

Social Structures, Aging, and Self-Regulation in the Elderly



Editors

**K. Warner Schaie
Laura L. Carstensen**

Social Structures, Aging, and Self-Regulation in the Elderly

Edited by
K. Warner Schaie, PhD
Laura L. Carstensen, PhD



Societal Impact on Aging Series

SPRINGER  **PUBLISHING COMPANY**
New York

Societal Impact on Aging Series

*K. Warner Schaie, PhD, Evan Pugh Professor of Human Development and Psychology College of Health and Human Development, Series Editor
The Pennsylvania State University, University Park, PA*

- 2006 Social Structures, Aging, and Self-Regulation in the Elderly**
K. Warner Schaie and Laura L. Carstensen, Editors
- 2005 Historical Influences on Lives and Aging**
K. Warner Schaie and Glen H. Elder, Jr., Editors
- 2004 Religious Influences on Health and Well Being in the Elderly**
K. Warner Schaie, Neal Krause, and Alan Booth, Editors
- 2003 Impact of Technology on Successful Aging**
Neil Charness and K. Warner Schaie, Editors
- 2002 Personal Control in Social and Life Course Contexts**
Steven H. Zarit, Leonard I. Pearlin, and K. Warner Schaie, Editors
- 2002 Effective Health Behavior in Older Adults**
K. Warner Schaie, Howard Leventhal, and Sherry L. Willis, Editors
- 2000 The Evolution of the Aging Self: The Societal Impact on the Aging Process**
K. Warner Schaie and Jon Hendricks, Editors
- 2000 Mobility and Transportation in the Elderly**
K. Warner Schaie and Martin Pietrucha, Editors
- 1997 Impact of Work on Older Adults**
K. Warner Schaie and Carmi Schooley, Editors
- 1997 Societal Mechanisms for Maintaining Competence in Old Age**
Sherry L. Willis, K. Warner Schaie, and Mark Hayward, Editors
- 1996 Older Adults' Decision-Making and the Law**
Michael Smye, K. Warner Schaie, and Marshall B. Kapp, Editors
- 1995 Adult Intergenerational Relations: Effects of Societal Change**
Vern L. Bengtson, K. Warner Schaie, and Linda K. Burton, Editors
- 1993 Societal Impact on Aging: Historical Perspectives**
K. Warner Schaie and W. Andrew Achenbaum, Editors

About the Authors

K. Warner Schaie, Ph.D., is the Evan Pugh Professor of Human Development and Psychology at the Pennsylvania State University. He also holds an appointment as Affiliate Professor of Psychiatry and Behavioral Science at the University of Washington. He received his PhD in psychology from the University of Washington, an honorary Dr. phil. from the Friedrich-Schiller University of Jena, Germany, and an honorary ScD degree from West Virginia University. He received the Kleemeier Award for Distinguished Research Contributions from the Gerontological Society of America, the MENSA lifetime career award, and the Distinguished Scientific Contributions award from the American Psychological Association. He is author or editor of 48 books including the textbook *Adult Development and Aging* (5th edition, with S. L. Willis) and the *Handbook of the Psychology of Aging* (6th edition, with J. E. Birren). He has directed the Seattle Longitudinal Study of Cognitive Aging since 1956, and is the author of more than 275 journal articles and chapters on the psychology of aging. His current research interest is the life course of adult intelligence, its antecedents and modifiability, the early detection of risk for dementia, as well as methodological issues in the developmental sciences.

Laura L. Carstensen, Ph.D., is Professor of Psychology at Stanford University, where she is also Chair of the Psychology Department. She has published extensively about emotional, cognitive, and motivational changes with age and formulated socioemotional selectivity theory. She is a fellow in the American Psychological Society, American Psychological Association, and the Gerontological Society of America. Dr. Carstensen received the Kalish Award for Innovative Research from the Gerontological Society in 1993, and was selected as a Guggenheim Fellow in 2003.

Copyright © 2006 Springer Publishing Company, Inc.

All rights reserved.

No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without the prior permission of Springer Publishing Company, Inc.

Springer Publishing Company, Inc.
11 West 42nd Street
New York, NY 10036

Acquisitions Editor: Sheri W. Sussman
Managing Editor: Mary Ann McLaughlin
Production Editor: Emily Johnston
Cover design by Joanne E. Honigman
Composition: Apex Covantage

06 07 08 09 10/ 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data

Laura L Carstensen

Social structures, aging and self-regulation in the elderly/edited by K. Warner Schaie,

Laura L Carstensen

p. cm.— (Societal impact on aging)

Edited conference papers.

Includes bibliographical references and index.

1. Aging—Social aspects—Congresses. 2. Cognition in old age—Congresses.

I. Schaie, K. Warner (Klaus Warner), 1928– II. Carstensen, Laura L.

III. Pennsylvania State University. Gerontology Center. IV. Series.

HQ1061.S6486 2006

305.26—dc22

2006012820

Printed in the United States of America by Edwards Brothers.

Contents

| | |
|--|------------|
| <i>Contributors</i> | <i>vii</i> |
| <i>Preface</i> | <i>ix</i> |
| 1. Societal Influences That Affect Cognitive Functioning in Old Age | 1 |
| <i>Selam Negash and Ronald C. Petersen</i> | |
| Commentary: Societal Factors in Cognitive Aging: One Eye Wide Shut? | 9 |
| <i>Naftali Raz</i> | |
| Commentary: Societal Influences on Cognition in Historical Context | 13 |
| <i>K. Warner Schaie</i> | |
| 2. Wisdom in Social Context | 33 |
| <i>Ursula M. Staudinger, Eva-Marie Kessler, and Jessica Dörner</i> | |
| Commentary: The Dynamic Relationship Between Age Stereotypes and Wisdom in Old Age | 55 |
| <i>Becca R. Levy and Corey Pilver</i> | |
| 3. Social Influences on Adult Personality, Self-Regulation, and Health | 69 |
| <i>Daniel K. Mroczek, Avron Spiro III, Paul W. Griffin, and Shevaun D. Neupert</i> | |
| Commentary: From Kindling to Conflagration: Self-Regulation and Personality Change | 85 |
| <i>Brent W. Roberts</i> | |

| | |
|--|-----|
| Commentary: Can Self-Regulation Explain Age Differences in Daily, Weekly, and Monthly Reports of Psychological Distress? | 95 |
| <i>David M. Almeida, Daniel K. Mroczek, and Michelle Neiss</i> | |
| 4. Social Norms, Rules of Thumb, and Retirement: Evidence for Rationality in Retirement Planning | 123 |
| <i>Gary Burtless</i> | |
| Commentary: Modeling the Retirement Decision | 161 |
| <i>Joseph F. Quinn</i> | |
| Commentary: Numbers Are Just Numbers | 175 |
| <i>Ellen Peters</i> | |
| 5. Race and Self-Regulatory Health Behaviors: The Role of the Stress Response and the HPA Axis in Physical and Mental Health Disparities | 189 |
| <i>James S. Jackson and Katherine M. Knight</i> | |
| Commentary: Mechanisms of Health Disparities: Variations in Health Care Provider Attitudes and Their Impact on Older Minority Adults | 209 |
| <i>Stephanie L. Garrett and Toni P. Miles</i> | |
| Commentary: Adding Paths to Resilience and Daily Accounts to an Already Rich Field of Inquiry: A Brief Commentary on James Jackson's "Social Structure and Health Disparities" | 215 |
| <i>Alex J. Zautra, Kate E. Murray, and Brendt P. Parish</i> | |
| <i>Index</i> | 240 |

Contributors

David M. Almeida, PhD

The Pennsylvania State University
State College, PA

Gary Burtless, PhD

The Brookings Institution
Washington, DC

Jessica Dörner, PhD

Jacobs Center for Lifelong
Learning and Institutional
Development
International University Bremen
Bremen, Germany

Stephanie L. Garrett, MD

University of Louisville
Louisville, KY

Paul W. Griffin, PhD

Pace University
Pleasantville, NY

James S. Jackson, PhD

Institute for Social Research
University of Michigan
Ann Arbor, MI

Eva-Marie Kessler, PhD

Jacobs Center for Lifelong
Learning and Institutional

Development

International University Bremen
Bremen, Germany

Katherine M. Knight

Institute for Social Research
University of Michigan
Ann Arbor, MI

Becca R. Levy, PhD

Yale University
New Haven, CT

Toni P. Miles, MD, PhD

Wise-Nelson Chair, Clinical
Geriatrics Research
Professor, Family & Geriatric
Medicine
University of Louisville
Louisville, KY

Daniel K. Mroczek, PhD

Purdue University
West Lafayette, IN

Kate E. Murray, M.S.

Arizona State University
Tempe, AZ

Salem Negash, PhD

Alzheimer's Disease Research

Center
Mayo Clinic College of Medicine
Rochester, MN

Michelle Neiss, PhD
Oregon Health and Science
University
Portland, OR

Shevaun D. Neupert, PhD
North Carolina State University
Raleigh, NC

Brendt P. Parish
Arizona State University
Tempe, AZ

Ellen Peters, PhD
Decision Research
University of Oregon
Eugene, OR

Ronald C. Petersen, PhD, MD
Alzheimer's Disease Research
Center
Mayo Clinic College of Medicine
Rochester, MN

Corey Pilver, PhD
Yale University
New Haven, CT

Joseph F. Quinn, PhD
Boston College
Chestnut Hill, MA

Naftali Raz, PhD
Institute of Gerontology
Department of Psychology
Wayne State University
Detroit, MI

Brent W. Roberts, PhD
University of Illinois
Urbana-Champaign
Champaign, IL

Avron Spiro III, PhD
Boston VA Health System &
Boston University School of Public
Health
Boston, MA

Ursula M. Staudinger, PhD
Jacobs Center for Lifelong
Learning and Institutional
Development
International University Bremen
Bremen, Germany

Alex J. Zautra, PhD
Arizona State University
Tempe, AZ

Preface

This is the 18th volume in a series on the broad topic of “Societal Impact on Aging.” The first five volumes of this series were published by Erlbaum Associates under the series title of “Social Structure and Aging.” The present volume is the 13th published under the Springer Publishing Company imprint. It is the edited proceedings of a conference held at the Pennsylvania State University, October 4–5, 2004.

The series of Penn State Gerontology Center conferences originated from the deliberations of a subcommittee of the Committee on Life Course Perspectives of the Social Science Research Council, chaired by Matilda White Riley in the early 1980s. That subcommittee was charged with developing an agenda and mechanisms that would serve to encourage communication between scientists who study societal structures that might affect the aging of individuals and those scientists who are concerned with the possible effects of contextual influences on individual aging. The committee proposed a series of conferences that would systematically explore the interfaces between social structures and behavior, and in particular to identify mechanisms through which society influences adult development. When the first editor was named director of the Penn State Gerontology Center in 1985, he was able to implement this conference program as one of the center’s major activities.

