

learning a language increases cognitive reserve and delay's cognitive decline and dementia

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Introduction:

With the growing aging population in the world there is also increase in conditions such as cognitive decline and dementia which commonly occurs in the older age. Dementia is a deterioration in cognitive functioning, affecting a individual's memory, thinking and behaviour to the extent that it affects a person's ability to perform day to day activities. There are different types of dementia among which the predominant one is alzheimer's dementia which accounts to 60 to 70% of dementia cases. Cognitive reserve is the resilience provided by the brain against neuropathological damage^{1,2}. Brain with higher cognitive reserve is able to more efficiently use the existing neural networks or alternative neural networks to sustain the ill effects of damage before reaching a threshold for clinical expression^{1,3}. By increasing cognitive reserve, the onset of dementia can be delayed². Non pharmacological interventions that improve cognitive function are healthy diet, physical activity and cognitive training⁴. Among the cognitively stimulating activities that a person can do to improve cognitive functioning is learning a new language. Learning a foreign language provides healthy cognitive aging. This article is a brief overview on studies that have shown a delay in dementia onset and how neuroplasticity and cognitive reserve can contribute to delaying cognitive decline.

Brain reserve , neural and cognitive reserve

The extent of brain damage or brain pathology is not propotional to the clinical manifestation of that damage³. The term reserve is used to bridge this gap between the extent of brain damage and its outcome³. Brain reserve refers to the brain size or the neuronal count⁵. Brain reserve indicates to inherent differences in neuronal count or structure of the brain⁶. It is measured by estimation of brain volume and by postmortem determination of neuronal number or size and synaptic density⁶. Larger brains can withstand more pathology as there is sufficient neural substrate remaining before the threshold is reached for clinical manifestation of the disease⁵. Cognitive reserve indicates to discrepancies in the way individuals take advantage of adaptive cognitive system and enlist neural networks to sustain normal cognition in the presence of neuropathologic burden⁶. Cognitive reserve is represented using markers such as general ability level, and lifestyle elements like cognitive, social, education, occupation and physical leisure activities⁶.

Cognitive reserve refers to the flexible and efficient use of the cognitive networks when doing tasks in the existence of brain pathology^{5,7}. Reserve can be approximately divided into active and passive models⁸. Passive model is an example of brain reserve, which derives from neuronal count or brain size⁸. Due to the presence of adequate amount of neural substrates to support normal function in larger brains, they can withstand more insult before clinical deficit surfaces⁸. This concept of reserve is expressed in the threshold model for acquired brain injury, which explains the idea of brain reserve capacity⁸. The model shows that there are individual variations in brain reserve capacity⁸. This model assumes that once brain reserve capacity is lost beyond a certain fixed critical threshold specific functional or clinical impairment occurs⁸. Therefore individual variations in brain reserve capacity causes variations in clinical expression of certain level of damage to the brain⁸. In this model it is assumed that a certain type of brain damage will have the same result in each person and instances where brain

damage occurs repeatedly is summed together. Individuals vary only in their overall brain capacity and so the brain damage is enough or not enough to deplete the brain reserve capacity to some critical level for clinical expression of the disease⁸. Active model is an example of cognitive reserve. Active model states that although two patients may have the same amount of brain, when a brain damage occurs the brain actively tries to cope with the damage by using the preexisting cognitive processes or by incorporating compensatory processes⁸. Even if two patients have the same amount of brain reserve capacity, the patient with higher cognitive reserve will be able to better tolerate larger lesion than the other patient before clinical deficit becomes evident⁸. Thus the active model does not consider a definitive cut-off or threshold at which functional deficit will occur, it rather concentrates on the processes that causes individuals to uphold brain damage and maintain function. The distinction between brain reserve and cognitive reserve is not clear cut⁸. The differences in cognitive processing foreseen by the cognitive reserve model should also have a physiologic basis, as in the brain should finally mediate all cognitive function. The physiologic changes incorporated by cognitive reserve is at the level of changes in synaptic organisation or in relative utilisation of certain brain areas. Thus cognitive reserve refers to anatomic changeability at the level of brain networks whereas brain reserve refers to variation in the quantity of available neural substrate⁸. Higher IQ and cognitively stimulating activities increase brain volume. Exercise and cognitive stimulation aids neurogenesis in the dentate gyrus and regulate factors that increase neuronal plasticity and provides opposition to cell death⁸. One such cognitively stimulating activity is learning a foreign language⁸. Cognitive reserve is not fixed at any point of time in a person's life rather is influenced by a combination of exposures⁸. Higher education, occupational attainment, and leisure activities independently contributes to cognitive reserve. Alzheimer's disease occurs later in individuals with higher cognitive reserve⁸. Greater reserve is related to significantly lower risk for developing dementia. Neural basis of cognitive reserve and brain behaviour variations that occur with aging can be studied using neuroimaging techniques⁸.

