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# CORRELATION BETWEEN AGING AND ALZHEIMER'S DISEASE: A REVIEW

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**ABSTRACT:** Dementia, commonly Alzheimer's disorder (AD), is a developing pandemic that presents profound challenges to healthcare systems, families, and societies in the course of the world. Alzheimer's disorder, referred to as an innovative multifarious neurodegenerative disorder, is the essential motive for dementia in a late person's life; brain developing older performs a pivotal feature in the pathogenesis and improvement of Alzheimer's disease. Oxidative stress, a circumstance of unbalance between reactive oxygen species and antioxidants, provides a robust contribution to the excessive incidence of AD in old age subjects. The normal getting older technique is related to declines in positive cognitive abilities, such as processing speed and some factors of memory, language, visuospatial function, and executive function. Research in neurology has mounted decreases in the quantity of gray and white matter and upgrades in the feature of white matter that might also lead to cognitive changes determined with aging. It is necessary to think about these natural cognitive modifications because, first, they can have an effect on the daily functioning of an older adult, and second, they can assist differentiate normal from disorder states. In this study, we will pay attention to the correlation and impact of aging on cognitive functioning and memory loss in aged human beings due to Alzheimer's disease, with early prevention and remedy to limit the cause of Alzheimer's disease at an early stage of aging.

**INTRODUCTION:** In 1906, Dr. Alois Alzheimer diagnosed the brain of a female who misplaced her existence due to the truth of certain exceptional symptoms, such as memory loss, during his study [1]. Alzheimer's disorder (AD) is one of the most regular neurodegenerative problems in aged people and accounts for larger than 80% of the world's cases of dementia. This leads to a gradual deterioration of emotional, behavioral, useful impairment and getting to know ability [2].

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As age is the most strong risk aspect for growing AD, Wilson mentioned that any attempt to account for the heterogeneity of AD need to likely begin with age [3]. Memory loss and other cognitive deficiencies in the elderly have been regarded as a consequence of the aging process for several years and are therefore referred to as 'senile dementia,' whose frequency and the incidence rises exponentially with the population [4].

As individuals age, cognition is essential for functional independence, including whether someone can live independently, handle money, correctly take drugs and drive safely. Furthermore, intact cognition is important for human beings to interact effectively, including the processing and integration of sensory input and the proper response to others.

With age, cognitive skills also decrease. Due to the increasingly rising number of adults over the age of 65 and the growing prevalence of age-related neurodegenerative dementias, it is important to consider the impact of age on cognition [5]. Changes in memory are one of the most common cognitive complaints among older adults. Indeed, as a group, older adults do not perform on several learning and memory tests and younger adults. Memory changes due to age can be attributed to slow processing speed, decreased ability to ignore irrelevant information, and decreased use of learning and memory enhancement techniques [6]. While dementia requires new functional dependency based on progressive cognitive impairment, it is a clinical diagnosis.

With age, the incidence of dementia increases, making it an increasingly common occurrence in our aging population. The essence of the symptoms means that people with dementia, both socially and in terms of physical and mental wellbeing, are more dependent and fragile, presenting changing challenges to society and our health systems [4]. In this review, we will discuss the occurrence of Alzheimer's disease in the aging population, associated with declines in certain cognitive abilities with earlier prevention.

**Pathophysiology of Alzheimer Disease:** The atrophy of the hippocampus and cerebral cortex is at the macroscopic level, which occurs more sharply due to age in AD. The formation of amyloid plaques or senile plaques, which are amorphous structures of Abeta, and the accumulation of hyper-phosphorylated Tau protein, which means the formation of neurofibrillary tangles and extensive neuronal tangles, can be observed microscopically. Recent research has shown that different mechanisms, ranging from genetic imprint elements to family history and apolipoprotein-containing mechanisms are associated with the formation of these AD markers, the oxidation mechanism that contributes to the neurodegeneration process [7]. In the hippocampus, amygdala, entorhinal cortex, and cortical affiliation regions of the frontal, temporal and parietal cortices, neuronal loss and/or pathology may also be specifically viewed. However, subcortical nuclei such as the serotonergic dorsal raphe and the cholinergic basal nucleus are also included [8].

