

Distinguishing Cognitive Decline Due to Normal Aging from Alzheimer’s Disease in Older Adults: A Systematic Review

P1-001

BRUNO ONYINYE UMUNAKWE

Amyloid positive (Aβ+) and Amyloid negative (Aβ–) are both linked to different processes and measurements related to Alzheimer’s disease

INTRODUCTION	METHODS	RESULTS	CONCLUSIONS	REFERENCES
- Alzheimer’s disease (AD) is linked to the early buildup of amyloid-beta (Aβ) in the brain before cognitive problems appear. - Clinical evidence shows that amyloid-positive individuals exhibit a significantly faster rate of decline in episodic memory than amyloid-negative individuals. - Deficits in associative memory and delayed recall are deeply linked to amyloid deposition, distinguishing them from the more gradual changes seen in normal aging.	- Qualitative methodology was used to obtain information from relevant literature on the meaning and roles of amyloid in memory loss. - Data were analysed using systematic review to explain the prevalence and outcomes of amyloid-positive and amyloid-negative in relation to memory loss.	- Alzheimer’s disease starts when abnormal proteins build up in the brain, forming amyloid plaques and neurofibrillary tangles. - Though amyloid-beta (Aβ) normally helps with brain function, too much buildup of it shows an early sign of Alzheimer’s disease (AD). - The change from amyloid-beta negative (Aβ–) to amyloid-beta positive (Aβ+) shows the progression of the AD, but the factors that predict this change are still not clinically clear.. - Aβ+ is linked to a faster rate of brain tissue loss (atrophy) when compared to Aβ-. - Clinical Studies show that among individuals without dementia, amyloid positivity is more prevalent with an increased age and is more frequent in women than in men.	- Individuals of Aβ+ and Aβ– show memory decline, but those with amyloid-positive experience a faster and more severe decline, especially in associative memory and delayed recall. - Addressing memory loss requires a multimodal approach that will incorporate sensitive cognitive assays, structural MRI, and core AD biomarkers for accurate differentiation in refining prognostic accuracy, enable timely intervention, and ensure the appropriate allocation of individuals into clinical trials and preventative care pathways.	- Lahiri, D., Roncero, C.T., Lima, B.S., Verhoeff, N.P.L.G., Freedman, M., Al-Shamaa, S. and Chertkow, H. (2023), A comparative study of Amyloid positive and negative individuals with “Alzheimer Syndrome”. <i>Alzheimer's Dement.</i>, 19: e075037. https://doi.org/10.1002/alz.075037. - Chiotis, K., Yingbing, W., La Joie, R. & Rabinovici, G.D. (2025). The Role of Amyloid-b and Tau PET in the New Era of Alzheimer Disease Therapies Konstantinos. <i>Journal of Nuclear Medicine</i>, jnumed.124.268339; DOI: https://doi.org/10.2967/jnumed.124.268339. - Vahid, A., Zamani, J. & Hadi Hosseini, S. Predicting future amyloid conversion: the role of baseline amyloid distribution, genetic and cognitive resilience. <i>npj Dement.</i> 1, 40 (2025). https://doi.org/10.1038/s44400-025-00041-z. CONTACT  umunnabruno@gmail.com 