World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(REVIEW ARTICLE)

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The review on versatile study of Alzheimer's Disease (AD) and their etiology, pathophysiology, common mechanism of action of drugs used in current treatment

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World Journal of Advanced Research and Reviews, 2026, 29(02), 631-644

Publication history: Received on 31 December 2025; revised on 09 February 2026; accepted on 11 February 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.29.2.0330

Abstract

Alzheimer's complaint (announcement) is the most common habitual neurodegenerative complaint worldwide and a leading cause of madness. It results in cognitive impairments similar as aphasia and agnosia, along with behavioral and cerebral symptoms that place a significant emotional and fiscal burden on cases and their families. First described by Alois Alzheimer in 1907, announcement is classified into domestic and sporadic forms. presently, over 47 million people are affected encyclopedically, with figures anticipated to rise sprucely by 2050. The complaint is characterized by amyloid pillars and neurofibrillary befuddlements formed by amyloid- β and tau proteins. Announcement is multifactorial, with the cholinergic and amyloid suppositions being central to its pathogenesis. This review summarizes recent advances in announcement mechanisms, memory impairment, opinion, treatment strategies, and ongoing clinical exploration.

Keywords: Alzheimer's; Dementia; Cognitive Impairment; Tau Protein; Amyloid Beta

1. Introduction

Alzheimer's disease (AD) is the most common cause of dementia and is characterized by progressive memory loss and decline in cognitive functions. It accounts for nearly 60–80% of all dementia cases worldwide. AD is a neurodegenerative disorder in which nerve cells in the brain gradually lose their function and eventually die. Since neurons in the central nervous system cannot regenerate, the damage caused by AD is irreversible.

The disease was first described in 1907 by German psychiatrist Alois Alzheimer, who observed abnormal protein deposits and neuronal loss in the brain of a patient with memory and behavioral problems. These abnormalities include amyloid-beta plaques and neurofibrillary tangles formed by tau protein.

Alzheimer's disease develops slowly, often beginning years before symptoms appear, and mainly affects older adults. As the disease progresses, it severely interferes with daily activities, creating a major social, emotional, and economic burden worldwide.

1.1. Types of Dementia

There is a difference in the opinions amongst the scientists with regard to the categorization of Alzheimer's disease. Some of them consider it to be a factor that causes dementia, while others define the condition as a type of dementia. The latter opinion puts Alzheimer's alongside other types of dementia, including:

- Mild cognitive impairment (MCI)

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- Creutzfeldt-Jakob disease (CJD)
- Dementia with Lewy bodies (DLB)
- Vascular dementia
- Alcohol-related brain damage (ARBD)
- Young-onset dementia
- Frontotemporal dementia (FTD)
- HIV-related cognitive impairment

Alzheimer's, with the currently available knowledge, is classified into different subtypes according to severity, inflammatory response, and type of onset or trigger.

Figure 1 Alzheimer's disease (AD)

Aim and objectives

The review focus on the basis information about Alzheimer's disease (AD) and their etiology, Pathophysiology, common mechanism of action of drugs, drugs used in their current treatment.

2. Types Of Alzheimer's Disease

2.1. Classification Based on the Severity

Based on the intensity of the typical Alzheimer's symptoms, it can be classified into the following subtypes

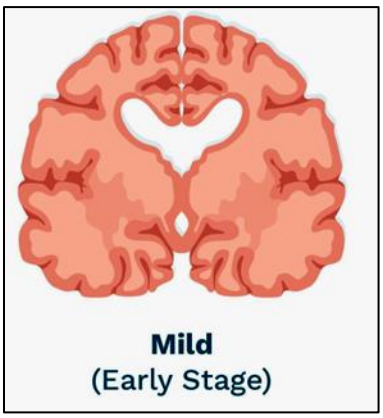

Figure 2 Early stages

2.2. Mild Alzheimer's

This includes the beginning of cognitive impairment that causes difficulties in remembering daily routines such as tasks at work, paying bills, and others. Because these symptoms are not very serious, the patients at this stage manage to remain functional with a certain amount of difficulty. They take longer to perform the same task which they used to do quicker before, and this becomes a pattern.

