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ADVANCES IN TARGETED DRUG DELIVERY SYSTEMS FOR ALZHEIMER'S DISEASE: EMERGING TECHNOLOGIES AND THERAPEUTIC OPPORTUNITIES

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1. INTRODUCTION

Alzheimer's Disease is the most common cause of dementia and is characterized by progressive cognitive decline and memory impairment. It mainly affects elderly individuals and accounts for nearly 60–70% of dementia cases worldwide. The disease is associated with neuronal degeneration, especially in the hippocampus and cerebral cortex. The major pathological hallmarks include extracellular amyloid-β plaques and intracellular neurofibrillary tangles formed by hyperphosphorylated tau protein. Clinical symptoms include memory loss, impaired reasoning, confusion, behavioral changes, and reduced ability to perform daily activities. Age is considered the strongest risk factor, while genetics, cardiovascular diseases, diabetes, and lifestyle factors also contribute to disease progression.[1] Oxidative stress, mitochondrial dysfunction, and neuroinflammation further aggravate neuronal damage. Diagnosis involves cognitive assessment, neuroimaging techniques such as MRI and PET scans, and biomarker analysis. Current pharmacological therapies include cholinesterase inhibitors and NMDA receptor antagonists, which mainly provide symptomatic relief.[2] Recent research focuses on nanotechnology-based drug delivery systems and disease-modifying therapies for improved brain targeting and therapeutic efficacy. Early diagnosis and preventive lifestyle modifications may help delay disease progression and improve patient quality of life.[3]

Alzheimer's Disease is one of the fastest-growing neurological disorders worldwide and represents a major public health challenge. More than 55 million people globally are living with dementia, with Alzheimer's disease accounting for nearly 60–70% of cases. The prevalence is projected to increase rapidly due to aging populations and increased life expectancy. Low- and middle-income countries are expected to experience the highest rise in disease burden. Alzheimer's disease causes progressive cognitive impairment, disability, and loss of independence, significantly affecting patient quality of life. The disease also places substantial emotional and physical stress on caregivers and families. Global dementia-related healthcare expenditures exceed US$1 trillion annually, including medical treatment, nursing care, and informal caregiving costs. Reduced productivity and long-term dependency further intensify the socioeconomic burden. Limited awareness and inadequate healthcare resources in developing nations often delay diagnosis and management. Hence, improved healthcare policies, early diagnosis, and advanced therapeutic strategies are essential to reduce the global impact of Alzheimer's disease.[4,5]

Conventional therapies for Alzheimer's disease mainly provide symptomatic relief and do not completely halt or reverse disease progression. Currently approved drugs such as cholinesterase inhibitors (donepezil, rivastigmine, and galantamine) and the NMDA receptor antagonist memantine offer only temporary improvement in cognition and behavior. These therapies are less effective in advanced stages of the disease and are often associated with adverse effects including nausea, vomiting, dizziness, and gastrointestinal disturbances. One major limitation is the inability of many therapeutic agents to efficiently cross the blood–brain barrier, resulting in poor drug delivery to the brain.[6,7] Conventional drugs also exhibit low bioavailability, short half-life, and non-specific distribution, which may reduce therapeutic efficacy and increase systemic side effects. In addition, Alzheimer's disease involves multiple pathological pathways such as amyloid-β aggregation, tau hyperphosphorylation, oxidative stress, and neuroinflammation, whereas conventional therapies generally target only a single mechanism. Delayed diagnosis and late initiation of treatment further limit clinical outcomes. Therefore, there is a growing need for advanced therapeutic strategies such as targeted drug delivery systems and nanotechnology-based approaches

to improve treatment efficacy and disease management.[8]

