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Editorial

Aging of the brain, sensorimotor, and cognitive processes☆

Aging is becoming an important research focus of various disciplines of the life and behavioral sciences in the 21st century. Average life expectancy at birth in most developed countries has increased from about 45 years in 1900 to about 75 years in 1990 [10]. Alas, this impressive 30-year gain in longevity is not necessarily accompanied by maintenance of a high level of cognitive and sensorimotor 'fitness' in old age. A major challenge for research on aging is not only to give age to life, but also to give life to old age-helping old individuals to maintain their cognitive and sensorimotor functioning by reducing decline or enhancing individual and environmental compensatory functions [2,3,29]. Therefore, with the rapid growth of aging populations worldwide there is an urgent need to obtain integrated understanding of mechanisms and processes contributing to cognitive and sensorimotor aging at different levels.

Gerontological research has traditionally been investigated by researchers working in various subdisciplines, each focusing on a specific level or aspect of aging. Current journals of aging and gerontology are commonly subdivided into separate sections for the medical/biological sciences and for the behavioral/psychological sciences. These disciplinary boundaries notwithstanding, the phenomena of aging occur simultaneously in multiple aspects and at multiple levels.

Regarding aging at the neurobiological level, processes of brain aging can be characterized by a number of neurobiological changes, including anatomical, metabolic, neurochemical changes, as well as changes in functional circuitry [1,6,24,27]. With respect to anatomical changes, for decades a common view has been that cortical neurons are lost during aging and that this may lead to cognitive decline [5]. However, more recent studies have revealed much stability in the number of cortical neurons during normal aging (see Refs. [20,21] for reviews). Moreover, in some cases neurogenesis in response to experience- and environment-dependent stimulation has demonstrated preserved neuronal plasticity in the aging brain (see Refs. [14,23] for reviews). However, the question remains: In the absence of gross structural changes, what other specific structural and neurochemical changes underlie the functional impairments seen in old people? It has been suggested that while there is no global loss, the number of certain subpopulations of neurons might decline. Substantial reduction of specific dendritic spines has also been recently demonstrated in the prefrontal cortex [22]. There is also aging-related decline in various neurotransmitter systems. For instance, molecular shifts in intact circuits have been found in the dentate gyrus of the monkey: While hippocampal information processing can deteriorate during normal aging in the absence of significant neuronal loss, there is evidence suggesting that circuit-specific alteration in the ratio of NMDA receptors may underlie memory impairments in old age [13]. Aging-related decline of the catecholaminergic system, particularly dopamine, in various striatal and extrastriatal regions and the functional significance of the ensuring impaired dopaminergic modulation on cognitive and sensorimotor deficits have been intensely studied (see Refs. [1,11,30] for reviews). Most recently, functional neuroimaging studies have shown adult age differences in the brain's functional circuitry on a large scale. For instance, there is new empirical consensus that many processes taking place separately in the two hemispheres in young adults co-activate both hemispheres in old people. The interpretations of such findings are currently being discussed. Some accounts suggest compensatory plasticity [6,25] and others non-selective recruitment [17].

Brain aging has consequences for various aspects of sensorimotor and cognitive functions. Moreover, sensorimotor and cognitive processes may not be as segregated as previously thought. Theories of motor learning and postural control [19,33] postulate that these processes involve complex and dynamic neural computation integrating multiple sensory inputs and motor commands, and that attentional and executive cognitive control processes may be implicated in the integration process. Furthermore, besides the bottom-up effects from neurobiology to behavior and cognition, top-down effects from experiential and environmental influences [14,18,23] on the aging sensorimotor and cognitive processes

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should also not be neglected. Generally, a main concern of applied and clinical gerontological research is interventions that may either delay or ameliorate the impact of aging-related functional decline [8]. In rodents, it is well established that diet and caloric restrictions have a significant effect on longevity. Currently, there is much discussion and research on comparable effects in primates and humans [28,31,32]. There are also other lines of evidence [7,9,12] suggesting that maintaining physical and mental exercise are prerequisites for what has been called 'successful aging' [2,26], although definite answers may only be revealed in the next decades. Taken together, in general there is agreement that aging should not be considered a fixed and immutable process, but that it is amenable to a variety of interventions. However, developing specific treatments or methods to either delay aging processes or to 'rehabilitate' the aging brain will depend on the progress in understanding the relations between basic mechanisms and effects of aging at various levels.

