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ALZHEIMER'S DISEASE IN THE 21ST CENTURY: CHALLENGES, INNOVATIONS, AND THE ROAD AHEAD

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ABSTRACT

Neurodegenerative diseases, such as Alzheimer's, Parkinson's, Huntington's, and amyotrophic lateral sclerosis, are defined by a gradual loss of neuron structure and function, which ultimately results in cognitive, motor, and functional decline. Even though these disorders can manifest in various ways, they often share similar pathological mechanisms, including protein misfolding, mitochondrial dysfunction, oxidative stress, neuroinflammation, and impaired autophagy. This review focuses on our current knowledge of these common molecular pathways, recent advancements in diagnostic biomarkers, and new therapeutic strategies aimed at modifying the disease. A better understanding of the overlapping and distinct mechanisms of neurodegeneration may lead to the development of more effective, targeted interventions. When it comes to treating neurological disorders, the primary focus is on relieving symptoms, slowing the disease's progression, improving overall quality of life, and helping individuals maintain their independence. Depending on the condition at hand, treatment strategies might aim to tackle the underlying issues, adjust neurotransmitter functions, decrease neuroinflammation, or support nerve protection and healing. Getting an early diagnosis and providing personalized, collaborative care are key to optimizing results and minimizing long-term challenges

In this paper we will review the diagnosis, etiology, genetics, epidemiology, course, and treatment of AD.

KEYWORDS:*AD, Dementia, Amyloid-beta (Aβ), Tau protein / tauopathy, Memory loss*

INTRODUCTION

Alzheimer's disease is the primary cause of dementia and is swiftly becoming one of the most expensive, deadly, and challenging diseases of this century. Since the Seminar was released in 2016, we've seen crucial advancements in our grasp of the disease's underlying pathology, the acknowledgment of several causative and protective genes, the discovery of new blood-based and imaging biomarkers, and the initial hopeful signs of effective disease-modifying treatments and lifestyle interventions. The goal of this new Seminar is to offer readers the most current insights into Alzheimer's disease. [1] Alzheimer's disease (AD) is a progressive neurodegenerative condition and the most prevalent cause of dementia, particularly in older adults. It features a gradual decline in cognitive function, memory impairment, challenges with reasoning, language difficulties, and shifts in behavior.

The principal risk factor for Alzheimer's disease is age. The incidence of the disease doubles every 5 years after 65 years of age, with the diagnosis of 1275 new cases per year per 100,000 persons older than 65 years of age. Data on centenarians show that Alzheimer's disease is not necessarily the outcome of aging nevertheless, the odds of receiving the diagnosis of Alzheimer's disease after 85 years of age exceed one in three. As the aging population increases, the prevalence will approach 13.2 to 16.0 million cases in the United States by mid-century. [2]

History Of AD

In 1901, a 51-year-old woman named Auguste Deter was admitted to the City Asylum for the Insane and Epileptic, known as "Irrenschloss" or the Castle of the Insane, in Frankfurt am Main, Germany. Up until eight months before her admission, she had been living a normal life, married and seemingly well. However, she began experiencing troubling psychological and neurological issues, including memory and language difficulties, paranoia, disorientation, and hallucinations. Alois Alzheimer, a doctor at the asylum, took a particular interest in her case. He was intrigued by her age; while senile dementia was recognized at the time, it typically didn't manifest until people were in their early to mid-sixties. What made Auguste's situation even more remarkable was the swift progression of her dementia, which developed in just eight months from the onset of her first symptoms to her commitment. During one of his examinations, Alzheimer asked Ms. Deter to complete some simple writing tasks. When she struggled to even write her name, she poignantly remarked, "I have lost myself, so to speak" ("Ich habe mich sozusagen selbst verloren"). In 1902, Alzheimer left the Frankfurt hospital to work alongside Emil Kraepelin at the Psychiatric University Hospital in Heidelberg-

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Bergheim. By 1903, both he and Kraepelin had moved to Ludwig Maximilian University in Munich. After Ms. Deter passed away from septicemia on April 8, 1906, Alzheimer was notified, and her brain was sent to Munich for further study. Under the microscope, he discovered neurofibrillary tangles and clusters of beta-amyloid plaque—two key characteristics of the disease. On November 3, 1906, Alzheimer shared his findings about Auguste's case at the Conference of South-West German Psychiatrists in Tübingen, and he published his research in 1907. In 1910, Emil Kraepelin officially named the condition 'Alzheimer's disease.' Typically, this disease begins to affect individuals in their late 60s or early 70s**.** [3]

Signs and Symptoms of Alzheimer's Disease

Alzheimer's disease unfolds gradually, with symptoms that intensify as time goes on. Generally, these symptoms are divided into three stages: **early**, **middle**, and **late**.