According to Del Maschio.N et al Delay in cognitive decline observed in bilingual seniors can be explained using two neurocognitive mechanisms⁹, they being the neural reserve and cognitive reserve. Both these constructs appears to be brought about by enhanced cognitive load for executive functions that bilingualism involve across the lifetime⁹. Neural reserve can be explained as the capacity to withstand the expected age associated decline in cognitive function and pathology of the brain⁹. Anatomic indicators such as grey matter density, brain size, branching of dendrites and synaptic count are recognised as active measures of neural reserve and related with occurrence, risk and intensity of dementing disorders⁹. Cognitive reserve can be explained as the difference between the radical levels of age associated cognitive decline or pathology and identified cognitive or functional effectiveness. Dissimilar to neural reserve, cognitive reserve utilises the alternative or back up brain networks or other cognitive approach as a means of compensation for cognitive deterioration induced by age and pathology⁹.

Cognitive processing to a certain extent can withstand neurodegenerative pathology in bilinguals with Alzheimer's disease when compared to monolinguals⁹. Thus although it is likely that neural reserve is related to greater cognitive reserve in healthy aging, neural reserve and cognitive reserve are divisible components in bilingual seniors⁹.

Positive effect of bilingualism on vocabulary acquisition and executive function

Antoniou M and all in 2013 conducted a study to estimate whether learning a foreign language is expected to make a large contribution to building cognitive reserve¹. On testing a variety of cognitive

tasks, older adults who spoke two or more languages performed better than adults who spoke only a single language especially in tasks that demand executive control or selective attention^{1,10,11}. This could be because the event of learning two languages, involved a different set of attention and control procedure¹. Learning two languages depends highly on functions of executive control and attention, so learning two languages trains the executive function better resulting in more developed executive function in bilinguals than in monolinguals¹¹. Bialystok E and all in 2009 showed that in individuals who learnt another language had a greater density of grey matter in the left inferior parietal cortex which is a region responsive to vocabulary acquisition¹². He also showed that knowing a second language has a positive effect on executive function^{11,12}. In children who know a second language had the capacity to solve problems that had deceptive and conflicting cues at a earlier age than children who did not know a second language¹². Perani.D and all in 2015 also highlights instances where bilinguals outperform monolinguals in episodic recall tasks and general intelligence independent of age¹¹.

The aim of the study conducted by Del Maschio et al was to examine the hypothesis that by practicing bilingualism throughout life is related to a higher cognitive and neural reserve in healthy aging by checking the neurostructural changes in areas of interest in the brain which are known to play a role in the executive function of the young and elderly bilinguals⁹. Here the comparison between bilingual and monolingual speakers were done matched for age⁹. The author's main hypothesis was based on previous theories that bilingual individuals highly depend on executive control to be able to speak in one language without interference from another language⁹. This may result in a improved training of executive control function in bilinguals than monolinguals, so that bilinguals perform better than monolinguals in tasks that demand conflict monitoring and resolution⁹. Evidence from neuroimaging indicates that elaborate use of executive function has functional and structural influence in areas of cognitive control network such as the prefrontal cortex, anterior cingulate cortex, inferior parietal lobule and dorsal striatum that intermediates particular demand of bilingual language processing⁹.

Neural reserve and neural compensation

Two neural mechanism can explain how bilingualism safeguards the aging brain, one is neural reserve and the other is neural compensation¹⁰. In accordance with the neural reserve concept, Lifelong usage of two languages brings about structural changes in the brain like increase in grey and white matter density in certain networks of the brain. These networks are the domain of executive functioning and language learning¹⁰. The rational that underlie this concept is lifelong increased use of executive function, which controls the two language process enables the speaker to speak in one language without intrusion from the other language, this stimulates plastic changes in the brain leading to neural reserve¹⁰. This neural reserve makes the brain resilient to aging effects on the brain¹⁴.

