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Review

Alzheimers Disease (Senile Dementia)



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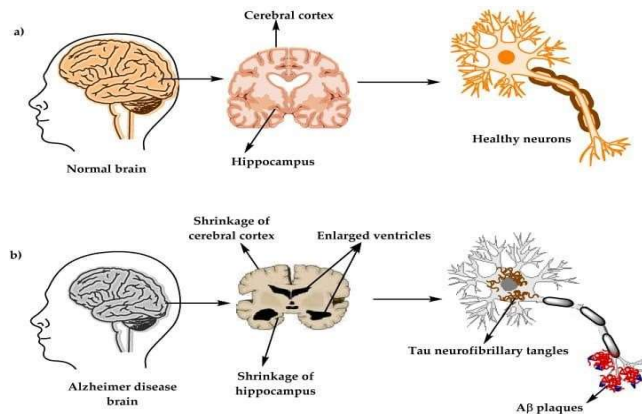
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	Abstract
Published on: 14 Jul 2025	<p>The term Alzheimer's disease was first used by Emil Kraepelin in 1910 to differentiate between presenile dementia and senile dementia. Alzheimer's disease (AD) is a brain disorder that ultimately results in loss of independence and impaired cognitive function due to degeneration of brain cell. Although multifaceted in nature, AD is commonly categorized by cholinergic and amyloid theories. Several risk factors for the development of AD have been identified, and include but are not limited to age, genetics, prior head injuries, vascular disease, infections, and environment. At present, there are only two approved drugs for the treatment of AD, the cholinesterase enzyme inhibitors and N-methyl d-aspartate (NMDA) antagonists. Research grants focused on understanding the pathology of AD, which targets the mechanisms such as the abnormal metabolism of tau protein, increased levels of β-amyloid, inflammation, and the cholinergic and free radical damage associated with each. Future hypotheses which could lead to new therapies include DMT (disease-modifying therapeutic), chaperones, and other natural compounds.</p>
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INTRODUCTION

Alzheimer's disease (AD) (named after the German psychiatric Alois Alzheimer) is the most common type of dementia and can be defined as a slowly progressive neurodegenerative disease characterized by neuritis plaques and neurofibrillary tangles (Figure 1) as a result of amyloid-beta peptide's ($A\beta$) accumulation in the most affected area of the brain, the medial temporal lobe and neocortical structures [1]. Alois Alzheimer noticed a presence of amyloid plaques and a massive loss of neurons while examining the brain of his first patient that suffered from memory loss and change of personality before dying and described the condition as a serious disease of the cerebral cortex. Emil Kraepelin named this medical condition Alzheimer's disease for the first time in his 8th edition psychiatry handbook [2]. Progressive loss of cognitive functions can be caused by cerebral disorder like Alzheimer's disease (AD) or other factors such as intoxications, infections, abnormality in the pulmonary and circulatory systems, which causes a reduction in the oxygen supply to the brain, nutritional deficiency, vitamin

B12 deficiency, tumors, and others [3].



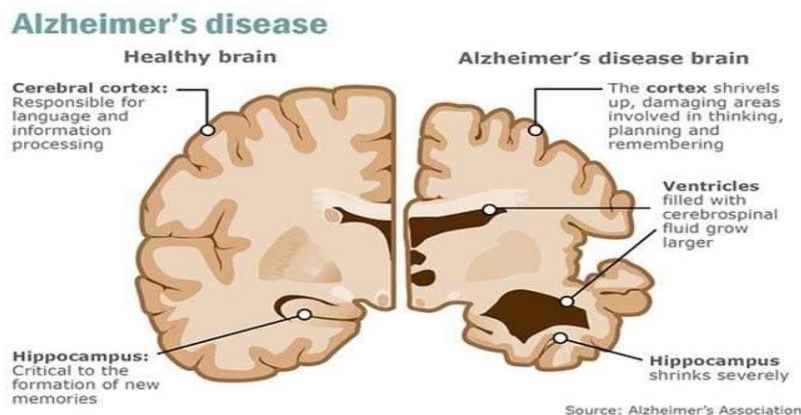
The physiological structure of the brain and neurons in (a) healthy brain and (b) Alzheimer's disease (AD) brain.

