ResearchGate

See discussions, stats, and author profiles for this publication at: https://www.researchgate.net/publication/305083836

Neurocognitive Markers of Aging

Chapter · December 2015

DOI: 10.1007/978-981-287-080-3_302-1

CITATIONS
2

READS
22

2 authors:

Tanya Dash

19 PUBLICATIONS **96** CITATIONS

SEE PROFILE

Yves Joanette

Université de Montréal

362 PUBLICATIONS **8,729** CITATIONS

SEE PROFILE

Some of the authors of this publication are also working on these related projects:

Project Right brian damaged and verbal communication View project

Project Neurofunctional reorganization in healthy aging View project

All content following this page was uploaded by Tanya Dash on 26 November 2020.

The user has requested enhancement of the downloaded file.

N

Neurocognitive Markers of Aging

Tanya Dash[1,2] and Yves Joanette[1]
[1]Centre de recherche, Institut universitaire de gériatrie de Montréal, Montréal, Canada
[2]École d'orthophonie et d'audiologie, Faculté de médecine, Université de Montréal, Canada

Synonyms

Cognitive markers; Neurobiological markers; Neurofunctional markers

Definition

Neurocognitive markers correspond to the components of cognition, along with their neurobiological and neurofunctional bases, that exhibit changes along the trajectory of normal aging.

Introduction

The aging of societies and globalization of activities both characterize the twenty-first century. Sustaining active aging, and particularly cognitive health, is one of the leading global public health priorities (WHO 2015). Aging is a dynamic process that spans across the entire life course. In order to distinguish between the normal evolution of cognition across the lifespan and cognitive impairments due to neurodegenerative diseases, it is critical to recognize the different neurocognitive markers in aging. This entry provides a description of those markers at both the functional and structural level, while also addressing the neurofunctional reorganization that occurs and is responsible for the relative preservation of cognitive abilities in normal aging.

Hedden and Gabrieli (2004) describe the age-related cognitive changes in three broad categories – *lifelong decline, late-life decline*, and *lifelong stability* – thus indicating that not every cognitive function follows a similar trajectory during the aging process. According to their view, a number of cognitive components are characterized by a process of *lifelong decline*, including general speed of processing, working memory (the transient holding of new and already stored information), divided attention (the ability to execute more than one action at a time), executive function (the management of cognitive processes), and encoding of information in episodic memory (memory of events). These components of cognition show a gradual change from early adulthood and continue throughout one's life. On the other hand, components of cognition that are well practiced on a day-to-day basis appear to be affected only in the later decades of life and are thus included in the *late-life decline* category. Examples of such components are short-term memory (the capacity to hold information for a short amount of time) and semantic memory

© Springer Science+Business Media Singapore 2016
N.A. Pachana (ed.), *Encyclopedia of Geropsychology*,
DOI 10.1007/978-981-287-080-3_302-1

(general world knowledge accumulated over one's lifetime). Finally, some components of cognition are comparatively less affected, or show no changes in aging, suggesting that these cognitive skills exhibit *lifelong stability*. Examples of the latter category include autobiographical memory, emotional processing, word knowledge, and automatic memory processes (Hedden and Gabrieli 2004). These three different trajectories of cognitive changes may themselves differ in their lifelong deployment, from a steady decline over the years to a sudden, steeper deterioration at one point during the life course.

Regardless of the lifelong pattern, these age-related changes in cognition result from the complex influence of genetic determinants, which are further shaped by lifelong cognitive experiences such as those involving novel items and skills. At the same time, structural changes in the brain may begin to occur as early as the second decade of life. These changes are a product of some level of genetic, lifelong wear and tear mechanism that is part of the adaptive process. Aging is no exception to the general rule, where systems try to maintain a balance within its subcomponents to give rise to optimal performance. These structural changes are not uniform across aging individuals, or within cognitive functions. In order to recognize the neurocognitive markers in aging, it is pivotal to understand both the neurobiological changes that arise and the neurofunctional adjustments that occur in response to these changes and lifelong experiences. On one hand, the neurobiological changes that affect cognition include the loss of synaptic connections, significant neural loss in different parts of the brain, and synaptic alterations in distinctive cerebral circuits. On the other hand, lifelong changes in the neurofunctional organization of the brain are a product of adaptation to the neurobiological process and fine-tuning through life experiences. These life experiences are a crucial determinant in building what is referred to as *cognitive reserve*, or the ability to perform tasks as efficiently as possible with the use of available resources (Stern 2002). Experiences such as engaging in leisure activities, educational or occupational attainment, learning multiple languages, or skill building all contribute to the cognitive reserve mechanism (Scarmeas and Stern 2003).