According to the concept of neural compensation, bilingualism, brings about Increased cognitive load on executive function which results in stronger intrinsic functional connectivity in the fronto parietal control network which is related to executive control^{10,11}. This stronger functional connectivity makes the brain able to cope more with loss of neurons and brain atrophy that may occur with aging, neurodegeneration and dementia¹⁰. Only individuals with regular and life long usage of second language, show significant neuroprotective effects¹⁰. Study by Perani D and all in 2017 showed evidence that among AD patients those with higher relative use and exposure to a second language, exhibited a superior neural compensation¹⁰. This study reflects how bilingualism acts as a powerful source of cognitive reserve¹⁰. This protective bilingual effect was independent of confounding factors such as gender, education, occupation, and urban vs rural dwelling¹⁰.

Studies showing delayed onset of dementia in bilinguals

Certain life experiences contributes to reserve and protects from the development of Alzheimer's pathology⁵. One such life experience is learning a new language. Peak brain volume can make better

the effects of pathology in the brain on cognitive function and signs of dementia¹³. Higher level of cognitive reserve may delay the appearance of symptoms of dementia¹³. Study by Bialystok E and all in 2007 showed that in individuals who knew another language had delayed onset of dementia by four years when compared to individuals who did not know another language¹³.

A study was conducted by Alladi S and all in 2013 by reviewing case records of patients diagnosed with any one of the following types of dementia i.e alzheimer's dementia, frontotemporal dementia, Vascular dementia, dementia with lewy bodies and mixed dementia from memory clinic of a university hospital in Hyderabad. The effect of bilingualism on dementia was examined. There was a delay in onset of dementia symptoms by around 4.5 years in bilinguals in comparison with monolinguals. This effect of bilingualism on age of onset of dementia was independent of confounding factors such as gender, education, occupation, cardiovascular risk factors and urban vs rural dwelling¹⁴.

In a study conducted by Woumans.E and all in 2015 among non immigrant homogenous population in Belgium; homogenous with respect to culture, ethnicity, environment, and patterns of language use. In bilinguals there was a delay in the clinical manifestation of alzheimer's disease by 4.6 years and diagnosis of alzheimer's disease by 4.8 years¹⁵. The study participants were all patients with a probable diagnosis of alzheimer's disease¹⁵. Age of acquiring second language did not impact this effect¹⁷. This study was conducted after controlling for confounding variable such as gender, education and occupation¹⁵.

Learning a new language has global effects on cognitive functioning

Higher reserve is significantly and robustly associated with diminished cognitive decline^{16,17}. The fact that knowing another language can delay the onset of dementia shows how a psychological factor can influence a biologically based disease state¹³. Unlike the concept that plasticity traced to a certain experience is specific for that particular skill, the study by Bialystok E and all in 2007 showed that considerable experience in one type of activity, like learning a new language can have global effects on general cognitive functioning¹³.

Mild cognitive impairment

Mild cognitive impairment (MCI) refers to a set of individuals who have some cognitive deficit but which is of inadequate severity to be termed as dementia¹⁸. MCI usually occurs following the preclinical stage of alzheimer's disease, before it progresses to mild dementia due to alzheimer's disease¹⁰. MCI is a transition between cognitive changes of normal ageing and very early dementia¹⁸, however not all MCI develop into dementia or Alzheimer's disease¹⁸. Foreign language instruction during childhood is associated with lower risk of acquiring MCI¹⁹. Such exposures to early life instruction, leads to cognitive and brain development, which increases cognitive reserve and delays the onset of MCI¹⁹. Perani.D and all in 2015 mentions about studies which reveal that in bilinguals incidence of MCI is lower than in monolinguals¹¹. The risk of MCI was 30 % less in individuals who before the age of 18 years were exposed to more than 4 years of foreign language training

Neuroplasticity may delay cognitive decline

In humans connections among brain cells reorganises itself from the time the brain starts to develop in utero till the day we die^{20,21,,22}. When something new is learnt we create new connections between brain cells and the internal structure of the available synapses change^{21,22}. When we excel in a particular domain the region of the brain concerned with the development of that skill grows²¹. For example in bilinguals learning a second language produces functional changes in the brain²¹. The left inferior parietal cortex which is concerned with language is larger in the bilingual brain than monolingual brain²¹.

