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A Review on Alzheimer's Disease

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ABSTRACT

Alzheimer's is one of the most common causes of dementia that influence nerve cells in various parts of the brain. Pathologically it is caused because of intracellular neurofibrillary tangles and extracellular amyloidal protein and results in the deposition of plaques that obstruct the communication between the nerve cells resulting in this neurodegenerative disease. The genetic risk factor found to be associated with this disease is a mutation in APP, PSEN1, and PSEN2 genes. Also, diet and nutrition play quite an important role in the development as well as prevention of Alzheimer's Disease. The biomarker used for the detection of the disease should be able to differentiate between different causes of dementia and should be able to detect an early stage. Further, the use of Induced pluripotent stem cells has proven to be an effective treatment for the cure of this disease. The objective of this review is to highlight the pathway that leads to this disease and stem cell treatment of this disease.

Keywords: Pluripotent stem cells, Amyloidal protein, Dementia

1. INTRODUCTION

The most prevalent form of dementia, Alzheimer's disease (AD), is named after the German psychiatrist Alois Alzheimer and is characterized by neuritic plaques and neurofibrillary tangles (Figure 1) as a result of amyloid-beta peptide (A) buildup in the medial temporal lobe and neocortical structures, which are the most affected regions of the brain [1]. When Alois Alzheimer examined the brain of his first patient, who had memory loss and a change in personality before passing away, he found amyloid plaques and detected a significant loss of neurons[2, 3]. He classified the illness as a serious disease of the cerebral cortex[4]. The term "Alzheimer's disease" was first used by Emil Kraepelin in his psychiatry text, 8th edition.[5,6,7]

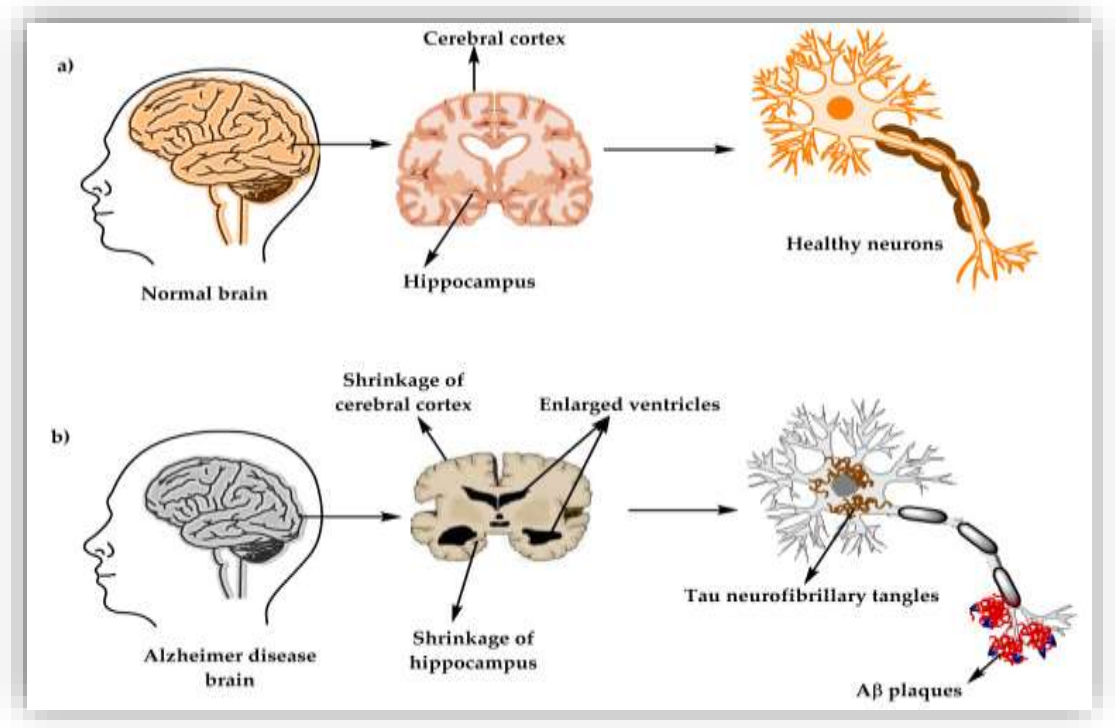


Fig.1 The physiological structure of the brain and neurons in a healthy brain and (b) Alzheimer's disease (AD) brain

2. ETIOLOGY

Risk factors from both the environment and genetics can contribute to the development of AD. Age is the biggest risk factor. The chance of getting AD is approximately 3% at age 65 and increases to almost 30% by age 85 [8]. It is less clear how common AD is in people under the age of 65, but estimates indicate that this age group accounts for about 3% of AD cases [8]. Age-specific incidence appears to be declining in several nations, despite the fact that overall numbers are rising as the population ages [9–11]. AD can be categorised according to when it appears and if it is hereditary. Over 95% of cases [12] of late-onset Alzheimer's disease (LOAD) manifest beyond age 65, whereas early-onset Alzheimer's disease (EOAD) manifests before age 65[13,14].

