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Increasing Burden of Alzheimer's Disease by Aging

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Alzheimer's disease (AD), the most common cause of dementia in elderly individuals, is a devastating chronic disorder characterized by a progressive loss of memory, cognitive decline and functional impairment causing difficulties in performing activities of daily living. Major neuropathological hallmarks include deposits of anomalous misfolded proteins in the brain, i.e., the extraneuronal plaques composed of amyloid β peptide (Aβ), and the intraneuronal neurofibrillary tangles (NFT) composed of hyperphosphorylated tau protein (1).

With the increase in life expectancy and the absence of effective disease-modifying agents, the number of people with AD is expected to triple within the upcoming 40 years (2). The prevalence and incidence of AD has been increasing exponentially with the advancing of age, with a new case occurring every seven seconds globally, and every 15 minutes in Korea. AD itself is becoming a slow pandemic over time (3).

Despite the remarkable improvements in our understanding of the cause of sporadic AD (more than 90% of all AD) over last several decades, the accurate etiopathomechanism of AD remains unclear. Several independent hypotheses have been proposed to address the pathological lesions and neuronal cytopathology in connection with apolipoprotein E (ApoE) genotyping, hyperphosphorylation of cytoskeletal proteins, oxidative stress, abnormal cell cycle re-entry, neuroinflammation and Aβ metabolism. It is because of these complexities, AD has been considered as a multifactorial disease.

Over the past decade, research efforts have concentrated largely on therapeutic strategies that aim to prevent the formation and deposition of Aβ and tau, or accelerate their clearance; however, novel drug development based on these hypotheses thus far has been unsuccessful. Drugs currently available are acetylcholine esterase inhibitors (AChEIs) and an N-methyl D-aspartic acid (NMDA) receptor modulator, which provide only modest symptomatic effects. There is an urgent unmet need to delay onset or to halt the progression of dementia and identify new approaches to manage the existing disease.

The accumulating care burdens associated with the progression of AD place considerable stress on caregivers and families of the person with AD (4). The global burden of AD has estimated that dementia accounts for 11.2% of disability years in people > 60 years of age, a value higher than the disability years attributable to stroke (9.5%), cardiovascular diseases (5.0%), musculoskeletal disorders (8.9%) and all forms of cancer (2.4%) (3). These facts and figures emphasize the importance of prevention of this debilitating disease to reduce the burden on society, as well as, ease suffering of those affected and their families.

Barnes and Yaffe (2) calculated the population-attributable risk for seven lifestyle risk factors that seem most promising in terms of primary prevention. Given the expected dramatic increase in the incidence and prevalence of AD, the identification of successful prevention and treatment strategies is critical. As a result, prevention of dementia through risk factor identification, and modification is of the utmost importance until disease-modifying agents prove efficacious. As a result, increasing attention is being paid to factors that might prevent or delay the onset of dementia. If preventative strategies can delay dementia onset by two years, then up to 25% of dementia cases may be prevented (5). In particular, interventions that combine a number of factors such as healthy nutrition, along with cognitive, social, and physical activity should be encouraged. In the most optimistic view, dementia could be delayed or even prevented by these interventions.

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