IJBPAS, August, 2024, 13(8): 4075-4089

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

PERSPECTIVE ON RECENT UPDATES IN ALZHEIMER'S DISEASE

SINGH S*, KAKADIYA J, JAMSA A AND MANDAL S

Department of Pharmacology, Parul Institute of Pharmacy and Research, Parul University, Limda, Waghodia, Vadodara-391760, Gujarat, India

***Corresponding Author: Ms. Sonali Singh: E Mail: sonaliss1230@gmail.com**

Received 15th May 2023; Revised 18th Aug. 2023; Accepted 4th Nov. 2023; Available online 1st Aug. 2024

https://doi.org/10.31032/IJBPAS/2024/13.8.8264

ABSTRACT

Alzheimer's disease is a debilitating neurological condition that affects millions of people throughout the world. Major development has been achieved in discovering the underlying processes and developing viable therapies for this illness in recent years. This viewpoint post gives a succinct review of recent changes and breakthroughs in Alzheimer's disease study.

The abstract opens by emphasising Alzheimer's disease's worldwide significance, demonstrating its extent and its importance in healthcare. It also highlights that ongoing studies have given major insights into the biology associated with the illness, adding to improved knowledge of its early stages and certainly.

It additionally highlights the development as well as evaluation of innovative therapy techniques, such as pharmaceutical therapies, lifestyle interventions, and recently developed technology. These therapies attempt to slow cognitive decline and improve the standard of life for those who are impacted.

The article continues by underlining the significance of ongoing study and cooperation in order to comprehend in full exacerbated aspect of dementia. It enables researchers and health care professionals to cooperate in order to identify new approaches and enhance the chances for dementia prevention, early identification, and successful management.

Overall, this perspective of an article gives a thorough account of recent advances in Alzheimer's disease studies as well as provides an outline for future research aimed at finally finding a solution for this difficult neurological disorder.

Keywords: Acetylcholinesterase inhibitors, Alzheimer's diseases, dementia with Lewy bodies and mild cognitive impairment

1. INTRODUCTION

Alzheimer's disease constitutes a neurodegenerative ailment which gradually impairs cognition to recall and remember along with the capacity to do simply the most basic initiatives. The most typical form of dementia is this one. The neurological condition bears the term Alzheimer's after Dr. Alois Alzheimer, who initially identified "a peculiar disease" during 1906 which was marked with severe impairment of memory and microscopic brain alterations **[1]**.

Alzheimer's disease is a degenerative illness that commences with minimal cognitive impairment and can impair one's abilities to converse or react to their surroundings. Cognitive impairment usually least during the earliest phases in the condition, but across several years, signs indicating dementia become more severe. This condition affects areas of the central nervous system that manage thinking, recalling information, and speech, so it may result in a significant impact overall an individual's capacity for carrying out everyday tasks **[1, 2]**.

Above the age range of 65, the prevalence of dementia rises twice every five years, so the possibility of developing it rises with as one gets older. 5.8 million Americans were estimated to be suffering from dementia in 2020, and in the year 2060, 14 million individuals are predicted to be impacted by the disease. The development of Alzheimer's tends to be less prevalent in youngsters yet it is still possible **[2]**.

Although the exact etiology of Alzheimer's disease is unknown, it is likely to involve a variety of issues including hereditary factors, way of life, including factors related to the environment. Dementia-related illnesses presently have no cure in sight, however there are a few medications that can assist in alleviating certain associated complications. Despite the onset of signs and symptoms, those suffering from Alzheimer's disease may survive for several years, however this might vary **[1]**.

Symptoms of Alzheimer's disease include: Memory loss, especially of recent events, difficulty with problem-solving, planning, and completing familiar tasks, Confusion, disorientation, and getting lost in familiar places, Difficulty with speech and language and changes in mood and personality will be affected **[1]**.

