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Stem Cell Therapy: A New Horizon in Alzheimer's Disease Treatment

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ABSTRACT
Alzheimer's disease (AD) is a progressive neurodegenerative disorder marked by memory loss, amyloid-β plaque accumulation, and neurofibrillary tangles leading to the neuronal death. Current therapies offer only symptomatic relief without halting disease progression. Stem cell therapy has emerged as a potential regenerative approach capable of restoring lost neurons, reducing inflammation, and improving cognitive function. Different stem cell types—embryonic, induced pluripotent, neural, and mesenchymal—have shown therapeutic promise through neuronal differentiation and secretion of neurotrophic factors. Preclinical and early clinical studies, especially with mesenchymal and umbilical cord–derived stem cells, demonstrate safety and modest benefits. However, challenges such as ethical concerns, delivery methods, and long-term efficacy remain. Advances in genetic engineering, intranasal delivery, and 3D bioprinting continue to refine this approach, making stem cell therapy a hopeful future strategy for treating Alzheimer's disease.

Keywords: Amyloid-β plaque, Neurofibrillary tangles ,Blood-brain barrier ,Nanoparticles ,Stem cell therapy, Tau Protein .

I. INTRODUCTION

Alzheimer's disease is a progressive disorder of the brain that causes a continuous decline in cognitive abilities, which includes memory loss, rationality, linguistic system as well as the ability to perform daily activities (1). Dementia in older adults is mostly caused due to this. AD is a chronic neurodegenerative condition which is marked by buildup of β-amyloid plaques and neurofibrillary tangles comprising hyper-phosphorylated tau protein in the brain which leads to forfeiture of neurons as well as disrupt the signaling of synapse primarily in cerebral region of brain. AD can be determined by the beginning of the pre-clinical stage; mild cognitive impairment (MCI) which further progresses to chronic AD characterized by weight loss, loss of verbal abilities, loss of physical abilities (2,3). As per the reports of World Alzheimer's 2022, there are 55 million people affected due to AD across the globe in 2019 and the numbers are estimated to rise by 139 million by 2050. Mostly, 75% of the cases are not diagnosed in underdeveloped countries due to unawareness. The foremost reason for dementia in almost 76% of the population is AD. It is mostly prevalent amongst the people of age >65 years (4). The side effects of AD extend beyond the individual to affect family, friends, and caregivers. Henceforth early diagnosis and promising treatments are utmost important before the further progression of disease.

AD starts with progressive memory loss, cognitive decline, language difficulties, disorientation, impaired judgment, behavioral changes, and loss of ability to perform daily activities and possibly premature death. Surprisingly the molecular mechanisms which cause the AD are not yet known. The 2 key features causing the disease however are the accumulation of amyloid plaques and neurofibrillary tangles which causes synaptic dysfunction, neuronal degeneration, and ultimately symptoms (5).

There is a breakdown of amyloid precursor protein (APP) by the β and γ-secretase which causes the formation of two forms of amyloid-β (Aβ) namely Aβ-42 (42 amino acids long) and Aβ-40 (40 amino acids long). Aβ42 is the most responsible for the plaques which are accumulated in the brain of AD patients.

Moreover, due to amyloid-β there is activation of specific kinases which causes tau hyper phosphorylation. Tau proteins play an important role as they help to stabilize the neuronal skeleton. The hyper phosphorylation of tau proteins leads to dissociation from microtubules and forms insoluble filaments called neurofibrillary tangles (NFTs) (6). The diagnostic techniques used in

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detecting AD are traditional computed tomography (CT) and magnetic resonance imaging (MRI) which detect any changes in ventricular size, cerebral hemispheric asymmetries, and cerebral blood flow. The exact morphological changes can be estimated by brain biopsy (7). However, there is no availability of tests which can determine AD in asymptomatic patients.

Progress is being made in the treatment of AD: the US FDA has approved some cognitiveenhancing drugs and some agents for reduction of agitation in AD, and few disease-modifying therapies (DMTs). The possible treatments and drugs are mentioned in table 1 (8,9,10).

