ACADEMIA | Letters

# *The Social Pathology of Dementia*

Mario Garrett

---

When Richard Katzman combined senile ("old age") dementia with Alzheimer's disease in 1976[1], he gained a larger constituency to promote the mission of the nascent United States National Institute on Aging (NIA). But this confabulation resulted in Alzheimer's disease research becoming confused and lacking a theoretical rudder[2]. The resulting confusion led the NIA to start promoting the diagnoses of Alzheimer's disease based on biological markers and not on clinical diagnosis[3],[4].

However, evidence contradicts the NIA's aspiration of diagnosing Alzheimer's disease purely on biological markers. These biological markers—colloquially referred to as plaques and tangles[5]—are not consistently correlated to Alzheimer's disease or dementia. Half of clinically diagnosed oldest-old with dementia have insufficient neuropathology to account for their dementia[6],[7] while thirty to fifty percent of older adults without dementia meet the neu-

[1]Katzman, R. (1976). The prevalence and malignancy of Alzheimer disease: a major killer. Archives of neurology, 33(4), 217-218.

[2]Garrett, M. D., & Valle, R. (2016) A Century of Confusion in Researching Alzheimer's Disease. Dementia: The International Journal of Healthcare 2(2), 13-22.

[3]Jack Jr, C. R., Albert, M. S., Knopman, D. S., McKhann, G. M., Sperling, R. A., Carrillo, M. C.,…& Phelps, C. H. (2011). Introduction to the recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's & dementia, 7(3), 257-262.

[4]Jack Jr, C. R., Bennett, D. A., Blennow, K., Carrillo, M. C., Dunn, B., Haeberlein, S. B.,…& Liu, E. (2018). NIA-AA research framework: toward a biological definition of Alzheimer's disease. Alzheimer's & Dementia, 14(4), 535-562.

[5]Selkoe, D. J. (2001). Alzheimer's disease: genes, proteins, and therapy. *Physiological reviews*.

[6]Crystal, H. A., Dickson, D., Davies, P., Masur D., Grober, E., Lipton, R.B. (2000). The relative frequency of "dementia of unknown etiology" increases with age and is nearly 50 percent in nonagenarians. Arch Neurol, 57(5):713-719.

[7]Polvikoski T., Sulkava R., Myllykangas L., Notkola I.L., Niinisto L., Verkkoniemi A., et al (2001). Prevalence of Alzheimer's disease in very elderly people: a prospective neuropathological study. Neurology, 56(12):1690-

 

**Corresponding Author:** Mario Garrett, mgarrett@mail.sdsu.edu
**Citation:** Garrett, M. (2021). The Social Pathology of Dementia. *Academia Letters*, Article 271.
https://doi.org/10.20935/AL271.

ropathological criteria for Alzheimer's disease[8],[9],[10],[11],[12],[13],[14]. Among older people, the correlation between Alzheimer's disease neuropathology and its clinical expression declines[15]. It is disturbing that we have known this fallacy for more than a century[16]. We therefore need to think more creatively if we want to be in a different place a hundred years from now.

A recent review identified close to a hundred documented causes of dementia[17]. These causes are so broad that some researchers suggest that dementia might be a biological strategy[18]. Because the brain is incredibly complex, it attracts, isolates, and insulates biological attacks. The biological theory suggests that this short-term strategy leads to long-term liability for dementia. But even these broad biological causes do not explain all the cases of dementia. Another process must be influencing its development.

Surprisingly, one of the avenues that might explain this process is psychology. By looking at what happens with memory at older age. Following a decrease in learning in older age, this affects memory and starts a cascade of negative events. Older adults, because of their ample experience, have developed a complex and reliant model of their environment—an isomorphic

1696.

[8]Katzman R., Terry R.D., DeTeresa R., Brown T., Davies P., Fuld P., et al. (1988). Clinical, pathological, and neurochemical changes in dementia: a subgroup with preserved mental status and numerous neocortical plaques. Ann Neurol, 23(2): 138-144.

[9]Tomlinson B.E., Blessed G. & Roth, M. (1970). Observations on the brains of demented old people. Journal of the neurological sciences, 11(3): 205-242.

[10]Dickson, D. W. (1997). Neuropathological diagnosis of Alzheimer's disease: a perspective from longitudinal clinicopathological studies. Neurobiology of aging,18(4): S21-S26.

[11]Snowdon, D. A., Greiner, L. H., Mortimer, J. A., Riley, K. P., Greiner, P. A., & Markesbery, W. R. (1997). Brain infarction and the clinical expression of Alzheimer disease: the Nun Study. Jama, 277(10), 813-817.

[12]Knopman, D. (2003). Pharmacotherapy for Alzheimer's disease: 2002. Clinical neuropharmacology, 26(2), 93-101.

[13]Price, J. L., & Morris, J. C. (1999). Tangles and plaques in nondemented aging and "preclinical" Alzheimer's disease. Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society, 45(3), 358-368.

