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Review

The Impact of L2 Learning on Cognitive Aging

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Abstract

It has become a multidisciplinary research area to overcome cognitive decline caused by aging. Many factors can affect cognitive aging and the influence of second language learning (L2 learning) cannot be ignored. The recent decade has witnessed much pathological, behavior and neuroimaging research that L2 experience may help maintain the cognitive function in the elderly, resist cognitive decline, and even delay the onset of Alzheimer's disease (AD). This work is to review available literature concerned and elucidate the neural mechanisms under which L2 learning (training) may modify or sculpt the brain from perspectives of cognitive reserve, plasticity and overlapping networks. Future directions concerning length of learning, frequency of use, comparison with other cognitively stimulating activities are put forward so as to clarify the relationship between language experience and cognitive aging.

Keywords

Cognitive decline; L2 learning; cognitive reserve (CR); neuroplasticity; overlapping network

Introduction

Aging is increasingly serious with the rapid growth of the global population. According to a recent UN report, people over 60 are the world's fastest growing age group, accounting for 700 million so far, which will increase to 2 billion by 2050 [1]. Cognitive aging has become an emergency as cases of neurodegenerative diseases, Alzheimer's diseases (AD) in particular, increase considerably. Currently, the prevalence of Alzheimer's diseases is about 5% among those aged over 60, at the speed of which a conservative estimate is that over one hundred million old people will have been diagnosed with AD by 2050 [2]. Caring for those who cannot provide for themselves has become a major burden of families and the society. Therefore, in the next few decades, it is vital to promote successful cognitive aging for both individuals and public health institutions and overcoming cognitive decline due to aging is becoming a multidisciplinary research field and one of the biggest challenges we face.

Apart from age, many factors affect cognitive aging, including heredity, diseases, education, occupation, lifestyle, etc [3]. It is remarkable that language experience has been found to relate to cognitive advantage. Over half a century, psycholinguists and neuroscientists consent at the benefits of L2 acquisition, rendering

plausible explanations based on language processing and cognitive plasticity, which both enrich theories of bilingualism and shed a deeper insight into the relationship between language and cognition: language not only reflects the mind, but also shapes the brain to a certain degree [4]. Most notable in recent years are several intriguing reports that bilingualism can significantly delay the onset of Alzheimer's diseases. It provides new train of thought for both pathological research and clinical treatments of cognitive degeneration [5-10]. In view of these, our review is aimed to explore the impact of L2 learning on cognitive aging and possible mechanisms, and also present challenges as well as directions for future research.

Connotation and categories of cognitive aging

Under the framework of biology, cognitive aging reflects that the body's protective function is no longer sufficient to offset a wide variety of pressure sources [11], the process involving damage of oxidative stress, loss of free radical detoxification, decline of mitochondrial function, accumulation of potentially harmful proteins, any one of which can lead to decreased neuronal membrane integrity, altered normal metabolic functions, even the death of neurons [12]. On the pattern of aging process, views of the past was that all the components of nervous system showed a similar degree of decline with the growth of age [13], while current researchers argue that some specific regions and networks are more vulnerable than others, such as gray matter in the prefrontal cortex (resulting in impaired executive function) or subcortical white matter (resulting in slow information processing speed), hippocampus (resulting in decline in memory) [14].

Generally, cognitive aging is categorized into normal aging and pathological aging. Normal aging, also known as "non-pathological aging" or "successful cognitive aging", entails predictable changes to cognitive function along with the increase of age. That is, no symptoms of the diseases can be identified to have negative effects on the central nervous system (such as AD, cerebrovascular disease). Otherwise, it is viewed as "pathological aging". However, many people insist that aging process is a continuum with no clear boundary between the two categories. Some even argue that dementia is still a part of normal aging due to the fact that quite a lot of old people aged over 85 would have clinical symptoms of dementia [15] and a large proportion of old people who are cognitively normal also manifest some degree of pathological phenomena (especially the amyloid protein or neurons winding) and cerebrovascular diseases. Research has shown that mild cognitive impairment caused by the AD may start as early as ten years before clinical onset [16], which means that many senior people who seem normal have actually been accumulating pathological phenomena. So it is very difficult to decide whether cognitive decline is caused by diseases or the normal aging. However, it is well accepted that typical manifestations may be memory loss and decline of executive control, poor working memory and slow information processing. Of course language and communication are also affected, including bad speech perception and production, reduced semantic comprehension in natural language environment, poor vocabulary memory, among others [17].

Advances on the impact of L2 learning on cognitive aging

Early studies last century mainly concerned cognitive effects of bilingual experiences of children within so-called "critical period", rather than young adults and the aged. Not until the beginning of this century did scholars begin to pay close attention to the influence of bilingual experiences on cognitive aging. The main research wisdom is to determine the differences of cognitive function between the older bilinguals and monolinguals by comparing their performances on various behavior tasks (such as Simon task, Flanker task, Stroop tasks, etc.). Recently, with the aid of electrophysiological and neuroimaging technologies, such as event-related potentials(ERP), functional magnetic resonance imaging (FMRI), positron emission

tomography (PET), researchers can obtain more accurate data about brain function and structure in comparison to behavioral tasks, allowing deeper probe into the relationship between L2 learning and cognitive aging. Below we will retrospect on current research progress along two channels: normal aging and pathological aging.