History

Ancient Greek and Roman philosophers and medicine linked old age to dementia. German psychiatrist Alois Alzheimer described the first case of Alzheimer's disease (which was later named after him) in 1901 in a 50-year-old woman called Auguste D. The disease would first be made public in 1906 [4]. Development of knowledge of the disease progressed in the next five years with a total of eleven more similar cases documented in the medical literature, some referring to the disease as Alzheimer's disease [5]. Emil Kraepelin initially described Alzheimer's disease as its own disease subtype, obscuring the clinical and pathological features in regard Auguste D. Kraepelin first included Alzheimer's disease as a subtype of senile dementia, in the 1910 Textbook of Psychiatry [6].

What is alzheimers

Short-term memory loss is a hallmark of Alzheimer disease; a kind of dementia that develops gradually and usually strikes people in their middle years or later. y, slowness of cognition and decline in conduct [7].



It is most common type of dementia .50-70% cases of dementia are diagnosed as Alzheimer's Disease.

Types of Alzheimer's Disease

Alzheimer's Classification are of three types:

Mild Alzheimer's

- Significant cognitive impairment limiting the patient's ability to recall some daily tasks and responsibilities.
- Patients will be functional, but they may experience some problems.
- Patients will require longer to complete tasks than previously [8].

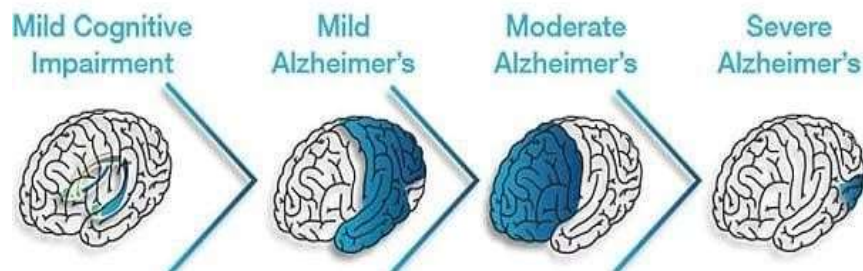
Moderate Alzheimer's

- More severe symptoms assumed the most intense symptoms are attributable to additional neuronal damage.
- Confusion and memory loss will be increased in intensity and duration.

- Patient will increase dependence on others [9].
- Patient may be physically able to move but unable to perform their regular responsibilities due to delusions.

Severe Alzheimer's

As the plaques and tangles spread, the brain cells start dying. This results in shrinkage of brain tissue [10]. The patients with this condition are typically bedridden and are hardly able to communicate.



The brain cells begin to die as the plaques and tangles grow. The brain tissue shrinks as a result. Patients suffering with this illness are usually immobile and have limited communication skills.

Alzheimer's Disease Subtypes and Classification

Inflammatory Response

Alzheimer's disease is classified into three subtypes based on inflammatory response:

Inflammatory: with symptoms of behavioural and cognitive changes, high serum albumin to globulin ratio, and high C-reactive protein [11].

non-inflammatory: with no elevated inflammatory biomarkers, but often with metabolic abnormalities.

Cortical: caused by a zinc deficiency in brain regions, causing abnormal brain functioning abnormalities [12].

Onset or Trigger Type

- Early-Onset Alzheimer's: a rare subtype affecting people below 65 years old, affects 5 out of 100 Alzheimer's patients.
- Changes typically occur when patients reach the age of 65.

