Perspective
Volume 9:2, 2023

Archives of Surgical Oncology

ISSN: 2471-2671 Open Access

Treatment of Neurological Conditions with Stem Cells

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Introduction

The disorders of the central and peripheral nervous systems that make up neurological disease are a diverse group and collectively account for the majority of global disease burden. The extent of treatment choices for neurological sickness is restricted, and drug endorsement rates for further developed therapies stay unfortunate when contrasted and other remedial regions. Although stem cell therapy offers hope to many patients, it is important to keep in mind that the scientific and medical communities have yet to fully comprehend the complexities of stem cell biology and provide sufficient data to support the rational, evidence-based therapeutic application of these cells. Starting with the fundamentals, we describe the current state of clinical trials and the advancements made in the last ten years as we provide an overview of the use of stem cells in neurological diseases. Neural, haematopoietic, and mesenchymal stem cells are among the many types of stem cells that can be used in stem cell therapy. Induced pluripotent stem cells and differentiated embryonic stem cells, two types of cell therapies, are also beginning to gain prominence. According to clinicaltrials.gov, more than 200 clinical studies using various stem cell treatments to treat neurological diseases have been registered to date. The majority of these studies target multiple sclerosis, stroke, and spinal cord injuries. We found 17 neurological indications that are currently in the clinical stage of development. With randomized clinical trial designs, few studies have progressed to large, pivotal studies. In the future, the approval and acceptance of such studies as mainstream treatments will depend on their findings.

Using retroviral vectors to introduce four genes that encode transcription factors Oct4, Sox2, Klf4, and c-Myc into mouse somatic cells, our research culminated in the 2006 induction of pluripotency. These cells were referred to as iPS cells by us. We were successful in creating human iPS cells in 2007 by employing genes that encode the same four transcription factors. By introducing key genes into differentiated adult cells, the cells could be reset to a state in which they possessed pluripotency at a very early stage of development, despite the fact that the developmental process was thought to be irreversible. This means that the results showed that the differentiation process could be reversed. The embryology textbooks had to be rewritten due to this shocking discovery.

Description

An effective strategy for producing microglia like cells from human ES and iPS cells from a variety of healthy and diseased subjects is described in this paper. We demonstrate that multiple ES and iPS cell lines can be efficiently used to produce and enrich microglia like cells by utilizing the most recent insights into microglial differentiation. The recently established consensus signature that distinguishes microglia from other macrophages is reflected in the expression signatures of this pluripotent stem cell derived microglia, which resemble those of purified human fetal microglia maintained in the same culture conditions. They respond to canonical stimuli and perform the functions of professional phagocytes, are positive for microglial markers like TMEM119. Human microglia maturation and steady state behavior in a highly defined cellular environment, as well as their involvement in disease onset, progression, and resolution, can now be studied using our findings as a framework.

Adult neurogenesis is also heavily influenced by hormones, cytokines, growth factors, and neurotrophins. Mostly through a variety of neurotransmitter systems, pharmacological manipulations can regulate various phases of adult neurogenesis. The olfactory bulb and dentate gyrus are both enriched with inputs from numerous brain regions that release various neuropeptides and neurotransmitters. Glutamate, GABA, and possibly acetylcholine are classic neurotransmitters that directly control the migration, maturation, integration, and survival of newly formed neurons. In the majority of other instances, it is unclear whether pharmacological manipulations affect neural precursors and newly formed neurons directly or indirectly through niche modulation. It is interesting to note that changes in serotonin and nonrepinephrine levels caused by the use of antidepressants in clinics increase the survival of newly formed neurons in the adult hippocampus, accelerate dendritic development, and increase the proliferation of neural progenitor cells.

Di Domenico et al., compared the cells with iPSC derived astrocytes and dopaminergic neurons from healthy controls to generate iPSC derived astrocytes and dopaminergic neurons from familial PD patients carrying the G2019S mutation. They demonstrated that synuclein, which was secreted by the PD iPSC derived astrocytes, caused a non-cell autonomous neuronal dysfunction and a neurotoxic effect on the dopaminergic neurons in the vicinity. They also found that the PD iPSC derived astrocytes had progressive synuclein accumulation, impaired macroautophagy,

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Received: 09 December, 2022, Manuscript No. ASO-22-82995; **Editor assigned:** 12 December, 2022, PreQC No. ASO-22-82995 (PQ); **Reviewed:** 27 December, 2022, QC No. ASO-22-82995; **Revised:** 03 March, 2023, Manuscript No. ASO-22-82995 (R); **Published:** 13 March, 2023, DOI: 10.37421/2471-2671.2023.9.39

and dysfunctional Chaperone Mediated Autophagy (CMA). Through the removal of synuclein accumulation, the reactivation of CMA also safeguarded the PD iPSC derived astrocytes and dopaminergic neurons. Astrocytes' non-cell autonomous role in PD pathogenesis was described in these findings.

Wada and co. using gene editing, human ESCs overexpressing the SOD1 mutant (G93A) were transformed into spinal motor neurons and astrocytes. It was discovered that the astrocytes produced substances that were harmful to the spinal motor neurons. Tyzack and co. showed that injured motor neurons have higher levels of the Ephrin type-B receptor (EphB1) and that EphB1 causes astrocytic STAT3 signaling, which is followed by an astrocyte protective and anti-inflammatory signature. They demonstrated that EphB1 and the subsequent pathway are affected by the SOD1 mutation (D90A) and produced astrocytes from ALS patient derived iPSCs. The neuroprotective astrocytic response was found to be dysfunctional and astrocytic toxicity increased in these ALS studies.

Conclusion

Numerous neurological disorders are mediated by astrocytes, but specifics are still lacking. Astrocytes derived from human PSCs with disease specific gene mutations can be an effective tool for *in vitro* disease mechanisms and cellular phenotypes research. It is anticipated that these models will provide insight into the mechanisms underlying how astrocytes contribute to neurological disorders.

How to cite this article: Yamanaka, Shinya. "Treatment of Neurological Conditions with Stem Cells." *Arch Surg Oncol* 9 (2023): 39.