Bilingualism contributing to better cognitive ability in healthy elderly adults

Although a few studies concerning children and young adults have found weaknesses of speaking more than one language, for example, slower reaction time and higher error rate on the picture naming tasks, poor fluency on either language and so on, more research has shown that bilinguals do excel in learning strategies, problem solving, conflict resolution, attention regulation, executive control in comparison to their counterparts [18]. More importantly, positive effects are assumed to extend to the elderly, helping ward off age-related decline of the cognitive levels [19-22]. For example, by comparing the performances of the middle-aged and old bilinguals with those of their monolingual counterparts in Simon task and the Visual Stroop task (executive control), Bialystok et al. found bilingual advantage in both groups, especially in old age group [19,23]. Second, through working memory span tasks (spatial WM) which are also known to be closely related to executive control, Luo et al., found the bilinguals outperformed the monolinguals in spatial WM rather than in verbal WM, although there was no interaction effect of bilingual experience and aging. They explained that this might be because bilingual deficit in verbal process offsets the positive impact on working memory as a function of their superior executive control [24]. Notably, Kave et al. (2008), with a follow-up study of 814 healthy old man over a period of 13 years, found that the number of languages they spoke could predict the performance on various cognitive tests after controlling for some demographic variables (such as age, gender, place of birth, age and education level). They proposed for the first time that the more languages old people spoke on regular basis, the higher their cognitive levels would be [21]. It is known as the cumulative effect of languages, which is consistent with the results of Luxembourg's study that mastering one more language would increase the probability of protection against cognitive impairment by more than four times (of course, a threshold existed) and the earlier they became multilingual, the greater the protection would be [25]. It is a pity that this research didn't involve monolinguals, which makes impossible the comparison between monlolinguals and bilinguals or multilinguals (people speaking more than two languages).

Recently, Bak and his colleagues from University of Edinburgh obtained encouraging results with Lothian Birth Cohort (about 1100 Irish born in 1936 in and near Edinburgh) [26]. Although this study was not meant to explore the influence of language, cognitive states were tracked toward the age of 70 for 853 of the participants, all of whom coincidentally spoke only English before the age of 11 and almost a third or 262 people, had learned to speak another one language ever since. This provides the absolute homogeneous sample for the longitudinal study on cognitive effects of foreign languages (the interference of the original intelligence ruled out). After conducting a series of cognitive tests for participants including intelligence test, and comparing the results with their own test scores at the age of 11, the researchers found that those who mastered two or more languages had significantly better cognitive ability, especially in terms of general intelligence and reading, irrespective of when to learn the second language. It may be inferred that the aging brain of adult learners will also benefit from speaking a second language even if their L2 cannot achieve the proficiency of their mother tongue, which turns out to be a great incentive for millions of adults around the world who are trying to learn a second language.

Bilingualism protecting against the onset of AD

At the end of last century, Graves and his colleagues published a study regarding the influence of cultural factors on the pathological process of a group of older Japanese Americans [27]. After controlling for many variables, such as immigration history, years of formal education in Japan, religion, diet, the proficiency of reading and writing in Japanese and so on, they found that comprehensive level of Japanese could serve as a most powerful indicator to predict the lower risk of cognitive decline among all participants]. This raised an interesting possibility that mastering one more language may affect cognitive decline [28]. The connection between language ability and aging echoed with the finding of another autopsy study by Snowdon et al. that language ability in youth (English self-introduction at the age of 19-27) was in significantly negative correlation with its AD pathology (age spots or nerve filament winding) in some cerebral areas (such as frontal lobe, temporal lobe and parietal lobe) among 74 elderly (ages 78-92), although their language experiences (bilingual or not) were mentioned in the study [28]. The breakthrough must be attributable to Canadian scientists, Bialystok, Craik and Freeman, who for the first time confirmed that L2 learning experience had significantly positive influence on pathological aging by analysing medical records of 184 old patients diagnosed with various types of dementia (70 % AD) in a Toronto hospital. After interviewing patients and families, eliminating various factors which may affect the statistical results (age, gender, education, economy, immigration status, etc.), they were surprised to find that the average age of bilinguals diagnosed with AD were four years later than monolingual patients (71.4 years for monolingual while 75.5 years for bilinguals) [5]. Once published in the well-known journal Neuropsychologia, it immediately aroused great interest from scholars and language learners throughout the world. In 2010, with a larger sample of 211 patients (100 % AD), the same research team not only sustained their earlier conclusion but found those with bilingual or multilingual experience had onset time delayed by up to 5 years, after controlling for language level, occupation, gender, immigration history and other factors [6]. Another group in Canada, with an even larger cohort of 632 people in Montreal (90% monolingual or multilingual immigrants), found that their results still mirrored those of Bialystok's team, especially in the immigrant population [7].

Shortly afterwards, different research teams in different regions or countries replicated the results of Bialystok et al. In 2011, Gollan et al. studied 44 Spain-English bilingual older people in Disease Research Center of University of California and explored the relationship between proficiency of either of two languages and onset age of the dementia, finding that those with higher proficiency of second language are less likely to get symptoms of AD and other kinds of dementia (the higher proficiency, the lower the incidence) [8]. More recently, Indian scientists continued to push forward the research theme in journal Neurology by investigating language abilities of 648 Indians diagnosed with different degrees of different types of dementia (AD and Frontal Temporal Dementia, vascular dementia, etc.). They reported that those who could speak two languages developed dementia 4.5 years later than those who could speak one, irrespective of other potential confounding factors such as education, sex, occupation, and living environments [9]. In contrast to previous studies, all the participants in this study were natives of India, which ruled out of the confounding effect of immigration status completely.

However, the subjects in most studies reviewed above are either patients diagnosed with dementia or healthy old people, with individuals in between unconsidered. As mentioned at the beginning, some scholars believe that there is a progressive transition stage between the healthy and demented elderly, known as amnestic mild cognitive impairment (aMCI) [30]. By examining the effect of bilingualism on the age of diagnosis in individuals with single-or multiple-domain aMCI through a battery of

neuropsychological tests and questionnaires, Michigan scholars identified the early protective advantage of bilingual experience against aMCI, which was further narrowed down to single domain aMCI, one specific subtype most likely to progress to AD. Hopefully, the study verified the inevitable link between L2 experience and the defense against AD in the whole temporal course and proved the continuity of the defense [4].