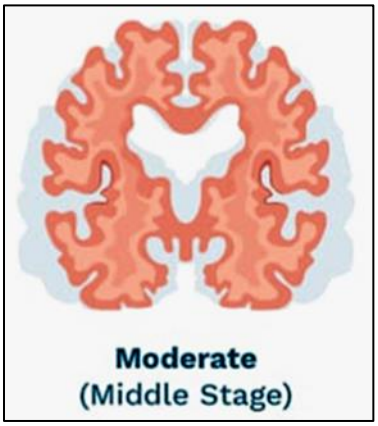

Figure 3 Moderate stage

2.3. Moderate Alzheimer's

Because of a significant amount of neuronal damage, the symptoms of moderate Alzheimer's are more intense. The confusion becomes worse and due to the amount of memory loss, they become increasingly dependent on others. These individuals, even though physically agile, are not able to perform routine tasks as the delusions take over the sensory processing of their thoughts.

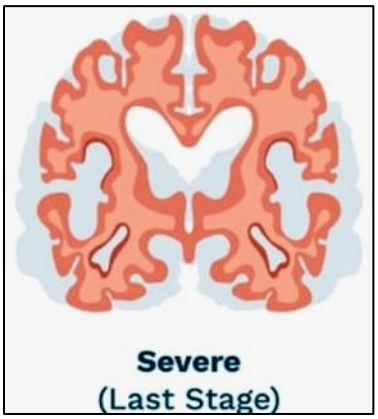

Figure 4 Last stage

2.4. Severe Alzheimer's

As the plaques and tangles spread, the brain cells start dying. This results in shrinkage of brain tissue. The patients with this condition are typically bedridden and are hardly able to communicate. These subtypes are more like stages of the disease, and it often progresses from a milder to a more severe form. The sooner the patient is diagnosed with the condition, the better are the chances of treating and preventing its progression.

2.5. Classification Based on the Inflammatory Response

Alzheimer's is categorized into three subtypes based on inflammatory response.

2.5.1. Inflammatory

In addition to the behavioral and cognitive symptoms, this subtype exhibits a high serum albumin-to-globulin ratio and a high level of C-reactive protein in response to neuroinflammation.

2.5.2. Non-inflammatory

This subtype of Alzheimer's does not exhibit elevated inflammatory biomarkers. However, other metabolic abnormalities are usually associated with this condition.

2.5.3. Cortical

A cortical subtype is caused by a deficiency of zinc throughout various regions of the brain. Even though there is no inflammatory response associated with this subtype, it causes abnormalities in normal brain functioning, which lead to Alzheimer's disease.

