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Understanding the pathophysiology of alzheimer's disease: Insights for early diagnosis

Mohammed Mesfer Musaed AL Khathami
KSA, National Guard Health Affairs

Anood Fhid Alanazi
KSA, National Guard Health Affairs

Razan Walid Alomran
KSA, National Guard Health Affairs

***Abstract*---Background:** Neurodegenerative diseases, particularly Alzheimer's disease (AD), pose a significant health challenge globally, with projections indicating nearly 152 million affected individuals by 2050. AD accounts for 60% to 80% of neurodegenerative cases, manifesting primarily as sporadic Alzheimer's disease (SAD) after age 65. **Aim:** This review aims to elucidate the pathophysiology of AD, focusing on the early identification of biomarkers for diagnosis and the exploration of potential therapeutic interventions. **Methods:** A comprehensive literature review was conducted, examining the biological mechanisms underpinning AD, particularly the role of amyloid plaques and neurofibrillary tangles, along with the impact of lipid nutrients and nanotechnology in treatment delivery. **Results:** Key findings indicate that soluble amyloid-beta oligomers are critical in AD pathogenesis, contributing to synaptic dysfunction and cognitive decline. Moreover, recent advancements in nanotechnology, particularly through nanoliposomes, show promise for enhancing drug delivery across the blood-brain barrier. **Conclusion:** Understanding the complex interplay of genetic, environmental, and pathological factors in AD can inform early diagnostic strategies and therapeutic approaches. The role of lifestyle and dietary interventions is crucial, and future research should focus on leveraging nanotechnology for effective treatment delivery.

***Keywords*---**alzheimer's disease, neurodegeneration, amyloid-beta, biomarkers, nanotechnology, therapeutic interventions.

Corresponding author: *akk29337@gmail.com*
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Introduction

Neurodegenerative diseases (ND) pose a significant health challenge, particularly due to aging populations and lifestyle factors. Currently, over 50 million individuals globally are affected by these conditions, a figure projected to nearly triple to 152 million by 2050 if effective preventive or therapeutic solutions are not developed [1,2]. Alzheimer's disease (AD) is the most prevalent neurodegenerative disorder, accounting for 60% to 80% of cases [3]. Since its identification in 1907, AD has been recognized to have multiple etiologies, yet its precise causes remain unclear. To date, no curative treatment has emerged. AD manifests in two forms: (1) genetic or autosomal-dominant AD (ADAD), which occurs before age 65 and represents less than 1% of cases, and (2) sporadic AD (SAD), which typically arises after age 65, with the risk doubling every five years [4]. This review specifically addresses sporadic AD.

The pathology of AD is characterized by both structural and functional damage within the central nervous system (CNS). Key lesions include amyloid plaques formed by beta-amyloid peptides (Aβ) that accumulate outside neurons [5], and neurofibrillary tangles caused by hyperphosphorylated tau protein that aggregates within neurons [6]. AD progression involves a series of biochemical, neurophysiological, neuroanatomical, and cognitive impairments [7]. Initial oligomerization of soluble Aβ in the brain leads to localized dysfunctions in dendrites, axonal processes, and synapses. Recent research has focused on soluble Aβ oligomers (AβO), identified as more toxic and relevant to AD pathology than other forms of Aβ [8,9]. AβO are seen as pathological agents emerging before the initial neuropathological signs of AD [10,11]. Over time, these agents contribute to the development of brain lesions and neuronal loss in specific regions, initially without clinical symptoms [12,13,14,15]. Ultimately, AD manifests through memory loss and cognitive decline [16]. Numerous therapeutic strategies have been explored over the decades, but existing treatments primarily address symptoms rather than providing curative solutions [17,18,19]. This has shifted research focus towards prevention and the reduction of AD risk. Studies suggest that over 30% of AD cases could be attributable to modifiable risk factors, highlighting promising targets for prevention strategies aimed at reducing cognitive decline related to AD and potentially other neurodegenerative conditions [3,20,21]. Enhancing early detection of the disease at preclinical stages remains a significant challenge [22].