Neuroimaging evidence

The exciting behavioral and pathological results aforementioned were in line with a large body of neuroimaging studies. Firstly, some researchers recently explored with FMRI the impact of speaking a second language on brain functional connectivity in the elderly. Relative to the monolingual counterparts, bilinguals performed better on perceptual switching tasks while with significantly less activation in three key regions of the brain responsible for executive control, left DLPFC (dorsolateral prefrontal cortex), left VLPFC (ventrolateral prefrontal cortex) and ACC (anterior cingulate cortex) , with other conditions controlled for [31]. This suggests that the bilingual experience, especially constant daily switching between languages, may enhance the executive control system, resulting in less consumption of cognitive resources on the same cognitive tasks.

Secondly, Schweizer et al. proved for the first time by CT (Computed Tomography) the differences of brain structure between bilingual and monolingual patients diagnosed with AD after matched on level of cognitive performance and years of education [32]. Through linear analysis into CT images of 40 subjects, they found Bilingual patients exhibited substantially greater amount of brain atrophy than monolinguals in traditionally AD related brain regions, specifically, medial temporal lobe. It is implied that bilinguals could tolerate more brain atrophy or brain structural changes without symptoms, or maintain comparable cognitive level with their counterparts but with less brain atrophy. Therefore, bilingual experience could protect against neuropathology of AD, delaying the onset of the disease.

The volume of gray matter (GMV) will decrease with normal aging [33], but the extent may be regulated by external experiences, such as speaking another language on daily basis. The left anterior temporal pole (LATP) is considered as a conceptual hub and characteristic region of brain plasticity in bilingual speakers, because they require it to store and distinguish the lexical concepts of two different languages in both comprehension and production processes [28]. Abutalebi et al. employed VBM (voxel-based morphometry) to compare the brain structure of 23 Chinese/English bilingual elders (mandarin or Cantonese) and monolingual Italian elders [34]. After ruling out all kinds of confounding factors, they found a wider range of age-related decreases in GMV in monolingual brains and a significant increase in left temporal pole in bilingual brains. Meanwhile, ROI (region of interested) analysis showed that bilinguals' performance in picture-naming tasks in second language was positively correlated with GMV in left temporal lobe. This indicates that the temporal pole is vulnerable during normal aging and speaking another language contributes a lot to holding up the development of age-related GMV decreases and maintaining the health of the elderly. Additionally, the same team had found that the advantage of GMV in ACC related to executive control in young adult bilinguals [35].

The decrease in white matter integrity is also known to accompany aging [36], and the extent may be also mitigated by bilingual experience. Through DTI (Diffusion tensor imaging), Luk et al. found higher WM integrity in older bilinguals in the corpus callosum extending to the superior and inferior longitudinal fasciculi comparative to monolinguals controlling for some demographic and neuropsychological data. Resting-state functional connectivity analysis showed stronger anterior to posterior functional connectivity

of bilingual frontal lobe regions adjacent to WM areas, which reflected stronger white matter (WM) connectivity between brain regions, facilitating information transfer and executive performance [37]. In a more recent study, both white matter (WM) integrity and gray matter volumes (GMV) were detected and compared between older adult lifelong bilinguals and monolinguals who were matched on a number of relevant cognitive test scores and controlled for some confounding variables such as education, socioeconomic status and intelligence. Results showed that, when the two groups performed in a draw on a series of neuropsychological tasks, significantly lower WM integrity in above-mentioned corpus-callosum-centred tracts was observed in the bilingual group, such as the inferior longitudinal fasciculus and inferior fronto-occipital fasciculus bilaterally, while no significant difference in GMV [38]. This seems inconsistent with Luk's results. The authors interpreted that this inconsistency could be due to the fact that their bilingual subjects were more likely to be at a preclinical AD stage, who might have suffered moderate neuron degeneration but maintained a comparable normal cognitive function with monolinguals since the decrease of FA (fractional anisotropy) and the increase of DR (radial diffusivity) were well-recognized as the neuroimaging characteristics of aMCI [17,39]. They also suggested that bilinguals did not necessarily have increased WM, but might actually compensate for their WM degeneration through the use of their more efficient executive function networks [17].

Mechanisms of L2 Learning influencing cognitive aging

The studies reviewed in former section provide plausible evidence that both epidemiological and neuroanatomical effects can be induced by long-term experience with learning a new language. Most exciting are sufficient findings that speaking two or more languages can serve as a safeguard to maintain cognitive function in the elderly, delay cognitive decline and even protect against the onset of neurodegenerative diseases such as AD. What remains to be understood are the questions of why and how second language learning can affect cognitive aging. In this section we will explore the reasons and the neural mechanisms from the three prominent aspects which are cognitive reserve, neural plasticity and brain networks in order to provide a synthesis of available studies, and build up a basis for future research in this domain.

Cognitive Reserve Hypothesis

In today's world where the aging problem is increasingly serious, it is vital to study the issue of cognitive reserve [40]. The cognitive reserve hypothesis are proposed by Stern in contrast to the twin concept of brain reserve [41], which is generally believed to be quantitatively measured by the number of synapses or brain size. For an individual it is assumed to have a critical threshold, beyond which either clinical or functional disorders will be manifested. But due to individual differences in brain sizes, the threshold varies a lot, such that the "passive" brain reserve cannot explain the common phenomena that many older individuals display distinct cognitive ability although with similar degree of brain atrophy or injury [41]. On the other hand, CR is dynamic and experience-dependant, which can better justify the discrepancy between the degree of neuropathology and clinical symptoms. It highlights the individuals' ability that they employ existing brain reserves or alternative networks to complete the tasks flexibly and effectively when brain reserve is insufficient or optimal network is disrupted. It applies to both healthy old individuals and those with brain injury [42]. Neural reserve and neural compensation are accepted as two main mechanisms by which CR functions. The former posits that some individuals with neuropathology maintain cognitive functioning by making more efficient use of the same networks engaged by healthy individuals [43]. In this case, individuals with greater neural reserve require less neural activation within the optimal

