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EMERGING PHARMACOLOGICAL STRATEGIES IN ALZHEIMER'S DISEASE: A REVIEW OF NOVEL THERAPEUTIC TARGETS AND DRUG INNOVATIONS

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ABSTRACT

Alzheimer's disease (AD) is the most prevalent neurodegenerative disorder worldwide and constitutes a significant public health concern due to its rising incidence and the absence of curative therapies. This review synthesizes recent pharmacological progress in the treatment of AD, with particular emphasis on emerging therapeutic strategies and investigational drug classes. The analysis draws on clinical trial data, regulatory documents, and peer-reviewed literature published between 2017 and 2025, identified through major scientific databases including PubMed, Scopus, and ClinicalTrials.gov. The most promising advances are associated with monoclonal antibodies directed against amyloid-beta pathology, such as aducanumab, lecanemab, and donanemab, which show potential to modify disease progression but also raise concerns related to efficacy, safety, and regulatory approval. Additional innovative approaches, including tau-targeted therapies, gene editing technologies such as CRISPR-Cas9, and RNA interference (RNAi), present new therapeutic opportunities, though they remain limited by challenges such as amyloid-related imaging abnormalities (ARIA), restricted delivery across the blood–brain barrier, and uncertainties regarding long-term clinical outcomes. While currently available pharmacological options are insufficient to halt or reverse AD, recent advancements, particularly in antibody-based therapies, represent an important step toward a new therapeutic era. Nevertheless, cautious interpretation of preliminary findings and rigorous clinical validation remain essential before these strategies can be translated into widespread clinical practice.

KEYWORDS

Alzheimer's Disease, Aducanumab, Lecanemab, Donanemab, Monoclonal Antibodies

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Introduction.

Alzheimer's disease (AD) poses one of the most critical health challenges of the 21st century, both from epidemiological and societal perspectives. As of 2019, an estimated 57, 4 million people worldwide were living with dementia, with projections indicating a rise to 152.8 million by 2050. (GBD 2019 Dementia Forecasting Collaborators, 2022) Alzheimer's disease is the primary cause of dementia, accounting for approximately 60–70% of all cases (WHO, 2025). In the United States alone, there were an estimated 6.9 million individuals aged 65 and older living with Alzheimer's disease in 2024, a figure expected to double to 13.8 million by 2060. (Alzheimers Dement, 2024)

Caregivers of individuals with dementia frequently experience significant emotional, physical, and financial strain, which may contribute to a deterioration of their own health. A 2023 study found that caregivers of individuals with dementia reported higher levels of stress and depressive symptoms compared to caregivers of patients with mild cognitive impairment. (Meyer OL et al., 2024) The rising number of people affected by AD, coupled with its societal and economic burden, highlights the urgent need for effective strategies for prevention, early diagnosis, and treatment.

Pharmacological management of AD currently relies on cholinesterase inhibitors (AChEIs) and memantine, which primarily provide symptomatic relief of cognitive decline. While AChEIs demonstrate moderate efficacy, their effectiveness varies by dementia subtype, with more noticeable effects in Parkinson's disease dementia than in AD. (Knight R et al., 2018) Memantine, an NMDA receptor antagonist, may alleviate certain symptoms, but its impact on apathy and other neuropsychiatric features remains limited. Additionally, AChEI therapy is often associated with adverse events, including gastrointestinal symptoms, dizziness, confusion, and headaches, which can reduce treatment tolerability. (Chin E et al., 2022)

Combination therapy (AChEI + memantine) has shown statistically significant benefits in cognitive performance and global clinical impression, though the clinical relevance of these outcomes remains debatable.

(Glinz D et al., 2019) Therefore, there is an urgent need to develop novel disease-modifying therapies that are both more effective and better tolerated.

In summary, Alzheimer's disease represents an escalating global medical and societal concern. Currently approved treatments offer only limited symptomatic relief, and the absence of effective disease-modifying drugs underscores the necessity of pursuing innovative pharmacological strategies that could more meaningfully alter disease progression.

Methodology

This review was conducted as a narrative synthesis of recent advances in pharmacological treatment strategies for Alzheimer's disease. Literature published between January 2017 and May 2025 was systematically identified through searches in PubMed, Scopus, and ClinicalTrials.gov, using combinations of keywords such as"Alzheimer's disease, ""pharmacological treatment, ""monoclonal antibodies, ""amyloid-beta, ""tau therapies, ""gene editing, " and"RNA interference."