Table 1: Possible Treatments and their limitations

Treatment Category	**Drugs used**	**Mode of action**	**Limitations**
Cholinesterase Inhibitors	Donepezil, Rivastigmine, Galantamine	Increase acetylcholine levels to improve memory and cognition	Only symptomatic relief; No effect on eliminating progression of disease; side effects: bradycardia
NMDA Receptor Antagonist	Memantine	Regulates glutamate activity to protect neurons from excitotoxicity	Limited benefit in moderate-to-severe stages; does not cure disease completely
Combination Therapy	Donepezil + Memantine	Targets both cholinergic and glutamatergic systems	Still only moderate improvements; side effects notable
Anti-Amyloid Antibodies (Disease-Modifying)	Aducanumab, Lecanemab, Donanemab	Target amyloid-β plaques to slow progression	Expensive ; infusionbased; risk of brain swelling/bleeding (ARIA); modest effect sizes; not universally effective; no patient compliance
Anti-Tau Therapies (Investigational)	Tau aggregation inhibitors, anti-tau antibodies (e.g., semorinemab, gosuranemab)	Prevent tau tangles that drive neurodegeneration	Mostly in trials; no conclusive evidence of slowing progression yet
Anti-Inflammatory & Microglial Modulation	NSAIDs, microglial modulators (e.g., AL002)	Reduce neuroinflammation	Mixed or inconclusive results; final stage trials unsuccessful
Insulin/Metabolic Approaches	Intranasal insulin, GLP-1 analogues (liraglutide, semaglutide)	Improve brain metabolism & insulin signaling	Still in experimental stage ; long-term efficacy not known
Lifestyle & Non-Pharmacological	Cognitive training, physical exercise, diet,	Improve brain resilience, delay decline	Cannot cure the disease; require long-term adherence

	sleep optimization		
Symptom Management (Behavioral/Psychiatric)	Antidepressants, antipsychotics, anxiolytics	Manage agitation, depression, psychosis	Do not affect underlying disease; side effects significant in elderly
Experimental Immunotherapies & Vaccines	Aβ vaccines (e.g., CAD106), DNA vaccines	Induce immune response against amyloid or tau	Mostly in clinical trials; safety and effectiveness not yet proven

Current treatments are focusing more on managing the symptoms and not actually treating the disease. Meanwhile, Stem cell therapy holds promise by regenerating lost neurons, modulating inflammation, and potentially reversing cognitive decline. It offers a regenerative, disease modifying approach, addressing the limitations of existing therapies and meeting the urgent need for more effective treatments in Alzheimer's disease.

II. POTENTIAL OF STEM CELL THERAPY IN TREATMENT OF ALZHEIMER

Stem cells are able to differentiate into different cell types. Two of the main characteristics of a stem cell are its ability of self-renewal and its potential to distinguish into a particularized adult cell. (11) This is found in the embryonic, fetal, and adult phases of life and goes into cellular differentiation to develop specialized cells that are the important constituents of tissue and organs. (12) Just like , "During early developmental stages, a single fertilized oocyte undergoes a series of processes to generate a multicellular organism, wherein individual cells and tissues acquire specialized identities and differentiated fates to execute defined biological functions." When embryos begin to start developing, cells that have identified their role start to multiply and expand their growth, supporting tissues and various organs to grow. (13) "The remarkable capacity of embryos to generate diverse cell types, along with the ability of certain adult tissues to undergo regeneration throughout life, is fundamentally attributed to stem cells—nature's pivotal contribution to multicellular life. These cells possess the dual capabilities of self-renewal and, to divide and generate more stem cells (12,13). Self- renewal and totipotency are the main properties of a stem cell. They're present in each and every human body from the early years of development till the last. Stem cells are a unique category of undifferentiated cells. These fundamental properties make stem cells essential not only during the embryonic development stage but also in the maintenance and repair of adult tissues (14,15). Research in stem cell biology has opened new pathways in regenerative medicine and holds therapeutic potential for treating a wide range of degenerative and chronic diseases.

For decades, scientists have been studying stem cell use and its treatment in various diseases such as bone marrow transplant, memory loss etc.

Recently, clinical trials are being performed to test the effectiveness of "stem cell therapy" to treat various neuro-degenerative disorders as they seem to replace dead/damaged cells with healthy cells (16).

Categorial classification of Stem Cells which can be effective in treatment of Alzheimer's disease is provided in Table 2

Table 2: Classification of Stem Cells

Name	Source	Potency	Use
Embryonic Stem Cell	Early stage Embryos	Pluripotent	Research and regenerative therapies
Neural stem cells	Found in specific regions like spinal cord and brain	Multipotent	Neurological disease treatment
Induced Pluripotent stem cells	Adult cell to behave as embryonic stem cell	Pluripotent	Promising for personalized medicine and disease modelling
Mesenchymal stem cells	Found in bone marrow and adipose tissues	Multipotent	Treatment of inflammatory and autoimmune disease

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2.1 Classification based on stem cell origin

1. Embryonic stem cells (ESCs)

Embryonic stem cells are extracted from Pre-gastrulation embryos. These cells possess the ability to differentiate into virtually any cell type in the human body, which includes neurons as well as glial cells that are essential for brain function.

