Journal of Research High School

Vol. 2026 (1) 12 – 19
ISSN 2688-3651 [online]

Exploring Stem Cell Therapy as a Regenerative Approach to Alzheimer's Disease

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Received March 16, 2025; Revised August 24, 2025; Accepted September 29, 2025

Abstract

Alzheimer's Disease (AD) is a devastating neurodegenerative disease that has multiple pathologies, including the accumulation of amyloid plaques and hyperphosphorylated tau proteins. AD lacks curative therapies, and current treatments only offer temporary symptom relief. The main objective of this review is to investigate the therapeutic potential of different types of stem cells in addressing AD pathologies. Stem cell therapy emerged as a new therapeutic approach that directly tackles some of the key pathologies of AD. Stem cells of various origins can be used to treat AD, including embryonic stem cells (ESCs), neural stem cells (NSCs), induced pluripotent stem cells (iPSCs), and mesenchymal stem cells (MSCs). Numerous preclinical studies and clinical trials have tested the efficacy of these stem cells. Most studies indicated positive developments like restoration of cognitive function, reduction of neuroinflammation, and increased synaptic plasticity with mild adverse effects, making stem cell therapy a promising potential treatment option. This review also discusses the mechanisms of stem cell action, methodological approach to reviewing the literature, and challenges such as ethical and safety concerns. Some types of stem cells such as ESCs, however, raise ethical considerations, which is a factor to be considered for the actual use of the treatment. Ultimately, stem cell therapy offers hope for regenerative medicine in AD, but further investigation is necessary to ensure its safety, efficacy, and practical application.

Keywords: Alzheimer's Disease, Stem cell, Regenerative, Neuroinflammation, Cognitive function

1. Introduction

1.1 Overview of Alzheimer's Disease

Alzheimer's Disease (AD) is a progressive neurodegenerative disorder characterized by changes in the brain, mainly caused by the accumulation of amyloid beta and hyperphosphorylated tau proteins (Vasic et al., 2019). This build-up leads to neuronal loss and, hence, brain atrophy, causing memory loss, cognitive dysfunction, and, in some cases, motor dysfunction (Vasic et al., 2019). AD is the most common form of dementia, with an estimated 50 million people affected worldwide, a number projected to triple by 2050 (Liu et al., 2020).

Although anti-amyloid beta monoclonal antibodies, the first disease-modifying therapies for AD such as Lecanemab (a recently approved monoclonal amyloid beta-directed antibody therapy), achieved slowing of clinical decline, certain side effects and accessibility limits their use. Therefore, symptomatic management composes the majority of treatment. The available primary treatments — memantine and cholinesterase inhibitors, such as donepezil, galantamine, and rivastigmine — slow the progression of the disease temporarily; however, drugs that aim to tackle some of the key pathologies of the disease, such as reducing amyloid beta production or inhibiting tau aggregation, have significant limitations (Liu et al., 2020). This could be because of AD's etiology, which is too complex, often

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involving genetic background, environmental factors, and other parameters such as chronic stress, oxidative stress, and inflammation (Vasic et al., 2019).

It is known that pathology in the brain starts approximately 10 years before the clinical symptoms arise, causing the drugs to intervene at a later stage of the disease. This suggests the need for an alternative and innovative treatment method (Liu et al., 2020). The treatment option should preferably stop and even reverse neuronal loss in the brain, which would ultimately restore cognitive function. Recently, many new studies, animal models, and clinical trials have shown results for the possible use of stem cell therapy in treating patients with AD.

To bridge a critical gap in the literature, this review integrates the most recent findings on the regenerative potential of various types of stem cells. While previous reviews have addressed stem cell mechanisms or outcomes independently, this paper aims to combine insights, preclinical and clinical data, and a discussion of ongoing challenges to provide a comprehensive update accessible to a student readership.

1.2 Stem Cell Therapy

Stem cells are undifferentiated cells with the potential to develop into different specialized cell types, including neurons (Liu et al., 2020). Theoretically, stem cells should be able to replace the neurons lost due to AD and become a disease-modifying treatment option. One of the reasons why stem cell therapy is favorable is the fact that, unlike other treatment options, which often involve drugs that cannot pass the blood-brain barrier (BBB), stem cells manage to bypass the BBB (Chia, 2020).