Michael Merzenich known as the “father of neuroplasticity” established that human brain is highly plastic. Marian Diamond known as the “Mother of neuroplasticity”, was the first to prove that brain reduces in size with impoverishment and increases in size in the presence of enriched environment at any age²³. Environmental stimulation is necessary for increasing and maintaining cognitive function. One way of providing a enriched environment or environmental stimulation is learning a foreign language²³. Even aged brains retain a enhanced level of neuroplasticity, Therefore a promising approach to attain successful aging and to prevent age related cognitive decline is to boost and recruit neuroplasticity in a positive direction²³.

Among others intensity and frequency of language use brings about experience-dependent neural changes in the brain²⁴. Different neuroimaging methods have revealed that learning a second language even in late adulthood brings about behavioural and neural changes that is almost similar to patterns of learning the native or first language²⁴. Grey matter (GM) density was studied in bilinguals using voxel brain morphometry by Mechelli and all in 2004. Here bilinguals were participants who learned European language and when aged below five years were termed as early bilinguals and if learned the European language between ten and fifteen years were termed as late bilinguals. Overall bilinguals showed a higher GM density in the inferior parietal lobule (IPL) than monolinguals. IPL is a brain region linked to executive control¹⁹. IPL is associated with phonological working memory, lexical learning and semantic integration. The enlargement of this region may be due to the bilingual's acquisition and processing of a greater vocabulary as a result of second language learning²⁴. Also in more proficient language learners and language learners at a earlier age showed more GM²⁴. A study by Luk and all in 2011 assessed older bilinguals and monolinguals at an average age around 70 years, whose first language was English²⁴. All the bilinguals here had learned their second language before the age of 11 years and commonly used both the languages throughout their lives²⁴. White matter (WM) integrity and resting state functional connectivity were both examined²⁴. Bilinguals when compared to the monolinguals showed greater WM integrity in the corpus callosum projecting to the right inferior fronto occipital fasciculus (IFOF)²⁴. IFOF is associated with function of semantic language processing and goal oriented behaviour. Bilinguals also showed that white matter structural connectivity correlated with resting state functional connectivity, specifically for the frontal region showing parallel structure function changes due to lifelong bilingualism. In alzheimer's disease there is GM atrophy, which may be compensated by higher WM integrity in bilinguals. This could account for the neural basis for the concept of cognitive reserve²⁴. Practicing bilingualism for all life may serve as a major block to the onset of age related cognitive decline¹². The concept that bilingualism protects against age related cognitive decline was examined in another study by Abutalebi and all in 2014 which showed that bilingualism protected against fast decrease of GM volume in older adults²⁴. Increase in WM integrity and GM volume, specifically in aging populations provides neural reserve which may in due course serve to protect against age related cognitive decline^{25,11}.

Studies showing increased grey matter density and microstructural integrity of white matter pathways as well as a delay in clinical dementia by around 4 to 5 years all indicate to that the bilingual exposure acts as a possible shield against neurodegeneration⁹. In general it can be put forth that bilingualism imparts the brain with more safeguard to brain atrophy and potential age associated disease. This the brain does either by increasing the neural substrates or by recruiting neural compensatory mechanisms, to hone cognitive performance²³. However it cannot be yet assumed that neural reserve is related to cognitive reserve at the functional level in bilingual healthy aging⁹.

The study by Del Maschio et al tested if in young and aging population matched for socio economic status and education bilingualism may distinctly foretell executive control capabilities in the groups under examination⁹. Grey matter volume was taken as a structural measure of neural reserve and Flanker test was considered as a behavioural indicator of cognitive efficiency. Flanker test was used as a standard test for attention and conflict supervised studies. This test expected aging to be associated with reduced grey matter volume and reduced achievement in flanker test in both

