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Does Bilingualism Contribute to Cognitive Reserve? Cognitive and Neural Perspectives

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Abstract

Objective—*Cognitive reserve* refers to how individuals actively utilize neural resources to cope with neuropathology in order to maintain cognitive functioning. The present review aims to critically examine the literature addressing the relationship between bilingualism and cognitive reserve in order to elucidate whether bilingualism delays the onset of cognitive and behavioral manifestations of dementia. Potential neural mechanisms behind this relationship are discussed.

Method—Pubmed and PsychINFO databases were searched (through January 2014) for original research articles in English or Spanish languages. The following search strings were employed as keywords for study retrieval: 'bilingual AND reserve', 'reserve AND neural mechanisms', and 'reserve AND multilingualism'.

Results—Growing scientific evidence suggests that lifelong bilingualism contributes to cognitive reserve and delays the onset of Alzheimer's disease symptoms, allowing bilingual individuals affected by Alzheimer's disease to live an independent and richer life for a longer time than their monolingual counterparts. Lifelong bilingualism is related to more efficient use of brain resources that help individuals maintain cognitive functioning in the presence of neuropathology. We propose multiple putative neural mechanisms through which lifelong bilinguals cope with neuropathology. The roles of immigration status, education, age of onset, proficiency and frequency of language use on the relationship between cognitive reserve and bilingualism are considered.

Conclusions—Implications of these results for preventive practices and future research are discussed.

Keywords

cognitive reserve; bilingualism; brain reserve; neural mechanisms; Alzheimer's disease

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Introduction

One of the most exciting findings in neuroscience is that the brain is not rigid, but flexible and sensitive to experience. Research consistently demonstrates that the environment can influence the brain by modifying its physical structure and functional organization throughout the lifetime. This phenomenon is known as neuroplasticity. The neuroplasticity hypothesis posits that the brain is constantly adapting itself to the environment at both neuronal (e.g., synapse strengthening, neurogenesis, synaptogenesis) and cognitive (e.g. new task-related pathways) levels (Galván, 2010). These structural and functional changes may result from developmental processes, learning and experience, or responses to injury. For instance, taxi drivers who memorize the spatial arrangement of London and become experts at navigating the city show increased volume of the posterior hippocampus (Maguire et al., 2000), a brain structure that is critical for learning and memory, and is known to play an important role in spatial navigation. Similarly, the rich sensorimotor experience of practicing a musical instrument can induce functional and structural modifications in the hippocampus, the auditory cortex and other brain regions in expert musicians (Herdener et al., 2010; Pantev & Herholz, 2011).

Similar to memorizing complex spatial information or playing an instrument, knowing and managing more than one language can be quite cognitively demanding, leaving a trace on our brain. Bilingualism has been characterized as the regular use of two (or more) languages (Grosjean, 1989). It has been estimated that more than half of the world's population speaks two or more languages regularly. In the United States, approximately 20% of the population speaks a language at home other than English (Census Bureau, 2007), yet this might very well change in the years ahead. Cross-cultural boundaries are gradually fading and business, the search for employment, education, relationships and new adventures lead to an increasing necessity to communicate in languages other than our mother tongue. Questions have been raised as to whether being bilingual or multilingual offers advantages beyond facilitating communication. In other words, how does knowing more than one language affect the brain and how do these changes influence cognition?

Much of the research addressing these questions has focused on the relationship between bilingualism and executive functioning, and a large body of literature suggests that bilingualism is associated with enhanced performance in tasks of executive control. However, recent research has demonstrated that activities requiring similar levels of mental activity as managing more than one language modify the clinical effects of brain damage or neuropathology caused by injuries or diseases such as Alzheimer's disease, a phenomenon that has been conceptualized as reserve. Specifically, it has been proposed that the continuous mental activity in which bilinguals engage protects against some of the deleterious effects of aging and disease.

Several reviews have examined the relationship between bilingualism and cognitive functioning, and the influence of bilingualism on the brain (Bialystock, Craik, Green, & Gollan, 2009; Bialystock, Craik, & Luk, 2012; Luk, Green, Abutalebi, & Grady, 2012). Some of these briefly discuss the relationship between bilingualism and dementia, but no article to date has examined in depth the literature concerning bilingualism and the delay of symptom onset of dementia (i.e., reserve), in addition to the potential mechanisms underlying this relationship. The present review aims to critically address the literature on how bilingualism might contribute to reserve, e.g., by modifying the relationship between brain damage and clinical performance. Second, it discusses the potential neural mechanisms underlying the relationship between bilingualism and reserve. Finally, implications for practice and future research are briefly discussed. Although reserve can be studied in any situation where the brain sustains injury, in this review it is discussed in the context of dementia since the vast majority of published studies concern this population. The recent increase in studies examining this phenomenon, the need for further research, and the alarming rate at which the prevalence of dementia is increasing, make this review timely and of potential importance to researchers in this field.

Search Strategy

Pubmed and PsychINFO databases were searched until January of 2014 for original research articles in English or Spanish languages. The following search strings were employed as keywords for study retrieval: 'bilingualism AND cognitive reserve', 'reserve AND neural mechanisms', 'reserve AND multilingualism'. Fifteen empirical articles that evaluated the relationship between bilingualism and reserve were included. Two articles were excluded given that they did not directly address how bilingualism is related to cognitive reserve (Kempler & Goral, 2008; López-Higes, Rubio-Valdehita, Prados, & Galindo, 2013). Articles that explored the relationship between bilingualism and dementia but not bilingualism as a possible contributor to reserve were not included. Finally, only studies discussing the mechanisms of cognitive reserve were selected for this review.

Reserve

A large number of studies have evidenced a positive association between neuropathology and cognitive impairment. For instance, the amount of amyloid-β and neurofibrillary tangles are strongly related to severity of cognitive impairment and increase the likelihood for a diagnosis of Alzheimer's disease of the dementia type (Roe et al., 2010; SantaCruz, Sonnen, Pezhouh, Desrosiers, Nelson, & Tyas, 2011; Solé-Padullés et al., 2011; Vemuri et al., 2011). Yet, multiple studies have failed to find this association in some individuals and instead have identified Alzheimer's disease neuropathology in the brain of high-functioning individuals who do not (or never did) exhibit Alzheimer's disease symptomatology. This puzzling phenomenon has been reported in studies using diverse measures of neuropathology including neuroimaging techniques (Scarmeas et al., 2003), postmortem examinations (SantaCruz et al., 2011), and measurements of cerebrospinal fluid (CSF; Solé-Padullés et al., 2011), as well as with distinct methodological procedures. For instance, both the Nun Study and the Adult Changes in Thought study found Braak stage V to VI pathology, which is usually a marker of late-stage Alzheimer's disease, in 8 to 12 percent of previously non-demented individuals, as well as in a larger group of individuals who had been diagnosed with dementia (SantaCruz et al., 2011). Snowdon (2003) further illustrated these individual differences by describing two Catholic sisters from the Nun Study who had significant Alzheimer's disease neuropathology as measured by postmortem examinations, yet one of them never expressed clinical symptoms of dementia and functioned at a high level while the other was described as having been cognitively and physically disabled. These observations prompt intriguing questions: What did the former Sister have or do that the latter did not? What forms the shield that prevents the brain from manifesting its damage behaviorally in some individuals?

The theoretical concept of reserve has been put forward to explain individual differences in the relationship between neuropathology and clinical performance (Jones et al., 2010). Specifically, reserve has been proposed to moderate the relationship between neuropathology and clinical performance, such that individuals with high reserve are able to withstand more neuropathology before cognitive function is affected (as in the case of the first Sister), whereas those with low reserve demonstrate the expected positive association between neuropathology and cognitive performance. Reserve has been further divided into two independent but related types: brain reserve and cognitive reserve. Although the concept of brain reserve will be discussed, this review will focus mainly on cognitive reserve.

Brain reserve refers to “passive” factors (e.g. brain volume, synapse count) that confer a particular capacity to endure neuropathological processes until a critical threshold is reached, after which cognitive and functional impairments are expressed (Bartrés-Faz & Arenaza-Urquijo, 2011; Chételat et al, 2010; Borenstein-Graves et al., 2001; Nithianantharajah & Hannan, 2010; Satz, 1993; Stern, 2002). In other words, the brain reserve hypothesis proposes that individuals differ in the amount of neurons and synapses available to be lost before clinical symptoms emerge such that individuals with a larger brain (or more synaptic connections, etc.) might tolerate more neuropathology than those with a smaller brain (Stern, 2012). On the other hand, brain function instead of brain structure is what is relevant in cognitive reserve. Cognitive reserve refers to individual differences in the ability to adaptively use brain resources to cope with neuropathology and maintain cognitive functioning (Bartrés-Faz et al., 2011; Stern, 2009). Therefore, if two individuals of similar age, sex and with comparable neuropathology (e.g., similar brain atrophy) are evaluated, the one with high cognitive reserve will perform better in tests of cognitive functioning, presumably by using brain resources more effectively.

