

MYTHBUSTERS

Myth: Dementia is a Normal Part of Aging

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Abstract

It is commonly assumed that cognitive function deteriorates with age. While some age-related changes in cognitive function exist, they do not exist to the extent that is widely believed. The pathologic changes seen in patients with dementia differ significantly from those seen in healthy elderly persons. Additionally, many seniors exhibit virtually no changes at all and continue to lead fruitful lives well beyond the average life expectancy. There are important implications of assuming that dementia is a normal part of aging: this myth contributes to poorer cognitive performance in seniors, to stigma and to a lack of initiative in preventing cognitive decline. Here the author demonstrates the prevalence of this myth and then challenges it, taking into account current evidence regarding normal age-related cognitive changes and pathologic cognitive changes as seen in patients with dementia.



There remains a commonly held assumption that cognitive impairment is an inevitable aspect of aging, despite evidence to support the contrary.² While select age-related cognitive changes certainly exist, a decline in overall functioning is not observed in healthy older adults.

Many examples of this myth are present in the media, scientific literature and everyday life. A particularly striking example was published in the media three years ago after an unfortunate on-air moment involving Don Cherry. Cherry verbally attacked the actions of Toronto's City Hall and was subsequently described in the *National Post* as "your basic senile grandpa."³ While Cherry's remarks could have been more eloquently worded, so could the reporter's remarks from the *National Post*. The comments in the article not only attribute Cherry's verbal attack to his age, but also assume that senility is a "basic" grandfatherly trait. In a separate article from the UK⁴, an author criticizes the idea of abolishing the age of retirement due to concerns of, "a workforce suffering from senile dementia".

This sweeping generalization that older workers will inevitably suffer from dementia is, needless to say, in stark contrast to the true prevalence of dementia.⁵

Several articles in the scientific literature have more formally demonstrated the prevalence of the myth that dementia is a normal part of aging. They include the articles, "Facts on Aging Quiz: A Review of Findings" by Palmore⁶, "Re-Vision of Older Television Characters: A Stereotype - Awareness Intervention"⁷ and, "Do Older Adults Expect to Age Successfully?" by Sarkisian et al.⁸ Palmore's groundbreaking publications have demonstrated that the majority of people believe "senility" is inevitable after the age of 65 when in fact only 4% of people over the age 65 have dementia and only 10% over the age of 65 have mild cognitive impairment. Mild cognitive impairment is defined as an intermediate stage between normal cognitive aging and dementia that does not affect activities of daily living.

Current research in the field of cognitive neuroscience is beginning to provide insight into the process of normal, healthy cognitive aging and how this differs from the pathologic changes observed in dementia. Importantly, research demonstrates that aging in itself does not lead to a global cognitive deficit², despite age-related changes in the brain. In other words, there are components of cognitive functioning that undergo predictable changes with age. However, in healthy people this does not lead to a significant overall decline in functioning due to compensatory mechanisms. Additionally, research has shown that dementia is not an inevitable result of aging, despite age being the greatest risk factor for dementia.²

The normal brain shows distinct patterns of aging, including both structural and functional changes. In general, a decrease in volume is seen, which accelerates after the age of 70.⁹ This decrease in volume is most prominent in the frontal lobe and in the hippocampal area,¹⁰⁻¹² although the hippocampal decline is substantially less than what is observed in Alzheimer's Disease. A decrease in white matter density is also seen, which follows a similar pattern to the volume changes.¹³⁻¹⁵ Functional changes in the aging brain consist of a general slowing of functional and resting blood flow and oxygen consumption¹⁶, as well as the recruitment of additional areas of activation.¹⁷ It is thought that the recruitment of additional areas of activation assists to compensate for a decrease in volume and processing speed secondary to the changes mentioned above thus helping to prevent a global decline. These brain changes translate to specific alterations in cognitive function. Age-related decreases are seen predominantly in effortful or active cognitive processes, whereas passive or automatic processes tend to be unaffected.¹⁸ Specifically, selective and divided attention¹⁹⁻²¹, active/novel problem solving²², and explicit²³, contextual²⁴ and prospective memory²⁵ tend to diminish with age. Interestingly, it appears that the ability to encode new information is largely unaffected by age, but rather the ability to retrieve new information becomes strained.²⁶ As well, the cumulative effect of learning over one's lifespan actually increases semantic memory or factual knowledge.²⁷⁻²⁸