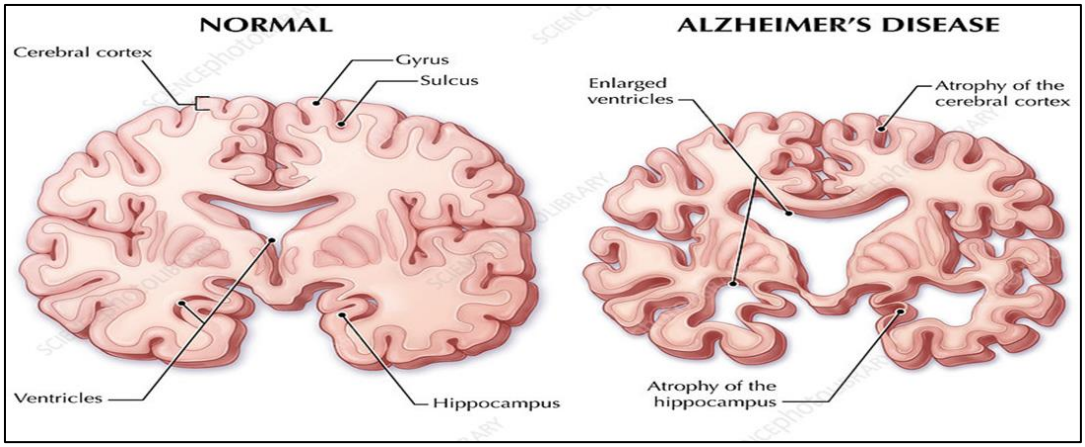

Figure 5 Difference between normal brain and disease affected brain

3. Classification Based on the Onset or Trigger Type

3.1. Early-Onset Alzheimer's

The subtype of Alzheimer's disease affecting people below 65 years of age is referred to as early-onset Alzheimer's. This condition is very rare (5 out of 100 Alzheimer's patients). The changes usually happen when the patients reach their late 40s or early 50s. Distinct features of this condition are considered the outcome of a defect in Chromosome 14.

3.2. Late-Onset Alzheimer's Disease

Late-onset Alzheimer's disease is the most common form of Alzheimer's, usually affecting individuals over 65 years of age. The exact genetic cause is still unknown; however, several risk factors such as age, lifestyle, and environmental influences have been identified, and research is ongoing to better understand the disease.

3.3. Risk Factors

Alzheimer's disease (AD) can be classified based on the age at which symptoms first appear. Early-onset AD occurs before the age of 65 and accounts for about 4–6% of cases, while late-onset AD affects individuals aged 65 years and older. These two forms differ in clinical presentation, neuropsychological features, and brain pathology. Genetic factors play a major role, particularly in early-onset AD, while late-onset AD is influenced by both genetic and environmental factors.

Diet is an important modifiable risk factor for AD. Diets rich in saturated and Transfats and low in antioxidants are associated with a higher risk of developing the disease. In contrast, the Mediterranean diet, which is rich in unsaturated fats, fruits, vegetables, and antioxidants, is linked to a reduced risk. Nutrients such as omega-3 fatty acids (DHA and EPA), vitamins B6, B12, C, and E, folate, selenium, and antioxidants support neuronal membrane integrity, reduce oxidative stress, and help protect against neurodegeneration.

3.3.1. Risk factors for Alzheimer's disease including following factors.

- Older age
- Family history and genetics
- Down syndrome

- Sex assigned at birth
- Mild cognitive impairment
- Head injury
- Air pollution
- Heavy alcohol use
- Poor sleep patterns
- Lifestyle and heart health

3.3.2. Hearing loss

- Vision loss that is not treated
- Lifelong learning and social engagement

3.4. Older age

The strongest known risk factor for Alzheimer's disease is getting older. Alzheimer's disease is not typical of aging. According to one study, there are four new diagnoses for every 1,000 people between the ages of 65 and 74. There were 32 new diagnoses for every 1,000 people aged 75 to 84. There were 76 new diagnoses for every 1,000 people over the age of 85.

3.5. Family History and Genetics

Having a first-degree relative with Alzheimer's disease increases an individual's risk, with genetic influences believed to be complex and multifactorial. The APOE ε4 allele is the strongest known genetic risk factor for late-onset AD, while rare mutations in three specific genes almost guarantee disease development but account for less than 1% of cases.

3.6. Down syndrome

Alzheimer's disease affects many people with Down syndrome. The presence of three copies of chromosome 21 is probably to blame for this. The gene on chromosome 21 is responsible for the production of the protein that causes beta-amyloid to form. In the brain, beta-amyloid fragments can form plaques. Compared to the general population, people with Down syndrome typically experience symptoms 10 to 20 years earlier.

3.7. Sex assigned at birth

Overall, there are more women with the disease because they tend to live longer than men.

3.7.1. Mild Cognitive Impairment (MCI)

Mild cognitive impairment involves noticeable decline in memory or thinking abilities beyond normal aging, while daily functioning remains largely intact. Individuals with MCI—especially memory-related MCI—have an increased risk of progressing to Alzheimer's disease, making early monitoring and lifestyle interventions important.