task-specific network in response to a given increase in task demand compared with those with less neural reserve, as a result of which those with greater neural reserve can withstand more neuropathology before the optimal task-specific network is disrupted [44]. On the other hand, neural compensation highlights individual differences in the capacity to recruit alternative networks or brain structures for the sake of maintaining cognitive functioning when the optimal task-specific networks of a given tasks have been disrupted [44-45]. That is, individuals with greater CR can produce new and compensatory networks to make up for the defects caused by neuropathology or brain damage. For example, a meta-analysis of more than 29,000 people showed that individuals with high CR were 46 % less likely to be diagnosed with dementia as opposed to those with lower CR [46].

Much literature shows that quite a few indexes contribute to the construct of CR, such as childhood intelligence, education, occupation accomplishment, social economic status, leisure life style and personality [47,48]. However, it is well-acknowledged that these indicators are unable to stop the development of neuropathology or other brain injuries, but modify the clinical manifestations or pathological symptoms of diseases. And many of these indexes are practically inseparable, and overlap considerably to contribute to general construct of CR, which makes it an urgent future task to specify their respective contributions and causal mechanisms [28].

Bilingualism and Cognitive Reserve

In recent years, as one of the key indicators to measure CR, the leisure life style has become a hot research topic in cognitive aging [47]. The leisure activities are generally categorized into cognitive or intellectual training (such as reading, and playing cards), social networking (such as visiting friends) and physical activity (such as walking) [49,50]. With converging evidence that bilingual experience significantly delays onset of dementia, It is no wonder that learning and speaking a second language is featured as a cognitively stimulating activity that affect the CR, not to mention some even take it as an independent indicator of CR [28]. Literature on bilingualism shows that bilinguals have to inhibit the unattended language while using the target one since both two languages are always active in most communication situations [51]. As a matter of fact, bilinguals are engaged in substantial exercise on brain subconsciously on daily basis over lifetime [52]. There arises the question how such exercise contributes to greater CR and further exerts positive impact on cognitive aging.

Much of the research addressing this question has focused on the assumption that countless inhibition exercise in bilinguals over a long period of time is bound to enhance the central executive control of the brain, which declines gradually with aging [53]. Thus, speaking two languages from early years is supposed to forge a much stronger “executive control system” to protect against brain impairment or injury [54] since almost all the types of dementia even normal aging are associated with the impairment and dysfunction of central executive control system [55]. Literature shows language switching network overlaps significantly with general executive function network (non-linguistic, such as perception switching network) in the LPFC (left prefrontal cortex) and ACC (known as the hub of executive control) [56,57]. In view of this, it is justifiable that older bilinguals’ reduced activation in overlapping areas in perceptual task-switching experiment by Gold at al. are consistent with a negative interaction between language competence and cognitive control demands (better language, less demand on control) in another longitudinal study spanning a year with fifteen bilingual children [38,56]. The mechanism involved may be that L2 learning increases neural efficiency of executive control regions to benefit even nonlinguistic tasks.

On the other hand, Schweizer and his colleagues' research mentioned above provides direct evidence that neural compensation may help determine when symptoms of dementia may set on in old people [32]. More atrophy in medial temporal lobe (possibly more AD neuropathology) in bilingual patients indicates that a certain amount of brain regional atrophy or neural network failure does not mean the ever-lasting loss of functions or network collapse but can be compensated for through actively recruiting other remaining regions or networks in the whole brain. For example, Luk et al. (2011) implied that the greater WM integrity around corpus callosum and the surrounding fiber bundles in bilinguals may compensate for the growing decrease of GMV due to aging or neurodegenerative diseases [37]. To sum up, lifelong bilingual experience may serve as a major deterrent to the onset of age-related cognitive decline, which provides the neural basis for the idea of "cognitive reserve".

L2 Learning and neuroplasticity

Further support for the relationship between cognitive reserve and bilingualism comes from abundant literature that the enriched environment can influence the brain by modifying its physical structure and functional organization throughout the lifetime, the phenomenon known as neuroplasticity. It posits that structural changes may result from learning and experience, or responses to brain damage [58]. For instance, London taxi drivers show increased volume of the posterior hippocampus [59], a region which is vital for spatial memory. Likewise, L2 learning can also bring about structural changes in brain in terms of increased GMV, increased cortical thickness (CT), or enhanced WM integrity. A number of recent studies have identified such changes induced by L2 learning in a series of brain regions in healthy adults, such as the prefrontal cortex (PFC), the temporo-parietal cortex, anterior cingulate cortex (ACC), hippocampus, caudate nucleus as well as putamen [58].

One of the early prominent studies using VBM to examine GMV in language learners was conducted by Mechelli and his colleagues, who found greater GMV in the left inferior parietal lobule (IPL) of bilinguals than that of monolinguals, and the effect was modulated by the age of L2 learning (the earlier they learnt, the more the GMV) and the proficiency (more proficient, more GM) [60]. Subsequent studies replicated their results and confirmed that bilinguals had greater GM density than monolinguals in the temporo-parietal cortex, such as inferior parietal lobule (IPL) and the posterior supramarginal gyrus (SMG) [58, 61]. In addition, Abutalebi's team found young bilinguals had GM advantage over monolinguals in ACC through Flanker task [62], and in left putamen through a picture naming task[63]. Interestingly, Zou et al. found that advanced language learners (Chinese sign language) had much greater GMV in the left caudate nucleus than monolinguals, which is so far the first attempt to illustrate the effect of different modal language learning [64].