There are a few types of stem cells that can be used for therapeutic purposes: embryonic stem cells (ESCs), neural stem cells (NSCs), induced pluripotent stem cells (iPSCs), and mesenchymal stem cells (MSCs) (Vasic et al., 2019). Each stem cell origin has its own advantages and disadvantages. Embryonic stem cells are pluripotent stem cells derived from early-stage embryos; their pluripotency allows them to differentiate into different cells, but, in some cases, the differentiation can occur in any direction, leading to undesirable results (Liu et al., 2020). ESCs are not, in many cases, optimal because of the challenges faced during purification and preservation, as well as because of ethical considerations (Karvelas et al., 2022). Different countries have varying regulations on the use of ESCs for research purposes, making them unavailable as a therapeutic approach in some regions. Researchers also consider iPSCs, generated by the reprogramming of adult somatic cells into stem cells (Liu et al., 2020). Similar to ESCs, iPSCs can differentiate into neurons, while avoiding ethical controversies. However, issues related to tumor formation and immune rejection are major disadvantages for iPSCs (Karvelas et al., 2022). Another potential therapy for AD could be the use of neural stem cells (NSCs), which exist in specific areas of the adult human brain, particularly the subventricular zone and the granular layer of the dentate gyrus (Liu et al., 2020). NSCs could be derived from the adult's own brain, avoiding immunological reactions. Mesenchymal stem cells (MSCs) are found in adult bone marrow and adipose tissue, with the capability to differentiate into various mature cell types. The evidence supports that MSCs improve synaptic plasticity and cognitive function (Bruno et al., 2024). Researchers continue to study the use of different types of stem cells to treat AD.

1.3 Research Question

Given the nature of AD and the potential of stem cell therapy, this review explores the following research question: Can stem cell therapy serve as a regenerative approach to treating Alzheimer's Disease by reversing or mitigating its primary pathologies? The present review provides an overview of the various types of stem cells currently under investigation for AD treatment and the mechanisms by which they may influence disease pathology. Additionally, it integrates recent findings from preclinical and clinical findings, offering insights into the potential and challenges of stem cell-based therapies in AD.

2. Methodology

This paper reviews recent peer-reviewed literature on Alzheimer's disease and stem cell–based therapeutic approaches. Sources were gathered from PubMed and Google Scholar using keywords such as "Alzheimer's Disease,"

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"stem cell therapy," "neuroregeneration," and "clinical trials." Studies that addressed mechanisms of action, preclinical models, and clinical applications were included. Both positive and negative outcomes were considered to provide a balanced perspective.

3. Mechanisms of Stem Cell Action in Alzheimer's Disease

3.1 Neuronal Replacement and Neurotrophic Support

One of the most promising aspects of stem cell therapy is its ability to replace the neurons lost due to the progression of AD (Liu et al., 2020). Its ability to provide neuronal replacement and neurotrophic support differentiates stem cell therapy from other treatment methods. Several types of stem cells have been researched for their potential to differentiate into neurons and glial cells.

Regeneration can be separated into two broad categories: endogenous regeneration and engrafted regeneration (Vasic et al., 2019). Endogenous regeneration is, as the name implies, the body's own mechanism to generate neurons in the dentate gyrus, a process also shared by neural stem cells (NSCs). However, while millions of neurons are lost during the progression of AD, the body can only regenerate about 700 per day, highlighting the limits of this natural process (Vasic et al., 2019). Another therapeutic possibility is induced regeneration through neurogenesis-stimulating drugs. Figure 1 demonstrates neurogenesis in the dentate gyrus.

Engrafted regeneration, on the other hand, is the process by which stem cells are directly transplanted surgically into the brain (Vasic et al., 2019). This method proceeds similarly to endogenous regeneration. The transplantation of stem cells allows for neuronal regeneration as well as neurotrophic support to the endogenous neurons: MSCs, for example, have been found to release neurotrophins such as Brain-Derived Neurotrophic Factors, causing remyelination — generation of myelin sheaths in the central nervous system (Uccelli et al., 2011).