3.7.2. Head injury

Several large studies found that people age 50 or older who had a traumatic brain injury, also called TBI, had a higher risk of getting dementia and Alzheimer's disease. The risk is even higher in people with serious TBIs or multiple TBIs.

3.7.3. Air pollution

Studies in animals have found that air pollution particulates can speed the breakdown of the nervous system. Human studies have found that air pollution exposure especially from traffic exhaust and burning wood is linked to a higher risk of dementia.

3.7.4. Heavy alcohol use

Drinking large amounts of alcohol has long been known to cause brain changes. Several large studies and reviews found that alcohol misuse is linked to a higher risk of dementia, especially early-onset dementia.

3.7.5. Poor sleep patterns

Research has shown that poor sleep patterns, such as trouble falling asleep or staying asleep, are linked to a raised risk of Alzheimer's disease. Sleep apnea also may raise the risk of dementia.

3.7.6. Lifestyle and heart health

Research has shown that the same risk factors for heart disease also may increase the risk of dementia. It's not clear if these factors raise risk by worsening Alzheimer's changes in the brain or by leading to blood vessel changes in the brain. The factors include:

- Lack of exercise.
- Obesity.
- Smoking or exposure to secondhand smoke.
- High blood pressure.
- High cholesterol.
- Poorly managed type 2 diabetes.

High situations of low- viscosity lipoprotein, known as LDL, cholesterol in middle age, in particular, raise the threatof madness. Research has set up that people youngish than 65 with high LDL cholesterol situations have anadvanced threatof madness. But taking drugs to lower LDL cholesterol did not raise the threat. These factors can all be modified, so changing life habits can to some degree alter your threat. For illustration, regular exercise and a healthy low- fat diet rich in fruits and vegetables are related to a lower threat of Alzheimer's complaint.

3.8. Hearing loss

Studies have found that people who have hearing loss are at risk of dementia. The worse the hearing loss, the higher the risk. However, wearing hearing aids protects against getting dementia.

3.8.1. Vision loss that is not treated

Newer research suggests vision loss that isn't treated is a risk factor for cognitive impairment and dementia. The link may be due to a disease such as diabetes that can increase the risk of both vision loss and dementia. But some research suggests vision loss itself may increase the risk of dementia.

3.8.2. Lifelong learning and social engagement

Studies have found that being social and doing activities that stimulate the mind throughout life can lower the risk of Alzheimer's disease. Low education levels — less than a high school education — appear to be a risk factor for Alzheimer's disease.

3.9. Complications

Alzheimer's disease can lead to a variety of complications. Symptoms such as memory loss, language loss, impaired judgment and other brain changes can make it harder to manage other health conditions. A person with Alzheimer's disease may not be able to.

- Tell someone about being in pain.
- Explain symptoms of another illness.
- Follow a treatment plan.
- Explain medicine side effects.

As Alzheimer's disease moves into its last stages, brain changes begin to affect physical functions. The changes can affect the ability to swallow, balance, and manage stool and bladder movements. These effects can lead to other health issues such as

- Inhaling food or liquid into the lungs.
- Flu, pneumonia and other infections.
- Falls.
- Fractures.
- Bedsores.
- Poor nutrition or dehydration.
- Constipation or diarrhea.

4. Sign Symptoms of Alzheimer's Disease Include

4.1. Memory loss

- A person may have difficulty taking in new information and remembering information. This can lead to:
- Repeating questions or conversations
- Losing objects
- Forgetting about events or appointments
- Wandering or getting lost

4.1.1. Cognitive deficits

A person may experience difficulty with reasoning, complex tasks, and judgment. This can lead to:

- A reduced understanding of safety and risks
- Difficulty with money or paying bills
- Difficulty making decisions
- Difficulty completing tasks that have several stages, such as getting dressed

4.1.2. Problems with recognition

A person may become less able to recognize faces or objects or less able to use basic tools, even if they can see them clearly.