[14]Davis, D. G., Schmitt, F. A., Wekstein, D. R., & Markesbery, W. R. (1999). Alzheimer neuropathologic alterations in aged cognitively normal subjects. Journal of neuropathology and experimental neurology, 58(4), 376-388.

[15]Savva, G. M., Wharton, S. B., Ince, P. G., Forster, G., Matthews, F. E., & Brayne, C. (2009). Age, neuropathology, and dementia. New England Journal of Medicine, 360(22), 2302-2309.

[16]Garrett, M. D. (2015). Politics of Anguish: How Alzheimer's disease became the malady of the 21st century. Kindle. USA

[17]Garrett, M. D. (2020). Multiple Causes of Dementia as Engineered Senescence. European Journal of Medical and Health Sciences, Vol2 (2).

[18]Goldgaber, D., Schwarzman, A. I., Bhasin, R., Gregori, L., Schmechel, D., Saunders, A. M.,…& Strittmatter, W. J. (1993). Sequestration of Amyloid β-Peptide a. Annals of the New York Academy of Sciences, 695(1), 139-143.

 

**Corresponding Author:** Mario Garrett, mgarrett@mail.sdsu.edu
**Citation:** Garrett, M. (2021). The Social Pathology of Dementia. *Academia Letters*, Article 271.
https://doi.org/10.20935/AL271.

representation[19]. When it comes to problem solving, older adults are more likely to rely on this constancy in favor of looking at the current environment. Their isomorphic representation becomes self-reliant, needing little input from outside. Older adults become so efficient in their regular activities (e.g., reading, driving, communicating, talking, and anticipating events) that any divergence away from this routine becomes difficult. It seems that for most older adults, both age and brain development bring about more control and inhibition, especially as found with eidetic imagery[20], [21],[22]. Finding that most of the answers reside in our experiences, the brain restricts learning from the environment and retrieves the information from our past. Although this system is very efficient, it eventually subverts the role of memory. Memory's role is not just to recall past events, it is to recall past event so that we might use them as a lesson for the present, in order to better anticipate our future. We should ask not what memory is, but what memory is for[23].

The brain, which in earlier times looked for answers from the outside, slowly becomes introspective as increasingly our internal representation supplies the information we need. The self-reliance on the isomorphic representation ensures that the outside world becomes increasingly ignored and discounted. As we reduce learning, we stop using memory as it was intended for, and it becomes increasingly redundant. This is the social pathology of dementia.

As a result, we start noticing faults with short-term memory recall—the first stage of memory. We attempt to rationalize these memory lapses and we find a ready-made stereotype for what we are experiencing: dementia. Then we substantiate it through "confirmatory bias" and "inattentive/change blindness" even when there is no biological evidence[24]. These perceptual biases (there are over 134 of them[25]) ensure that we reinforce our self-description by discounting evidence that negate this judgement and highlighting evidence that support it. Once we make these judgements it is difficult, if not impossible, to reverse—even if there is evidence to the contrary[26].

[19]Dror, I. E., Schmitz-Williams, I. C., & Smith, W. (2005). Older adults use mental representations that reduce cognitive load: mental rotation utilizes holistic representations and processing. Experimental aging research, 31(4), 409-420.

[20]Giray, E. F., Altkin, W. M., Vaught, G. M., & Roodin, P. A. (1976). The incidence of eidetic imagery as a function of age. Child development, 1207-1210.

[21]Gray, C. R., & Gummerman, K. (1975). The enigmatic eidetic image: a critical examination of methods, data, and theories. Psychological bulletin, 82(3), 383.

[22]Siipola, E. M., & Hayden, S. D. (1965). Exploring eidetic imagery among the retarded. Perceptual and Motor Skills, 21(1), 275-286.

[23]Glenberg, A. M. (1997). What memory is for. Behavioral and brain sciences, 20(1), 1-19.

[24]Merckelbach, H., Dalsklev, M., van Helvoort, D., Boskovic, I., & Otgaar, H. (2018). Symptom self-reports are susceptible to misinformation. Psychology of Consciousness: Theory, Research, and Practice, 5(4), 384.

[25]Garrett, M. D. (2021) Social Dementia: The role of memory. Kindle, USA.

[26]Merckelbach, H., Jelicic, M., & Jonker, C. (2012). Planting a misdiagnosis of Alzheimer's disease in a

 

**Corresponding Author:** Mario Garrett, mgarrett@mail.sdsu.edu
**Citation:** Garrett, M. (2021). The Social Pathology of Dementia. *Academia Letters*, Article 271.
https://doi.org/10.20935/AL271.

