Intelligence

M Lövdén and U Lindenberger, Max Planck Institute for Human Development, Berlin, Germany

© 2007 Elsevier Inc. All rights reserved.

Glossary

Covariance Dedifferentiation – Stronger associations between the fluid mechanics and the crystallized pragmatics in old age as compared to young adulthood.

Crystallized Pragmatics – Content-rich abilities strongly driven by cultural systems of influence; typical examples are verbal knowledge and professional expertise.

Directionality Dedifferentiation – Decline in both the fluid mechanics and the crystallized pragmatics seen in very old age.

Fluid Mechanics – Abilities representing measurable outcomes of biological influences on intellectual development; basic information processes that are content poor and universal, such as processing speed and working memory.

Terminal Decline – Changes in intelligence associated with impending death. These changes may reflect causal structures other than those underlying normative age-graded changes, including cognition-influencing diseases.

Two-Component Models of Life Span Cognition – Models of intelligence disputing the existence of a unitary general intelligence construct and positing that development of intellectual abilities can be mapped onto biological versus cultural ensembles of influence.

Introduction

This article summarizes psychometric theorizing and evidence about intelligence in old age, with a focus on normal aging. First, two-component theories of life span changes in intelligence are introduced, and evidence regarding average population age gradients in intellectual abilities is summarized. Then, hypothesized normative and basic determinants of decrements in intellectual performance in old age as well as the extent and possible causes of individual differences in such decrements are reviewed. Finally, evidence is highlighted suggesting that many aging individuals possess sizable capacity for learning and plasticity, despite aging-related losses in average intellectual performance.

Two-Component Models of Life Span Changes in Intelligence

Two-component models of life span changes in intelligence posit that development of intellectual abilities can be mapped onto biological versus cultural ensembles of influences (see **Figure 1**). Typical contemporary examples for two-component models are the distinction between fluid and crystallized intelligence advanced by Raymond B. Cattell and John

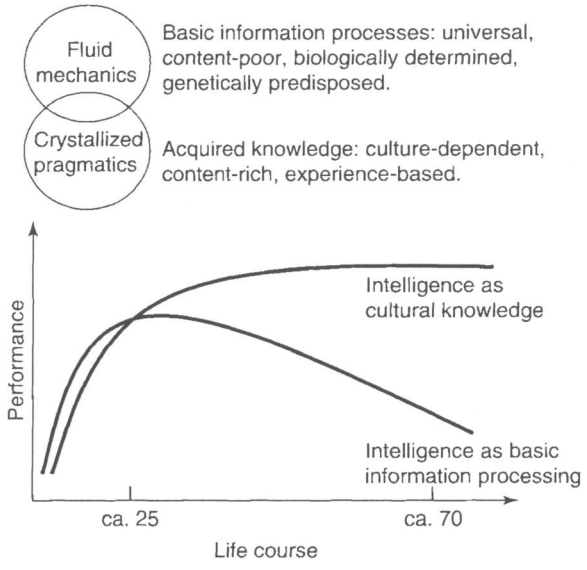

Figure 1 Two-component models of cognition. The top section defines the two categories of intellectual abilities and the bottom section illustrates postulated life span trajectories. In very old age, the trajectories become less differentiated because the fluid mechanics increasingly constrain the acquisition, expression, and representation of pragmatic knowledge.

Horn and the decomposition of cognition into mechanics and pragmatics proposed by Paul Baltes. Two-component models of life span changes in intelligence have a long history dating back to Johann Nicolaus Tetens (1736–1807), a philosopher and psychologist of the enlightenment era who noted that well-trained skills are less likely to decline with advancing age than the basic abilities underlying their acquisition. Thus, and most importantly, two-component theories dispute the validity of a unitary general intelligence construct in understanding intellectual development across the life span. Instead, at least two broad, ontogenetically intertwined but separable categories of abilities are needed to describe the basic properties of intellectual development.

The first collection of intellectual abilities represents measurable outcomes of the influence of the biological component on development. It manifests itself in cognitive processes involving extrapolation, reorganization, and transformation of novel information (i.e., reasoning) and in basic information processes such as working memory (i.e., the ability to maintain information online while manipulating it), processing speed (i.e., the speed with which elementary processing operations can be performed), and cognitive control (i.e., the top-down coordination and control of lower-level processing). Henceforth, these processes are referred to as the fluid mechanics of intelligence.