4.1.3. Problems with spatial awareness

A person may have difficulty with their balance, trip over, or spill things more often, or they may have difficulty orienting clothing to their body when getting dressed.

4.1.4. Problems with speaking, reading, or writing

A person may develop difficulties with thinking of common words, or they may make more speech, spelling, or writing errors.

4.1.5. Personality or behavior changes: A person may experience changes in personality and behavior that include

- Becoming upset, angry, or worried more often than before
- A loss of interest in or motivation for activities they usually enjoy
- A loss of empathy
- Compulsive, obsessive, or socially inappropriate behavior

4.2. Diagnostic processes

Alzheimer's disease (AD) is both a clinical and neuropathological disorder, with definitive diagnosis possible only through brain biopsy or autopsy. Clinically, AD is diagnosed with high accuracy using established criteria for probable and possible AD, based on patient history, cognitive decline, and neurological examination, while excluding other causes of memory loss. Differential diagnosis includes reversible conditions such as delirium and depression, as well as other dementias.

Other causes of dementia include vascular dementia, Parkinson's disease, dementia with Lewy bodies, front temporal dementia, prion diseases, and structural or metabolic brain disorders. Careful clinical evaluation is essential, as several conditions can mimic or coexist with AD, and early, accurate diagnosis helps guide appropriate management and treatment strategies.

4.3. Volumetric Data

- MRI-based volumetric analysis measures volume changes in specific brain regions to help predict progression from mild cognitive impairment to Alzheimer's disease. Hippocampal atrophy is a key biomarker, but due to limited sensitivity, volumetric MRI supports diagnosis rather than confirming AD on its own.

4.3.1. Diffusion Tensor Imaging (DTI)

- Diffusion tensor imaging assesses the diffusion of water molecules to evaluate micro structural changes in brain white matter and cortical mini columns. Alterations detected by DTI progress from normal aging to MCI and Alzheimer's disease, making it a useful marker of neurodegeneration.

4.3.2. PET Scan

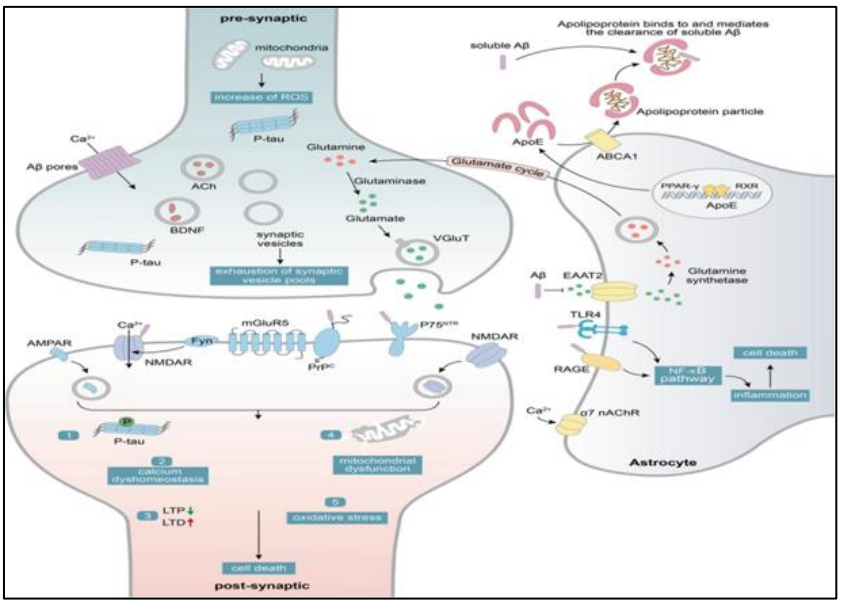

Figure 6 Neuroinflammation

- PET scans detect the accumulation of amyloid-β and hyperphosphorylated tau in the brain, serving as reliable biomarkers for Alzheimer's disease. Amyloid deposition occurs before noticeable cognitive decline, while tau buildup correlates with disease progression, aiding diagnosis and monitoring.