Targeted drug delivery systems have gained significant importance in Alzheimer's disease (AD) treatment due to the limitations associated with conventional therapies. One of the major challenges in AD management is the presence of the blood–brain barrier (BBB), which restricts the entry of most therapeutic agents into the brain. As a result, many drugs exhibit poor brain bioavailability and limited therapeutic efficacy.[9] Conventional administration also leads to non-specific drug distribution, systemic side effects, and rapid drug degradation. Since Alzheimer's disease involves multiple pathological mechanisms such as amyloid-β plaque formation, tau protein aggregation, oxidative stress, and neuro-inflammation, effective treatment requires precise and sustained delivery of therapeutics to affected brain regions. Targeted drug delivery systems, including nanoparticles, liposomes, dendrimers, and polymeric carriers, can improve drug stability, enhance BBB penetration, provide controlled drug release, and increase drug concentration at the target site. These systems may also reduce dosing frequency and minimize adverse effects. Furthermore, targeted nano-carriers can be functionalized with ligands for receptor-mediated transport across the BBB, thereby enhancing therapeutic outcomes. Therefore, targeted drug delivery approaches offer promising opportunities for improving the safety and efficacy of Alzheimer's disease treatment.[10]

This review focuses on recent advances in targeted drug delivery systems for the management of Alzheimer's Disease and their potential to overcome the limitations of conventional therapies. The review discusses the pathophysiology of Alzheimer's disease and the major barriers associated with effective brain drug delivery, particularly the blood–brain barrier (BBB). It highlights various nanotechnology-based delivery approaches including liposomes, nanoparticles, dendrimers, micelles, nano-emulsions, and polymeric carriers designed for improved brain targeting and controlled drug release/[11] The review also examines ligand-mediated and receptor-targeted strategies that enhance BBB penetration and therapeutic efficacy. Furthermore, the role of targeted systems in reducing toxicity, improving bioavailability, and achieving sustained drug delivery is critically analyzed. Emerging therapeutic approaches such as gene delivery, immunotherapy, and multifunctional nano-carriers are also discussed. In addition, the review addresses current challenges, safety concerns, clinical translation issues, and future opportunities in targeted therapy for Alzheimer's disease. The primary objective of this review is to provide a comprehensive understanding of modern targeted drug delivery strategies and their potential to improve the diagnosis, treatment, and overall management of Alzheimer's disease.

2. Principles of Targeted Drug Delivery Systems

In the treatment of Alzheimer's Disease, targeted drug delivery system systems aim to improve drug transport across the blood–brain barrier (BBB) and enhance drug accumulation in affected brain regions by minimizing exposure to health cells. Different targeted drug delivery approaches are shown in **Figure 1**.

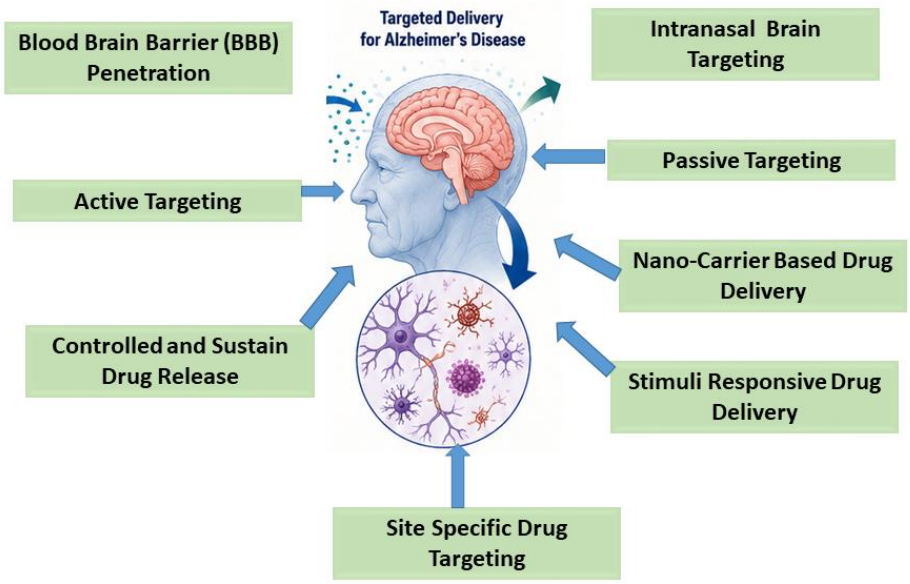

Figure 1: Different Targeted drug delivery system of Alzheimer's Disease.