The previous seventeen volumes in this series have dealt with the societal impact on aging in psychological processes (Schaie & Schooler, 1989); age structuring in comparative perspective (Kertzer & Schaie, 1989); self-directedness and efficacy over the life span (Rodin, Schooler, & Schaie, 1991); aging, health behaviors, and health outcomes (Schaie, House, & Blazer, 1992); caregiving in families (Zarit, Pearlin, & Schaie, 1993); aging in historical perspective (Schaie & Achenbaum, 1993); adult intergenerational relations (Bengtson, Schaie, & Burton, 1995); older adults’ decision making and the law (Smyer, Schaie, & Kapp, 1996); the impact of social structures on decision making in the elderly (Willis, Schaie, &

Hayward, 1997); the impact of the workplace on aging (Schaie & Schooler, 1998); the evolution of the aging self (Schaie & Hendricks, 2000); mobility and transportation in the elderly (Schaie & Pietrucha, 2000); societal impact on health behavior in the elderly (Schaie, Leventhal, & Willis, 2002); mastery and control in the elderly (Zarit, Pearlin, & Schaie, 2002); impact of technology on the elderly (Charness & Schaie, 2003); religious influences on health and well being in the elderly (Schaie, Krause, & Booth, 2004); and historical influences on lives and aging (Schaie & Elder, 2005).

The strategy for each of these volumes has been to commission reviews on three major topics by established subject-matter specialists who have credibility in aging research. We then invited two formal discussants for each chapter—usually one drawn from the writer's discipline and one from a neighboring discipline. This format seems to provide a suitable antidote against the perpetuation of parochial orthodoxies as well as to make certain that questions are raised in regard to the validity of iconoclastic departures in new directions.

To focus each conference, the organizers chose three aspects of the conference topic that are of broad interest to gerontologists. Social and behavioral scientists with a demonstrated track record were then selected and asked to interact with those interested in theory building within a multidisciplinary context.

The present volume focuses on the interplay of social structures and self-regulation in the elderly. No one doubts that the social contexts in which individuals develop exert strong influence on life trajectories. Those born into environments that provide high quality education, supportive social relations, and economic assets do better in old age than those born into environments bereft of the same resources. However, the extent of this influence is only beginning to be revealed. Recent research shows that life experiences influence basic brain structures (e.g., the influence of musical training on neural organization) and functions (e.g., inflammatory processes), and that social embeddedness may even protect against Alzheimer's disease. Similarly, education increasingly appears to have a real effect on neural integrity. Thus, societal contexts may not simply open or close doors for individuals; they may influence self-regulatory processes at the most basic levels of functioning.

Although social structures are generally thought of as the independent variables that affect individual aging, it is also possible to think of a lifetime development of self-regulatory processes leading to behaviors in old

age that can have impact on and modify societal structures. Hence, we first consider self-regulation as the dependent variable, asking how social contexts influence cognitive, emotional, and self-regulatory processes. We then reverse the question, by treating self-regulation as the independent variable and consequences for retirement and physical health as dependent variables. This approach allows us to consider the question as to how the effectiveness of self-regulation influences physical and economic outcomes in old age.

The first topic in this volume is concerned with the complex bidirectional influences between brain and environment. In particular, consideration is given to the effects of lifelong experiences as they affect cognitive functioning in old age. The major review lists a variety of societal contexts and in particular focuses on the protective role of education. Two commentaries caution, first, that many individuals who profit from favorable environments seek out or are selected into such environments because of genetic predisposition, and second, that the environmental impact may differ across historical contexts and in subsets of the population studied.

The second topic is concerned with individual resilience in societal context. A major impact of societal context on individual aging is through providing the experiences necessary for learning how to navigate one's life, adapting to the complex societal and cultural rules and mores within which the individual life course progresses. It is well known that the attainment of wisdom requires not only high levels of cognitive competence, but also the successful experience of many complex life situations: a lifelong process. However, experiential learning not only affects problem-solving behavior, it also affects the development of life-stage-appropriate identity and self-schemas that affect adult personality organization. It is also likely to affect personality processes such as assimilation and accommodation in old age. While the first review chapter on this topic considers the acquisition of wisdom in societal context, the second directly addresses the role of self-regulation as an important personality construct.

The third topic addresses the physical and economic outcomes of effective self-regulation across the adult lifespan. The issues and literature on certain behaviors that may have major impact on major societal structures are explored. The position is taken that successful aging results from effective self-regulation over the lifespan; that is, functional status in old age reflects, in large part, the lifelong culmination of economic decisions, health behaviors, and health care utilization. It also reflects social stand-

ing and the favorable or adverse consequences that social status affords. Specifically, sample cases examined in this chapter are the effects of self-regulation on the timing of retirement and the effects of self-regulation upon health-related behaviors in different ethnic communities and social classes.

We are grateful for the financial support of the conference provided by conference grant R13 AG 09787 from the National Institute on Aging that led to this volume, and for the additional support from the College of Health and Human Development of the Pennsylvania State University. We are also grateful to Chriss Schultz for handling the conference logistics, and to Jenifer Hoffman for coordinating the manuscript preparation and preparing the indexes.

K. Warner Schaie
January, 2006

REFERENCES

- Bengtson, V. L., Schaie, K. W., & Burton, L. (Eds.). (1995). *Adult inter-generational relations: Effects of societal changes*. New York: Springer Publishing Co.
- Charness, N., & Schaie, K. W. (Eds.). (2003). *Impact of technology on the aging individual*. New York: Springer Publishing Co.
- Kertzner, D., & Schaie, K. W. (Eds.). (1989). *Age structuring in comparative perspective*. Hillsdale, NJ: Erlbaum.
- Rodin, J., Schooler, C., & Schaie, K. W. (Eds.). (1991). *Self-directedness and efficacy: Causes and effects throughout the life course*. Hillsdale, NJ: Erlbaum.
- Schaie, K. W., & Achenbaum, W. A. (Eds.). (1993). *Societal impact on aging: Historical perspectives*. New York: Springer Publishing Co.
- Schaie, K. W., & Elder, G. H., Jr. (Eds.). (2005). *Historical influences on lives and aging*. New York: Springer Publishing Co.
- Schaie, K. W., & Hendricks, J. (Eds.). (2000). *Evolution of the aging self: Societal impacts*. New York: Springer Publishing Co.
- Schaie, K. W., House, J., & Blazer, D. (Eds.). (1992). *Aging, health behaviors, and health outcomes*. Hillsdale, NJ: Erlbaum.

- Schaie, K. W., Krause, N., & Booth, A. (Eds.). (2004). *Religious influences on health and well-being in the elderly*. New York: Springer Publishing Co.
- Schaie, K. W., Leventhal, H., & Willis, S. L. (Eds.). (2002). *Societal impacts on health behaviors in the elderly*. New York: Springer Publishing Co.
- Schaie, K. W., & Pietrucha, M. (Eds.). (2000). *Mobility and transportation in the elderly*. New York: Springer Publishing Co.
- Schaie, K. W., & Schooler, C. E. (Eds.). (1989). *Social structure and aging: Psychological processes*. Hillsdale, NJ: Erlbaum.
- Schaie, K. W., & Schooler, C. E. (Eds.). (1998). *Impact of the work place on older persons*. New York: Springer Publishing Co.
- Smyer, M., Schaie, K. W., & Kapp, M. B. (Eds.). (1996). *Older adults' decision-making and the law*. New York: Springer Publishing Co.
- Willis, S. L., Schaie, K. W., & Hayward, M. (Eds.). (1997). *Impact of social structures on decision making in the elderly*. New York: Springer Publishing Co.
- Zarit, S. H., Pearlin, L., & Schaie, K. W. (Eds.). (1993). *Social structure and caregiving: Family and cross-national perspectives*. Hillsdale, NJ: Erlbaum.
- Zarit, S. H., Pearlin, L., & Schaie, K. W. (Eds.). (2002). *Mastery and control in the elderly*. New York: Springer Publishing Co.

1

Societal Influences That Affect Cognitive Functioning in Old Age

Selam Negash and Ronald C. Petersen

INTRODUCTION

With the increasing of the population of older adults, there is a growing interest in improving quality of life in old age, and in early detection and prevention of cognitive declines with aging. One important aspect of this endeavor pertains to achieving and maintaining cognitive vitality, which is shown to be essential to quality of life and survival in old age (Fillit et al., 2002). Although it is well documented that aging is accompanied by a decline in several domains of cognitive functions, studies have also shown that such a decline is not common to all older people, and that some older adults can in fact enjoy optimal aging without experiencing a significant cognitive impairment. For example, in successful aging, where individuals function effectively and successfully as they age, there is likely very little cognitive decline over the life span, and cognitive function can be preserved well into the 10th decade or beyond (Kaye et al., 1994). Further, even in typical aging, where individuals encounter co-morbidities such as hypertension and coronary artery disease as part of aging, it has generally been observed that some cognitive functions such as those involving crystallized knowledge (e.g., vocabulary) remain preserved or even improve with age (Lindenberger & Baltes, 1997).

Researchers over the past several decades have been investigating whether there are factors, intrinsic or extrinsic, that would allow some individuals to age successfully while others experience the consequences of normal aging or even pathological forms of aging such as Alzheimer's disease (AD). The literature in the field implicates an association between

cognitive decline and factors such as educational and occupational attainment, cognitive and physical activity, and nutrition (Fratiglioni, Paillard-Borg, & Winblad, 2004). The present chapter discusses some of these lifestyle factors that are thought to influence cognitive vitality in normal aging and dementia, and also the mechanisms that are speculated to underlie this relationship.

Terminology

In discussing cognitive declines with aging, it is important to consider some of the terms that have been used over the years to describe the different degrees of cognitive impairment associated with aging. Historically, attempts at characterizing cognitive changes at the normal tail end of the continuum have produced several terms such as benign senescent forgetfulness (Kral, 1962), age-associated memory impairment (AAMI) (Crook et al., 1986), and age-associated cognitive decline (AACD) (Levy, 1994). These terms are meant to reflect the extremes of normal aging in general. Recently, increased attention is being given to the boundary between normal aging and very early AD, and this transitional zone has been termed mild cognitive impairment (MCI) (Petersen et al., 1999). MCI represents a condition where individuals show memory impairment greater than expected for their age, but otherwise are functioning well and do not meet the commonly accepted criteria for dementia. The construct of MCI has become recognized as a pathological condition rather than a manifestation of normal aging, and it has recently received a great deal of attention as a useful entity for possible therapeutic intervention.

The most typical scenario of MCI pertains to the amnesic subtype (a-MCI), where individuals show impairment in a single domain of memory with other cognitive domains remaining relatively intact. More recently, as the field of MCI has advanced, it has become apparent that other clinical subtypes exist as well (Petersen, 2003). *Multiple domain amnesic MCI* pertains to individuals who, in addition to memory deficit, also have impairments in at least one other cognitive domain such as language, executive function, or visuospatial skills. *Multiple domain non-amnesic MCI*, on the other hand, pertains to individuals who have impairments in multiple cognitive domains, but not including memory. *Single domain non-amnesic MCI*, which is the least common subtype,

pertains to individuals with impairment in a single non-memory domain of language, executive function, or visuospatial skills. Other cognitive domains, including memory, are essentially normal. Individuals in this subtype likely have a different outcome from those with memory impairment. It is also imperative that individuals in all of these subtypes of MCI have minimal impairments in functional activities that do not represent a significant change in function from a prior level, and also do not meet the criteria for dementia. Clinical subtypes of MCI have been proposed to broaden the concept and include prodromal forms of a variety of dementias. As the field matures, and as we learn more about the various subtypes of MCI and their ability to predict various forms of cognitive impairment, these could lay the groundwork for therapeutic interventions that are tailored for specific prodromal forms of dementia.

