Journal of Clinical and Experimental Transplantation

Editorial Open Access

Huntington's disease Stem Cell Transplantation's Potential Therapeutic Application and Pathogenesis

Diana Jacobs*

Department of Oncology, CMS Medical School, United Kingdom

Abstract

Repetitive CAG or glutamine expression along the Huntington gene's coding region results in the progressive neurodegenerative disorder known as Huntington's disease (HD). Certain abnormalities in movement, emotional disturbances, dementia, and cognitive impairments are the effects of this disease. To this date, there could be no legitimate remedy for this uncommon and deadly neurological condition yet there have been sure headways in the field of hereditary creature model examination studies to explain the comprehension of the pathogenesis of this condition. Currently, HD follows a specific therapeutic approach that only treats symptoms and does not address the disease's underlying cause. Stem cell therapy has the potential to be a game-changer in the search for a cure. The pathogenesis, efficacy, and clinical viability of the therapeutic application of stem cell transplantation in Huntington's disease have all been discussed in this review. A brief list and analysis of the applications of this revolutionary therapy on genetically modified animal models has been provided.

Keywords: Transplantation of Huntington's disease; Stem cells; Pathogenesis therapeutics

Introduction

The intellectual and cognitive function of the central nervous system deteriorates in neurodegenerative diseases as a result of a progressive loss of structure over time [1]. These diseases account for approximately 6.3% of all diseases [2]. Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS), and Huntington's disease are examples of neurodegenerative conditions that are typically observable in the elderly population. However, the underlying causes of this degradation are poorly understood. The therapeutic medications that have been approved by the Food and Drug Administration (FDA) are unable to stop disease progression or extend life expectancy due to the variety of these diseases and the fact that the clinical symptoms vary depending on the region of brain injury. Understanding the pathogenesis of various neurodegenerative diseases is essential to comprehending the therapeutic value of stem cells. The pathogenesis, efficacy, and clinical viability of the therapeutic application of stem cell transplantation in Huntington's disease will all be discussed in this review.

Stem cells and properties

Stem cells are types of cells that are capable of self-renewal and differentiation into a variety of cell types. The embryonic stem cells (ESCs) and the adult stem cells (ASCs) that are found in various adult tissues are the two types of mammalian stem cells. With these two types of stem cells, a new type of stem cell known as induced pluripotent stem cells (iPSCs) has been introduced. IPSC can be generated from fibroblasts and other types of cells [3]. The undifferentiated inner mass cells of a human embryo that are capable of producing all of the specialized tissues in the human body are the source of ESCs. ASCs are non-reproductive, undifferentiated cells that come from specific, differentiated human tissues. Somatic stem cells are another name for ASCs, which are not reproductive cells. There are a few different kinds of ASCs: Neural Stem Cells (NSCs) and Mesenchymal Stem Cells (MSCs) are two types of stem cells that are utilized in various therapeutic procedures. iPSCs are determined in the lab in a medium among ESCs and ASCs. Neurodegenerative disease research has utilized embryonic stem cells (ESCs), MSCs, brain-derived neural stem cells (NSCs), and induced pluripotent stem cells (iPSCs) among these stem cells.

A variety of cognitive and physical symptoms are associated with Huntington's disease (HD), an inherited neurodegenerative disorder characterized by selective neurodegeneration in the striatum [4]. It is an autosomal prevailing sickness brought about by a change in the huntingtin quality (HTT). In healthy individuals, this mutation increases the number of CAG trinucleotide repeats in exon1 above a critical threshold of 35 [5]. When this CAG repeat is translated in HTT, the polyglutamine (poly Q) tract at the huntingtin gene's N-terminus gets bigger. In HD, this Poly Q expanded HTT—also known as mutant HTT or mHTT—causes toxic function phenotypes like cytotoxicity and biochemical dysfunction [6]. The mHTT protein is thought to be causing a variety of transcriptional problems, axonal transport problems, and proteostasis problems.

A polyglutamine stretch (polyQ) is found at the N terminus of the Heat repeat of Human HTT. A variety of harmful N-Terminal fragments are produced by various proteolytic cleavages, including caspase 6 and other proteases. In addition, an alternative method for generating mutant fragments is the aberrant splicing of the first exon of the huntingtin protein [7]. These mutant fragments are correlated with the rising toxicity brought about by the formation of less toxic aggregates in the cytoplasm as opposed to the nuclear region, which may be a factor in differences in cell susceptibility.