This special issue of *Neuroscience and Biobehavioral Reviews* provides a collection of articles that bring together a broad overview of recent empirical and theoretical developments in the research on aging neurobiological, sensorimotor, and cognitive processes. All of the selected articles review recent findings across at least two levels and/or two aspects of aging phenomena from animal, human, or computational studies. At the substantive level, a sub-selection of the articles addresses the relations between a variety of cognitive processes and specific structural, neurochemical, or functional changes in the aging brain as well as genetic factors. Another sub-selection of the articles focuses on the relation between decline in the somatosensory cortex and sensorimotor processing as well as possible interactions between aging sensorimotor and cognitive processes at various levels. Furthermore, findings reviewed in some of the articles address the issue of preserved plasticity, allowing the aging sensorimotor and cognitive processes to still open to experiential, environmental, and cultural influences. In addition, at the technical level the utilities and challenges associated with using animal (van der Staay in this issue) and computational models (Braver and Barch and S.-C. Li and Sikström in this issue) in aging research are also exemplified. In what follows, we briefly highlight the various substantive topics.

The brain-mind/behavior relations. Specifically, on the brain-mind/behavior continuum [16], aging at the neurobiological level affects sensorimotor and cognitive processes. For instance, structural changes in dendrites and synapses of neurons in layer 1 of the neocortex and the breakdown of myelin sheaths might have implications for cognitive decline such as the slowing of information processing (see Peters in this issue). Aside from structural changes, aging-related neurochemical declines may particularly affect the prefrontal cortex's working memory, attentional, and other cognitive control functions (see Braver and Barch, Kaasinen and Rinne, and S.-C. Li and Sikström in this issue). At the behavioral level, meta-analyses show differential aging effects on different aspects of attention. While there is no age-related deficit specific to selective attention other than what is attributable to the slowing of information processing, divided attention assessed by the dual-task paradigm involves a deficit over and beyond general slowing (see Verhaeghen and Cerella in this issue). Aside from working memory functions and associated processes such as context representation and maintenance that play roles in cognitive control (e.g. Braver and Barch in this issue), there are also aging-related declines in semantic, episodic, and prospective memory. Old people seem to benefit less from encoding and retrieval supports in episodic or semantic memory tasks. Research on the neural and genetic substrates for such effects during normal and pathological aging has begun (see Nyberg et al. and Nilsson et al. in this issue). Aging-related decline of average event-related brain potentials in response to memory cues is related to aging deficits in prospective memory (see West et al. in this issue). As for sensorimotor processes, deterioration in the topographical maps of the rat's hindpaw representation in the somatosensory cortex is related to the walking behavior of old rats (see Godde et al. in this issue).

Relations between sensorimotor and cognitive processes. While traditionally the aging of sensorimotor and cognitive processes have mostly been studied independently, recent experimental behavioral aging data indicate that sensorimotor and cognitive processes might not be as segregated as previously thought. During aging, cognitive involvement in sensory and sensorimotor processes seems to increase. On the one hand, experimentally simulated loss in sensory/sensorimotor inputs affect cognitive performance; and on the other hand, increasing cognitive load affects sensory/sensorimotor performance (see Bock and Schneider and K.Z.H. Li and Lindenberger in this issue). Consider also a different line of evidence: neuronal activity in the motor-brain area (e.g. somatosensory cortex and premotor cortex) seems to subserve the individual's ability to improve their memory for actions under an encoding enactment condition (see Nyberg et al., in this issue). At the neurochemical level, aging-related decline in brain dopamine activity is implicated both in cognitive and sensorimortor impairments [30]. Besides being associated with various cognitive deficits, aging-related decline in dopaminergic modulation of the basal ganglia motor loop [4] is specifically related to movement disorder in Parkinson patients, for instance (see Kaasinen and Rinne, Nyberg et al., and S.-C. Li and Sikström in this issue).