Given the importance of ensuring quality of life in old age, there is a need to better understand both the natural course and the determinants of healthy cognitive aging. Understanding the typical neurocognitive changes in aging is essential in order to be able to distinguish between the effects of aging and those of neurological diseases that may occur in aging. Factors affecting these neurocognitive processes can help spread awareness and provide support for active lifestyle changes. The knowledge presently available regarding these questions comes from converging methodologies including psychophysics, behavioral studies, neuroimaging, and electrophysiological studies in the aging population. But prior to studying this question, it is important to discuss what is currently known about cognition and aging.

Cognition and Aging

Age-related changes in the different cognitive domains are relatively heterogeneous throughout the lifespan. Such heterogeneity can be found among different cognitive domains, as well as between individuals (Salthouse 2004). Memory, attention, executive function, visual perception, and language abilities are discussed in this order of priority in the literature related to aging. Different components of cognition show age-related changes that can differ in quality and quantity. Factors such as motivation, adaptation, or persistence can influence the detrimental consequences of cognitive decline due to aging. Furthermore, the magnitude of decline seen in older adults due to anatomical brain changes can be compensated through environmental factors and lifestyle choices. The following sections will discuss the age-related changes in memory, attention, executive functions, visual perception, and language abilities.

Memory

Age-related decline in memory processes can take many trajectories, from linear to curvilinear.

Among the different memory processes, episodic memory and working memory are the most affected by aging (Brickman and Stern 2009).

Working memory enables the processing of information from the current environment and of already stored information within a transient period of time. It is sensitive to age-related changes in executive control processes, speed of processing, and memory load. Distinct areas in the prefrontal cortex are activated in young and older adults, suggesting differences in the execution of working memory tasks (Park and Reuter-Lorenz 2009).

Episodic memory enables the registration of an event in space and time and its key feature lies in its ability to bind different fragments of the event together into a cohesive unit. This process can range from perceptual feature binding to the binding of higher-order memory process, namely, executive functions (Shing et al. 2010). There is an age-related linear and continuous decline in episodic memory from 20 to 60 years of age, followed by a steep deterioration (Brickman and Stern 2009). This decline is not influenced by the stimuli type or modality in which episodic memory is assessed (e.g., face vs. word recall; verbal vs. nonverbal task).

Implicit memory is generally preserved in older adults. Thus, the deficits in implicit memory performance, when present, mostly reflect a decline in processing speed and processing of stimuli. In the aging literature, it is usually assessed with priming tasks (mediated through frontal and occipital regions in aging) and sequence and categorical learning tasks (mediated through frontostriatal regions in aging). However, when implicit memory deficits are present, they appear to be linked with changes in the frontostriatal circuitry (Dennis and Cabeza 2008).

Semantic memory and procedural memory are comparatively spared during the aging process and can even show some improvement with aging (Brickman and Stern 2009). This suggests that decline in semantic memory is relatively slow. Also, its decline is seen only after the seventh decade of life (Brickman and Stern 2009). The tip-of-the-tongue phenomenon is associated with the subjective accounts of memory decline by older adults (Brickman and Stern 2009). In terms of how the brain supports these types of memories, procedural memory depends on the basal ganglia and the cerebellar structures, while semantic memory is related to the posterior neocortex.

Multiple neuroimaging studies report changes in brain activation as a function of aging in areas related to memory (Grady 2008). The literature reports both positive and negative changes in neural activities depending on the task, the type of memory process, and the type of training induced. Grady (2008) highlights the existence of a distributed pattern of brain activation in older adults compared to young adults. Greater activation was reported in the ventral and dorsal prefrontal cortex in older adults during the memory task, and frontal and parietal activation was detected during the attention task. Furthermore, the medial temporal area, and more specifically the hippocampus, showed a reduced activation in memory encoding and retrieval (Grady 2008).