The second, more disparate category of intellectual abilities refers to procedural and declarative knowledge common to a given culture (e.g., verbal knowledge), but also to specialized and sometimes highly idiosyncratic (person-specific) knowledge such as occupational expertise, as well as to knowledge about the meaning and conduct of life. Henceforth, these processes are referred to as the crystallized pragmatics of intelligence. For examples of specific abilities and functions related to each of the two components, see Figure 2.

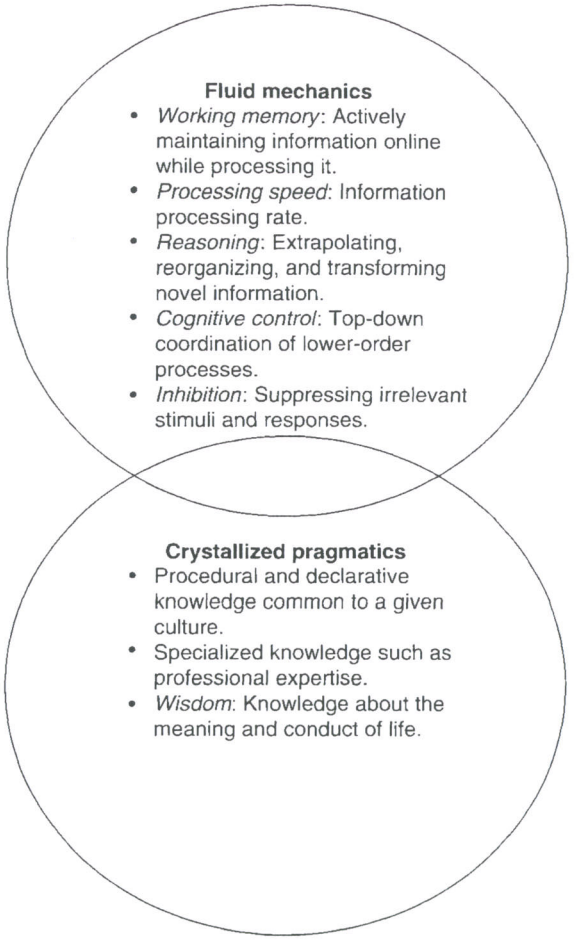

Figure 2 Exemplary functions and abilities in the broad domains of the fluid mechanics and the crystallized pragmatics.

Average Aging Patterns of Fluid Mechanics and Crystallized Pragmatics

The fluid mechanics are expected to decline after maturity. In contrast, the crystallized pragmatics are proposed to increase over the life span as long as knowledge maintenance and knowledge acquisition outweigh the adverse consequences of losses in the mechanics (see Figure 1). Empirical evidence is largely consistent with these propositions. Starting in young or middle adulthood, cross-sectional evidence