There is evidence that stem cell therapy causes neurogenesis and neurotrophic support; however, the complex molecular mechanisms of the differentiation of stem cells into neurons have not been fully understood yet. Further research will be able to fully grasp the underlying mechanisms of a potentially promising therapeutic approach.

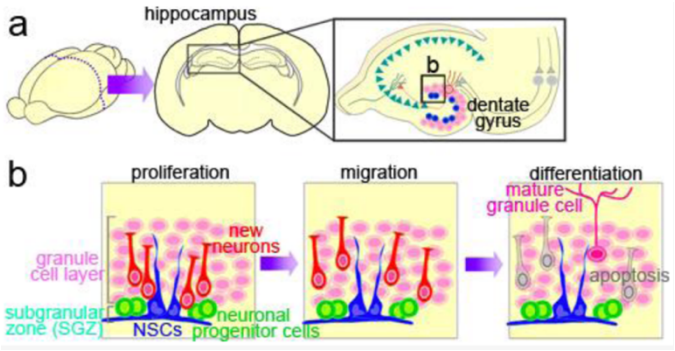

Figure 1. NSCs in the hippocampus. (a) Location, structure, and neuronal circuitry of the dentate gyrus in the hippocampus of the adult rodent brain (b) Neurogenesis in the dentate gyrus. NSCs (blue) and neuronal progenitor cells (light green) reside in the subgranular zone, where they proliferate, and generate immature new neurons (red) (left). The new neurons migrate into the granule cell layer (middle), where some of them differentiate into mature granule cells (pink), and the rest are eliminated by apoptotic cell death (gray) (right) (Kaneko et al., 2011).

3.2 Modulation of Neuroinflammation and Amyloid Beta Clearance

It has been observed that stem cell therapy has also been helpful in mitigating neuroinflammation and the accumulation of amyloid beta plaques. AD is characterized by neuroinflammation in the central nervous system leading to cognitive dysfunction (Si et al., 2023). Engrafted stem cells directly decrease neuroinflammation by diverting microglia (primary immune cells of the nervous system) from the inflammatory M1 phenotype to the M2 phenotype after stroke, which mitigates neuroinflammation in diseases like AD (Anthony et al., 2022).

A reason that neuroinflammation is reduced can be the effect of stem cell transplantation on amyloid-beta plaque accumulation. A study on mice showed that the injections of NSC-derived extracellular vesicles significantly decreased the amyloid-beta accumulation caused by AD (Apodaca et al., 2021). Another study showed that stem cells secrete inflammatory cytokines such as TNFα, IL-1, IL-2, IL-6, and IL-10, regulating neuroinflammation, cell growth, and apoptosis (Qin et al., 2021). Another study on the effect of human umbilical cord blood-derived mesenchymal

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ISSN 2688-3651 [online]

stem cells (hUCB-MSC) on amyloid-β accumulation demonstrated that hUCB-MSCs secrete a protein named sICAM-1, which causes an important amyloid beta (Aβ)-degrading enzyme to be induced, as shown in Figure 2 (Kim et al., 2011).

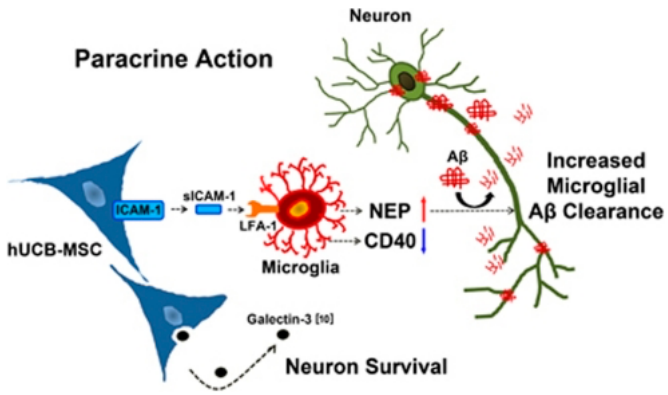

Figure 2. Therapeutic potential of hUCB-MSCs in AD. When hUCB-MSCs meet microglia, hUCB-MSCs secrete high levels of sICAM-1, which induces neprilysin (NEP) expression, an Aβ-degrading enzyme in microglia (Kim et al., 2011).