Only peer-reviewed original studies, clinical trials, and regulatory reports available in English were included. Editorials, conference abstracts, case reports, and studies lacking primary data were excluded.

A critical appraisal of clinical relevance, therapeutic mechanisms, and reported outcomes was performed to highlight the most significant pharmacological innovations. Particular emphasis was placed on monoclonal antibody therapies and emerging experimental approaches, with attention to both therapeutic potential and limitations related to efficacy, safety, and translational feasibility.

Results

1. Emerging Pharmacological Strategies for the Treatment of Alzheimer's Disease

Alzheimer's disease remains the leading cause of dementia in the elderly population. Despite decades of intensive research, the development of disease-modifying treatments had long remained elusive. In recent years, however, a significant breakthrough has emerged with the advent of targeted therapies aimed at beta-amyloid - a peptide implicated in the formation of amyloid plaques within the brains of individuals with AD. Among the most prominent agents are monoclonal antibodies such as aducanumab and lecanemab, which have been engineered to facilitate the clearance of pathogenic amyloid aggregates. (Figure 1)

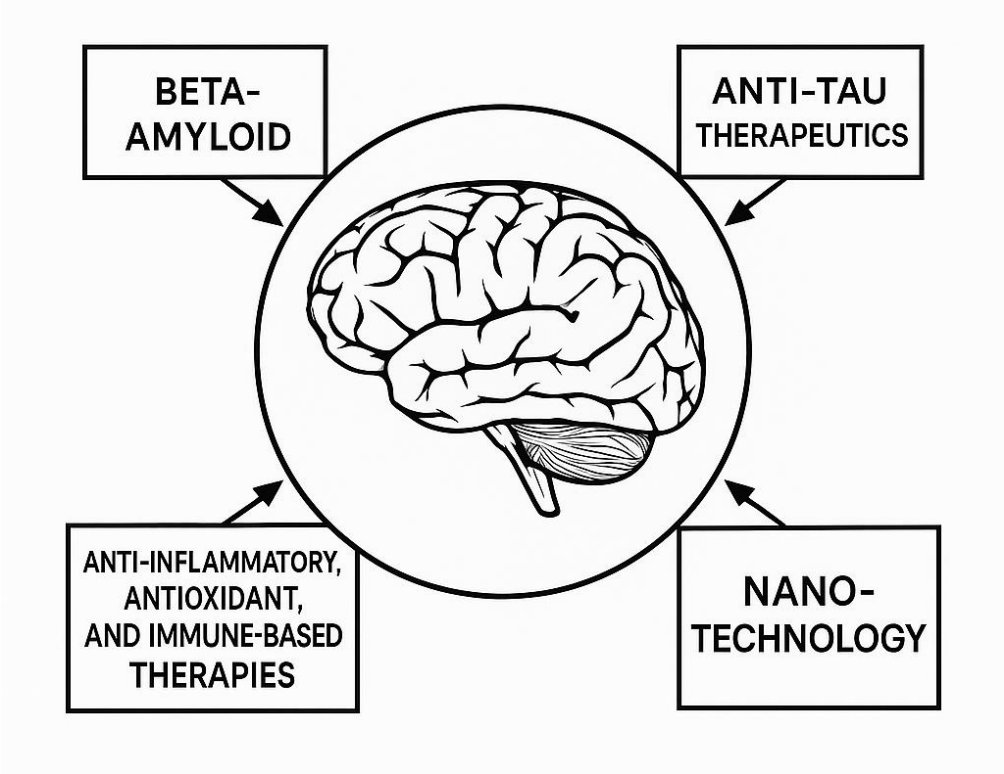

Fig. 1. *Emerging pharmacological strategies in Alzheimer's Disease*

1.1 Therapeutic Strategies Targeting Beta-Amyloid Pathology

Lecanemab is a humanized IgG1 monoclonal antibody designed to selectively bind to soluble beta-amyloid protofibrils, a particularly neurotoxic form of amyloid-beta aggregates implicated in the pathogenesis of Alzheimer's disease. By targeting and facilitating the clearance of these protofibrils, lecanemab aims to reduce amyloid-related neurotoxicity and slow the progression of cognitive decline in individuals with early-stage Alzheimer's disease. (van Dyck CH et al., 2022)