In contrast to cognitive changes of normal aging described above, pathologic changes in patients with dementia result in "a persistent state of serious cognitive, functional, and emotional deterioration from a previously higher level of functioning."²⁹ Very generally speaking, the pathologic changes seen in various types of dementia consist of changes that are also observed with advancing age, but to a greater degree. This includes the accumulation of beta amyloid plaques³⁰, reduction in brain volume, especially in the frontal lobe and hippocampus, and white matter changes. Additionally, many disease-specific changes can occur, such as the phosphorylation and accumulation of tau-protein in Alzheimer's Disease³¹, and the deposition of lewy-bodies in Lewy Body Dementia. Dementias are a heterogeneous group of syndromes that can be caused by different types of insults, including vascular insult, infection, malignancy and substance abuse. Interestingly, recent studies have shown that the degree of pathology seen on autopsy is not as closely related to clinical symptoms of dementia as previously assumed. It is likely that factors such as years of formal education can modulate the threshold at which dementia will

appear clinically.³² This phenomenon is known as "cognitive reserve".

The assumption that cognitive decline is inevitable in old age has significant implications. Seniors who hold this view actually have lower levels of cognitive performance.⁸ The myth also contributes to a lack of contact with health care professionals when needed, to a lack of initiative to prevent decline and to stigma and a lower quality of life.⁸ Recent evidence indicates that engagement in physical and intellectual activities can help prevent and slow the progression of cognitive impairment.^{33,34} The most significant intervention appears to be aerobic exercise, the mechanism of which has not yet been clearly defined.³³ Intellectual activities are of the most benefit to components of cognitive function related to the specific activity.³⁴ For example, a game of chess requires reasoning and will not necessarily enhance memory capacity, but may enhance reasoning capabilities. The Alzheimer's Society recognizes the importance of these implications and provides educational resources for the general public on their website.³⁵

This myth is part of a larger set of stereotypes regarding aging. Among other things, it contributes to a "hidden curriculum" in medical school and a lack of interest in elderly care among medical students, despite the high need for geriatric specialists.³⁶ Physicians have been shown to demonstrate a fatalistic approach in dealing with cognitive impairment in the elderly.³⁷ It is often assumed, even among physicians, that impairment is inevitable and that a diagnosis of dementia will not alter the course of the disease. Conversely, appropriate management in the early stages of dementia, including the initiation of cholinesterase inhibitors, has been shown to significantly decrease the speed of progression.³⁸ Consequences of these assumptions have led to both under diagnosis and under treatment of dementia, as well as to the under diagnosis of reversible causes of cognitive impairment.³⁹ Patients showing clinical signs of cognitive impairment but who have not received a formal diagnosis are less likely to have been worked up for serious medical causes of impairment.⁴⁰ This points to the importance of careful evaluation and treatment of patients presenting with cognitive impairment.

In conclusion, the assumption that cognitive impairment naturally occurs with age is embedded in our society. Degenerative changes do occur with age, but in healthy seniors these changes are largely compensated with adaptive mechanisms. Cognitive changes include increased effort with active, novel

tasks, while implicit memories and abilities remain intact. As well, semantic and factual knowledge increases with age. Overall, research shows that global cognitive decline is not a part of normal aging and steps can be taken to significantly reduce the likelihood of decline.



This article was inspired by the author's grandmother, Margaret Cusack, who was a source of wisdom for her family to the very end of her long life.