1) **Early-Stage Symptoms (Mild)**
- **Memory loss**, especially recent events or conversations
- **Difficulty finding words** or naming familiar objects (anomia)
- **Misplacing items** (e.g., keys, wallet)
- **Trouble planning or solving problems** (e.g., difficulty following a recipe)

2) **Middle-Stage Symptoms (Moderate)**
- Increased memory loss and confusion
- Difficulty recognizing familiar people (e.g., family or friends)
- Problems with language (e.g., repeating phrases, struggling with vocabulary)
- Wandering and getting lost

3) **Late-Stage Symptoms (Severe)**
- Inability to communicate coherently
- **Loss of awareness** of surroundings and recent experiences
- Complete dependence on others for personal care
- Difficulty swallowing and eating

Although memory loss is the most commonly recognized early indicator, Alzheimer's disease gradually affects various cognitive, behavioral, and physical abilities. It's vital to identify and diagnose the condition early to manage symptoms effectively and ensure proper care planning.

Epidemiology

Global and regional prevalence:- Current Prevalence: In 2021, approximately 57 million people worldwide were living with dementia, with nearly 10 million new cases emerging annually

Forms of Dementia: Alzheimer's disease accounts for **60–70%** of dementia cases.

Death and Disability: Dementia is the **seventh leading cause of death** globally and a significant contributor to disability among older adults

Economic Impact: In 2019, dementia-related costs globally reached **US $1.3 trillion**, with half attributed to the informal care provided by families and friends

Gender Disparity: More women are affected than men, both in terms of cases and care roles

Back in 2005, a team of international experts brought together by Alzheimer's Disease International (ADI) came to a consensus about the prevalence of dementia across different WHO regions. Unfortunately, the quality and coverage of the evidence they found were lacking. There were only a handful of published studies from places like Latin America, Africa, the Middle East, Eastern Europe, and Russia, and the estimates from other less developed areas were often inconsistent and patchy. [4]

Dementia is becoming a significant public health issue worldwide, particularly in low- and middle-income countries. We're seeing regional increases linked to both an aging population and overall population growth, with notable differences across various areas. While the total number of cases is on the rise, the rate per capita remains steady, suggesting that demographic changes are the key factor. Women are hit harder by this condition, facing greater risks and shouldering more of the caregiver responsibilities. It's becoming increasingly crucial to adapt public health strategies to align with regional trends and demographics.

Age distribution and gender differences

Several studies indicate that women are more severely and frequently impacted by Alzheimer's disease (AD) compared to men. Yet, other research has not supported this view, making the topic a bit of a debate. The discrepancies often stem from different research approaches and the varying life expectancies of males and females. [5]

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Age is the strongest risk factor—incidence doubles approximately every 5 years after 65.
Women bear a disproportionate Alzheimer's burden, largely due to longer life needs but also influenced by biology and social determinants. Regional patterns vary, but a consistent trend is higher burden in women, especially in high-age brackets.

Pathophysiology of Alzheimer's disease (AD)

Amyloid-β plaques are a key feature of Alzheimer's disease and play a crucial role in what's known as the "amyloid cascade hypothesis." This theory proposes that the buildup of Aβ is one of the first steps, and perhaps even the trigger, in the development of Alzheimer's.

Amyloid-β plaques are deposits made up of misfolded protein fragments that form outside of cells and are crucial in the progression of Alzheimer's disease. Their buildup is one of the earliest signs we can detect in the brains of Alzheimer's patients and is thought to trigger a chain reaction of neurodegenerative events.

Aβ is a peptide that our bodies produce naturally throughout our lives, and amyloid plaques are a key sign of Alzheimer's disease. It's quite interesting that, despite being one of the most researched proteins, the normal role of APP remains a bit of a mystery. The potential normal function of Aβ is even less clear. However, we do know that synaptic activity—one of the most distinctive and essential functions of our nervous system actually boosts the production and release of Aβ. So, the creation of this small Aβ peptide, which can be up to 42 or 43 amino acids long, isn't necessarily harmful and might even serve a useful purpose in our bodies, even though amyloid plaques are often associated with toxicity. [6]

In the brain, amyloid-β plaques form when Aβ protein fragments misfold and accumulate outside of cells. These plaques are one of the two main indicators of Alzheimer's disease, alongside neurofibrillary tangles composed of tau protein.
Back in 1984, researchers discovered that Aβ was the main ingredient in the extracellular amyloid plaques found in Alzheimer's disease (AD), which is a key feature of the condition. Then, in 1992, Hardy and Higgins introduced the "amyloid cascade hypothesis." They suggested that the buildup of Aβ in the brain triggers the onset of AD, leading to the formation of tau tangles, loss of neurons, and cognitive decline.