The clinical efficacy and safety of lecanemab were rigorously evaluated in the phase III CLARITY AD trial, which enrolled 1, 795 participants with mild cognitive impairment or mild dementia due to Alzheimer's disease. Over an 18-month treatment period, lecanemab demonstrated a statistically significant 27% reduction in the rate of cognitive decline compared to placebo. In addition to these clinical effects, biomarker analyses revealed a substantial reduction in cerebral amyloid burden, as confirmed through positron emission tomography (PET), as well as decreased plasma levels of phosphorylated tau at threonine 181 (p-tau181), a marker associated with neurodegeneration. (McDade E et al., 2022) Lecanemab was generally well tolerated, although amyloid-related imaging abnormalities (ARIA), including vasogenic edema (ARIA-E) and cerebral microhemorrhages (ARIA-H), were observed. Importantly, the incidence of these adverse events was lower than that reported with earlier-generation anti-amyloid therapies, such as aducanumab.

Based on the strength of these findings, lecanemab received full approval from the U.S. Food and Drug Administration (FDA) in July 2023, marking a significant milestone in the treatment landscape for Alzheimer's disease. As a second-generation anti-amyloid antibody, it reflects the growing refinement in immunotherapeutic strategies targeting disease-modifying pathways in neurodegeneration. (Park A, 2023, McDade E et al. 2022, Kim BH et al., 2025, Kim AY et al., 2024)

Aducanumab, developed by Biogen, became the first anti-amyloid monoclonal antibody to receive accelerated approval from the U.S. Food and Drug Administration (FDA) in 2021 for the treatment of Alzheimer's disease. The drug works by selectively targeting and removing beta-amyloid aggregates from the brain one of the hallmark pathologies of Alzheimer's. Despite its promising mechanism, aducanumab's clinical performance has been highly controversial.

The phase III clinical trials, EMERGE and ENGAGE, produced conflicting outcomes. While EMERGE suggested a modest slowing of cognitive decline at the highest doses, ENGAGE failed to replicate these results. This inconsistency in efficacy, along with methodological concerns, fueled ongoing debate within the scientific and medical communities.

Additional concerns were raised about the drug's safety profile. A significant number of patients, particularly those carrying the APOE ε4 allele, experienced amyloid-related imaging abnormalities (ARIA), such as brain swelling or microhemorrhages. These side effects, combined with the high cost and limited clinical benefit, led to scrutiny from regulators. While the FDA approved the drug, the European Medicines Agency (EMA) declined authorization, citing insufficient evidence to support its use.

Ultimately, due to limited adoption, controversy over its effectiveness, and challenges in real-world implementation, Biogen announced in 2024 that it would discontinue the commercialization of aducanumab (marketed as Aduhelm). (Budd H et al., 2022, Rahman A et al., 2023, Nisticò R et al., 2021, Alexander GC et al., 2021, Sharma A et al., 2025)

Meta-analyses comparing the efficacy of lecanemab and aducanumab indicate that lecanemab exhibits a superior clinical profile, demonstrating enhanced effectiveness in amyloid reduction as well as a more favorable safety profile. These reviews emphasize that therapeutic strategies targeting protofibrils may represent a more effective approach than those aimed at the removal of mature plaques. (Chhabra A et al., 2024, Wu W et al., 2023)

Donanemab is a monoclonal IgG1 antibody that targets a pathologically modified form of beta-amyloid known as N3pG. By selectively binding to these abnormal aggregates, donanemab facilitates their accelerated clearance from the brain, aiming to slow cognitive deterioration in individuals with early symptomatic Alzheimer's disease. A unique feature of this therapy is that treatment is discontinued once a predefined threshold of amyloid clearance is reached, potentially reducing the treatment burden and associated healthcare costs. (Sims JR et al., 2023)

In the phase 3 TRAILBLAZER-ALZ 2 trial, which enrolled 1, 736 participants with early symptomatic Alzheimer's disease, donanemab treatment led to a 29% reduction in cognitive decline across the overall population over 76 weeks, as measured by the integrated Alzheimer's Disease Rating Scale (iADRS). In a subgroup of patients with low to moderate tau pathology, the benefit was even greater, with a 35% reduction in the rate of cognitive decline compared to placebo. Functional outcomes also improved, with a 40% slower

decline in daily living activities (ADCS-iADL) and a 39% reduction in the risk of clinical progression. Notably, 47% of treated patients experienced no clinical worsening after one year, compared to 29% in the placebo group. (Sims JR et al., 2023; Manap AS et al. 2024)