In terms of WM integrity, Luk et al. (2011) found higher white matter integrity in healthy bilingual older adults, primarily in the corpus callosum and increased anterior-posterior connectivity [37]. As discussed above, this result suggests bilingualism is associated with better maintenance of WM integrity in the course of cognitive aging [65]. However, Cummine and Boliek found greater integrity for adult monolinguals over bilinguals in the right inferior fronto-occipital fasciculus and the anterior thalamic radiation [66], which seems contradictory to Luck's findings (2011), but more consistent with those of Mohades et al. (2012) in which no significant differences were observed between monolingual and bilingual children (aged 8 to 11) [67]. These three studies may reflect a developmental trajectory concerning the establishment of lifelong WM integrity, which may take as much as few decades of L2 experience to a stable degree [58].

Most of the studies reviewed above are about bilingual speakers who have long-term (sometimes

lifelong) L2 experiences. Confusion remains about whether short-term learning or intensive training lead to the same type of structural changes. Several recent longitudinal studies have been intended to clear the confusion, and some exciting results are achieved. Stein et al. (2012) found that college students who went to Switzerland learning German over the course of 5 months increased GM density in the left IFG (inferior frontal gyrus) and the left anterior temporal lobe, two areas that are implicated to serve lexical access and semantic integration [58,68]. Mårtensson et al. (2012) examined 14 young military students who went through intensive language training for 3 months in preparation for interpreters and found, as compared to controls matched for age and cognitive abilities, the future interpreters had increased CT in left PFC as well as increased right hippocampal volume [69]. Meanwhile, Schlegel et al. (2012) found greater WM density for 11 American college students who had had 9-month Chinese intensive language course in opposition to 16 controls [70]. Further, Hosoda, et al. found, after a 16-week English vocabulary training in the laboratory, 24 college students showed both increased GM and WM density in the right IFG (inferior frontal gyrus), and an average increase of 6 % in the volume of right prefrontal lobe as compared to the controls [71].

In summary, as far as neural plasticity is concerned, the aforementioned studies with balanced or unbalanced young bilinguals (even beginners), help explore possible neural mechanism by which L2 may sculpt the brain. It could be concluded that a period of language learning or practice, whether short-term or long term, may lead to changes in the structure of the brain. Accordingly, we attempt to believe that if such changes are maintained to a certain amount as brain reserve, individuals may be able to withstand reductions of the neurons, the deterioration of cell death or nerve fiber winding, which are accompanied by aging in the long run.

Overlapping between language and aging networks

As discussed above with the connotation of cognitive aging, although the mechanisms of pathological aging and normal aging are not the same, they are both accompanied by decline in cognitive functions, ranging from the general (e.g., reduced working memory and executive control) to the language-specific (e.g., slower lexical retrieval and poor fluency). The cognitive decline maybe typically derive from brain atrophy, loss of neuronal synaptic connections (functional connectivity), and signs of neuropathology associated with dementia [71]. A few brain imaging studies have established that the brain network involved in cognitive aging is extensive, including prefrontal cortex, medial temporal lobe and subcortical structures such as the hippocampus, ACC, etc. In normal aging without clinically significant neurodegenerative diseases, the shrinkage of GM rather than WM is found to be the principal cause of total brain volume reduction. Between the ages of 30 to 90, the brain's GM will diminish by 15 % to 25 %, with the atrophy mostly occurring in frontal and temporal cortexes, such as the prefrontal cortex, in which GM reduction is said to be associated with the decline or damage of executive control in elders [72]. In the presence of cognitive impairment which is not sufficiently severe to meet criteria for a dementia diagnosis, hippocampal atrophy emerges as the most consistent symptom [73]. Wolf et al. (2001) suggests that the turning point from normal cognitive aging to earlier Alzheimer’s disease may be detected by hippocampal atrophy. Meanwhile, WM density decreases in the pertinent brain regions responsible for information processing speed and executive function due to the effect of demyelinization caused by aging [17].

However, quite a few studies have found that language learning network overlaps extensively with the brain network engaged in cognitive aging [74]. The brain network involved in language learning in adulthood can be characterized into several sub-networks [75]. Specifically, the left IFG (inferior frontal gyrus) and left MFG (middle frontal gyrus) in prefrontal cortex are key regions in the articulatory network.

Additionally, medial temporal lobe is involved in acoustic–phonetic processes and the acquisition of meaning [76]. The hippocampus may be critically involved in the memory processes or vocabulary acquisition [77]. The learning of grammatical rules is linked to the frontal-striatal system, which connects frontal lobe regions with caudate nucleus and putamen [78]. L2 learning largely involves the same neural structures as the native language, except that more activity in left prefrontal areas is typically observed in those who acquire the second language later in life and whose proficiency has not reached native-like level [79]. As reviewed in last section, the anterior cingulated cortex (ACC), the IPL (Inferior parietal lobe), and subcortical regions including the basal ganglia, particularly the left caudate and the putamen are involved in bilingual control network [80,58]. The exact functional roles of these sub-networks are sometimes controversial, however, few would deny that the acquisition of a new language involves a large brain network with regions similar to those implicated in cognitive aging described above. Therefore, we attempt to hypothesize that it is the overlapping of these two kinds of networks that make the L2 learners keep strengthening brain areas which are prone to cognitive decline. For example, WM density enhancement Schlegel (2012) found in fiber bundles going through corpus callosum in young L2 learners [70], is amazingly consistent with Luk's suggestion (2011) that life-long language experience entails the maintenance of WM in similar regions of older healthy people [37].