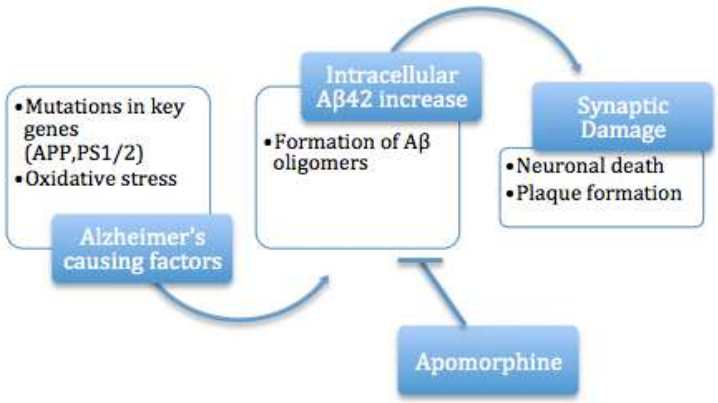

Figure 1 Amyloid-β plaques

Formation

According to the amyloid hypothesis, the key issue lies in the accumulation of extracellular amyloid beta (Aβ) deposits. Evidence for this theory comes from the positioning of the gene for the amyloid precursor protein (APP) on chromosome 21. People with trisomy 21, or Down syndrome, who have an extra copy of this gene, typically start showing early symptoms by the age of 40. Another important factor is a specific isoform of apolipoprotein called APOE4, which is a major genetic risk factor. While apolipoproteins usually aid in breaking down amyloid beta, some forms, like APOE4, are less effective, resulting in amyloid buildup in the brain. [7]

Tau protein aggregation:- Tau protein is at the heart of the neurodegeneration we see in Alzheimer's disease. While the accumulation of amyloid-β (Aβ) might initiate the disease, it's the tau pathology that has a stronger connection to the worsening and severity of cognitive decline.

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Diagnosis

When it comes to Alzheimer's Disease (AD), the clinical signs can include issues with memory and language, difficulties with spatial orientation, and challenges in higher-level thinking. On the noncognitive front, you might see personality shifts, reduced judgment, wandering tendencies, psychosis, mood fluctuations, agitation, and problems with sleep.

Clinical Evaluation

A. Medical & Neurological History

- Family history of AD or dementia
- Onset and progression of symptoms (e.g., memory loss, confusion)
- Medical conditions that can mimic dementia (e.g., depression, B12 deficiency)

B. Neuropsychological Testing

- Mini-Mental State Examination (MMSE)
- Montreal Cognitive Assessment (MoCA)
- Tests assess memory, attention, language, problem-solving, and orientation.

Brain Imaging

A. Structural Imaging

- MRI or CT scan
- Rule out strokes, tumors, or hydrocephalus
- Show brain atrophy (especially hippocampus and cortex)

B. Functional Imaging

- FDG-PET: Detects reduced glucose metabolism in the brain
- Amyloid PET scans: Visualizes amyloid plaques
- Tau PET scans: Detect abnormal tau protein build-up (research/advanced use)

Age Related Tau Pathologies

The amyloid hypothesis suggests that the buildup of extracellular amyloid beta (Aβ) is the primary culprit behind certain conditions. This idea is supported by the fact that the gene for the amyloid precursor protein (APP) is located on chromosome 21. Interestingly, individuals with trisomy 21, or Down syndrome, who have an extra copy of this gene, often show early symptoms by the time they reach 40. Additionally, a specific form of apolipoprotein known as APOE4 is a significant genetic risk factor. While apolipoproteins generally help break down amyloid beta, some variants, like APOE4, are not as effective, which can lead to an accumulation of amyloid in the brain [9]