Additionally, 52% of participants reached the amyloid clearance threshold within the first year, allowing for treatment discontinuation, which highlights an advantage over other anti-amyloid therapies such as lecanemab, which does not offer a fixed-duration approach. However, donanemab is also associated with a higher incidence of amyloid-related imaging abnormalities (ARIA). ARIA-E (cerebral edema) occurred in 24% of treated patients 6.1% of whom experienced symptoms—while ARIA-H (microhemorrhages) was reported in 31.4%. Although most ARIA cases were mild or moderate and manageable, three treatment-related deaths were reported. (Sims JR et al., 2023; Barakos J et al., 2022)

Despite its promising efficacy, safety concerns have delayed full regulatory approval. In mid-2023, the FDA postponed its decision on donanemab and requested an independent review of the trial data, particularly in light of the ARIA-related adverse events and mortality. (Sims JR et al., 2023; Manap AS et al. 2024)

Table 1. Summary of Targeted Therapeutics Aimed at Beta-Amyloid in Alzheimer's Disease

Drug	Number of Participants	Efficacy Summary	Common Adverse Effects	FDA Approval Status
Lecanemab	1,795 (CLARITY AD trial)	27% reduction in cognitive decline over 18 months; significant reduction in amyloid burden (PET imaging); decreased plasma p-tau181 levels; improved biomarker and clinical outcomes in early-stage AD patients.	ARIA-E (edema), ARIA-H (microhemorrhages); lower incidence compared to aducanumab.	Fully approved by FDA in July 2023.
Aducanumab	~3,300 (EMERGE + ENGAGE)	EMERGE showed modest slowing of cognitive decline at high doses; ENGAGE failed to replicate findings. High inconsistency and limited benefit. Amyloid plaque reduction confirmed, but clinical efficacy remains controversial.	High incidence of ARIA, particularly in APOE ε4 carriers; brain swelling, microhemorrhages.	Accelerated FDA approval in 2021; commercializati on discontinued in 2024; not approved by EMA.
Donanemab	1,736 (TRAILBLA ZER-ALZ 2)	29% reduction in overall cognitive decline (iADRS); 35% in low-moderate tau subgroup; 40% slower decline in ADLs (ADCS-iADL); 39% reduced risk of disease progression; 47% showed no worsening after 1 year. 52% reached amyloid clearance threshold within 12 months, enabling treatment discontinuation.	ARIA-E in 24% (6.1% symptomatic); ARIA-H in 31.4%; 3 treatment-related deaths reported.	FDA decision delayed in 2023 pending independent safety review; not yet fully approved.

1.2 Advances in Anti-Tau Therapeutics for Alzheimer's Disease

Tau protein, which undergoes pathological hyperphosphorylation and aggregation in the brains of Alzheimer's disease (AD) patients, has emerged as a crucial therapeutic target in recent years. Ongoing research focuses on diverse strategies to modulate tau, including inhibition of its phosphorylation, prevention of aggregation, immunotherapy, and modulation of glycosylation pathways. (Guo Y et al., 2022) One of the most advanced approaches targets glycogen synthase kinase-3β (GSK-3β), a key enzyme involved in tau hyperphosphorylation. Tideglusib, an irreversible inhibitor of GSK-3β, demonstrated in preclinical models a reduction in phosphorylated tau levels, decreased β-amyloid plaque deposition, and cognitive improvement.

However, phase 2 clinical trials failed to show significant benefits in patients with mild to moderate AD, leading to discontinuation of its development. (Guo Y et al., 2022)

Lithium, a reversible GSK-3β inhibitor, has also been evaluated for its neuroprotective potential. Animal studies suggest that chronic lithium administration may prevent tau phosphorylation, but clinical evidence supporting its efficacy in humans remains inconclusive. (Xia et al., 2021)

Another promising therapeutic avenue involves molecules that inhibit tau aggregation. TRx0237 (also known as LMTM) showed encouraging preclinical results; however, phase 3 trials did not demonstrate a significant impact on slowing cognitive decline. (Manap AS et al., 2024)

A novel molecule, RI-AG03, binds simultaneously to two distinct sites on tau protein, effectively inhibiting its aggregation. Preclinical studies in Drosophila models and cell lines have confirmed its ability to reduce tau pathology, supporting the advancement to clinical trials. (Swartz T., 2024)