Stem cell therapy not only provides neurogenesis and neurotrophic support, but it also removes amyloid beta plaques from where they migrate and reduces neuroinflammation. These are vital aspects that directly tackle the pathologies of AD, showing that stem cell therapy has immense potential to be used as a disease-modifying therapeutic approach.

3.3 Synaptic Plasticity and Cognitive Enhancement

One of the contributing factors to cognitive dysfunction in AD is the disruption of synaptic plasticity, often caused by amyloid-beta accumulation or phosphorylated tau aggregation (Chang et al., 2023). Stem cell therapy has shown restoration of hippocampal neuroplasticity, the ability of synapses to change in response to stimuli, constituting better cognitive ability (Duan et al., 2023). By synaptogenesis, the formation of new synapses, the brain is able to restore some of its functions. The signaling pathways of synaptogenesis include stimulating the production of BDNF and NGF (nerve growth factor) for remyelination (Qin et al., 2021). It has also been shown that exogenous NSCs increased long-term potentiation and the strength of synapses in response to stimulation in the CA1 region of mice models (Chang et al., 2023). Numerous studies have demonstrated that stem cell therapy decreases long-term depression and reduces the efficacy of synapses for a period while increasing synaptic connections, synaptic density, number of synapses, dendritic branches, and dendritic spine density (Chang et al., 2023). Improvement in synaptic plasticity translates to restoration of cognitive function.

4. Preclinical Studies and Clinical Trials of Stem Cell Therapy in AD

4.1 Animal Models of Alzheimer's Disease Using Stem Cells

There have been many animal models and preclinical studies on the use of stem cells as a means of therapy for Alzheimer's Disease. Almost all of the studies included rodents as the subjects (Duan et al., 2023). A significant proportion of studies favored MSCs or NSCs over other stem cell types—25 of the 42 studies used MSCs, 8 used NSCs, with the remainder testing other types in very small numbers (Duan et al., 2023). This uneven distribution raises questions about whether conclusions can be generalized across stem cell types. The advantage of animal models is the ability to test stem cell therapy on a tissue level; however, the translatability of results on rodents to AD patients remains uncertain, as rodent models often do not fully replicate the complex pathology of human disease.

One of the most prevalent outcomes found from these studies was the effect of stem cell therapy in the reduction of Aβ plaque. Taoufiq Harach and his colleagues' study on the effect of MSCs on transgenic APPPS1 mice, for example, found that human adult ischemia-tolerant mesenchymal stem cells (hMSCs) both reduced cerebral soluble Aβ levels and increased some enzymes like insulin-degrading enzyme (IDE), which is known for its function to degrade Aβ (Harach et al., 2017). In most animal models, there was also a noticeable change in the behavioral symptoms or cognitive dysfunction (Duan et al., 2023).

There were also a couple of in vitro studies on the use of stem cells. MSCs, iPSCs, and NSCs were the most common stem cell types examined in the in vitro studies, and these studies generally mitigated the symptoms of AD, like amyloid-beta clearance (Duan et al., 2023).

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4.2 Clinical Trials Using Stem Cells

The number of clinical trials in humans is lower than in animal models using stem cell therapy for AD. The reason is self-explanatory: the risk of going through a new therapeutic approach and the individualization needed for the use of stem cells in humans (Duan et al., 2023). Most clinical trials use iPSCs since the cells are generated from the somatic cells of specific patients (Duan et al., 2023). A phase I trial investigating the 12-week effect of the implantation of hUCB-MSC found that it is safe with mild adverse effects such as nausea, headache, and fever (Kim et al., 2021). The patients were evaluated with some cognitive tests such as Alzheimer's Disease Assessment Scale–Cognitive Subscale and Mini-Mental State Examination during the study, and the mean change from the baseline to week 12 showed a positive development in their scores at most tests (Kim et al., 2021). Yet, results across clinical trials remain mixed. Some patients demonstrate measurable improvement, while others show no significant benefit. A key issue is variability in stem cell quality, delivery methods, and patient selection criteria. The long-term nature of AD renders clinical trials difficult. In some studies, patients are observed for up to 100 weeks, which is time and resource-consuming both for the patients and the researchers (Kim et al., 2021). Before moving on with more clinical trials, the safety of the approach should be validated, and the quality of stem cells should be further tested (Chang et al., 2023). The lack of precedent for clinical trials also causes some limitations like estimation of doses based on animal models, which is not always optimal (Kim et al., 2021).

