDS; Grigsby, Kaye, & Robbins, 1992), ILS Health & Safety Scale (Loeb, 1992) and the Controlled Oral Word Association Test (Benton & Hamscher, 1997).

Basic functional capacity was assessed using both a self-report as well as performance-based measures of basic/physical and instrumental activities of daily living (BADL and IADL). Physical functional capacity was measured with the NIA Short Physical Performance Battery (SPPB; Guralnik et al., 1994) and Physical Performance and Mobility Examination (PPME; Guralnik & Winograd, 1994). The performance-based measures include selected subtests of the Direct Assessment of Functional Status—Revised (DAFS-R; Loewenstein et al., 1989). The measures and Performance Rating Scale were taken from OARS (Fillenbaum, 1988; see Davey et al., 2010). Additional information about the methodological considerations necessary to study centenarians is discussed in Poon and Perls (2008).

These norms from the Georgia Centenarian study are complemented by extensive norms from the Maine–Syracuse Longitudinal Study and the Framingham Heart Study, which provide cross-sectional normative data on health and many cognitive measures organized in the decades extending from early adulthood to the years immediately preceding centenarian status (Dore, Elias, Robbins, Elias, & Brennan, 2007; M. F. Elias et al., in press; Au et al., 2004).

Aging and Health

The aging of the population and the increasing prevalence of chronic diseases pose challenges to the U.S. health-care system. One out of 10 deaths among Americans each year are from chronic diseases. Heart disease, cancer and stroke account for more than 50% of all deaths each year (Kung, Hoyert, Xu, & Murphy, 2008), and 133 million Americans—almost 1 out of every 2 adults—had at least one chronic illness (Wu & Green, 2000). Obesity has become a major health concern; 1 in every 3 adults is obese (Ogden, Carroll, McDowell, & Flegal, 2007).

About one-fourth of people with chronic conditions have one or more daily activity limitations (Anderson, 2004). Healthcare costs for individuals with at least three chronic conditions accounted for 89% of Medicare's annual budget (RWJ Web site; see Anderson, 2004). Multimorbidity is associated with poor quality of life, increased physical disability, high health care use, use of multiple medications, and increased risk for adverse drug events and mortality (Hoffman, Rice, & Sung, 1996). Optimizing care for this population is a national priority (Anderson, 2003). Consider that, just for hypertension, the residual lifetime risk for hypertension for middle-aged and elderly individuals is 90% (Vasan et al., 2002); we need to better consider how we capture these comorbidities and what we do with this information.

INCREMENTAL FINDINGS FROM REVIEW CHAPTERS

The psychology of adult development and aging uses edited volumes as a useful way to integrate information. As well, undergraduate text books in adult development and aging now routinely include information on physical and mental health (e.g., Hoyer & Roodin, 2009). We have recently completed companion chapters that may be useful to the readers of this chapter. Siegler and colleagues (2009) review how changes in conceptualizations of normal aging have had an impact on our understanding of psychiatric syndromes, which in turn has implications for treatment. In particular, this chapter gives a good brief summary of the implications of the 50 years of research on the Seattle Longitudinal Studies of intellectual development and the data on cognitive interventions with older persons; reviews of findings in neuroimaging, personality, and health-related cognitive changes from cardiovascular diseases; a summary of findings from centenarian studies around the world; findings on personality and disease at midlife; and as a predictor of survival and a discussion of the stresses of caregiving as experienced by minority families.

Hooker, Hoppmann, and Siegler (2010) focus on lifespan personality and health, and highlight the role of the UNC Alumni Heart Study. Siegler and Davey (2012) was written to evaluate the role of risk factors with age over the lifecycle in a volume focused on adult development, and uses data from a variety of sources to develop a synthetic cohort approach from age 40 to 100.

Siegler, Hooker, Bosworth, Elias, and Spiro (2010) have written the aging chapter of a new volume on

behavioral medicine, which covers a wide range of data but also has particular interest in the measurement of age and the treatment of time as a variable. The volume (Suls, Davidson, & Kaplan, 2010) shows the expansion of behavioral medicine into treatment, evidence-based medicine, and public health.