4.4. Pathophysiology (Summarized)

4.4.1. Cholinergic Hypothesis

Loss of cholinergic neurons results in decreased acetylcholine levels, impairing memory and cognition.

4.4.2. Amyloid Hypothesis

Excessive accumulation of amyloid-β peptides initiates neuronal damage and synaptic dysfunction.

4.4.3. Tau Hypothesis

Hyperphosphorylated tau forms neurofibrillary tangles, leading to neuronal death.

4.4.4. Neuroinflammation

Activated microglia and astrocytes release pro-inflammatory cytokines, exacerbating neuronal injury.

4.5. Oxidative Stress

Increased ROS production damages neuronal membranes, proteins, and DNA.

4.5.1. Metal Ion Dishonesties

Abnormal iron, copper, and zinc levels contribute to oxidative stress and protein aggregation.

4.5.2. Glutamatergic Excitotoxicity

Over activation of NMDA receptors leads to calcium overload and neuronal death.

4.5.3. Gut-Brain Axis

Micro biotic imbalance induces systemic inflammation affecting brain health.

4.5.4. Abnormal Autophagy

Defective clearance of mis folded proteins promotes amyloid and tau accumulation.

4.6. Diagnosis

- Clinical history and neurological examination
- Neuropsychological testing
- MRI (hippocampal atrophy)
- PET scans (amyloid and tau imaging)
- CSF biomarkers (Aβ42, P-tau)
- Blood-based biomarkers (emerging)
- Definitive diagnosis requires post-mortem histopathology.

Table 1 AD current treatment approaches in clinical trials approved drug

Agent	Therapeutic purpose	CADRO target	Mechanism of action	Clinical trial	Date of initiation	Estimated primary completion date
ALN-APP	Disease - modifying biologic	Amyloid beta	RNAi to decrease APP and downstream Aβ-related events	NCT05231785	Feb 2022	Jul 2025
ALZ-101	Disease - modifying biologic	Amyloid beta	Amyloid beta-directed vaccine	NCT05328115	Sep 2021	Dec 2023
APNmAb005	Disease - modifying biologic	Tau	Anti-tau antibody	NCT05344989	May 2022	Mar 2024
AV-1959	Disease - modifying biologic	Amyloid beta	Anti-amyloid vaccine	NCT05642429	Feb 2023	Feb 2026
Bacillus Calmette Guerin	Disease - modifying biologic	Inflammation	Vaccine to stimulate resilience to Alzheimer-related processes	NCT06078891	Jul 2023	Jul 2024
BMS-984923	Disease - modifying small molecule	Amyloid beta	Silent allosteric modulator (SAM) of mGluR5	NCT05804383 NCT05817643	Mar 2023 Jan 2023	Oct 2024 Feb 2023
Cannabidiol	Neuropsychiatric symptom	Neurotransmitter	Cannabinoid	NCT04075435	Jan 2021	Sep 2024
Centellaasiatica product	Disease - modifying small molecule	Synaptic Plasticity /Neuroprotection	Antioxidant anti-inflammatory agent with synaptic and neuroprotective effects	NCT05591027	Dec 2022	Nov 2024

Choline	Disease - modifying small molecule	Metabolism and Bioenergetics	Stabilizes the lipid metabolism and concomitantly restoring normal cell function by increasing phosphatidylcholine activity via the Kennedy pathway	NCT05880849	Jun 2023	Jun 2025
CpG1018	Disease - modifying biologic	Inflammation	Toll-like receptor nine agonist leading to reduced Aβ plaques and tau pathology	NCT05605414	Mar 2023	Nov 2024
CS6253	Disease - modifying biologic	ApoE, Lipids and Lipoprotein Receptors	Adenosine triphosphate-binding cassette transporter A1 (ABCA1) transfers lipids to ApoE, and increases clearance of A-beta from the brain	NCT05965414	Oct 2023	Sep 2024