The reported histopathological properties of AD are also extracellular aggregates of Aβ plaques and intracellular aggregations of neurofibrillary tangles. In the neocortex, hippocampus, amygdala, diencephalon, and basal ganglia in the basal, temporal, and later forming at some point, Aβ plaques advance at the beginning. In basic cases, Aβ is also found at some point in the mesencephalon, brain stem decrease, and cerebellar cortex. The tau tangle located in the locus coeruleus, transentorhinal, and entorhinal regions of the brain is triggered by this recognition of Aβ. [9]. The main pathological hallmarks of AD are called senile plaques (SPs) and neurofibrillary tangles (NFTs). Based on the different causative factors to describe this multi factorial condition, several theories have been put forward.

The cholinergic hypothesis, the Amyloid-beta hypothesis, the tau hypothesis, the Inflammation hypothesis are some of those included. The mechanism of occurrence of AD is detailed in the Amyloid Cascade hypothesis and Cholinergic hypothesis. Amyloid plaque development occurs at a later age, leading to cognitive decline due to neurotoxicity due to these Amyloid-beta oligomers. Widespread cell dysfunction & degeneration can lead to cholinergic neurons being primarily affected by neurotransmission defects. Loss of cholinergic activity is corrected with AD severity as the condition progresses as the cholinergic neurons begin to expand in the hippocampus region alongside these nicotine receptors and the cortex is also diminished, but the presynaptic nicotine receptors manipulate the release of acetylcholine [2].

**The Biology of the Aging Mechanism:** Aging is an unavoidable and irreversible process, synonymous with changes in physical appearance and function in the individual organism that are readily recognizable and characteristic. Studies have identified aging-related molecular and cellular processes and biomarkers and classified them into primary, antagonistic and integrative hallmarks. In the field of aging science, these hallmarks are commonly used [10]. Genomic instability, gene expression, epigenetic changes, and proteostasis loss are the main hallmarks. As a significant driving force of aging, genomic instability is known to be the core subject of one of the dominant aging theories.

Mitochondrial dysfunction, cellular senescence, and deregulated nutrient sensing are the antagonistic hallmarks of compensatory or antagonistic responses to primary injury. These reactions initially mitigate the damage, but eventually, they may themselves become deleterious. The integrative characteristics of stem cell fatigue and altered intercellular connectivity develop as a result of accumulated damage incurred by primary and antagonistic characteristics and are largely responsible for the functional deterioration associated with aging [11].

**Changes in Cognition with Normal Aging:** Because of the increasingly growing number of adults over the age of 65 and the increasing incidence of age-related neurodegenerative dementia, it is important to consider the impact of age on cognition. The life expectancy for both males and females has significantly increased over the past century. In 1910, for example, a man's life expectancy was 48 years and a woman's was 52 years. This has risen to 76 years for males and 81 for females in 2010. Because many more people live longer, the number of people with age-related neurodegenerative dementia is also increasingly growing [12].

The Alzheimer's Association reports that in 2014, 5.2 million people in the United States had a clinical diagnosis of Alzheimer's disease (AD). The number of people with an AD diagnosis is expected to grow to 13.8 million by 2050 unless successful prevention or prevention treatment measures are established. Therefore, it is important to understand how age affects cognition and what preventive or therapeutic approaches can maintain cognition into advanced age.

Any solution that could minimize the adverse cognition effects of age or reduce the risk of developing neurodegenerative dementia will significantly impact the quality of life of millions of older adults. Cognitive skills, including concentration, memory, executive cognitive function, language, and visuospatial skills, can be divided into many distinct cognitive domains [12]. During senescence, only a minority of aged individuals retain their full cognitive performance level. Most healthy people over the age of 50, episodic memory decreases concerning the previous output of the person; they experience good aging. Episodic memory loss associated with age usually affects free recall rather than recognition. A decline in cognitive processing speed and executive functions are other cognitive changes associated with natural aging. Cognitive aging usually does not affect cognition globally but leaves particular cognitive domains unchanged, such as familiarity judgment [13].