As well, we have listed many of the longitudinal aging studies that are continuing to produce important new data:

Longitudinal studies are now maturing. Investigators are publishing data that has been collected for over 50 years (Seattle Longitudinal Study, Schaie, 2005; Baltimore Longitudinal Study of Aging; [Terracciano, Lockenhoff, Zonderman, Ferrucci & Costa], 2008; Normative Aging Study, Mroczek & Spiro, 2007). In addition, findings from a second generation of psychologically oriented longitudinal studies (e.g., Berlin Aging Study; Baltes & Mayer, 1999; Gerstorf et al., 2008; Victoria Longitudinal Study; MacDonald, Hultsch & Dixon, 2008) have incorporated broadly measured health and cognitive measures into their designs, resulting in an increased understanding of stability and change in basic psychological processes and their implications for health and survival.

—Siegler et al., 2010, p. 147

Measuring Health

In our chapter in the previous edition, we reviewed various measures of functional health, health-related quality of life, and self-rated health, as well as medical and psychiatric comorbidity (Siegler et al., 2003, pp. 430–434). The issues are still the same. Possibly because they don't have objective measures of disease, or cannot afford them, many psychologists have rushed prematurely to establish self-report of health variables as "just as good as" objective measures. What this literature has established is that self-report of general health is important in its own right as a predictor of mortality (Singh-Manoux et al., 2007) and reflects the fact that individuals can report accurately how they feel but cannot necessarily diagnose the cause of their discomfort. Nonetheless, research has shown that self-rated health does predict major cardiovascular events, depending on assessment of functional status (Rutledge et al., 2010). Few medical practitioners or epidemiologists will agree that asking someone whether they have hypertension or diabetes is as good as measuring it objectively. Survey research generally asks about reports of disease conditions that have been diagnosed by a physician, or treatments of invasive procedures, or medications taken. There is a large literature in epidemiology that has verified such conditional reports and suggests that in well-educated populations with access to medical care, they can

be reliable in terms of identifying the presence of the particular condition or disease, but these are not necessarily adequate measures of severity of disease (e.g., presence of hypertension, but not necessarily actual blood pressure levels). In general, the level of reliability of self-report measures is different for each disease condition, and it is important to consider the role of both reporter (e.g., self versus proxy reports) and potential moderating variables (e.g., cognition), particularly for the oldest old (Mitchell et al., 2011).

In the UNC Alumni Heart Study, which is a mail survey, for example, we ask about full range of doctor-diagnosed diseases and coronary procedures, and we collect medical records for reported cardiovascular events. We have found that the self-report of coronary procedures is excellent and is always verified by the records. The outcomes of diagnostic procedures and heart attacks are less so. The records we receive about these suggest that the person is reporting accurately in that the status of the diagnostic procedure is often ambiguous. People can only tell you accurately what they know. Furthermore, there are methodological techniques that can be used, such as those derived by Davey and Salva (2010) to evaluate whether designing studies with some inferior and some gold standard methods is, in fact, more cost efficient (Davey & Salva, 2010) leading toward the establishment of the appropriate uses of various methods of disease assessment.

UNDERSTANDING RISK AND PREDICTIONS OF COGNITIVE AND FUNCTIONAL DECLINES

Petersen and colleagues (2009) provide a 10-year review of the construct of mild cognitive impairment and the extent to which it converts to AD, as well as the problems with it as a construct. Findings from Washington University (Johnson, Storandt, Morris, & Galvin, 2009) speak to transition from healthy aging to AD; longitudinal data on multiple aspects of cognitive functioning in those who develop dementia is clear about 3 years before the clinical diagnosis, suggesting the increased power of repeated measures in a longitudinal design.

Many risk factors for cardiovascular disease are related to quality of life, cognitive performance, and dementia (Smith, 2008; Waldstein & Elias, in press). These include: rising blood pressure, hypertension, diabetes, obesity, atherosclerosis, atrial fibrillation, smoking, low or no alcohol consumption in some studies, low physical activity, a diet low in fish, rate of decline in body mass

index (BMI), low education, low intake of antioxidants, raised markers of inflammation, raised plasma total homocysteine, low plasma concentrations of folate, vitamin B₆, vitamin B₁₂, low testosterone in men, hormone replacement therapy in women after age 65, nonuse of NSAIDs, low thyroxine-stimulating hormone (TSH), head injury in men, depression, poor perceived health, and low level of social and mental activities (Siegler, Elias, & Bosworth, in press; Smith, 2008). In addition to environmental and lifestyle risk factors, there are genetic risk factors for decline in cognition and dementia—for example, carrying one or more of the ApoE-e4 alleles. Among other functions, the ApoE gene is involved with neuronal repair. Cognitive deficit is exacerbated in diabetics (e.g., Dore, Elias, Robbins, Elias, & Nagy, 2009) or persons with high homocysteine values who carry at least one ApoE-e4 allele (e.g., M. F. Elias, Goodell, & Waldstein, in press).