A highly trained professional may do additional tests, including cerebral imaging if required, analyse the signs in more depth and establish a medical as well as therapeutic approach if dementia is detected.

1.1. Epidemiology

Alzheimer's disease is a global health concern that affects millions of people worldwide. According to recent estimates, approximately 47 million people live with

dementia across the globe, and Alzheimer's is the single most common cause of dementia, comprising 70% of all cases. In the United States, approximately 5.7 million people are living with Alzheimer's, and it is the 6th leading cause of death in the country. The prevalence of Alzheimer's disease is projected to increase in the coming years, with the number of people affected expected to double every 20 years until at least 2040 **[3]**.

Age is a major risk factor for Alzheimer's disease, and the disease is predominantly a disease of aging. Females are more likely to develop Alzheimer's compared to males, partially because they live longer. Genetic factors also play a role in the development of Alzheimer's disease, and cerebrovascular disease is also a risk factor for the disease **[4, 5]**.

In conclusion, Alzheimer's disease is a major public health issue that affects millions of people worldwide. The prevalence of the disease is projected to increase in the coming years, and age, gender, genetics, and cerebrovascular disease are all risk factors for the development of the disease **[3, 5]**.

1.2. State of brain during the Alzheimer's diseases

Multiple brain changes brought on by Alzheimer's disease cause an overtime decline of remembering and ability to think clearly. The consequences of Alzheimer's disease on the brain are summarised as follows:

1. Neuronal Damage: In the beginning stages of this conditions, synapses and the connections which exist between them in memory-related regions of the brain, such as the entorhinal cortex and hippocampus, are targeted and destroyed by Alzheimer's disease. These areas are essential for generating and retaining recent recollections. Additional regions of the cerebral cortex important for cognitive function and social interaction are affected as the disease worsens.

2. Disruption of Communication: Alzheimer's disease interferes with the exchange of electrochemical and biochemical impulses, which are crucial for the interpretation and dissemination of data. This interference causes a decline of functionality and subsequently the death of cells.

3. Shrinkage of the Brain: A person's brain typically shrinks to a certain extent with regular ageing. In contrast, there is significant damage in Alzheimer's disease, which results in numerous synapses ceasing operating, losing connectivity to other brain cells, and eventually dying. The brain begins to shrink as a result of this.

4. Specific Brain Regions: Certain areas of the brain are impacted by Alzheimer's disease, which causes

various kinds of cognitive deficits. The harmed regions consist of:

- Hippocampus: The region of the hippocampus oversees creating new recollections. Initial decline in memory in Alzheimer's patients is a result of damage to this area
- Frontal Lobe: The frontal region has an impact on cognition, judgement, and conduct.
- Temporal Lobe: The retrieval of memory involves the brain's temporal lobe. The decline in memory is further exacerbated by destruction of this area.
- Parietal Lobe: The region of the parietal lobe contributes to the thought process of speech. Language acquisition may be impacted by impairment to this area.

As a whole, Alzheimer's disease impairs crucial brain functions involving metabolic processes, transmission, and restoration, resulting in the gradual deterioration of recollection, cognitive abilities, and everyday function.

1.3. Difference between Alzheimer's disease and other types of dementia

Although Alzheimer's disease is a particular kind of dementia, there are other varieties of dementia with different distinctions from Alzheimer's. The following are some significant distinctions amongst dementias including Alzheimer's disease:

1. Cause: The formation of aberrant accumulations of proteins in the brain, such as plaques containing amyloid and tau tangles, is the primary hallmark of Alzheimer's disease. Additional underlying triggers can contribute to other kinds of dementia, such as diminished blood supply to the brain or the development of aberrant protein deposits known as Lewy bodies, which can result in vascular memory loss or Lewy body dementia **[6]**.

2. Progression: Alzheimer's disease normally develops progressively over an extended period, with indications progressively getting severe across several periods. Distinct variations in advancement could be present for additional types of dementia. For instance, vascular dementia could develop gradually, with symptoms abruptly getting exacerbated shortly after a cerebral infarction or other vascular incident **[6]**.