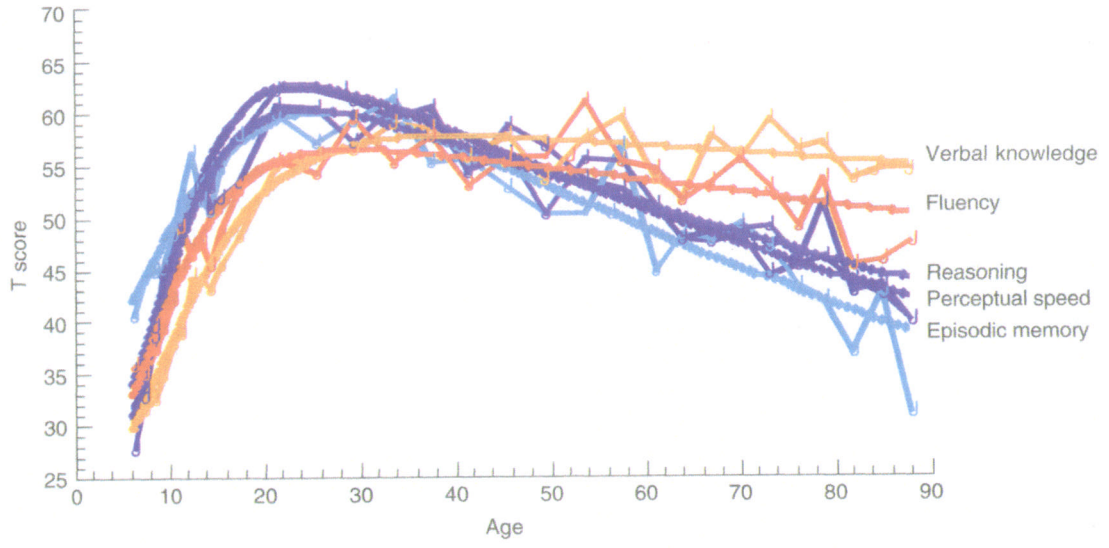

Figure 3 Cross-sectional life span age gradients for five different intellectual abilities. Verbal knowledge is a marker of intellectual ability from the domain of the crystallized pragmatics, whereas reasoning, perceptual speed, and episodic memory are intellectual abilities from the domain of the fluid mechanics. Fluency is generally regarded as a hybrid intellectual ability because it requires fast access to stored knowledge. The empirically observed age gradients are consistent with two-component theories of life span cognition. Adapted from Li S-C, Lindenberger U, Hommel B, Aschersleben G, Prinz W, and Baltes PB (2004) Transformations in the couplings among intellectual abilities and constituent cognitive processes across the life span. *Psychological Science* 15: 158.

reveals continuous decrements for abilities in the broad fluid domain. Age gradients in the domain of the crystallized pragmatics show greater variation within and between individuals in the transition from early to late adulthood, with the modal patterns of increments in young adulthood typically followed by asymptotic plateaus in middle and late adulthood, until modest decrements are discernible in old age (e.g., around 60–80 years of age) or very old age (e.g., over 80 years of age). For illustration, see **Figure 3**.

The maintenance of levels of intellectual performance in abilities belonging to the crystallized pragmatics well into old age is not confined to psychometrically assessed intellectual abilities from this domain such as verbal knowledge. Rather, maintenance is also observed for specialized and person-specific bodies of knowledge and for knowledge about the meaning and conduct of life. For example, reasoning about difficult life problems (e.g., a 14-year-old pregnant girl seeking advice) is unrelated to chronological age from early adulthood up to old age, and older individuals with an idoneous constellation of enabling personality dispositions, life experiences, and cognitive styles are among the top performers.

Longitudinal findings suggest a greater degree of similarity between the age gradients of the fluid mechanics and the crystallized pragmatics than cross-sectional evidence. Here, the fluid mechanics show decelerating increases across young adulthood, peaks in middle age, and accelerating declines thereafter. The dominant pattern of the pragmatics is one of stable, or even increasing, performance until old or very old age. Note, however, that longitudinal findings are often obtained from individuals who show more positive changes than the population from which they originate, and that retest effects may further bias age gradients in the positive direction.

In summary, and as predicted by two-component theories of life span cognition, available evidence underscores the multidirectionality of adult intellectual development. Considerable decline in the fluid mechanics and stability followed by late-life decline in the crystallized pragmatics constitute the classical pattern of the aging of adult intelligence.

The Dedifferentiation Hypothesis

The onset and amount of decline in the crystallized pragmatics during the transition from old to very old age is more variable than the divergence in age gradients between the fluid mechanics and the crystallized pragmatics observed during earlier periods of the life span. Whereas some studies report small negative age trends in crystallized pragmatics during early old age, with noticeable decrements appearing only at later ages, others report age-related decrements starting at about age 50. There is general agreement, however, that the preservation of crystallized pragmatics often observed for groups of older adults does not generalize to groups of very old

individuals. That is, for the period from early to late adulthood, cross-sectional evidence reveals monotonic decline in the fluid mechanics but stable or increasing performance levels for the crystallized pragmatics. However, in very old age, negative age gradients generally prevail for both the fluid mechanics and the crystallized pragmatics. Hence, in very old age, the difference in the directionality of the age gradients diminishes and eventually ceases to exist. In relation to the life span development of the fluid mechanics and the crystallized pragmatics, this pattern has been termed directionality dedifferentiation. Importantly, this pattern of directionality dedifferentiation also holds for longitudinal data. Furthermore, age-associated changes in the structure of intelligence are consistent with the notion of stronger relations among different intellectual abilities in old age relative to early adulthood. This finding has been referred to as covariance dedifferentiation.