ESCs are used to develop neurons and other glial cells that can potentially replace those neurons which are lost due to Alzheimer's. Not only this but also, they serve as valuable resources for modeling the disease in the lab scale and help in formulating new drugs.

Despite their promising benefits, embryonic stem cells (ESCs) encounter considerable moral objections because of their origin .There are also some sort of technical risks which include immune rejection. This also might cause uncontrolled cell growth eventually leading to tumor formation (17).

2. Induced Pluripotent Stem Cells (iPSCs)

Induced pluripotent stem cells also termed as adult cells, derived from somatic cells, such as those from the skin or blood, which have been genetically altered to convert into a pluripotent condition. Similar to ESCs , iPSCs also possess the ability to differentiate into any cell type.

Moreover iPSCs enable researchers to generate patient-specific neurons, which can be useful for studying genetic forms of Alzheimer's so that they can test various personalized treatments. They also commit to develop individualized cell replacement therapies with considerably lower possibilities of immune rejection.

iPSCs sidestep many ethical issues associated with ESCs. However, they can still hold risks like genetic instability and tumor formation, and their use in clinical transplantation is still in underdevelopment stages (18).

3. Neural Stem Cells (NSCs)

These are multi-potent cells possessing a tendency to differentiate into neurons, astrocytes, and oligodendrocytes (forms myelin sheaths). These cells can be isolated from brain tissue or can be formed from ESCs and iPSCs respectively.

Neural stem cells (NSCs) are in their investigation phase because of their ability to get directly implanted into the brain to restore or substitute lost or injured neurons. They may also secrete neurotrophic factors that enhance neuron survival and activity.

However, the unhealthy state of the brain due to Alzheimer, creates complications for the survival, incorporation and adequate function of transplanted NSCs. Prolonged therapeutic benefit and tolerability needs to be studied; henceforth clinical trials are on-going (19).

4. Mesenchymal Stem Cells (MSCs)

These are adult stem cells often detected in bone marrow, adipose tissue, umbilical cord, and many such other body tissues. Their primary function often tends to be their abilities to differentiate into bone, fat, and cartilage cells. However adding to this MSCs also play a substantial role as they inherit immuno-modulatory and anti-inflammatory properties as well.

Unlike other cells which tend to replace neurons, MSCs are rather utilized to regulate the immune system and reduce neuronal-inflammation, which is a central aspect of Alzheimer's pathology. Not only this but they also release certain factors which might help to protect neurons and encourage their healing.

In comparison to other stem cells it is quite less challenging to isolate MSCs as they have less chances of developing immune rejection. On the challenging note, it is often found that they do not have direct ability to get converted into functional neurons. As a result it can be concluded that MSCs require more fields of research to be used for such neurodegenerative disorders (20).

Certain additional categorical MSCs are there which have the potential to cure neurodegenerative disorders.

Menstrual Blood derived Mesenchymal Stem cells

This menstrual blood derived mesenchymal stem cells (Me-Sc) have shown to provide treatment in Alzheimer's disease through secretion of BDNF (21,22).

This stem cell improves functioning in patients having Alzheimer's by reducing pro-inflammatory cytokines (TNF-α, IL-1β) (23,24).

Wharton's jelly-derived mesenchymal stem cells

A porous connective tissue of mucus-like is called Wharton's Jelly which are found surrounding vessels of the umbilical cord. This jelly provides protection to the following growth factors: (a) insulin growth factor (IGF), (b) fibroblast growth factor (FGF), and the (c) transforming growth factor-β (TGF-β) (25). WJ-MSCs are generally found in conditions of hypoxia and high pressure(26). Though, an increase in

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pressure and hypoxia increases WJ- MSC's capacity to multiply (27).

