Human Cognitive Aging

Maintenance Versus Dedifferentiation

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***Abstract*— Human cognitive aging differs between and is malleable within individuals. In the absence of a strong genetic program, it is open to a host of hazards, such as vascular and metabolic risk, but also open to protective and enhancing factors, such as experience-dependent cognitive plasticity. Longitudinal studies suggest that leading an intellectually challenging, physically active, and socially engaged life might mitigate losses and consolidate gains, but results need to be interpreted with caution, as individuals are not randomly assigned to lifestyles. In this presentation, I will report on (i) the degree to which individual differences in cognitive decline generalize across abilities; (ii) the role education in adult cognitive development; (iii) the search for domain-general causes of cognitive aging that reduce the distinctiveness of representations and processing pathways (i.e., dedifferentiation); (iv) brain maintenance as a potential mechanism for mitigating dedifferentiation and cognitive decline.**

Keywords-cognitive aging; individual differences; education; dedifferentiation; maintenance

I. INTRODUCTION

Human cognitive aging is likely to reflect limitations in somatic maintenance, resulting in buildup of damage. For any given individual, the shape and course of cognitive aging has been sampled from a range of potential developmental trajectories available to that person. Hence, modifiers and modulators are of key importance.

The recent history of the study of human cognitive aging can be subdivided into three overlapping periods. Following conceptual observations by Tim Salthouse [1] and others, researchers in the 1980s began to realize that they tend to interpret adult age differences in cognition in terms of specific experimental paradigms instead of attempting to identify causal mechanisms that generalize across paradigms. Accordingly, the 1990s were marked by a search for "cognitive resources" whose age-related declines might act as a common cause on multiple manifestations of cognitive aging. However, the postulated resources often did not have a clear biological or conceptual meaning, and the attempts to identify them empirically were generally based on statistical methods that cannot capture the dimensionality of change [2, 3, 4]. Fortunately, starting in the early 2000s, increasing connections between cognitive aging research and neuroscience led to the emergence of a new research field, the cognitive neuroscience of aging [5, 6]. In the following, I will report important findings and current debates from this field.

II. THE DIMENSIONALITY OF COGNITIVE CHANGE IN ADULTHOOD

With advancing age, healthy adults typically exhibit decreases in performance across many different cognitive abilities such as memory, processing speed, spatial ability, and abstract reasoning. However, there are marked individual differences in rates of cognitive decline, with some adults declining steeply and others maintaining high levels of functioning. To move toward a comprehensive understanding of cognitive aging, it is critical to know whether individual differences in longitudinal changes interrelate across different cognitive abilities.

In a recent meta-analysis, Elliot Tucker-Drob, Andreas Brandmaier, and I investigated the degree to which cognitive changes are correlated across different cognitive abilities [7]. We identified 89 effect sizes representing shared variance in longitudinal cognitive change from 22 unique datasets composed of more than 30,000 unique individuals, which we meta-analyzed using a series of multilevel metaregression models. An average of 60% of the variation in cognitive changes was shared across cognitive abilities. Shared variation in changes increased with age, from approximately 45% at age 35 years to approximately 70% at age 85 years. There was a moderate-to-strong correspondence between the extent to which a variable indicated general intelligence and the extent to which change in that variable indicated a general factor of aging-related change. Shared variation in changes did not differ substantially across cognitive ability domain classifications. In a sensitivity analysis based on studies that carefully controlled for dementia, shared variation in longitudinal cognitive changes remained at upward of 60%, and age-related increases in shared variation in cognitive changes continued to be evident.

These results together provide strong evidence for a general factor of cognitive aging that strengthens with advancing adult age. As next steps, cognitive researchers need to identify the dimensionality of brain changes [8|, and link the two change spaces to one another [9].

III. THE ROLE OF EDUCATION IN COGNITIVE AGING

It has been proposed that education early in life might mitigate cognitive decline. Recently, Martin Lövdén and

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colleagues reviewed the available evidence [10]. They confirmed that (i) educational attainment has positive effects on cognitive function; (ii) cognitive abilities are associated with selection into longer education; (iii) common factors, such as parental socioeconomic resources, affect both educational attainment and cognitive development. However, contrary to widely held assumptions, associations between education and aging-associated cognitive declines were found to be negligible.

Based on this evidence, the authors conclude that educational attainment exerts its influences on late-life cognitive function primarily by contributing to individual differences in early adult cognitive skills that persist into older age. It follows that improving the conditions that shape development during the first decades of life carries great potential for improving cognitive ability levels in early adulthood and for reducing public health burdens related to cognitive aging and dementia development.

IV. A Dedifferentiation View of Cognitive Aging

About two decades ago, Shu-Chen Li and I introduced a connectionist model of cognitive aging [11, 12], based on the observation that dopaminergic neuromodulation decreases throughout adulthood and old age. According to the model, suboptimal neuromodulation leads to less distinct representations and processing pathways [13]. The model accounts for a wide range of key cognitive aging phenomena, such as decrements in mean levels of performance, increase in between-person differences, increase in within-person fluctuations, increase in covariation between tasks across individuals, and greater age-related deficits with more difficult tasks.