A long-standing hypothesis in life span developmental research asserts that the functional organization of intellectual abilities undergoes decompression (differentiation) during maturation and again becomes more compressed (dedifferentiated) in old age. The differentiation-dedifferentiation hypothesis promotes a dynamic and process-oriented view of the structure of intellectual abilities. During childhood and old age, the operations and expressions of the diversity of cognitive abilities are posited as being constrained by the developmental status of the biological substrates of intelligence. In contrast, during maturation and in adulthood, biological-developmental constraints are relaxed, and other factors, such as interest and educational opportunities, occupy more prominent roles as determinants of individual differences in intellectual performance and development, leading to greater diversity in levels of functioning in different abilities. In old and very old age, the space of intellectual abilities becomes again increasingly dominated by an ensemble of common biological constraints on intellectual development. In terms of the two-component view of life span intelligence, old age decrements in abilities belonging to the domain of the crystallized pragmatics are induced by decline in the fluid mechanics. Put differently, the biological changes indicated by declines in the fluid mechanics hinder the acquisition, expression, and representation of pragmatic knowledge in old age when falling below thresholds that may vary from skill to skill. Conversely, at higher levels of functioning, development of the crystallized pragmatics is more dependent upon cultural-experiential factors, though biological factors continue to play a role.

The dedifferentiation hypothesis was sparked by early findings of increasing associations among different intellectual abilities from early adulthood to old age. More recent empirical evidence has generally bolstered the early findings, but some prominent exceptions are also available. Importantly, recent findings by Paolo Ghisletta and Ulman Lindenberger support the specific account of the dedifferentiation hypothesis, suggesting that decline in the fluid mechanics ultimately limits the acquisition, expression, or maintenance of the pragmatics in old age and thus drives late-life decline in the crystallized pragmatics. It follows that, with advancing adult age, between-person differences in levels of crystallized pragmatics, such as verbal knowledge, are increasingly determined by the functioning of the fluid mechanics, such as processing speed. Thus, in old age, expressing, maintaining, and acquiring knowledge are more and more dependent on biological vitality.

Basic Determinants of Decline in Fluid Mechanics

If decline in the fluid mechanics drives late-life decline in the crystallized pragmatics, then what drives decline in the fluid mechanics? Recent decades have witnessed a great number of predominantly cross-sectional studies attempting to identify information-processing mechanisms that act as domain-general pacemakers of decline in the fluid mechanics in old age. Among the most prominent constructs are processing speed, working memory capacity, and the ability to suppress irrelevant information (inhibition). Evidence supporting each of these three constructs as primary determinants of decline in a wide variety of fluid mechanics is available. However, all three constructs are relatively weakly grounded at the neurophysiological level, suffer from difficulties in making differential predictions, and often have been investigated in age-heterogeneous cross-sectional data sets, which are severely limited in inferential conclusiveness. Strengthening the link between aging changes at behavioral and neuronal levels of analysis probably offers a more viable route toward the identification of domain-general mechanisms of decline in the fluid mechanics.

In this vein, the emerging field of the cognitive neuroscience of aging has highlighted the central role of adult age changes in neuromodulation for intellectual aging. In particular, theoretical work by Shu-Chen Li and colleagues using neurocomputational modeling has suggested that age-based reductions in levels of the neurotransmitter dopamine is

apt to explain key phenomena of intellectual aging such as decrements in mean levels of performance, greater age deficits with more difficult tasks, lower maximum levels of performance, as well as increments in covariation between tasks across individuals, performance fluctuation, and interindividual differences. In addition, there are strong empirical relations among dopamine markers, adult age, and intellectual performance.