LIFESTYLE FACTORS

In the following section, we summarize the main findings from studies that have examined the associations between cognitive functioning in old age and lifestyle factors, such as attainment level, cognitive and physical activity, and nutrition. The link between these factors and the risk for dementia will also be briefly discussed.

Educational and Occupational Attainment

Several studies have reported an association between cognitive decline late in life and educational and occupational attainment. Cross-sectional and longitudinal data show that individuals with low levels of education perform poorly on tests of cognitive function and exhibit greater decline over time compared to those with higher education levels (Evans et al., 1993; Farmer, Kittner, Rae, Bartko, & Regier, 1995). Low levels of educational attainment have also been linked to increased risk of dementia. Prevalence studies conducted in various parts of the world have shown that the incidence and prevalence of AD was higher in individuals with low levels of education (Katzman, 1993; Stern et al., 1994; Zhang et al., 1990; Callahan et al., 1996). There is also evidence that education modifies the relation of AD pathology to cognitive functions; the relation of senile plaques and level of cognitive function has been shown to differ by years of education

(Bennett et al., 2003). Similarly, data derived from autobiographies of sisters participating in the Nun Study have shown that poor linguistic ability (as measured by idea density and grammatical complexity) in early life was a strong predictor of poor cognitive function and AD in late life (Snowdon et al., 1996).

The link between occupational attainment and AD has also been documented. Many studies have shown that the incidence of AD was higher in individuals with low levels of occupational attainment (Callahan et al., 1996; Dartigues et al., 1992), although there have been some exceptions (Beard, Kokmen, Offord, & Kurland, 1992). Studies have also found a relationship between occupational demands and cognitive decline, where individuals in occupations involving lower mental demands and greater use of manual skills showed poor cognitive performance and higher prevalence of dementia (Jorm et al., 1998). Similarly, AD patients have been shown to engage in significantly lower mental occupational demands and significantly higher physical occupational demands compared to control participants (Smyth et al., 2004).

The mechanisms underlying the above associations, however, are not fully understood. Nevertheless, some plausible explanations have been put forth by several studies. Katzman (1993) has proposed that the protective effect of education against cognitive decline is due to a neuronal reserve, where higher educational attainment increases synaptic density, allowing individuals to delay the onset of symptoms of cognitive impairment. The extensive work in animal studies also appears to support this hypothesis in that it shows brain weight and numbers of neuronal processes in rodents are related to environmental stimulation (Kempermann, Kuhn, & Gage, 1998; Sirevaag, Black, Shafron, & Greenough, 1998). However, the extent to which such findings can be extrapolated to humans is unclear. Another support for this view comes from imaging studies, which have shown prominent regional cerebral blood flow deficits, suggestive of more advanced pathology, in AD patients with higher educational and occupational attainment (Stern, Alexander, Prohovnik, & Mayeux, 1992; Stern et al., 1995). The authors in these studies concluded that individuals with more reserve are able to tolerate more AD pathology, and that at any level of clinical severity, the underlying pathology is more advanced in patients with higher levels of attainment. Consistently, they have proposed that increased educational and occupational attainment may reduce the risk of AD, either by decreasing ease of clinical detection or by supplying a reserve that allows

individuals to cope longer before AD is clinically expressed. However, as proponents of the above view also note, the concept of a reserve must be weighted against the alternate explanation of a detection bias. That is, it is possible that low educational or occupational attainment enables the clinician to diagnose dementia at an earlier point in time, in which case the attainment effect would confound the diagnostic criteria (Katzman, 1993). Although this remains a possibility, the above findings have also been replicated in cases involving a more careful application of the diagnostic criteria in order to reduce the likelihood of a spurious effect attainment level, thus suggesting that links between higher incidence of AD and low levels of educational or occupational attainment are not simply a result of detection bias (Stern et al., 1994). In general, then, the protective effects of educational and occupational attainment against cognitive decline late in life appear to be supported by the literature.

Cognitive Activity

Recent research indicates that frequent participation in cognitively stimulating activities may also protect against cognitive decline and reduce the risk of AD (Hultsch, Hertzog, Small, & Dixon, 1999; Wilson et al., 2002). The construct of cognitive activity is generally operationalized in the literature through use of a number of scales that measure frequency of cognitive activities that are judged to primarily involve seeing or processing information (Wilson & Bennett, 2003). These may include reading a book, playing a game such as chess or crosswords, or listening to a radio program. Studies that have examined the relationship between cognitive activity and cognitive decline in old age have found that individuals who participate in cognitive activity and are engaged in their environment show the least decline in cognitive function compared to those with disengaged lifestyles (Hultsch et al., 1999; Schaie, 1984). Engaging in cognitively stimulating activities during childhood has also been associated with a higher cognitive function in old age (Everson-Rose, Mendes de Leon, Bienias, Wilson, & Evans, 2003). In addition, cognitive activity has been hypothesized to be associated with incidence of AD. Several studies have found that individuals who reported lower participation in cognitively stimulating activities were at a higher risk of developing AD compared to those reporting frequent cognitive activity

(Scarmeas, Levy, Tang, Manly, & Stern, 2001; Wang, Karp, Winblad, & Fratiglioni, 2002; Wilson et al., 2002).

The mechanisms that might account for the association between cognitive activity and risk of AD are also unknown. One hypothesis, similar to that mentioned previously for the attainment level, is that cognitive activity contributes to the development and maintenance of neural systems that underlie cognitive processes, thereby requiring more AD pathology before these processes are disrupted and the disease is expressed clinically (Wilson & Bennett, 2003). The considerable data from animal studies appear to be consistent with this view, and indicate that environmental enrichments influence increased neurons, synapses, and dendritic arbors in several brain regions including the hippocampus and cerebellum (Kempermann, Kuhn, & Gage, 1997; Kleim, Vij, Ballard, & Greenough, 1997).

Physical Activity and Nutrition

There is also evidence that cognitive function in old age is associated with physical exercise and nutrition. For example, animal model studies have demonstrated that physical exercise, such as access to a running wheel, enhanced neurogenesis and improved performance on learning and memory tasks in adult mice (Praag, Kempermann, & Gage, 1999). The results from human studies, on the other hand, have been mixed. While several studies report exercise-related benefits such as improvements in cognitive function (Weuve et al., 2004) and reduced risk of dementia (Abbott et al., 2004), others studies have not. For example, cross-sectional data have shown that individuals who had engaged in aerobic exercise performed better than non-exercisers on several tasks, such as reasoning, working memory, and vigilance monitoring (Bunce, Barrowclough, & Morris, 1996; Powell & Pohndorf, 1971). Other studies, however, have not found such association (Madden, Blumenthal, Allen, & Emery, 1989). Some epidemiological studies have also reported a link between physical exercise and reduced risk of dementia (Abbott et al., 2004; Laurin, Verreault, Lindsay, MacPherson, & Rockwood, 2001), while others have not (Wilson et al., 2002). The inconsistent findings among these studies may have resulted from several factors such as differences in the types of exercise and aspects of cognition examined by the different studies. Nonetheless, it appears generally that the animal and human data support some beneficial effect of physical activity on cognitive functioning in old age.

Likewise, the relationship between nutrition and cognitive function late in life has been investigated by various studies in several research areas. Some animal studies have found that caloric restriction may have beneficial effects on cognition in old age (Mattson, Duan, Lee, & Guo, 2001). Other studies, however, have not found an association (Bellush, Wright, Walker, Kopchick, & Colvin, 1996), and as such, the results with regards to caloric restriction have been mixed. Further, caloric restriction is not generally recommended in older adults because it may promote malnutrition, which could cause long-term cognitive impairments. Studies have also examined the relationship between dietary fat consumption and cognitive decline, and have found that high consumption of total fats, saturated fatty acids, and cholesterol was associated with cognitive decline in elderly people (Ortega et al., 1996), while consumption of fish and n-3 fatty acids correlated with reduced risk of AD (Morris et al., 2003). Further, some studies have also reported an association between higher serum levels of cholesterol and increased risk of dementia (Notkola et al., 1998), although others have not (Engelhart et al., 2002). More research is needed in this area to understand the relationship between diet and risk of dementia.

Another area of investigation that has gained increased interest, especially in recent years as the focus of AD research moves toward prevention, is the effect antioxidants may have against cognitive decline in aging. Antioxidants such as Vitamins E and C help neutralize free radicals that can cause oxidant injury, and they have been associated with various benefits such as promoting cardiovascular health (Frei, 1995). Vitamin E has also recently gained attention in the clinical trials for MCI. A recent study involving Vitamin E and donepezil, however, has indicated no therapeutic effect for Vitamin E while donepezil reduced the risk of progressing to AD for the first 12 months of the trial (Petersen et al., 2005). Further investigation is needed in this area, and additional work is likely to continue.

SUMMARY

In general, then, the literature indicates a strong association between various lifestyle factors and cognitive functioning late in life, although some studies have not found such associations. Some of this variability may be the result of several factors, including differences in the design of studies (e.g., longitudinal versus cross-sectional), the operational definitions of variables under consideration, and the types of assessments of cognitive

performance (Fratiglioni et al., 2004). Nonetheless, despite such limitations, the literature provides enough evidence to support the hypothesis that an active and socially integrated lifestyle influences cognitive vitality in old age. A lifestyle such as this may also serve to buffer against cognitive decline and perhaps aid in delaying the diagnosis of dementia. It remains unclear, however, what mechanisms underlie these associations, and whether this effect can be causal. It is possible that the aforementioned associations are markers of innate characteristics such as genetic background. Indeed, the link between genetic markers such as apolipoprotein E (apoE) and risk of dementia has been established, in that individuals with an E4 allele were found to be at increased risk for AD (Corder et al., 1993; Strittmatter et al., 1993). The presence of an E4 allele in MCI patients as a predictor of progression to AD has also been reported (Petersen et al., 1995). However, not all persons with the E4 allele develop dementia, and this underscores the likelihood that the E4 allele is a risk factor that interacts with other factors to cause dementia. As such, these observations stress the importance of genetic and environmental interactions in influencing cognitive vitality late in life, and highlight the importance of gaining an understanding of these interactions for early detection and prevention of cognitive decline and dementia.

ACKNOWLEDGMENTS

Supported by the National Institute of Aging Alzheimer's Disease Patient Registry, AG 06576 and Alzheimer's Disease Research Center, AG 16574.

Commentary: Societal Factors in Cognitive Aging: One Eye Wide Shut?