The process of diagnosing Alzheimer's Disease (AD) is largely clinical and requires a thorough evaluation of the patient's cognitive abilities, medical history, and behavior. It usually starts with a detailed account from the patient and their close family or caregivers, focusing on how memory loss has developed and any changes in behavior, language, or daily activities. Cognitive assessments, such as the Mini-Mental State Examination (MMSE) or the Montreal Cognitive Assessment (MoCA), are used to gauge memory, attention, language, and problem-solving skills. To rule out other potential causes of dementia, laboratory tests are conducted, checking for issues like vitamin deficiencies or thyroid disorders. Brain imaging techniques, like MRI or CT scans, are employed to spot characteristic changes such as hippocampal atrophy and to exclude other structural brain diseases. In some instances, advanced methods like PET scans and cerebrospinal fluid (CSF) analysis are used to identify abnormal amyloid-beta and tau proteins, which are key markers of Alzheimer's pathology. The diagnosis is guided by criteria from the DSM-5 or the National Institute on Aging–Alzheimer's Association (NIA-AA), which consider both clinical features and, when available, biomarker evidence.

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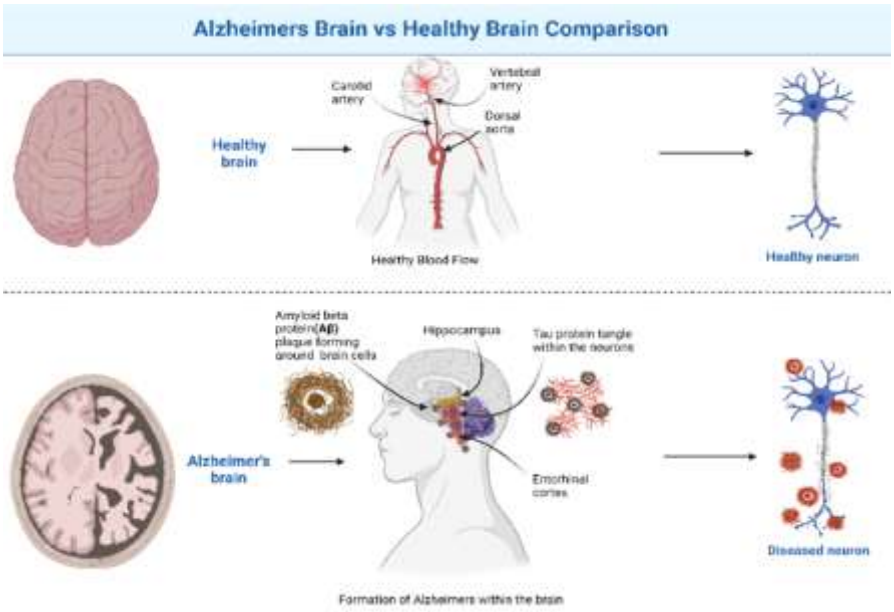

Figure 2 Healthy brain and Alzheimer's brain atrophy [10]

Pharmacological Treatment

Drug Class	Generic Name	Brand Name	Mechanism of Action	Indication/Stage	Common Side Effects
Cholinesterase Inhibitors	Donepezil	Aricept	Inhibits acetylcholinesterase, increasing acetylcholine levels	Mild, Moderate, and Severe AD	Nausea, diarrhea, insomnia, bradycardia
	Rivastigmine	Exelon	Inhibits acetylcholinesterase and butyrylcholinesterase	Mild to Moderate AD (also in patch form)	GI upset, weight loss, dizziness
	Galantamine	Razadyne	Inhibits acetylcholinesterase and modulates nicotinic receptors	Mild to Moderate AD	Nausea, vomiting, anorexia
NMDA Receptor Antagonist	Memantine	Namenda	Blocks NMDA receptors to reduce glutamate excitotoxicity	Moderate to Severe AD	Dizziness, confusion, headache
Combination Therapy	Donepezil + Memantine	Namzaric	Combines cholinesterase inhibition with NMDA antagonism	Moderate to Severe AD	Combination of above side effects
Anti-Amyloid Monoclonal Antibodies	Lecanemab (FDA approved)	Leqembi	Targets and clears amyloid-beta plaques from the brain	Early (mild) AD with confirmed amyloid pathology	ARIA (edema or microhemorrhages), infusion reactions
	Aducanumab *(limited use)*	Aduhelm	Reduces amyloid-beta plaques (controversial efficacy)	Early AD with amyloid confirmation	ARIA, headache, confusion

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Some of the usual functions of tau include cellular signaling, helping with neuronal development, providing neuroprotection, and regulating apoptosis. However, scientists are currently looking into some unusual roles for tau, such as its contribution to chromosome stability and its interactions with the cellular transcriptome.