Research has highlighted the critical role of lipid nutrients in brain health and cognitive function. The brain contains high levels of omega-3 polyunsaturated fatty acids (n-3 PUFA), integrated into phospholipids that form neuronal membranes [23,24]. Numerous studies suggest that these fatty acids have neuroprotective properties that support neuronal function and synaptic plasticity [25]. Given the importance of diet as a source of PUFA, nutritional strategies targeting CNS lipid composition are viable for prevention [26]. Another challenge in AD treatment lies in efficiently delivering therapeutic agents to the brain, hindered by the protective barriers of the CNS [27,28]. The blood-brain barrier (BBB) safeguards the CNS from neurotoxins and harmful substances in the bloodstream but also limits the accessibility of therapeutic drugs [27]. Nanoparticles (NPs), ranging in size from 10 to 1000 nm, offer a potential solution

by encapsulating therapeutic molecules and facilitating their transport across the BBB to specific brain regions [29,30,31]. Soft nanoparticles, such as nanoliposomes (NL) or exosomes, are particularly effective for drug delivery, protecting therapeutic agents and enabling targeted release [32,33]. Increasing evidence suggests that NLs exhibit restorative effects in cellular and animal models of neurological disorders, including stroke, Parkinson's disease, and AD, indicating enhanced bioavailability in the CNS [34,35,36].

Furthermore, nanotechnology can enhance the bioavailability of PUFA via NLs. These spherical vesicles, constructed from phospholipid bilayers dispersed in an aqueous medium, can be engineered to incorporate n-3 PUFA-rich phospholipids, offering neuroprotective benefits. NLs can encapsulate a variety of molecules, including hydrophilic and hydrophobic drugs, proteins, and DNA [40]. Their biofilm characteristics closely mimic cell membranes, providing an effective drug delivery mechanism. Additionally, NLs form a protective barrier against degradation by enzymes, digestive juices, and intestinal microorganisms [41,42].

Clinical Spectrum of AD

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by a gradual decline in cognitive functions, often manifesting as insidious onset cognitive disorders. This condition leads to multiple cognitive deficits that progressively worsen over time. Key symptoms include memory impairment, particularly in acquiring and recalling new information [13], alongside one or more associated dysfunctions such as aphasia, apraxia, agnosia, or dysexecutive syndrome. AD is often conceptualized as an amnestic syndrome of the hippocampal type. These neuropsychological disorders significantly impair activities of daily living and result in a notable decline in cognitive and functional abilities compared to prior levels of functioning [16]. In recent years, there has been an increasing focus on neuropsychiatric symptoms and behavioral disorders associated with AD, including psychotic symptoms, depression, apathy, aggression, and sleep disturbances [43,44,45]. In 1996, the International Psychogeriatric Association introduced the concept of behavioral and psychological symptoms of dementia (BPSD) to describe disturbances in perception, thought content, mood, and behavior commonly observed in individuals with neurodegenerative diseases [46]. AD can be understood as a process involving chemical, physiological, and anatomical changes in the brain, which may be identifiable many years prior to the emergence of clinically significant cognitive-behavioral syndromes (CBS) [47].

Pathophysiology of AD

Alzheimer's disease (AD) is characterized by significant structural and functional damage in the central nervous system (CNS), with two primary histological lesions: amyloid plaques and neurofibrillary tangles (NFTs). NFT formation initiates in the internal temporal lobe, often present in hippocampal structures before any observable cognitive decline. As NFTs evolve, they spread to the external temporal lobe and subsequently to posterior cortical associative areas and the entire cortex, paralleling the progression of AD symptoms [6,48]. In contrast to the more localized topography of NFTs, amyloid deposits exhibit a