Research suggests that different traits and exposures contribute to cognitive reserve during the life course, including formal educational attainment, occupational attainment, premorbid intelligence, socioeconomic status (SES), early-life linguistic ability, and cognitive activities (Koenen, et al., 2009; Reed et al., 2011; Sattler, Toro, Schönknecht, & Schröder, 2012; Snowdon, Kemper, Mortimer, Greiner, Wekstein, & Markesbery, 1996). It is important to acknowledge that none of these variables protect individuals from developing neuropathology or other brain damage. Instead, they appear to mitigate the impact of neuropathology on the clinical expression of diseases such as Alzheimer's disease. And it should also be underscored that many of these variables are undoubtedly related, and would overlap considerably in contributing to the general construct of cognitive reserve. Sorting out specific contributions and the causal mechanisms is a task for future research.

Measuring cognitive reserve

Cognitive reserve is estimated by measuring the aforementioned variables (e.g., education, premorbid intelligence, SES), where those with higher educational attainment, SES or intelligence are considered to have higher cognitive reserve. One limitation of this approach is that it is difficult to determine exactly how each variable influences cognitive performance since many of these variables might confound with each other (Jones et al., 2011). For instance, formal education, the most frequently used measure of cognitive reserve, has been suggested to be the most robust contributor of cognitive reserve (Jefferson et al, 2011; Sattler et al., 2012). However, it is difficult to isolate the contribution of formal education to cognitive reserve from that of other variables, such as SES or premorbid intelligence, since these can highly influence educational attainment (Jones et al., 2011). Thus, it is necessary to measure or account for multiple contributors of cognitive reserve instead of only examining one when studying the relationship between neuropathology and cognitive functioning. Doing so might offer a more accurate estimate of the individual's cognitive reserve.

Measures of neuropathology and cognitive functioning are also necessary for examining how cognitive reserve influences clinical outcome. Neuropathology can be measured by examining brain atrophy (e.g., structural Magnetic Resonance Imaging, Computed Tomography scan), CSF, cerebral blood flow patterns (e.g., functional Magnetic Resonance Imaging), postmortem brain tissue, among others. Although each of these techniques has its own limitations (e.g., failure to distinguish between types of neuropathology, inability to capture all neuropathology, etc.), they are the most reliable measures of neuropathology available thus far. However, these techniques are not always accessible. In these cases, severity of disease and the presence of neuropathology are often approximated with measures of function such as the Mini-Mental State Examination (MMSE), the Montreal Cognitive Assessment (MoCA), and other measures of dementia severity. These measures of cognitive function only offer a rough estimate of amount of neuropathology and do not provide information about the specific location or type of neuropathology. Moreover, they confound the predictors with the outcomes when measuring individuals' cognitive reserve. Therefore, it becomes more difficult to draw conclusions about the role of cognitive reserve in the relationship between neuropathology and cognitive functioning. On the other hand, measuring cognitive functioning is less controversial than measuring neuropathology, since cognitive functioning is typically measured by administrating valid neuropsychological tests (i.e., tests measuring memory, visuospatial abilities, processing speed, among others) and questionnaires of daily functioning.

In sum, because cognitive reserve cannot be measured directly, and in vivo measures of neuropathology are often unavailable, operationalizing and studying how cognitive reserve moderates the relationship between neuropathology and clinical outcome has proven to be quite challenging. Moreover, it is extremely challenging to establish cause-and-effect relationships between measures of cognitive reserve and outcome variables (usually onset of diagnosis), given the nature of these variables, and thus far, only correlations have been established. Despite these challenges, research using different methodologies and techniques offers converging evidence supporting the existence of this concept and that factors such as education, premorbid intelligence, and SES contribute to it. Further, regardless of the concept that we choose to use, it is evident from hundreds of research studies that these variables influence the clinical trajectory of many diseases such as Alzheimer's disease.

Another individual characteristic that has been suggested to be a contributor to cognitive reserve is bilingualism, given recent research suggesting that bilinguals exhibit a delay in the onset of symptoms of dementia compared to monolinguals. This finding is not surprising given the large amount of studies evidencing that bilinguals perform better than monolinguals in multiple cognitive tasks. The relationship between bilingualism and cognitive functioning will be first briefly discussed in order to understand the cognitive changes that have been associated with bilingualism, followed by a more in depth discussion of the relationship between bilingualism and cognitive reserve.

Bilingualism and Cognitive Performance

For a long time it was believed that bilingualism might actually have negative consequences for the developing mind. However, an extensive body of literature has falsified this belief. Although there is evidence suggesting that verbal skills (e.g., picture-naming, comprehension and verbal fluency) of bilinguals are significantly weaker in each language compared to verbal skills of monolinguals even when they are only tested in their dominant language (Bialystok, Craik, Green, & Gollan, 2009), there is growing evidence showing that bilinguals demonstrate better executive control (i.e., inhibition, attention, task-switching) than monolinguals matched on different demographic variables.

Several models have been put forward to explain how bilinguals control both languages (e.g., Costa, Miozzo, & Caramazza, 1999; Green, 1998). The inhibition control model proposes that both languages are simultaneously activated in bilinguals, which results in a necessity to attend to one language while inhibiting the other non-target language in order to communicate effectively (Kroll, Bobb, Misra, & Guo, 2008). It has been proposed that the “bilingual advantage” (i.e., better executive control) is in part driven by the need for inhibition control with simultaneous activation of both languages (Bialystok, Craik, & Luk, 2012), which has been observed to happen even when the context requires the use of only one language. In addition, bilinguals often have to switch from one language to the other, depending upon the context of interaction and interlocutors. Interestingly, bilinguals consistently show enhanced performance in non-verbal tasks that require similar skills. That is, bilinguals usually perform better than monolinguals in tasks requiring inhibition, attention, anticipation, monitoring and task-switching (for a review see Bialystok, Craik, & Luk, 2012; Bonifacci, Giombini, Bellocchi, & Contento, 2011). However, it must be noted that some studies have failed to find such advantages and instead have concluded that bilinguals and monolinguals do not perform significantly different on tasks of executive control (Duñabeitia et al., 2013; Kousaie & Phillips, 2012; Paap & Greenberg, 2013). Nonetheless, most studies suggest that the skills that are frequently employed by bilinguals when managing language generalize to other non-verbal domains. This “bilingual advantage” has been observed in all age groups, from children to older adults, suggesting that enhanced executive control in bilinguals is present throughout the lifetime (Bialystok, Craik, Klein, & Viswanathan, 2004). Studies have reported this phenomenon even after controlling for potential confounding variables such as socioeconomic status, education, culture, immigration status and proficiency (Bialystok & Viswanathan, 2009; Engel de Abreu, Cruz-Santos, Tourinho, Martin & Bialystok, 2012). Better performance in executive control tasks has also been observed in bilingual children from lower-income and less educated families compared to middle-class monolinguals (Carlson & Meltzoff, 2008) and in young children growing up in underprivileged conditions and environments that might negatively impact or even impede healthy brain development and impact executive control performance (Engel de Abreu et al., 2012). This suggests that the advantages of bilingualism can transcend social disadvantages that often challenge children's cognitive development. Therefore, bilingualism can potentially sustain or improve functioning even when significant adverse conditions are present, which is consistent with the cognitive reserve hypothesis.

Bilingualism and Cognitive Reserve

In 1999, Graves and colleagues published a study evaluating the influence of cultural factors on the progression of pathologic processes in a sample of Japanese-Americans. In this study, researchers evaluated multiple variables including migration history, years of formal education, number of years lived in Japan before age 18, religion, diet, age at which English became the main language spoken at home, language spoken at home at present, currently facility with reading and writing Japanese, and whether participants had Japanese friends. Those with a steeper decline in cognition had less formal education, were older, and had slower reaction time and lower income. They also tended to have a specific apolipoprotein E allele (ApoE-ε4), which has been associated with an increased risk for Alzheimer's disease (Chartier-Harlin et al., 1994), and practiced an Eastern religion. The strongest indicators of lower risk for cognitive decline were having lived in Japan early in life, and speaking, reading and writing Japanese, in addition to English. This study raised the interesting possibility that knowing more than one language could influence the progression of cognitive decline. The relationship between bilingualism and cognitive decline was further explored by Bialystok and colleagues (2007) in a retrospective study aimed to examine whether bilingualism is associated with a delay in the appearance of the symptoms of dementia. Medical records of individuals from a Memory Clinic in Toronto, Canada with diverse dementia diagnosis but similar cognitive status as measured by the MMSE were evaluated. Most bilingual patients, defined as those who had spent most of their lives (at least during early adulthood) regularly using two languages or more, had migrated to Canada and were not native English speakers. In fact, there were 25 different first languages spoken by the participants in total. Results showed a delay of 4.1 years in the onset of symptoms of dementia in the bilingual group compared to the monolingual group, even after taking into account immigration status. Interestingly, bilinguals had significantly fewer years of schooling compared to monolinguals (see below for further discussion). Based on the reported data, the researchers concluded that bilingualism delays the onset of dementia by enabling the brain to better tolerate the accumulated neuropathology. Although this study raises exciting possibilities, it also has several limitations and potential confounding factors. For instance, age of memory impairment onset was estimated by a family member and can introduce information bias. Also, both groups of participants were quite heterogeneous in dementia diagnosis and cultural experiences, especially since many of the bilinguals resided in Europe during World War II. Thus, it is possible that many of these patients were exposed to factors that are likely to influence cognitive functioning.