Naftali Raz

The process of aging is marked by significant individual variability, the sources of which are multiple and, by and large, ill-defined. Nonetheless, as longitudinal studies of brain and cognition show, individual differences may be quite stable and are transferred reliably along the path of late life development (Deary et al., 2004; Hertzog & Schaie, 1986; Raz et al., 2005). In an attempt to explicate some reasons for variability in trajectories of aging, Negash and Petersen turn their attention to the factors that until recently have been underestimated in biomedical and cognitive gerontology. Specifically, they present a brief review of the evidence pertaining to the role of educational and occupational attainment, cognitive and physical activity, and nutrition in cognitive decline and stability. They conclude, on the balance of the surveyed evidence, that all of the listed factors exert significant influence on the individual paths of aging and affect the likelihood of crossing a diagnostic line between normal aging and dementia. Although they mention the role of a specific genotypic variation (ApoE-E4 allele) in cognitive decline, the authors assume that all of the factors considered in their brief survey are societal in nature.

On the surface this seems true. However, there may be valid reasons to doubt the exclusivity and even the primacy of society in the influences discussed by Negash and Petersen. The aim of the following brief commentary is to broaden the Negash and Petersen argument for paying attention to the aforementioned so-called societal factors by highlighting the non-societal aspects that fall beyond the scope of their review. More specifically, I will argue that social and cultural influences notwithstanding, a significant portion of the variance in the modifiers of cognitive aging identified by the authors can be attributed to genetic sources that reside

outside the realm of society and family environment, and need to be taken into account in determining the mechanisms of cognitive aging.

Education is the first of the societal factors Negash and Petersen mention. Indeed, an institutionalized education process is implemented within the confines of culture and society and is therefore a good candidate for a non-biological modifier of aging. However, population genetics studies present evidence that complicates that expectation. A significant genetic component for educational achievement was found in large European cohorts (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Heath et al., 1985). Research of the past several decades has established that whatever intelligence quotient (IQ) may reflect, and no matter how malleable it may appear, educational achievement is heritable to a substantial degree (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). Regardless to the discussion of causality, educational attainment is linked to IQ and cognitive performance in the general population and in older adults (Pedersen, Plomin, Nesselroade, & McClearn, 1992; Pedersen, Reynolds, & Gatz, 1996). Moreover, the association between achievement and IQ is also influenced by genotype (Bartels et al., 2002; Heath et al., 1985), and age-related changes in heritability of cognitive traits are unlikely to be large (McArdle & Hamagami, 2003). Notably, in genetically related individuals, education does not predict incidence of pathological cognitive decline (Gatz et al., 2001). Thus, if education has any effect on how we age, it is likely to act as an expression of genetic endowment and early experiences, including, and maybe especially, biological events of embryogenesis rather than societal events of adulthood.

Recent studies of a well-documented Scottish cohort support the hypothesis that cognitive skills measured at age 11 determine to a large extent cognitive performance at age 79 (Deary et al., 2004). The Nun Study (Snowdon et al., 1996) cited by Negash and Petersen in support of societal influences on cognitive aging, can actually provide the evidence against them. As the lifestyle within religious order is tightly controlled and societal influences are minimized, the variability in age trajectories of cognitive performance is probably determined by non-societal, mainly biological, factors, many of which are inherited. The findings that persons engaged in occupations with reduced mental demands evidence “poor cognitive performance and higher prevalence of dementia” can be just as parsimoniously explained by variation in levels of cognitive prowess registered before entering a specific occupation.

The next societal factor considered by Negash and Petersen is enriched cognitive activity. They conclude that “individuals who reported lower

participation in cognitively stimulating activities were at a higher risk of developing AD compared to those reporting frequent cognitive activity.” The problem with this argument is twofold. First, cognitive activity is not independent of general cognitive abilities and skills, thus referring this proposition to the previously discussed role of premorbid and inherited cognitive abilities. Second, propensity for cognitive activity and other recreational interests show a reasonably high level of heritability (Lykken, Bouchard, McGue, & Tellegen, 1993). Thus, “active lifestyles” are not assigned to individuals at random, but may be predicated on interaction between their genetic endowment and their early life history, not current societal influences.

Vascular risk factors exert negative influence on brain and cognitive aging even at the levels that used to be considered benign (e.g., Raz, Rodrigue, & Acker, 2003). The authors consider the effects of potential positive modifiers of vascular risk, such as physical activity and nutrition on cognitive aging. Indeed there is growing evidence of beneficial effects of exercise on the brain and behavior of older adults (e.g., Colcombe et al., 2003). However, activity levels and propensity to engage in exercise and sports (An et al., 2003; Maes et al., 1996), as well as important biological determinants of tolerance to high levels of exercise (VO_{2max}), are heritable (De Geus, Boomsma, & Snieder, 2003). Moreover, although exercise is a reasonable way to reduce blood pressure (Bassuk & Manson, 2005), the response of blood pressure to behavioral interventions is heritable to a significant degree (Rice et al., 2002). As for nutrition, genetic factors also play a significant role in shaping the extent of risk and the risk-enhancing interactions with the environment, with high heritability observed for such indices of obesity as body mass index (Speakman, 2004). Significant heritability has been found for the daily amount of food, alcohol, and water consumption, especially for the meal portion size, whereas the familial environment effect on those indices is negligible (de Castro, 1993). The response to reactive oxygen species (free radicals) that are linked to nutrition and play a role in emergence of vascular disease may also depend on specific genes (Kachiwala et al., 2005). Thus, societal modifications of exercise and nutrition conducted in an attempt to introduce healthier lifestyles are constrained to a significant extent by genetic factors.

In conclusion, Negash and Petersen’s attention to the relatively neglected and potentially powerful influences on cognitive aging is welcome and commendable. However, the term societal when applied to education,

occupation, nutrition, mental engagement, and physical activity may be a misnomer. Although we need to learn more about how the genetic variability and societal influences contribute to cognitive aging, more important may be to understand how the heritable complex traits interact with societal constraints in shaping the patterns of cognitive stability and declines observed in older adults. It may be surprising, but this conclusion, derived from a different platform, is not fundamentally at odds with that of the authors, who issue a broad appeal for understanding the “importance of genetic and environmental interactions in influencing cognitive vitality.” However, such understanding will necessitate an understanding of the genetics of cognitive aging beyond ApoE, and an acknowledgement that all behavioral and social traits are shaped by the workings of the genes in the complex environment, and not by gene-free societal forces.

ACKNOWLEDGMENT

The author is supported by the grant 2R37AG011230 from the National Institutes of Health.

Commentary: Societal Influences on Cognition in Historical Context

K. Warner Schaie

INTRODUCTION

Negash and Petersen have identified and discussed a number of societal influences and lifestyle factors that mediate or moderate the path from normal cognitive functioning, through mild cognitive impairment, to the diagnosis of full-blown dementia. In this commentary, I will first describe the different forms of cognitive aging as characterized by differences in level and slope of cognitive trajectories across the adult years. I will then comment on the various risks and protective factors inherent in differential social status characteristics and lifestyles, and indicate how the role of these factors is modified by changes in historical context. Finally, I will provide some illustrations of these changes with data from the Seattle Longitudinal Study (SLS).

FORMS OF COGNITIVE AGING

It is readily apparent that there are vast individual differences in patterns of cognitive change across adulthood. Scrutiny of a variety of longitudinal studies of cognition (cf., Schaie & Hofer, 2001) suggest that four major patterns will describe most of the observed differences, although further subtypes could, of course, be considered. These patterns would classify individuals into those who age normally, those who age successfully (the super-normals), those who develop mild cognitive impairment, and finally those who become clinically diagnosable as suffering from dementia. Figure 1.1 shows commonly found cognitive trajectories as reported in the SLS (Schaie, 2005), separately from young adulthood through midlife, and from midlife to advanced old age.

The most common pattern is what we could denote as the *normal aging* of cognitive abilities. This pattern is characterized by most individuals

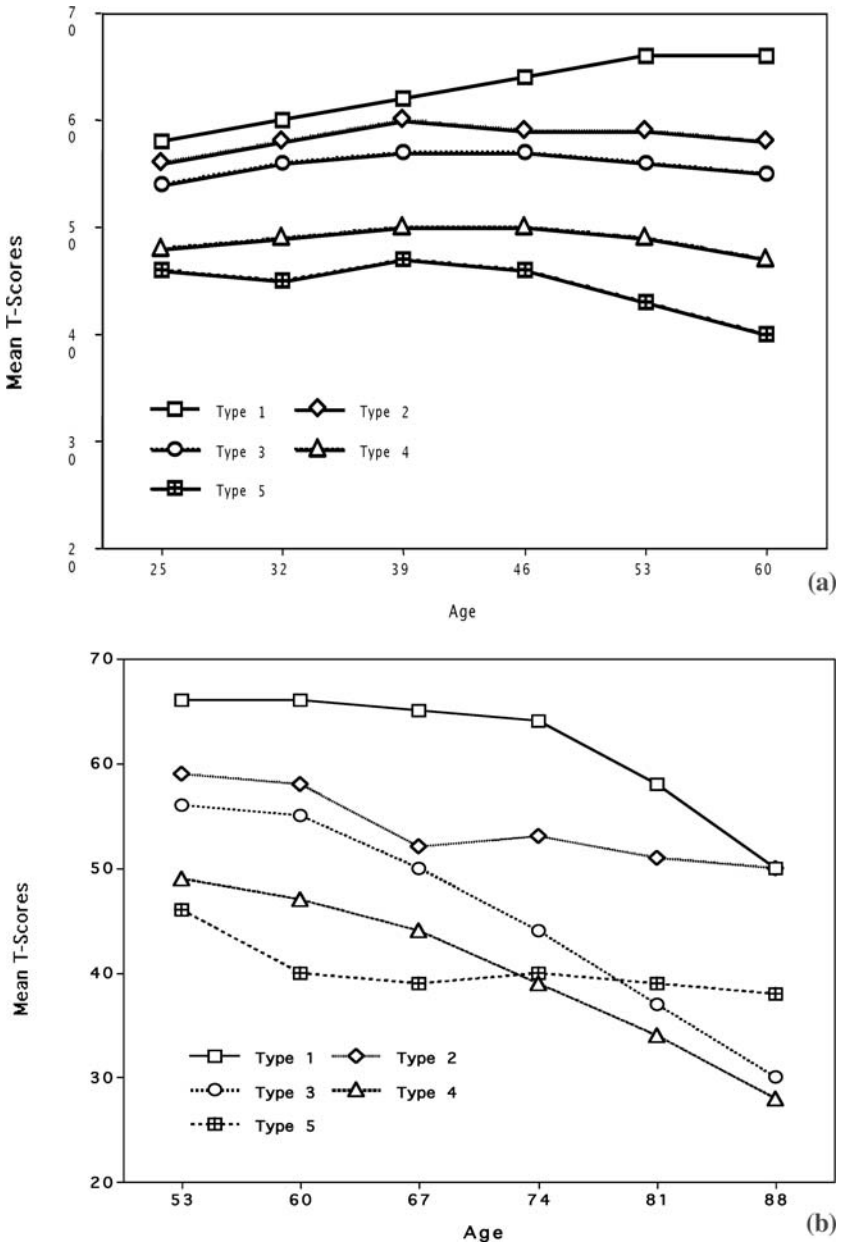


FIGURE 1.1 Alternate cognitive trajectories from young adulthood to midlife (a), and from midlife to advanced old age (b).

reaching an asymptote in early midlife, maintaining a plateau until the late 50s or early 60s, and then showing modest decline on most abilities through the early 80s, with more marked decline in the years prior to death (cf., Bosworth & Schaie, 1999). Among those whose cognitive aging can be described as normal, we can distinguish two subgroups. The first includes those individuals who reach a relatively high level of cognitive functioning who can remain independent until close to their demise even if they become physically frail. On the other hand, the second group includes those who only reach a modest asymptote in cognitive development, and may require greater support and be more likely to experience a period of institutional care in old age.