diffuse distribution. These plaques form initially in the neocortex, then spread to the hippocampus, subcortical nuclei, and cerebellum [5]. Amyloid plaques arise from the aggregation and abnormal accumulation of the amyloid beta (Aβ) peptide in the extracellular space outside neurons. This peptide is generated via the amyloidogenic pathway through the sequential cleavage of amyloid precursor protein (APP) by β- and γ-secretases [49]. Soluble Aβ oligomers can interact with cell membranes, disrupting signal transduction pathways and altering neuronal activities, which leads to the release of neurotoxic mediators by microglia. This cascade results in early synaptic dysfunction and impaired plasticity [50]. The oligomerization of soluble Aβ initiates synaptic deterioration, affects axonal transport, and influences glial cell functions, contributing to oxidative stress, insulin resistance, tau phosphorylation, and selective neuronal death [51,52]. Additional factors, such as dysregulated lipid and glucose metabolism, neuroinflammation, cerebrovascular abnormalities [53], and endosomal pathway blockages [54], also contribute to AD pathology [55]. Impaired vascular function hinders the delivery of blood and nutrients to the brain, leading to chronic inflammation driven by activated astrocytes and microglia. The E4 isoform of apolipoprotein E (ApoE), a significant risk factor for AD, is associated with increased Aβ production and reduced clearance. Cleavage of ApoE4 can yield toxic fragments that disrupt the cytoskeleton and impair mitochondrial function [56], directly impacting Aβ clearance mechanisms.

Hyperphosphorylation of tau proteins leads to the formation of NFTs, which aggregate inside neurons, contrasting with the extracellular nature of amyloid plaques. Tau detachment from microtubules disrupts intracellular transport, resulting in neuronal dysfunction, potential brain atrophy, and cell death [12]. Notably, while NFT presence correlates with symptom progression, the distribution of amyloid deposits does not correlate with clinical symptoms [48]. The amyloid cascade hypothesis suggests that Aβ accumulation triggers NFT formation, heightening neuronal vulnerability and leading to cell death [57]. However, recent models highlight the central role of soluble Aβ oligomers in AD pathogenesis, shifting focus from the traditional amyloid cascade hypothesis [11]. While abnormal metabolism of Aβ and tau proteins are established hallmarks of AD, research indicates that amyloid and tau pathologies can arise independently, influenced by genetic and environmental factors [59,60]. Although the sequence of pathological events remains complex, both amyloid plaques and NFTs undoubtedly accelerate neurodegenerative processes. Initially, prior to the formation of plaques and NFTs, the presence of soluble Aβ aggregates contributes to synaptic destruction, neurotransmitter dysfunction, and glutamatergic excitotoxicity, adversely affecting cholinergic and glutamatergic systems critical for memory and cognition [61]. These systems, integral to neuronal plasticity, exhibit deficits correlated with cognitive decline in AD, with cholinergic deficits detectable even at early histopathological stages [63,64]. The early emergence of pathogenic Aβ aggregates positions them as promising targets for therapeutic and diagnostic interventions [51].

Diagnostic Criteria for Alzheimer's Disease
Criteria of the National Institute of Aging and the Alzheimer's Association (NIA-AA)

The diagnostic criteria for Alzheimer's Disease (AD) were initially established in 1984 by the National Institute on Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association, primarily focusing on clinical-pathologic criteria, particularly memory disorders [67]. However, these criteria were limited, as some individuals could exhibit AD biomarkers without cognitive impairment, and vice versa. In response, the NIA and Alzheimer's Association revised the criteria in 2011 to include mild cognitive impairment (MCI) associated with AD [68,69]. The diagnosis of MCI is now grounded in clinical, functional, and cognitive assessments. The most prevalent form of MCI linked to AD is amnestic MCI (aMCI), which is characterized by memory impairment that is below expectations for the individual's age, gender, and educational level, though it does not meet the criteria for dementia. AD can be diagnosed prior to dementia onset if other factors, such as amnestic hippocampal syndrome and specific AD biomarkers, are present.

