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MODERN APPROACHES IN THE TREATMENT AND MANAGEMENT OF ALZHEIMER'S DISEASE: FROM PATHOGENESIS TO THERAPY

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ABSTRACT

Herein, Alzheimer's disease (AD) is a progressive neurodegenerative disorder and the most common cause of dementia worldwide, characterized by gradual memory loss, cognitive impairment, and behavioural disturbances. The global prevalence of Alzheimer's disease is increasing rapidly due to aging populations, creating a significant medical, social, and economic burden. The pathological hallmarks of AD include the accumulation of extracellular amyloid-β plaques, intracellular neurofibrillary tangles composed of hyperphosphorylated tau protein, synaptic dysfunction, and progressive neuronal loss. Despite decades of research, effective disease-modifying therapies remain limited, and currently available treatments primarily provide symptomatic relief rather than halting disease progression. Recent years have witnessed significant advances in understanding the molecular mechanisms underlying Alzheimer's disease, leading to the development of novel therapeutic strategies. Modern approaches in AD management focus not only on targeting classical pathological pathways such as amyloid and tau aggregation but also on addressing neuroinflammation, oxidative stress, mitochondrial dysfunction, and synaptic degeneration. In addition, emerging therapeutic modalities including monoclonal antibodies, small-molecule inhibitors, gene-based therapies, and neuroprotective agents are being actively investigated. Alongside pharmacological interventions, non-pharmacological strategies such as cognitive training, lifestyle modifications, dietary interventions, and digital therapeutics are increasingly recognized as important components of comprehensive disease management. This review aims to provide a comprehensive overview of the current understanding of Alzheimer's disease pathogenesis and highlight recent advances in therapeutic strategies for its treatment and management. Particular emphasis is placed on emerging drug targets, innovative treatment approaches, and integrated management strategies that may contribute to improved clinical outcomes. Furthermore, the review discusses ongoing challenges and future perspectives in Alzheimer's research, emphasizing the need for multidisciplinary approaches to develop more effective and disease-modifying therapies.

KEYWORDS: Alzheimer's disease, Neurodegeneration, Amyloid-beta plaques, Tau protein pathology, Monoclonal antibody therapy, Biomarkers, Artificial intelligence in diagnosis, Neuroprotective strategies.

1. INTRODUCTION

Alzheimer's Disease is a progressive neurodegenerative disorder that primarily affects memory, thinking ability, and behavior. It is considered the most common cause of dementia, especially among the elderly population. The disease gradually damages neurons in the brain, leading to cognitive decline, memory loss, impaired judgment, and difficulty in performing daily activities.[1] As the condition progresses, patients experience severe neurological impairment, which ultimately affects their independence and quality of life. Due to its chronic and irreversible nature, Alzheimer's disease has become a major concern for healthcare systems and researchers worldwide. Globally, the prevalence of Alzheimer's disease has increased significantly in recent decades, mainly due to the aging population and improved life

expectancy.[2] Millions of individuals worldwide are currently affected by this disorder, and the number is expected to rise rapidly in the coming years. The increasing burden of Alzheimer's disease not only affects patients but also places significant emotional, social, and economic pressure on caregivers, families, and healthcare infrastructures. Therefore, understanding the disease and developing effective management strategies has become a critical priority in modern medical research. The pathological characteristics of Alzheimer's disease are mainly associated with two hallmark features in the brain: amyloid plaques and neurofibrillary tangles. Amyloid plaques are extracellular deposits primarily composed of amyloid-beta peptides that accumulate between neurons and disrupt cell communication. In contrast, neurofibrillary tangles are formed due to abnormal aggregation of hyperphosphorylated tau protein inside neurons.[3] These pathological changes lead to neuronal dysfunction, synaptic loss, and progressive brain atrophy. In recent years, the development of modern therapeutic strategies has become an important focus in Alzheimer's research. Advances in pharmacological therapies, monoclonal antibodies, neuroprotective agents, and lifestyle-based interventions have provided new possibilities for improving disease management. These modern approaches aim not only to alleviate symptoms but also to target the underlying disease mechanisms and slow disease progression.[4] The aim of this review is to provide a comprehensive overview of the current understanding of Alzheimer's disease and highlight modern approaches used in its treatment and management. This review also discusses recent advances in therapeutic strategies and explores potential future directions for improving patient outcomes.[5]

Figure 1: Overview of Alzheimer's Disease as a Progressive Neurodegenerative Disorder.