Alzheimer's disease is chronic and progressively debilitating for 65+ populations; the late-onset variety dominates [13]. The genetic trigger of Alzheimer's disease is undetermined, but there are many risk factors identified. A small irregular category is familial Alzheimer's disease (FAD), similarly classified as Alzheimer's disease and diagnosed only by the particular genotypic pattern of illness in family members. Early identification is much more important for treatment and prevention, to either prevent or slow down the "progressing" disease; the first symptom that occurs is memory loss [14]. As a society, we do not tend to observe memory loss for granted as a symptom of dementia, but instead, associate it with normal aging processes; we acknowledge memory loss only in retrospect, where memory relied on a prior level of cognition, that continues to decline, and events are frankly misinterpreted or mis-remembered. The process is insidious, so that one first experiences mild memory loss, and then struggles physically to retrieve memory and words, while knowing there is an intention of speaking [15]. Clearly, it is only when memory loss begins to interact with significant social and work activity, or someone else observes changes stating dementia is occurring, there is simply suspicion. People experience emotional changes, and major depression occurs in 24-32% of cases, anxiety occurs in 17-27% of cases, apathy can occur in up to 41%, and delusions in about 23%.

Symptoms

Alzheimer's Disease Signs:

Memory loss: Forgetting where things are located, forgetting what you've learned moments/ days ago, forgetting where you are going to.

Language issues: Difficulty in following conversations; repeating questions; remembering common words.

Getting lost/disoriented: Unknown place/unknown time.

Mood variations: Becoming anxious, irritable, depressed or withdrawn.

Difficulty performing tasks: Tasks like planning- determining how to potentially solve a problem, bring it to conclusion.

Personality changes: Loss of interest in activities / hobbies, or loss of initiative.

10 warning signs of Alzheimer

Cognition and memory loss in aging: a symptom of dementia

- Memory loss is a symptom of dementia, which is a progressive reduction in memory, thinking, and reasoning skills [16].
- Alzheimer's disease is the most common dementia diagnosis.
- The Alzheimer's Association has developed a checklist of common signs:
 - o memory loss
 - o difficulty with familiar tasks
 - o problems with language
 - o disorientation to time and place
 - o poor or decreased judgment
 - o abstraction problems
 - o misplacing items
 - o mood changes
 - o personality changes
 - o loss of initiative.

Risk factors

Alzheimer's Risk Factors

Age: Increased risk of Alzheimer's increases with age, especially after 65 and 85.

Family History and Genetics: Certain genes like the APOE-e4 gene can increase the risk [17].

Gender: Women are more likely to develop Alzheimer's than men, possibly due to hormonal differences or life expectancy differences.

Down Syndrome: Individuals with Down syndrome are at higher risk due to the extra copy of chromosome 21, which contains the gene that produces amyloid precursor protein (APP) [18].

Head Injuries: History of significant head trauma or repeated concussions can increase the risk.

Heart Health: Conditions like high blood pressure, high cholesterol, diabetes, and obesity can damage blood vessels and reduce blood flow to the brain [19].

Education and Cognitive Engagement: Lower levels of education and lack of mentally stimulating activities may increase the risk.

Social and Physical Activity: Regular physical exercise and active social life can protect brain health [20].

Sleep Disorders: Sleep problems, including sleep apnea, can interfere with brain functions and increase amyloid plaque buildup.

Depression: Evidence suggests a link between depression and Alzheimer's risk.

Diagnosis

The process of diagnosing Alzheimer's disease:

Medical history and symptoms: This includes taking a medical history, such as when the patient noticed symptoms like memory loss, confusion, difficulty completing daily tasks [21].

Physical and neurological exam: A physical exam is done to screen out possible causes [22].

Cognitive and neuropsychological tests: Assesses memory, problem-solving, attention, and language.

Brain imaging: MRI or CT scans are taken to look for brain shrinkage and amyloid plaques [23].

Blood tests: Blood tests are done to rule out other causes of similar symptoms.

Genetic tests: Genetic tests look for gene mutations.

Formal diagnosis: A formal diagnosis is made when all causes are ruled out and clinical criteria are met [24].