4.3 Emerging Strategies for Enhancing Stem Cell Therapy

To improve the efficacy of stem cell therapy, one of the strategies being considered is genetically modifying stem cells in order to enhance their survival rate, especially in hostile environments (Phillips & Tang, 2008). By adding the nerve growth factor gene to NSCs, stem cells are able to contact the existing neurons, resulting in neuronal regeneration (Patwardhan & Belemkar, 2021). Ex vivo gene editing can also be used to prevent potential immune rejection (Pacheco-Herrero et al., 2021).

Gene therapy is another approach that could be used in the future to target AD. Patients with mutations in the apolipoprotein E (ApoE) gene, for instance, are predisposed to AD; hence, targeting a gene like ApoE, which is currently being tested in clinical trials, could be a step in treating AD (Patwardhan & Belemkar, 2021).

There are also strategies that aim to improve the engraftment of stem cells rather than changing the therapy itself. A study came up with two distinct approaches that improve cell engraftment (Figure 3): preconditioning the stem cells to create resistant stem cells and preconditioning the host tissue to create a less hostile microenvironment (Ezquer et al., 2017).

The first strategy suggests that since stem cells are going to experience hypoxia after being transplanted, hypoxia will be simulated ex vivo in order to train the cells (Ezquer et al., 2017). An animal model of idiopathic pulmonary fibrosis has shown that this strategy increased the number of cells four times more in comparison to untreated stem cells (Ezquer et al., 2017). The other strategy uses regional hepatic irradiation with X-rays before the engraftment of stem cells, which has been shown to decrease inflammation in animal models (Ezquer et al., 2017). With new approaches and strategies being found, stem cell therapy will become a more viable and safer approach.

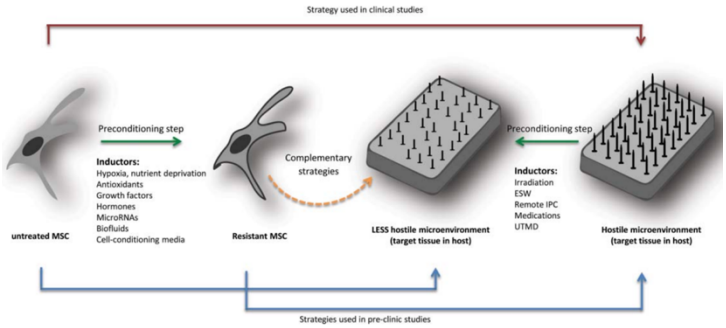

Figure 3. Summary of MSC based strategies to improve cell engraftment. The red arrow represents the strategy used in clinical trials where untreated MSCs have been transplanted into the target tissue in the host (hostile microenvironment symbolized as a bed of nails). Blue arrows represent the strategies used in pre-clinic studies where MSCs or target tissue received a preconditioning procedure to promote cell engraftment. The orange dotted arrow represents the combination of both strategies suggested in this review. Abbreviations: ESW, Extracorporeal shock waves; IPC, ischemic postconditioning; UTMD, ultrasound-target microbubble destruction (Ezquer et al., 2017).

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5. Challenges in Stem Cell Therapy for AD

5.1 Ethical Considerations and Safety

Although having a huge potential, there are also some ethical questions related to the use of stem cells, mainly Embryonic Stem Cells (ESCs). ESCs are derived from early-stage embryos, and this raises concerns about the moral status of the embryo. Many argue that as harvesting stem cells from an embryo destroys a potential human life, it is ethically wrong to carry out this process. Hence, other types of stem cells such as iPSCs or MSCs seem like a better alternative. However, there are also some ethical concerns regarding the use of iPSCs as their unlimited differentiation potential could be used for human cloning (Duan et al., 2023).