Episodic memory and working memory are the subcomponents of memory that are the most affected in aging. The age-related changes seen in other memory processes are mainly due to general decline in processing speed (Brickman and Stern 2009). In addition to memory, other cognitive domains are affected by aging. Attention and executive functions, which are discussed in the next section, are closely related to memory processes.

Attention and Executive Functions in Aging

Attention is usually conceived under three broad headings: selective attention, divided attention, and sustained attention. In the older population, attentional deficits are more evident in highly demanding situations. Minimal deficits are seen in selecting relevant information (selective attention) and maintaining attention for an extended period of time (sustained attention). In general, divided attention, inhibitory control, and executive attention are three processes that are influenced by aging. Inhibitory processes, which correspond to the ability to ignore irrelevant information in the environment, experience a decline

with aging. These attentional processes are influenced by memory load and executive attention (Ansado et al. 2013). Executive attention is usually discussed with both attentional and memory processes and plays a crucial function in higher-order planning and programming in day-to-day activities. Verhaeghen and Cerella (2002) report higher costs in response time for older adults but only in divided attention tasks (dual-task performance and global task switching cost) with minimal signs of deficits in selective attention. The performance deficits seen in divided attention tasks are not only caused by the effect of general slowing down but are also associated with memory load. Another approach in studying executive attention is by looking at the role of error monitoring in older adults. The anterior cingulate cortex is usually associated with error monitoring in both young and older adults, with increased activation in older adults (Dennis and Cabeza 2008). Paxton et al. (2008) report a shift in performance in older adults from a proactive (more automatic) to a more reactive (stimuli-driven) strategy of cognitive control for effective performance in any task. This is described by multiple neuroimaging studies suggesting compensatory strategies at the neural level. Grady (2008) stresses the importance of frontoparietal regions for attentional processes.

Regarding executive functions, most studies suggest an enhanced activation in different brain areas as a function of age. Enhanced activation is frequently seen in the frontal and parietal areas when performing nonverbal executive function tasks (Madden 2007). Providing a different perspective, Jonides et al. (2000) reported less activation in the prefrontal cortex in older adults. Reduced executive function is mostly influenced by changes in frontostriatal circuits in older adults (Park and Reuter-Lorenz 2009). In addition to greater neural activation in the prefrontal cortex, a decrease in occipital neural activation in older adults during tasks requiring the participant to ignore irrelevant information is also reported (Madden 2007). In contrast to this frontal increase in brain activation, Madden (2007) reports no changes in the activation of frontal regions in older adults in an oddball paradigm; in this particular study, more activation was reported in the striatum, the thalamus, and the insula in a task involving processing of irrelevant stimuli. Similar reduced activation was reported in the left dorsal prefrontal cortex in auditory attention induced by a dichotic listening task (Thomsen et al. 2004). Selective attention, which was measured by a visual search task as well as a Stroop task, was found to be less affected in aging, suggesting an overall decline in the processing of information rather than in selective attention per se. Moreover, Persson et al. (2007) reported an increased degree of deactivation with the increase in selection demand in the default mode network, which was suspended when engaged in a verb generation task.

In summary, the execution of any goal-directed behavior changes with aging. The prefrontal cortex and the anterior cingulate cortex, which show alterations in activation, are considered to be the key areas associated with these changes. One of the major steps in allocating attention stems from the perceptual analysis of internal and external stimuli, which will be the focus of the next section.

Visual Perception and Visual Cognition in Aging

Visual perception comprises visual cognition, perception, and visually guided behavior. The perceptual deficits seen in aging are associated with attentional demands (e.g., load and complexity), semantic memory, and sensory acuity. The trajectory of visual working memory performance peaks in the early twenties and exhibits thereafter a linear decline with age. Studies on the age-related changes in regional cerebral blood flow report an increase in the prefrontal cortex activation coupled with a decrease in occipital activation. This is predominantly seen in ventral visual pathways that are involved in word and face processing (Grady 2008; Madden 2007). Age-related variations are also seen in motion perception in older adults when compared to young adults. This change is more prominent in second-order motion perception (e.g., changes in contrast, flicker, and spatial frequency). The perception of luminance changes, corresponding to

first-order motion perception, shows limited age-related differences. Thus, the increase in the level of complexity in motion perception is a key factor for age-related changes in older adults. In a virtual reality environment, dynamic variations in tracking speed were seen in older adults, a phenomenon that was attributed to capacity limitation. Some neurofunctional studies report changes in the visual cortex as a function of aging during various cognitive tasks. For visually oriented cognitive tasks, enhanced activation was seen in the frontal and occipital regions, whereas the medial temporal lobes (parahippocampal gyrus) showed reduced activation (Greenlee and Sekuler 2014).