A small subgroup of adults experience what is often described as *successful aging* (Fillit et al., 2002; Rowe & Kahn, 1987). Members of this group are often genetically and socioeconomically advantaged, and tend to continue cognitive development later than most, typically reaching their cognitive asymptotes in late midlife. While they too show some very modest decline on highly speeded tasks, they are likely to maintain their overall level of cognitive functioning until shortly before their demise. These are the fortunate individuals for whom the mortality curve has been virtually squared and whose active life expectancy comes very close to their actual life expectancy.

The third pattern, *mild cognitive impairment* (MCI), includes that group of individuals who typically experience greater than normative cognitive declines in early old age (Petersen et al., 1999). Various definitions, mostly statistical, have been advanced to assign membership to this group. Some have argued for a criterion of 1 *SD* of performance compared to the young adult average, while others have proposed a rating of 0.5 on a clinical dementia rating scale, where 0 is normal and 1.0 is probable dementia. Previously the identification of MCI required the presence of memory loss, in particular. However, more recently the diagnosis has been extended to include decline in other cognitive abilities. There has also been controversy surrounding the question of whether individuals with the diagnosis of MCI inevitably progress to dementia, or whether this group of individuals represents a unique entity; which could perhaps be described as experiencing *unsuccessful aging* (cf., Petersen, 2003).

The final pattern includes those individuals who are diagnosed with *dementia* in early or advanced old age. Regardless of the specific cause of the

dementia, what these individuals have in common is dramatic impairment in cognitive functioning. However, the pattern of cognitive change, particularly in those whose diagnosis at postmortem turns out to be Alzheimer's disease, is very different from those experiencing normal aging. When followed longitudinally, at least some of these individuals show earlier decline, perhaps starting in midlife. Figure 1.2 shows the longitudinal ability patterns over a 35-year period prior to death for two women, one who was diagnosed with Alzheimer's disease, and another who died from a Cerebral Vascular Accident (CVA) and presented a virtually so-called clean cortex.

SOCIETAL INFLUENCES THAT MEDIATE OR MODERATE COGNITIVE CHANGE WITH AGE

Of the whole array of societal structures that can influence cognitive change in cognitive functioning across adulthood (cf., Nagesh & Petersen, this volume), the most thoroughly documented ones appear to be educational and occupational attainment. Although there are differences among the studies cited by Nagesh and Petersen, their sum total suggests that educational attainment in particular may have some protective value or at least postpone the onset of dementia (e.g., Katzman, 1990). It should be immediately noted that these influences are not constant, but vary across changing historical contexts (cf., Schaie & Elder, 2005; Schaie, Willis, & Pennak, 2005).

Educational Attainment

There has been a dramatic increase in educational attainment in the United States over the past century. One of the major historical events that particularly contributed to the increase of educational attainment in men was the GI Bill, which was originally designed to reduce potential veteran unemployment after the end of World War II (cf., Laub & Sampson, 2005). Disregarding changes in quality of education, the fact remains that the duration of the educational exposure has increased markedly. Data from the SLS suggest that there has been an average increase of approximately 5 ½ years of educational exposure over a 70-year period (see Figure 1.3).

Not only have there been dramatic increases in educational exposure, but there have also been dramatic changes in educational practice that have

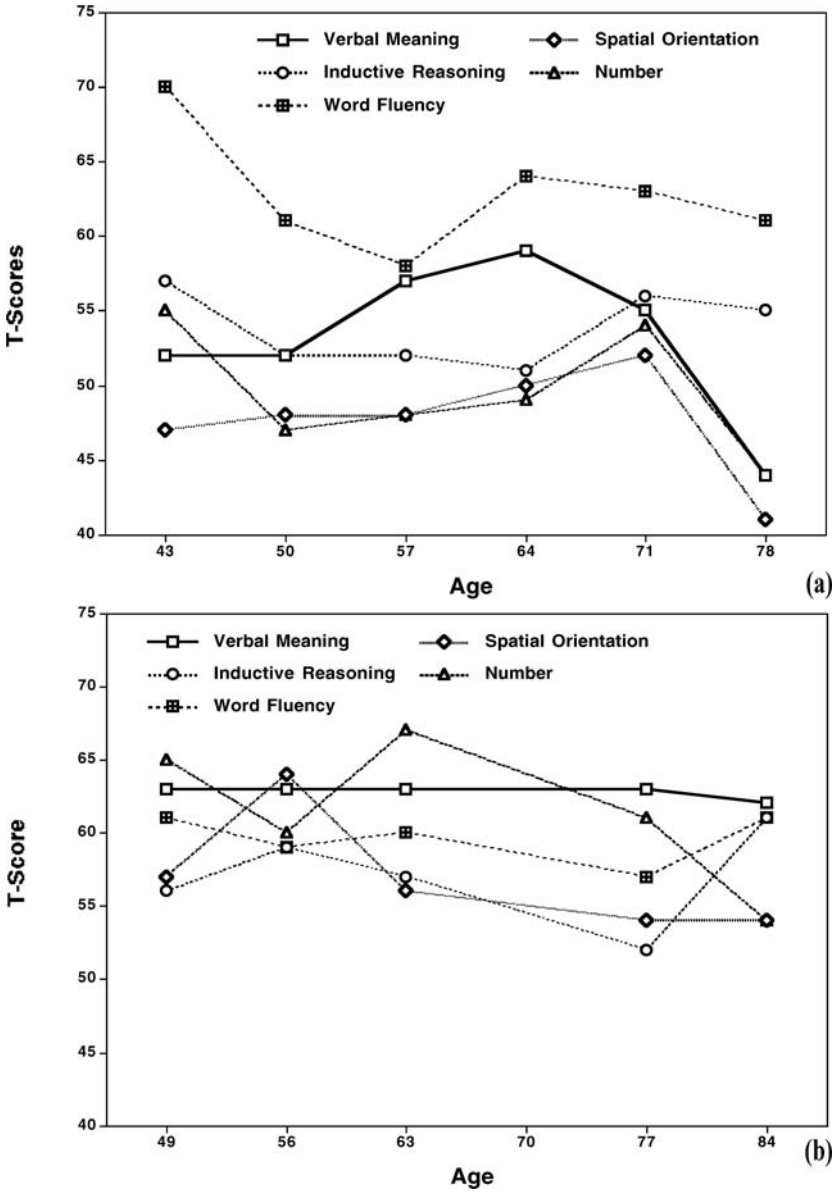


FIGURE 1.2 Longitudinal cognitive profiles over 35 years prior to death of two women, one diagnosed with Alzheimer's disease (a) and the other with no cerebral abnormalities (b) upon post mortem.

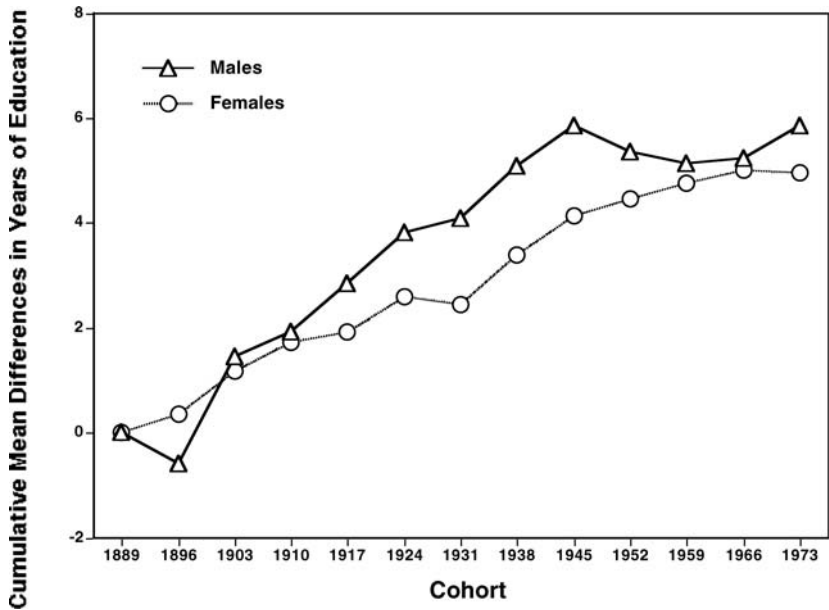


FIGURE 1.3 Cumulative cohort difference in years of education (from Schaie, 2005).

markedly affected performance on mental abilities. Some of these changes in practice have been positive, such as the increased emphasis on problem solving skills, supporting gains in executive function for many. The basic abilities affected are, in particular, Verbal Ability (due to increased educational exposure) and Inductive Reasoning (due to increased *emphasis* on discovery methods in problem solving).

Evidence for these effects has been found in the SLS (Schaie, 2005). The association between educational exposure and Verbal Ability is particularly striking. Figure 1.4 shows a lifelong advantage for the well-educated, in that the verbal ability of the average 80-year-old with post-college education remains at the level of the average 25-year-old grade school graduate. Similar findings are also shown for the association of educational attainment and Reasoning Ability.