Because cardiovascular risk factors can be present very early in life (Waldstein, 1995), it is important to prevent or detect and treat them as early as possible. While these cardiovascular risk factors herald dementia and mild cognitive dysfunction, they are also responsible for lower cognitive performance in persons free from stroke and dementia. Lowered cognitive functioning is, in turn, a risk factor for MCI and dementia (M. F. Elias et al., 2000; Smith, 2008).

With advancing age, the likelihood of multiple cardiovascular risk factors increases, and risk factors act in a synergistic manner to work their adverse effects on quality of life and cognitive ability. The greater the number of risk factors, the lower the cognitive performance (M. F. Elias, Elias, Robbins, Wolf, & D'Agostino, 2001). Stroke-free persons who, by virtue of cardiovascular disease risk factors, are at the highest risk of acute stroke based on the Framingham Stroke Risk Profile exhibited the lowest levels of cognitive functioning in the Framingham Heart Study (M. F. Elias, Sullivan, et al., 2004).

Next to diabetes mellitus, hypertension has probably received the greatest attention in the adult developmental psychology literature. Prospective designs led to the discovery that untreated blood pressure (BP) levels are inversely related to cognitive performance measured in midlife (M. F. Elias et al., 2001). More importantly, for both young adults and middle-aged adults, the higher the BP at baseline, the greater the acceleration in decline in fluid, but not crystallized-verbal abilities, over time (M. F. Elias, Robbins, Elias, & Streeten, 1998), and the rate of decline over time is the same for young and middle-aged adults (P. K. Elias, Elias, Robbins, & Budge, 2004). However, for pulse pressure (systolic–diastolic

blood pressure), an index of arterial stiffness, accelerated decline in cognition was only observed when baseline BP was related to change for middle-aged individuals (see M. F. Elias, Robbins, et al., 2004 for review).

The age range over which blood pressure and cognition is tracked is important. These age \times hypertension interactions may change in very old age or when the longitudinal study involves only elderly individuals (Wilkie & Eisdorfer, 1971). Here, old age may be a disadvantage in the presence of sustained high blood pressure. Curvilinear relations between blood pressure and cognition have been seen in the Baltimore Longitudinal Study of Aging (Waldstein, Giggey, Thayer, & Zonderman, 2005). For hypertension and for other risk factors, more longitudinal studies are needed and will be seen as the longitudinal studies in progress mature. It is important to note that lower is not necessarily better with regard to blood pressure among older adults. Hypotension is a risk factor for lower cognitive performance and dementia, and the elderly are especially vulnerable given its effect on decreased cerebral blood flow and the fact that blood perfusion of the brain decreases across the life span (de la Torre, in press).

While representing only a small fraction of the literature and one risk factor, the literature on hypertension illustrates that one must take a life-span approach to understanding risk in relation to cognitive change and that one must not assume that what holds for one risk factor holds for all others, that youth protects against the adverse behavioral consequences of hypertension, or that lower is better under all circumstances. This is also made complex by the fact that changes in diastolic blood pressure are problematic for young/middle adults, but rarely do older adults have problems with diastolic blood pressure; rather, the majority of hypertension is systolic hypertension in older adults.

Space does not permit examples from the literature with other risk factors. However, we direct the reader to edited volumes by Waldstein and Elias (2001), Waldstein and Elias (in press), Siegler et al. (in press), Siegler, Hooker, Bosworth, Elias, and Spiro (2010), and M. F. Elias, Robbins, and colleagues (2004) for this literature.