monolingual and bilingual. It was also expected that practicing bilingualism for entire life would safeguard bilingual seniors from age related cognitive decline⁹. Therefore it was expected that there would be a increased grey matter volume in important structures of executive control network like the prefrontal cortex and anterior cingulate cortex in bilinguals when compared to monolinguals. It was also expected increase grey matter volume to be related to improved performance in the flanker test i.e associated with faster response time in flanker test. As expected the results showed reduced grey matter volume in seniors than young adults⁹ Also as expected bilingual seniors showed a better neural reserve than monolingual senior⁹ i.e in all regions of interest except for the bilateral caudate there was increased grey matter volume for bilinguals than monolinguals, this finding holds good for both the young and older bilinguals in comparison with their monolingual counterparts. Increased grey matter volume in bilinguals was noted extending from young adulthood to older adulthood, exhibiting that speaking and regulating for two or more languages, causes early neurostructural changes that occurs across lifespan. It was assumed that anterior cingulate cortex had increased grey matter volume in bilinguals because of the ever existing challenge of correlating between languages while simultaneously preventing interference from the other language. Anterior cingulate cortex plays a important role in the executive control network, with respect to conflict and error supervision in both the non verbal and verbal domains. One consequence of this study is that neural reserve in bilinguals develops from a early age and higher neural reserve relies on practicing bilingualism for a lifetime⁹.

Another study shows a increased grey matter volume in broad areas of cortical regions, including prefrontal cortex and right inferior parietal lobule. Prefrontal cortex is concerned with response selection and inhibition in background of presenting interfering information⁹. Grey matter volume can be assumed to be increased in the prefrontal cortex in bilingual speakers due to their continuous ability to be committed in their overruling predominant responses from the dominant language⁹. Further neuroimaging evidence shows increased neural reserve for bilinguals in the bilateral inferior parietal lobule which can be credited to the role this region plays in the language switching contexts⁹. Therefore results of this study shows how knowledge in a second language enhances cognitive reserve even in the presence of cognitive decline⁹. Strengthening of the synapses and neural connections of the executive control organisation may permit bilinguals to adapt more effectively to healthy brain aging⁹.

Previous studies have shown that the effect of language exposure on the brain is the influence it exerts on the language control process²⁵. When a bilingual is less exposed to one language in comparison to the other, then the automaticity of word finding with the less exposed language becomes less simply because the less exposed language becomes less available for spontaneous word production²⁵. If automaticity in lexical access reduces with decreased exposure, there will be a enhanced competitive demand to select the less exposed language, in other words an increased neural effort for the use of less exposed language and increased inhibition of the more exposed language²⁵. So Tu.L et al hypothesised that the language a bilingual uses less for a short period maybe in higher need of mental control. The bilinguals here spoke Cantonese (L1) and Mandarin (L2). The effect of differential language exposure on the network that governs language control was studied. This network included brain regions such as bilateral prefrontal cortex (PFC), bilateral caudate nuclei (CN), and the anterior cingulate gyrus (ACG). Language exposure in this study reflected the degree of L1 and L2 usage, calculated as the duration spent in speaking, writing listening and reading of the respective language. The effects of both the languages on the brain was evaluated using two blood oxygenation level dependent (BOLD) functional magnetic resonance imaging (fMRI) scans done in a interval of three months²⁵. The first fMRI was done during the semester when there was almost equal usage of L1 and L2. Two months after the first imaging subjects began their summer vacation when there was a significant higher usage of L1 than L2, for atleast 30 days. The second fMRI was done immediately after summer holidays, to account for the differential language exposure²⁵. There was a functional effect exerted on the brain due to the short 30 day of differential language exposure. Reduced exposure and usage of L2 showed a heightened involvement of left anterior cingulate gyrus which is

concerned with controlling and monitoring languages. Imaging results showed increased activation of left pars opercularis in bilinguals than monolinguals. Left pars opercularis which was found to play a important role in language production is also found to be involved in language control. This increased activation of left pars opercularis in bilinguals than monolinguals could be because of the increased need in controlling verbal interference. Due to the thirty day period of increased use of L1 words, the activation threshold for L2 words has risen i.e due to the decreased exposure of L2 words has made access to L2 words cumbersome. This study shows that decreased use of a certain language leads to increased involvement of language control regions that maybe essential to regulate the particular language system in bilinguals²⁵.