Bearing these limitations in mind, Chertkow and colleagues (2010) undertook the task of replicating these findings in a sample of individuals with memory complaints who were later diagnosed with probable Alzheimer's disease. The MMSE was administered at two different times (during the diagnostic session and the annual follow-up visit), age of symptom onset was assessed in a subset of patients by interviewing a family member, and language history was obtained from caregiver and patient interviews. However, instead of classifying patients into two groups (bilinguals and monolinguals), patients were assigned to one of three groups: monolingual, bilingual, or multilingual, according to the definition set out by Bialystok et al. (2007). It was estimated that approximately half of multilinguals were immigrants, whereas only 6% of monolinguals had migrated to Canada, all of whom had less formal education on average compared to natives. Outcomes revealed a positive association between the number of languages spoken and age of Alzheimer's disease diagnosis and symptom onset, a finding that is consistent with previous research suggesting that knowing multiple languages leads to a better cognitive state in older adults (Kavé, Eyal, Shorek, & Cohen-Mansfield, 2008). Moreover, the data suggested that knowing more than two languages is particularly protective. The authors also evaluated the influence of bilingualism on age of Alzheimer's disease diagnosis in Canadian-born individuals whose first language was English vs. French. A trend toward a protective effect was observed for native-born bilinguals whose first language was French, but not for native-born bilinguals whose first language was English. However, when examining only the immigrant subgroup, analyses showed that bilinguals were diagnosed an average of five years after the monolingual group. Furthermore, the delay in years increased as the number of spoken languages increased. As will be discussed later, these findings raise questions about the role of migration status in the delay of symptom onset. Overall, these findings partially support those reported by Bialystok and colleagues (2007), and add a nuance by revealing an additive benefit for individuals who speak multiple languages, a finding that was recently replicated in a population of older adults from Luxemburg (Perquin et al., 2013). In this study, actively-practiced multilingualism protected against the onset of cognitive impairment. Specifically, researchers found that the probability of not developing cognitive impairment increased up to four times with each practiced language, although there was a threshold after which the probability of not developing cognitive impairment did not increase. These results remained true even after considering other factors that are known to influence cognitive reserve (e.g., sociocultural activities, formal and non-formal education, among others). Further, results showed that those who learned to speak more than two languages early in life were more protected against cognitive impairment than those who acquired the languages at an older age. Unfortunately, monolingual individuals were not included in the sample, and differences between monolinguals and bilinguals could not be examined. Nonetheless, these studies provide further insight into the relationship between bilingualism and cognitive reserve by suggesting that protection against cognitive decline increases with the number of spoken languages, especially when individuals are multilinguals from an early age.

Bilingualism and preclinical stages of Alzheimer's disease

It has been reported that bilinguals show a symptom-onset delay in the preclinical stages of Alzheimer's disease. After administrating a neuropsychological test battery to individuals with single and multiple domain amnesic mild cognitive impairment, researchers found that bilinguals in the single-domain amnesic mild cognitive impairment group (i.e., those who exhibited only memory problems) were almost five years older at the time of diagnosis compared to monolinguals (Ossher, Bialystok, Craik, Murphy, & Troyer, 2013). This study adds to the existing literature by demonstrating that bilingualism contributes to cognitive reserve by staving off the manifestations of Alzheimer's disease.

The Influence of Immigration

Chertkow and colleagues (2010) reported that immigrant bilinguals had an advantage over non-immigrants and participants from most studies examining the relationship between cognitive reserve and bilingualism are immigrants. This raises the question of whether the delay in dementia diagnosis reported in these studies was mainly due to bilingualism or immigration status. This is particularly plausible since immigration status has been shown to influence cognitive performance. For instance, a study examining performance in the Stroop task by young and older non-immigrant bilinguals failed to find a “bilingual advantage,” suggesting that migration status might be strongly related to cognitive performance (Kousaie et al., 2012). Although migrating to a different country can be stressful, it is also very stimulating and individuals are often learning new things and adapting to the demands of a new environment. It could be that these challenges result in more efficient cognitive processing. However, the study by Perquin et al. (2013) challenges this notion since they found multilingualism to be protective in a non-immigrant population. In addition, bilingualism has been suggested to delay Alzheimer's disease diagnosis for almost five years, even after taking into account immigration status (Craik et al., 2010), and has been associated with maintained cognitive level when evaluating a non-immigrant sample only (Schweizer, Craik, & Bialystok, 2013), suggesting that bilingualism in itself protects against the clinical expression of Alzheimer's disease. Further support for this phenomenon in non-immigrants comes from a compelling study conducted in India (Alladi et al., 2013). In this study, researchers reviewed 648 records of patients with a diagnosis of dementia who were part of a longitudinal dementia registry project. Unlike most studies, onset of dementia was based on a comprehensive examination by health professionals (e.g., neurologists, psychologists) and not on self-report. As with most studies, language history was obtained by interviewing a family member. All participants were born and raised in India, and more than half spoke two or more languages. Most bi- or multilingual participants had acquired languages simultaneously, and used them with regularity. Researchers also considered factors such as educational attainment, occupation, rural vs. urban dwelling, sex, and cardiovascular risk factors, among others, in their analysis. Similar to what has been reported previously, bilinguals and multilinguals were 4.5 years older at the age at onset of dementia compared to monolinguals, even after controlling for the aforementioned factors. Contrary to findings from previous research, the researchers did not find significant differences between number of languages spoken and onset of dementia. The authors speculated that the strong multilingual environment where the study was conducted forces individuals to constantly use more than one language, and that it could be that those who speak two languages reached a maximum “level of switching.” This implies that additional languages would not be of significant benefit. Although this could be possible, more research is needed to clarify the nature of these discrepancies. Regardless of whether there is a difference in onset of dementia based on amount of languages spoken, this study illustrates that immigration status does not solely explain the delay in onset of dementia since none of the participants were immigrants. Given that studies with non-immigrants only have also found a relationship between bilingualism and dementia symptom delay, it seems accurate to say that although migration status might influence cognitive performance, bilingualism in itself is related to cognitive reserve.

Neuroimaging Studies Examining Bilingualism and Cognitive Reserve

Further support for the relationship between cognitive reserve and bilingualism comes from a study evaluating structural differences and cognitive performance in individuals who had consistently used two languages during most of their life and monolinguals with Alzheimer's disease (Schweizer, Ware, Fischer, Craik, & Bialystok, 2012). In this study, bilingual patients with Alzheimer's disease did not significantly differ from monolingual patients on cognitive function as measured by the Behavioral Neurology Assessment and MMSE, age of diagnosis, years of formal education or on a measure of activities of daily living. However, a head computed tomography (CT) scan revealed that bilingual patients had significantly more medial temporal lobe atrophy than monolingual patients (and possibly more Alzheimer's disease neuropathology in this region). Despite having more atrophy, bilingual patients' cognitive and daily functioning did not differ from that of monolinguals. Furthermore, these differences could not be accounted for by factors such as occupation or educational attainment since monolinguals had higher job status and were slightly more educated than bilinguals. This study was the first to measure neuropathology more reliably by using a neuroimaging technique, instead of inferring extent of brain damage based on disease severity as determined by measures of cognitive functioning such as the MMSE. Consistent with these findings, a recent study examining white matter integrity and gray matter volumetric patterns in older lifelong bilinguals and monolinguals found that bilinguals maintained cognitive performance despite moderate neurodegeneration (Gold, Johnson, & Powell, 2013). In this study, researchers examined healthy older bilinguals and monolinguals whose first language was English, and matched them for age, sex, education, SES, intelligence, MMSE, and multiple neuropsychological scores. Although no significant differences in gray matter were observed, findings showed that bilinguals had significantly lower white matter integrity in tracts that are prominently affected in Alzheimer's disease (i.e., fornix, inferior longitudinal fasciculus, among others) even when they performed similar to monolinguals in a range of neuropsychological tests. Thus, these studies offer convergent evidence supporting the hypothesis that bilingualism modifies the relationship between neuropathology and cognitive functioning.