1. *Overview of Alzheimer's Disease as a Progressive Neurodegenerative Disorder*

Alzheimer's disease is a chronic and progressive neurodegenerative disorder that primarily affects cognitive functions such as memory, reasoning, learning ability, and decision-making. It is widely recognized as the most common cause of dementia among elderly individuals and represents a major public health concern worldwide.[6] The disease is characterized by gradual neuronal degeneration in different regions of the brain, particularly in areas responsible for memory and cognitive processing such as the hippocampus and cerebral cortex. As the disease progresses, patients experience a continuous decline in cognitive abilities, which eventually interferes with their ability to perform everyday tasks. In the early stages, individuals may experience mild memory loss, difficulty in recalling recent events, or challenges in organizing daily activities. However, as the disease advances, symptoms become more severe and may include confusion, language impairment, personality changes, and behavioral disturbances. In the later stages, patients may lose the ability to communicate effectively, recognize family members, or maintain independent living.[7] The progressive nature of Alzheimer's disease results from the gradual damage and death of neurons within the brain. This neuronal loss disrupts communication between brain cells, leading to impairment in cognitive functions. Over time, the brain undergoes structural changes such as shrinkage and loss of neural connections. These changes contribute to the severe neurological deficits observed in advanced stages of the disease. Because Alzheimer's disease develops slowly over several years, early detection and proper management are essential for improving patient outcomes. Researchers and clinicians continue to investigate the underlying biological mechanisms of the disease in order to develop more effective therapeutic

strategies. Understanding Alzheimer's disease as a progressive neurodegenerative disorder is therefore crucial for the development of improved diagnostic tools, treatment options, and preventive approaches.[8]

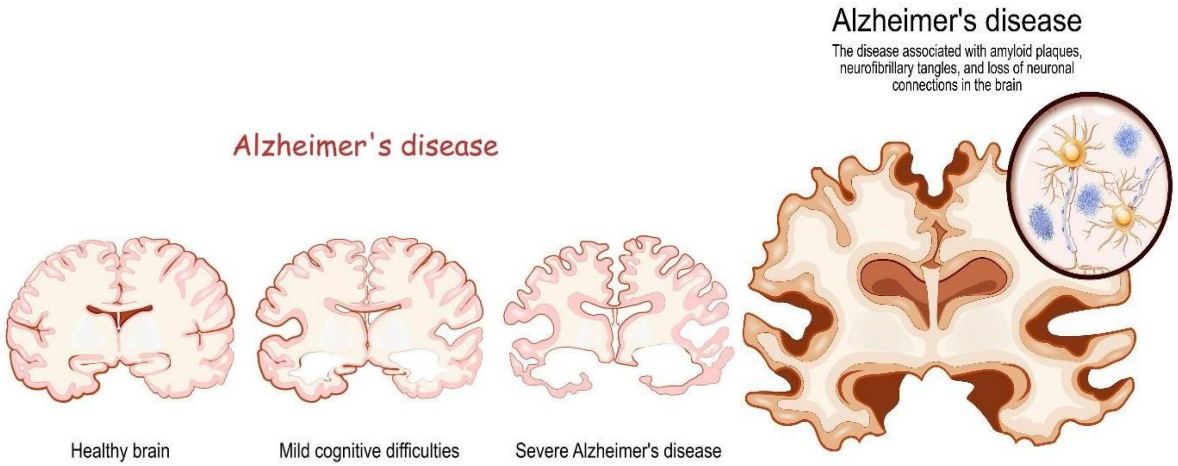

Figure 2: Spread of Alzheimer's disease.

2. ***Global Prevalence and Increasing Burden***

The global prevalence of Alzheimer's Disease has increased dramatically over the past few decades, largely due to the rapid growth of the aging population and increased life expectancy. Alzheimer's disease currently represents one of the most significant neurological health challenges worldwide. According to recent epidemiological studies, millions of people are living with Alzheimer's disease and other forms of dementia, and this number is expected to rise substantially in the coming years.[9] The burden of Alzheimer's disease extends beyond the affected individuals and significantly impacts families, caregivers, and healthcare systems. Patients with Alzheimer's disease require long-term medical care, psychological support, and assistance with daily activities. As the disease progresses, the level of care required increases considerably, often placing emotional and financial stress on caregivers and family members. In addition to personal and social impacts, Alzheimer's disease also imposes a substantial economic burden on healthcare systems.[10] The cost of treatment, long-term care, and support services for individuals with Alzheimer's disease continues to rise globally. Many countries are facing increasing healthcare expenditures due to the growing number of patients requiring specialized neurological care. Addressing this growing burden requires collaborative efforts in research, healthcare policy, and community awareness. Improved diagnostic techniques, effective treatment strategies, and preventive measures are essential to reduce the global impact of Alzheimer's disease and improve the quality of life of affected individuals.[11]