Occupational Status

Over the past century there have been dramatic changes in the workplace. Nevertheless, it remains true that the workplace characteristics that are most likely to affect maintenance of high levels of cognitive

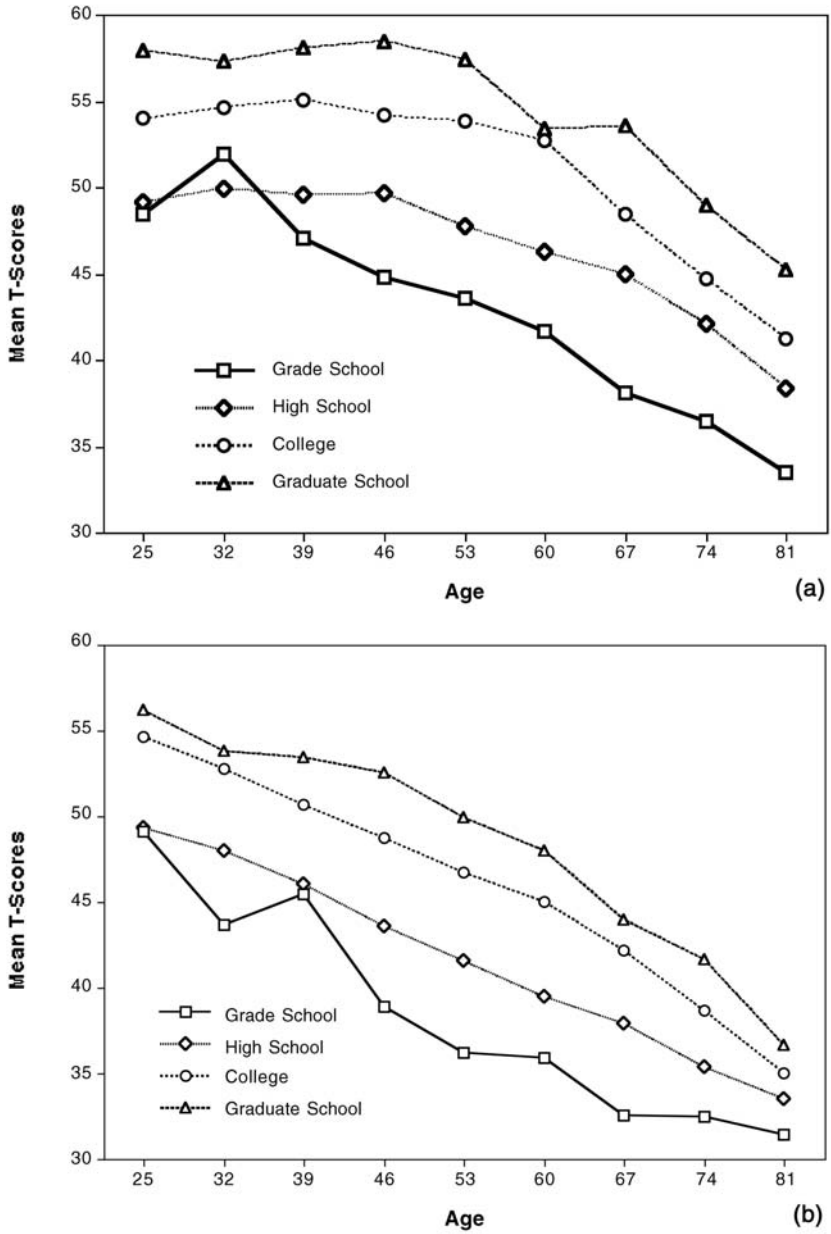


FIGURE 1.4 Association between level of educational attainment and performance on tests of Verbal Ability (a) and Inductive Reasoning (b).

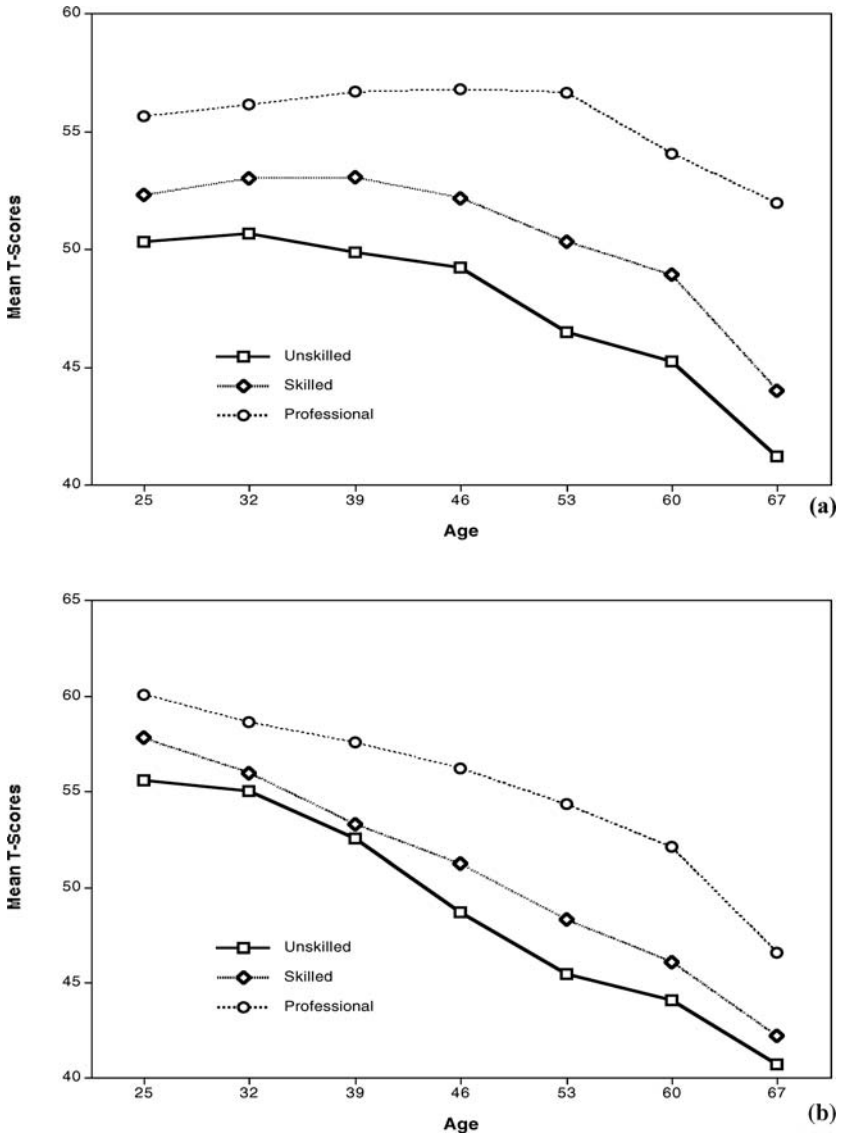


FIGURE 1.5 Association between occupational status and performance on tests of Verbal Ability (a) and Inductive Reasoning (b).

functioning are the extent to which the worker/employee is in control of his time, engages in non-routine work activities, and engages in interpersonally challenging work pursuits (cf., Schooler, Mulatu, & Oates, 2004).

Similar to the relationship with education, striking differences in longitudinal patterns of cognitive abilities for different occupational levels can also be found. When study participants are classified into unskilled, skilled, and professional workers, at the end of their work life, those termed professional still outperform skilled or unskilled workers at any life stage on Verbal Ability, and remain at an advantage on Reasoning Ability throughout life (see Figure 1.5).

LIFESTYLE FACTORS THAT AFFECT COGNITIVE FUNCTION THROUGHOUT ADULTHOOD

The most thoroughly investigated lifestyle influences that appear to affect cognitive functioning throughout adulthood appear to be nutrition, exercise, and intellectually stimulating activities. All of these factors have been subject to dramatic historical changes that seem to put cohorts born in later generations at a great advantage in comparison to cohorts at similar ages born in earlier generations. These factors include not only the increase in educational exposure discussed earlier, but also changes in educational practice that may positively or negatively affect performance on cognitive abilities (see below; Schaie et al., 2005). Marked changes have also occurred in occupational distribution. Over the past century, following a shift from agriculture to manufacturing as the principal source of employment, there was a shift to a service economy that has demanded the acquisition of computer skills and other more specialized competencies.

Although the evidence on the impact of changes in lifestyles on maintenance of cognitive functions into old age is still limited, generational differences in the practice of exercise, dietary restraint, and the utilization of preventive health care are substantial (cf., Zanjani, 2004). Also impressive is the increase in the availability of cognitively stimulating activities known to have protective effects against cognitive decline (cf., Wilson et al., 2002).

CUMULATIVE COHORT DIFFERENCES IN COGNITIVE ABILITIES

The historical changes described above have had significant impact on levels of performance on cognitive abilities. Figure 1.6 provides evidence of cumulative cohort differences measured in the SLS by obtaining averages between successive cohorts over all available ages. Over the 84-year

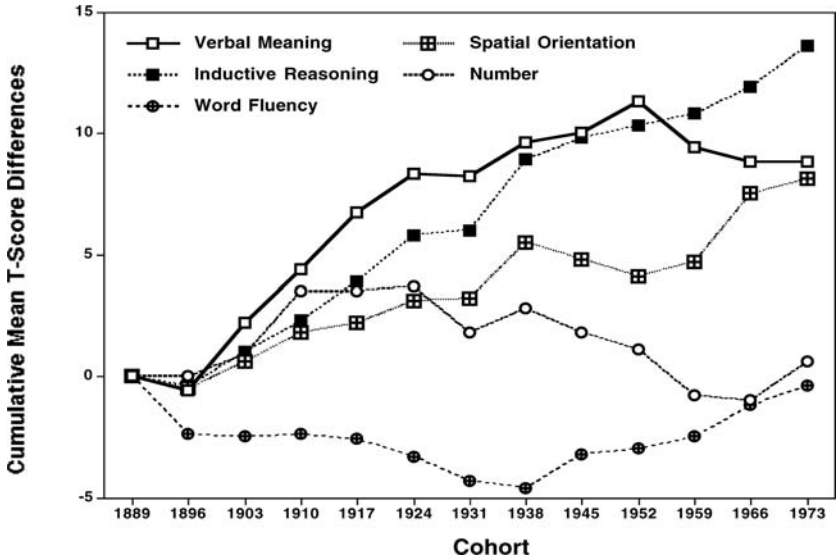


FIGURE 1.6 Cumulative generational difference of five mental abilities for birth cohorts in seven-year intervals from 1889–1973.

Source: Schaie, K. W. (2005). *Developmental influences on adult intelligence: The Seattle Longitudinal Study*. New York: Oxford University Press, p. 137. Reproduced by permission of the publisher.

period for which data is available, it will be noted that there is a positive cohort gradient for those abilities most directly impacted by educational and occupational changes in our society: Inductive Reasoning, Verbal Ability, and Spatial Ability. Numeric ability reached an asymptote early in the 20th century and has been steadily declining, while Word Fluency declined until the 1930s and then started returning to the level observed in our oldest study participants, those born in the late 19th century.

To illustrate differences of within-cohort trajectories of mental abilities across age, we selected three cohorts who, among them, in our study, cover the age range from 25–81 years of age and who overlap each other at least at three ages. The cohorts chosen are cohort 2 (born 1893–1899), cohort 6 (born 1921–1927), and cohort 10 (born 1949–1955). We are thus comparing baby boomers with the generations of their parents and grandparents. Figure 1.7 again focuses on the abilities of Verbal Ability and Inductive Reasoning. Note that the within-cohort slopes for Verbal Meaning differ primarily in old age, while the slopes for Inductive Reasoning differ significantly at all ages covered.

