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A STUDY OF ALZHEIMER DISEASE CURRENT TREATMENT AND NEW STRATERGIES: A REVIEW

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ABSTRACT

Alzheimer's disease (AD) is a progressive and irreversible neurodegenerative disorder characterized by memory loss, cognitive impairment, and behavioral abnormalities. Affecting millions worldwide, it places a major burden on patients, families, and healthcare systems. The complex pathology of AD includes β-amyloid plaques, tau protein hyperphosphorylation, synaptic dysfunction, oxidative stress, and chronic neuroinflammation. Current treatments primarily offer symptomatic relief but fail to halt or reverse disease progression. In recent years, significant advancements have emerged involving monoclonal antibodies, gene-modifying therapies, stem cell interventions, nanomedicine, and multi-target drug approaches. This review highlights the current pharmacological therapies and explores promising new strategies under investigation for more effective management of Alzheimer's disease.

Keywords: Alzheimer's Disease, Β-Amyloid, Tau Protein, Neurodegeneration, Monoclonal Antibodies, Novel Therapies, Cholinesterase Inhibitors.

1. INTRODUCTION

Alzheimer's disease (AD) is the leading cause of dementia, accounting for nearly 60–70% of cases globally. First described by Alois Alzheimer in 1906, the disease has since become one of the most challenging health issues affecting the aging population. AD is marked by progressive cognitive decline, including memory loss, language impairment, reduced problem-solving ability, and changes in personality and behavior.

The pathophysiology of AD is complex and multifactorial. Two hallmark features define the disease:

1. **Extracellular β-amyloid (Aβ) plaques**
2. **Intracellular neurofibrillary tangles (NFTs) formed by hyperphosphorylated tau protein**

Other important contributors include mitochondrial dysfunction, oxidative stress, synaptic loss, dysregulated calcium homeostasis, and chronic neuroinflammation.

Despite the rising prevalence of AD, curative treatments remain unavailable. Current therapies provide only symptomatic improvement and temporary slowing of disease progression. This gap has encouraged scientific exploration of innovative therapies such as monoclonal antibodies, gene therapy, neuroprotective agents, nanotechnology-based drug delivery, and lifestyle-modification strategies.

This article provides a detailed overview of the established treatments and highlights the newest developments in AD research.

2. METHODOLOGY

This review is based on an extensive analysis of published literature from:

- PubMed
- ScienceDirect
- Google Scholar
- Alzheimer's Association database
- ClinicalTrials.gov

Recent journals from 2010–2025, including peer-reviewed articles, clinical trial data, and meta-analyses, were evaluated. Emphasis was placed on:

- Current approved treatment options
- Mechanistic insights into AD pathology
- Novel drug candidates
- Emerging therapeutic strategies

Studies were selected for relevance, scientific accuracy, and up-to-date clinical evidence.

3. CURRENT TREATMENTS FOR ALZHEIMER'S DISEASE

Current AD treatments fall under **two categories**:

A. Symptomatic Treatments

1. **Cholinesterase Inhibitors**

- Donepezil
- Rivastigmine
- Galantamine

Mechanism: Increase acetylcholine levels in the brain to improve memory and cognitive function.

2. **NMDA Receptor Antagonist**

- Memantine

Mechanism: Regulates glutamate activity to prevent excitotoxicity and neuronal death.

3. **Combination Therapy**

- Memantine + Donepezil

Shows enhanced efficacy in moderate to severe AD.

B. Recently Approved Disease-Modifying Therapies

1. **Aducanumab (Aduhelm®)**

- Monoclonal antibody targeting β-amyloid
- Removes amyloid plaques
- Controversial approval due to mixed clinical outcomes

2. **Lecanemab (Leqembi®)**

- FDA approved (2023)
- Slows cognitive decline by reducing amyloid burden
- Better clinical evidence than aducanumab

3. **Donanemab (Pending Approval/Approved in Some Regions)**

- Targets Aβ plaques
- Shows promise for early-stage AD

4. RESULTS AND DISCUSSION

4.1 Amyloid Cascade Hypothesis

Excessive accumulation of β-amyloid peptides leads to plaque formation, neurotoxicity, synaptic damage, and neuron death.

4.2 Tau Pathology

Hyperphosphorylated tau causes intracellular tangles disrupting nutrient transport and destabilizing microtubules.

4.3 Neuroinflammation

Activated microglia and astrocytes release inflammatory mediators that exacerbate neurodegeneration.

4.4 Oxidative Stress & Mitochondrial Dysfunction

Leads to energy deficiency, ROS accumulation, and neuronal apoptosis.

5. NEW & EMERGING THERAPEUTIC STRATEGIES

5.1 Monoclonal Antibody Therapy

Designed to remove amyloid plaques or inhibit tau accumulation.

5.2 Anti-Tau Therapies

- Anti-tau antibodies
- Tau aggregation inhibitors
- Microtubule stabilizers

5.3 Gene Therapy

- APOE4 gene modification
- CRISPR-based gene editing

- Antisense oligonucleotides targeting harmful protein production

5.4 Stem Cell Therapy

Mesenchymal stem cells (MSCs) show potential in regenerating neurons and reducing inflammation.

5.5 Nanotechnology-Based Drug Delivery

Nanoparticles enable targeted delivery across the blood-brain barrier and enhance drug bioavailability.

5.6 Neuroprotective Agents Under Study

- Curcumin analogues
- Resveratrol
- BDNF modulators

5.7 Lifestyle & Non-Pharmacological Interventions

- Mediterranean diet
- Aerobic exercise
- Cognitive training
- Sleep optimization
- Social engagement

6. CONCLUSION

Alzheimer’s disease remains a major global health challenge, with increasing prevalence and significant socioeconomic impact. Despite advances, current approved therapies offer limited disease-modifying benefits. However, recent breakthroughs—especially monoclonal antibodies, gene therapy, stem cell-based therapies, and nanomedicine—represent encouraging steps toward more effective treatments. Continued research, early diagnosis, and personalized medicine strategies have the potential to transform AD management in the coming decades.

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