III. THE STEM CELLS AID IN DEALING THE ALZHEIMER BY FOLLOWING THE SUBSEQUENT PATTERN

Table 3
Certain Chemicals used in differentiation of stem cell

Chemicals	Function
Retinoic acid	Promotes neuronal development and acts as signaling molecule by regulating gene expression
Wnt (Wingless related integration site)signaling	Control neural induction
Noggin, SHH (Sonic Hedgehog)	Guides cell future (which type of neuron to become)
FGF2 (Fibroblast Growth Factor)	Regulates proliferation and survival of NPCs

1. Differentiation

Stem cells begin in an undifferentiated form in the initial phase. In order to get developed into neurons, they need to differentiate into neural progenitor cells (NPCs) and later into mature neurons. Researchers use molecular indicators, specifically the ones which are usually used in development of a brain. This process includes the use of certain chemicals.
The stem cells begin to express neural markers (e.g., Nestin, βIII-tubulin, NeuN) and lose pluripotency markers like Oct4 and SOX2. (28) (NEURAL MARKER - a protein which acts like a marker for neural stem and progenitor cells which helps in developing human brain.

2. Migration – commuting towards damaged site

After administration into the brain (or spinal fluid), stem cells need to commute towards the damaged location. The damaged tissue of brain releases certain chemicals such as chemokines and cytokines which pull stem cells towards them. The chemicals responsible and their roles are mentioned in table 4 respectively.

Table 4
Chemicals and their role in migration

Molecule	Role
CXCR4	It drives neural progenitor migration towards injury and promotes axon growth
SDF-1 (stromal-derived factor-1)	It restricts the premature differentiation and binds to the receptor of the stem cell.
MCP-1, IL-6	Foster stem cell migration via inflamed tissues
VEGF(vascular endothelial growth factor)	Elevates blood vessel permeability and attracts cells.

Stem cells travel along these molecular gradients to reach the sites of amyloid plaques, tangles, or neuronal death. Hence stem cells may attempt to repair the damage caused in the brain because of plaques (29).

3.Differentiation (Phase 2) – Becoming Specific Neuron types

Once the stem cells reach the injury site, they tend to be subjected towards specialization to convert into the right type of neuron which includes cholinergic neurons (important for Alzheimer), dopaminergic neurons (for Parkinson's), or interneurons. The factors essential for these along with their role are mentioned in table 5.

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Table 5
Essential transcription regulators

Chemical	Role
Ngn 2(Neurogenin 2)	Initiates Neurogenesis
Mash1 (mammalian achaete scute homolog 1)	Promotes a stem cell to convert into a neuron.
Lhx -8 , Isl1	Promotes Cholinergic identity
Neuro D1	Stabilises the neuronal phenotype.

After this process the stem cells now initiate to show proteins such as acetyltransferase which plays an important role for synthesis of neurons which were lost in Alzheimer. Later, these help in synthesizing, storage and release of Acetylcholine (30).

4.Incorporation – Blending into the brain

Now this step can be considered to be most crucial and complicated as for the proper functioning of neurons it needs to be integrated into the existing circuit of the brain. The functions of the cell happen to be the following.
(a)Develop axons toward target neurons
(b)Extend dendrites to receive signals
(c)Build synapses
(d)Release neurotransmitters
The tools required for these processes are mentioned in table 6.

Table 6
Various tools for incorporation of stem cell & their functions

System	Function
Growth cones	Sense chemical signals and promote axon growth
Synaptic vesicles	Store and release neurotransmitters
Ion channels (Na^+ , K^+)	Generate action potentials

Newly developed neurons set themselves up and start signaling each other which eventually restores memory and cognition power (31).

5. Assisting Roles – Anti-inflammatory Action:

Even the stem cells which were unable to transform to neurons also play the following roles for improving sustainability:
-Secretes Trophic Factors:
-BDNF (Brain-Derived Neurotrophic Factor): Helps in survival of neurons.
-NGF (Nerve Growth Factor): Helps in development, maintenance, and survival of neuronsin the brain.
-GDNF (Glial cell line-Derived Neurotrophic Factor): Enhances synaptic plasticity.

Reduce Inflammation:
MSCs release IL-10, TGF-β, and PGE2, which suppress hyperactivation of microglia (32).
Final Outcome: Returning to normal environment of brain by stem cells includes;
(1)Becoming neurons
(2)Migrating to damaged areas
(3)Integrating into circuits

IV. SECRETING HEALING FACTORS.

Ongoing Clinical Trials on this therapy along with their current status and its progress

Table 7 Current clinical trials being conducted on various Stem cell .