Morphological alterations and decreases in brain volume of the prefrontal cortex also have been associated with normal aging. Given that brain activity in the prefrontal cortex is functionally related to performance in central abilities of the fluid mechanics, such as reasoning and working memory, these findings suggest that anatomical, chemical, and functional changes in the prefrontal cortex and associated neuronal circuitries play a central role in late-life intellectual decline. Brain alterations in the medial temporal lobe and diencephalic systems are observed to a lesser extent with normal aging and appear to be more specifically linked to the onset and progression of age-associated dementing illnesses.

Normative and Non-normative Influences on Intellectual Development in Old Age

Average age gradients and the search for basic determinants of cognitive decline in old age serve important purposes. However, to understand the development of intelligence in old age, it is necessary to unpack the ingredients contributing to a particular score, for a particular individual, at a particular point in time. Intellectual development in old age is shaped by a wealth of additional developmental sources such as learning history, health status, distance from death, onset of pathology, and non-normative events, all of which are only loosely linked to chronological age. Thus, as suggested by Paul Baltes and others, three distinct but overlapping influences on intellectual changes in old age need to be set apart: normative age-graded influences, normative history-graded influences, and non-normative (idiosyncratic) influences. Normative in this context refers to influences that are relatively general to the population of aging individuals, such as declines in basic determinants of the fluid mechanics. Age-graded influences refer to those biological and environmental factors that are highly related to age and therefore shape the intellectual aging processes of most individuals. History-graded influences differ across birth cohorts, historical periods, or both. For example, substantial associations between birth cohorts and intelligence scores have been observed during the past century at the age of enrollment into military services. Finally, non-normative influences are more or less individualized conditions and events (e.g., rare illnesses, winning a lottery).

Between-Person Differences in Old-Age Intellectual Change: Risk and Protective Factors

Differences in intellectual performance between individuals are pronounced in groups of older individuals. In addition to stable differences in levels of intellectual performance, which are large throughout life, between-person differences in intellectual changes are also substantial within old age and are actually more pronounced in old age than during earlier periods of the adult life span. Furthermore, for the periods of old and very old age, between-person differences in change tend to be greater for the fluid mechanics than for the crystallized pragmatics, suggesting a predominantly biological origin. The following sections list some of the causal factors that contribute to the diversity of developmental patterns in intellectual functioning observed in old and very old age.

Terminal Decline

One important source of between-person differences in intellectual development in old age is terminal decline, denoting changes associated with impending death. These changes may reflect causal structures other than those underlying normative age-graded changes, for example, specific cognition-influencing diseases or global breakdowns of the biological system. Mounting evidence points to an association between performance on intelligence tests and longevity as well as between changes in intelligence and mortality. Importantly, directionality dedifferentiation might be driven by a subset of individuals in close proximity to death. Put differently, in a person entering the terminal decline phase, the crystallized pragmatics may grow increasingly dependent on the fluid mechanics, whereas the intellectual ability structure of a person of the same age who is more distant from death may remain more differentiated.

The existence of mortality-related changes in intelligence is suggestive of profound heterogeneity in development and warns against the uncritical practice of averaging individual differences in intelligence over a particular age. Average trends based on groups that are made up of mixtures of different types of individuals may yield a picture of intellectual development in old age that approximates, at best, an unknown proportion of the individuals constituting

this mixture. Thus, a focus on the individual, or at least on subgroups of individuals, is needed to understand intellectual development in late adulthood and old age. In the case of distance to death, aging-related changes and dying-related changes are confounded when performances of relatively healthy individuals and individuals experiencing terminal decline are averaged. Of course, if some of the causal structures promoting intellectual decline in old age are indeed mortality related, one would expect most individuals to sooner or later evince a pattern of dedifferentiation; that is, from a life span perspective, normally aging and terminally declining individuals do not denote mutually exclusive groups, but different sections of a developmental pathway.