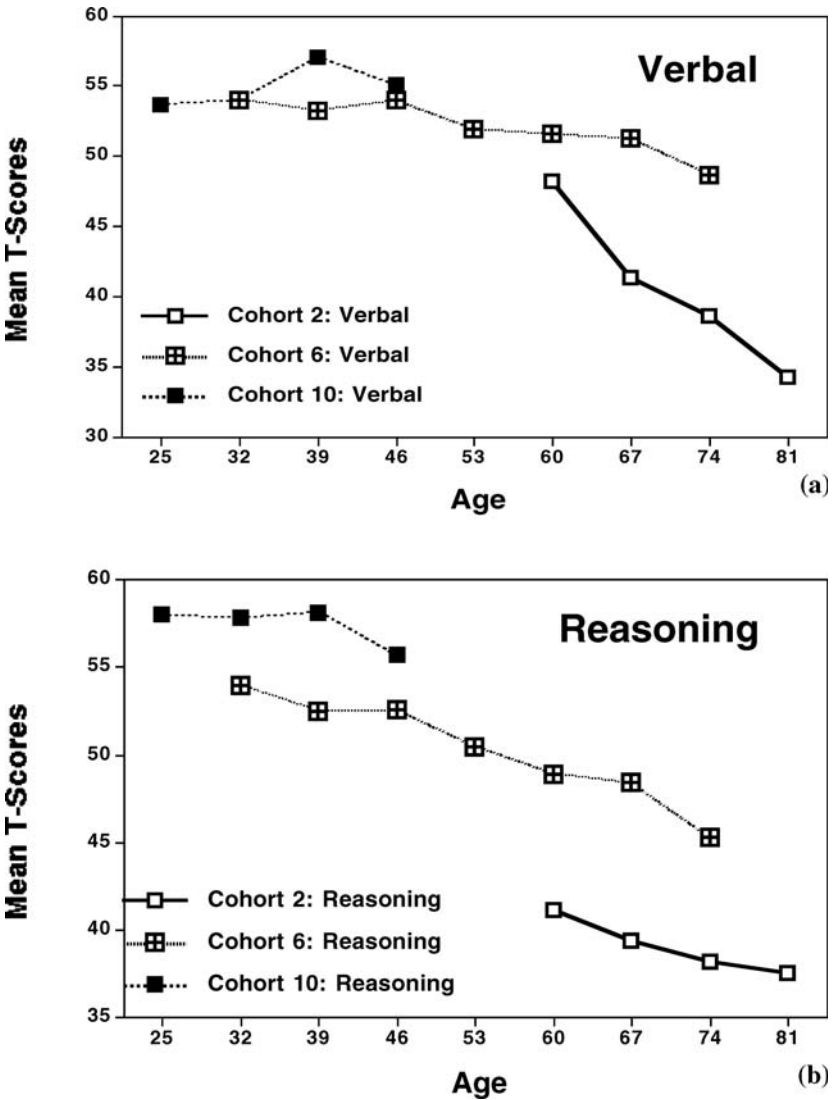


FIGURE 1.7 Within-cohort developmental trajectories for three cohorts born 28 years apart on tests of Verbal Ability (a) and Inductive Reasoning (b).

CONCLUSIONS

My interpretation of the review offered by Negash and Petersen and the data I have presented from mine and my colleagues' work in the SLS lead me to five major conclusions about the role of societal influences on cognition over the adult life course:

First, it is clear that societal influences ensure that there is no single uniform pattern of cognitive aging. Hence, there is need to discover the different types of individual trajectories that characterize different forms of cognitive aging. Second, there is conclusive evidence of generational differences in cognitive trajectories. That is, successive generations age differently. Third, it follows that historical changes in societal practices and allocation of resources will affect both individual cognitive trajectories as well as the prevalence of differential trajectories. Fourth, given the currently observable pattern of generational succession in cognitive level and rate of decline, it is most likely that future generations will have greater prevalence of more positive cognitive trajectories. Fifth, and finally, it is likely that the greater availability of societal resources that serve as protective factors against cognitive decline will serve to compensate for the impact of many cognitive risks now leading to unfavorable cognitive trajectories in late life.

REFERENCES

- Abbott, R. D., White, L. R., Ross, G. W., Masaki, K. H., Curb, J. D., & Petrovitch, H. (2004). Walking and dementia in physically capable elderly men. *Journal of the American Medical Association, 292*, 1447–1453.
- An, P., Perusse, L., Rankinen, T., Borecki, I. B., Gagnon, J., Leon, A. S., et al. (2003). Familial aggregation of exercise heart rate and blood pressure in response to 20 weeks of endurance training: The HERITAGE family study. *International Journal of Sports Medicine, 24*, 57–62.
- Bartels, M., Rietveld, M. J., Van Baal, G. C., & Boomsma, D. I. (2002). Heritability of educational achievement in 12-year-olds and the overlap with cognitive ability. *Twin Research, 5*, 544–553.
- Bassuk, S. S., & Manson, J. E. (2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *Journal of Applied Physiology, 99*, 1193–1204.

- Beard, C. M., Kokmen, E., Offord, K. P., & Kurland, L. T. (1992). Lack of association between Alzheimer's disease and education, occupation, marital status, or living arrangement. *Neurology*, *42*, 2063–2068.
- Bellush, L. L., Wright, A. M., Walker, J. P., Kopchick, J., & Colvin, R. A. (1996). Caloric restriction and spatial learning in old mice. *Physiology and Behavior*, *60*, 541–547.
- Bennett, D. A., Wilson, R. S., Schneider, J. A., Evans, D. A., Mendes de Leon, C. F., Arnold, S. E., et al. (2003). Education modifies the relation of AD pathology to level of cognitive function in older persons. *Neurology*, *60*, 1909–1915.
- Bosworth, H. B., & Schaie, K. W. (1999). Survival effects in cognitive function, cognitive style, and sociodemographic variables in the Seattle longitudinal Study. *Experimental Aging Research*, *25*, 121–139.
- Bouchard, T. J., Lykken, D. T., McGue, M., Segal, N. L., & Tellegen, A. (1990). Sources of human psychological differences: The Minnesota study of twins reared apart. *Science*, *250*, 223–228.
- Bunce, D. J., Barrowclough, A., & Morris, I. (1996). The moderating influence of physical fitness on age gradients in vigilance and serial choice responding tasks. *Psychology and Aging*, *11*, 671–682.
- Callahan, C. M., Hall, K. S., Hui, S. L., Musick, B. S., Unverzagt, F. W., & Hendrie, H. C. (1996). Relationship of age, education, and occupation with dementia among a community-based sample of African Americans. *Archives of Neurology*, *53*, 134–140.
- Colcombe, S., Erickson, K. I., Raz, N., Webb, A. G., Cohen, N. J., McAuley, E., & Kramer, A. (2003). Aerobic fitness reduces brain tissue loss in aging humans. *Journal of Gerontology*, *58A*, 176–180.
- Corder, E. H., Saunders, A. M., Strittmatter, W. J., Schmechel, D., Gaskell, P., Small, W., et al. (1993). Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science*, *261*, 921–923.
- Crook, T., Bartus, R. T., Ferris, S. H., Whitehouse, P., Cohen, G. D., & Gershon, S. (1986). Age-associated memory impairment: Proposed diagnostic criteria and measures of clinical change—Report of a National Institute of Mental Health Work Group. *Developmental Neuropsychology*, *2*, 261–267.
- Dartigues, J. F., Gagnon, M., Letenneur, L., Barberger-Gateau, P., Commenges, D., Evaldre, M., et al. (1992). Principal lifetime occu-

- pation and cognitive impairment in a French elderly cohort (Paquid). *American Journal of Epidemiology*, *135*, 981–988.
- Deary, I. J., Whiteman, M. C., Starr, J. M., Whalley, L. J., & Fox, H. C. (2004). The impact of childhood intelligence on later life: Following up the Scottish mental surveys of 1932 and 1947. *Journal of Personality and Social Psychology*, *86*, 130–147.
- de Castro, J. M. (1993). Genetic influences on daily intake and meal patterns of humans. *Physiology and Behavior*, *53*, 777–782.
- De Geus, E. J., Boomsma, D. I., & Snieder, H. (2003). Genetic correlation of exercise with heart rate and respiratory sinus arrhythmia. *Medicine and Science in Sports and Exercise*, *35*, 1287–1295.
- Engelhart, M. J., Geerlings, M. I., Ruitenberg, A., Swieten, J. C., van Hofman, A., Witteman, J.C.M., et al. (2002). Diet and risk of dementia: Does fat matter? The Rotterdam Study. *Neurology*, *59*, 1915–1921.
- Evans, D. A., Beckett, L. A., Albert, M. S., Hebert, L. E., Scherr, P. A., Funkenstein, H. H., et al. (1993). Level of education and change in cognitive function in a community population of older persons. *Annals of Epidemiology*, *3*, 71–77.
- Everson-Rose, S. A., Mendes de Leon, C. F., Bienias, J. L., Wilson, R. S., & Evans, D. A. (2003). Early life conditions and cognitive functioning in later life. *American Journal of Epidemiology*, *158*, 1083–1089.
- Farmer, M. E., Kittner, S. J., Rae, D. S., Bartko, J. J., & Regier, D. A. (1995). Education and change in cognitive function. The Epidemiologic Catchment Area Study. *Annals of Epidemiology*, *5*, 1–7.
- Fillit, H. M., Butler, R. N., O'Connell, A. W., Albert, M. S., Birren, J. E., Cotman, C. W., et al. (2002). Achieving and maintaining cognitive vitality with aging. *Mayo Clinic Proceedings*, *77*, 681–696.
- Fratiglioni, L., Paillard-Borg, S., & Winblad, B. (2004). An active and socially integrated lifestyle in late life might protect against dementia. *Lancet Neurology*, *3*, 343–353.
- Frei, B. (1995) Cardiovascular disease and nutrient antioxidants: Role of low-density lipoprotein oxidation. *Critical Reviews in Food Science and Nutrition*, *35*, 83–98.
- Gatz, M., Svedberg, P., Pedersen, N. L., Mortimer, J. A., Berg, S., & Johansson, B. (2001). Education and the risk of Alzheimer's disease: Findings from the study of dementia in Swedish twins. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, *56*, 292–300.

- Heath, A. C., Berg, K., Eaves, L. J., Solaas, M. H., Corey, L. A., & Sundet, J., et al. (1985). Education policy and the heritability of educational attainment. *Nature*, *314*, 734–736.
- Hertzog, C., & Schaie, K. W. (1986). Stability and change in adult intelligence: 1. Analysis of longitudinal covariance structures. *Psychology and Aging*, *1*, 159–171.
- Hultsch, D. F., Hertzog, C., Small, B. J., & Dixon, R. A. (1999). Use it or lose it: Engaged lifestyle as a buffer of cognitive decline in aging? *Psychology and Aging*, *14*, 245–263.
- Jorm, A. F., Rodgers, B., Henderson, A. S., Korten, A. E., Jacomb, P. A., Christensen, H., et al. (1998). Occupation type as a predictor of cognitive decline and dementia in old age. *Age and Aging*, *27*, 477–483.
- Kachiwala, S. J., Harris, S. E., Wright, A. F., Hayward, C., Starr, J. M., Whalley, L. J., et al. (2005). Genetic influences on oxidative stress and their association with normal cognitive aging. *Neuroscience Letters*, *386*, 116–120.
- Katzman, R. (1993). Education and the prevalence of dementia and Alzheimer's disease. *Neurology*, *43*, 13–20.
- Kaye, J. A., Oken, B. S., Howieson, D. B., Howieson, J., Holm, L. A., & Dennison, K. (1994). Neurologic evaluation of the optimally healthy oldest old. *Archives of Neurology*, *51*, 1205–1211.
- Kempermann, G., Kuhn, H. G., & Gage, F. H. (1997). More hippocampal neurons in adult mice living in an enriched environment. *Nature*, *386*, 493–495.
- Kempermann, G., Kuhn, H. G., & Gage, F. H. (1998). Experience-induced neurogenesis in the senescent dentate gyrus. *The Journal of Neuroscience*, *18*, 3206–3212.
- Kleim, J. A., Vij, K., Ballard, D. H., & Greenough, W. T. (1997). Learning-dependent synaptic modifications in the cerebellar cortex of the adult rat persist for at least four weeks. *The Journal of Neuroscience*, *17*, 717–721.
- Kral, V. A. (1962). Senescent forgetfulness: Benign and malignant. *Canadian Medical Association Journal*, *86*, 257–260.
- Laub, J. H., & Sampson, R. J. (2005). Coming of age in wartime: How World War II and the Korean War changed lives. In K. W. Schaie & G. H. Elder, Jr. (Eds.), *Historical influences on lives and aging* (pp. 208–228). New York: Springer Publishing Co.