In summary, the limitation in attentional resources and the decline in processing speed can partially account for the visual perceptual deficits reported in the literature. However, positive perceptual changes do occur in aging, particularly for more complex visual stimuli.

Language and Aging

Language abilities are considered to be generally stable in aging compared to other cognitive domains. Although, there are considerable reports of decline in language in older adults, comprehension largely shows an unimpaired performance compared to language production. In general, comprehension deficits in older adults are reported in studies carried out in noisy environments, which rather suggests challenges in auditory perception. It is usually supposed that language deficits are due to impairment in central cognitive processes (Shafto and Tyler 2014). Semantic processing is usually preserved at both the sentential and word level. In fact, word knowledge continues to improve with age. Among the brain areas involved in syntactic processing tasks in older adults, the left frontotemporal network shows changes with age in the form of an increase in frontal activities in the right hemisphere, which is even detected in low-load tasks (Shafto and Tyler 2014). On the other hand, there seems to be less of a compensatory increase in prefrontal activities during semantic and phonological tasks, a phenomenon also reported in the attention and memory domains of cognitive processes. Some of the changes seen in language abilities can be considered as resulting from the impact of deficits in working memory, executive attention, and visual/auditory sensory perceptual skills (evident as a modality-specific effect: spoken/written and reading/writing). Thus, language is the cognitive domain that is the least affected in aging. As mentioned previously, some of its components (e.g., word knowledge, discourse skills) may even show improvement with age.

In sum, the different components of cognition are differentially affected by aging within and across cognitive domains. Working memory, episodic memory, divided attention, and executive functions are more susceptible to changes related to aging. These cognitive changes are usually linked to alterations in the frontal regions of the brain. The underlying cognitive and neural architecture of the system and the interrelationship between different cognitive domains are key to understand cognitive aging. The following section addresses the neuroanatomical and neurofunctional bases of aging as a function of these cognitive domains.

Neural Bases of Cognitive Aging

The age-related changes in the neural bases of cognition occur at both the neurobiological and neurofunctional level.

Neurobiological Modifications in Aging

The main neurobiological changes in aging consist of structural and functional changes that largely occur in the medial temporal lobe, the prefrontal cortex, and the white matter tracts. The rate of atrophy varies within and between the lobes of the brain. The frontal, parietal, and temporal lobes show more atrophy compared to the occipital lobe. Within the frontal and parietal cortices, there is a steeper decline in gray matter in the inferior subregions of the lobes (Dennis and Cabeza 2008). Compared to other lobes, the frontal region of the brain shows a faster rate of decline, with an average decline rate of 0.9–1.5 % per annum (Raz et al. 2005). On the other hand, the occipital lobe shows minimal or no significant age-related atrophy (Dennis and

Cabeza 2008). There is a 0.12 % rate of decline per year in the cerebral cortex in young adults, whereas older adults show a 0.35 % decline per year as of 52 years of age (Dennis and Cabeza 2008; Raz et al. 2005). Due to the importance of these structures in memory, specific regions of the temporal lobes are extensively studied such as the entorhinal cortex, the hippocampus, and the parahippocampal gyrus. Raz et al. (2005) carried out a longitudinal study and found considerable atrophy in the hippocampus compared to the entorhinal cortex in healthy adults, a change that is common in Alzheimer's disease. In normal aging, hippocampal atrophy ranges from 1.18 % to 1.85 % per annum after the age of 50 (Raz et al. 2005). In addition to cortical atrophy, there are reports of shrinkage of subcortical (Dennis and Cabeza 2008; Raz et al. 2005) and cerebellar structures (Raz et al. 2005), and of the corpus callosum (Sullivan and Pfefferbaum 2006). In terms of neuronal loss, subcortical structures show maximum change in the caudate nucleus, followed by the putamen and the globus pallidus. Similarly, the cerebellar cortex showed more shrinkage compared to the pons and the vermis. The anterior part of the corpus callosum is more susceptible to change compared to its posterior part (Sullivan and Pfefferbaum 2006). As a result of white and gray matter loss, the rate of expansion of the ventricles increases from 0.43 % in young adults up to 4.25 % in older adults after the age of 70 (Dennis and Cabeza 2008; Raz et al. 2005).