Preclinical Dementia

Dementing illnesses, such as dementia of the Alzheimer type, are often preceded by a prolonged preclinical phase. During this period of time, dementia-related cognitive deficits are present but have not yet reached a diagnostic threshold. For instance, cognitive impairments several years prior to diagnosis have been shown to be sensitive markers of later dementia diagnosis. Preclinical cognitive deficits tend to be global, with impairments in episodic memory apparently being most prominent. Furthermore, impairments in functions that are normally relatively well preserved in old age, such as the crystallized pragmatics, may distinguish early dementia from aging. Given the accelerating increase of dementia incidence in old age, the proportion of individuals with preclinical dementia in the population is also bound to increase. This age-associated increase in the proportion of individuals suffering from preclinical dementia has led researchers to reconsider conclusions about normative age-related changes in intelligence. In analogy to removing individuals with terminal decline, removing individuals who later will develop dementia reduces the estimated size of the negative relation between intellectual performance and age.

Other Diseases

Many specific health-related conditions that affect intellectual performance increase in old age. Much research has addressed the effects of disorders to the circulatory system on intellectual performance. In general, this research has shown that circulatory disturbances, ranging from hypertension and cardiovascular conditions to stroke and vascular dementia, have profound effects on the brain and intellectual performance, especially in the fluid mechanics. Medial-temporal structures such as the hippocampus and related intellectual functions such as episodic memory appear to be especially sensitive to cardiovascular conditions including hypertension.

Elderly adults with diabetes tend to evince impaired intellectual performance. Furthermore, non-diabetic older individuals who have impaired glucose tolerance tend to show similar but less pronounced impairments in performance. Thus, there might be a continuum of intellectual performance deficits in diabetes that progresses from subclinical to the clinical phase.

As in earlier age periods, depression is related to lower performance on intelligence tests in old age. Again, the severity of depressive symptoms is related to intellectual performance, with individuals suffering from depressive symptoms performing at higher levels than clinically depressed individuals but at lower levels than non-symptomatic individuals.

Finally, various markers related to nutrition show an association with intellectual performance in old age. For example, relatively small (e.g., subclinical) deficiencies in vitamin B_{12} and folic acid appear to negatively affect intellectual performance.

Engaged Lifestyle

On the positive side, older individuals living an engaged and active life on average perform better on tests of intelligence than less active individuals. In addition, changes in intellectual performance in old age are associated with changes in lifestyle factors, such as engagement in leisure activities, activities in a social context, and intellectually stimulating activities. When analyzing longitudinal data from the Berlin Aging Study with dynamic modeling techniques, Martin Lövdén, Paolo Ghisletta, and Ulman Lindenberger recently provided direct evidence for the widely held but untested assumption that staying mentally and socially active attenuates intellectual decline in old and very old age.

The mechanisms through which engaged lifestyles influence intellectual development in old age are not yet well understood. The disuse hypothesis proposes that changes in lifestyle during old age (e.g., retirement, loss of spouse, and subsequent social isolation) may result in reduced levels of mental stimulation and subsequent magnification of cognitive decline. The cognitive reserve hypothesis asserts that life experiences during earlier periods of the life span, such as educational attainment, provide greater readiness for compensatory changes in response to age-related and disease-related neurophysiological decline and thus contribute to the resilience of late-life intelligence. Finally, lifestyle factors may also modify the course of neurophysiological changes underlying intellectual aging in more direct ways. For instance,

mechanisms of neuronal plasticity (see below) may function more efficiently in individuals with engaged lifestyles.

Most likely, several of these causal paths are operating in concert. Some of the behaviors associated with engaged lifestyle, such as physical activity and healthy nutrition, may reduce age-related negative influences on non-neural components of the brain, such as vascular changes including decreased blood flow, oxygen extraction, and glucose transport. For instance, mental stimulation may directly influence neurophysiological processes, whereas physical exercise may improve brain functioning indirectly but effectively through the cardiovascular route. Recent evidence based on animal models suggests that the two forms of stimulation may interact and reinforce each other.