Preserved plasticity. Aside from aging-related decline, there is also preserved plasticity at the neurobiological, cognitive, and behavioral level (see Ref. [15] for review). For instance, there is evidence for experience-/use-dependent plasticity in old rat's somatosensory cortex. When old rats were kept under enriched environment for several months, less aging-related alteration of the hindpaw's representation in the somatosensory cortex was observed (see Godde et al.,

in this issue). In humans, cortical activities associated with various cognitive processes are less asymmetrical in the aging brain which, in some cases, reflect adaptive functional compensation (see Dolcos et al., in this issue). At the information-processing and behavioral level, old people are still flexible in reorganizing and allocating resource between concurrent tasks, although the cost of resource sharing is often greater for older adults, indicating compensatory trade-offs (see K.Z.H. Li and Lindenberger in this issue).

Experiential, environmental, and cultural influences. Given growing evidence for functional plasticity at different levels even in old age, it becomes clear that research efforts should not only be devoted towards exploring the unidirectional, bottom-up neurobiology-to-behavior effects. Studies of reciprocal experiential/environmental influences on aging sensorimotor and cognitive processes should also be emphasized in future research. Current findings give some directions for further investigations in this area. For instance, what are the boundary conditions [2] for old people's performance still able to be enhanced by experiential/environmental support (see Nyberg et al., and Nilsson et al., in this issue)? How may lifelong experience and expertise in specific skills interact with the aging process? (see Krampe in this issue). What are the benefits and methodological challenges in adding a cross-cultural dimension to study the neuroscience of aging (see Park and Gutchess in this issue)?

The aging brain is still endowed with plasticity in its implementations of sensorimotor and cognitive processes and is situated in experiential/environmental contexts. To give 'life' to old age, research fields need to reach toward more comprehensive understandings of the relations between aging mechanisms at different levels. By bringing together the collection of articles in this special issue, we hope to open a forum for broader conceptions of the relations between the different levels and facets of aging phenomena.

References

[1] Arnsten AFT. Catecholamine modulation of prefrontal cortical cognitive function. Trends Cogn Sci 1998;2:436–47.

[2] Baltes PB. The aging mind-potential and limits. Gerontologist 1993;33:580–94.

[3] Baltes PB. On the incomplete architecture of human ontogeny—selection, optimization, and compensation as foundation of developmental theory. Am Psychol 1997;52:366–80.

[4] Bar-Gad I, Bergman H. Stepping out of the box: information processing in the neural networks of the basal ganglia. Curr Opin Neurobiol 2001;11:689–95.

[5] Brody HD. Organization of the cerebral cortex. III. A study of aging in the human cerebral cortex. J Comp Neurol 1955;1023:511–56.

[6] Cabeza R. Hemispheric asymmetry reduction in old adults: the HAROLD model. Psychol Aging 2002;17:85–100.

[7] Compton DM, Bachman LD, Brand D, Avet TL. Age-associated changes in cognitive function in highly educated adults: emerging myths and realities. Int J Geriatr Psychiatry 2000;15:75–85.

[8] Dinse HR. The aging brain. Introductory remarks. In: Elsner N, Kreutzberg GW, editors. The neurosciences at the turn of the century, Stuttgart: Thieme, 2001. p. 356–63.

[9] Elward K, Larson EB. Benefits of exercise for older adults. A review of existing evidence and current recommendations for the general population. Clin Geriatr Med 1992;8:35–50.

[10] Kannisto V. Development of oldest-old mortality, 1950–1990: evidence from 28 developed countries. Odense, Denmark: Odense University Press, 1994.

[11] Kaasinen V, Vilkman H, Hietala J, Nagren K, Helenius H, Olsson H, Farde L, Rinne JO. Age-related dopamine D2/D3 receptor lss in exstrastriatal regions of the human brain. Neurobiol Aging 2000; 21:683–8.