The Influence of Education

Alladi and colleagues (2013) reported a significant difference in age of dementia diagnosis between monolingual and bilingual illiterates, providing support for bilingualism as an independent contributor to cognitive reserve. Given that educational attainment contributes highly to cognitive reserve (Meng et al., 2012), is it intriguing that bilinguals in many of the aforementioned studies had lower years of formal education and a later onset of dementia compared to monolinguals (Bialystok et al., 2007; Chertkow et al., 2010). This observation was granted further attention in a study conducted by Gollan and colleagues (2011) who reported that bilinguals with lower educational attainment (11 or less years of education) tended to have a later onset of dementia, whereas this relationship was weaker in the group of highly educated individuals (12 or more years of education). Why does education not provide additional significant benefit to bilinguals? Gollan and colleagues (2011) suggest that bilinguals with high formal education reach a threshold that decreases the contribution of bilingualism to cognitive reserve, which contradicts the notion of different cognitive reserve contributors having an additive effect on cognitive reserve (Sánchez-Rodríguez et al, 2011, Solé-Padullés et al., 2009; Sattler et al., 2012; Vemuri et al., 2011). Thus, it is possible that education and bilingualism employ a similar mechanism to compensate for brain damage, perhaps because a second language is often learned through formal education. However, this is speculative and more research is needed to clarify this phenomenon.

Language Proficiency and Age of Acquisition

Other factors, in addition to formal education, seem to influence the association between bilingualism and clinical outcome. For instance, language proficiency has been suggested to be associated with the onset of dementia (Gollan et al., 2011). In this study, more proficient individuals showed clinical manifestations of the disease later compared to less proficient individuals. Yet, most studies do not systematically account for language proficiency but only consider whether participants speak all languages regularly since or before early adulthood. This is an important aspect to consider since both language proficiency and age of acquisition of the second language are associated with structural and functional changes in the brain. For instance, results from a study using voxel-based morphometry found that native English-speaking bilinguals who acquired the second language before the age of five and had used both languages regularly since then, had greater grey-matter density in the left and right inferior parietal cortex compared to those who acquired it between the ages of 10 and 15 (Mechelli et al., 2004) with comparable age and educational attainment. The same study reported a statistically significant correlation between language proficiency and grey-matter density in the inferior parietal cortex on a group of Italian native speakers whose second language was English. Further, it has been observed that bilinguals with lower proficiency recruit more brain resources compared to highly proficient bilinguals (Leonard et al., 2011), suggesting that more proficient bilinguals use the brain's resources more efficiently. Thus, it is possible that the influence of bilingualism on cognitive reserve might be modulated by either proficiency or age of acquisition, a possibility that has not been studied systematically. It is also possible that more proficient bilinguals have more brain reserve, which also contributes to better cognitive functioning.

Does the Specific Language Matter?

The language in which the individual is more proficient also seems to be of importance. A study by Kavé and colleagues (2008) showed that participants who were less proficient in their mother tongue performed better in a cognitive-screening test compared to those who were less proficient in their second language. The authors suggest that these participants might have been investing more cognitive effort and time into learning an additional language, an effort that might have provided them with more cognitive reserve. A matter that has not been directly addressed in these studies is whether the languages spoken by the individual influence cognitive reserve. However, although Chertkow et al. (2013) observed (near significant) differences between French native speakers and English native speakers in their study, it seems unlikely that the specific language spoken substantially influences the age of Alzheimer's disease diagnosis. Studies examining the relationship between cognitive reserve and bilingualism have recruited individuals who share the same first and second languages, individuals whose first language is English but differ in their second-language or individuals who differ in all languages. Yet, a delay in the onset of dementia has been observed regardless of the languages spoken. Furthermore, although some studies might suggest otherwise (Bick, Goelman, & Frost, 2011), others indicate that speaking English and Spanish or English and Japanese (or any two languages for that matter) does not have a significant effect on cognitive performance (Perani et al., 1998) beyond the general effects for bilingualism across studies.

Challenging Bilingualism as a Contributor to Cognitive Reserve

Each study mentioned so far has supported bilingualism as a contributing factor to cognitive reserve. However, two longitudinal studies failed to find a relationship between bilingualism and cognitive reserve. The first study evaluated whether proficiency with written Japanese delayed the onset of dementia symptoms, as measured by the Cognitive Abilities Screening Instrument (CAS) administered in English, in a group of second-generation immigrants who were raised in Hawaii. After controlling for multiple variables—education, head circumference, among others—the data suggested that neither use of written or spoken Japanese reduced the risk for dementia (Crane et al., 2009). This same group of investigators later examined whether the use of spoken and written Japanese in this English-speaking population protected against cognitive decline (Crane et al., 2010). Most participants were educated in English at school and "were able to speak and understand at least some Japanese." Consistent with their previous findings, results do not indicate that written proficiency or speaking Japanese in midlife protects against cognitive decline in late life. Something to consider is that the frequency of use of the second language was not reported, whereas all studies reporting a difference between groups in diagnosis delay have recruited participants who report being proficient and using both languages regularly. It is possible that bilingualism contributes to cognitive reserve only if the individual uses both languages on a regular basis, which is consistent with what we know about how neuroplasticity works —i.e., you either use it or lose it. That is, if you do not use both languages then you are at risk of losing its beneficial effects on cognition. In fact, it has been proposed that the constant switching between languages (and therefore, inhibition of one language as proposed by the inhibition control model described earlier) is what drives the benefits associated with bilingualism (Bialystok et al., 2010). Therefore, it is possible that the constant experience of two or more languages is what influences the course of dementia. A third longitudinal study from a different group of scientists also failed to find a positive influence of language use on risk of incident dementia (Sanders, Hall, Katz, & Lipton, 2012). In fact, the authors concluded, "bilingual activity is more burdensome than beneficial in cognitive aging" (p. 104), since those with at least 16 years of formal education showed a four-fold increased risk for dementia compared to individuals with less formal education. Thus, their results are in part consistent with those by Gollan et al. (2011) and suggest that educational attainment modifies the relationship between speaking two languages and risk of incident dementia. Yet, they did not find bilingualism to delay onset of dementia even in the group with lower years of formal education. A strength of this study, as well as of the ones by Crane and colleagues (2009, 2010), is the fact that the data was longitudinal, which may reduce recall bias and also permits a more objective measure of incident dementia. Nevertheless, the authors acknowledged several limitations that might account for the findings. For instance, information about proficiency or frequency of use in the mother tongue was not collected and it was unknown whether participants spoke a third or fourth language. Further, those identified as native English speakers (NES) were not asked whether they spoke a second language. Therefore, it is possible that the NES group was not strictly monolingual. Finally, a recent study evaluating the relationship between bilingualism and development of dementia in a group of Spanish-speaking immigrants residing in New York failed to find a difference between monolinguals and bilinguals (Zahodne, Schofield, Farrell, Stern, & Manly, 2013). Contrary to previous studies, these culturally similar participants were followed prospectively for up to 23 years. Participants self-reported their proficiency level by answering how well they spoke English, and a subset of the sample completed the English-language Wide Range Achievement Test—Version 3, which yielded results consistent with self-reports. Also, all participants were tested in their preferred language, including on tests of memory, language, executive functions, and processing speed. Consistent with the literature, bilingualism was associated with better initial performance on tests of episodic memory, executive function, and task switching ability. Interestingly, rates of change over time in these cognitive domains were not significantly different between monolinguals and bilinguals. Strengths of this study include its prospective nature, the relative homogeneity of the sample (i.e., cultural background, spoken languages) and use of well validated neuropsychological tests. However, three limitations complicate interpretation of the findings, 1) Frequency of use of both languages is unknown and was not considered when selecting participants, 2) Participants learned English as adults, and 3) The vast majority of participants reported speaking English less than “well.” Further, higher educational attainment was correlated with greater degree of bilingualism, and educational attainment was related to the development of dementia. The authors concluded that becoming bilingual (in adulthood) does not significantly influence cognitive decline or dementia onset above and beyond factors such as education. However, it would have been interesting to explore whether individuals with lower educational attainment but comparable degree of bilingualism showed a difference in the development of dementia (Gollan et al., 2011).

Multiple factors could account for the discrepancy in results between studies, including how symptom onset was determined (prospective vs. retrospective), criteria used to select participants, definition of bilingualism, and lack of standardization of measures of bilingualism. Nonetheless, the majority of these studies, which have examined different populations, different languages, considered different variables and used different methods, suggest that environmental factors such as bilingualism can influence the clinical manifestation of untreatable diseases such as Alzheimer's disease. Specifically, bilingualism appears to delay the onset of dementia. There is still a need to better clarify the circumstances under which bilingualism contributes to cognitive reserve and the role that variables such as proficiency, age of acquisition, frequency of use, immigration status, among others, play in bilingualism as a moderator between neuropathology and clinical outcome. However, much of the evidence points to frequency of use of both languages as a crucial factor in the relationship between bilingualism and cognitive reserve, possibly because—based on the inhibition control model—those who constantly use more than one language are constantly inhibiting one language while paying attention to another and switching between languages. That is, such persons are challenging their brain in a way that monolinguals might not be. Despite the limitations in the literature, bilingualism seems to reflect aspects of lifelong experiences that can protect against cognitive decline in older adults. But exactly how does this happen?