Table 2 AD current treatment approaches in clinical trials approved drug

Agent	**Therapeutic purpose**	**CADRO TARGET**	**Mechanism of action**	**Clinical trial**	**Start date**	**Estimated primary completion date**
IBC-Ab002	Disease - modifying biologic	Inflammation	Anti-programmed death-ligand 1 (PD-L1) Immune checkpoint inhibitor	NCT05551741	Feb 2023	Oct 2024
LX1001	Disease - modifying biologic	Ape, Lipids and Lipoprotein Receptors	Adeno-associated virus (AAV) gene transfer vector expressing the cDNA coding for human apolipoprotein E2 (APOE2) directly to the CNS/CSF of APOE4 homozygotes	NCT05400330	May 2023	Nov 2028
Muramyl amine	Cognitive enhancement	Neurotransmitter Receptors	Nicotinic antagonist	NCT04129060	Mar 2020	Mar 2024
MK- 2214	Disease - modifying biologic	Tau	Anti-tau monoclonal antibody	NCT05466422	Sep 2022	May 2025
Nicotinamide Riboside	Disease - modifying small molecule	Metabolism and Bioenergetics	Mitochondrial function enhancer and antioxidant	NCT04430517	Mar 2022	Apr 2025

NIO752	Disease - modifying biologic	Tau	Anti-tau antisense oligonucleotide	NCT05469360	Feb 2023	Oct 2024
OLX-07010	Disease - modifying small molecule	Tau	Inhibits tau self-aggregation	NCT05696483	Jan 2023	Dec 2024

Table 3 AD current treatment approaches in clinical trials approved drug

AGENT	**THERAPEUTIC PURPOSE**	**CADRO target**	**MECHANISM OF ACTION**	**CLINICAL TRIAL**	**START DATE**	**ESTIMATED PRIMARY COMPLETION DATE**
50561	Disease - modifying small molecule	Synaptic plasticity/ neuroprotection	RAC1inhibitor (RACfamily small GTPase inhibitors) Enhance dendritic spine morphogenesis and synaptic plasticity	NCT05811442	Apr 2023	May2024
ABBV-552	Disease - modifying small molecule	Synaptic plasticity/ neuroprotection	Synapticvesicleglycoprotein2 A(SV2A) modulator	NCT0577148	Apr 2023	Jun2024
ABBV-916	Disease - modifying biologic	Amyloid beta	Anti-amyloid antibody	NCT0529124	Aug 2022	Jan2030
ACI-24.066	Disease - modifying biologic	Amyloid beta	Vaccine stimulates antibodies against amyloid beta protein	NCT05462106	Jun 2022	Jun2026
AL002	Disease - modifying biologic	Inflammation	Monoclonal antibody targeting TREM2 receptors	NCT04592874 NCT05744401	Jan2021 Jan2023	Sep2024 Sep2025
Allopregnanolone	Disease - modifying small molecule	Neurogenesis	Allosteric Modulator of GABA-A Receptors	NCT0483831	Aug 2023	Apr 2025
ALZN002	Disease - modifying biologic	Amyloid beta	AutologousBeta-AmyloidMutant Peptide-pulsedDendriticCells	NCT0583426	Jul2023	Mar2028
APH-1105	Disease - modifying small molecule	Amyloid beta	Alpha-secretase modulator (amyloidprecursorprotein secrete as modulator)	NCT0380648	Jun 2023	Sep2024
Astragalus	Cognitive enhancement	Inflammation	Undisclosed	NCT0564743	Feb 2024	May2025