The literature on hypertension, with its emphasis on cerebral blood flow, has stimulated an interest in heart and cognition in general. Each of the following heart diseases increases the burden of reduced blood flow to the brain with advancing age and has been associated with dementia (de la Torre, in press): (a) low ejection fraction or low cardiac output; (b) atrial fibrillation; (c) aortic and mitral valve prolapse; (d) hypertension; (e) heart failure;

and (f) coronary artery disease. The model for this process appears to be straightforward. Glucose is the primary molecule used to create energy fuel for mammalian brain cells, and thus the brain depends on a continuous and optimal flow of blood to maintain normal brain cell activity and structural integrity. Brain cells receive 21% less blood flow at age 60 compared to age 22, so any additional burden that lowers cerebral perfusion can damage or kill vulnerable neurons (de la Torre, in press). As this line of investigation progresses, life-span studies will be essential. It is important to track the progression of reduction in brain perfusion from normal to stages identified as reductions in cerebral blood flow (brain hypoxia) and chronic and acute cerebral ischemia. Essential tools include batteries of cognitive tests measuring different domains of functioning, neuroimaging methods, and cerebral blood flow studies.

MEDICAL SELF-MANAGEMENT AND NEW MODELS OF CARE

Since the previous edition's chapter was written, there has been a new emphasis on patient-centered care, the medical home, and self-management; treatment of disease has expanded past the province of physicians now. Chronic diseases, specifically, cardiovascular diseases (CVD), have become the leading cause of death and disability in most countries in the world (Lawes, Vander Hoorn, & Rodgers, 2008). In the United States, an estimated 81.1 million persons have CVD, and coronary heart disease (CHD) and stroke remain the first and third leading causes of death, respectively. CVD also carries an enormous personal and financial burden; the total direct and indirect cost of heart disease and stroke in the United States for 2010 is estimated at \$503.2 billion (American Heart Association, 2010).

Managing a chronic illness is a time-consuming and complex process. Patients and their informal caregivers are required to make day-to-day decisions about such actions as how to respond to new symptoms, what and how much to eat, whether to take their medication, or whether to exercise—all of which can have substantial effects on their clinical outcomes, particularly when the decisions are aggregated over months and years. These day-to-day decisions and tasks are referred to as self-management, which was formally defined by Barlow and colleagues as “the individual’s ability to manage the symptoms, treatment, physical and psychosocial consequences and lifestyle changes inherent in living with a

chronic condition” (Barlow, Wright, Sheasby, Turner, & Hainsworth, 2002). All patients with chronic diseases self-manage; the question is how well they self-manage and the influence of self-management on the patient’s experience of chronic disease and health outcomes.

The potential benefit of interventions to improve patients’ self-management and subsequent health behaviors exceeds that of interventions aimed at health-care providers, in part because unhealthy behaviors may contribute more than inadequate health care does to poor health and premature death. Unhealthy behaviors such as smoking, poor diet, and sedentary lifestyles account for as much as 40% of premature deaths in the United States, whereas deficiencies in health-care delivery account for only 10% (Stampfer et al., 2000).

Self-management is more than simple adherence to provider recommendations, because it also incorporates the psychological and social management of living with a chronic condition. Indeed, self-management consists of the following components: engaging in activities that promote physical and psychological health; interacting with health-care providers and adhering to treatment recommendations; monitoring health status and making associated care decisions; and managing the impact of the illness on physical, psychological, and social functioning (Bosworth, Powers, & Oddone, 2010). To a great extent, patients’ outcomes will be dictated by the degree to which these choices lead to further reductions in risk. As we continue to understand the relationship between diseases like hypertension and CVD and their impacts on cognition, it will become increasingly important to examine ways to reduce the burden of these chronic diseases.

END OF LIFE ISSUES

Issues surrounding how we die are different than they were when previous chapters addressing these questions were published. More people now die later in life, when the cause may not be due to a specific disease (Fried et al., 1998; Nuland, 1995). At least one-third of all disease burden in developed countries can be attributable to five modifiable risk factors: tobacco use, blood pressure level, cholesterol level, obesity, and alcohol use (World Health Organization, 2002). How will the control of these risk factors, if achieved, change aging and the end of life? What role will psychosocial factors play in the development of geriatric syndromes? Once thought to be a reflection of hypertension and diabetes, obesity (especially central adiposity) is now known to be independently

associated with lower cognitive performance (Dore, Elias, Robbins, Budge, & Elias, 2008; M. F. Elias, Elias, Sullivan, Wolf, & D'Agostino, 2003; Waldstein & Katzel, 2001; Wolf, Davis, Tilson, Bass, & Parker, 2006). The role of self-management and health psychology becomes apparent when one realizes that these five modifiable factors that explain a majority of CVD and diabetes, for example, are attributed to behavior (Stampfer et al., 2000).