The rate of decline in white matter volume is also said to accelerate after the age of 50 and is characterized by a loss of synaptic connections. Many studies have reported a greater loss of white matter compared to gray matter with increasing age, particularly after the age of 50 (Dennis and Cabeza 2008). Furthermore, atrophy is more important in the anterior regions than in the posterior regions (Sullivan and Pfefferbaum 2006). A loss of white matter and associative track integrity is believed to be associated with the age-related decrease in processing speed (Penke et al. 2010). In fact, the diminished processing speed may be responsible for the age-related deficits in executive attention, episodic memory (Penke et al. 2010), and in the general slowing of performance.

Regarding the regional cerebral metabolic rate of glucose and oxygen, the results reported in the literature are more diverse. Through the use of different methodologies, it has been established that cerebral blood flow is altered as a function of age. Similar results were found for regional cerebral metabolic rate of oxygen (Dennis and Cabeza 2008). More recently, positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies using blood-oxygen-level dependent (BOLD) and hemodynamic response amplitude (HDR) measures have shown that there is more decline in the activation of the visual cortex and less so in the motor cortex. Some studies report stability in hemodynamic responses in face processing up to the ninth decade of life. Changes in the dopaminergic system have also been extensively studied as examples of the age-related modifications in neurotransmitters. The changes in the dopaminergic system seem to be mostly associated with the decline in episodic memory, executive function, and motor performance, and the related deficits appear to be at the level of the striatum. Extrastriatal decline is also evident in the frontal, temporal, and occipital lobes, as well as in the hippocampus and the thalamus (Dennis and Cabeza 2008).

In summary, the anatomical alterations in the form of gray and white matter decline and of changes in cerebral blood flow, neurotransmitters, and hemodynamic responses represent most of the age-related decrement at the structural level. However, cognitive abilities along the age continuum do not appear to change proportionately to the extent of these structural and physiological changes. It is thus important to understand the neurofunctional reorganization that occurs in aging and that is thought to be responsible for this phenomenon.

Neurofunctional Modifications in Aging: Brain-Behavior Interface

Numerous studies using neuroimaging techniques (e.g., PET and fMRI) offer critical information on the neurobiological reorganization in aging

reported in visual perception, memory, attention, and executive function. As summarized in the previous section, the evolution of cognitive performance with age includes a number of cognitive markers such as speed of performance, available resources, recollection of information, processing of sensory information, and inhibition. However, these cognitive changes are in many cases less important than what could be expected given the age-related structural and physiological decline. Such a difference stems from the existence of a neurofunctional reorganization that occurs with age as an expression of constant adaptations. The fact that the brain is capable of a neurofunctional reorganization that limits the decrease in cognitive abilities has introduced the concept of cognitive reserve. The neurofunctional reorganization in brain activation with age has been captured within two observed phenomena and two frameworks. The phenomena described correspond to: (a) the existence of a hemispheric asymmetry reduction in older adults (HAROLD) and (b) the presence of a posterior to anterior shift in activation (PASA). On the other hand, the two frameworks related to the age-related neurofunctional reorganization are the *compensation-related utilization of neural circuit hypothesis* (CRUNCH) and the *scaffolding theory of aging and cognition* (STAC) (Park and Reuter-Lorenz 2009; Dennis and Cabeza 2008).

The HAROLD and PASA phenomena have been introduced to describe the adaptive nature of the aging brain by providing some explanation concerning the localized account of neurofunctional changes.

The PASA phenomenon has been identified in studies showing an anterior shift in brain activation in older adults who maintained an adequate performance in tasks involving attention, working memory, and executive functioning.

The HAROLD phenomenon refers to a decrease in the interhemispheric differences of brain activation in older adults in memory and attentional tasks.

The CRUNCH and STAC frameworks seek to provide some insight on the causes of the neurofunctional reorganization.