Another concern about the use of stem cells is the safety concerns associated with them. After the engraftment of stem cells, there is potential for immune rejection, tumor formation, and off-target differentiation. Although most preclinical and clinical trials test the short-term safety of stem cells, the long-term safety and efficacy are not studied as much due to the recency of the therapeutic approach (Pacheco-Herrero et al., 2021). While some studies show potential adverse effects, other studies have shown only mild effects. A study involving the engraftment of ESCs to mice, for example, has shown that these cells "have a high degree of cellular heterogeneity and proliferation, uncharacterized growth and tumor-forming potential" (Fujikawa et al., 2005). In a study testing the safety of intracerebroventricular injection of hUCB-MSC into the lateral ventricle, however, it was noted that most patients did not experience serious adverse effects: the most common mild side effects were headache, nausea, vomiting, myalgia, or chills (Kim et al., 2021).

Immune rejection is a prominent issue in the safety of stem cells. In some cases, "cells from amniotic fluid, placental tissue, umbilical cord tissue, and even unknown sources of cells from different donors are used," which can cause immune rejection (Pacheco-Herrero et al., 2021). This issue can potentially be tackled by the use of immunosuppressive therapies, treatment methods that weaken the function of the body's immune system, but this could cause other risks such as susceptibility to infections (Pacheco-Herrero et al., 2021).

The number of clinical trials about the use of stem cells increases rapidly, which would make stem cell therapy more reliable. To resolve the ethical questions regarding the use of ESCs, other types of stem cells can be utilized as a therapeutic approach.

5.2 Technical Challenges and Research Needs

The development of safe and effective stem cell therapies to treat AD presents numerous technical challenges that need to be further explored. Although NSCs derived from adult brains present a great option for a therapeutic approach, their numbers are insufficient and they have a limited range of expansion (Fan et al., 2014). Some other types of stem cells impose unique challenges to overcome, like immune rejection due to the incompatibility of the ESCs with the patient (Fan et al., 2014). There could also be instances where cells do not survive or form connections with other neurons due to the inflammatory environment of an AD brain (Pacheco-Herrero et al., 2021). Lastly, a major issue faced in stem cell therapy is the potential for off-target differentiation (Fujikawa et al., 2005).

Further research is needed to create a more precise mechanism for ensuring that stem cells differentiate into the correct neurons and glial subtypes. This would remarkably increase the efficacy of the approach. Additionally, supplementary treatment methods that increase the possibility of the survival of newly generated neurons would be highly beneficial. With research on stem cell therapy rapidly increasing, these issues will be addressed, creating a more reliable, viable, effective, and safer treatment option.

6. Conclusion

Stem cell therapy shows immense promise for Alzheimer's Disease, offering regenerative benefits beyond traditional treatments. It has demonstrated potential in neuronal replacement, neuroinflammation reduction, and synaptic repair. Yet, challenges such as ethical issues, immune rejection, and inconsistent clinical outcomes remain.

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Continued research into improving safety, refining delivery methods, and expanding clinical trials is essential for translating this therapy into reliable clinical use.

References

Anthony, S., et al. (2022). Neuroinflammation, Stem Cells, and Stroke. *Stroke*, 53(5), 1460–1472. https://doi.org/10.1161/STROKEAHA.121.036948

Apodaca, L. A., et al. (2021) Human neural stem cell-derived extracellular vesicles mitigate hallmarks of Alzheimer's disease. *Alz Res Therapy* 13, 57. https://doi.org/10.1186/s13195-021-00791-x

Bruno, A., et al. (2024). Perinatal Tissue-Derived Stem Cells: An Emerging Therapeutic Strategy for Challenging Neurodegenerative Diseases. *Int. J. Mol. Sci.* 25, 976. https://doi.org/10.3390/ ijms25020976

Chang, J., et al. (2024). Neural stem cells promote neuroplasticity: a promising therapeutic strategy for the treatment of Alzheimer's disease. *Neural regeneration research*, 19(3), 619–628. https://doi.org/10.4103/1673-5374.380874

Chia, Y. C., et al. (2020). Stem Cell Therapy for Neurodegenerative Diseases: How Do Stem Cells Bypass the Blood-Brain Barrier and Home to the Brain?. *Stem cells international*, 8889061. https://doi.org/10.1155/2020/8889061

Duan, Y., Lyu, L., & Zhan, S. (2023). Stem Cell Therapy for Alzheimer's Disease: A Scoping Review for 2017-2022. *Biomedicines*, 11(1), 120. https://doi.org/10.3390/biomedicines11010120