Genetic Influences

Examination of associations between genetics and intellectual functioning in late life has been divided into two types. One class of studies has examined the overall influence of genetic variation by comparing dizygotic and monozygotic twins. In general, heritability in intellectual functioning (i.e., between-person differences accounted for by genetic differences) increases from about 35% during childhood to about 80% in middle adulthood. In old age, heritability tends to decrease to about 60%, perhaps pointing to a gradual breakdown of genome expression during the postreproductive period of the human life span. The other class of studies has focused on the role of specific genetic polymorphisms on intellectual functioning in old age. Here, the gene coding for the apolipoprotein E (APOE) has received most attention because carriers of a variant of this gene are considerably more likely to develop dementia. Even in samples of older adults screened for dementia, genetic variations in the APOE polymorphism are related to intellectual functioning. It remains to be seen whether this association reflects the presence of preclinically demented individuals. The influence of genetic variations on individual differences in neuronal organization and intellectual functioning is likely to become an active area of research in coming years.

Intellectual Plasticity in Old Age

Intellectual plasticity, or the ability to improve one's intellectual performance through experience (e.g., learning), continues to be present in healthy old age. For instance, training programs targeted at intellectual performance in the domain of the fluid mechanics do indeed lead to performance improvements. However, training gains are considerably smaller in older adults than in young adults. For instance, after instruction and training in a mnemonic technique for the recall of word lists, the distributions of young and older adults' memory performances become close to non-overlapping. In very old age, training gains are further restricted in size and compromised in quality.

Intellectual plasticity in old age probably reflects, for the most part, reactivation of existing and, to some extent, acquisition of novel intellectual strategies, rather than modification of fluid mechanic efficiency per se. As a consequence, positive transfer of training gains in a specific intellectual task to other tasks from the same or related intellectual abilities is generally small or absent. On a more promising note, recent aerobic fitness interventions with healthy older adults have been found to positively affect performance on a range of cognitive tasks that impose high demands on executive functions or cognitive control, that is, on mechanisms that are at the heart of the fluid mechanics. These positive changes appear to be accompanied by structural and functional alterations in relevant parts of the brain such as areas in the prefrontal cortex. Future research needs to further examine the amount, mechanisms, and moderators of neuronal and behavioral plasticity in late adulthood and old age.

Conclusion

Intelligence is a multidimensional construct that undergoes structural transformation from early to late adulthood and advanced old age. Two-component theories of life span intelligence posit two broad systems of influence, one biological and the other cultural. In early adulthood, the biology-based fluid mechanics and the culture-based crystallized pragmatics of intelligence are loosely coupled and display different age gradients, with decline for the fluid mechanics and stability or increase for the crystallized pragmatics. In old and very old age, declines in the fluid mechanics compromise the acquisition, expression, and representation of the pragmatic knowledge. The onset and nature of this transition differ from person to person and may be more dependent upon distance from death or cognition-related pathology than upon distance from birth. In the course of their lives, individuals experience unique constellations of normative, age-graded, and nonnormative influences. Intelligence is not necessarily structured similarly in different individuals just because they happen to be born at similar points in time. General regularities across groups of aging individuals in patterns of intellectual change in old and very old age can be discerned after sources of individual differences, such as

age-related pathology and terminal decline, have been identified and taken into account. Thus, understanding intellectual development in old age requires a multivariate, dynamic, and person-centered perspective.

See also: Information Processing/Cognition; Learning; Life Span Theory.

Further Reading

Baltes PB (1987) Theoretical propositions of life-span developmental psychology: on the dynamics between growth and decline. *Developmental Psychology* 23: 611–626.

Baltes PB, Lindenberger U, and Staudinger UM (in press) Life-span theory in developmental psychology. In: Lerner RM (ed.) *Handbook of Child Psychology*, 6th edn. *Vol. 1. Theoretical Models of Human Development*. New York: Wiley.

Cabeza R, Nyberg L, and Park DC (eds.) (2004) *Cognitive Neuroscience of Aging: Linking Cognitive and Cerebral Aging*. New York: Oxford University Press.

Colcombe SJ, Kramer AF, Erickson KI, Scalf P, McAuley E, Cohen NJ, *et al.* (2004) Cardiovascular fitness, cortical plasticity, and aging. *Proceedings of the National Academy of Sciences* 101: 3316–3321.

Craik FIM and Salthouse TA (eds.) (2000) *The Handbook of Aging and Cognition*, 2nd edn. Mahwah, NJ: Lawrence Erlbaum Associates.