**Neurocognitive Changes in Aging:** The understanding of the cognitive and behavioral symptoms of dementia and their relationship to underlying brain disease is one of the major challenges faced by neuropsychologists over the last 50 years. With the aging population and the age-related presence of many neurodegenerative diseases that produce dementia, this problem has increased significantly over the years [14]. In scientific literature, cognitive modification as a natural aging mechanism has been well established. Some cognitive skills are immune to brain aging, such as vocabulary, and may even develop with age. Other skills degrade steadily over time, such as logical thinking, memory and processing speed. In the rate of decline in certain skills, such as measurements of perceptual reasoning and processing speed, there is substantial variability among older adults [6].

In 1907, the symptoms of a 51-year-old woman, Auguste Deter, who was under his care at the state asylum in Frankfurt, Germany, were carefully identified by Aloysius 'Alois' Alzheimer. The first neuropsychological interpretation of the condition is almost definitely Alzheimer's explanation of her symptoms: "Her memory has been significantly compromised.

When things are presented to her, she correctly calls them, but she has forgotten everything almost immediately afterward. She skips from line to line while reading a test or reads by individually spelling the words or rendering them meaningless by her pronunciation. When Auguste Deter died, Alzheimer's used the modern histological technique of silver staining to microscopically analyze her brain. The neuritic plaques, neurofibrillary tangles, and amyloid angiopathy that were to become the hallmarks of the disease were found [14].

**Aging as a Risk Factor for Neurodegenerative Disease:** Age is the biggest danger factor for probable AD. The effects of adverse events accumulate over our lifespan and can eventually affect the brain and raise the risk of neurodegenerative disease (an indirect consequence of aging). Alternatively, changes that raise the risk of AD, could be correlated with the aging process itself, a direct effect of aging [13].

**Indirect Consequences of Aging:** The correlation between probable AD and mid-life hypertension, diabetes mellitus, and hyperhomocysteinemia is demonstrated by epidemiological studies. A significant risk factor for the development of AD is clinical stroke as well. The presence of white matter hyperintensity often raises the risk of dementia in the absence of a clinical stroke [15].

**Direct Consequences of Aging:** Aging and the hypothesis of amyloid cascade Ab rates increase with age in humans, primates, and transgenic mice. Logically, through three main processes, this may happen:

- Increased development of (Amyloid beta) Ab.
- Improved Ab 42 ratios over Ab 40 ratios
- Reduced Ab 422 clearance [15].

**Aging Factors that Lead to Cause Alzheimer's disease:**

**Cardiovascular Disease:** Heart disease is a growing concern, and in other organs, including the brain, the resulting vascular insufficiency can impair function. There is an expanding body of literature concerning heart disease as a dementia risk factor. Reduced cerebral blood flow (CBF) due to any form of heart disease worsens the brain's vascular homeostasis. It magnifies any cognitive difficulties caused by the accumulation of tau and Aβ proteins [16]. The role of cardiometabolic health in cognitive health and decline is increasingly recognized, including vascular stiffness and reactivity. In patients older than 80 years, higher aortic stiffness was associated with a more pronounced decrease in cognitive function. Microvascular function was significantly reduced in patients with dementia compared to controls. Some metabolic changes, such as obesity or type 2 diabetes mellitus, may increase the risk of age-related cognitive impairment, especially in the elderly [17]. Type 2 diabetes mellitus is a disease in which increased hepatic glucose output, decreased insulin production by pancreatic beta cells, the impaired release of insulin in response to hyperglycaemic stimuli result in a high level of blood glucose. Type 2 diabetes mellitus (T2DM) and Alzheimer's Disease (AD) are also more prevalent with aging, and hyperinsulinemia and insulin resistance can lead to memory failure in patients with T2DM [18].