Specific AD Biomarkers

According to the NIA-AA and the International Working Group (IWG), AD is considered a slowly progressive neurological disease that commences before clinical symptoms manifest. AD is conceptualized as a continuum encompassing three stages: asymptomatic (preclinical AD), predementia (MCI due to AD), and dementia (due to AD) [22,70,71].

Although the AD diagnosis is predominantly clinical, it is strengthened by evidence of biomarkers indicative of AD-related pathophysiological processes. The new diagnostic criteria mandate the inclusion of cerebrospinal fluid (CSF) biomarkers, such as total and hyperphosphorylated Tau protein levels, Aβ42 levels, and the Aβ42/Aβ40 ratio, along with positron emission tomography (PET) imaging for tau and amyloid to assess the likelihood (high, medium, or low) of underlying neurodegenerative processes contributing to clinical observations [1,22,72]. In 2018, a new biological framework and model for AD biomarkers was proposed, outlining a progressive sequence of neurophysiological, biochemical, and neuroanatomical abnormalities that can be identified years before noticeable cognitive-behavioral syndromes (CBS) [58]. Abnormal deposits of Aβ and Tau proteins remain critical markers of AD pathology, enabling differentiation from other neurodegenerative diseases [71]. Key pathophysiological markers of AD include amyloid pathology (e.g., decreased CSF Aβ1-42 or amyloid tracer accumulation in PET) and Tau pathology (elevated CSF Tau and phosphorylated Tau levels or Tau tracer accumulation in PET). Additionally, topographical markers indicative of AD include changes in brain volume (temporoparietal and hippocampal atrophy, cortical thickness) assessed through magnetic resonance imaging (MRI) and glucose hypometabolism measured by fluorodeoxyglucose (FDG)-PET [73]. The NIA-AA framework categorizes these biomarker criteria into three groups: amyloid, tau, and neurodegeneration.

The Different Stages of the Sporadic Form of AD: The Alzheimer's Spectrum

The Early Asymptomatic Stage: Preclinical Stage

Neuropathological changes in AD can begin 15 to 20 years prior to clinical manifestation [74]. Early alterations in Aβ, including oligomerization in the brain, disrupt dendrites, axonal processes, and synapses. The origins of these abnormal Aβ aggregates remain unclear [14]. During this preclinical phase, lesions develop slowly, typically without any clinical symptoms (patients do not report issues in daily functioning) [12,14,15]. Preclinical AD is characterized by the presence of Aβ biomarkers indicating pathological changes (e.g., PET amyloid retention, low CSF Aβ42) in cognitively healthy individuals or those with subtle cognitive impairments [71]. Research has underscored the concept of cognitive reserve in AD, suggesting that cognition can remain stable despite the presence of Aβ lesions due to compensatory mechanisms—especially those linked to educational background—until the transition to the symptomatic stage (MCI). Consequently, AD may present later in individuals with higher cognitive reserves, as these patients can leverage more extensive neural networks to mitigate the disease's impacts, with symptoms appearing only at advanced stages [75,76,77,78]. For the asymptomatic stage, these criteria are primarily utilized in clinical research rather than for diagnostic purposes.

The Early Symptomatic Stage: Amnesic Mild Cognitive Impairment (aMCI)

In individuals with aMCI, cognitive complaints or deficits are noted by themselves or their close contacts, but these do not significantly interfere with daily living activities. Timely and accurate diagnosis of AD at this stage is crucial, as it allows for the implementation of non-pharmacological interventions and/or pharmacotherapy, potentially even in the preclinical phase. Numerous clinical studies are currently investigating this early diagnosis approach [79,80]. During this early symptomatic stage, testing typically reveals positive signs of amyloid and tau pathology biomarkers [71], while neurodegenerative syndromes remain absent. Conversely, a lack of these biomarkers indicates a low probability of AD progression. The presence of two key CSF biomarkers—amyloidopathy (characterized by low CSF Aβ levels) and neuronal degeneration (elevated CSF Tau and phosphorylated Tau levels)—is associated with a high risk of conversion to AD [71].