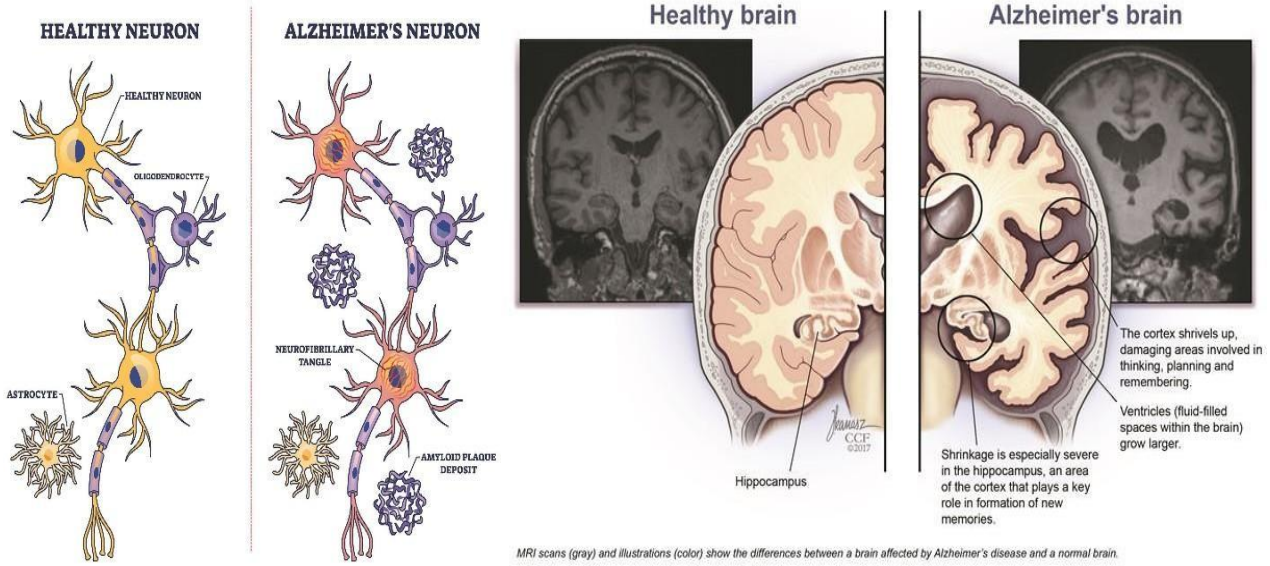

Figure 3: Alzheimer's disease improve In the patient.

3. ***Amyloid Plaques and Tau Tangles***

The pathological characteristics of Alzheimer's Disease

are mainly defined by the presence of two hallmark abnormalities in the brain: amyloid plaques and neurofibrillary tangles. These pathological features play a central role in the development and progression of the disease and are widely used as diagnostic markers in Alzheimer's research. Amyloid plaques are extracellular deposits composed primarily of aggregated amyloid-beta peptides.[12] These peptides are derived from the cleavage of a larger protein known as amyloid precursor protein (APP). In Alzheimer's disease, abnormal processing of APP leads to excessive accumulation of amyloid-beta peptides in the brain. Over time, these peptides aggregate and form insoluble plaques between neurons.[13] The accumulation of amyloid plaques interferes with neuronal communication, disrupts synaptic function, and triggers inflammatory responses that contribute to neuronal damage. Another important pathological feature of Alzheimer's disease is the formation of neurofibrillary tangles.[14] These tangles consist of abnormal aggregates of a protein known as tau. Under normal conditions, tau proteins help stabilize microtubules within neurons, which are essential for maintaining the structural integrity and transport systems of the cell. However, in Alzheimer's disease, tau proteins become abnormally phosphorylated, causing them to detach from microtubules and aggregate into twisted filamentous structures inside neurons.[15] The presence of these tangles disrupts the internal transport system of neurons, ultimately leading to neuronal dysfunction and cell death. The combined effects of amyloid plaque accumulation and tau tangle formation contribute to progressive neuronal degeneration, synaptic loss, and brain atrophy observed in Alzheimer's disease. Understanding these pathological mechanisms has been crucial for the development of modern therapeutic strategies aimed at targeting amyloid and tau pathways in Alzheimer's treatment.[16]

4. *Importance of Modern Therapeutic Approaches*

The increasing prevalence and complex pathology of Alzheimer's Disease have highlighted the urgent need for effective therapeutic strategies. Traditional treatment options have mainly focused on symptomatic relief rather than addressing the underlying causes of the disease. Although these treatments may temporarily improve cognitive symptoms, they do not significantly slow the progression of neuronal degeneration.[17] As a result, researchers have increasingly focused on developing modern therapeutic approaches that target the fundamental biological mechanisms involved in Alzheimer's disease. Modern therapeutic strategies aim to interfere with key pathological processes such as amyloid-beta accumulation, tau protein aggregation, oxidative stress, and neuroinflammation. Advances in molecular biology and neuroscience have led to the development of targeted therapies, including monoclonal antibodies designed to remove amyloid plaques from the brain. These therapies represent a promising direction for modifying disease progression rather than merely treating symptoms. In addition to pharmacological treatments, modern approaches also emphasize the importance of non-pharmacological interventions.[18] Lifestyle modifications such as regular physical activity, balanced nutrition, cognitive stimulation, and proper sleep patterns have been shown to play a supportive role in maintaining brain health and reducing the risk of cognitive decline. Furthermore, advances in digital health technologies, including artificial intelligence-based diagnostic tools and biomarker-based detection methods, are improving early diagnosis and disease monitoring. The integration of these modern therapeutic approaches has the potential to significantly improve patient outcomes. By targeting multiple aspects of the disease process, researchers hope to develop more effective treatment strategies that can delay disease progression and enhance the quality of life for patients living with Alzheimer's disease.[19]