Bilingualism and Reserve: A Neural Perspective

Naturally, the disparity between neuropathology and cognitive performance in some individuals has spiked curiosity among neuroscientists. Questions have been raised as to precisely what the neural mechanisms underlying this phenomenon might be. For example, how do these activities modify the brain throughout the lifetime in a way that is later reflected in cognitive performance? How does bilingualism modify the brain to prolong functioning in the presence of extensive brain damage? Answering these questions will not only be of interest to researchers in the field, but could potentially inform public health and the development of interventions and prevention practices.

It remains unclear exactly how neural processing differs as a function of cognitive reserve. However, research has pointed to several possibilities. Three neural mechanisms that are consistent with a neuroplasticity hypothesis have been proposed to mediate the relationship between cognitive reserve and clinical performance: neural reserve, neural compensation and a common network (Steffener, Reuben, Rakitin, & Stern, 2011; Steffener & Stern, 2012).

Neural mechanisms

The three previously mentioned mechanisms are thought to reflect how the brain uses its resources more efficiently and effectively. Neural reserve posits that some individuals with neuropathology maintain cognitive functioning by making more efficient use of the same networks engaged by healthy individuals (Bartrés-Faz et al., 2011; Steffener et al., 2011). In this case, when there is a given increase in task demand, individuals with greater neural reserve require less of an increase in neural activity compared to those with less neural reserve, possibly because they have greater resources within the optimal task-specific network (Steffener et al., 2011). As a result, individuals with greater neural reserve can withstand more neuropathology before the optimal task-specific network is disrupted. On the other hand, neural compensation refers to individual differences in the ability to recruit alternative networks or brain structures when the tasks' optimal task-specific networks have been disrupted, in order to maintain cognitive functioning (Steffener et al., 2011; Stern et al., 2005). More recently, a third mechanism has been proposed to mediate the relationship between cognitive reserve and clinical performance. According to the common network theory, there is a generic cognitive reserve network that is unrelated to task-related activation (Steffener et al., 2012). This approach aims to explain how cognitive reserve allows individuals with extensive neuropathology to preserve function in multiple cognitive tasks (Steffener et al., 2012). Sánchez-Rodríguez and colleagues (2011) further contribute to this discussion by suggesting that over the lifespan, individuals with high cognitive reserve have gradually developed an extensive range of cognitive strategies for solving complex problems, which leads to the development of multiple neuronal pathways for performing the same cognitive processes. In turn, this results in a larger preservation of cognitive functions in the presence of neuropathology. In addition to hypothesizing about the mechanism of cognitive reserve, this proposal implies that cognitive reserve is not fixed or static but is rather developed throughout the lifetime.

The proposed mechanisms are based primarily on neuroimaging findings from studies using years of formal education and indices of premorbid intelligence as contributors to cognitive reserve (Boyle, Wilson, Schneider, Bienias, & Bennett, 2008; Steffener et al., 2011). No study to date has directly examined the neural mechanisms of cognitive reserve using bilingualism as a proxy. However, several mechanisms seem plausible that are consistent with findings from studies using other contributors of cognitive reserve.

Neural Reserve

Much of the available literature suggests neural networks in bilinguals are more flexible and less susceptible to disruption compared to those of monolinguals. In a recent study, Gold and colleagues (2013) examined the functional neuroanatomical basis of bilingual cognitive control advantages in a group of lifelong older proficient bilinguals who had learned a second language before the age of 10, and spoke both languages on a daily basis. Monolinguals and bilinguals were matched across multiple demographic and neuropsychological scores, most of which have been proposed to influence cognitive control performance—e.g., educational attainment, SES and intelligence. Groups did not significantly differ in regional brain volume. Results revealed that bilingual older adults performed better than monolinguals in a task-switching experiment (i.e., were significantly faster at switching between perceptual tasks) while showing decreased activation in the left dorsolateral prefrontal cortex, the left ventrolateral prefrontal cortex and the anterior cingulate cortex, areas that have been previously associated with the task-switching network (Gold, Kim, Johnson, Kryscio, & Smith, 2013). Further, a similar pattern of activation was observed in younger bilingual adults, supporting that this is not a compensatory mechanism. Thus, these findings evidence increased neural efficiency in bilinguals, a potential mechanism through which bilinguals may withstand neuropathology while maintaining cognitive function.

Neural Compensation

Another possibility is that bilinguals display compensatory mechanisms when the optimal network has reached neural capacity—i.e, is too affected by neuropathology. This is reflected in a study by Luk and colleagues (2011), which showed enhanced white matter connectivity and more distributed resting-state functional connectivity in frontal regions—close to where white matter structural differences were observed—on individuals who had successfully managed two languages since early age. It is important to note that bilingual older adults did not differ from monolinguals in a range of demographic variables including age and years of formal education, or in neuropsychological performance on standardized tests. The authors express that these findings reflect how enriched experience protects white matter against age-related deterioration (Luk et al., 2011). They also suggest that these results, in conjunction with those from Schweizer and colleagues (2012), indicate white matter could provide reserve by compensating for grey matter damage, which in turn results in sustained cognitive performance in lifelong bilinguals. In addition to considering this evidence as an illustration of a compensatory mechanism, it raises the question of whether this also reflects brain reserve. Perhaps lifelong bilingualism provides individuals with enhanced white matter connectivity (brain reserve), which in turn influences brain functioning (cognitive reserve). Although these results seem to partially contradict those by Gold and colleagues (2013), the authors suggest that the difference in the direction of white matter integrity might be due to a possible higher incidence of preclinical Alzheimer's disease in their bilingual group given the pattern of reduced integrity in some of the tracts, suggesting that these individuals are engaging compensatory mechanisms in order to maintain cognitive functioning. It would be interesting to explore how white matter integrity and functional connectivity differ as a function of proficiency, age of acquisition or frequency of language use, and whether a similar pattern can be observed in bilinguals with Alzheimer's disease.

Other Mechanisms

Further, although this possibility has received less attention, it could be that bilingualism contributes to cognitive reserve by operating through a general cognitive reserve network that is not linked to a task-specific function (Bartrés-Faez et al., 2011). Finally, the noradrenergic theory of cognitive reserve posits that noradrenaline moderates the relationship between cognitive reserve and reduced risk of Alzheimer's disease (Robertson, 2013). This hypothesis argues that many contributors of cognitive reserve—formal educational attainment, mental and social engagement, intelligence—involve upregulation of the noradrenergic system. In turn, upregulation of noradrenaline results in compensatory mechanisms (e.g., increased cortical volume, neurogenesis, synaptogenesis, increase in brain-derived neurotrophic factor (BDNF), increased cortical connectivity) and disease modification mechanisms (e.g., reduced amyloid burden, plaque size and aggregation, anti-inflammatory processes, and rescue of cholinergic and dopaminergic cells). Thus, optimization of noradrenergic activity driven by the different contributors of cognitive reserve might give rise to a bigger and better connected brain capable of reorganizing itself in the presence of neuropathology. In other words, it is possible that optimization of noradrenaline facilitates neural compensation or neural reserve. Since bilingualism has been considered to be a form of mental activity, it is possible that it could also influence noradrenaline activity. However, this deserves further investigation.

Overall, lifelong bilinguals seem to have a greater critical threshold and make more efficient use of the brain's resources, perhaps resulting in maintained cognitive functioning even when neuropathology is present. As discussed, it is possible that bilingualism contributes to cognitive reserve by increasing the efficiency of existing neural networks, providing compensatory mechanisms, engaging a general cognitive reserve network or through a combination of mechanisms, resulting in a delay in Alzheimer's disease symptom onset. More neuroimaging studies or studies with postmortem pathological data are needed to elucidate the relationship between bilingualism and cognitive reserve and its neural mechanisms. Yet, despite the challenges that accompany investigating this phenomenon, research so far seems promising and could potentially inform prevention practices.