Bacillus Calmette-Guerin	Disease-modifying biologic	Inflammation	Vaccine to stimulate resilience toAlzheimer-related processes	NCT05004 68	Mar 2022	Oct 2023
Baricitinib	Disease - modifying small molecule	Inflammation	Janus kinase (JAK)inhibitor	NCT05189 16	Dec 2022	Jul2024
Bepranemab	Disease-modifying biologic	Tau	Anti-tau monoclonal antibody binding to central region of tau	NCT04867 66	Jun 2021	May2024
BIIB080	Disease-modifying biologic	Tau	Anti sense oligonucleotide that inhibits translation of tau mRNA into the tau protein	NCT05399 88	Aug 2022	Nov2027
Brivaracetam	Cognitive enhanceme nt	Neurotransmi tter receptors	Anticonvulsant with high affinity for synaptic vesicle protein 2A	NCT05899 74	Jun 2023	Jun2028
Bumetanide	Disease - modifying small molecule	ApoE, lipids and lipoprotein receptors	Reversal of ApoE-specific AD signatures	NCT06052 13	Oct 2023	Oct 2025
Canakinumab	Disease-modifying biologic	Inflammation	Anti-IL-1-beta monoclonal antibody	NCT04795 46	Oct 2021	Mar2024

Table 4 AD current treatment approaches in clinical trials approved drug

Agent	**Therapeutic purpose**	**CADRO target**	**Mechanism of action**	**Clinical trial**	**Start date**	**Estimated primary completion date**
Cannabidiol	Neuropsychiatric symptom	Neurotransmitter receptors	Endo cannabinoid receptor agonist	NCT058223 62	Jan2024	Apr2028
Chinese Traditional Medicine	Cognitive enhancement	Metabolism andbioenergetics	Three herbs (Rhizoma Acori Tatarinowii, Poriacum Radix Pini, Radix Polygalae); mechanism unknown	NCT055385 07	Jun2022	Jun2024
CORT108297	Cognitive enhancement	Growth factors and hormones	Selective glucocorticoid receptor antagonist	NCT046010 38	Jun2021	Jun2025

CST-2032	Cognitive enhancement	Neurotransmitter receptors	Noradrenergic agonist.	NCT05104463	Apr2022	Nov2023
Dalzanemdor	Disease-modifying small molecule	Synaptic plasticity/neuroprotection	Enhances synaptic function through NMDA receptor blockade	NCT05619692	Dec2022	Dec2024
Dasatinib+ Quercetin	Disease-modifying small molecule	Inflammation	Dasatinizb induces apoptosis in senescent cells to allow their removal; quercetin is a flavonoid	NCT04685590 NCT04785300 NCT05422885	Dec2021 Jul2022 May2022	Jan2025 Dec2023 Jun2024
Dexmedetomidine	Neuropsychiatric symptom	Neurotransmitter receptors	Presynaptic alpha-2 adrenoceptor agonist to inhibit release of nor epinephrine	NCT06052254	Dec2023	Dec2024
Dronabinol+ PEA	Neuropsychiatric symptom	Neurotransmitter receptors	Cannabinoid	NCT05239390	Dec2021	Jun2023
E2814	Disease-modifying biologic	Tau	Anti-tau monoclonal antibody	NCT04971733	Jan2021	Mar2025

4.7. Treatment Strategies

- Currently Approved Drugs
- Cholinesterase inhibitors: Donepezil, Rivastigmine, Galantamine
- NMDA receptor antagonist: Memantine
- Disease-Modifying Therapies (Under Trial)
- Anti-amyloid monoclonal antibodies
- Anti-tau therapies
- Anti-inflammatory agents
- Neuroprotective compounds
- Gene and RNA-based therapies
- Non-Pharmacological Approaches
- Cognitive stimulation
- Physical exercise
- Dietary interventions
- Social engagement

4.8. Complications

- Infections (pneumonia)
- Malnutrition and dehydration
- Falls and fractures
- Bedsores
- Complete dependency

5. Conclusion

Alzheimer's disease is a complex, multifactorial neurodegenerative disorder with no definitive cure. Early diagnosis, lifestyle modification, and symptomatic treatment remain the cornerstone of management. Advances in biomarker research and disease-modifying therapies offer hope for future interventions that may delay or prevent disease progression.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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