**Anemia:** Anemia is associated with morbidity and mortality in older adults. A few recent findings indicate that anemia or abnormal hemoglobin levels are associated with an increased risk of dementia and progressive cognitive impairment in the elderly. Participants with anemia have a greater risk of developing dementia in the prospective sample of older adults without baseline dementia than those without anemia after adjustment for age, ethnicity, sex, and education. After adjusting for possible confounders, including comorbidities and other indicators of red cell health, the correlation was still substantial and meaningful. Cognitive dysfunction and dementia may also be associated with anemia due to a lack of micronutrients such as iron and vitamin B12. Iron deficiency can contribute to cerebral hypoxia and cognitive impairment since it plays an essential role in oxygen transport and storage in the brain [19]. Anemia is a significant problem among the medical conditions that follow the cognitive impairment in the pathological aging process. Studies investigating the effect of anemia on the cognitive decline were primarily inspired by certain unique samples, such as hospitalized elderly, end-stage renal disease, heart failure, malignancy, and fingering, suggesting that anemia was strongly correlated with cognitive impairment [20].

**Oxidative Stress:** The theory of oxidative stress in aging is based on the idea that age-related functional declines are due to the accumulation of damage caused by RONS. The imbalance between the development of reactive oxygen and nitrogen species (RONS) and these antioxidant defenses causes oxidative stress. Aging is a phenomenon marked by the gradual deterioration of tissue and organ function [21].

The brain is a highly metabolically active tissue that relies on oxidative phosphorylation as a way to retain energy. Free radicals are a typical product of natural cellular aerobic metabolism. The in-built antioxidant system of the body plays a key role in preventing any loss that free radicals cause. However, the imbalanced antioxidant defense mechanism, overproduction, or introduction of free radicals from the atmosphere into the living system contributes to extreme penalties leading to neurodegeneration. In neurodegenerative disorders, neural cells undergo functional or sensory failure. Oxidative stress (OS) leading to a free radical attack on neural cells leads to neurodegeneration, in addition to many other environmental or genetic factors. Although oxygen is essential for life, imbalanced metabolism and production of excess reactive oxygen species (ROS) result in several disorders such as Alzheimer's disease, Parkinson's disease, aging, and many other neural disorders [22].

**Neuroinflammation:** Neurodegeneration is a condition that arises through the marks associated with the loss of neuronal structure and function in the central nervous system. After viral insult and often in numerous so-called 'neurodegenerative disorders', neurodegeneration is primarily observed in the elderly, such as Alzheimer's disease, multiple sclerosis, Parkinson's disease, and amyotrophic lateral sclerosis that adversely affect mental and physical functioning [23]. Accumulating evidence shows that inflammation levels, and immune response, are rising in the body and the brain with age.

The effects of both acute and chronic inflammation on the brain have been related to cognitive impairment and the risk of dementia in older adults. Normal aging is associated with increased and sustained body-wide inflammation, and the brain is important for cognition. In turn, neurodegeneration, impaired neurogenesis, atherosclerotic processes, and chronic diseases are associated with recurrent elevated inflammation levels. Evidence also indicates that peripheral infection and the resulting stimulation of immune responses may intensify some neurodegenerative diseases [24]. The effect of age on neuroinflammatory responses, including glial activation, increased development of pro-inflammatory cytokines, and aberrant neuronal signaling, could increase the degradation of the microenvironment of the central nervous system in the disease and may lead to accelerated cognitive impairment [25].

The aging process is characterized by an organism's lifetime by gradual degradation of homeostatic functions. For instance, there is a balance between pro-inflammatory and anti-inflammatory cytokines in the adult brain, but this balance changes to a pro-inflammatory state with increased age [26].

**Blood Pressure:** Hypertension, particularly high blood pressure in the middle of life, has been linked to a greater risk of cognitive impairment and dementia, including Alzheimer's disease [27]. In elderly people, Alzheimer's disease (AD) and vascular dementia (VaD) are major causes of cognitive impairment. The incidence of dementia is expected to increase exponentially as a consequence of an aging population worldwide. In the pathogenesis of both AD and vascular dementia, vascular risk factors are involved. Midlife hypertension is especially linked to an increased risk of developing dementia. As the risk of stroke arises from cerebrovascular insults in cortical and subcortical areas responsible for memory and executive function, one could expect that the treatment of high blood pressure in midlife will minimize the risk of developing dementia [28]. A common risk factor for CVD, chronic hypertension, causes vessel wall thickening, decreased elasticity of the vessel, and narrowing of the lumen, especially in small vessels. These sequelae contribute to decreased cerebral blood flow, a prominent phase in both AD and CVD pathophysiology [29]. Hypertension, especially high midlife BP, has been associated with a higher risk of cognitive impairment and dementia [27].