Discussion

As Sneuronal division is impossible, the brain was thought to be a fixed system for a long time. However, with the development of

***Corresponding author:** Diana Jacobs, Department of Oncology, CMS Medical School, United Kingdom, E-mail: Diana33@hotmail.com

Received: 01-Nov-2022, Manuscript No: jcet-22-83426; **Editor assigned:** 04-Nov-2022, PreQC No: jcet-22- 83426 (PQ); **Reviewed:** 18- Nov-2022, QC No. jcet-22-83426; **Revised:** 22- Nov-2022, Manuscript No: jcet-22- 83426 (R); **Published:** 30- Nov-2022, DOI: 10.4172/2475-7640.1000148

Citation: Jacobs D (2022) Huntington's disease Stem Cell Transplantation's Potential Therapeutic Application and Pathogenesis. J Clin Exp Transplant 7: 148.

technology, it was discovered that neural stem cells originate from the hippocampus, followed by the olfactory bulbs, striatum, spinal cord, and septum. These cells can divide into neurons and glial cells through the generation of offspring [8]. Primate and rodent models have demonstrated remarkable potential for HD cell therapy. Motor function has improved, aggregate formation has decreased, and lifestyle improvements have been observed with NSC transplants [9]. IV organization of NSCs causes acceptance of useful recuperation by moving to the striatum and diminishes striatal decay in rat sore models of HD [10]. NSCs can be great for the treatment of HD for which further examination is extremely fundamental however the presence of moral restrictions may be a downside in the improvement of this cell treatment.

Conclusion

This article examines the positive effects of several stem cell-based neural repair strategies on neurological, cognitive, and electrophysiological changes in various HD rodent models. Stem cell reprogramming technology makes it possible to generate patient-derived cellular models of HD, which can assist us in a variety of ways, including comprehending the disease's pathogenesis and identifying potential therapeutic strategies. However, despite the varying results obtained with various types of stem cells on other rodent models, it remains unclear which strategy will be most effective.

Acknowledgement

The study was designed by YA. The study was designed by YA and SP. The study was directed by YA. SS and MD composed the primer draft composition. The manuscript's preliminary draft was looked over by YA and SP. The manuscript was edited, revised, and finalized by SS, MD, YA, and SP. The manuscript was read and approved by all authors.

Conflict of Interest

The authors declare no conflict of interests.

References

1. Kaufmann SH (2008) Immunology's foundation: the 100-year anniversary of the Nobel Prize to Paul Ehrlich and Elie Metchnikoff. Nat Immunol 9(7): 705-712.
2. Lahaie YM, Watier H (2017) Contribution of physiologists to the identification of the humoral component of immunity in the 19th century. MAbs 9(5): 774-780.
3. Kuruvilla J, Shepherd JD, Sutherland HJ, Nevill TJ, Nitta J, et al. (2007) Long-term outcome of myeloablative allogeneic stem cell transplantation for multiple myeloma. Biol Blood Marrow Transplant 13(8): 925-931.
4. Holstein SA, Suman VJ, Owzar K, Santo K, Benson DM Jr, et al. (2020) Long-Term Follow-up of CALGB (Alliance) 100001: Autologous Followed by Nonmyeloablative Allogeneic Transplant for Multiple Myeloma. Biol Blood Marrow Transplant 26(8): 1414-1424.
5. Musto P, Simeon V, Todoerti K, Neri A (2016) Primary Plasma Cell Leukemia: Identity Card 2016. Curr Treat Options Oncol 17(4): 19-25.
6. Nakajima R, Tate Y, Yan L, Kageyama T, Fukuda J (2021) Impact of adipose-derived stem cells on engineering hair follicle germ-like tissue grafts for hair regenerative medicine. J Biosci Bioeng 131(6): 679-685.
7. Kageyama T, Nanmo A, Yan L, Nittami T, Fukuda J (2020) Effects of platelet-rich plasma on in vitro hair follicle germ preparation for hair regenerative medicine. J Biosci Bioeng 130(6): 666-671.
8. Matsuzaki T, Yoshizato K (1998) Role of hair papilla cells on induction and regeneration processes of hair follicles. Wound Repair Regen 6(6):524-530.
9. Pedroza-González SC, Rodriguez-Salvador M, Pérez-Benítez BE, Alvarez MM, Santiago GT (2021) Bioinks for 3D Bioprinting: A Scientometric Analysis of Two Decades of Progress. Int J Bioprint 7(2): 3-33.
10. Kageyama T, Chun YS, Fukuda J (2021) Hair follicle germs containing vascular endothelial cells for hair regenerative medicine. Sci Rep 11(1): 6-24.