3. Symptoms: Although dementia is a typical symptom of various forms of dementia, memory loss is a prevalent sign of Alzheimer's disease. For instance, frontotemporal dementia may bring about alteration in habits, personality traits, and language skills, whereas Lewy body dementia is

commonly associated with visual delusions and Parkinson's-like movement symptoms **[6]**.

4. Age of Onset: Alzheimer's disease frequently manifests in people over 60, usually during their mid-60s or afterwards. Nevertheless, some dementias can develop in people those who are younger. As an example, people in their 40s and 50s are frequently affected with frontotemporal dementia.

5. Brain Regions Affected: Numerous dementias can impact various parts of the brain. The hippocampus, frontal lobes, temporal lobes, and parietal lobes are frequently impacted in Alzheimer's disease. According to the fundamental cause, several kinds of dementia may affect various parts of the brain **[6]**.

It is essential to keep in mind that these are broad distinctions and that every individual's encounter with dementia will probably vary. Regarding the purpose of identifying the precise form of dementia and delivering the necessary treatment and assistance, accurate assessments and diagnosis by medical experts are essential **[6]**.

1.4. Types of Alzheimer's

Alzheimer's disease comes in a variety of forms, and it is often grouped with other dementias. Here are a few of the dementias linked to and caused by Alzheimer's disease:

1. Alzheimer's Disease (AD): Particularly prevalent type of dementia is Alzheimer's disease. The gradual loss of recall and mental abilities can be triggered by the buildup of aberrant accumulations of proteins in the brain, that include amyloid plaques and tau tangles.

2. Vascular Dementia: The subsequent most prevalent kind of dementia is vascular dementia. Memory loss happens whenever there is a lack of blood supply to the brain, which is frequently caused by a cerebral infarction or other vascular diseases **[7]**.

3. Dementia with Lewy Bodies (DLB): Lewy bodies, aberrant protein deposits, are a hallmark of DLB and can be detected in the brain. It can result in cognitive deterioration, movement indications, and visual delusions, which makes it comparable to either Parkinson's disease and Alzheimer's disease **[8]**.

4. Frontotemporal Dementia (FTD): The frontal and temporal lobes of the brain are largely affected by FTD, the least prevalent form of dementia. Alteration in actions, nature, and linguistic abilities are its defining traits **[9]**.

5. Mixed Dementia: Mixed dementia is the concurrent occurrence of various kinds of dementia, which includes

Alzheimer's disease and vascular dementia. It is typical for people to experience several distinct forms of dementia **[10]**.

6. Young-Onset Dementia: When hallmarks of dementia appear earlier the age of 65, the condition is referred to as young-onset dementia. It can involve a variety of dementias, such as frontotemporal dementia and early-onset Alzheimer's disease **[11]**.

The above are merely a few forms of dementias associated with Alzheimer's disease, it is crucial to remember them. Each kind may have unique traits, inherent reasons, and sequences of development. For the purpose of identifying the precise form of dementia and delivering adequate treatment and assistance, an accurate identification by medical specialists is crucial.

1.5. Pathophysiology of Alzheimer's diseases

Alzheimer's disease has a complicated pathogenesis which comprises several elements, such as the buildup of aberrant accumulations of proteins in the brain, harm to the neurons, and inflammation. It is primarily identified by the aberrant buildup among the disease's marker proteins, amyloid beta and hyperphosphorylated tau. The central nervous system contains the extremely essential protein tau, which tends to reside in synapses or neuronal cells. Its main objective is to sustain the microtubule structure, that is essential for preserving the structural integrity and arrangement of synapses. The migration of nutrients, biological elements, and additional vital chemicals across neurons is also facilitated by these microtubules. As assuring the integrity and effective operation of microtubules, tau serves a crucial part in stimulating optimal activity in neurons when it is in a state of good health. Tau proteins, on the other hand, develop hyperphosphorylation in Alzheimer's disease, which means they pick up a substantial quantity of phosphate groups. Tau becomes detached off microtubules because of such hyperphosphorylation, abandoning the potential to stabilise the microtubule structure. Consequently, neurofibrillary tangles, which are tangled filaments made of tau proteins, commence to develop in neurons. The buildup of neurofibrillary tangles interferes with typical activity within cells, which causes synaptic malfunction and ultimately the death of cells.