5. *Aim and Scope of the Review*

The growing impact of Alzheimer's Disease on global health has made it essential to continuously evaluate current research developments and therapeutic strategies. Over the past few decades, significant progress has been made in understanding the molecular mechanisms, pathological features, and clinical manifestations of Alzheimer's disease. These advances have opened new opportunities for the development of innovative treatment approaches aimed at improving disease management and patient outcomes.[20] The primary aim of this review is to provide a comprehensive overview of Alzheimer's disease with particular emphasis on modern treatment and management strategies. This review highlights recent scientific developments in pharmacological therapies, emerging drug targets, and innovative therapeutic approaches designed to slow or modify disease progression. By examining current research findings, the review aims to present a clear understanding of how modern scientific advancements are shaping the future of Alzheimer's disease management. In addition to pharmacological therapies, this review also explores non-pharmacological interventions and lifestyle-based strategies that may contribute to improved cognitive health.[21] The role of emerging technologies, including biomarker-based diagnostics and artificial intelligence-assisted disease detection, is also discussed as part of modern approaches to Alzheimer's disease management. The review aims to identify existing challenges and research gaps in the development of effective treatments for Alzheimer's disease.[22] Despite extensive research efforts, many therapeutic strategies remain under investigation, and further studies are required to improve treatment efficacy and accessibility. The scope of this review is to summarize current knowledge, highlight recent advancements, and provide insights into future directions for improving the treatment and management of Alzheimer's disease.[23] The information presented in this review may serve as a valuable resource for researchers, clinicians, and healthcare professionals involved in the study and treatment of this complex neurodegenerative

disorder. Additionally, for upcoming treatments, only trials from 2015 until 2023 were obtained. From there, a manual selection for appropriate drugs and drug categories was done to ensure the inclusion of any additional publications or trials that might have been missed by the electronic search and the exclusion of any articles ineligible or irrelevant to the systematic review.[24]

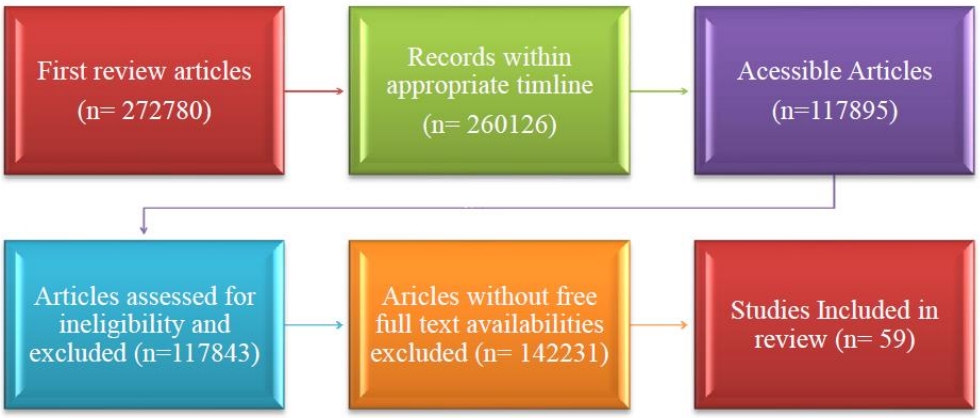

Figure 4: Flow chart of inclusion and exclusion articles for review paper.

Pathophysiology of Alzheimer's Disease

2.1 Amyloid Cascade Hypothesis

- ***Role of amyloid-beta aggregation***

The amyloid cascade hypothesis is one of the most widely accepted explanations for the development of Alzheimer's disease. According to this hypothesis, the abnormal accumulation of amyloid-beta (Aβ) peptides in the brain initiates a series of pathological events that ultimately lead to neuronal damage and cognitive decline.[25] Amyloid-beta peptides are generated through the enzymatic cleavage of amyloid precursor protein (APP). Under normal physiological conditions, these peptides are cleared efficiently from the brain. However, in Alzheimer's disease, an imbalance between production and clearance results in the accumulation of amyloid-beta. These peptides aggregate to form insoluble amyloid plaques that deposit in the extracellular spaces of the brain.[26] The presence of these plaques interferes with neuronal communication, disrupts synaptic transmission, and triggers inflammatory responses. Furthermore, amyloid-beta aggregation is believed to promote oxidative stress and activate microglial cells, leading to further neuronal injury. Over time, this cascade of pathological processes contributes to progressive neurodegeneration and cognitive impairment characteristic of Alzheimer's disease.[27]