2.1. Blood Brain Barrier penetration

The Blood–Brain Barrier (BBB) is a highly selective physiological barrier that separates the circulating blood from the brain extracellular fluid. It protects the central nervous system (CNS) from toxins, pathogens, and harmful substances while maintaining brain homeostasis.[12] The BBB represents the greatest obstacle to effective drug delivery in Alzheimer's disease, as it

prevents approximately 98% of small-molecule drugs and nearly all large biomolecules from entering the brain. Different strategies for BBB Crossing are: Passive diffusion of lipophilic drugs, Receptor-mediated transcytosis, Carrier-mediated transport, Adsorptive-mediated transcytosis and Intranasal brain targeting (**Figure 2**).

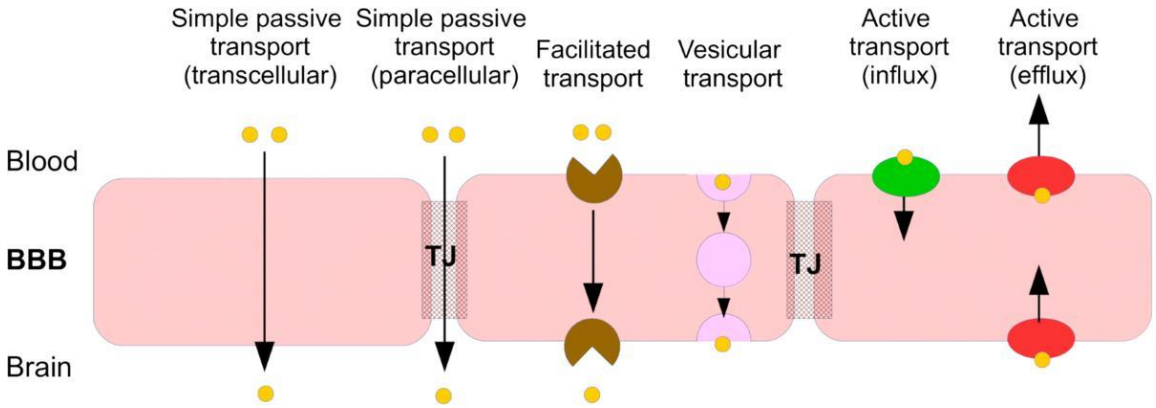

Figure 2: Transport mechanism of medicine through BBB.

2.2. Active targeting strategies

Active targeting involves surface modification of carriers with ligands, antibodies, peptides, or receptors that specifically bind to target cells or BBB transport proteins. Controlled and sustained drug release is another important principle, ensuring prolonged therapeutic action and reduced dosing frequency. These systems also enhance drug solubility, bioavailability, and stability while minimizing systemic toxicity and adverse effects. Additionally, targeted delivery systems can facilitate receptor-mediated endocytosis and transcytosis for efficient BBB penetration. Therefore, targeted drug delivery offers a promising approach for improving the efficacy and safety of Alzheimer's disease therapy.[13]

2.3. Controlled and sustain drug release

Controlled and sustained drug release involves the gradual and predictable release of therapeutic agents over an extended period, maintaining effective drug concentrations in the brain while minimizing fluctuations, toxicity, and dosing frequency. In AD treatment, sustained drug release is particularly important because the disease requires long-term therapy, and many therapeutic agents exhibit short half-lives, poor brain bioavailability, and rapid systemic clearance.[14]