Individuals with the same amount of neuropathological burden exhibited different level of cognitive function, this difference can be explained in terms of reserve. There are two types of reserve, brain reserve and cognitive reserve (CR)²⁶. According to C.Groot et al, Cognitive reserve either by making use of the present neural networks or by recruiting alternative neural network tries to maintain the cognitive function despite the presence of neuropathology²⁶. Cognitive reserve is measured as educational attainment²⁶. Brain reserve (BR) is the amount of neural resources that is utilised to defend the brain against emerging neuropathology. Brain reserve is measured using neuroimaging techniques as the intracranial volume²⁶. In this study by C.Groot et al the independent and additional effects of Brain reserve and cognitive reserve on attention,visuospatial,memory and executive function on Alzheimer disease biomarker positive participants was determined after controlling for the extent of neurodegeneration which is measured by cerebral atrophy²⁶. The final study sample included participants comprising of 201 subjects with predementia, 70 subjects with subjective cognitive decline, 131 subjects with mild cognitive impairment, and 462 subjects with probable Alzheimer disease dementia²⁶. The study results indicated that In patients with biomarker positive alzheimer's disease adjusted for cerebral atrophy both cognitive reserve and brain reserve had positive influence on cognition²⁶. The influence of cognitive reserve on executive functioning and attention, were higher in predementia participants compared to participants with dementia²⁶. Overall influence of cognitive reserve was higher than influence of brain reserve²⁶. A linear trend for improved cognitive performance was observed in all domains in all the following groups CR+ve/BR+ve, CR-ve/BR+ve, CR+ve/BR-ve, CR-ve/BR-ve groups.

Better education and higher intracranial volume had a positive impact on cognitive trajectory of participants with Alzheimer's disease²⁶. Cognitive reserve and brain reserve differentially alleviate cognitive symptoms in AD with CR being particularly useful in the predementia stages, and the influence of CR in general was higher than BR²⁶. C.Groot et al states that CR and BR are not interchangeable terms rather atleast partially independent components of a larger conception which is reserve. The safeguard rendered by cognitive reserve could be because of physiologic components such as strengthened network reliability, novel cognitive approach, and modulation of functional connectivity which is all related to higher education²⁶. Increased resistance to neuropathology rendered by brain reserve could be attributed to presence of increased quantities of premorbid brain parenchyma. In the incidence of occurrence of neurodegeneration the structural integrity essential to sustain normal cognitive function will be preserved for a longer time for participants with a greater BR than participants with a lower BR. CR has the highest ability to delay cognitive deterioration in Alzheimer's disease²⁶. The positive effects of CR are more evident when CR is higher during earlier phase of the disease so interventions aiming to increase cognitive reserve when given during the early stages of alzheimer's disease are more effective²⁶. Interventions to enhance CR when given to participants with greater BR show better treatment effects²⁶

Conclusion :

Human brain cells continuously reorganises itself from the day we are born till we die. When we become skilled in a specific task the region of the brain corresponding to that task also grows. All the

above studies throws light on the concept that doing cognitively stimulating activities like learning a language or in general learning any subject strengthens the existing synapses and forms new neural connections associated with learning that specific skill. So learning a language increases cognitive reserve and even delays the development of Alzheimer's dementia or any other neurodegenerative disease.

References .