Treatment

Overview of Alzheimer's Disease Treatments

Medications:

Cholinesterase inhibitors (ACE inhibitors): These medications activate and prolong cell-to-cell communication and are effective in maintaining a chemical messenger, which is reduced in the brain, by Alzheimer's disease and is the most common first therapeutic trials. Most show some modest improvement in symptoms [25].

Memantine (Namenda): This medication slows decline in symptoms with moderate to severe Alzheimer's disease, and can sometimes be paired with a cholinesterase inhibitor [26].

Newer Medications:

Lecanemab-irmb (Leqembi): This is administered as an IV infusion every two weeks, with side effects of fever, flu-like symptoms, nausea, vomiting, dizziness, change in heart rate, and respiratory issues [27].

Donanemab-azbt (Kisunla): This is administered as an IV infusion every four weeks, with side effects of flu-like symptoms, nausea, vomiting, headache, respiratory issues, and change in blood pressure [28].

- Side effects may also include brain swelling or small brain bleeds, with the FDA recommending an MRI of

the patient's brain prior to starting treatment and follow-up MRIs during treatment.

- Patients with a specific form of the APOE e4 gene may be at risk for having these serious side effects [29].
- Blood-thinning medications may amplify the risk of a brain bleed. There is insufficient information about the risks of taking lecanemab and donanemab, and how effective they will be for individuals at risk of having Alzheimer's disease.

Alternative Medicine

Vitamin E: Based on evidence, 2,000 international units (IU) daily may be used to delay worsening of symptoms in people with mild to moderate disease. Valid research is needed to confirm safety for long-term use in people with dementia (daily dose=2,000 IU) [30].

Omega-3 fatty acids: May reduce the risk of dementia, clinical trials have shown no benefits for the treatment of symptoms associated with Alzheimer's disease [31].

Curcumin: Herb derived from turmeric with anti-inflammatory and anti-oxidant properties; clinical trials have demonstrated no benefit for treatment of Alzheimer's disease [32].

Ginkgo: Plant extract demonstrated no effect on the prevention of Alzheimer disease or delay its symptoms [33].

Melatonin: supplement that helps with sleep for individuals, some studies have found it may worsen mood for some individuals with dementia [34].

CONCLUSION

Alzheimer's disease (AD) is a brain disorder that causes dementia and the inability to maintain independence, due to the degenerative process of brain cells associated in the disease. Two basic types of theories (cholinergic and amyloid) have been derived from the disease: there are neuritis plaques and neurofibrillary tangles. The disease is classified into 3 categories, mild, moderate, and severe. Signs and symptoms of AD include impairment in memory; impairment with language; changes in mood; difficulty with simple tasks; and personality disorders. AD will affect 80% of the population, with an increased risk associated with age (risk 1 in 14 with over 70 years of age), female gender, family history, genetics, head trauma or injury of a serious nature, cardiovascular health, education, level of cognitive activity, level of social and physical activity, level of sleep activity, and depression. To make a diagnosis of AD a physician needs to take a comprehensive medical history, perform a physical and neurological examination, order cognitive and neuropsychological examination, order brain imaging and blood tests. First line medications include cholinesterase inhibitors, memantine; alternatives are Vitamin E, omega-3 fatty acids, curcumin, ginkgo, and melatonin.