There are new findings in terminal drop. Thirty-six years after Klaus Riegel introduced the notion to psychology in terms of changes in intellectual functioning (K. Riegel & Riegel, 1972), Gerstorf and colleagues (2008) reported on similar changes in life satisfaction. Harel, Hofer, Hoffman, Pedersen, and Johansson (2007) present models for reaching valid longitudinal inferences with terminal drop. Kurland, Johnson, Egelston and Diehr (2009) present some new methods useful in analysis of old data, while Wilson, Beck, Bienias, and Bennett (2007) provide detailed data showing terminal decline in different cognitive functions beginning about 3.5 years before death.

Issues of mortality and terminal drop underscore the importance of explicitly considering issues of selection out of the population when making inferences about the nature of longitudinal change. To date, very few studies take these issues adequately into account. The importance of selection grows as the length of time over which inferences are being made increases and as research grows to include samples of individuals who are increasingly rare in the population. A related issue is the effect of inclusion and exclusion criteria in prospective studies as an increasing proportion of the population experiences a particular condition (e.g., cognitive impairment, hypertension).

By way of example, Stone, Schwartz, Broderick, & Deaton (2010) present data from an extremely interesting cross-sectional study of well-being, using a sample of 340,847 individuals aged 18 to 85 in the United States. The paper is important, interesting, theoretically derived,

and carefully interpreted. But it does not consider the potential effects of mortality on its cross-sectional conclusions regarding well-being. The authors conclude that "relative to prior studies, the present results broaden the case that WB, including positive affect, increases with age" (p. 3). We would like to suggest that an alternative perspective, namely selection out of the population, is likely to be at least as important in interpreting these data.

It has been well established that constructs associated with negative affect (depression and depressive symptoms) predict mortality (e.g., Barefoot, Mortensen, Helms, Avlund, & Schroll, 2001). To show the effects of selection out of the population on cross-sectional inferences, we began with a stable, standardized ($M = 0$, $SD = 1$) variable that was completely unassociated with age. Next, we calculated the effects on mean levels of this standardized variable of dropping individuals from the bottom of the distribution in proportion of mortality data published in the Social Security Administration life tables, separately for men and women. Changes in population mean levels associated with age are due solely to selection out of the population due to mortality, and as such, there is absolutely nothing "developmental" affecting the data (i.e., individual levels of well-being are constant over time).

As can be seen in Figure 17.1, well-being in the (surviving) population is seen to increase, slowly at first, beginning early in midlife, and then in a rapidly accelerating fashion in the 50s and 60s, which is consistent with when Stone and colleagues (2010) first reported changes. Considered across the entire adult life span, these selection effects are hardly inconsequential. The effects of selection are equivalent to a small ($d = .2$) effect size by age 54 years for men and 62 years for women; to a medium ($d = .5$) effect size by age 71 years for men and 77 years by women, and to a large ($d = .8$) effect size by age 79 years for men and 84 years for women. It is

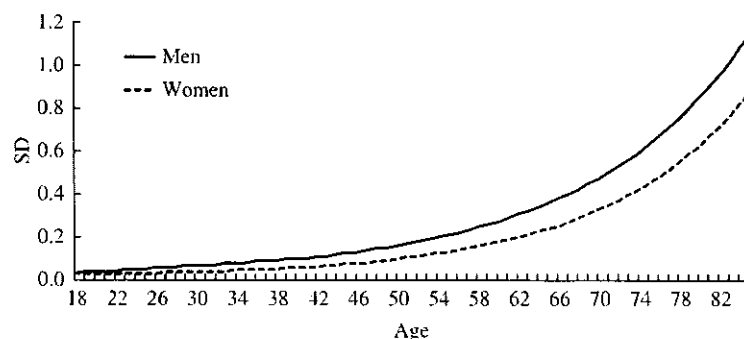


Figure 17.1 Selection effects with age

not difficult to think of research applications in which the effects of selection may easily trump those of the substantive variable under investigation. As a result, we strongly advocate more explicit consideration of the role of selection in developmental psychology with older adults.