The CRUNCH framework suggests that the age-related neurofunctional reorganization in aging occurs as a response to demanding situations when there are cognitive limitations such as deficient memory load or capacity limitation. The CRUNCH principle has been reported in several studies using different cognitive functions and describes a saturation of the system at higher load levels. In the CRUNCH framework, it is suggested that older adults require more resources to process the same amount of information as young adults. CRUNCH complements the STAC framework well, suggesting that a sort of scaffolding process could account for the compensation in cognitive aging. The STAC is thus presented as a recruitment principle of additional areas or networks when the primary areas are less efficient or need assistance. It provides a holistic account of neuronal plasticity in older adults as a response to the neurobiological changes. According to this framework, an alternative network is recruited, which is less focal than in young adults. Thus, the CRUNCH and STAC explain the nature of interaction between "demand" and "supply" in a particular system, aging being only one of the examples.

In sum, the aging brain undergoes significant neurobiological changes. At the same time, there is evidence suggesting that a number of neurofunctional reorganization processes occur during the same period. The question, however, remains in our ability to reconcile the presence of such neurobiological changes with the relatively preserved cognitive abilities in aging and to link them with the ongoing neurofunctional reorganization. This reconciliation requires the introduction of a new concept: cognitive reserve.

Reconciling Structure and Function: Cognitive Reserve

The notion of cognitive reserve has been introduced to explain the existence of functionally appropriate cognitive performance in older individuals, in spite of the known anatomical and physiological changes. The reason for such a mismatch is thought to relate to the existence of

adaptive changes in the brain, also referred to as cognitive reserve, that protect the individual from the adverse effects of aging. According to Stern (2002), the notion of *reserve* can be conceived from either a structural or functional standpoint. As previously discussed, there are many structural differences in the aging brain, such as brain volume, brain size, and decline in synaptic count. However, there is clear evidence of a functional reorganization that allows for the aging brain to perform in an optimal manner. These functional changes, which are considered to *sustain the existence of a cognitive reserve*, include the recourse to neural networks that are less susceptible to disruption and the involvement of previously unused networks or components of networks (Stern 2002). Cognitive reserve is now a widely used concept in the aging literature and has a vital role in contributing to the understanding of some of the determinants of the trajectory of cognitive changes as a function of age. Cognitive reserve can be useful in discussing the reduced effect of age on cognition as a function of life exposure. Life exposure includes education, occupational attainment, and engagement in leisure as well as social activities. At the same time, there are factors that can adversely affect cognitive aging. The resilience to the neurobiological changes and neuropathological damage stems from different adaptive processes essentially corresponding to either a change (1) in cognitive strategies (which include speed of processing, resource allocation, etc.) or (2) in the recruitment of brain networks.

Factors Affecting Cognitive Reserve

Various factors are now known as having a differential impact on the nature and extent of cognitive aging.

Education and occupation – Individuals with higher levels of educational and occupational attainment show less age-related cognitive decline. It is the lifelong process of skill building that results in the development of cognitive reserve (Stern 2002), possibly leading to the selection of more efficient strategies for optimal functioning in old age.

Multilingualism – Multilingual environments also play a crucial role in building a strong cognitive reserve. Cognitive advantages in bilingual and multilingual individuals have been well documented in various studies conducted over the last 10 years (Bialystok et al. 2007). In its simplest term, it is the lifelong ability to cognitively juggle two or more languages that appears to lead to better cognitive flexibility/cognitive control. However, this advantage is not only linked to the knowledge of languages but also to the proficiency level of a particular language, thus adding to the level of cognitive flexibility.

Cognitive training – Another determinant of cognitive reserve consists of the lifelong exposure to cognitive training. Erickson et al. (2007) reported training-induced changes in the dorsal and ventral prefrontal cortex on a dual-task training program in older adults. This could be considered as a way to reduce the age-related cognitive and neural decline. In fact, improvement in older adults has been found with perceptual-cognitive training in motion discrimination and tracking speed thresholds (Legault et al. 2013).