Stem cell category	Dose and ROA	Phase / identification	Sample & Condition	Status	Conclusions drawn
hUC-MSCs	(I) 3×10⁶ cells/brain [3] IC;	Phase I / NCT01297218 & Phase I/IIa / NCT01696591	9 pts / probable AD; >50 years; K-MMSE 10–	Completed	Safe; no doselimiting toxicity. Cognitive

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	(II) 6×10⁶ cells/brain [6] IC		24; PIB-PET+		decline observed (KMMSE ↓; ADAS-Cog ↑)
hUC-MSCs	Stage 1: (I) 1×10⁷ cells/brain [3] ICV ×3 infusions ; (II) 3×10⁷ cells/brain [6] ICV ×3. Stage 2: (I) 3×10⁷ cells/brain [24] **ICV ×3; (II) placebo [12] ICV ×3**	Phase I/IIa / NCT02054208	46 pts / probable AD; 50–85 years; K-MMSE 18–26; amyloid PET+	Completed	Transient fever, CSF leukocytosis in 9 pts. Biomarkers improved (↓ttau, p-tau, Aβ42). Cognitive change minimal.
hUC-MSCs	Null	Phase I/IIa / NCT03172117	Follow-up from NCT02054208	Ongoing	Null
hUC-MSCs	Null	Null / NCT04954534	Placebo pts from NCT02054208	Not yet recruiting	Null
hUC-MSCs	0.5×10⁶ cells/kg IV ×8	Phase I/II / NCT01547689	30 pts / probable AD; 50–85 years; MMSE 3–20	Not known	Null
hUC-MSCs	(I) 0.5×10⁶ cells/kg IV ×8; (II) placebo IV ×8	Phase I/II / NCT02672306	16 pts / probable AD or mixed dementia; 50–85 years; MMSE 10–26; on standard therapy	Not known	Null
hUC-MSCs	1×10⁷ cells IV ×4	Phase I / NCT04040348	6 pts / AD or probable AD; 50–85 years; MMSE 20–26; amyloid PET positive	Ongoing	Null
Human amniotic & UC-MSCs	IV, intranasal, or nebulizer (per condition)	Phase I / NCT03899298	~5000 pts / multiple conditions; >18 years	Not yet recruiting	Null
Human amniotic & UC-MSCs	Route on the basis of patient profile	Phase I/II / NCT04684602	~5000 pts / multiple conditions; >18 years	Ongoing	Null

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Human placenta MSCs (CB-AC-02)	Stage 1: (I) 2×10^8 cells IV ×1; (II) 2×10^8 cells IV ×2. Stage 2: (I) Arm1 (KMMSE 20–26): 2×10^8 cells IV ×2; (II) Arm2 (K-MMSE 10–19): 2×10^8 cells IV ×2; (III) placebo [12] ICV ×2	Phase I/II / NCT02899091	24 pts / probable AD; >50 years ; MMSE 10–26; amyloid PET positive	Ongoing	Null

huc MSCs (Human umbilical cord derived mesenchymal stem cells) ; IC (Intracerebral) (stereotactic injection); ICV (Intracerebro-ventricular) (via Ommaya reservoir); IV (intravenous infusion); MMSE / K-MMSE (Mini-Mental State examination) (cognitive test) ;ADAS-Cog (Alzheimer's Disease Assessment Scale–Cognitive Subscale);PET (positron emission tomography) ; pts(patients).

V. CHALLENGES AND DRAWBACKS FOR INTRODUCING THIS THERAPY INTO MARKET

1. Complexity of Alzheimer's disease Pathology

Stem cell therapy generally aims to replace or repair lost neurons, but this aims to treat only a certain part of Alzheimer. It has no promising effect on treating inflammation or removal of toxic protein buildup in the brain. A simultaneous therapy would be needed to comprehensively work on inflammation and protein aggregation which is not fulfilled by the stem cell therapy alone (33).

2.Stem Cell Type, Source, and Standardization:

The cells isolated are different in all the individuals and it can be different in different donors.
As the time passes, certain stem cells may lose the capacity to regenerate.

Producing large quantities of identical, high-quality cells under Good Manufacturing Practice (GMP) is technically complex and expensive.(34)

3.Effective Delivery to the Brain:

Administration of stem cells to the brain is quite a complex process. The viable routes of administration along with their benefits and limitations are mentioned in table 8.

Table 8
Routes of administration of stem cell along with their drawback

Route of Administration	Pros	Cons
Intravenous (IV)	Minimally invasive; easiest for clinical application.	High "first-pass" effect.
Intranasal	Non-invasive; directly bypasses the Blood-Brain Barrier (BBB) via olfactory pathways.	Still under development;
Intrathecal	Delivered directly into Cerebrospinal Fluid (CSF); bypasses the BBB.	Moderate invasiveness (spinal tap); cells may distribute in CSF but not

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		integrate into brain tissue.
Intracerebral (Stereotactic injection)	Extremely precise targeting; delivers cells directly to the site of neurodegeneration (e.g., Hippocampus).	Highly invasive; requires neurosurgery.