Diagnostic Approaches:- Despite the many strides we've taken in medicine, figuring out how to diagnose Alzheimer's disease (AD) before someone passes away is still a significant hurdle for scientists. This challenge arises because the diagnosis is primarily based on clinical signs, making it tricky to differentiate AD from other neurodegenerative disorders. When we diagnose AD—whether it presents typically or atypically we're often working with probabilities. Therefore, it's crucial to identify the biological features of this disease to establish clear markers that reflect the neurological changes happening beneath the surface.

Currently Available Pharmacological Treatment-

Donepezil- Donepezil is a cholinergic medication derived from piperidine. It acts as a reversible, non-competitive inhibitor of acetylcholinesterase, the enzyme that breaks down acetylcholine. Additionally, donepezil influences the molecular and cellular processes involved in Alzheimer's disease. It helps to curb glutamate-induced excitotoxicity, decreases the early production of inflammatory cytokines, encourages a neuroprotective variant of AChE, and mitigates the impacts of oxidative stress. [11]

Donepezil is a type of cholinesterase inhibitor that's often prescribed for Alzheimer's disease (AD). It works by temporarily blocking the enzyme acetylcholinesterase, which leads to higher levels of acetylcholine in the brain. This boost helps improve communication between the nerve cells that are impacted by AD. Donepezil is approved for all stages of Alzheimer's—whether it's mild, moderate, or severe. Typically, treatment begins with a daily dose of 5 mg, and after a few weeks, this can be increased to 10 mg if the patient tolerates it well. In more severe cases, a higher extended-release dose of 23 mg might be an option.[12]

Rivastigmine- Rivastigmine is a type of carbamate derivative that works by reversibly inhibiting both acetyl- and butyryl-cholinesterase (AChE and BuChE, respectively). When taken orally, it reaches its highest concentration in the bloodstream within an hour. What's interesting is that this drug doesn't rely on cytochrome P450 isoenzymes for its metabolism, which helps reduce the risk of drug interactions. Numerous clinical trials indicate that rivastigmine can significantly enhance memory and other cognitive functions. In contrast to rivastigmine capsules, the patches have a notable impact on language abilities. These patches are particularly useful for treating patients with mild to moderate Alzheimer's disease. [13]

Rivastigmine is a cholinesterase inhibitor used to manage mild to moderate Alzheimer's disease, and also Parkinson's disease dementia. Like donepezil, it is a symptomatic treatment it does not cure AD but can help improve cognitive function temporarily.
Rivastigmine is a type of cholinesterase inhibitor that's commonly used to treat mild to moderate Alzheimer's disease. It works by blocking the action of two enzymes, acetylcholinesterase and butyrylcholinesterase, which are responsible for breaking down acetylcholine. By doing this, it boosts cholinergic transmission in the brain. This dual action might provide extra benefits for some patients compared to other cholinesterase inhibitors. You can find rivastigmine in oral capsules or as a transdermal patch, with the patch often being easier on the stomach due to fewer gastrointestinal side effects. Typically, the oral version starts at a low dose that's gradually increased to help reduce nausea, vomiting, and loss of appetitecommon side effects. The patch is applied once a day and offers a steady release of the medication, which can improve adherence and tolerability. While rivastigmine may lead to modest improvements or stabilization in memory, cognition, and daily activities, it's important to note that, like other drugs in its class, it doesn't stop the progression of Alzheimer's disease. It's also approved for treating dementia related to Parkinson's disease.

Galantamine

Galantamine is a selective alkaloid from the isoquinoline group that works as a competitive and reversible inhibitor of acetylcholinesterase. It also boosts the action of acetylcholine on nicotinic receptors. The FDA approved this drug in 2001. You can take it orally, with doses available in 4, 8, 12, 16, and 24 mg, either as a quick-release solution (twice a day) or as extended-release capsules (once a day). The initial recommended dose for treatment is 8 mg per day, which can be increased to a maintenance dose of 16 mg per day, taken twice daily after 4 to 8 weeks. What's really intriguing about galantamine in relation to Alzheimer's disease is its ability to work effectively in the central nervous system while having minimal effects on the peripheral system. [14]

Memantine

Memantine is known for its uncompetitive blockade of the NMDA receptor, which may help protect neurons from dying and improve symptoms by aiding the recovery of damaged neurons. It's usually prescribed starting at 5 mg per day, with weekly increases of 5 mg, capping out at 20 mg. Generally, it's well tolerated and has fewer side effects compared to cholinesterase inhibitors, though some people might experience dizziness, headaches, drowsiness, constipation, or high blood pressure. Studies indicate that memantine offers modest benefits for those with moderate to severe Alzheimer's disease, but there's limited evidence for its effectiveness in milder cases. Additionally, adding memantine to donepezil treatment can be beneficial for patients with mid-stage Alzheimer's or those showing cognitive decline. It's important to note that neither memantine nor donepezil is effective for mild cognitive impairment.[15]