PREDICTIONS FOR THE FUTURE AND THE ROLE OF DEVELOPMENTAL PSYCHOLOGY

The “brave new world of aging” may or may not have anything to do with psychology. Life-span or life-course approaches are recognized in epidemiology (Kuh & Ben-Shlomo, 2004). Personality change as a signal of early dementia may prove to be useful (Siegler et al., 1991; Siegler, Dawson, & Welsh, 1994; Balsis, Carpenter, & Storandt, 2005; Duchek, Balota, Storandt, & Larsen, 2007) and studies of the role of personality as factor in cancer risk may be retired (see Ranchor, Sanderman, & Coyne, 2010). Psychological factors and their role in disease etiology and progression must be studied disease by disease. There are many studies showing that psychological factors predict all-cause mortality, that is, they predict age at death.

Dementia may be predictable up to 10 years before onset from cerebral spinal fluid (CSF) indicators (De Meyer et al., 2010). What are the implications for us? If dementia can be identified early and prevented, will it become like polio? Like AIDS? Or will there be a new set of unanticipated problems? The way in which developmental psychology studies constructs in specific areas (cognition vs. personality vs. emotion) may not be useful as we integrate our findings with other disciplines that consider all we do as “psychosocial stuff,” and indeed research within the field may also be moving in that direction (Carstensen, Mikels, & Mather, 2006).

Recognition of the biology of aging is evidenced by the awarding of the Nobel Prize in Physiology in 2009 for work on telomeres—a “key mechanism that cells use to protect their genetic information” (see Vogel & Pennisi, 2009)—as well as new data on the sirtuin gene (Haigis & Guarente, 2006) and potential identification of longevity genes (Jazwinski et al., 2010). Has the technology gotten so complex that the models are not understandable by non-experts? Have we been oversold on the role of genetics? For all the genetic predictors of cardiovascular disease, they do not add much to the Framingham CVD risk score, which consists of diabetes status, smoking status, LDL, age, and SBP (Melander et al., 2009; Shah & de Lemos,

2009). These advances have also spurred biotechnology and drug companies to invest in the biology of aging. Is this the start of a new understanding of basic aging processes and a change in fundamental understanding of aging and development—or not? It may be too early to tell, but we may indeed be on the verge of the true aging revolution. There is a lot of attention in the press and in biotech investments; it may take another 10–20 years to see if these discussions represent hype or real changes (Verghese, 2010; Weiner, 2010).

CONCLUSIONS

In sum, in the past 10 years, most of the work studying aging health and disease has been incremental within psychology of adult development and aging and has been finding great promise in the study of particular disease endpoints combining the traditional methods of adult development and aging—careful longitudinal studies with well-characterized disease outcomes (Siegler, 2007). Major changes in the next 10 years will not necessarily come from psychology of adult development and aging. If there is a treatment for Alzheimer’s disease, it will change later life and the health psychology of aging. If not, we will be faced with larger and larger numbers of dependent older persons with tremendous needs for long-term care, and requirements for caregiving will escalate. We already know that the demographics of this group are fixed—the first Boomers will be 65 in 2011, and 20 years away from the massive increase in incident dementia at age 85 and beyond. It is unlikely that health disparities and income disparities that interact with age and disability will moderate in the next 10 years (see Crimmins & Hagedorn, 2010).

Furthermore, genetic hypotheses will continue to accumulate, as will critical genetic data—but 10 years is too soon for this important area to revolutionize aging (see Kaiser, 2011) on the 10th anniversary of the Human Genome project. At this point in time, major issues remain about sharing genetic information with patients, parents, and subjects in research studies (Couzin-Frankel, 2011). Depending on one’s place in the age and status hierarchy continuity, the next 10 years may not be a problem for those middle-aged and older. However, as we are now seeing changes in the meaning of adulthood, it is more likely that there will be major shifts for younger generations, who will find adult development and aging different from the way it is experienced presently.