Conclusion

Taken together, given the possible mechanisms under which L2 learning may affect cognitive aging, we attempt to hypothesize that the ultimate result of second language learning may be that the integrity of the brain structures involved is maintained to attenuate atrophy or lesion, and more potential neural networks and sub-networks available could allow for compensation or substitute of age-related cognitive declines. However, there are still many researchers who have questioned this hypothesis. For example, in the two longitudinal studies with Japanese Americans who began to learned Japanese as adults, Crane et al. found there were no connection between the incidence of dementia later and Japanese proficiency, either written or oral [81]. Sanders et al. (2012) also failed to find speaking English may be a cognitive activity associated with the lower risk of developing dementia for non-native English speakers [82]. In a more recent study of a larger sample of Hispanic immigrants in Manhattan communities over a time-span of 23 years, still no protective effect of bilingualism against normal aging and dementia was identified in spite of the findings that bilingualism was associated with better initial performance on tests of memory, executive function, and task switching[83]. It is possible that the discrepancy in results between studies is due to some factors with regard to the methods to determine dementia, criteria to select participants, definition and measurements of bilingualism, the dissolution of language experience from education, among others. Nonetheless, these contradictory results undoubtedly pose an unprecedented challenge to our hypothesis. Urgency turns out to be clarification-making about circumstances under which second language learning can delay the cognitive aging and the roles some variables play in bilingualism as a moderator between neuropathology and clinical onset, such as migration status, language proficiency, frequency of use and so on [28]. More research in the future is needed to achieve a breakthrough in these respects.

Most of the studies discussed above concerns those early language learners who spoke two or more languages over the course of a lifetime, they have high proficiency of each language and the frequency of using it is also high, two variables known to play a crucial part in constituting bilingual advantage over monolinguals [8,60]. It is no doubt that the effect of language experience promoting healthy aging against cognitive decline become more obvious and stable in this population [5]. Moreover, more exciting is much

evidence that short-term foreign language learning or training in young adults can also lead to positive structural and functional changes [68-71]. It seems quite possible that, if these changes can be maintained to the elderly, they are bound to constitute cognitive reserve, suppressing the decrease of neurons or nerve fibers winding causing age-related cognitive decline. Given that the aged brain retains somewhat plasticity [40], foreign language learning initiated in old age is also likely to have analogous effects, and be used as an effective therapy to Alzheimer's disease [17]. However, up to present, few empirical studies have been conducted to give definite evidence to this end. Therefore, more longitudinal studies are needed in future work to determine whether learning a foreign language in the elderly (aged 65 or above) can promote cognitive functions or even produce positive structural changes in their brain as in young adults.

Many studies have shown other cognitively stimulating activities, such as reading newspapers, bridge, chess, crossword puzzle, among others, can also improve the cognitive function and delay the symptoms of AD and mild cognitive impairment [84,85]. In order to seek optimal non-medicinal approaches to prevent or treat dementia, it is required that magnitude of cognitive improvement should be compared between varieties of cognitively stimulating activities. It remains to be verified whether L2 learning could serve as a major deterrent or preferable therapy to age-related cognitive decline, and whether stronger neural networks or connections are engaged in L2 learning than other forms of cognitive trainings that have been investigated.

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References

[1] Department of Economic and Social Affairs, *World Population Ageing*. United Nations Publications; New York: 2007.

[2] B.L. Plassman, K.M. Langa, G.G. Fisher, S.G. Heeringa, D.R. Weir, M.B. Ofstedal, J.R. Burke, M.D. Hurd, G.G. Potter, W.L. Rodgers, D.C. Steffens, R.J. Willis, R.B. Wallace, *Neuroepidemiology* **29**(1-2) (2007) 125-132.

[3] L.L. Drag, L.A. Bieliauskas, *Journal of geriatric psychiatry and neurology* **23**(2) (2010) 75-93.

[4] C. Kaiwen, D. Yanhui, Y. Dezhong, *Advances in Psychological Science* **22**(11) (2014) 1723-1732.

[5] E. Bialystok, F.I.M. Craik, K.J. Freedman, K.J. Murphy, A.K. Troyer, *Neuropsychologia* **45**(2) (2007) 459-464.

[6] F.I.M. Craik, E. Bialystok, M. Freedman, *Neurology* **75**(19) (2010) 1726-1729.

[7] H. Chertkow, V. Whitehead, N. Phillips, C. Wolfson, J. Atherton, H. Bergman, *Alzheimer Disease & Associated Disorders* **24**(2) (2010) 118-125.

[8] T.H. Gollan, D.P. Salmon, R.I. Montoya, D.R. Galasko, *Neuropsychologia* **49**(14) (2011) 3826-3830.

[9] S. Alladi, T.H. Bak, V. Duggirala, B. Surampudi, M. Shailaja, A.K. Shukla, J.R. Chaudhuri, S. Kaul, *Neurology* **81**(22) (2013) 1938-1944.

[10] L. Ossher, E. Bialystok, F.I.M. Craik, K.J. Murphy, A.K. Troyer, *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* **68**(1) (2013) 8-12.

[11] L.J. Whalley, I.J. Deary, C.L. Appleton, J.M. Starr, *Ageing research reviews* **3**(4) (2004) 369-382.

[12] M. Tosato, V. Zamboni, A. Ferrini, M. Cesari, *Clinical interventions in aging* **2**(3) (2007) 401-412.

[13] U. Lindenberger, P.B. Baltes, *Psychology and aging* **9**(3) (1994) 339-355.

[14] K.R. Daffner, *Journal of Alzheimer's disease* **19**(4) (2010) 1101.

[15] L.X. Hy, D.M. Keller, *Neurology* **55**(2) (2000) 198-204.

[16] A. La Rue, L.F. Jarvik, *The International Journal of Aging & Human Development* **25**(2) (1987) 79-89.

[17] M. Antoniou, G.M. Gunasekera, P.C.M. Wong, *Neuroscience & Biobehavioral Reviews* **37**(10) (2013) 2689-2698.

[18] H. Wenguang, C. Baoguo, *Advances in Psychological Science* **19**(11) (2011) 1615-1624.