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Natureal product in the management of AD

There's a growing interest in natural products for managing Alzheimer's disease (AD), thanks to their neuroprotective, anti-inflammatory, and antioxidant effects. Compounds from plants like curcumin (from turmeric), resveratrol (from grapes), ginkgo biloba extract, and ginsenosides (from ginseng) have been thoroughly researched for their potential to stop amyloid-beta aggregation, decrease oxidative stress, and influence the pathways that support neuronal health. These natural substances could play a role in slowing down AD progression or easing its symptoms by boosting cognitive function and minimizing neurodegeneration. Although clinical evidence is still emerging, these natural products provide a valuable complement to standard treatments and highlight the need for further exploration of phytochemicals in the safe and effective management of AD.

A number of medicinal plants have demonstrated neuroprotective, antioxidant, anti-inflammatory, or anti-cholinesterase properties that could be beneficial in managing symptoms of Alzheimer's disease or even slowing its progression. Although most of these plants aren't FDA-approved for Alzheimer's, they are extensively studied in the fields of ethnopharmacology and both preclinical and clinical research.

Ginkgo Biloba

Ginkgo biloba leaves have a long history in traditional medicine, often touted for their ability to boost memory and combat age-related decline. The leaf extracts are packed with phytochemicals, including flavonoids, organic acids, and terpenoids, all of which contribute to their neuroprotective properties. Research has shown that Ginkgo biloba extracts can help protect against β-amyloid-induced cytotoxicity, likely due to their ability to scavenge free radicals, mitigate mitochondrial dysfunction, activate JNK and ERK pathways, and prevent the death of neurons.[16]

Table3 Mechanism of Herbal Druds

Mechanism	Effect in AD Management
Antioxidant activity	Scavenges free radicals; protects neurons from oxidative damage
Anti-inflammatory effects	Reduces neuroinflammation linked to amyloid and tau pathology
Cholinesterase inhibition	Increases acetylcholine levels → may improve cognition
Inhibition of Aβ aggregation	May prevent amyloid-β plaque formation (in vitro evidence)
Enhances cognition & memory	Improves spatial learning and memory in animal studies
Neuroprotective gene expression	Modulates pathways related to apoptosis and neuron survival

Crocus Sativus

Recent research has suggested that water-soluble carotenoids, particularly crocins, play a significant role in the learning and memory processes associated with Crocus sativus L. extract. This study specifically looked at how crocins affect sporadic Alzheimer's disease, which was induced in male rats through intracerebroventricular (icv) administration of streptozocin (STZ). The findings indicate that crocin (30 mg/kg) effectively counteracts the cognitive deficits caused by STZ-icv in these rats, highlighting its potential as a treatment for neurodegenerative diseases like Alzheimer [17]

Strawberry

Strawberry intake by just one serving was linked to a 24% lower risk of developing Alzheimer's dementia. This finding was adjusted for factors like age, sex, education, physical activity, engagement in cognitive activities, Apo-ε4 status, and the consumption of other fruits, as well as total calorie intake. We also took into account other foods known to support better cognitive health, such as leafy greens and seafood. Even after these additional adjustments, the relationship between strawberry consumption and Alzheimer's risk remained significant (HR = 0.79, 95% CI: 0.62, 0.99). To explore whether this connection was influenced by dietary impacts on heart health, we also factored in conditions like hypertension, diabetes, and previous heart attacks. [18]

Green Tea (Camellia sinensis)

Green tea, which comes from the Camellia sinensis plant, has been found to have a protective effect on the brain in various neurodegenerative diseases, particularly in addressing memory loss related to Alzheimer's disease (AD). However, it's still unclear if other teas from Camellia sinensis, like red and black tea, offer the same benefits [19]

Natural products show great potential in managing Alzheimer's disease thanks to their diverse biological activities, which include antioxidant, anti-inflammatory, and neuroprotective effects. While they aren't a cure, compounds like curcumin, resveratrol, and ginkgo biloba might help slow down the progression of the disease and enhance quality of life when used alongside traditional treatments. That said, we still need more thorough clinical trials to confirm their effectiveness, safety, and the best dosages. Ongoing research into these natural products could pave the way for more effective and holistic treatment options for Alzheimer's.