References

1. Irony Design. Clip Art Image: Old Dog No New Tricks. Permission to use image was received from Donna Leger, Irony Design on February 28, 2013.
2. Drag L & Bieliauskas L. Contemporary Review 2009: Cognitive Aging. *J Geriatr Psychiatry Neurol*;2010 23:75.
3. Arthur B. Back Talk; The freedom to rebut wayward sports figures is an inalienable right that must be defended. *National Post (Canada)*. 2010 Dec 9;S1.
4. Ball C. Letter; Abolition of DRA will raise concerns but can be addressed. *Recruiter (UK)*. 2010 Sept 15;Pg.18.
5. Plassman B.L., Langa K.M., Fisher G.G. Prevalence of dementia in the United States: the aging, demographics, and memory study. *Neuroepidemiology*. 2007;29:125-132.
6. Palmore E. The facts on aging quiz: a review of findings. *Gerontologist*. 1980 Dec;20(6): 669-72.
7. Donlon MM, Ashman O, Levy BR. Re-Vision of Older Television Characters: A Stereotype-Awareness Intervention. *Journal of Social Issues*. 2005;61(2): pp 307-19.
8. Sarkisian C, Hays R & Mangione C. Do Older Adults Expect to Age Successfully? The Association Between Expectations Regarding Aging and Beliefs Regarding Healthcare Seeking Among Older Adults. *J Am Geriatr Soc* 50:1837-1843, 2002.
9. Raz N, Lindenberger U, Rodrigue K, et al. Regional brain changes in aging healthy adults: general trends, individual differences and modifiers. *Cereb Cortex*. 2005; 15(11):1679-1689.
10. Haug H. Anatomical changes in the aging brain: morphometric analysis of the human prosencephalon. In Cervos-Nacarro J, Sarkander H. eds., *Brain Aging Neuropathology and Neuropharmacology*. New York: Raven Press; 1983:1-12.
11. Haug H, Eggers R. Morphometry of the human cortex cerebri and corpus striatum during aging. *Neurobiol Aging*. 1991;12(4): 336-338.
12. Raz N, Gunning FM, Head D, et al. Selective aging of the human cerebral cortex observed in vivo: differential vulnerability of the prefrontal gray matter. *Cereb Cortex*. 1997;7(3):268-282.
13. Pfefferbaum A, Sullivan EV. Frontal, parietal, and callosal degradation in aging: a quantitative DTI fiber tracking study. *Neuropsychopharmacology*. 2005;30(S1):S80-S81.
14. Bartzokis G. Age-related myelin breakdown: a developmental model of cognitive decline and Alzheimer's disease. *Neurobiol Aging*. 2004;25(1):5-18.
15. Head D, Buckner RL, Shimony JS, et al. Differential vulnerability of anterior white matter in nondemented aging with minimal acceleration in dementia of the Alzheimer type: evidence from diffusion tensor imaging. *Cereb Cortex*. 2004;14(4):410-423.
16. Gazzaley A, D'Esposito M. Bold functional MRI and cognitive aging. In: Cabeza R, Nyberg L, Park D, eds. *Cognitive Neuroscience of Aging*. Oxford: Oxford University Press; 2005:107-131.
17. Cabeza R, Daselaar S, Dolcos F, Prince SE, Budde M, Nyberg L. Task-independent and task-specific age effects on brain activity during working memory, visual attention, and episodic retrieval. *Cereb Cortex*. 2004;14(4):364-375.
18. Jacoby L, Jennings J, Hay J. Dissociating automatic and consciously - controlled processes: implications for diagnosis and rehabilitation of memory deficits. In: Herrmann D, Johnson M, McEvoy C, Hertzog C, Hertel P, eds. *Basic and Applied Memory Research: Theory in Context*. Mahwah, NJ: Lawrence Erlbaum Associates; 1996:161-193.
19. Parasuraman R, Nestor P. Attention and driving skills in aging and Alzheimer's Disease. *Human Factors*. 1991;33(5):539-557.
20. Verhaeghen P, Cerella J. Aging, executive control, and attention: a review of meta-analyses. *Neurosci Biobehav Rev*. 2002;26(7):849-857.