Another significant pathogenic characteristic found in the brains of people with Alzheimer's disease is neurofibrillary tangles. These complex structures are aberrant buildups of tau, a protein that is essential for stabilising the cellular organisation of cells in the nervous system or synapses. Tau protein attaches to and

maintains microtubules, which serve as critical for preserving the configuration of each cell along with aiding the transit of nutrients and various other substances throughout the cell, in functional neurons. Tau, however, experiences aberrant changes in chemicals in Alzheimer's disease and several additional neurodegenerative illnesses, which causes it to aggregate forming undissolved clusters across the nerve cells. The tangles that make up the neurofibrillary are composed of twisted filaments constituted by these accumulated tau proteins. Neurofibrillary tangles prevent synapses from interacting with adjacent cells and impede their typical function, which eventually leads to cell death. These tangles expand over different brain areas as the illness worsens, which contributes to the intellectual and behavioural deficits that are typical of AD.

For synaptic plasticity, along with the potential of connections to grow or decrease in accordance with neuronal activity, exocytosis thus performs an essential function. The foundation of both memory and learning is synaptic plasticity. Exocytosis problems might hinder synaptic plasticity and exacerbate cognitive deterioration in AD. It is believed that a disturbance in brain formation and removal is what causes Aβ (Amyloid β protein) buildup. This is a neurodegenerative condition that worsens over time and is brought on by the loss of neurons. In contrast, it progressively begins in the entorhinal cortex of the hippocampus and expands through time into additional regions of the brain. In addition, plaques as well as neurofibrillary tangles (NFT) are linked to gradual decline in cognition, neurological dysfunction, and degeneration of the neurons. An inflammation and oxidative stress both consequently lead to the pathogenesis of AD.

However, there is an additional component that causes the cholinergic neurons over time to decline especially when they came from the basal forebrain and extend to different parts of the central nervous system that are important in thinking and remembering, such the cerebral cortex and hippocampus. Acetylcholine levels in various areas of the brain are significantly decreased as a result of this deterioration. Acetylcholine insufficiency is an underlying cause in the intellectual loss and cognitive decline associated with Alzheimer's disease. As a result, one of the key treatment approaches for treating the clinical signs of this neurodegenerative condition is the suppression of acetylcholinesterase (AChE). Acetylcholinesterase inhibitors (AChEIs) are drugs that prevent the acetylcholine from undergoing break down as quickly, elevating the amount of acetylcholine in terminals and boosting cholinergic neurotransmission. As

a result, AChEIs may benefit some AD sufferers by enhancing their mental abilities, recall, and ability to perform daily tasks. Donepezil, rivastigmine, and galantamine are a few of the typical acetylcholinesterase inhibitors recommended in the management of Alzheimer's disease. These medications help relieve symptoms as well as briefly enhance cognitive performance, specifically for those with initial mild to moderate phases of Alzheimer's disease, however they do not change the fundamental path of the condition. Research is underway to gain insight into AD and create viable therapies since the precise processes behind the illness have not been fully known **[12, 13]**.

1.6. The roles of amyloid beta and tau proteins

By means of a variety of methods, amyloid beta and tau protein buildup within the brain can cause cognitive deterioration among individuals with Alzheimer's disease. The following are some of the ways that the buildup of these proteins might impact mental function:**[14, 15]**.