**High Cholesterol:** W risk factor for AD, dyslipidemia has been reported. With aging, the synthesis of lipoproteins undergoes many hepatic and hormone-mediated changes. Longitudinal studies have shown that total cholesterol levels rise in men until about 50 years of age after the start of puberty, followed by a plateau until 70 years of age when the concentration of serum cholesterol decreases slightly. Total cholesterol levels are comparable in women and men between the ages of 55 and 60 and higher in women than in men after the age of 60. Changes in total serum cholesterol

concentration due to age are mostly due to an increase in LDL cholesterol levels [30]. This term refers to elevated blood lipid or lipoprotein levels, including high low-density lipoprotein (LDL-C), low high-density lipoprotein (HDL-C), total cholesterol (TC), and triglyceride levels (TG). The connection between cholesterol and AD has been extensively investigated over the last ten years, particularly in longitudinal epidemiological studies. Data indicates a link between mid- and late-life elevated blood cholesterol levels and the development of dementia [31].

**Obesity:** And there is a substantial correlation between younger and middle-aged measured overweight and obesity with an increased risk of incident dementia [32]. The increased risk of Alzheimer's disease (AD) and underlying neurodegenerative changes are also associated with obesity and dementia.

Analysis studies provide evidence that obesity and dementia contribute to an early memory crisis in old age. Obesity, which causes vascular dementia, not only reduces the blood flow to the brain but also increases the fat cells that destroy the white matter of the brain, resulting in cognitive and intellectual behavior loss.

Adipocyte-secreted proteins and inflammatory cytokines clarify the association between obesity and increased dementia risk. In modern medical science, obesity and dementia are two critical topics, as both are prevalent at an alarming pace. Obesity is described as a condition in which too much body fat is found in the person, a concept that is misunderstood by many.

This condition can be attained by a person who consumes more calories than he or she burns, which leads to different complications of cardiovascular disease, cognitive disabilities, sleep disorders, and joint complications. Increased body fat content will promote a reduction in blood flow to the brain, encouraging vascular dementia. Loss of blood supply will result in brain ischemia, leading to an increased risk of memory loss and hence long-term dementia. Also, obese individuals appear to have higher levels of cytokine-released adipokines or fat cells. These cells have been associated with decreasing white matter in the brain, which is responsible for brain-wide nerve connections. Decreased neuronal activity and decreased brain vascular supply obsolete with brain atrophy and thus loss of normal brain function [33].

**Prevention of Cognitive Decline Through Nutritional and Other Lifestyle Interventions:** Preclinical and clinical research provides useful evidence about the impact on cognitive performance of particular dietary habits and specific nutrients. For example, as main environmental factors for oxidative stress and brain disorders in older adults, deleterious dietary habits (overfeeding or intake of low antioxidant nutrients) and sedentary lifestyles, or emotional stress, have been recorded

**Micronutrients:** Important antioxidant trace elements such as Zinc (Zn), Selenium (Se)) and/or insulin sensitizers (Chromium (Cr), Zn) are profoundly involved in brain defense, as stated by numerous studies in animals and humans. *I.e.*, selenoproteins, Protect brain cells from oxidative stress with Selenoprotein P and Glutathione peroxidase.

A low Se status increases the risk of cognitive decline. The greater the drop in plasma Se, the greater the risk of cognitive decrease. By enhancing insulin sensitivity and reducing inflammation and oxidative stress, Zn also works positively on brain health. However, no aging brain benefits have been identified to date after human Zn supplementation, and even a possible neurotoxic effect of Zn accumulation has been found in patients with AD. Insulin resistance and increased oxidative stress have resulted from Cr deficiency. Indeed, higher Cr intakes are associated with better cognitive functions in insulin-resistant states (T2DM ) [17].