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ARTIFICIAL INTELLIGENCE (AI) IN ALZHEIMER'S CARE

Area	AI Role
Early Detection	Brain imaging, biomarker analysis, cognitive pattern recognition
Diagnosis	Pattern recognition in clinical and imaging data
Treatment	Personalization, response prediction, digital therapeutics
Monitoring	Wearables, speech/activity tracking, telehealth integration
Caregiving	Virtual assistants, robotic help, safety monitoring
Research	Drug discovery, trial optimization, biomarker development

In the United States, as of 2020, around 5.8 million people were living with Alzheimer's disease (AD). This number is projected to nearly triple to 14 million by 2050. The rising prevalence of AD affects not just physical health, but also cognition, emotional well-being, and the ability to live independently. This situation highlights the urgent need for better care strategies.[20]

Deep learning (DL) can be a powerful tool for creating models that track how diseases progress. Unlike the early predictions of Alzheimer's disease (AD), which aim to identify it before it fully develops, the focus here is on patients who have already received a diagnosis. By using DL techniques, we can evaluate the level of cognitive decline in these individuals. Today, MRI scans are primarily used for diagnosing AD. In the realm of image processing, convolutional neural networks (CNNs) shine when it comes to analyzing neuroimaging data. They can extract crucial pathological features from these images, assisting doctors in monitoring how the disease is advancing in their patients.[21]

Artificial intelligence (AI) is stepping up as a vital force in the early detection, diagnosis, monitoring, and management of Alzheimer's disease (AD). With the help of machine learning and deep learning algorithms, AI can dive into extensive and complex datasets—such as brain imaging, genetic data, and electronic health records—to uncover patterns and biomarkers that signal the onset and progression of AD. AI-driven tools are being designed to catch subtle cognitive declines in speech, handwriting, or daily activities, which allows for earlier diagnoses than what traditional clinical methods can offer. Additionally, AI plays a crucial role in drug discovery by predicting how new compounds may influence AD-related proteins or pathways. In summary, AI holds significant promise for enhancing the accuracy, efficiency, and personalization of Alzheimer's care, but its successful integration into clinical practice will require ongoing validation, ethical considerations, and collaboration between technology and healthcare experts.

In recent years, artificial intelligence has undergone a remarkable transformation, largely thanks to the advent of machine learning and deep learning. These cutting-edge technologies are becoming more popular because they excel at analyzing vast datasets, making predictions, and uncovering insights that were once out of reach. With the ever-increasing volume of data and advancements in computing power, it's becoming more obvious that machine learning and deep learning have the potential to revolutionize different industries and change the world as we know it.[22]

AI, machine learning, and speech technologies

Technology	**Role in AD**
Machine Learning	Predict disease onset, analyze imaging/biomarkers, forecast progression
Speech & NLP	Detect early cognitive decline, monitor progression, support virtual screening
AI in Monitoring	Track behavioral/speech changes, provide passive and remote assessment
Virtual Assistants	Support independent living and memory
AI in Research	Optimize trials, identify drug targets, analyze big data

Use of AI in Drug Discovery

The integration of artificial intelligence (AI) into the realm of drug discovery for Alzheimer's disease has truly transformed the quest for effective treatments. It has made the drug development process not only faster but also more efficient. With the help of AI algorithms, researchers can sift through massive amounts of biological and chemical data to pinpoint potential drug candidates. These algorithms can even predict how these candidates will interact with key Alzheimer's-related targets like amyloid-beta and tau proteins, while also evaluating their safety and effectiveness