Heterogeneity in Cognitive Aging

Our understanding of the patterns of cognitive aging has evolved over the last decades. Initially, cognitive domains were thought of as modular systems, and thus the influence of one domain on another was rarely addressed. However, it is now known that different cognitive domains can share some basic processes, which explains why changes in some cognitive processes may affect multiple domains (Hedden and Gabrieli 2004; Brickman and Stern 2009; Grady 2008). At the same time, other cognitive processes contribute in a more restricted manner to a specific cognitive domain. With this in mind, it is crucial to describe not only within but also across domain variability. Heterogeneity in the changes occurring between cognitive domains and between individuals can provide information on individual differences in the normal aging process or on differences in various cognitive domains. Heterogeneity is also present in the way a given individual's cognitive pattern evolves over time (Salthouse 2004). As discussed in earlier sections of this entry, there

are cognitive domains that are more affected than others (executive function, episodic memory, working memory > visual perception, language, semantic memory) (Grady 2008). However, beyond the existence of actual differences in the age-related changes between cognitive domains, and within these domains along the age continuum, there are also many possible sources of variability among studies reporting changes in the different components of cognition in aging that have to be taken into consideration. These sources include: (1) different types of variables measured, (2) different sample sizes, (3) longitudinal vs. cross-sectional studies, (4) differences in the amount of cognitive demand required per task, and (5) lack of control over extraneous variables (e.g., education, occupation, skill-set learning, cardiovascular disorders, diabetes, etc.) (Valdois et al. 1990).

Conclusion and Future Directions

Aging is undeniably associated with changes in cognition. However, not all cognitive abilities are changing in the same way during the aging process. In some cases such as language, some skills are nearly unaffected by aging, while other aspects of cognition show significant decline. However, in general, the magnitude of the changes in cognitive abilities in aging is not as important as it could be predicted by the extent of the changes in the brain. The disparity between the neurobiological and cognitive dimensions of aging is reassuring as it expresses the existence of neurofunctional processes that allow the aging brain to benefit from its lifelong learning and experiences and to engage in such a way that it can achieve similar or even superior cognitive performance compared to young adults, with an apparently diminished neurobiological basis. Some of the obvious determinants of this remarkable evolution are beginning to emerge such as education, bilingualism/multilingualism, and cognitive training, which are collectively referred to as contributing to cognitive reserve. It will become important to better understand these determinants of cognitive reserve and the exact link between such lifelong activities and their impact on cognitive abilities. It will also be important to enhance the efficiency of such measures in order to counteract the impact of the aging brain for as long as possible. The neurocognitive markers of aging are starting to be better known – research should now turn its focus toward understanding the underlying determinants of age-related changes in cognition and empowering individuals to curb the impact of aging and brain diseases in aging as much as possible.

Cross-References

- ▶ Aging and Inhibition
- ▶ Aging and Quality of Life, Aging and Attention
- ▶ Aging and Semantic Memory
- ▶ Cognitive and Brain Plasticity in Old Age
- ▶ Cognitive Changes
- ▶ Executive Functions
- ▶ Memory: Autobiographical
- ▶ Memory: Episodic
- ▶ Memory: Implicit
- ▶ Memory: Training Methods and Benefits
- ▶ Normal and Age-Related
- ▶ Working Memory in Older Age

References

Ansado, J., Monchi, O., Ennabil, N., Deslauriers, J., Jubault, T., Faure, S., & Joanette, Y. (2013). Coping with task demand in aging using neural compensation and neural reserve triggers primarily intra-hemispheric-based neurofunctional reorganization. *Neuroscience Research, 75*(4), 295–304.

Bialystok, E., Craik, F. I., & Freedman, M. (2007). Bilingualism as a protection against the onset of symptoms of dementia. *Neuropsychologia, 45*(2), 459–464.

Brickman, A. M., & Stern, Y. (2009). Aging and memory in humans. In L. R. Squire (Ed.), *Encyclopedia of neuroscience* (pp. 175–180). Oxford: Academic.

Dennis, N. A., & Cabeza, R. (2008). Neuroimaging of healthy cognitive aging. In F. I. M. Craik & T. A. Salthouse (Eds.), *Handbook of aging and cognition* (3rd ed., pp. 1–54). Mahwah: Erlbaum.

Erickson, K. I., Colcombe, S. J., Wadhwa, R., Bherer, L., Peterson, M. S., Scalf, P. E., . . . Kramer, A. F. (2007). Training-induced plasticity in older adults: Effects of

training on hemispheric asymmetry. *Neurobiology of Aging*, *28*(2), 272–283.