2.4. Intranasal brain targeting

Intranasal brain targeting is a non-invasive strategy that delivers drugs directly to the brain while bypassing the blood–brain barrier (BBB). The nasal cavity connects to the brain through the olfactory and trigeminal pathways, enabling rapid drug transport to the CNS. This route avoids first-pass metabolism, enhances bioavailability, and reduces systemic side effects. Nanocarriers such as polymeric nanoparticles, liposomes, nanoemulsions, solid lipid nanoparticles (SLNs), and nanostructured lipid carriers (NLCs) improve drug stability and brain delivery. Mucoadhesive polymers like chitosan increase nasal residence time and drug absorption. Several anti-Alzheimer agents, including donepezil, rivastigmine, memantine, curcumin, and quercetin, have shown improved brain uptake through intranasal administration. The approach is also being explored for delivering anti-amyloid agents, neurotrophic factors, siRNA, and gene therapies. Although challenges such as mucociliary clearance and enzymatic degradation remain, intranasal delivery offers rapid onset of action, better patient compliance, and enhanced therapeutic efficacy, making it a promising strategy for Alzheimer's disease treatment.[15]

2.5. Passive targeting strategies

Passive targeting relies on the physicochemical properties of carriers, including particle size, surface charge, and hydrophobicity, to improve brain distribution. Passive targeting enhances the accumulation of therapeutic agents in the brain without the use of specific ligands or receptors. Nanoparticles, liposomes, solid lipid nanoparticles, and polymeric micelles can improve drug stability and prolong circulation time, thereby increasing the probability of crossing the blood–brain barrier (BBB).

Passive targeting also utilizes the altered permeability and retention characteristics associated with pathological brain regions. Surface modification with hydrophilic polymers such as polyethylene glycol (PEG) helps nanoparticles evade rapid clearance by the reticuloendothelial system and enhances brain bioavailability. Small-sized nano-carriers are capable of diffusing through endothelial barriers and facilitating drug accumulation in neuronal tissues. Controlled and sustained drug release from these carriers further improves therapeutic efficacy while reducing systemic toxicity and dosing frequency. Although passive targeting improves brain delivery to some extent, it lacks high specificity toward diseased neurons compared to active targeting approaches.[16]

2.6. Nano-carriers based drug delivery

Nano-carrier-based drug delivery systems offer an effective approach for treating Alzheimer's disease by overcoming the limitations of conventional therapies. These nanosized carriers enhance drug solubility, stability, and bioavailability while facilitating transport across the blood–brain barrier (BBB).[17] Common nano-carriers include polymeric nanoparticles, liposomes, solid lipid nanoparticles (SLNs), nanostructured lipid carriers (NLCs), dendrimers, and nano-emulsions. They protect drugs from degradation and enable controlled and sustained release. Surface modification with targeting ligands further improves brain-specific delivery. Nano-carriers have been used to deliver cholinesterase inhibitors, antioxidants, anti-amyloid agents, and gene therapeutics. They also enhance the therapeutic potential of natural compounds such as curcumin and resveratrol. By increasing drug accumulation in the brain and reducing systemic side effects, nanocarriers improve treatment efficacy. Thus, nano-carrier-based drug delivery represents a promising strategy for the management of Alzheimer's disease.[18]

2.7. Stimuli responsive drug delivery

Stimuli-responsive drug delivery systems, also known as smart drug delivery systems, are advanced nano-carriers that release therapeutic agents in response to specific internal or external stimuli. In Alzheimer's disease (AD), these systems are designed to respond to pathological conditions such as altered pH, elevated reactive oxygen species (ROS), enzymes, temperature changes, or amyloid-β accumulation. The drug remains encapsulated during circulation and is released only when the carrier encounters the target stimulus, improving site-specific delivery and reducing systemic side effects.[19] Common stimuli-responsive nano-carriers include polymeric nanoparticles, liposomes, hydrogels, and dendrimers. These systems enhance blood–brain barrier (BBB) penetration, improve drug stability, and provide controlled release of anti-Alzheimer therapeutics. Stimuli-responsive delivery has been investigated for cholinesterase inhibitors, antioxidants, anti-amyloid agents, and gene therapies. By increasing drug concentration at the diseased site while minimizing off-target effects, smart drug delivery systems offer significant potential for improving Alzheimer's disease treatment.[20]