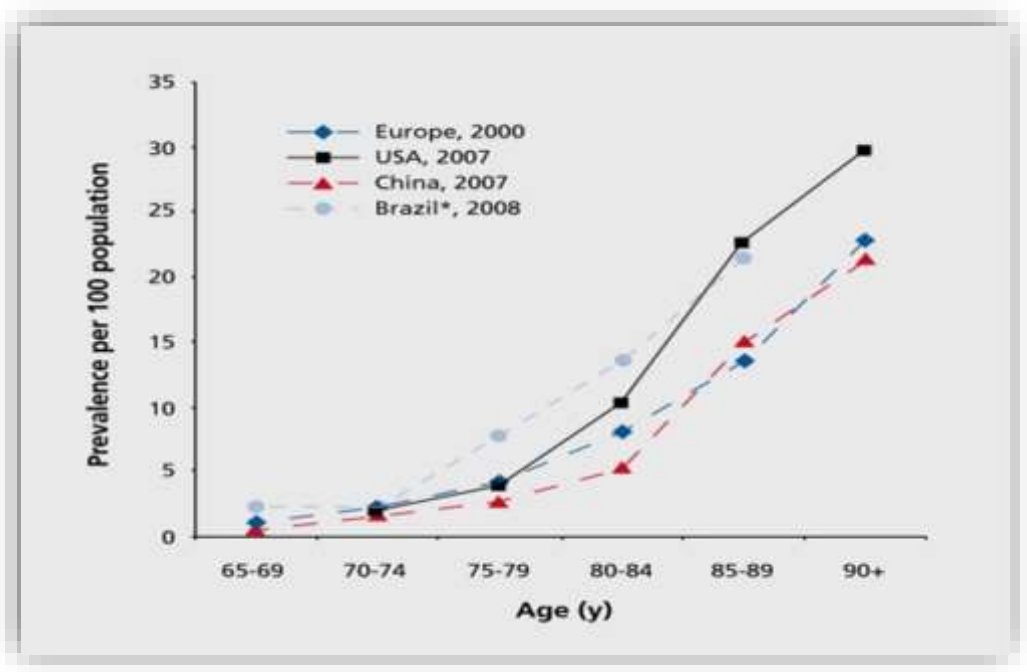
3. EPIDEMIOLOGY

Epidemiologic studies on dementia and AD in low- and middle-income nations have received a lot of attention lately. According to a comprehensive analysis, the overall prevalence of AD in developing nations was estimated to be 3.4% (95% CI, 1.6% - 5.0%).[15]The 10/66 Dementia Research Group discovered that, compared to industrialized nations, the prevalence of dementia (as measured by DSM-IV criteria) among those 65 and older in seven developing nations ranged widely from less than 0.5% to more than 6%.[16] In fact, rural areas of Latin America and India had dementia prevalence rates that were about one-fourth as high as those in European nations. However, in China's metropolitan regions, the prevalence of AD in people 65 and older was 3.5%, and after accounting for negative screening results, it was even higher (4.8%)[17,18]

Fig.2. Age-specific prevalence of Alzheimer's disease (per 100 population) across continents and countries. *, the prevalence of all types of dementia

4. CLINICAL PRESENTATION

The most typical AD presentation involves an aged person who is experiencing an insidious progression of cognitive decline, which is mostly characterized by memory loss.[19] Early signs can include declines in non-memory components of cognition such as difficulty locating words, problems with eyesight or spatial awareness, and weakened judgment or reasoning. Patients currently fit the description of mild cognitive impairment [20]. Nearly all AD patients also experience neuropsychiatric symptoms at some point in the course of their illness, with despair and apathy being the most prevalent early on. Aggression, both verbal and physical, is regularly seen in all phases[21,22]. Delusions, hallucinations, and violence increase in frequency as the disease worsens, and circadian sleep-wake rhythms become more pronounced in comparison to those associated with healthy aging like the illness[23-25]



5. GENETIC RISK FACTORS

AD can be categorized based on when the first symptoms appeared. About 4-6% of cases of AD are early-onset, which affects those under 65; late-onset AD, on the other hand, affects people 65 and older[26]. In addition to the age at which symptoms first appear, there are also clinical, cognitive, neuropathological, and neuroimaging differences between the early and late forms of AD [27,28]. Ballard et al. (2011) estimate that genetics account for

around 70% of the chance of getting AD[29]. Early Alzheimer's disease (AD) is typically caused by mutations in the APP, PSEN1, and PSEN2 (genes for the amyloid precursor protein, presenilin 1 and 2, respectively), whereas late-form AD is primarily linked to an APOE gene polymorphism, particularly[30,31]

6. PREVENTION

Nutritional therapy through nutrition may be able to decrease the onset of dementia and presumably enhance the quality of life for AD patients without having any impact on survival rates [32,33,34]. It has been proven that eating things like fish, fruits, vegetables, nuts, and even Indian spices can cut your risk of AD by up to 45%[35]. As was stated before in our review, fructose intake should be no more than 25 g per day[36,37]. a reduction in Alzheimer's symptoms when the brain contains a sufficient amount of magnesium[38]. Because of its ability to strengthen the immune system and reduce inflammation, vitamin D also has positive benefits on AD[39]. Consuming a diet high in omega-3 fatty acids and vitamin B12 is recommended. by way of astrocytes In a study using adult and newborn mice raised in culture, astrocytes were injected into the hippocampi of AD animals[40].

7. CONCLUSION

Alzheimer's disease is not a life-threatening condition. If the patient is provided the appropriate care and management from the time the condition is diagnosed until the patient passes away, it can be controlled. As there is no known treatment to prevent Alzheimer's disease. The main goal should be to lessen the severity of the patient's symptoms. To help the patient regain his capacity to lead a regular life, support should be provided. The condition is deadly, but the symptoms can be managed with medication and the right physical and mental care to lessen the patient's suffering. The numerous medications used to treat Alzheimer's disease include cholinesterase inhibitors Donepezil and Memantine, among others.

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