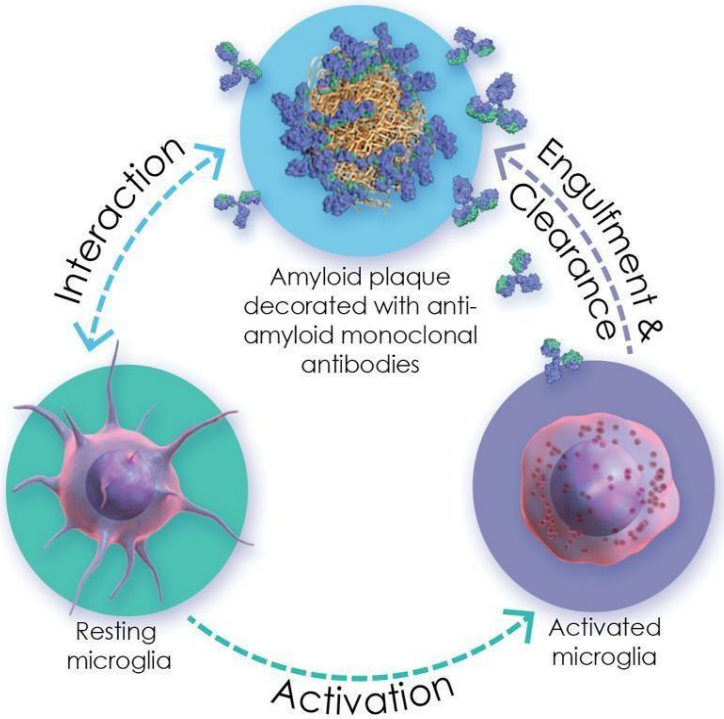

Figure 5: Amyloid Cascade Hypothesis Pharmacological action.

2.2 Tau Protein Pathology

Tau protein plays an essential role in stabilizing microtubules within neurons, which are responsible for maintaining cell structure and facilitating intracellular transport. In Alzheimer's disease, tau protein undergoes abnormal hyperphosphorylation, which alters its normal function.[28] As a result, tau molecules detach from microtubules and begin to aggregate inside neurons. These aggregated tau proteins form twisted filamentous structures known as neurofibrillary tangles. The formation of these tangles disrupts the transport of essential nutrients and organelles within neurons, leading to cellular dysfunction and eventual neuronal death. Neurofibrillary tangles are primarily observed in brain regions associated with memory and cognition, such as the hippocampus and cerebral cortex.[29] The severity of tau pathology often correlates strongly with the degree of cognitive decline in Alzheimer's patients. Therefore, targeting tau protein aggregation has become an important focus in the development of disease-modifying therapies.[30]

2.3 Neuroinflammation

- ***Microglial activation and inflammatory response***

Neuroinflammation is another critical factor contributing to the progression of Alzheimer's disease. The accumulation of amyloid-beta plaques and damaged neurons activates microglial cells, which are the primary immune cells of the central nervous system. Under normal conditions, microglia help maintain brain homeostasis by clearing cellular debris and protecting neurons.[31] However, in Alzheimer's disease, prolonged activation of microglia leads to the release of pro-inflammatory cytokines, chemokines, and reactive oxygen species. These inflammatory mediators contribute to neuronal damage and exacerbate disease progression.[32] Chronic neuroinflammation also disrupts synaptic function and accelerates neuronal degeneration. Recent studies suggest that targeting inflammatory pathways may provide a promising strategy for slowing the progression of Alzheimer's disease. As a result, anti-inflammatory therapies are currently being investigated as potential therapeutic interventions.[33]

2.4 Oxidative Stress and Mitochondrial Dysfunction

Oxidative stress plays a significant role in the pathogenesis of Alzheimer's disease. It occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the brain's antioxidant defense mechanisms. Neurons are particularly vulnerable to oxidative damage because of their high metabolic activity and oxygen consumption. In Alzheimer's disease, excessive oxidative stress damages cellular components such as lipids, proteins, and DNA.[34] This damage impairs neuronal function and contributes to cell death. Mitochondrial dysfunction further worsens this process by reducing cellular energy production and increasing ROS generation. The combined effects of oxidative stress and mitochondrial impairment accelerate neurodegeneration and cognitive decline. Consequently, antioxidant and mitochondrial-protective agents are being explored as potential therapeutic strategies for Alzheimer's disease.[35]