2.8. Site specific drug targeting

Site-specific drug targeting delivers therapeutic agents directly to diseased regions of the brain, improving treatment efficacy and reducing systemic side effects. In Alzheimer's disease, nano-carriers such as liposomes, polymeric nanoparticles, and solid lipid nanoparticles are functionalized with targeting ligands to cross the blood–brain barrier (BBB). These carriers selectively accumulate in areas affected by amyloid-β plaques and neurofibrillary tangles. Site-specific delivery enhances drug bioavailability, minimizes off-target toxicity, and improves therapeutic outcomes. Therefore, it represents a promising strategy for effective Alzheimer's disease management.[21]

3. Targeting Strategies for Alzheimer's Disease

3.1. Receptor-mediated targeting

(a) Transferrin receptor

The transferrin receptor (TfR) is highly expressed on the endothelial cells of the BBB. Nano-carriers functionalized with transferrin receptor antibodies cross the BBB efficiently and enhances the accumulation of therapeutic agents in the brain. Various nanocarrier such as liposomes, polymeric nanoparticles, and dendrimers, have been engineered with transferrin ligands to improve brain targeting. TfR drug delivery enhanced drug bioavailability, neuronal uptake, and improved therapeutic outcomes in AD.[22]

(b) Insulin receptor

Insulin receptor are highly expressed on BBB endothelial cells and facilitate receptor-mediated transcytosis. Nano-carriers conjugated with insulin-mimicking peptides enhance the delivery of drugs to the central nervous system by improving brain penetration. Insulin receptor targeting also impaired insulin signaling which is commonly observed in AD. This strategy has the potential to reduce amyloid-beta accumulation and improve neuronal function.

(c) Low-density lipoprotein receptor-related proteins

Low-density lipoprotein receptor-related proteins (LRPs) play a vital role in lipid transport and cellular uptake processes within the brain. Nanocarriers modified with apolipoprotein E (ApoE) or related ligands can interact with LRPs and undergo receptor-mediated transport across the BBB. This mechanism enhances the brain delivery of anti-amyloid drugs, neuroprotective compounds, and genetic materials.

3.2. Peptide- and antibody-mediated targeting

Peptide- and antibody-mediated targeting strategies provide high specificity for delivering therapeutics to the brain and diseased neuronal tissues. Peptides such as TAT, RVG, and other cell-penetrating peptides can facilitate nanoparticle transport across the BBB and improve cellular uptake. Similarly, monoclonal antibodies can be designed to recognize specific receptors on BBB endothelial cells or pathological proteins associated with AD. Furthermore, dual-functional systems combining therapeutic and targeting capabilities have demonstrated improved efficacy in preclinical AD models, making peptide- and antibody-mediated targeting a promising avenue for future therapies.

3.3. Amyloid-beta targeted delivery

Amyloid-beta (Aβ) plaques are among the most prominent pathological hallmarks of Alzheimer's disease and serve as important therapeutic targets. Amyloid-beta-targeted delivery systems are designed to selectively recognize and bind Aβ aggregates within the brain.

Nanocarriers can be functionalized with anti-Aβ antibodies, peptides, or aptamers that specifically interact with amyloid plaques. Once localized near these pathological structures, the carriers release therapeutic agents that inhibit Aβ aggregation, promote plaque degradation, or enhance clearance mechanisms. This targeted approach increases local drug concentration at the site of pathology while reducing systemic toxicity. Additionally, some nano-carriers incorporate imaging agents, enabling simultaneous diagnosis and treatment, known as theranostics. Preclinical studies have shown that Aβ-targeted nanoparticles can reduce plaque burden, alleviate neurotoxicity, and improve cognitive performance. As a result, amyloid-beta-targeted delivery remains a major focus of advanced AD treatment strategies.[23]