21. Verhaeghen P, Steitz D, Sliwinski M, Cerella J. Aging and dual-task performance: a meta-analysis. *Psychol Aging*. 2003;18(3):443-460.
22. Schaie K, Willis S. Age difference patterns of psychometric intelligence in adulthood. *Psychol Aging*. 1993;8(1):44-55.
23. Maylor E. Remembering versus knowing television theme tunes in middle-aged and elderly adults. *Br J Psychol*. 1995;86(1):21-25.
24. Dennis N, Hayes S, Prince S, Madden DJ, Huettel SA, Cabeza R. Effects of aging on the neural correlates of successful item and source memory encoding. *J Exp Psychol Learn Mem Cogn*. 2008;34(4):791-808.
25. Henry J, MacLeod M, Phillips L, Crawford J. A meta-analytic review of prospective memory and aging. *Psychol Aging*. 2004;19(1):27-39.
26. Stebbins G, Carillo M, Dorfman J, et al. Aging effects on memory encoding in the frontal lobes. *Psychol Aging*. 2002;17(1):44-55.
27. Kemper S, Sumner A. The structure of verbal abilities in young and older adults. *Psychol Aging*. 2001;16(2):312-322.
28. Singer T, Verhaeghen P, Ghisletta P, Lindenberger U, Baltes PB. The fate of cognition in very old age: six-year longitudinal findings in the Berlin Aging Study (BASE). *Psychol Aging*. 2003;18(2):318-331.
29. Apostolova LG, DeKosky ST, Cummings JL. Chapter 66 - Dementias. In: *Neurology in clinical practice*, 6th ed. Walter G. Bradley, Editor. 2012; pp 1534-1581.
30. Soscia SJ et al. The Alzheimer's disease-associated amyloid beta-protein is an antimicrobial peptide. *PLoSOne*. 2010; 5(3): p.e9505.
31. Pritchard SM et al. The toxicity of Tau in Alzheimer Disease: Turnover, targets and potential therapeutics. *J Cell Mol Med*, 2011.
32. Stern Y. Cognitive reserve in ageing and Alzheimer's disease. *Lancet Neurol* 2012; 11: 1006-12.
33. Hamer M, Chida Y. Physical activity and risk of neurodegenerative disease: a systematic review of prospective evidence. *Psychol Med*. 2009;39(1):3-11.
34. Kemoun G, Thibaud M, Roumagne N, Carrette P, Albinet C, Toussaint L, et al. Effects of a physical training programme on cognitive function and walking efficiency in elderly persons with dementia. *Dement Geriatr Cogn Disord*. 2010;29(2):109-114.
35. Myths and Reality about Alzheimer's Disease. Alzheimer's Society of Nova Scotia, 2013. Webpage on the internet available at: <http://www.alzheimer.ca/en/ns/About-dementia/Alzheimer-s-disease/>

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- Myth-and-reality-about-Alzheimer-s-disease#DynamicId_3
36. Higashi R, Tillack AA, Steinman M, Harper M, Johnston CB. Elder care as “frustrating” and “boring”: Understanding the persistence of negative attitudes toward older patients among physicians-in-training. *Journal of Aging Studies* 26 (2012) 476–483.
 37. Boise L, Camicioli R, Morgan D, et al. Diagnosing dementia: perspectives of primary care physicians. *Gerst.* 1999;39:457–464.
 38. Trinh NH, Hoblyn J, Mohanty S, Yaffe K. Efficacy of cholinesterase inhibitors in the treatment of neuropsychiatric symptoms and functional impairment in Alzheimer disease: a meta-analysis. *JAMA.* 2003;289(2):210.
 39. Boise L, Neal MB and Kaye J. Dementia Assessment in Primary Care: Results From a Study in Three Managed Care Systems. *Journal of Gerontology*; 2004; Vol. 59A, No. 6, pp 621–626.
 40. Callahan CM, Hendrie HC, Tierney WM. Documentation and evaluation of cognitive impairment in elderly primary care patients. *Ann Intern Med.* 1995;122:422–429

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