REFERENCES

- American Heart Association. (2010). *Heart disease and stroke statistics—2009 update*. Dallas, TX: American Heart Association.
- Anderson, G. F. (2003). Physician, public, and policymaker perspectives on chronic conditions. *Archives of Internal Medicine*, *163*, 437–442.
- Anderson, G. (2004). Chronic conditions: Making the case for ongoing care. Baltimore, MD: Johns Hopkins University. Retrieved from www.rwjf.org/reports/npreports/betterlives.htm
- Arnett, J. J., & Tanner, L. J. (Eds.). (2006). *Emerging adults in America: Coming of age in the 21st century*. Washington, DC: APA Press.
- Arnett, J. J. (2010). *Emerging adulthood*. Oxford University Press.
- Arnold, J., Dai, J., Nahapetyan, L., Arte, A., Johnson, M. A., Hausman, D. B., . . . Poon, L. W. (2010). Predicting successful aging in a population-based sample of Georgia Centenarians. *Current Gerontology and Geriatrics Research*, *1*–9.
- Au, R., Seshadri, S., Wolf, P. A., Elias, M. F., Elias, P. K., Sullivan, L., . . . D'Agostino, R. B. (2004). New norms for a new generation: Cognitive performance in the Framingham Offspring Cohort. *Experimental Aging Research*, *30*, 333–358.
- Balsis, S., Carpenter, B. D., & Storandt, M. (2005). Personality change precedes diagnosis of dementia of the Alzheimer type. *Journal of Gerontology: Psychological Sciences*, *60B*, 98–101.
- Baltes P. B., & Mayer, K. U. (Eds.). (1999). *The Berlin aging study: From 70 to 100*. New York, NY: Cambridge University Press.
- Barefoot, J. C., Mortensen, E. L., Helms, M. J., Avlund, K., & Schroll, M. (2001). A longitudinal study of gender differences in depressive symptoms from age 50 to 80. *Psychology and Aging*, *16*, 342–345.
- Barlow, J., Wright, C., Sheasby, J., Turner, A., & Hainsworth, J. (2002). Self-management approaches for people with chronic conditions: A review. *Patient Education and Counseling*, *48*(2), 177–187.
- Benton, A., & Hamsher, K. (1997). *Multilingual aphasia examination*. Iowa City, IA: University of Iowa.
- Bosworth, H. B., Powers, B. J., & Oddone, E. Z. (2010). Patient self-management support: novel strategies in hypertension and heart disease. *Clinical Cardiology*, *28*(4), 655–663.
- Carstensen, L. L., Mikels, J. A., & Mather, M. (2006). Aging and the intersection of cognition, motivation and emotion. In J. Birren & K. W. Schaie (Eds.), *Handbook of the psychology of aging* (6th ed., pp. 343–362). San Diego, CA: Academic Press.
- Couzin-Frankel, J. (2011). What would you do? *Science*, *331*, 662–665.
- Crimmins, E. M., & Hagedorn, A. (2010). The socioeconomic gradient in healthy life expectancy. In K. E. Whitfield (Ed.), *Annual review of gerontology and geriatrics: Focus on behavioral perspectives on health in later life* (pp. 305–321). New York, NY: Springer.
- Davey, A., Elias, M. F., Siegler, I. C., Lele, U., Martin, P., Johnson, . . . Poon, L. W. (2010). Cognitive function, physical performance, health, and disease: Norms from the Georgia Centenarian Study. *Experimental Aging Research*, *36*, 394–425.
- Davey, A., & Salva, J. (2010). *Statistical power analysis with missing data: A structural equation modeling approach*. Philadelphia, PA: Routledge.
- de la Torre, J. C. (in press). Cardiovascular disease promotes cognitive dysfunction that can signal AD and vascular dementia. In S. Waldstein & M. F. Elias (Eds.), *Neuropsychology of cardiovascular disease* (2nd ed.). Mahwah, NJ: Erlbaum.
- De Meyer, G., Shapiro, F., Vanderstichele, E., Engelborghs, S., De Deyn, P. P., Coart, . . . Trojanowski, J. Q. (2010). Diagnosis-independent biomarker signature in cognitively normal elderly people. *Archives of Neurology*, *67*, 949–956.
- Derks, E. M., Dolan, C. V., & Boomsma, D. I. (2007). Statistical power to detect genetic and environmental influences in the presence of data missing at random. *Twin Research and Human Genetics*, *10*, 159–167.