[19] E. Bialystok, F.I.M. Craik, R. Klein, M. Viswanathan, *Psychology and aging* **19**(2) (2004) 290-303.

[20] E. Bialystok, F.I.M. Craik, J. Ryan, *Journal of Experimental Psychology: Learning, Memory and Cognition* **32**(6) (2006) 1341-1354.

[21] G. Kavé, N. Eyal, A. Shorek, J. Cohen-Mansfield, *Psychology and aging* **23**(1) (2008) 70-78.

[22] B.T. Gold, C. Kim, N.F. Johnson, R.J. Kryscio, C.D. Smith, *The Journal of Neuroscience* **33**(2) (2013) 387-396.

[23] E. Bialystok, F.I.M. Craik, G. Luk, *Journal of Neurolinguistics* **21**(6) (2008) 522-538.

[24] L. Luo, F.I.M. Craik, S. Moreno, E. Bialystok, *Psychology and aging* **28**(1) (2013) 28-34.

[25] M. Perquin, M. Vaillant, A.-M. Schuller, J. Pastore, J.F. Dartigues, M.L. Lair, N. Diederich, *PLoS ONE* **8**(4) (2013) e62030. doi:10.1371/journal.pone.0062030.

[26] T.H. Bak, J.J. Nissan, M.M. Allerhand, I.J. Deary, *Annals of neurology* **75**(6) (2014) 959-963.

[27] A.B. Graves, L. Rajaram, J.D. Bowen, W.C. McCormick, S.M. McCurry, E.B. Larson, *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* **54**(3) (1999) S154-S161.

[28] E. Guzmán-Vélez, D. Tranel, *Neuropsychology* **29**(1) (2015) 139-150.

[29] D.A. Snowdon, S.J. Kemper, J.A. Mortimer, L.H. Greiner, D.R. Wekstein, W.R. Markesbery, *JAMA* **275**(7) (1996) 528-532.

[30] R.C. Petersen, *The New England journal of medicine* **364**(23) (2011) 2227-2234.

[31] B.T. Gold, C. Kim, N.F. Johnson, R.J. Kryscio, C.D. Smith, *The Journal of Neuroscience* **33**(2) (2013) 387-396.

[32] T.A. Schweizer, J. Ware, C.E. Fischer, F.I. Craik, E. Bialystok, *Cortex* **48**(8) (2012) 991-996.

[33] N. Raz, F. Gunning-Dixon, D. Head, K.M. Rodrigue, A. Williamson, J.D. Acker, *Neurobiology of aging* **25**(3) (2004) 377-396.

[34] J. Abutalebi, M. Canini, P.A. della Rosa, L.P. Sheung, D.W. Green, B.S. Weekes, *Neurobiology of aging* **35**(9) (2014) 2126-2133.

[35] J. Abutalebi, P.A. della Rosa, D.W. Green, M. Hernandez, P. Scifo, R. Keim, S.F. Cappa, A. Costa, *Cerebral Cortex* 2011: bhr287.

[36] D.J. Madden, I.J. Bennett, A.W. Song, *Neuropsychology review* **19**(4) (2009) 415-435.

[37] G. Luk, E. Bialystok, F.I.M. Craik, C.L. Grady, *The Journal of Neuroscience* **31**(46) (2011) 16808-16813.

[38] B.T. Gold, N.F. Johnson, D.K. Powell, *Neuropsychologia* **51**(13) (2013) 2841-2846.

[39] B. Bosch, E.M. Arenaza-Urquijo, L. Rami, R. Sala-Llonch, C. Junqué, C. Solé-Padullés, C. Peña-Gómez, N. Bargalló, J.L. Molinuevo, D. Bartrés-Faz, *Neurobiology of aging* **33**(1) (2012) 61-74.

[40] E. Bialystok, F.I.M. Craik, G. Luk, *Trends in cognitive sciences* **16**(4) (2012) 240-250.

[41] R. Katzman, *Neurology* **43**(1) (1993) 13-20.

[42] N. Scarmeas, Y. Stern, M.X. Tang, R. Mayeux, J.A. Luchsinger, *Annals of neurology* **59**(6) (2006) 912-921.

[43] D. Bartrés-Faz, E.M. Arenaza-Urquijo, *Brain topography* **24**(3-4) (2011) 340-357.

[44] J. Steffener, A. Reuben, B.C. Rakitin, Y. Stern, *Brain imaging and behavior* **5**(3) (2011) 212-221.

[45] Y. Stern, C. Habeck, J. Moeller, N. Scarmeas, K.E. Anderson, H.J. Hilton, J. Flynn, H. Sackeim, R. van Heertum, *Cerebral Cortex* **15**(4) (2005) 394-402.

[46] M.J. Valenzuela, P. Sachdev, *Psychological medicine* **36**(04) (2006) 441-454.

[47] B.R. Reed, M. Dowling, S. Tomaszewski Farias, J. Sonnen, M. Strauss, J.A. Schneider, D.A. Bennett, D. Mungas, *Journal of the International Neuropsychological Society* **17**(04) (2011) 615-624.

[48] C. Sattler, P. Toro, P. Schönknecht, J. Schröder, *Psychiatry research* **196**(1) (2012) 90-95.

[49] L. Fratiglioni, S. Paillard-Borg, B. Winblad, *The Lancet Neurology* **3**(6) (2004) 343-353.

[50] N. Scarmeas, Y. Stern, *Journal of clinical and experimental neuropsychology* **25**(5) (2003) 625-633.

[51] Y.J. Wu, G. Thierry, *The Journal of Neuroscience* **30**(22) (2010) 7646-7651.

[52] J. Diamond, *Science(Washington)* **330**(6002) (2010) 332-333.