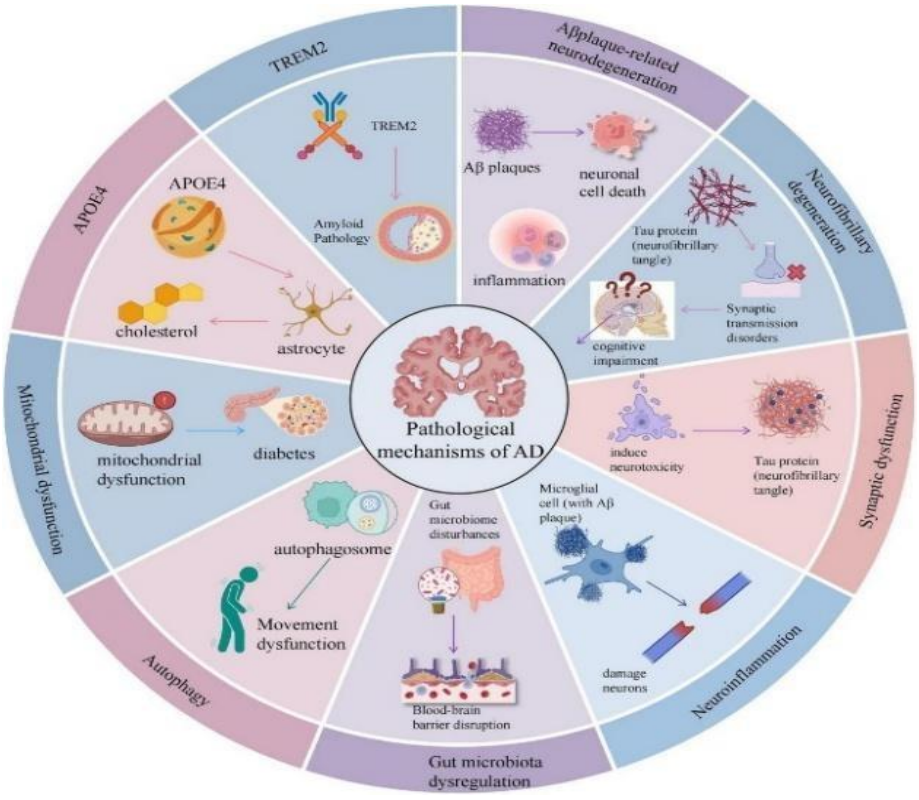

Figure 6: Formation of neurofibrillary chat flow.

3. Conventional Pharmacological Treatments

3.1 Cholinesterase Inhibitors

Examples:

- Donepezil
- Rivastigmine
- Galantamine

Cholinesterase inhibitors are among the most commonly prescribed medications for the symptomatic treatment of mild to moderate Alzheimer's disease. These drugs work by inhibiting the enzyme acetylcholinesterase, which is responsible for the breakdown of acetylcholine in the brain. Acetylcholine is an important neurotransmitter involved in learning and memory processes.[36] In Alzheimer's disease, the levels of acetylcholine decrease significantly due to the degeneration of cholinergic neurons. By inhibiting acetylcholinesterase, these drugs increase the availability of acetylcholine in the synaptic cleft, thereby improving communication between neurons. Donepezil, rivastigmine, and galantamine have been shown to provide modest improvements in cognitive function, daily activities, and behavioral symptoms. However, these medications do not stop the progression of the disease and mainly provide temporary symptomatic relief.[37,38]