REFERENCES

1. Lane, Christopher A., John Hardy, and Jonathan M. Schott. "Alzheimer's disease." *European journal of neurology* 25.1 (2018): 59-70. <https://onlinelibrary.wiley.com/doi/full/10.1111/ene.13439>
2. Masters, Colin L., et al. "Alzheimer's disease." *Nature reviews disease primers* 1.1 (2015): 1-18. <https://www.nature.com/articles/nrdp201556>
3. Scheltens, Philip, et al. "Alzheimer's disease." *The Lancet* 388.10043 (2016): 505-517.
4. Tanzi, Rudolph E. "A brief history of Alzheimer's disease gene discovery." *Journal of Alzheimer's disease* 33.s1 (2012): S5-S13. <https://journals.sagepub.com/doi/abs/10.3233/JAD-2012-129044>
5. Yang, Hyun Duk, Sang Bong Lee, and Linn Derg Young. "History of Alzheimer's disease." *Dementia and neurocognitive disorders* 15.4 (2016): 115-121. <https://synapse.koreamed.org/articles/1120231>
6. Reichman, William E., and Nathan S. Rose. "History and experience: the direction of Alzheimer's disease." *Menopause* 19.7 (2012): 724-734.
7. Richards, Marcus, and Carol Brayne. "What do we mean by Alzheimer's disease?" *BMJ* 341 (2010). <https://www.bmj.com/content/341/bmj.c4670.full>
8. Braak, Heiko, et al. "Vulnerability of select neuronal types to Alzheimer's disease." *Annals of the New York Academy of Sciences* 924.1 (2000): 53-61. <https://nyaspubs.onlinelibrary.wiley.com/doi/abs/10.1111/j.1749-6632.2000.tb05560.x>
9. Griffin, W. Sue T., et al. "Interleukin-1 expression in different plaque types in Alzheimer's disease: significance in plaque evolution." *Journal of Neuropathology & Experimental Neurology* 54.2 (1995): 276-281. <https://academic.oup.com/jnen/article-abstract/54/2/276/2610392?login=false>
10. Rossor, M. N., et al. "Neurochemical characteristics of early and late onset types of Alzheimer's disease." *Br Med J (Clin Res Ed)* 288.6422 (1984): 961-964. <https://www.bmj.com/content/288/6422/961.abstract>
11. Ferreira, Daniel, Agneta Nordberg, and Eric Westman. "Biological subtypes of Alzheimer disease: A systematic review and meta-analysis." *Neurology* 94.10 (2020): 436-448. <https://www.neurology.org/doi/full/10.1212/WNL.0000000000009058>