[12] Kramer AF, Hahn S, Cohen NJ, Banich MT, McAuley E, Harrison CR, Chason J, Vakil E, Bardell L, Boileau RA, Colcombe A. Ageing, fitness and neurocognitive function. Nature 1999;400:418–9.

[13] Gazzaley AH, Siegel SJ, Kordower JH, Mufson EJ, Morrison JH. Circuit-specific alterations of *N*-methyl-D-aspartate receptor subunit 1 in the dentate gyrus of aged monkeys. Proc Natl Acad Sci USA 1996;93:3121–5.

[14] Gross CG. Neurogenesis in the adult brain: death of a dogma. Nat Rev 2000;1:67–73.

[15] Li S-C. Biocultural orchestration of developmental plasticity across levels: the interplay of biology and culture in shaping the mind and behavior across the lifespan. Psychol Bull 2002.

[16] Llinas RR, Churchland PS. The mind–brain continuum: sensory processes. Cambridge, MA: MIT Press, 1996.

[17] Logan JM, Sanders AL, Snyder AZ, Morris JC, Buckner RL. Underrecruitment and nonselective recruitment: dissociable neural mechanisms associated with againg. Neuron 2002;33:827–40.

[18] Maguire EA, Gadian DG, Johnsrude IS, Good CD, Ashburner J, Frackowiak RSJ, Frith CD. Navigation-related structural changes in the hippocampi of taxi drivers. Proc Natl Acad Sci USA 2000;97: 4398–403.

[19] Manchester D, Woollacott M, Zederbauer-Hylton N, Marin O. Visual, vestibular, and somatosensory contributions to balance control in the older adults. J Gerontol Med Sci 1989;44:M118–27.

[20] Morrison JH, Hof PR. Life and death of neurons in the aging brain. Science 1997;278:412–29.

[21] Peters A, Morrison JH, Rosene DL, Hyman BT. Feature article: are neurons lost from the primate cerebral cortex during normal aging? Cereb Cortex 1998;8:295–300.

[22] Peters A, Sethares C, Moss MB. The effects of aging on layer 1 in area 46 of prefrontal cortex in the rhesus monkey. Cereb Cortex 1998; 8:671–84.

[23] van Praag H, Christie BR, Sejnowski TJ, Gage FH. Running enhances neurogenesis, learning, and long-term potentiation in mice. Proc Natl Acad Sci USA 1999;96:13427–31.

[24] Raz N. Aging of the brain and its impact on cognitive performance. In: Craik FIM, Salthouse TA, editors. Handbook of aging and cognition, Erlbaum, 2000. p. 1–90.

[25] Reuter-Lorenz PA, Jonides J, Smith EE, Hartley A, Miller A, Marshuetz C, Koeppe RA. Age differences in the frontal lateralization of verbal and spatial working memory revealed by PET. J Cogn Neurosci 2000;12:174–87.

[26] Rowe JW, Kahn RL. Human aging: usual and successful. Science 1987;237:143–9.

[27] Schneider EL, Rowe JW, Johnson TE, Holbroo NJ, Morrison JH, editors. Handbook of the biology of aging New York: Academic Press, 1996.

[28] Sohal RS, Weindruch R. Oxidative stress, caloric restriction, and aging. Science 1996;273:59.

[29] Stern PC, Carstensen LL, editors. The aging mind: opportunities in cognitive research Washington DC: National Academy Press, 2000.

[30] Volkow ND, Gur RC, Wang G-J, Fowler JS, Moberg PJ, Ding Y-S, Hitzemann R, Smith G, Logan J. Association between decline in brain dopamine activity with aging and cognitive and motor impairment in healthy individuals. Am J Psychiatry 1998;155:344–9.

[31] Walford RL. The extension of maximum life span. Clin Geriatr Med 1985;1:29–35.
[32] Wanagat J, Allison DB, Weindruch R. Caloric intake and aging: mechanisms in rodents and a study in nonhuman primates. Toxicol Sci 1999;52:35–40.
[33] Wolpert DM, Ghahramani Z, Flanagan JR. Perspectives and problems in motor learning. Trends Cogn Sci 2001;5:487–94.

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