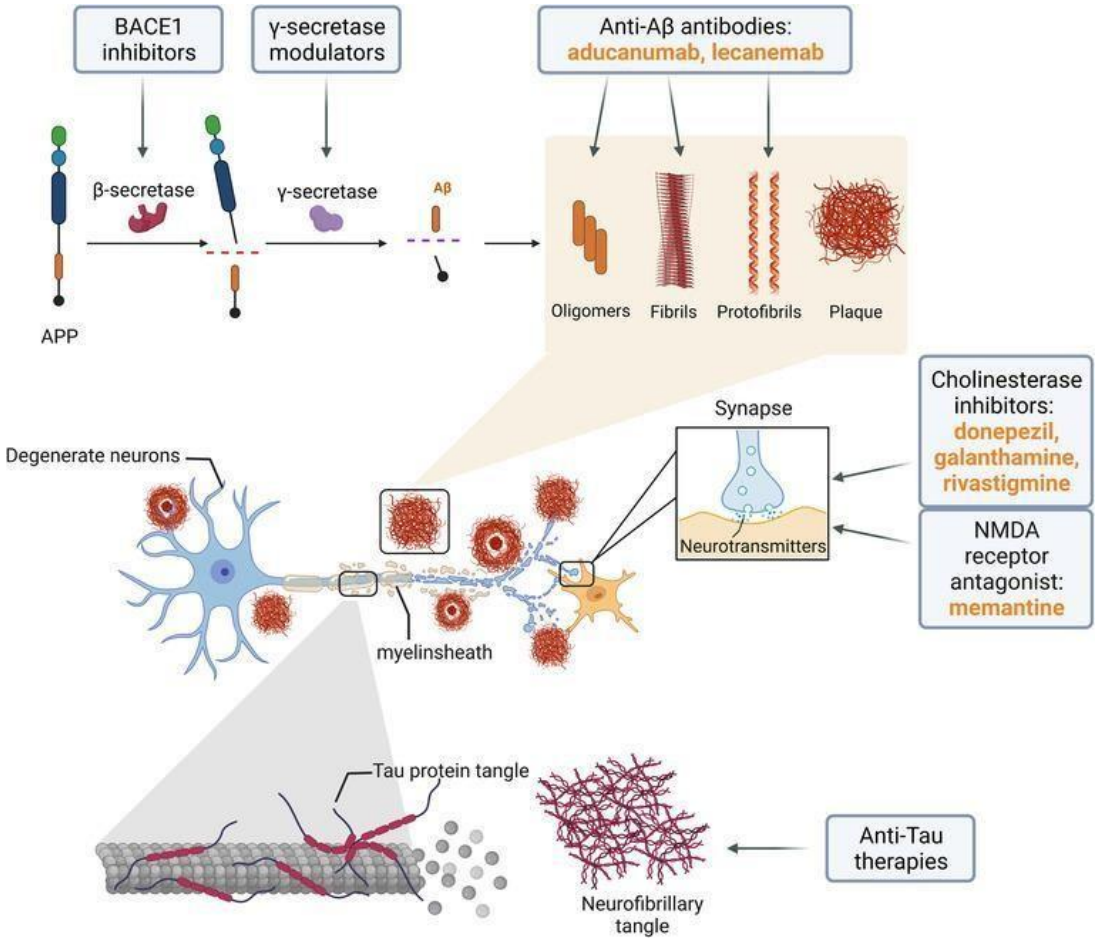

Figure 7: Mechanism of action of Pharmacological Treatments with drug.

3.2 NMDA Receptor Antagonists

Memantine is another commonly used drug in the treatment of moderate to severe Alzheimer's disease. It acts as an antagonist of the N-methyl-D-aspartate (NMDA) receptor, which is involved in glutamate-mediated excitatory neurotransmission in the brain.[39] In Alzheimer's disease, excessive activation of NMDA receptors by glutamate can lead to excitotoxicity, a process that damages neurons and contributes to neurodegeneration. Memantine works by regulating NMDA receptor activity and preventing excessive calcium influx into neurons. By reducing excitotoxic neuronal damage, memantine helps improve cognitive function and daily living activities in some patients.[40] It is often used alone or in combination with cholinesterase inhibitors for better symptomatic management.[41]

3.3 Limitations of Current Therapies

Although conventional pharmacological treatments provide symptomatic benefits, they have several limitations. Most importantly, these drugs do not address the underlying causes of Alzheimer's disease such as amyloid accumulation or tau pathology. The clinical benefits of current medications are generally modest and

temporary, and their effectiveness may vary among patients. Additionally, some patients experience side effects such as gastrointestinal disturbances, dizziness, or confusion. Because of these limitations, there is a growing need to develop disease-modifying therapies that can slow or halt the progression of Alzheimer's disease rather than only managing symptoms.[42,43]

4. Lifestyle Interventions

- Physical exercise
- Mediterranean diet
- Sleep regulation

Lifestyle interventions play an important role in the prevention and management of Alzheimer's disease. Although these strategies cannot completely cure the disease, they may significantly contribute to slowing cognitive decline and improving overall brain health. Increasing evidence suggests that lifestyle-related factors influence neuronal function, neuroplasticity, and inflammation in the brain. Regular physical exercise has been widely associated with improved cognitive function and reduced risk of neurodegenerative disorders.[44] Exercise enhances cerebral blood flow, promotes the release of neurotrophic factors such as brain-derived neurotrophic factor (BDNF), and supports synaptic plasticity. These effects help maintain neuronal health and may delay the progression of Alzheimer's disease. Dietary habits also have a strong influence on brain health.[45] The Mediterranean diet, which includes fruits, vegetables, whole grains, fish, olive oil, and nuts, has been associated with a reduced risk of cognitive decline. This diet is rich in antioxidants and anti-inflammatory compounds that help protect neurons from oxidative stress and inflammation.[46] Studies have shown that individuals who follow a Mediterranean-style diet often demonstrate better cognitive performance and slower disease progression. Sleep regulation is another critical factor in maintaining cognitive health. During sleep, the brain clears metabolic waste products, including amyloid-beta peptides. Poor sleep quality or chronic sleep deprivation may lead to increased amyloid accumulation and accelerate neurodegenerative processes. Therefore, maintaining healthy sleep patterns may help reduce the risk and progression of Alzheimer's disease.[47]

4.1. Behavioural and Psychological Therapy

Behavioural and psychological therapies are important components of comprehensive Alzheimer's disease management. As the disease progresses, patients often experience behavioural and psychological symptoms such as agitation, anxiety, depression, irritability, and mood disturbances.[48] These symptoms can significantly affect both the patient's quality of life and the well-being of caregivers. Non-pharmacological psychological interventions aim to reduce these symptoms through supportive therapy, counselling, and structured behavioural programs. Cognitive behavioural therapy (CBT), reminiscence therapy, and supportive social interactions can help patients manage emotional distress and maintain a sense of identity and well-being.[49] Behavioural therapy also focuses on modifying environmental and social factors that may trigger agitation or confusion. Establishing regular routines, maintaining familiar surroundings, and providing emotional reassurance can help reduce behavioural disturbances. In addition, caregiver education programs play a crucial role in helping families manage challenging symptoms effectively. Overall, behavioural and psychological therapies complement pharmacological treatments and contribute to improved patient care and quality of life.[50]