Alzheimer's Disease (AD)

In the typical form of sporadic Alzheimer's Disease (SAD), patients exhibit a range of symptoms, predominantly marked by progressive and significant episodic memory impairment, alongside other cognitive deficits, such as executive dysfunction, apraxia, aphasia, and agnosia. Neuropsychiatric disorders are also common, affecting many individuals, including apathy (49%), depression (42%), aggression (40%), anxiety (39%), and sleep disturbances (39%) [81]. These symptoms significantly impact autonomy, often necessitating external assistance for daily activities. Diagnosis at the dementia stage relies on clinical behavioral assessments, where biomarkers serve primarily to enhance diagnostic certainty, particularly in atypical cases or younger patients [22]. The presence of biomarkers can also indicate the severity of AD [7,71]. These include decreased CSF Aβ levels,

increased CSF Tau and/or phosphorylated Tau levels, cortical thinning and hippocampal atrophy assessed via MRI, hypometabolism or hypoperfusion in the posterior cingulate and temporoparietal cortex via FDG-PET, and amyloid deposition detection through PET imaging. Ultimately, the certainty of an AD diagnosis is evaluated on a probabilistic scale, with definitive evidence achievable only through biopsy or autopsy.

Risk Factors for SAD

Current research suggests that the etiology of AD is multifactorial, involving both genetic and environmental risk factors, which can be categorized into modifiable and non-modifiable factors [83,84,85,86,87].

Non-Modifiable Risk Factors

Key non-modifiable risk factors identified in research include age, the presence of the APOE-ε4 allele, and gender [4,88].

Age: Age is the primary risk factor for SAD. The increasing life expectancy correlates with a heightened likelihood of developing neurodegenerative conditions, including AD [89,90]. Normal aging processes involve structural changes in the brain, affecting membrane fluidity, lipid composition, regional brain volume, cortical density, and microstructural integrity of both white and grey matter. This results in a gradual loss of neuronal synapses and a corresponding decline in neuronal density.

Genetic Risk Factors: While autosomal dominant AD (ADAD) is linked to mutations in genes involved in amyloid metabolism (APP, PSEN1, PSEN2), the most significant genetic risk factor for SAD is the APOE gene [91,92,93,94]. The APOE gene is crucial for lipid transport, including cholesterol, within peripheral tissues and the central nervous system. Its role in astrocytic cholesterol transport to neurons is vital for maintaining neuronal membrane integrity and facilitating brain repair processes. The APOE gene has three alleles: ε2, ε3, and ε4. The ε4 allele is associated with a significantly increased risk of SAD, linked to hippocampal atrophy, abnormal Aβ accumulation, and cerebral hypometabolism [95]. The ε4 allele is implicated in neurotoxic and neuroprotective mechanisms, affecting processes like Aβ metabolism, tauopathy, synaptic plasticity, and neuroinflammation [96]. Research indicates that possessing the ε4 allele increases the risk of developing AD by four times, whereas the ε2 allele appears to confer a protective effect, with the ε3 allele showing no significant impact. Notably, some ε4 allele carriers never develop AD, suggesting that additional unidentified factors may influence disease progression [88].

Gender: The prevalence and progression of AD symptoms are disproportionately higher in women [97,98,99]. Various factors, including APOE genotype, cardiovascular health, depression, hormonal changes, sociocultural influences, and specific sex-related risk factors, may contribute to this disparity. Further studies are essential to understand how gender influences biomarker evolution throughout life, including cognitive abilities and neuroimaging, particularly in

younger populations. Additionally, research focused on gender-specific therapeutic developments for AD is warranted.