Ghisletta P and Lindenberger U (2003) Age-based structural dynamics between perceptual speed and knowledge in the Berlin Aging Study: direct evidence for ability dedifferentiation in old age. *Psychology and Aging* 18: 696–713.

Li S-C, Lindenberger U, and Sikström S (2001) Aging cognition: from neuromodulation to representation. *Trends in Cognitive Science* 5: 479–486.

Li S-C, Lindenberger U, Hommel B, Aschersleben G, Prinz W, and Baltes PB (2004) Transformations in the couplings among intellectual abilities and constituent cognitive processes across the life span. *Psychological Science* 15: 155–163.

Lindenberger U (2001) Lifespan theories of cognitive development. In: Smelser NJ and Baltes PB (eds.) *International Encyclopedia of the Social and Behavioral Sciences*, Vol. 13, pp. 8848–8854. Amsterdam: Elsevier Science.

Lövdén M and Lindenberger U (2005) Development of intellectual abilities in old age: From age gradients to individuals. In: Wilhelm O and Engle RW (eds.) *Handbook of Understanding and Measuring Intelligence*, pp. 203–221. Thousand Oaks, CA: Sage.

Lövdén M, Ghisletta P, and Lindenberger U (2004) Cognition in the Berlin Aging Study: the first ten years. *Aging, Neuropsychology, and Cognition* 11: 104–133.

Lövdén M, Ghisletta P, and Lindenberger U (2005) Social participation attenuates decline in perceptual speed in old and very old age. *Psychology and Aging* 20: 423–434.

Schaie KW (1962) A field-theory approach to age changes in cognitive behavior. *Vita Humana* 5: 129–141.

Schaie KW (1996) *Intellectual Development in Adulthood. The Seattle Longitudinal Study*. New York: Cambridge University Press.

Sliwinski M, Lipton RB, Buschke H, and Stewart W (1996) The effects of preclinical dementia on estimates of normal cognitive functioning in aging. *Journals of Gerontology: Psychological Sciences and Social Sciences* 50B: P162–P170.

Small BJ and Bäckman L (1999) Time to death and cognitive performance. *Current Directions in Psychological Science* 8: 168–172.

Tetens JN (1777) *Philosophische Versuche über die menschliche Natur und ihre Entwicklung* [Philosophical essays about human nature and its development]. Leipzig: Weidmanns Erben und Reich.

Wohlwill JF (1973) *The Study of Behavioral Development*. New York: Academic Press.

ENCYCLOPEDIA OF
GERONTOLOGY

SECOND EDITION

EDITED BY

JAMES E. BIRREN

ELSEVIER

Amsterdam • Boston • Heidelberg • London • New York • Oxford
Paris • San Diego • San Francisco • Singapore • Sydney • Tokyo
Academic Press is an imprint of Elsevier

Academic Press is an imprint of Elsevier
The Boulevard, Langford Lane, Kidlington, Oxford OX5 1GB, UK
525 B Street, Suite 1900, San Diego, CA 92101-4495, USA

First edition 1996

Copyright © 2007 Elsevier Inc. All rights reserved.

The following articles are US Government works in the public domain and not subject to copyright:
Accelerated Aging
Atherosclerosis
Disability, Functional Status and Activities of Daily Living
Falls
Sleep

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 written permission of the publisher

Permissions may be sought directly from Elsevier's Science & Technology Rights Department in Oxford, UK: phone (+44) (0) 1865 843830; fax (+44) (0) 1865 853333; email: permissions@elsevier.com. Alternatively you can submit your request online by visiting the Elsevier web site at http://elsevier.com/locate/permissions, and selecting *Obtaining permission to use Elsevier material*

Notice
No responsibility is assumed by the publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, in particular, independent verification of diagnoses and drug dosages should be made

Library of Congress Catalog Number: 2006935560

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

ISBN–13: 978-0-1237-0530-3
ISBN–10: 0-12-370530-4

For information on all Elsevier publications
visit our website at books.elsevier.com

Printed and bound in the United Kingdom.

07 08 09 10 10 9 8 7 6 5 4 3 2

Working together to grow
libraries in developing countries
www.elsevier.com | www.bookaid.org | www.sabre.org
ELSEVIER BOOK AID International Sabre Foundation