3.4. Tau-Targeted Delivery Systems

Tau protein abnormalities, particularly hyper-phosphorylation and aggregation, contribute significantly to neurodegeneration in Alzheimer's disease. Tau-targeted delivery systems aim to prevent the formation and propagation of neurofibrillary tangles by delivering therapeutic agents directly to affected neurons. Nano-carriers can transport anti-tau antibodies, small interfering RNA (siRNA), antisense oligonucleotides, or small-molecule inhibitors that suppress tau pathology. Targeted delivery improves intracellular drug concentration and enhances therapeutic efficacy while minimizing systemic exposure. Functionalized nanoparticles can facilitate neuronal uptake and protect sensitive therapeutic molecules from enzymatic degradation. Several experimental studies have demonstrated reductions in tau aggregation, decreased neurotoxicity, and improved neuronal survival following targeted treatment. Since tau pathology correlates closely with cognitive decline in AD, tau-targeted delivery systems are increasingly viewed as promising disease-modifying therapeutic approaches.[24]

3.5. Neuro-inflammation-Targeted Nano-carriers

Neuro-inflammation plays a critical role in the initiation and progression of Alzheimer's disease. Activated microglia and astrocytes release pro-inflammatory cytokines, reactive oxygen species, and other mediators that contribute to neuronal damage. Neuro-inflammation-targeted nano-carriers are designed to selectively accumulate in inflamed brain regions and deliver anti-inflammatory therapeutics directly to activated immune cells. Surface modification with specific ligands allows these carriers to recognize inflammation-associated receptors and enhance cellular uptake. Such targeted delivery reduces cytokine production, suppresses inflammatory signaling pathways, and protects neurons from secondary damage. Controlled-release formulations further improve therapeutic effectiveness by maintaining sustained drug concentrations at the target site. By minimizing systemic immunosuppression and reducing adverse effects, neuro-inflammation-targeted nano-carriers offer a promising strategy for modulating disease progression and improving clinical outcomes in AD.[25]

3.6. Mitochondrial and Oxidative Stress Targeting

Mitochondrial dysfunction and oxidative stress are key contributors to neuronal degeneration and cognitive impairment in Alzheimer's disease. Excessive production of reactive oxygen species (ROS) damages cellular proteins, lipids, and DNA, ultimately leading to neuronal death. Mitochondrial-targeted nanocarriers are engineered to deliver antioxidants and neuroprotective agents directly to mitochondria, the primary source of oxidative stress. Ligands such as tri-phenyl phosphonium (TPP) facilitate selective mitochondrial localization and enhance intracellular drug accumulation. These systems improve mitochondrial function, restore energy production, and reduce oxidative damage within neurons. Controlled and sustained drug release further enhances therapeutic efficacy by providing prolonged protection against ROS-induced injury. In addition, combination strategies targeting both mitochondrial dysfunction and amyloid pathology have shown synergistic effects in preclinical studies. Therefore, mitochondrial and oxidative stress-targeted delivery systems represent an emerging and highly promising approach for the treatment of Alzheimer's disease.[26]

4. CONCLUSION

Targeted therapies for AD face several significant challenges. The BBB restricts efficient drug delivery to the brain. Many therapeutic agents exhibit poor penetration and limited accumulation at disease sites. Alzheimer's disease is multifactorial, involving amyloid-beta, tau pathology, neuro-inflammation, and oxidative stress, making single-target therapies less effective. Off-target drug distribution may cause unwanted side effects and reduce therapeutic efficacy. Long-term safety and biocompatibility of nanocarriers remain concerns. Variability among patients can lead to inconsistent treatment responses. Most targeted delivery systems have shown success in preclinical studies but limited clinical translation. Large-scale manufacturing, stability, and cost issues further hinder commercialization. Therefore, improving BBB penetration, target specificity, safety, and clinical effectiveness remains essential for successful AD therapy.

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