Modifiable Risk Factors

Modifiable risk factors are of great interest because they offer opportunities for preventive strategies. Cardiovascular health is particularly relevant, as the brain is supplied by an extensive network of blood vessels; thus, a healthy cardiovascular system can be viewed as neuroprotective [100]. Many risk factors for cardiovascular diseases overlap with those for Alzheimer's Disease (AD), including hypertension, dyslipidemia, diabetes, obesity, dietary habits, smoking, and physical inactivity. Consequently, lifestyle choices play a significant role, with intellectual, physical, and social activities, alongside diet, contributing to AD prevention [1,22,100,101,102,103].

Metabolic Disorders and Dyslipidemia

Although the brain constitutes only 2% of total body weight, it consumes approximately 20% of the body's oxygen and 25% of its glucose [101,104]. The brain is also the second most lipid-rich organ, following adipose tissue. Lipids are integral to gray matter, white matter, and nerve nuclei, supporting neuronal growth and synaptogenesis. The lipid composition of the brain includes about 50% phospholipids, 40% glycolipids, 10% cholesterol, cholesterol esters, and trace amounts of triglycerides [105].

Long-chain polyunsaturated fatty acids (LC-PUFAs), particularly docosahexaenoic acid (DHA) and arachidonic acid (AA), comprise 25–30% of total fatty acids in the central nervous system. Cholesterol and omega-3 fatty acids, especially DHA, play critical roles in brain function. Research indicates that lipid homeostasis imbalances correlate with an increased risk of AD [105,106]. The brain holds 25% of the body's total cholesterol, which is synthesized within the central nervous system. Disrupted cerebral cholesterol homeostasis may lead to neurite pathology, tau hyperphosphorylation, and amyloidogenic processes [49]. Elevated brain cholesterol levels and dyslipidemia are associated with a higher incidence of AD [49,55,100,107]. It is plausible that, akin to cardiovascular disease, diabetes, and obesity, lipid homeostasis disruptions may heighten the risk of age-related neurodegeneration and AD. Furthermore, dyslipidemia is often linked with obesity, which has been associated with insulin resistance and hyperinsulinemia in AD development [108,109]. Insulin resistance in the brain contributes to the formation of neurofibrillary tangles and amyloid plaques [110], leading to AD being referred to as type III diabetes. These findings suggest that strategies aimed at maintaining optimal brain lipid levels may be beneficial for preserving neuronal function and synaptic plasticity, thereby lowering AD risk. Dietary interventions can effectively promote proper lipid homeostasis [111,112,113].

Other Risk Factors

Lower levels of cognitive, social, and physical engagement are linked to an elevated risk of developing neurodegenerative diseases [3,83]. An enriched environment that fosters cognitive reserve—shaped by education, social

interactions, diverse leisure activities, and physical exercise—can offer protective benefits. However, these cognitive and physical factors are influenced by additional elements, including nutrition and environmental conditions that mitigate cardiovascular risks, subsequently reducing AD risk [87]. Psychological factors such as depression, anxiety, stress, and chronic psychological distress are associated with increased risks of mild cognitive impairment (MCI) and AD [114,115]. Additionally, excessive tobacco and alcohol consumption can exacerbate cognitive impairments [20]. A history of head trauma and hearing loss may also elevate the risk of AD [3,102,116]. Recently, studies have identified air pollution as a potential risk factor for neurodegenerative diseases [117]. A report identified 12 modifiable risk factors that account for approximately 40% of dementia cases globally: low education levels, hypertension, hearing impairment, smoking, obesity, depression, sedentary lifestyle, diabetes, poor social interaction, excessive alcohol consumption, history of head trauma, and air pollution [117]. These factors have been updated in recent studies [3].