Older people who report subjective cognitive decline are four times more likely to progress to dementia, and then these people are twice as likely to develop dementia when others substantiate their memory loss[27]. Our social world reinforces these negative expectations. One unexpected observation is that subjective memory decline only predicts dementia if this self-evaluation causes concern[28]. A "consensus trance" further promotes the self-evaluation, when other people—through stereotypes, expectations, and interactions—confirm these negative expectations[29]. The social pathology of dementia can play a key role in tipping the scales from memory lapses to dementia. This explains why longer-lived people free from dementia live in communities of older people (Blue Zones), where such negative stereotypes do not exist[30]. Many other perceptual biases may appear that then make this subjective negative evaluation come true. We become psychologically apathetic, not concerned so much with the world around us, as with focusing on the immediacy of our situation. We can see all these developments because apathy and depression correlate closely with dementia[31],[32] and they are indicative of a perceptual change[33]. These emotions suggests that a radical perceptual change has occurred, a change that modifies how we see the world.

Like a house of cards, the edifice of social learning slowly unhinges and then dismantles when we rely on established patterns of behavior, while disregarding novelty. With the loss of memory, we lose retrieval of memories that shape self-consciousness, self-knowledge, and self-image[34]. Our lack of access to these memories not only leads to a diminished sense of self and identity, but also slows down acquisition of new knowledge, in general, as this loss of

person's mind. Acta neuropsychiatrica, 24(1), 60-62.

[27]Koppara, A., Wagner, M., Lange, C., Ernst, A., Wiese, B., König, H. H.,…& Werle, J. (2015). Cognitive performance before and after the onset of subjective cognitive decline in old age. Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring, 1(2), 194-205.

[28]Mendonça, M. D., Alves, L., & Bugalho, P. (2016). From subjective cognitive complaints to dementia: who is at risk?: a systematic review. American Journal of Alzheimer's Disease & Other Dementias®, 31(2), 105-114.

[29]Brigham, D. D., & Toal, P. O. (1990). The Use of Imagery in a Multimodal Psychoneuroimmunology Program for Cancer and Other Chronic Diseases. In Mental Imagery (pp. 193-198). Springer, Boston, MA.

[30]Buettner, D. (2012). The blue zones: 9 lessons for living longer from the people who've lived the longest. National Geographic Books.

[31]Bock, M. A., Bahorik, A., Brenowitz, W. D., & Yaffe, K. (2020). Apathy and risk of probable incident dementia among community-dwelling older adults. Neurology.

[32]Ownby, R. L., Crocco, E., Acevedo, A., John, V., & Loewenstein, D. (2006). Depression and risk for Alzheimer disease: Systematic review, meta-analysis, and metaregression analysis. Archives of General Psychiatry,63, 530–538.

[33]Mowla, A., Ashkani, H., Ghanizadeh, A., Dehbozorgi, G. R., Sabayan, B., & Chohedri, A. H. (2008). Do memory complaints represent impaired memory performance in patients with major depressive disorder?. Depression and anxiety, 25(10), E92-E96.

[34]El Haj, M., Antoine, P., Nandrino, J. L., & Kapogiannis, D. (2015). Autobiographical memory decline in Alzheimer's disease, a theoretical and clinical overview. Ageing research reviews, 23, 183-192.

 

**Corresponding Author:** Mario Garrett, mgarrett@mail.sdsu.edu
**Citation:** Garrett, M. (2021). The Social Pathology of Dementia. *Academia Letters*, Article 271.
https://doi.org/10.20935/AL271.

self-importance reduces our motivation to learn[35]. Apathy, shadowed by depression, further reduces the motivation to learn. Activities that involve novelty-seeking—such as sharing, exchanging of ideas, and being social, the bedrock for challenging dementia[36]—are shunned. In the same way that placebos change our biology and outcomes, nocebos and negative perceptual biases similarly bring about negative physical and biological changes[37]. Social dementia translates to biological changes[38]. Like false memories[39], social dementia follows a set pattern of validation. Dementia is promoted by how we feel, our emotions, in this case apathy, depression and the lack of interest in learning about the world around us. The social pathology of dementia is an important part of the puzzle in understanding the causes of dementia.

[35]Amanzio, M., Geminiani, G., Leotta, D., & Cappa, S. (2008). Metaphor comprehension in Alzheimer's disease: Novelty matters. Brain and language, 107(1), 1-10.

[36]Stern, C., & Munn, Z. (2010). Cognitive leisure activities and their role in preventing dementia: a systematic review. International Journal of Evidence-Based Healthcare, 8(1), 2-17.

[37]Enck P, Benedetti F, Schedlowski M (2008). New insights into the placebo and nocebo responses. Neuron 59: 195–206

[38]Benedetti, F., & Shaibani, A. (2018). Nocebo effects: more investigation is needed.

[39]Loftus, E. (2020). The fiction of memory.

 

**Corresponding Author:** Mario Garrett, mgarrett@mail.sdsu.edu
**Citation:** Garrett, M. (2021). The Social Pathology of Dementia. *Academia Letters*, Article 271.
https://doi.org/10.20935/AL271.