Increasing tau glycosylation through inhibition of the O-GlcNAcase enzyme represents an emerging therapeutic strategy. ASN51, an OGA inhibitor developed by Asceneuron, is currently in phase 2 clinical trials. (Philpott J, 2025) Similarly, ceperogastat (LY3372689) has completed phase 1 studies with a favorable safety profile and is preparing for further clinical evaluation. (Kielbasa W et al., 2024)

Immunotherapy remains a critical pillar of anti-tau treatment. The active vaccine AADvac1 elicited strong immunogenicity in patients with mild AD, but this did not translate into a significant delay in disease progression. In contrast, ACI-35, another active vaccine, successfully induced antibodies against phosphorylated tau during phase 1 trials, with subsequent phases currently underway. (Chen Y et al., 2023, Chen H et al., 2023)

Advances in diagnostics are improving the evaluation of anti-tau therapies. Researchers at the University of Washington have developed a blood test capable of detecting the crystalline form of tau protein, which correlates strongly with disease progression. This tool holds promise for enhancing Alzheimer's disease diagnosis and monitoring treatment response. (Park A, 2025)

Although some therapeutic approaches, such as GSK-3β inhibitors, have demonstrated limited clinical efficacy, the development of new drug classes including OGA inhibitors and anti-tau vaccines offers renewed hope for Alzheimer's disease treatment. Ongoing preclinical and clinical studies may soon yield breakthroughs in addressing this currently incurable neurodegenerative disorder.

1.3 The Role of Anti-Inflammatory, Antioxidant, and Immune-Based Therapies in Alzheimer's Disease Management

Neuroinflammation is recognized as a key pathogenic factor in Alzheimer's disease. Recent studies highlight the involvement of signaling pathways such as NF-κB, MAPK, and the NLRP3 inflammasome in sustaining chronic inflammation within the brains of AD patients. Among promising therapeutic candidates are natural plant-derived compounds with anti-inflammatory properties, such as tanshinone IIA. This compound has demonstrated the ability to suppress microglial activation and reduce the expression of pro-inflammatory cytokines, thereby potentially mitigating neuronal damage and enhancing cognitive functions. (Zheng Y et al., 2023)

Simultaneously, there is growing interest in employing nanocarrier systems for targeted delivery of anti-inflammatory agents to the central nervous system. These advanced technologies facilitate crossing the blood-brain barrier and improve therapeutic efficacy while minimizing adverse effects. (Chu J et al., 2024)

Oxidative stress is a major contributor to neurodegeneration in AD. Numerous preclinical and clinical investigations focus on the role of natural antioxidants such as resveratrol, curcumin, and ferulic acid. Resveratrol activates the SIRT1/AMPK pathway, which helps reduce reactive oxygen species (ROS) levels and prevents mitochondrial dysfunction. It also inhibits neuroinflammatory transcription factors like NF-κB. (Conti Filho CE et al., 2023) Curcumin exhibits free radical scavenging properties and attenuates microglial and astrocyte activation, potentially slowing cognitive decline progression. (Twarowski B et al., 2023) However, the clinical efficacy of classical antioxidants such as vitamin E remains controversial, primarily due to poor bioavailability, insufficient CNS penetration, and the complexity of oxidative stress mechanisms in AD. (Pappolla MA et al., 2024)

Immune system modulation represents one of the most innovative therapeutic strategies in recent years. Remarkably, the Bacillus Calmette-Guérin (BCG) vaccine, originally developed against tuberculosis, has been associated with a reduced risk of developing AD. This effect is thought to stem from enhanced immune responses, decreased neuroinflammation, and improved clearance of amyloid-beta deposits in the brain. (Robson D, 2024) Another approach involves blocking pro-inflammatory mediators such as tumor necrosis factor-alpha (TNF-α).

TNF inhibitors like etanercept have shown potential in improving cognitive function in patients with mild AD by reducing microglial activation and pro-inflammatory cytokine levels. (Weinstock M, 2024)

Emerging therapeutic strategies in Alzheimer's disease clearly extend beyond the traditional amyloid and tau targets. Anti-inflammatory, antioxidant, and immunotherapeutic approaches offer promising avenues to modulate neurodegenerative processes and enhance patient quality of life. While many of these interventions remain under clinical or preclinical investigation, ongoing research may pave the way for breakthroughs in AD treatment.