- Dore, G. A., Elias, M. F., Robbins, M. A., Budge, M. M., & Elias, P. K. (2008). Relation between central adiposity and cognitive function in the Maine-Syracuse Study: Attenuation by physical activity. *Annals of Behavioral Medicine*, *35*, 341–350.
- Dore, G. A., Elias, M. F., Robbins, M. A., Elias, P. K., & Brennan, S. L. (2007). Cognitive performance and age: Norms from the Maine-Syracuse Study. *Experimental Aging Research*, *33*, 205–271.
- Dore, G. A., Elias, M. F., Robbins, M. A., Elias, P. K., & Nagy, Z. (2009). Presence of the *ApoE* $\epsilon 4$ allele modifies the relationship between type 2 diabetes and cognitive performance: The Maine-Syracuse Study. *Diabetologia*, *52*, 2551–2560.
- Duchek, J. M., Balota, D. A., Storandt, M., & Larsen, R. (2007). The power of personality in discriminating between healthy aging and early-stage Alzheimer's disease. *Journal of Gerontology: Psychological Sciences*, *62B*, 353–361.
- Elias, M. F., Beiser, A., Wolf, P. A., Au, R., White, R. F., & D'Agostino, R. B. (2000). The preclinical phase of Alzheimer's disease: A 22-year prospective study of the Framingham cohort. *Archives of Neurology*, *57*, 808–813.
- Elias, M. F., Dore, G. A., Davey, A., Robbins, M. A., & Elias, P. K. (2010). From blood pressure to physical disability: The role of cognition. *Hypertension*, *55*, 1360–1365.
- Elias, M. F., Dore, G. A., Goodell, A., Davey, A., Zilioli, M. K. C., Brennan, S., . . . Robbins, M. A. (in press). Normative data for elderly adults: The Maine-Syracuse Study. *Experimental Aging Research*.
- Elias, M. F., Elias, P. K., Robbins, M. A., Wolf, P. A., & D'Agostino, R. B. (2001). Cardiovascular risk factors and cognitive functioning: An epidemiological perspective. In S. Waldstein & M. F. Elias (Eds.), *Neuropsychology of Cardiovascular Disease* (pp. 83–105). Mahwah, NJ: Erlbaum.
- Elias, M. F., Elias, P. K., Sullivan, L. M., Wolf, P. A., & D'Agostino, R. B. (2003). Lower cognitive function in the presence of obesity and hypertension: The Framingham heart study. *International Journal of Obesity and Related Metabolic Disorders*, *27*, 260–268.
- Elias, M. F., Goodell, A. L., & Waldstein, S. R. (in press). Obesity, cognitive functioning and dementia: Back to the future. *Journal of Alzheimer's Disease*, *28*, 1–13.
- Elias, M. F., Robbins, M. A., Budge, M. M., Elias, P. K., Hermann, B. A., & Dore, G. A. (2004). Studies of aging, hypertension and cognitive functioning: With contributions from the Maine-Syracuse Study. In P. T. Costa & I. C. Siegler (Vol. Eds.), *Recent advances in psychology and aging* (pp. 89–131). Vol. 14 in M. P. Mattson (Series Ed.), *Advances in cell aging and gerontology*. Amsterdam, Netherlands: Elsevier.
- Elias, M. F., Robbins, M. A., Elias, P. K., & Streeten, D. H. (1998). A longitudinal study of blood pressure in relation to performance on the Wechsler Adult Intelligence Scale. *Health Psychology*, *17*, 486–493.
- Elias, M. F., Sullivan, L. M., D'Agostino, R. B., Elias, P. K., Beiser, A., Au, R., . . . Wolf, P. A. (2004). The Framingham stroke risk profile and lowered cognitive performance. *Stroke*, *35*, 404–409.
- Elias, P. K., Elias, M. F., Robbins, M. A., & Budge, M. M. (2004). Blood pressure-related cognitive decline: Does age make a difference? *Hypertension*, *44*, 631–636.
- Evert, J., Lawler, H., Bogan, H & Perls, T. (2003) Morbidity profiles of centenarians: Survivors, escapers and delayers. *Journals of Gerontology: Series A*, *58*, 232–237.
- Fillenbaum, G. (1988). *Multidimensional functional assessment of older adults*. Hillsdale, NJ: Erlbaum.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Minimal state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189–198.
- Fried, L. P., Kronmal, R. A., Newman, A. B., Bild, D. E., Mittelmark, M. B., Polak, J. F., . . . Gardin, J. M. (1998). Risk factors for 5-year