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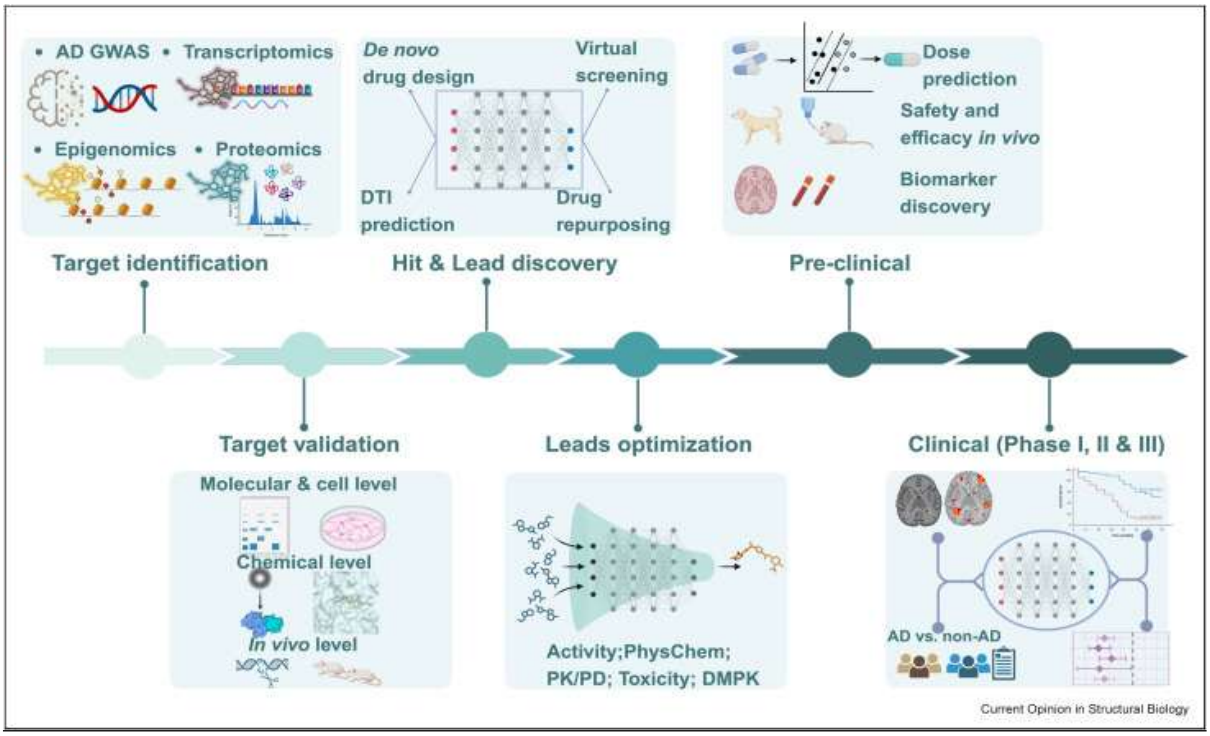

***Table 1*Artificial intelligence (AI) assistant drug discovery and development pipeline using Alzheimer's disease (AD) as a prototypical example[23]**

CAD Systems of Alzheimer's Disease

Tool/Platform	Key Features
Free Surfer	Brain image analysis and cortical reconstruction
ADNI (Alzheimer's Disease Neuroimaging Initiative)	Provides large datasets for training and testing
DeepAD	Deep learning-based AD diagnosis from MRI
SPM (Statistical Parametric Mapping)	Statistical analysis of brain imaging data
Clinica	Pipeline-based medical imaging analysis

Over the past twenty years, there's been a growing emphasis on developing strategies for Anti-Alzheimer's drug research. This shift likely stems from the fact that many cases of Alzheimer's remain largely mysterious, with only a handful of instances where genetic factors have been pinpointed. As the disease progresses, it brings about a range of symptoms, including cognitive decline, memory loss, unusual personality changes, confusion, aggression, mood swings, and irritability. Unfortunately, the current treatments available only provide symptomatic relief and don't address the underlying biololecular processes. Most therapies aimed at treating Alzheimer's focus on altering the amyloid cascade, which is believed to play a crucial role in the disease's development.[24]

Advantages of CAD for AD

1)Early and accurate diagnosis
2)Reduces diagnostic variability
3)Assists in clinical decision-making
3)Non-invasive and data-driven

Challenges

1)Data variability (different scanners, protocols)
2)Interpretability of AI models
3)Need for large annotated datasets
4)Regulatory approval and clinical integration

CONCLUSION

In conclusion, Alzheimer's disease is one of the most daunting neurodegenerative disorders, having a profound impact on individuals, families, and healthcare systems worldwide. Even though it's a complex challenge, the continuous progress in research—from early diagnostics and biomarker discovery to innovative treatment strategies and supportive technologies—provides hope for better management and a higher quality of life for patients. The integration of natural products, artificial intelligence, and

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personalized medicine is a promising avenue for more effective prevention, diagnosis, and treatment. Ongoing global collaboration, investment in research, and raising public awareness are vital to tackling this escalating health crisis and getting closer to a cure.
Alzheimer's disease is a progressive and challenging condition that poses a serious threat to global health. Although a cure is still out of reach, we've made great progress in understanding its mechanisms and in finding ways to detect, treat, and care for those impacted. New strategies, such as leveraging natural products, artificial intelligence, and precision medicine, are opening doors to more effective and personalized interventions. With continued research, innovation, and collaboration among scientists and healthcare professionals, there's hope for better outcomes, improved quality of life for patients and their caregivers, and even the potential to prevent or reverse this complex disease.

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