5. Emerging Technologies in Alzheimer's Management

5.1 Artificial Intelligence in Early Diagnosis

Artificial intelligence (AI) has emerged as a powerful tool in the early detection and diagnosis of Alzheimer's disease. AI-based systems can analyse large volumes of medical data, including brain imaging, genetic information, and clinical records, to identify patterns associated with the early stages of the disease.[51] Machine learning algorithms can detect subtle changes in brain structure and function from imaging techniques such as magnetic resonance imaging (MRI) and positron emission tomography (PET). These changes may appear long before clinical symptoms become evident. Early detection through AI-assisted analysis enables timely intervention and improved disease management. Furthermore, AI can assist healthcare professionals in predicting disease progression and evaluating treatment responses. As technology continues to advance, AI-based diagnostic tools may become an essential component of personalized Alzheimer's disease management.[52]

5.2 Biomarkers for Early Detection

Biomarkers have become increasingly important in the diagnosis and monitoring of Alzheimer's disease. Biomarkers are measurable biological indicators that reflect specific pathological processes occurring within the body. In Alzheimer's disease, several biomarkers are associated with amyloid-beta accumulation, tau protein pathology, and neurodegeneration. Cerebrospinal fluid (CSF) biomarkers such as decreased amyloid-beta levels and increased phosphorylated tau protein concentrations are widely used indicators of Alzheimer's pathology. Imaging techniques, including amyloid PET and tau PET scans, also provide valuable information about the presence and distribution of these pathological proteins in the brain.[53] More recently, researchers have been exploring blood-based biomarkers as a less invasive and more accessible diagnostic approach. These biomarkers may allow earlier detection of Alzheimer's disease and facilitate large-scale screening programs. The development of reliable biomarkers is expected to significantly improve diagnostic accuracy and enable earlier therapeutic interventions.[54]

5.3 Digital Therapeutics and Remote Monitoring

Digital therapeutics represent a rapidly growing field in the management of neurodegenerative diseases. These technologies involve the use of software-based interventions and digital tools to support cognitive training, behavioral therapy, and patient monitoring. Mobile applications and digital platforms can provide structured cognitive exercises designed to stimulate memory, attention, and problem-solving skills. Wearable devices and remote monitoring systems can track important health parameters such as sleep patterns, physical activity, and daily routines.[55] These technologies allow healthcare providers to monitor disease progression more effectively and adjust treatment strategies when necessary. Digital health tools also support caregivers by providing real-time information about patient status and facilitating communication with healthcare professionals.[56]

6. Challenges and Future Perspectives

- Blood–brain barrier limitations
- Challenges in drug development
- Personalized medicine approaches
- Future research directions

Despite extensive research efforts, several challenges continue to hinder the development of effective treatments for Alzheimer's disease. One of the major obstacles is the presence of the blood–brain barrier (BBB), a highly selective barrier that restricts the entry of many therapeutic agents into the brain. This barrier often limits the effectiveness of potential drugs designed to target pathological processes within the central nervous system.[57] Another major challenge lies in the complex and multifactorial nature of Alzheimer's disease. The disease involves multiple pathological mechanisms, including amyloid accumulation, tau pathology, inflammation, and oxidative stress. As a result, developing drugs that effectively target all these processes remains difficult. Many clinical trials have failed to demonstrate significant therapeutic benefits, highlighting the need for improved research strategies. Personalized medicine approaches are gaining increasing attention in Alzheimer's research. These strategies aim to tailor treatments based on individual genetic profiles, biomarker data, and disease characteristics. Personalized therapeutic strategies may improve treatment effectiveness and reduce adverse effects. Future research is likely to focus on developing multi-target therapies, advanced drug delivery systems capable of crossing the blood–brain barrier, and improved diagnostic technologies. Continued interdisciplinary collaboration between neuroscientists, clinicians, and pharmaceutical researchers will be essential for achieving meaningful progress in Alzheimer's disease treatment.[58,59]

7. CONCLUSION

Alzheimer's disease remains one of the most significant neurodegenerative disorders affecting aging populations worldwide. Although currently available treatments primarily focus on symptom management, recent advances in biomedical research have led to the development of modern therapeutic strategies aimed at modifying disease progression. Emerging therapies such as monoclonal antibodies targeting amyloid-beta, tau-directed treatments, and anti-inflammatory agents represent promising approaches for addressing the underlying pathology of the disease. In addition, non-pharmacological strategies including cognitive stimulation, lifestyle modifications, and psychological support play an essential role in improving patient care and quality of life.

Early diagnosis and timely therapeutic intervention are critical for achieving better clinical outcomes. Advances in artificial intelligence, biomarker research, and digital health technologies are expected to significantly enhance early detection and personalized treatment approaches.

Overall, continued research and innovation are necessary to develop more effective and accessible therapies. A comprehensive and multidisciplinary approach will be essential for improving disease management and reducing the global burden of Alzheimer's disease.

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