1. Antoniou M, Gunasekera GM, Wong PC. Foreign language training as cognitive therapy for age-related cognitive decline: a hypothesis for future research. *Neuroscience & Biobehavioral Reviews*. 2013 Dec 1;37(10):2689-98.
2. Perani D, Abutalebi J. Bilingualism, dementia, cognitive and neural reserve. *Current opinion in neurology*. 2015 Dec 1;28(6):618-25.
3. Stern Y. What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the international neuropsychological society*. 2002 Mar;8(3):448-60.
4. Klimova B. Learning a foreign language: A review on recent findings about its effect on the enhancement of cognitive functions among healthy older individuals. *Frontiers in human neuroscience*. 2018 Jul 30;12:305.
5. Stern Y. Cognitive reserve: implications for assessment and intervention. *Folia Phoniatrica et Logopaedica*. 2013;65(2):49-54.
6. Jicha GA, Rentz DM. Cognitive and brain reserve and the diagnosis and treatment of preclinical Alzheimer disease.
7. Lenehan, M. E., Summers, M. J., Saunders, N. L., Summers, J. J., Ward, D. D., Ritchie, K., & Vickers, J. C. (2016). Sending your grandparents to university increases cognitive reserve: The Tasmanian Healthy Brain Project. *Neuropsychology*, 30(5), 525
8. Stern Y. Cognitive reserve. *Neuropsychologia*. 2009 Aug 1;47(10):2015-28.
9. Del Maschio N, Sulpizio S, Gallo F, Fedeli D, Weekes BS, Abutalebi J. Neuroplasticity across the lifespan and aging effects in bilinguals and monolinguals. *Brain and cognition*. 2018 Aug 1;125:118-26.
10. Perani D, Farsad M, Ballarini T, Lubian F, Malpetti M, Fracchetti A, Magnani G, March A, Abutalebi J. The impact of bilingualism on brain reserve and metabolic connectivity in Alzheimer's fm dementia. *Proceedings of the National l xl Academy of Sciences*. 2017 Feb
11. Perani D, Abutalebi J. Bilingualism, dementia, cognitive and neural reserve. *Current opinion in neurology*. 2015 Dec 1;28(6):618-25
12. Bialystok E. Bilingualism: The good, the bad, and the indifferent. *Bilingualism: Language and cognition*. 2009 Jan;12(1):3-11.
13. Bialystok E, Craik FI, Freedman M. Bilingualism as a protection against the onset of symptoms of dementia. *Neuropsychologia*. 2007 Jan 1;45(2):459-64.
14. Alladi S, Bak TH, Duggirala V, Surampudi B, Shailaja M, Shukla AK, Chaudhuri JR, Kaul S. Bilingualism delays age at onset of dementia, independent of education and immigration status. *Neurology*. 2013 Nov 26;81(22):1938-44.

15. Woumans EV, Santens P, Sieben A, Versijpt JA, Stevens M, Duyck W. Bilingualism delays clinical manifestation of Alzheimer's disease. *Bilingualism: Language and Cognition*. 2015 Jul;18(3):568-74
16. Valenzuela MJ, Sachdev P. Brain reserve and cognitive decline: a non-parametric systematic review. *Psychological medicine*. 2006 Aug;36(8):1065-73.
17. Valenzuela MJ, Sachdev P. Brain reserve and dementia: a systematic review. *Psychological medicine*. 2006 Apr;36(4):441-54.
18. Petersen RC. Mild cognitive impairment as a diagnostic entity. *Journal of internal medicine*. 2004 Sep;256(3):183-94.
19. Wilson RS, Boyle PA, Yang J, James BD, Bennett DA. Early life instruction in foreign language and music and incidence of mild cognitive impairment. *Neuropsychology*. 2015 Mar;29(2):292.
20. Neuroplasticity. Retrieved from: https://www.google.com/search?q=neuroplasticity&sxsrf=ALeKk00ysEuL4jgtrmqntWZbj1bNr68JEg:1591342496246&source=lnms&tbn=isch&sa=X&ved=2ahUKEwi0kdHh1OrpAhXE4jgGHVfKDYQ_AUoAXoECBIQAw&biw=1280&bih=689#imgsrc=vsE1BC4rfRvZ9M
21. Dr. Michelon P. Brain plasticity: How learning changes your brain. 2008. Retrieved from : <https://sharpbrains.com/blog/2008/02/26/brain-plasticity-how-learning-changes-your-brain/>
22. Ackerman C.E. What is neuroplasticity? A psychologist explains [+14 exercises]. 2020. Retrieved from: <https://positivepsychology.com/neuroplasticity/>
23. Shaffer J. Neuroplasticity and clinical practice: building brain power for health. *Frontiers in Psychology*. 2016 Jul 26;7:1118.
24. Li P, Legault J, Litcofsky KA. Neuroplasticity as a function of second language learning: anatomical changes in the human brain. *Cortex*. 2014 Sep 1;58:301-24.
25. Tu L, Wang J, Abutalebi J, Jiang B, Pan X, Li M, Gao W, Yang Y, Liang B, Lu Z, Huang R. Language exposure induced neuroplasticity in the bilingual brain: A follow-up fMRI study. *Cortex*. 2015 Mar 1;64:8-19.
26. Groot C, van Loenhoud AC, Barkhof F, van Berckel BN, Koene T, Teunissen CC, Scheltens P, van der Flier WM, Ossenkoppele R. Differential effects of cognitive reserve and brain reserve on cognition in Alzheimer disease. *Neurology*. 2018 Jan 9;90(2):e149-56.