**Polyphenols and Polyphenol-rich Diets A:** In particular, it has been reported that polyphenols exercise their neuroprotective activities through the potential for their neuroprotective activities to protect neurons from neurotoxin-induced damage, The ability to inhibit neuroinflammation, and the ability to encourage memory, learning, and cognitive function. Polyphenols such as polyphenol-rich foods or drinks have beneficial effects on memory and learning in both animals and humans, especially tea, Gingko Biloba, cocoa,

and blueberry. However, recent data indicates that their beneficial effects include decreases in the signaling of oxidative/inflammatory stress, increases in protective signaling, and neurotic effects leading to the expression of antioxidant enzyme-encoding genes, phase 2 enzymes and neurotrophic cytoprotective protein factors. The sirtuin-FoxO pathway, the NF-kB pathway, and the Nrf-2/ARE pathway are basic examples of such pathways. These mechanisms function together to preserve brain homeostasis and play an important role in the adaptation of neuronal stress, such that polyphenols may prevent the development of neurodegenerative pathologies in the aging community [34].

**Flavonoids:** Flavonoids are present in high amounts in fruits, fruit and fruit juices, and other beverages such as tea, red wine and chocolate; plant extracts high in flavonoids from pine bark, Gingko Biloba and Pueraria lobata is regularly consumed for their reputed health-giving properties, especially to protect elderly people from cognitive decline [35].

**Vitamins:** In the nervous system, vitamins and minerals have several roles that are essential for brain health. Various vitamin and mineral supplements have been suggested to be effective in preserving cognitive function and slowing the development of dementia, for example, vitamin B, vitamin B12, vitamin E, vitamin C, play a significant role in cognitive function in aged individuals [36].

**Prevention of Cognitive Decline by Other Lifestyles:** Aging also contributes to a deterioration in cognitive skills. Severe cognitive dysfunction leads to reduced functioning and the need for medication. It will help to minimize the need for long-term care to prevent cognitive deterioration and prolong its progression. In late life, both genetic and environmental variables are major determinants of cognitive health. To avoid cognitive deterioration, a higher cognitive reserve helps. Instead of a structural reserve, the cognitive reserve is now known as a functional reserve. Through experience, cognitive reserves can be improved. People with higher levels of education tend to have higher reserves of cognition. A buffer will act as a stronger cognitive reserve. In late life, involvement in cognitively stimulating activities can avoid cognitive decline. Cognitive wellbeing is often enhanced by physical activity. Cognitive functions such as motor functions, cognitive speed, and auditory and visual attention are enhanced by aerobic exercises that enhance cardiorespiratory fitness.

Beneficial impacts are also reported on executive functions. Healthy diets are considered helpful in preserving cognitive health, particularly adherence to the Mediterranean diet. Social activity involvement may also decrease cognitive decline. It seems that promoting commitment to a balanced lifestyle and continuing to be mentally, socially, and cognitively active is a successful strategy to avoid cognitive decline [37].

**CONCLUSION:** In healthy adults, decreases in cognitive abilities with age occur during the adult lifespan. Cognition is closely linked to many health problems, such as CVD, diabetes, or obesity. Therefore, a holistic approach should be considered when determining dietary methods to promote healthy brain aging, including nutrition, exercise, and lifestyle factors that address the brain and overall cardiometabolic health.

Natural aging-related processes, including oxidative stress, neuroinflammation, and vascular dysfunction, are the same as those that contribute to neurological disease growth, however, the processes leading to aging are intensified in these pathological conditions and caused by multiple factors that may be hereditary or environmental.

This review article is regarding a correlation study between aging and dementai; we find that aging, in many ways, lead to cause Alzheimer disease. So by anticipating the above risk factors such as hypertension, high cholesterol level, and obesity like factor in further research, it can help reduce the cause of Alzheimer's disease in the early stage of aging by considering their prevention of cognitive decline through nutritional and other lifestyle interventions in early stage.

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