1. Formation of Plaques and Tangles: Plaques and tangles can form in the central nervous system as a result of the buildup of amyloid beta and tau proteins. Memory loss might result from such aberrant accumulations of proteins interfering with synapses' ability to operate appropriately **[15]**.

2. Neuronal Damage: Amyloid beta and tau protein buildup can potentially result in impairment of neurons and demise of cells. Cognitive impairment may result from this because there may be fewer functional neurons and less connectivity within neurons **[15, 16]**.

3. Inflammation: Amyloid beta and tau protein buildup might end up in a response of inflammation in the central nervous system, which may additionally harm neurons and impair cognitive function **[15, 16]**.

4. Disruption of Communication: The buildup of amyloid beta and tau proteins may interfere with the interaction between neurons and the exchange of electrical and chemical signals, both of which are crucial for the acquisition and transfer of material. This interference causes a loss of functionality and subsequently the death of cells **[14, 16]**.

In general, amyloid beta and tau protein buildup in the central nervous system may result in an accumulation of plaques and tangles, damaged neurons, inflammatory conditions, and interruption of neuronal transmission, any of which can trigger cognitive impairment in Alzheimer's disease **[4, 15]**.

2. FDA-approved treatments for Alzheimer’s

Even though there is no known cure for Alzheimer's, two approved by the FDA medicines target the underlying biology. Additional medications might assist with symptoms like disorientation and remembering loss **[17]**.

2.1. FDA-approved drugs for Alzheimer's

The FDA has authorised treatments that can be classified into two distinct groups: the ones that slow the advancement underlying the initial stages Alzheimer's disease in patients and those which can temporarily decrease the indications of Alzheimer's dementia. It is crucial to consult a medical professional before deciding on any course of therapy to ascertain the extent to which it is necessary. Individuals who utilise these drugs are advised to be closely monitored by a physician with expertise prescribing them, who will also make sure that the suggested parameters are rigorously followed **[17]**.

2.2. Drugs that change disease progression

By targeting the molecular basis of the medical condition process, medications in this category decrease the extent of the disorder. Treatments are designed to reduce the functional and cognitive deterioration of persons suffering Alzheimer's disease.

Anti-amyloid medications function by binding and then expelling beta-amyloid, a protein typically builds up in plaques in the brain. Each has a unique mechanism of action and addresses beta-amyloid at a distinct phase of plaque development **[17]**. Aducanumab (Aduhelm™) It is an intravenous (IV) infusion medication for anti-amyloid antibodies that has been permitted for the treatment for the earliest stages of Alzheimer's disease, comprising moderate to severe cognitive impairment (MCI) and mild dementia caused by Alzheimer's disease.

The initial treatment demonstrated the fact that getting rid of beta-amyloid from the brain slows down intellectual and functional deterioration in persons with early Alzheimer's was aducanumab. Alzheimer's disease is suggested as a therapy via aducanumab. The medication was tested on persons who had early-stage Alzheimer's disease, which entails those who had moderate cognitive impairment (MCI) or mild dementia associated with Alzheimer's disease and have amyloid plaque formation in their brains. For patients with the illness phase analysed in the clinical investigations, medication via aducanumab may be suitable. Beginning therapies at earlier or subsequent phases of the disorder that has been investigated does not seem to be safe or beneficial.

Aducanumab treatment resulted in a decrease in cognitive deterioration as assessed by mental and physical abilities in certain clinical study subjects.

Remembering and orientations are two examples of cognitive metrics. Executing own finances as well as carrying out domestic duties like housekeeping are included in functional measurements.

Perhaps the most frequent side effects are headaches, falls, and amyloid-related imaging abnormalities (ARIA). An allergic response is an additional side effect that might be quite harmful. ARIA is a typical side effect that, while typically causing no indications, can be severe. It generally begins as sudden swelling in some parts of the central nervous system and goes away as time passes. Whereas the majority of those experiencing inflammation in regions of the brain might not experience symptoms, some individuals might additionally have small spots of haemorrhage within or around the surface of the brain. Certain individuals may experience ARIA symptoms as headaches, nausea, dizziness, disorientation, and alterations in sight **[17]**.