1.4 Innovative Drug Delivery Strategies for Alzheimer's Disease: Nanotechnology Approaches to Overcome the Blood-Brain Barrier

One of the major challenges in effective Alzheimer's disease therapy remains the difficulty of delivering drugs to the brain due to the presence of the blood-brain barrier (BBB). In recent years, nanotechnology has opened new avenues for overcoming this barrier and enabling targeted delivery of therapeutics to the central nervous system.

Nanoparticles, which are drug carriers ranging in size from 1 to 100 nanometers, can be engineered to improve the pharmacokinetic properties of therapeutic agents. Various types of nanocarriers including liposomes, dendrimers, metallic nanoparticles, and polymer-based carriers have demonstrated the capacity to cross the BBB and allow for controlled, site-specific drug release within the brain. Through surface functionalization, such as ligand conjugation targeting specific receptors, these nanocarriers can selectively bind to brain endothelial cells and penetrate into the brain's interstitial space. This targeted delivery enhances therapeutic efficacy while minimizing systemic toxicity. (Dong N et al., 2025)

Recent nanotechnology-based drug delivery systems include both conventional cognitive-enhancing drugs and novel biologics, such as monoclonal antibodies. A notable example is trontinemab, which employs Brainshuttle™ technology to effectively traverse the BBB. Preclinical studies have shown that trontinemab binds amyloid-beta plaques and facilitates their clearance, with fewer adverse effects compared to traditional anti-amyloid therapies. (Pathak K et al., 2025)

Furthermore, noble metal nanoparticles, such as gold and silver, are being explored as carriers for anti-inflammatory, neuroprotective, and antioxidant agents. Their unique physicochemical characteristics support efficient BBB penetration and synergistic interaction with active therapeutic substances in AD treatment. (Panghal A et al., 2024)

Nanocarriers offer numerous advantages including improved drug bioavailability, protection from enzymatic degradation, reduced systemic toxicity, and controlled release of active compounds. Nevertheless, challenges remain, particularly concerning the long-term safety of nanoparticles, potential tissue accumulation, and difficulties in standardizing production for clinical applications. (Altinoglu G et al., 2020, Li L et al., 2023)

The ongoing development of nanotechnology has the potential to revolutionize AD therapy. Notably, research on"smart nanoparticles" capable of recognizing pathological brain structures and responding in a personalized manner paves the way for precision medicine approaches. However, further translational research and multicenter clinical trials are essential to validate the efficacy and safety of these innovative strategies. (Martín-Rapun R et al., 2017)

Overall, the application of nanotechnology in Alzheimer's treatment represents one of the most promising and rapidly advancing research fields. Innovative drug carriers facilitate efficient BBB penetration and targeted action on pathological brain structures. While many of these technologies are still at the experimental stage, early results are highly encouraging and may lead to breakthroughs in the management of this challenging disease.

2. The Future of Alzheimer's Disease Therapy

2.1 Genetic and Molecular Strategies for Therapeutic Intervention: RNAi and CRISPR

In Alzheimer's disease, gene and molecular therapies such as RNA interference (RNAi) and CRISPR-Cas9 genome editing represent innovative strategies aimed at modifying pathogenic mechanisms at the genetic level. RNAi approaches are being explored for their ability to reduce the production of disease-associated proteins, including beta-amyloid and tau, by selectively silencing the genes that encode them. Experimental studies using neural stem cells have demonstrated that RNAi targeting of BACE1, the gene encoding β-secretase, can effectively lower its expression, resulting in reduced β-amyloid generation. (Cummings et al., 2022)

CRISPR-based techniques allow for precise modification of genes such as APP and PSEN1, potentially addressing the root genetic causes of Alzheimer's disease. However, clinical translation faces significant challenges, including safe and efficient delivery across the blood–brain barrier, off-target genetic alterations,

immune responses, and a lack of long-term safety data. Although current preclinical studies and limited clinical trials have yielded promising results, they emphasize the need for further comprehensive investigation. (Barman et al., 2020)

Therefore, while RNAi and CRISPR hold considerable promise for transforming Alzheimer's treatment, their widespread clinical application will depend on extensive validation of their efficacy and safety. (Cummings et al., 2022; Barman et al., 2020; Tripathi et al., 2024; Bhardwaj et al., 2022)

2.2 The Role of Precision Medicine and Predictive Biomarkers in Advancing Alzheimer's Disease Treatment

Traditional therapeutic approaches often fall short due to the heterogeneous pathophysiology of AD. In response to these challenges, personalized medicine based on predictive biomarkers is gaining importance as a promising therapeutic strategy.