Even the administration of stem cells is successful, many of them might die due to restricted environments (inflammation, stress) or fail to commute and integrate into the brain (35).

4. Uncertain and Limited Clinical Efficacy

There is no sufficient evidence which proves long term No strong evidence yet of long-term functional recovery
Alzheimer's progression is slow and variable, requiring years-long follow-up to detect significant effects (36).

5. Safety Risks: Tumorigenesis & Immune Response

Tumor risk: There is risk of developing a tumor if stem cells are not thoroughly differentiated before transplantation. (Particularly iPSCs)

Immune rejection: There are chances of graft rejection by the immune system if Allogeneic stem cells (from donors) are recognized as foreign.
Inflammatory response: May intensify existing neuroinflammation in certain cases (37).

6. Regulatory and Ethical Hurdles

Stem cell therapies must comply with Good Manufacturing Practices (GMP).
Regulatory bodies (like the FDA) requires:
Long-term toxicity studies
Defined mechanism of action
Reproducibility and scalability
Transparent donor screening
The regulatory pathway is complex, specifically for personalized therapies wherein the reactions differ from patient to patient.

7. High Cost and Limited Accessibility

Stem cell therapies involve, cell collection (e.g., liposuction for adipose MSCs) , advanced delivery systems (e.g., surgical implantation) and post-treatment follow-ups.
Estimated costs:
$25,000–$100,000+ per patient, depending on cell source, delivery, and number of treatments
(38).

VI. RECENT ADVANCEMENTS IN STEM CELL THERAPY

1) Genetic Engineering

CRISPR-Cas9 Genetic Engineering iPSC Derived Microglia for In-Brain Delivery to Treat AD (in pre-clinical stage)

AD is characterized by the pathological accumulation of Amyloid Beta (Aβ)protein in the brain, which forms plaques that destroy the neuronal activity.Using CRISPR Editing, iPSC derived Microglia was formed to detect Amyloid Plaques & secrete Neprilysin (An Enzyme used to degrade Beta Amyloid under promoter which only activates around. In Alzheimer's mouse models, these engineered microglia reduced the buildup of betaamyloid and protected against damage to neurons and synapses, curbed inflammation, and even lowered a biomarker of neuronal injury in the blood.
Though in Pre-Clinical stage, it shows promising response in reducing plaque formation and reversal of neurodegeneration in mouse-models(39).

2) 3D Bioprinting

3D-engineered bioprinted in – vitro human neural stem cell self-assembling culture model ofAD.Twenty-two-month-old APP/PS1 mice received a first IN administration of MSC-CS and were tested 7 days later in the NORT, to verify if a single IN administration could replicate the memory recovery obtained with one IV injection.(40,41,42,43). It bypasses the BBB.

3) Intra Nasal Delivery of Msc

Intranasal delivery of mesenchymal stem cells (MSCs) is a non-invasive approach that enables direct transport of therapeutic cells to the brain via the olfactory and trigeminal neural pathways, bypassing the blood–brain barrier. This method enhances targeted delivery and has shown potential in improving neuroregeneration and reducing neuroinflammation in neurodegenerative disorders such as Alzheimer's disease. It is considered a promising strategy for CNS drug delivery due to its safety, ease of administration, and improved therapeutic efficacy.(44,45)

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VII. CONCLUSION

The stem cell therapy for Alzheimer's disease is still in the experimental stage, however ongoing progress in stem cell research, innovative disease models, and smarter clinical trial strategies are accelerating the path towards the possible practical therapies.

Preclinical studies have showcased promising results, and early-phase human trials suggest that stem cells may support neuro-protective and regenerative benefits.

However, due to the complexity of Alzheimer's pathology and the challenges associated with effective delivery of cell, safety, and long-term efficacy, clinical adoption on a large scale is currently unviable.

If scientific developments maintain their current pace, stem cell therapies are likely to become feasible treatment approaches over the next 10 to 15 years, particularly as Supportive treatments which aim to reduce the progression of disease, repairing neural damage, or improving cognitive function. However, the integration of these therapies into clinical practice will require evidence from myriad, well-controlled trials demonstrating their safety, efficacy, and consistency in treating the disease.

As an overall assessment it can be stated that, stem cell therapy has the potential to revolutionizethe treatment for Alzheimer's disease, but further scientific and clinical approval is essential before it can be introduced into the market (46,47,48,49).

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