Implications for Practice and Future Research

Scientific evidence indicates that although having a mentally stimulating lifestyle cannot prevent Alzheimer's disease or other such brain diseases, it can influence how the individual is able to cope with neuropathology at a cognitive level. That is, having high cognitive reserve could potentially allow maintenance of cognitive functioning despite the presence of brain damage. Since cognitive reserve is built throughout the life course by modifying the mechanisms used by the brain to perform a task, it could be argued that intense mental stimulation throughout the lifetime might provide individuals with greater resilience in facing neuropathology (Liberati, Raffone, & Olivetti Belardinelli, 2012). Therefore, if in fact bilingualism modifies the relationship between neuropathology and cognitive performance, then learning and successfully managing two (or more) languages for many years could delay the onset of symptoms of diseases such as Alzheimer's disease. This implies that the individual will be able to independently carry on daily activities and everyday tasks for a longer period of time compared to monolinguals. Does this mean that we should all start learning a second or third language? Perhaps, as suggested by Engel de Abreu et al. (2012), participating in foreign-language programs could potentially reduce the achievement gap between more- and less-advantaged children, which could in turn stimulate a “sound cognitive foundation that might help children to reach their full potential and improve their educational opportunities” (p. 1369). Similarly, it could be argued that teaching children more than one language and providing opportunities for the continued use of both languages could potentially impact the incidence of dementia in the future. It has also been suggested that learning a foreign language in older age could also help build cognitive reserve (Antoniou, Gunasekera, & Wong, 2013).

However, it is still unknown how much is enough, exactly what it is about bilingualism that contributes to cognitive reserve and how it interacts with other variables such as years of formal education and other sociocultural factors. Specifically, it appears as if the frequency of use of both languages is important in determining the role of bilingualism in the course of the clinical expression of dementia, possibly because individuals are constantly facing the challenge of managing two languages. In addition, age of second language acquisition and language proficiency deserve further consideration, especially since they modify brain organization and functioning (Mechelli et al., 2004). Further, it is important to consider whether all individuals can increase their reserve regardless of biological predispositions or if some individuals are born with a significant advantage for developing cognitive reserve, and whether there is something unique about bilingualism that contributes to the delay of Alzheimer's disease symptom onset. However, although some individuals might have a natural talent to learn multiple languages, individuals usually learn more than one language due to circumstances that require it (Craik, Bialystok, & Freedman, 2010), which is usually not the case for other cognitive reserve contributors such as education and intelligence. Nonetheless, this does not eliminate the possibility that similar results might be obtained if individuals continuously engage in demanding activities that involve task-switching, response inhibition, and the like, throughout their lives, a possibility that deserves further attention. Moreover, studies measuring neuropathology using structural and functional neuroimaging techniques in individuals at high risk for Alzheimer's disease (and other diseases), or measuring neuropathology postmortem are needed to better understand the role of bilingualism in the relationship between neuropathology and clinical outcome, and the neural mechanisms underlying this phenomenon. It would be interesting to explore whether bilinguals matched in multiple demographic variables and neuropsychological test scores to monolinguals, have more neuropathology at the time of death.

Another limitation deserving attention is the fact that many of these studies do not consider variables such as smoking, alcohol consumption, nutrition, exposure to toxins, personality characteristics and sleep habits, all of which could potentially interact with measures of cognitive reserve (Bartrés-Faez et al., 2011). Finally, the relationship between bilingualism and cognitive reserve has been studied in a population with dementia, particularly Alzheimer's disease. Yet, cognitive reserve has also been shown to impact other neurological and psychiatric diseases including multiple sclerosis (Sumowski, J. F., Chiaravalloti, N., & DeLuca, 2009), schizophrenia (Khandaker, Barnett, White, & Jones, 2011), Huntington's disease (Nithianantharajah et al., 2011) and Parkinson's disease (Armstrong et al., 2012). Thus, it would be interesting to examine whether the effects of bilingualism on the clinical progression of Alzheimer's disease could also be observed in other diseases such as those mentioned.

Taken together, does bilingualism contribute to cognitive reserve? Despite all limitations and caveats, evidence suggests that bilingualism does contribute to cognitive reserve, and possibly to brain reserve. Elucidating the circumstances under which bilingualism contributes to cognitive reserve and delays the onset of dementia will become even more imperative since the prevalence of Alzheimer's disease is estimated to substantially increase in the years ahead (Alzheimer's Association, 2012). This is without taking into account the other disorders (e.g., schizophrenia, multiple-sclerosis) that appear to benefit from factors that contribute to cognitive reserve. A better understating of how factors such as bilingualism and education delay the onset of dementia and other brain diseases could potentially help individuals learn about how to augment the possibilities of maintaining independence or lead a fulfilling life for a longer period of time. The lack of effective treatments makes the search for and study of preventive strategies even more urgent. This review has identified what we know so far about bilingualism and cognitive reserve, and has delineated areas that need further examination, with the hope of stimulating research that might elucidate how bilingualism (or the factors underlying it) protects against the early manifestation of symptoms of dementia or other debilitating brain diseases.

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References

Alladi S, Bak TH, Duggirala V, Surampudi B, Shailaja M, Shukla AK, Kaul S. Bilingualism delays age at onset of dementia, independent of education and immigration status. Neurology. 2013; 81:1938–1944. doi: 10.1212/01.wnl.0000436620.33155.a4. [PubMed: 24198291]

Alzheimer's Association. Alzheimer's Association facts and figures. 2012. Retrieved from http://www.alz.org/downloads/facts_figures_2012.pdf

Antoniou M, Gunasekera G, Wong PCM. Foreign language training as cognitive therapy for age-related cognitive decline: A hypothesis for future research. Neuroscience and Behavioral Reviews. 2013; 37:2689–2698. doi: 10.1016/j.neubiorev.2013.09.004.

Armstrong MJ, Naglie G, Duff-Canning S, Meaney C, Gill D, Eslinger PJ, Marras C. Roles of Education and IQ in Cognitive Reserve in Parkinson's Disease-Mild Cognitive Impairment. Dementia and Geriatric Cognitive Disorders Extra. 2012; 2:343–352. doi: 10.1159/000341782. [PubMed: 22962558]

Bartrés-Faz D, Arenaza-Urquijo EM. Structural and functional imaging correlates of cognitive and brain reserve hypotheses in healthy and pathological aging. Brain topography. 2011; 24:340–357. doi:10.1007/s10548-011-0195-9. [PubMed: 21853422]

Bialystok E. Global–local and trail-making tasks by monolingual and bilingual children: Beyond inhibition. Developmental Psychology. 2010; 46:93–105. doi:10.1037/a0015466. [PubMed: 20053009]

Bialystok E, Craik FIM, Freedman M. Bilingualism as a protection against the onset of symptoms of dementia. Neuropsychologia. 2007; 45:459–464. doi:10.1016/j.neuropsychologia.2006.10.009. [PubMed: 17125807]

Bialystok E, Craik FIM, Green DW, Gollan TH. Bilingual minds. Psychological Science in the Public Interest. 2009; 10:89–129.

Bialystok E, Craik FIM, Klein R, Viswanathan M. Bilingualism, aging and cognitive control: Evidence from the Simon task. Psychology and Aging. 2004; 19:290–303. [PubMed: 15222822]

Bialystok E, Craik FIM, Luk G. Bilingualism: Consequences for mind and brain. Trends in Cognitive Sciences. 2012; 16:240–250. doi:10.1016/j.tics.2012.03.001. [PubMed: 22464592]

Bialystok E, Viswanathan M. Components of executive control with advantages for bilingual children in two cultures. Cognition. 2009; 112:494–500. doi:10.1016/j.cognition.2009.06.014. [PubMed: 19615674]

Bick AS, Goelman G, Frost R. Hebrew brain vs. English brain: Language modulates the way it is processed. Journal of Cognitive Neuroscience. 2011; 22:2280–2290. doi: 10.1162/jocn.2010.21583. [PubMed: 20961169]

Bonifacci P, Giombini L, Bellocchi S, Contento S. Speed of processing, anticipation, inhibition and working memory in bilinguals. Developmental Science. 2011; 14:256–269. [PubMed: 22213899]

Borenstein-Graves A, Mortimer JA, Bowen JD, McCormick WC, McCurry SM, Schellenberg GD, Larson EB. Head circumference and incident Alzheimer's disease: Modification by apolipoprotein E. Neurology. 2001; 57:1453–1460. [PubMed: 11673588]

Brayne C, Ince PG, Keage HA, McKeith IG, Matthews FE, Polvikoski T, Sulkava R. Education, the brain and dementia: Neuroprotection or compensation? Brain. 2010; 133:2210–2216. doi: 10.1093/brain/awq185. [PubMed: 20826429]

Carlson SM, Meltzoff AN. Bilingual experience and executive functioning in young children. Developmental Science. 2008; 11:282–298. [PubMed: 18333982]

Census Bureau. Language use in the United States. 2007. Retrieved from http://www.census.gov/prod/2010pubs/acs-12.pdf