12. Jellinger, Kurt A. "Pathobiological subtypes of Alzheimer disease." *Dementia and Geriatric Cognitive Disorders* 49.4 (2021): 321-333. <https://karger.com/dem/article/49/4/321/103517/Pathobiological-Subtypes-of-Alzheimer-Disease>
13. Alismail, Sarah, Hind Bitar, and Swati Ramani. "How to Motivate Adults to Check for Alzheimer's Disease (AD) Using Three Types of Triggers." (2017).
14. Medeiros, Rodrigo, Meredith A. Chabrier, and Frank M. LaFerla. "Elucidating the triggers, progression, and effects of Alzheimer's disease." *Journal of Alzheimer's disease* 33.s1 (2012): S195-S210. <https://journals.sagepub.com/doi/abs/10.3233/JAD-2012-129009>
15. Erdoğan, Şükran. "Trigger Factors and Effects of Alzheimer's Disease." *Europeanatolia Health Sciences Journal* 2.1 (2024): 01-03. <https://europeanatolia.com/index.php/pub/article/view/25>
16. Benjamin, Anoop I., et al. "Identifying Alzheimer's disease through the "10 Warning Signs" approach: a useful tool for population-based screening in the elderly." *Asian J Med Res* 192 (2012): 40-4.
17. Parker, K., & Rhee, Y. (2020). Alzheimer's Disease Warning Signs: Gender and Education Influence Modifiable Risk Factors. A Pilot Survey Study. *Journal of the American College of Nutrition*, 40(7), 583–588. <https://doi.org/10.1080/07315724.2020.1812451>
18. Armstrong, Richard A. "Risk factors for Alzheimer's disease." *Folia neuropathologica* 57.2 (2019): 87-105. <https://www.termedia.pl/Risk-factors-for-Alzheimer-s-disease,20,36928,0,1.html>
19. Povova, Jana, et al. "Epidemiological of and risk factors for Alzheimer's disease: a review." *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub* 156.2 (2012): 108-114. <https://pdfs.semanticscholar.org/1830/50a9a5e5090a34405293d1f6862ae48856dd.pdf>
20. Lindsay, Joan, et al. "Risk factors for Alzheimer's disease: a prospective analysis from the Canadian Study of Health and Aging." *American journal of epidemiology* 156.5 (2002): 445-453. <https://academic.oup.com/aje/article-abstract/156/5/445/157983?login=false>
21. Dubois, Bruno, et al. "Clinical diagnosis of Alzheimer's disease: recommendations of the International Working Group." *The Lancet Neurology* 20.6 (2021): 484-496. [https://www.thelancet.com/journals/laneur/article/PIIS1474-4422\(21\)00066-1/abstract](https://www.thelancet.com/journals/laneur/article/PIIS1474-4422(21)00066-1/abstract)
22. Khachaturian, Zaven S. "Diagnosis of Alzheimer's disease." *Archives of neurology* 42.11 (1985): 1097-1105. <https://jamanetwork.com/journals/jamaneurology/article-abstract/584730>
23. Khachaturian, Zaven S. "Diagnosis of Alzheimer's disease." *Archives of neurology* 42.11 (1985): 1097-1105. <https://jamanetwork.com/journals/jamaneurology/article-abstract/586020>
24. Geldmacher, David S., and Peter J. Whitehouse Jr. "Differential diagnosis of Alzheimer's disease." *Neurology* 48.5_suppl_6 (1997): 2S-9S. https://www.neurology.org/doi/abs/10.1212/WNL.48.5_Suppl_6.2S
25. Kumar A, Sidhu J, Lui F, et al. Alzheimer Disease. [Updated 2024 Feb 12]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK499922/>
26. Matsunaga, Shinji, et al. "The efficacy and safety of memantine for the treatment of Alzheimer's disease." *Expert Opinion on Drug Safety* 17.10 (2018): 1053-1061. <https://www.tandfonline.com/doi/abs/10.1080/14740338.2018.1524870>
27. Schiller, Emily R., Bret David Silverglate, and George T. Grossberg. "Profiling lecanemab as a treatment option for Alzheimer's disease." *Expert Review of Neurotherapeutics* 24.5 (2024): 433-441. <https://www.tandfonline.com/doi/full/10.1080/14737175.2024.2333970>
28. Mintun, Mark A., et al. "Donanemab in early Alzheimer's disease." *New England Journal of Medicine* 384.18 (2021): 1691-1704. <https://www.nejm.org/doi/full/10.1056/NEJMoa2100708>
29. Rashad, Areeba, et al. "Donanemab for Alzheimer's disease: a systematic review of clinical trials." *Healthcare*. Vol. 11. No. 1. MDPI, 2022. <https://www.mdpi.com/2227-9032/11/1/32>
30. Lloret, Ana, et al. "The effectiveness of vitamin E treatment in Alzheimer's disease." *International journal of molecular sciences* 20.4 (2019): 879. <https://www.mdpi.com/1422-0067/20/4/879>
31. Jicha, Gregory A., and William R. Markesbery. "Omega-3 fatty acids: potential role in the management of early Alzheimer's disease." *Clinical interventions in aging* (2010): 45-61. <https://www.tandfonline.com/doi/full/10.2147/cia.s5231>
32. Chen, Min, et al. "Use of curcumin in diagnosis, prevention, and treatment of Alzheimer's disease." *Neural regeneration research* 13.4 (2018): 742-752.
33. Liao, Ziwei, et al. "Meta-analysis of Ginkgo biloba preparation for the treatment of Alzheimer's disease." *Clinical neuropharmacology* 43.4 (2020): 93-99.
34. Brusco, Luis I., Miguel Márquez, and Daniel P. Cardinali. "Melatonin treatment stabilizes chronobiologic and cognitive symptoms in Alzheimer's disease." *Neuroendocrinology Letters* 21.1 (2000): 39-42. <https://www.nel.edu/userfiles/articlesnew/NEL210100A04.pdf>