Grady, C. L. (2008). Cognitive neuroscience of aging. *The Annals of the New York Academy of Sciences, 1124*(1), 127–144.

Greenlee, M. W., & Sekuler, A. B. (2014). Visual perception and visual cognition in healthy and pathological ageing. *Frontiers in Psychology, 5*, 348.

Hedden, T., & Gabrieli, J. D. (2004). Insights into the ageing mind: A view from cognitive neuroscience. *Nature Reviews. Neuroscience, 5*(2), 87–96.

Jonides, J., Marshuetz, C., Smith, E., Reuter-Lorenz, P., Koeppe, R., & Hartley, A. (2000). Age differences in behavior and PET activation reveal differences in interference resolution in verbal working memory. *Journal of Cognitive Neuroscience, 12*(1), 188–196.

Legault, I., Allard, R., & Faubert, J. (2013). Healthy older observers show equivalent perceptual-cognitive training benefits to young adults for multiple object tracking. *Frontiers in Psychology, 4*, 323.

Madden, D. J. (2007). Aging and visual attention. *Current Directions in Psychological Science, 16*(2), 70–74.

Park, D. C., & Reuter-Lorenz, P. (2009). The adaptive brain: Aging and neurocognitive scaffolding. *Annual Review of Psychology, 60*, 173.

Paxton, J. L., Barch, D. M., Racine, C. A., & Braver, T. S. (2008). Cognitive control, goal maintenance, and prefrontal function in healthy aging. *Cerebral Cortex, 18*(5), 1010–1028.

Penke, L., Maniega, S. M., Murray, C., Gow, A. J., Hernández, M. C. V., Clayden, J. D., … Deary, I. J. (2010). A general factor of brain white matter integrity predicts information processing speed in healthy older people. *The Journal of Neuroscience*, *30*(22), 7569–7574.

Persson, J., Lustig, C., Nelson, J., & Reuter-Lorenz, P. A. (2007). Age differences in deactivation: A link to cognitive control? *Journal of Cognitive Neuroscience, 19*(6), 1021–1032.

Raz, N., Lindenberger, U., Rodrigue, K. M., Kennedy, K. M., Head, D., Williamson, A., … Acker, J. D. (2005). Regional brain changes in aging healthy adults: General trends, individual differences and modifiers. *Cerebral Cortex*, *15*(11), 1676–1689.

Salthouse, T. A. (2004). What and when of cognitive aging. *Current Directions in Psychological Science, 13*(4), 140–144.

Scarmeas, N., & Stern, Y. (2003). Cognitive reserve and lifestyle. *Journal of Clinical and Experimental Neuropsychology, 25*(5), 625–633.

Shafto, M. A., & Tyler, L. K. (2014). Language in the aging brain: The network dynamics of cognitive decline and preservation. *Science, 346*(6209), 583–587.

Shing, Y. L., Werkle-Bergner, M., Brehmer, Y., Müller, V., Li, S. C., & Lindenberger, U. (2010). Episodic memory across the lifespan: The contributions of associative and strategic components. *Neuroscience and Biobehavioral Reviews, 34*(7), 1080–1091.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8*(03), 448–460.

Sullivan, E. V., & Pfefferbaum, A. (2006). Diffusion tensor imaging and aging. *Neuroscience and Biobehavioral Reviews, 30*(6), 749–761.

Thomsen, T., Specht, K., Hammar, Å., Nyttingnes, J., Ersland, L., & Hugdahl, K. (2004). Brain localization of attentional control in different age groups by combining functional and structural MRI. *NeuroImage, 22*(2), 912–919.

Valdois, S., Joanette, Y., Poissant, A., Ska, B., & Dehaut, F. (1990). Heterogeneity in the cognitive profile of normal elderly. *Journal of Clinical and Experimental Neuropsychology, 12*(4), 587–596.

Verhaeghen, P., & Cerella, J. (2002). Aging, executive control, and attention: A review of meta-analyses. *Neuroscience and Biobehavioral Reviews, 26*(7), 849–857.

WHO. (2015). World report on aging and health. Extracted from http://www.who.int/ageing/publications/world-report-2015/en/

View publication stats