Biomarkers play a crucial role in identifying individuals at risk of developing AD and in monitoring disease progression. Genetic markers, such as the APOE ε4 allele, are strongly associated with an increased risk of AD and earlier disease onset. Studies have shown that individuals carrying one copy of this allele have a threefold higher risk, while those with two copies face a twelvefold greater risk of developing AD compared to individuals with the ε3 allele. (Bougea A et al., 2024) Furthermore, genome-wide association studies (GWAS) have identified over 30 genetic loci linked to the risk of late-onset AD, including genes involved in immune response, lipid metabolism, and synaptic function. (Bougea A et al., 2024)

Recent technological advances have enabled the development of non-invasive diagnostic tests, such as blood assays detecting phosphorylated tau protein (p-tau217), which demonstrate high accuracy in identifying early stages of AD (Schindler S, 2023). Additionally, analysis of circulating cell-free DNA (cfDNA) and its methylation patterns offers new possibilities for detecting epigenetic changes associated with AD, potentially leading to novel predictive biomarkers. (Bahado-Singh RO et al., 2023)

The integration of genetic, epigenetic, and clinical data using advanced analytical tools like artificial intelligence (AI) allows for the creation of personalized risk models and disease progression forecasts. AI also supports the identification of new therapeutic targets and treatment optimization. Despite promising progress, the implementation of personalized medicine in clinical practice faces challenges, including standardization of biomarker tests, technology accessibility, and ethical concerns related to genetic disease prediction. Continued research and infrastructure development are essential to facilitate the integration of personalized medicine into care for patients with AD. (Kale M et al., 2024; Zhou X et al., 2023)

2.3 An Integrated Approach to Alzheimer's Disease: Pharmacological and Lifestyle Interventions

Alzheimer's disease (AD) requires a comprehensive therapeutic approach due to the complex nature of its pathogenesis. Current pharmacological treatments, such as anti-amyloid antibodies (e.g., lecanemab, donanemab), show moderate effectiveness in slowing disease progression, particularly in its early stages. However, their impact may be enhanced when combined with lifestyle interventions.

Research indicates that a plant-based diet, regular physical activity, stress reduction, and social support positively influence cognitive function and disease biomarkers. Programs like the one developed by Dean Ornish have demonstrated improvements in cognitive function among individuals with mild cognitive impairment. (Park A, 2024)

An increasing body of evidence suggests that combining pharmacological treatment with lifestyle modifications can yield better outcomes than either approach alone. This integrated strategy may also facilitate the personalization of therapy based on biomarkers and genetic data. (Arora S et al., 2023)

3. Discussion and conclusions

Alzheimer's disease remains the most prevalent neurodegenerative disorder worldwide, presenting a growing medical and societal challenge due to aging populations and the lack of curative treatment. Currently approved pharmacological therapies, such as cholinesterase inhibitors and memantine, provide only limited symptomatic relief and do not significantly alter the disease course. Recently developed monoclonal antibodies targeting amyloid-beta pathology, such as aducanumab, lecanemab, and donanemab, offer some promise in slowing disease progression, but concerns persist regarding their efficacy, safety profiles, and regulatory acceptance. Emerging investigational approaches, including tau-targeted therapies, gene editing technologies (e.g., CRISPR-Cas9), and RNA interference (RNAi), introduce novel therapeutic possibilities.

However, their clinical application faces numerous challenges, such as drug delivery across the blood–brain barrier, adverse effects like amyloid-related imaging abnormalities (ARIA), and the need for long-term safety and efficacy data. Given the projected rise in AD prevalence and the significant burden on caregivers, there is an urgent need for better-tolerated and more effective disease-modifying therapies.

Despite recent progress, ongoing clinical research and the pursuit of innovative treatment strategies remain essential to meet the complex needs of patients and their families. Continued investment in new approaches is critical to advancing toward truly transformative care in Alzheimer's disease.

Disclosure:
Author Contributions:
The authors confirm contribution to the paper as follows:

Conceptualization: JS
Methodology: GS
Software: non applicable
Check: RF, ZK
Formal analysis: KG, AK
Investigation: WP, DG
Resources: AS
Data curation: RF, ZK
Writing - rough preparation: GS, JS
Writing - review and editing: GS, JS, GK
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