Conclusion

The exploration of Alzheimer's disease (AD) reveals a multifaceted condition characterized by significant neurodegenerative processes, primarily involving amyloid-beta (Aβ) oligomers and tau protein abnormalities. The pathophysiology underscores the complexity of AD, where both amyloid plaques and neurofibrillary tangles play pivotal roles in cognitive decline. Research indicates that these lesions may begin accumulating years prior to the manifestation of clinical symptoms, emphasizing the importance of early detection through biomarker identification. The integration of lipid nutrients into dietary strategies presents a viable pathway for preventive measures, particularly given the neuroprotective properties of omega-3 polyunsaturated fatty acids. Furthermore, advancements in nanotechnology, particularly in developing nanoliposomes, offer promising avenues for effective drug delivery across the blood-brain barrier. This could enhance therapeutic efficacy by targeting specific brain regions affected by AD pathology. Given that no curative treatment exists, the focus must shift towards prevention and risk reduction. Over 30% of AD cases are attributed to modifiable risk factors, highlighting the necessity for lifestyle changes that can mitigate cognitive decline. Future research should prioritize identifying novel biomarkers that enable the differentiation of AD from other neurodegenerative diseases, alongside developing therapeutic strategies that can effectively address the early stages of AD. The multidimensional nature of AD calls for a concerted effort among researchers, clinicians, and public health initiatives to raise awareness, improve diagnostic criteria, and foster preventive strategies. By enhancing our understanding of AD's pathophysiology, we can pave the way for innovative approaches to combat this growing global health challenge. As the aging population increases, addressing AD with a comprehensive strategy that incorporates early diagnosis, lifestyle modifications, and advanced therapeutic technologies will be crucial in managing this debilitating condition.

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فهم الفيزيولوجيا المرضية لمرض الزهايمر: رؤى للتشخيص المبكر

الملخص:
الخلفية :تشكل الأمراض التنكسية العصبية، وخاصة مرض الزهايمر (AD) ، تحدياً صحياً كبيراً على مستوى العالم، حيث تشير التوقعات إلى أن نحو 152 مليون شخص سيتأثرون بحلول عام 2050. يمثل مرض الزهايمر 60% إلى 80% من الحالات التنكسية العصبية، ويظهر في الغالب كمرض زهايمر عشوائي (SAD) بعد سن 65.
الهدف :تهدف هذه المراجعة إلى توضيح الفيزيولوجيا المرضية لمرض الزهايمر، مع التركيز على تحديد المؤشرات الحيوية للتشخيص في مراحل مبكرة واستكشاف التدخلات العلاجية المحتملة.
الطرق :تم إجراء مراجعة شاملة للأدبيات، لفحص الآليات البيولوجية التي underpin مرض الزهايمر، خاصة دور لويحات الأميلويد والتشابكات العصبية، بالإضافة إلى تأثير العناصر الغذائية الدهنية وتكنولوجيا النانو في توصيل العلاجات.
النتائج :تشير النتائج الرئيسية إلى أن أوليغومرات الأميلويد beta-القابلة للذوبان تلعب دورًا حاسمًا في مرض الزهايمر، مما يساهم في ضعف التشابك العصبي والانخفاض المعرفي. علاوة على ذلك، تُظهر التقدمات الحديثة في تكنولوجيا النانو، خاصة من خلال النانو لليبوسومات، وعدًا في تعزيز توصيل الأدوية عبر الحاجز الدموي الدماغي.
الخاتمة :يمكن أن تسهم فهم التفاعل المعقد بين العوامل الوراثية والبيئية والمرضية في مرض الزهايمر في توجيه استراتيجيات التشخيص المبكر والنهج العلاجية. يلعب نمط الحياة والتدخلات الغذائية دوراً حاسماً، ويجب أن تركز الأبحاث المستقبلية على استغلال تكنولوجيا النانو لتحقيق توصيل فعال للعلاج.
الكلمات المفتاحية :مرض الزهايمر، التنكس العصبي، الأميلويدbeta-، المؤشرات الحيوية، تكنولوجيا النانو، التدخلات العلاجية.