Lecanemab (LeqembiTM) is an intravenous (IV) infusion medication for anti-amyloid antibodies that has been authorised for treating early Alzheimer's with detection that it raises beta-amyloid. Individuals with moderate cognitive decline (MCI) or mild dementia brought on by Alzheimer's disease are eligible to use this prescription drug. The presence of raised beta-amyloid plaques in the brain must also be validated by these people as well.

Amyloid-related imaging abnormalities (ARIA), a frequent side effect that often provides no symptoms although can be dangerous, and infusion-related responses were the most frequently reported dangerous side effects. Usually, there is a brief enlargement of the brain in certain places. Typically, it gets better with time **[17]**.

2.3. Drugs that treat symptoms

2.3.1. Cognitive symptoms (memory and thinking)

- Alzheimer's disease improves cognitive symptoms when neuronal cells die and connectivity between cells are damaged. Although these drugs might not prevent the destruction of brain cells caused by Alzheimer's, but they may reduce or stabilise symptoms for a brief period by altering molecules associated with transmitting information via the nerve cells of the brain **[17]**. Listed drugs are recommended for the treatment of memory and thinking-related disorders.
- Cholinesterase inhibitors (Aricept®, Exelon®, Razadyne®)

  Treatment for problems with recall, thinking, language, judgement, and other mental processes involves the use of cholinesterase inhibitors. Acetylcholine is a chemical messenger crucial for memory and learning, and these drugs stop it from

breaking down. These medications aid in nerve cell transmission **[17]**.

- The cholinesterase inhibitors most prescribed are:

  Donepezil (Aricept®): permitted for the management of Alzheimer's disease throughout all phases.

  Rivastigmine (Exelon®): permitted for both mild to moderate dementia brought on by Parkinson's condition and mild to moderate Alzheimer's disease.

  Galantamine (Razadyne®): recommended for mild-to-moderate Alzheimer's disease phases.

  Although typically tolerated without issue side effects may arise. If they do, they tend to involve nausea, vomiting, appetite loss, and more frequent bowel movements.

- Glutamate regulators (Namenda®)

  Glutamate regulators are recommended to enhance cognition, speech, logic, and basic task performance. This kind of medication controls the function of glutamate, a separate chemical transmitter which assists in interpreting data in the brain. This medication is referred to as:

  Memantine (Namenda®): approved for treating moderate-to-severe Alzheimer's disease. Side effects such as headache, constipation, disorientation, and dizziness may possible **[17]**.

- Cholinesterase inhibitor + glutamate regulator (Namzeric®)

  This class of medication combines a glutamate modulator with a cholinesterase inhibitor. Donepezil and memantine (Namzaric®): approved for treating moderate-to-severe Alzheimer's disease. Nausea, vomiting, lack of appetite, more frequent bowel movements, headaches, constipation, disorientation and dizziness are just a few of the probable side effects.

2.3.2. Non-cognitive symptoms (behavioural and psychological symptoms)

Remembering and thinking are not the only things that Alzheimer's impacts. Numerous behavioural and psychological dementia symptoms, including agitation, hallucinations, and delusions, can have an influence on an individual's quality of life. While certain drugs temporarily target reducing these non-cognitive symptoms, it is vital to first attempt non-drug behaviour management techniques **[17]**.

The FDA has now authorised one medication to treat lack of sleep-in dementia patients, but clinical studies for medications to treat additional non-cognitive symptoms are also under progress.

- Orexin receptor antagonist (Belsomra®)

This medication, which is used to treat insomnia, blocks the function of orexin, a kind of neurotransmitter involved in the sleep-wake cycle:

Suvorexant (Belsomra®): It has been demonstrated in clinical studies to be beneficial for those with mild to severe Alzheimer's disease and has been approved for the treatment of sleeplessness.