Following pioneering functional magnetic resonance imaging (fMRI) work of Denise Park and colleagues [14], recent advances in fMRI allow for testing the proposition that representations become less distinct with advancing age. In line with this prediction, unpublished work by Myriam Sander and colleagues indicates that the stability or self-similarity of neural representations at the item level, relative to their similarity to other items of the same category, is negatively related to adult age, and positively related to memory performance [15].

V. Brain Maintenance

Cognitive aging researchers have searched for general mechanisms whose operations might attenuate adult cognitive decline. The notion of brain maintenance, proposed by Lars Nyberg and others, rests on the observation that individuals who show a relative lack of senescent brain changes also show more youth-like brain activation patterns and higher levels of cognitive performance [16]. Brain maintenance appears to operate both at the general level of brain metabolism [17] and at the level of specific circuits and functions, such as the hippocampal formation [18]. Physical exercise is likely to foster brain maintenance by reducing vascular risks [19].

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References

[1] T. A. Salthouse, A Theory of Cognitive Aging. Amsterdam: North-Holland, 1985.

[2] U. Lindenberger and U. Pötter, „The complex nature of unique and shared effects in hierarchical linear regression: Implications for developmental psychology,“ Psychological Methods, 3, pp. 218–230, 1998.

[3] S. M. Hofer, and M. J. Sliwinski, „Understanding ageing: An evaluation of research designs for assessing the interdependence of ageing-related changes,“ Gerontology, 47, pp. 341–352, 2001.

[4] U. Lindenberger, T. von Oertzen, P. Ghisletta, and C. Hertzog, „Cross-sectional age variance extraction: What's change got to do with it?,“ Psychology and Aging, 26, pp. 34–47, 2011.

[5] Cognitive Neuroscience of Aging, 1st ed., R. Cabeza, L. Nyberg, and D. Park, Eds. New York: Oxford University Press, 2005.

[6] U. Lindenberger, „Human cognitive aging: Corriger la fortune?,“ Science, 346, pp. 572–578, 2014.

[7] E. M. Tucker-Drob, A. M. Brandmaier, and U. Lindenberger, „Coupled cognitive changes in adulthood: A meta-analysis,“ Psychological Bulletin, 145, pp. 273–301, 2019.

[8] N. Raz, U. Lindenberger, K. M. Rodrigue, K. M. Kennedy, D. Head, A. Williamson, C. Dahle, D. Gerstorf, and J. D. Acker, „Regional brain changes in aging healthy adults: General trends, individual differences and modifiers,“ Cerebral Cortex, 15, pp. 1676–1689, 2005.

[9] R. A. Kievit, J.-W. Romeijn, L. J. Waldorp, J. M. Wicherts, H. S. Scholte, and D. Borsboom, „Mind the gap: A psychometric approach to the reduction problem,“ Psychological Inquiry, 22, pp. 67–87, 2011.

[10] M. Lövdén, L. Fratiglioni, M. M. Glymour, U. Lindenberger, and E. M. Tucker-Drob, ”Education and cognitive functioning across the lifespan,” Psychology in the Public Interest, in press.

[11] S.-C. Li and U. Lindenberger, „Cross-level unification: A computational exploration of the link between deterioration of neurotransmitter systems and dedifferentiation of cognitive abilities in old age,“ in Cognitive Neuroscience of Memory, L.-G. Nilsson and H. J. Markowitsch, Eds. Seattle: Hogrefe & Huber, 1999, pp. 103–146.

[12] S.-C. Li, U. Lindenberger, and S. Sikström, „Aging cognition: From neuromodulation to representation,“ Trends in Cognitive Sciences, 5, pp. 479–486, 2001.

[13] S.-C. Li and S. Sikström, „Integrative neurocomputational perspectives on cognitive aging, neuromodulation, and representation,“ Neuroscience & Biobehavioral Reviews, 26, pp. 795–808, 2002.

[14] D. C. Park, T. A. Polk, R. Park, M. Minear, A. Savage, and M. R. Smith, „Aging reduces neural specialization in ventral visual cortex,“ Proceedings of the National Academy of Sciences, 101, pp. 13091–13095, 2004.

[15] M. Kobelt, V. R. Sommer, A. Keresztes, M. Werkle-Bergner, and M. C. Sander, „Reduced stability of neural representations in early visual regions predict older adults' memory performance,“ unpublished.

[16] L. Nyberg and U. Lindenberger, “Brain maintenance and cognition in old age,” in The Cognitive Neurosciences, 6th ed., D. Poeppel, G. Mangun, and M. Gazzaniga, Eds. Cambridge, MA: MIT Press, 2020.

[17] N. Raz and A. M. Daugherty, „Pathways to brain aging and their modifiers: Free-Radical-Induced Energetic and Neural Decline in Senescence (FRIENDS) Model – A mini-review,“ Gerontology, 64, pp. 49–57, 2018.

[18] L. Nyberg and S. Pudas, „Successful memory aging,“ Annual Review of Psychology, 70, pp. 219–243, 2019.

[19] M. W. Voss, C. Soto, S. Yoo, M. Sodoma, C. Vivar, H. van Praag, „Exercise and hippocampal memory systems,“ Trends in Cognitive Sciences, 23, pp. 318–333, 2019.