Chartier-Harlin MC, Parfitt M, Legrain S, Pérez-Tur J, Brousseau AE, Berr C, Amouyel P. Apolipoprotein E, epsilon 4 allele as a major for sporadic early and late-onset forms of Alzheimer's disease: Analysis of the 19q13.2 chromosomal region. Human Molecular Genetics. 1994; 3:569–574. [PubMed: 8069300]

Chertkow H, Whitehead V, Phillips N, Wolfson C, Atherton J, Bergman H. Multilingualism (but not always bilingualism) Delays the onset of Alzheimer disease: Evidence from a bilingual community. Alzheimer Disease and Associated Disorders. 2010; 24:118–125. doi:10.1097/WAD.0b013e3181ca1221. [PubMed: 20505429]

Chételat G, Villemagne VL, Pike KE, Baron JC, Bourgeat P, Jones G, Rowe CC. Larger temporal volume in elderly with high versus low beta-amyloid deposition. Brain. 2010; 133:3349–3358. doi: 10.1093/brain/awq187. [PubMed: 20739349]

Craik FIM, Bialystok E, Freedman M. Delaying the onset of Alzheimer disease: Bilingualism as a form of cognitive reserve. Neurology. 2010; 75:1726–1729. doi:10.1212/WNL.0b013e3181fc2a1c. [PubMed: 21060095]

Crane PK, Gibbons LE, Arani K, Nguyen V, Rhoads K, McCurry SM, White L. Midlife use of written Japanese and protection from late life dementia. Epidemiology. 2009; 20:766–774. doi:10.1097/EDE.0b013e3181b09332. [PubMed: 19593152]

Crane PK, Gruhl JC, Erosheva EA, Gibbons LE, McCurry SM, Rhoads K, White L. Use of spoken and written Japanese did not protect Japanese-American men from cognitive decline in late life. The Journals of Gerontology Series B: Psychological Sciences and Social Sciences. 2010; 65:654–666. doi: 10.1093/geronb/gbq046.

Costa A, Miozzo M, Caramazza A. Lexical selection in bilinguals: Do words in the bilingual's two lexicons compete for selection? Journal of Memory and Language. 1999; 41:365–397.

Díaz-Orueta U, Buiza-Bueno C, Yanguas-Lezaun J. Cognitive reserve: Evidence, limitations and future research lines. Revista Española de Geriatría y Gerontología. 2010; 45:150–155. doi: 10.1016/j.regg.2009.12.007. [PubMed: 20167390]

Duñabeitia JA, Hernández JA, Antón E, Macizo P, Estévez A, Fuentes LJ, Carreiras M. The inhibitory advantage in bilingual children revisited. Experimental Psychology. 2013; 11:1–18.

Engel de Abreu PMJ, Cruz-Santos A, Tourinho CJ, Martin R, Bialystok E. Bilingualism enriches the poor: Enhanced cognitive control in low-income minority children. Psychological Science. 2012; 23:1364–1371. doi:10.1177/0956797612443836. [PubMed: 23044796]

Galván A. Neural plasticity of development and learning. Human Brain Mapping. 2010; 31:879–890. doi: 10.1002/hbm.21029. [PubMed: 20496379]

Garbin G, Sanjuan A, Forn C, Bustamante JC, Rodríguez-Pujadas A, Belloch V, Avila C. Bridging language and attention: Brain basis of the impact of bilingualism on cognitive control. Neuroimage. 2010; 53:1272–1278. doi: 10.1016/j.neuroimage.2010.05.078. [PubMed: 20558314]

Green DW. Mental control of the bilingual lexico-semantic system. Bilingualism: Language and Cognition. 1998; 1:67–81.

Gold BT, Kim C, Johnson NF, Kryscio RJ, Smith CD. Lifelong bilingualism maintains neural efficiency for cognitive control in aging. The Journal of Neuroscience. 2013; 33:387–396. doi: 10.1523/JNEUROSCI.3837-12.2013. [PubMed: 23303919]

Gold BT, Johnson NF, Powell DK. Lifelong bilingualism contributes to cognitive reserve against white matter integrity declines in aging. Neuropsychologia. 2013; 51:2841–2846. doi: 10.1016/j. [PubMed: 24103400]

Gollan TH, Salmon DP, Montoya RI, Galasko DR. Degree of bilingualism predicts age of diagnosis of Alzheimer's disease in low-education but not in highly educated Hispanics. Neuropsychologia. 2011; 49:3826–3830. doi:10.1016/j. [PubMed: 22001315]

Graves AB, Rajaram L, Bowen JD, McCormick WC, McCurry SM, Larson EB. Cognitive decline and Japanese culture in a cohort of older Japanese Americans in King County, WA: the Kame Project. The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences. 1999; 54:S154–161.

Grosjean F. Neurolinguists, beware! The bilingual is not two monolinguals in one person. Brain and Language. 1989; 36:3–15. [PubMed: 2465057]

Herdener M, Esposito F, di Salle F, Boller C, Hilti CC, Habermeyer B, Cattapan-Ludewig K. Musical training induces functional plasticity in human hippocampus. The Journal of Neuroscience. 2010; 30:1377–1384. doi: 10.1523/JNEUROSCI.4513-09.2010. [PubMed: 20107063]

Jefferson AL, Gibbons LE, Rentz DM, Carvalho JO, Manly J, Bennett DA, Jones RN. A life course model of cognitive activities, socioeconomic status, education, reading ability, and cognition. Journal of the American Geriatrics Society. 2011; 59:1403–1411. doi: 10.1111/j.1532-5415.2011.03499.x. [PubMed: 21797830]

Jones RN, Manly J, Glymour MM, Rentz DM, Jefferson AL, Stern Y. Conceptual and measurement challenges in research on cognitive reserve. Journal of the International Neuropsychological Society. 2011; 17:593–601. [PubMed: 21411036]

Kavé G, Eyal N, Shorek A, Cohen-Mansfield J. Multilingualism and cognitive state in the oldest old. Psychology and Aging. 2008; 23:70–78. doi:10.1037/0882-7974.23.1.70. [PubMed: 18361656]

Kempler D, Goral M. Language and dementia: Neuropsychological aspects. Annual Review of Applied Linguistics. 2008; 28:73–90.

Khandaker GM, Barnett JH, White IR, Jones PB. A quantitative meta-analysis of population-based studies of premorbid intelligence and schizophrenia. Schizophrenia Research. 2011; 132:220–7. doi: 10.1016/j.schres.2011.06.017. [PubMed: 21764562]

Kroll JF, Bobb SC, Misra M, Guo T. Language selection in bilingual speech: Evidence for inhibitory processes. Acta Psychologica. 2008; 128:416–430. doi:10.1016/j.actpsy.2008.02.001. [PubMed: 18358449]

Koenen KC, Moffitt TE, Roberts AL, Martin LT, Kubzansky L, Harrington H, Caspi A. Childhood IQ and adult mental disorders: a test of the cognitive reserve hypothesis. The American Journal of Psychiatry. 2009; 166:50–57. doi: 10.1176/appi.ajp.2008.08030343. [PubMed: 19047325]

Kousaie S, Phillips NA. Ageing and bilingualism: Absence of a "bilingual advantage" in Stroop interference in a nonimmigrant sample. Quarterly Journal of Experimental Psychology. 2012; 65:356–369. doi: /10.1080/17470218.2011.604788.

Leonard MK, Torres C, Travis KE, Brown TT, Hagler DJ Jr. Dale AM, Halgren E. Language proficiency modulates the recruitment of non-classical language areas in bilinguals. PLoS ONE. 2011; 6:e18240–e18240. doi: 10.1371/journal.pone.0018240. [PubMed: 21455315]

Liberati G, Raffone A, Olivetti Belardinelli M. Cognitive reserve and its implications for rehabilitation and Alzheimer's disease. Cognitive Processing. 2012; 13:1–12. doi:10.1007/s10339-011-0410-3. [PubMed: 21643921]

Liu Y, Julkunen V, Paajanen T, Westman E, Wahlund LO, Aitken A. Soininen H. Neuroradiology. 54:929–938. doi: 10.1007/s00234-012-1005-0.

Luk G, Bialystok E, Craik FIM, Grady CL. Lifelong Bilingualism Maintains White Matter Integrity in Older Adults. Journal of Neuroscience. 2011; 31:16808–16813. doi:10.1523/JNEUROSCI. 4563-11.2011. [PubMed: 22090506]

Luk G, Green DW, Abutalebi J, Grady CL. Cognitive control for language switching in bilinguals: A quantitative meta-analysis of functional neuroimaging studies. Language and Cognitive Processes. 2012; 27:1479–1488. [PubMed: 24795491]

Maguire EA, Gadian DG, Johnsrude IS, Good CD, Ashburner J, Frackowiak RS, Frith CD. Navigation-related structural change in the hippocampi of taxi drivers. Proceedings of the National Academy of Sciences. 2000; 11:4398–4403.