- Laurin, D., Verreault, R., Lindsay, J., MacPherson, K., & Rockwood, K. (2001). Physical activity and risk of cognitive impairment and dementia in elderly persons. *Archives of Neurology*, *58*, 498–504.
- Levy, R. (1994). Aging-associated cognitive decline. *International Psychogeriatrics*, *6*, 63–68.
- Lindenberger, U., & Baltes, P. B. (1997). Intellectual functioning in old and very old age: Cross-sectional results from the Berlin Aging Study. *Psychology and Aging*, *12*, 410–432.
- Lykken, D. T., Bouchard, T. J., Jr., McGue, M., & Tellegen, A. (1993). Heritability of interests: A twin study. *Journal of Applied Psychology*, *78*, 649–661.
- Madden, D. J., Blumenthal, J. A., Allen, P. A., & Emery, C. F. (1989). Improving aerobic capacity in healthy older adults does not necessarily lead to improved cognitive performance. *Psychology and Aging*, *4*, 307–320.
- Maes, H. H., Beunen, G. P., Vlietinck, R. F., Neale, M. C., Thomis, M., Vanden Eynde, B., et al. (1996). Inheritance of physical fitness in 10-year-old twins and their parents. *Medicine and Science in Sports and Exercise*, *28*, 1479–1491.
- Mattson, M. P., Duan, W., Lee, J., & Guo, Z. (2001). Suppression of brain aging and neurodegenerative disorders by dietary restriction and environmental enrichment: Molecular mechanisms. *Mechanisms of Aging and Development*, *122*, 757–778.
- McArdle, J. J., Hamagami, F. (2003). Structural equation models for evaluating dynamic concepts within longitudinal twin analyses. *Behavior Genetics*, *33*, 127–159.
- Morris, M. C., Evans, D. A., Bienias, J. L., Tangney, C. C., Bennett, D. A., Aggarwal, N., et al. (2003). Dietary fat and the risk of incident Alzheimer disease. *Archives of Neurology*, *60*, 194–200.
- Notkola, I. L., Sulkava, R., Pekkanen, J., Erkinjuntti, T., Ehnholm, C., Kivinen, P., et al. (1998). Serum total cholesterol, apolipoprotein E epsilon 4 allele, and Alzheimer's disease. *Neuroepidemiology*, *17*, 14–20.
- Ortega, R. M., Manas, L. R., Andres, P., Gaspar, M. J., Agudo, F. R., Jimenez, A., et al. (1996). Functional and psychic deterioration in elderly people may be aggravated by folate deficiency. *The Journal of Nutrition*, *126*, 1992–1999.

- Pedersen, N. L., Plomin, R., Nesselroade, J. T., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science*, 3, 346–353.
- Pedersen, N. L., Reynolds, C. A., & Gatz, M. (1996). Sources of covariation among Mini-Mental State Examination scores, education, and cognitive abilities. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*, 51, 55–63.
- Petersen, R. C. (2003). Conceptual overview. In R. C. Petersen (Ed.), *Mild cognitive impairment: Aging to disease* (pp. 1–14). New York: Oxford University Press.
- Petersen, R. C., Smith, G. E., Ivnik, R. J., Tangalos, E. G., Schaid, D. I., Thibodeau, S., et al. (1995). Apolipoprotein E status as a predictor of the development of Alzheimer's disease in memory-impaired individuals. *Journal of the American Medical Association*, 273, 1274–1278.
- Petersen, R. C., Smith, G. E., Waring, S. C., Ivnik, R. J., Tangalos, E. G., & Kokmen, E. (1999). Mild cognitive impairment: Clinical characterization and outcome. *Archives of Neurology*, 56, 303–308.
- Petersen, R. C., Thomas, R. G., Grundman, M., Bennett, D., Doody, R., Ferris, S., et al. (2005). Vitamin E and donepezil for the treatment of mild cognitive impairment. *New England Journal of Medicine*, 352(23), 2379–2388.
- Powell, R. R., & Pohndorf, R. H. (1971). Comparison of adult exercisers and nonexercisers on fluid intelligence and selected physiological variables. *Research Quarterly*, 42, 70–77.
- Praag, H., van Kempermann, G., & Gage, F. H. (1999). Running increases cell proliferation and neurogenesis in the adult mouse dentate gyrus. *Nature Neuroscience*, 2, 266–270.
- Raz, N., Rodrigue, K.M., & Acker, J.D. (2003). Hypertension and the brain: Vulnerability of the prefrontal regions and executive functions. *Behavioral Neuroscience*, 17, 1169–1180.
- Raz, N., Lindenberger, U., Rodrigue, K. M., Kennedy, K. M., Head, D., Williamson, et al. (2005). Regional brain changes in aging healthy adults: General trends, individual differences, and modifiers. *Cerebral Cortex*, 15, 1676–1689.
- Rice, T., An, P., Gagnon, J., Leon, A. S., Skinner, J. S., Wilmore, J. H., et al. (2002). Heritability of HR and BP response to exercise training

- in the HERITAGE Family Study. *Medicine and Science in Sports and Exercise*, 34, 972–979.
- Rowe, J. W., & Kahn, R. L. (1987). Human aging: Usual and successful. *Science*, 237, 143–149.
- Scarmeas, N., Levy, G., Tang, M. X., Manly, J., & Stern, Y. (2001). Influence of leisure activity on the incidence of Alzheimer's disease. *Neurology*, 57, 2236–2242.
- Schaie, K. W. (1984). Midlife influences upon intellectual functioning in old age. *International Journal of Behavioral Development*, 7, 463–478.
- Schaie, K. W. (2005). *Developmental influences on adult intelligence: The Seattle Longitudinal Study*. New York: Oxford University Press.
- Schaie, K. W., & Elder, G. H. Jr. (Eds.). (2005). *Historical influence on lives and aging*. New York: Springer Publishing Co.
- Schaie, K. W., & Hofer, S. M. (2001). Longitudinal studies in research on aging. In J. E. Birren & K. W. Schaie (Eds.), *Handbook of the psychology of aging* (5th ed., pp. 55–77). San Diego, CA: Academic Press.
- Schaie, K. W., Willis, S. L., & Pennak, S. (2005). A historical framework for cohort differences in intelligence. *Research in Human Development*, 2, 43–67.
- Schooler, C., Mulatu, M. S., & Oates, G. (2004). Occupational self-direction, intellectual functioning, and self-directed orientation in old workers: Findings and implications for individuals and societies. *American Journal of Sociology*, 110, 161–197.
- Sirevaag, A. M., Black, J. E., Shafron, D., & Greenough, W. T. (1998). Direct evidence that complex experience increases capillary branching and surface area in visual cortex of young rats. *Brain Research*, 471, 299–304.
- Smyth, K. A., Fritsch, T., Cook, T. B., McClendon, M. J., Santillan, C. E., & Friedland, R. P. (2004). Worker functions and traits associated with occupations and the development of AD. *Neurology*, 63, 498–503.
- Snowdon, D. A., Kemper, S. J., Mortimer, J. A., Greiner, L. H., Wekstein, D. R., & Markesbery, W. R. (1996). Linguistic ability in early life and cognitive function and Alzheimer's disease in late life: Findings from the Nun Study. *Journal of the American Medical Association*, 275, 528–532.
- Speakman, J. R. (2004). Obesity: The integrated roles of environment and genetics. *Journal of Nutrition*, 134, 2090S–2105S.

- Stern, Y., Alexander, G. E., Prohovnik, I., & Mayeux, R. (1992). Inverse relationship between education and parietotemporal perfusion deficit in Alzheimer's disease. *Annals of Neurology*, *32*, 371–375.
- Stern, Y., Alexander, G. E., Prohovnik, I., Stricks, L., Link, B., Lennon, M. C., et al. (1995). Relationship between lifetime occupation and parietal flow: Implications for a reserve against Alzheimer's disease pathology. *Neurology*, *45*, 55–60.
- Stern, Y., Gurland, B., Tatemichi, T. K., Tang, M. X., Wilder, D., & Mayeux, R. (1994). Influence of education and occupation on the incidence of Alzheimer's disease. *Journal of the American Medical Association*, *271*, 1004–1010.
- Strittmatter, W. J., Saunders, A. M., Schmechel, D., Pericak-Vance, M., Enghild, J., Salvesen, G. S., et al. (1993). Apolipoprotein E: High avidity binding to β -amyloid and increased frequency of type 4 allele in late-onset familial Alzheimer disease. *Proceedings of the National Academy of Science*, *90*, 1977–1981.
- Wang, H. X., Karp, A., Winblad, B., & Fratiglioni, L. (2002). Late-life engagement in social and leisure activities is associated with a decreased risk of dementia: A longitudinal study from the Kungsholmen project. *American Journal of Epidemiology*, *155*, 1081–1087.
- Weuve, J., Kang, J. H., Manson, J. E., Breteler, M. M., Ware, J. H., & Grodstein, F. (2004). Physical activity, including walking, and cognitive function in older women. *Journal of the American Medical Association*, *292*, 1454–1461.
- Wilson, R. S., & Bennett, D. A. (2003). Cognitive activity and risk of Alzheimer's disease. *Current Directions in Psychological Science*, *12*, 87–91.
- Wilson, R. S., Mendes de Leon, C. F., Barnes, L. L., Schneider, J. A., Bienias, J. L., Evans, D. A., et al. (2002). Participation in cognitively stimulating activities and risk of incident Alzheimer disease. *Journal of the American Medical Association*, *287*, 742–748.
- Zanjani, F.A.K. (2004). *Developmental trajectory of health behaviors across the adult lifespan*. Unpublished doctoral dissertation, The Pennsylvania State University.
- Zhang, M., Katzman, R., Salmon, D., Jin, H., Cai, G., Wang, Z., et al. (1990). The prevalence of dementia and Alzheimer's disease in Shanghai, China: Impact of age, gender, and education. *Annals of Neurology*, *27*, 428–437.