The risk of decreased mental acuity as well as coordination of movements (which includes impaired driving), worsening of depression or suicidal ideation, complex sleep behaviours (including sleepwalking and sleep driving), paralysis during sleep, and impaired respiratory function are just a few of the potential side effects **[17]**.

2.4. Treatments in brief [17].

Changes diseases progression

Treats cognitive symptoms (memory and thinking)

Name (Generic/Brand)	Approved for	Side effects
Aducanumab Aduhelm™	Alzheimer's disease (MCI or mild dementia)	ARIA, headache, and falls
Lecanemab Leqembi™	Alzheimer's disease (MCI or mild dementia)	ARIA, infusion-related reactions
Name (Generic/Brand)	**Approved for**	**Side effects**
Donepezil Aricept®	Mild to severe dementia due to Alzheimer's	Nausea, vomiting, loss of appetite, muscle cramps and increased frequency of bowel movements.
Galantamine Razadyne®	Mild to moderate dementia due to Alzheimer's	Nausea, vomiting, loss of appetite and increased frequency of bowel movements.
Rivastigmine Exelon®	Mild to moderate dementia due to Alzheimer's or Parkinson's	Nausea, vomiting, loss of appetite and increased frequency of bowel movements.
Memantine Namenda®	Moderate to severe dementia due to Alzheimer's	Headache, constipation, confusion, and dizziness.
Memantine + Donepezil Namzaric®	Moderate to severe dementia due to Alzheimer's	Nausea, vomiting, loss of appetite, increased frequency of bowel movements, headache, constipation, confusion, and dizziness.

Treats non-cognitive symptoms (behavioural and psychological)

Name (Generic/Brand)	Approved for	Side effects
Suvorexant Belsomra®	Insomnia, has been shown to be effective in people living with mild to moderate Alzheimer's disease	Impaired alertness and motor coordination, worsening of depression or suicidal thinking, complex sleep behaviours, sleep paralysis, compromised respiratory function.

CONCLUSION:

Finally, the perspective on recent dementia updates indicates a positive panorama of breakthroughs in research and knowledge of this complicated neurodegeneration condition. The worldwide impact of Alzheimer's disease highlights the critical need for effective ways to counteract its

devastating impacts on individuals and their families.

Recent advances in unravelling the underlying processes of Alzheimer's disease have resulted in a better understanding of its pathology. Furthermore, the introduction of novel treatment options, ranging from pharmaceutical therapies to lifestyle changes, has the potential to significantly decrease the disease's course and enhance the standard of life of individuals affected.

Nonetheless, problems persist, and there is much more to discover in the effort to eradicate Alzheimer's disease completely. Collaboration between academics, healthcare providers, and lawmakers will be critical in moving the research agenda ahead, converting findings into effective therapies, and eventually discovering a cure. As we continue to learn more about Alzheimer's disease, it is crucial to recognize the importance of supporting affected individuals and their caregivers. Public awareness and education initiatives can help reduce stigma, improve early detection rates, and ensure that those living with Alzheimer's disease receive the care and support they need.

Regarding conclusion that current advancements on Alzheimer's disease show that progress is being made, but the struggle to defeating this difficult ailment is ongoing. We may try to have a lasting effect by reducing the strain of Alzheimer's disease and enhancing the lives of people impacted by it through obedience and cooperation.

REFERENCES:

[1] Castellani RJ, Rolston RK, Smith MA. Alzheimer disease. Disease-a-month: DM. 2010; 56(9): 484-546. https://doi.org/10.1016/j.disamonth.2010.06.001

[2] It K, KNOW WD. What Is Alzheimer's Disease?. N Engl] Med. 1986; 314: 964-973.