Mechelli A, Crinion JT, Noppeney U, O'Doherty J, Ashburner J, Frackowiak RS, Price CJ. Neurolinguistics: Structural plasticity in the bilingual brain. Nature. 2004; 431:757–757. doi: 10.1038/431757a. [PubMed: 15483594]

Meng X, D'Arcy C. Education and dementia in the context of the cognitive reserve hypothesis: A systematic review with meta-analyses and qualitative analyses. PLoS ONE. 2012; 7:1–16. doi: 10.1371/journal.pone.0038268.t002.

Murray AD, Staff RT, McNeil CJ, Salarirad S, Ahearn TS, Mustafa N, Whalley LJ. The balance between cognitive reserve and brain imaging biomarkers of cerebrovascular and Alzheimer's diseases. Brain. 2011; 134:3687–3696. doi: 10.1093/brain/awr259. [PubMed: 22102649]

Nithianantharajah J, Hannan AJ. Mechanisms mediating brain and cognitive reserve: Experience-dependent neuroprotection and functional compensation in animal models of neurodegenerative diseases. Progress in Neuropsychopharmachology & Biological Psychiatry. 2011; 35:331–339. doi: 10.1016/j.pnpbp.2010.10.026.

Ossher L, Bialystok E, Craik FIM, Murphy KJ, Troyer AK. The effect of bilingualism on amnestic mild cognitive impairment. The Journals of Gerontology Series B: Psychological Sciences and Social Sciences. 2013; 68:8–12. doi:10.1093/geronb/gbs038.

Paap KR, Greenberg ZI. There is no coherent evidence for a bilingual advantage in executive processing. Cognitive Psychology. 2013; 66:232–258. doi: 10.1016/j.cogpsych.2012.12.002. [PubMed: 23370226]

Pantev C, Herholz SC. Plasticity of the human auditory cortex related to musical training. Neuroscience and Beobehavioral Reviews. 2011; 35:2140–2154. doi: 10.1016/j.neubiorev. 2011.06.010.

Perani D, Paulesu E, Galles NS, Dupoux E, Dehaene S, Bettinardi V, Mehler J. The bilingual brain: proficiency and age of acquisition of the second language. Brain. 1998; 121:1841–1852. [PubMed: 9798741]

Perquin M, Vaillant M, Schuller AM, Pastore J, Dartigues JF, Lair ML, MemoVie Group. Lifelong exposure to multilingualism: New evidence to support cognitive reserve hypothesis. PLoS One. 8:e62030. doi: 10.1371/journal.pone.0062030. [PubMed: 23646113]

Reed BR, Dowling M, Tomaszewski Farias S, Sonnen J, Strauss M, Schneider JA, Mungas D. Cognitive activities during adulthood are more important than education in building reserve. Journal of the International Neuropsychological Society. 2011; 17:615–624. doi: 10.1017/S1355617711000014. [PubMed: 23131600]

Robertson IH. A noradrenergic theory of cognitive reserve: Implications for Alzheimer's disease. Neurobiology of Aging. 2012; 34:298–308. doi: 10.1016/j.neurobiolaging.2012.05.019. [PubMed: 22743090]

Roe CM, Mintun MA, Ghoshal N, Williams MM, Grant EA, Marcus DS, Morris JC. Alzheimer disease identification using amyloid imaging and reserve variables: Proof of concept. Neurology. 2010; 75:42–48. doi: 10.1212/WNL.0b013e3181e620f4. [PubMed: 20603484]

Roe CM, Xiong C, Miller JP, Morris JC. Education and Alzheimer’s disease without dementia: Support for the cognitive reserve hypothesis. Neurology. 2007; 68:223–228. doi:10.1212/01.wnl.0000251303.50459.8a. [PubMed: 17224578]

Sattler C, Toro P, Schönknecht P, Schröder J. Cognitive activity, education and socioeconomic status as preventive factors for mild cognitive impairment and Alzheimer's disease. Psychiatry Research. 2012; 196:90–95. doi:10.1016/j.psychres.2011.11.012. [PubMed: 22390831]

Sánchez Rodríguez JL, Torrellas C, Martín J, José Fernandez M. Cognitive reserve and lifestyle in Spanish individuals with sporadic Alzheimer's disease. American Journal of Alzheimer's Disease and Other Dementias. 2011; 26:542–554. doi:10.1177/1533317511428150.

Sanders AE, Hall CB, Katz MJ, Lipton RB. Non-native language use and risk of incident dementia in the elderly. Journal of Alzheimer’s Disease. 2012; 29:99–108. doi: 10.3233/JAD-2011-111631.

SantaCruz KS, Sonnen JA, Pezhouh MK, Desrosiers MF, Nelson PT, Tyas SL. Alzheimer disease pathology in subjects without dementia in 2 studies of aging: The Nun Study and the Adult Changes in Thought Study. Journal of Neuropathology and Experimental Neurology. 2011; 70:832–840. doi: 10.1097/NEN.0b013e31822e8ae9. [PubMed: 21937909]

Satz P. Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for Threshold theory. Neuropsychology. 1993; 7:273–295.

Schweizer TA, Craik FIM, Bialystok E. Bilingualism, not immigration status, is associated with maintained cognitive level in Alzheimer’s disease. Cortex. 2013; 49:1442–1443.

Schweizer TA, Ware J, Fischer CE, Craik FIM, Bialystok E. Bilingualism as a contributor to cognitive reserve: Evidence from brain atrophy in Alzheimer’s disease. Cortex. 2012; 48:991–996. doi: 10.1016/j.cortex.2011.04.009. [PubMed: 21596373]

Snowdon DA. Healthy aging and dementia: Findings from the Nun Study. Annals of Internal Medicine. 2003; 139:450–454. [PubMed: 12965975]

Snowdon DA, Kemper SJ, Mortimer JA, Greiner LH, Wekstein DR, Markesbery WR. Linguistic ability in early life and cognitive function and AD in late life. Findings from the Nun study. JAMA: the Journal of the American Medical Association. 1996; 275:528–532.

Solé-Padullés C, Bartrés-Faz D, Junqué C, Vendrell P, Rami L, Clemente IC, Molinuevo JL. Brain structure and function related to cognitive reserve variables in normal aging, mild cognitive impairment and Alzheimer's disease. Neurobiology of Aging. 2009; 30:1114–1124. doi:10.1016/j.neurobiolaging.2007.10.008. [PubMed: 18053618]

Steffener J, Stern Y. Exploring the neural basis of cognitive reserve in aging. Biochimica et Biophysica Acta. 2012; 1822:467–473. doi:10.1016/j.bbadis.2011.09.012. [PubMed: 21982946]

Steffener J, Reuben A, Rakitin BC, Stern Y. Supporting performance in the face of age-related neural changes: Testing mechanistic roles of cognitive reserve. Brain imaging and Behavior. 2011; 5:212–221. doi:10.1007/s11682-011-9125-4. [PubMed: 21607547]

Stern Y. What is cognitive reserve? Theory and research application of the reserve concept. Journal of the International Neuropsychological Society. 2002; 8:448–60. [PubMed: 11939702]

Stern Y. Cognitive reserve and Alzheimer's disease. Neuropsychologia. 2009; 47:2015–2028. doi: 10.1016/j.neuropsychologia.2009.03.004. [PubMed: 19467352]

Stern Y. Elaborating a hypothetical concept: Comments on the special series in cognitive reserve. Journal of the Internation Neuropsychological Society. 2011; 17:639–642.

Stern Y. Cognitive reserve in ageing and Alzheimer's disease. Lancet Neurology. 2012; 11:1006–1012. doi: 10.1016/S1474-4422(12)70191-6.

Stern Y, Habeck C, Moeller J, Scarmeas N, Anderson KE, Hilton HJ, van Heertum R. Brain networks associated with cognitive reserve in healthy young and old adults. Cerebral Cortex. 2005; 15:394–402. doi:10.1093/cercor/bhh142. [PubMed: 15749983]

Sumowski JF, Chiaravalloti N, DeLuca J. Cognitive reserve protects against cognitive dysfunction in multiple sclerosis. Journal of Clinical and Experimental Neuropsychology. 2009; 31:913–926. doi: 10.1080/13803390902740643. [PubMed: 19330566]

Vemuri P, Weigand SD, Przybelski SA, Knopman DS, Smith GE, Trojanowski J, Alzheimer's Disease Neuroimaging Initiative. Cognitive reserve and Alzheimer's disease biomarkers are independent determinants of cognition. Brain: A Journal of Neurology. 2011; 134:1479–1492. doi:10.1093/brain/awr049. [PubMed: 21478184]

Zahodne LB, Schofield PW, Farrell MT, Stern Y, Manly JJ. Bilingualism does not alter cognitive decline or dementia risk among Spanish-speaking immigrants. Neuropsychology. 2013 doi: 10.1037/neu0000014.