[53] A.F. Kramer, S. Hahn, N.J. Cohen, M.T. Banich, E. McAuley, C.R. Harrison, J. Chason, E. Vakil, L. Bardell, R.A. Boileau, A. Colcombe, *Nature* **400**(6743) (1999) 418-419.

[54] E. Bialystok, F.I.M. Craik, *Current Directions in Psychological Science* **19**(1) (2010) 19-23.

[55] L.M. Duke, A.W. Kaszniak, *Neuropsychology review* **10**(2) (2000) 75-99.

[56] J. Abutalebi, D. Green, *Journal of neurolinguistics* **20**(3) (2007) 242-275.

[57] T. Guo, H. Liu, M. Misra, J.F. Kroll, *NeuroImage* **56**(4) (2011) 2300-2309.

[58] P. Li, J. Legault, K.A. Litcofsky, *Cortex* **58** (2014) 301-324.

[59] E.A. Maguire, D.G. Gadian, I.S. Johnsrude, C.D. Good, J. Ashburner, R.S. Frackowiak, C.D. Frith, *Proceedings of the National Academy of Sciences* **97**(8) (2000) 4398-4403.

[60] A. Mechelli, J.T. Crinion, U. Noppeney, J. O'Doherty, J. Ashburner, R.S. Frackowiak, C.J. Price, *Nature* **431**(7010) (2004) 757-757.

[61] P.A. della Rosa, G. Videsott, V.M. Borsa, M. Canini, B.S. Weekes, R. Franceschini, J. Abutalebi, *Cortex* **49**(2) (2013) 605-608.

[62] J. Abutalebi, P.A. della Rosa, D.W. Green, M. Hernandez, P. Scifo, R. Keim, S.F. Cappa, A. Costa, *Cerebral Cortex* **22**(9) (2012) 2076-2086.

[63] J. Abutalebi, P.A. della Rosa, G. Ding, B. Weekes, A. Costa, D.W. Green, *Cortex* **49**(3) (2013) 905-911.

[64] L. Zou, G. Ding, J. Abutalebi, H. Shu, D. Peng, *Cortex* **48**(9) (2012) 1197-1206.

[65] M.H. Davis, M.G. Gaskell, *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**(1536) (2009) 3773-3800.

[66] J. Cummine, C.A. Boliek, *Brain Structure and Function* **218**(2) (2013) 595-601.

[67] S.G. Mohades, E. Struys, P. van Schuerbeek, P. van de Craen, R. Luypaert, *Brain Research* **1435** (2012) 72-80.

[68] M. Stein, A. Federspiel, T. Koenig, M. Wirth, W. Strik, R. Wiest, D. Brandeis, T. Dierks, *Cortex* **48**(4) (2012) 458-465.

[69] J. Mårtensson, J. Eriksson, N.C. Bodammer, M. Lindgren, M. Johansson, L. Nyberg, M. Lövdén, *Neuroimage* **63**(1) (2012) 240-244.

[70] A.A. Schlegel, J.J. Rudelson, U.T. Peter, *Journal of cognitive neuroscience* **24**(8) (2012) 1664-1670.

[71] C. Hosoda, K. Tanaka, T. Nariai, M. Honda, T. Hanakawa, *The Journal of Neuroscience* **33**(34) (2013) 13663-13672.

[72] F.M. Gunning-Dixon, N. Raz, *Neuropsychologia* **41**(14) (2003) 1929-1941.

[73] H. Wolf, M. Grunwald, F. Kruggel, S.G. Riedel-Heller, S. Angerhöfer, A. Hojjatoleslami, A. Hensel, T. Arendt, H. Gertz, *Neurobiology of aging* **22**(2) (2001) 177-186.

[74] A. Rodríguez-Fornells, T. Cunillera, A. Mestres-Missé, R. de Diego-Balaguer, *Philosophical Transactions of the Royal Society of London B: Biological Sciences* **364**(1536) (2009) 3711-3735.

[75] G. Hickok, D. Poeppel, *Nature Reviews Neuroscience* **8**(5) (2007) 393-402.

[76] P.C.M. Wong, M. Ettlinger, J.P. Sheppard, G.M. Gunasekera, S. Dhar, *Ear and hearing* **31**(4) (2010) 471-479.

[77] M.H. Davis, M.G. Gaskell, *Philosophical Transactions of the Royal Society B: Biological Sciences* **364**(1536) (2009) 3773-3800.

[78] K.L. Sakai, *Science* **310**(5749) (2005) 815-819.

[79] J. Abutalebi, *Acta psychologica* **128**(3) (2008) 466-478.

[80] Y. Li, J. Yang, K.S. Scherf, P. Li, *Brain and language* **127**(3) (2013) 452-462.

[81] P.K. Crane, L.E. Gibbons, K. Arani, V. Nguyen, K. Rhoads, S.M. McCurry, L. Launer, K. Masaki, L. White, *Epidemiology* (*Cambridge, Mass*.) **20**(5) (2009) 766-774.

[82] A.E. Sanders, C.B. Hall, M.J. Katz, R.B. Lipton, *Journal of Alzheimer's Disease* **29**(1) (2012) 99-108.

[83] L.B. Zahodne, P.W. Schofield, M.T. Farrell, Y. Stern, J.J. Manly, *Neuropsychology* **28**(2) (2014) 238-246.

[84] J. Verghese, R.B. Lipton, M.J. Katz, C.B. Hall, C.A. Derby, G. Kuslansky, A.F. Ambrose, M. Sliwinski, H. Buschke, *New England Journal of Medicine* **348**(25) (2003) 2508-2516.

[85] R.S. Wilson, D.A. Bennett, J.L. Bienias, N.T. Aggarwal, C.F. Mendes De Leon, M.C. Morris, J.A. Schneider, D.A. Evans, *Neurology* **59**(12) (2002) 1910-1914.