[3] Qiu C, Kivipelto M, Von Strauss E. Epidemiology of Alzheimer's disease: occurrence, determinants, and strategies toward intervention. Dialogues in clinical neuroscience. 2022;11(2):111-128. https://doi.org/10.31887/DCNS.2009.11.2/cqiu

[4] Mayeux R, Stern Y. Epidemiology of Alzheimer disease. Cold Spring Harbor perspectives in medicine. 2012;2(8): a006239.

[5] Reitz C, Brayne C, Mayeux R. Epidemiology of Alzheimer disease. Nature Reviews Neurology. 2011; 7(3): 137-152. https://doi.org/10.1038/nrneurol.2011.2

[6] Karantzoulis S, Galvin JE. Distinguishing Alzheimer's disease from other major forms of dementia. Expert review of neurotherapeutics.

2011;11(11):1579-1591. https://doi.org/10.1586/ern.11.155

[7] Rizzi L, Rosset I, Roriz-Cruz M. Global epidemiology of dementia: Alzheimer's and vascular types. BioMed research international. 2014;2014. https://doi.org/10.1155/2014/908915

[8] Outeiro TF, Koss DJ, Erskine D, Walker L, Kurzawa-Akanbi M, Burn D, Donaghy P, Morris C, Taylor JP, Thomas A, Attems J. Dementia with Lewy bodies: an update and outlook. Molecular neurodegeneration. 2019; 14(1):1-18. https://doi.org/10.1186/s13024-019-0306-8

[9] Snowden JS, Neary D, Mann DM. Frontotemporal dementia. The British journal of psychiatry. 2002;180(2):140-143. https://doi.org/10.1192/bjp.180.2.140

[10] Custodio N, Montesinos R, Lira D, Herrera-Pérez E, Bardales Y, Valeriano-Lorenzo L. Mixed dementia: Review of the evidence. Dementia and Neuropsychology. 2017;11(4):364-370. https://doi.org/10.1590/1980-57642016dn11-040005

[11] Mendez MF. Early-onset Alzheimer disease. Neurologic clinics. 2017;35(2):263-281. https://doi.org/10.1016/j.ncl.2017.01.005

[12] Thakur AK, Kamboj P, Goswami K, Ahuja KJ. Pathophysiology and management of Alzheimer's disease: An overview. J anal pharm Res. 2018; 9(2): 226-235. https://doi.org/10.15406/japlr.2018.07.00230

[13] Abubakar MB, Sanusi KO, Ugusman A, Mohamed W, Kamal H, Ibrahim NH, Khoo CS, Kumar J. Alzheimer's disease: An update and insights into pathophysiology. Frontiers in Aging Neuroscience. 2022; 14: 742408. https://doi.org/10.3389/fnagi.2022.742408

[14] Ossenkoppele R, Pichet Binette A, Groot C, Smith R, Strandberg O, Palmqvist S, Stomrud E, Tideman P, Ohlsson T, Jögi J, Johnson K. Amyloid and tau PET-positive cognitively unimpaired individuals are at high risk for future cognitive decline. Nature Medicine. 2022;28(11):2381-2387. https://doi.org/10.1038/s41591-022-02049-x

[15] Paula VD, Guimarães FM, Diniz BS, Forlenza OV. Neurobiological pathways to Alzheimer's disease: Amyloid-beta, TAU protein or both?. Dementia &

Neuropsychologia. 2009; 3: 188-194. https://doi.org/10.1590/S1980-57642009DN30300003

[16] Sperling RA, Mormino EC, Schultz AP, Betensky RA, Papp KV, Amariglio RE, Hanseeuw BJ, Buckley R, Chhatwal J, Hedden T, Marshall GA. The impact of amyloid-beta and tau on prospective cognitive decline in older individuals. Annals of neurology. 2019;85(2):181-193. https://doi.org/10.1002/ana.25395

[17] Atri A. Current and future treatments in Alzheimer's disease. In Seminars in neurology. 2019; 39(02): 227-240. https://doi.org/10.1055/s-0039-1678581