Ezquer, F. E., et al. (2017). Two complementary strategies to improve cell engraftment in mesenchymal stem cell-based therapy: Increasing transplanted cell resistance and increasing tissue receptivity. *Cell adhesion & migration*, 11(1), 110–119. https://doi.org/10.1080/19336918.2016.1197480

Fan, X., et al. (2014). *Stem-Cell Challenges in the Treatment of Alzheimer's Disease: A Long Way from Bench to Bedside. Medicinal Research Reviews,* 34(5), 957–978. doi:10.1002/med.21309

Fujikawa, T., et al. (2005). Teratoma formation leads to failure of treatment for type I diabetes using embryonic stem cell-derived insulin-producing cells. *The American journal of pathology*, 166(6), 1781–1791. https://doi.org/10.1016/S0002-9440(10)62488-1

Harach, T., et al. (2017). Administrations of human adult ischemia-tolerant mesenchymal stem cells and factors reduce amyloid beta pathology in a mouse model of Alzheimer's disease. *Neurobiology of Aging,* 51, 83–96. doi:10.1016/j.neurobiolaging.2016.11.009.

Kaneko, N., Kako, E., & Sawamoto, K. (2011). Prospects and limitations of using endogenous neural stem cells for brain regeneration. *Genes,* 2(1), 107-130. https://doi.org/10.3390/genes2010107

Karvelas, N., et al (2022). Advances in stem cell therapy in Alzheimer's disease: a comprehensive clinical trial review. *Stem Cell Investigation*. https://dx.doi.org/10.21037/sci-2021-063

Kim, J. Y., et al. (2011). Soluble intracellular adhesion molecule-1 secreted by human umbilical cord blood-derived mesenchymal stem cell reduces amyloid-β plaques. *Cell death and differentiation*, 19(4), 680–691. https://doi.org/10.1038/cdd.2011.140

Kim, H. J., et al. (2021). Intracerebroventricular injection of human umbilical cord blood mesenchymal stem cells in patients with Alzheimer's disease dementia: a phase I clinical trial. *Alz Res Therapy* 13, 154. https://doi.org/10.1186/s13195-021-00897-2

Liu, X.-Y., Yang, L.-P., & Zhoa, L. (2020). Stem cell therapy for Alzheimer's disease. *World Journal of Stem Cells*, 787-802. https://dx.doi.org/10.4252/wjsc.v12.i8.787

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ISSN 2688-3651 [online]

Pacheco-Herrero, M., et al. (2021). Current Status and Challenges of Stem Cell Treatment for Alzheimer's Disease. *Journal of Alzheimer's disease : JAD*, 84(3), 917–935. https://doi.org/10.3233/JAD-200863

Patwardhan, A. G., & Belemkar, S. (2021). *An update on Alzheimer's disease: Immunotherapeutic agents, stem cell therapy and gene editing. Life Sciences,* 282, 119790. doi:10.1016/j.lfs.2021.119790

Phillips, M. I., & Tang, Y. L. (2008). Genetic modification of stem cells for transplantation. *Advanced drug delivery reviews*, 60(2), 160–172. https://doi.org/10.1016/j.addr.2007.08.035

Qin, C., Li, Y., & Wang, K. (2021). Novel Balance Mechanism Participates in Stem Cell Therapy to Alleviate Neuropathology and Cognitive Impairment in Animal Models with Alzheimer's Disease. *Cells*, 10(10), 2757. https://doi.org/10.3390/cells10102757

Si, Z. Z., et al. (2023). Targeting neuroinflammation in Alzheimer's disease: from mechanisms to clinical applications. *Neural regeneration research*, 18(4), 708–715. https://doi.org/10.4103/1673-5374.353484

Uccelli, A., et al. (2011). Neuroprotective features of mesenchymal stem cells. *Best practice & research. Clinical haematology*, 24(1), 59–64. https:// doi:10.1016/j.beha.2011.01.004

Vasic, V., Barth, K., & Schmidt, M. (2019). Neurodegeneration and Neuro-Regeneration — Alzheimer's Disease and Stem Cell Therapy. *International Journal of Molecular Sciences*, 1-21. http://dx.doi.org/10.3390/ijms20174272