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Therapeutically Growth in Oligodendroglia Fate Induction via Trans Differentiation of Stem Cells for Neurodegenerative Therapy

Darcy Armour*

Department of Microbiology, College of Medicine, Ethiopia

Abstract

The benefits of stem cell therapy and research are undeniable given how frequently they are used to treat demyelinating illnesses and neurodegenerative diseases. Prior to transplantation or disease modelling research, stem cells are induced into several cell lineages, both adult and progenitor - belonging to each germ layer - in cell replacement treatment. The nervous system contains a large number of glial cells, including oligodendrocytes, which can remyelinate axons that have lost or damaged myelin sheaths as well as myelinate newly formed neurons. Yet, myelin breakdown and regeneration are drastically outpaced by demyelinating illnesses. Mesenchymal stem cells (MSCs) have been stimulated to transdifferentiate in order to make up for this loss, examine the abnormalities in remyelination pathways, and accelerate full recovery in such individuals.

Keywords: Oligodendroglial fate induction; Stem cells; Neuroregenerative therapy

Introduction

Neurodegenerative illnesses frequently include progressive and untimely neuronal cell loss. In general, neurodegenerative illnesses are an outcome of a broad spectrum of factors like age, heredity, and environment, and the causation of each varies correspondingly [1]. The processes that cause neuronal degeneration are currently being investigated. The prevention and treatment of neurological illnesses must be improved immediately [2]. As they preserve the neurons in a functional state, the supporting cells of the nervous system known as glial cells are as important to a portion of neurodegenerative illnesses as the neurons themselves. The nervous system's homeostatic and protective neuroglia is comprised of very varied cellular populations with varying ancestry, structure, and function.

Mesenchymal stem cells

Glial cells called oligodendrocytes create myelin, the coating that covers axons and is rich in lipids. In the central nervous system (CNS), the myelin sheath is necessary for the salutatory conduction of action potentials. Any harm to the oligodendrocytes or the myelin sheath has a serious impact on how well neurons function. Demyelination of axons is an essential element causing function loss in many neurological conditions (multiple sclerosis, stroke, spinal cord injury (SCI), trauma, leukodystrophies, etc.). Unexpectedly, recent research on the deterioration and death of motor neurons found that oligodendrocytes (OLs) play an early involvement in the onset of multiple sclerosis (MS). The causes of MS-related neurodegeneration caused by the auto-inflammatory response are unclear. Another significant disadvantage is the lack of medicines that can take advantage of people with high risk levels who are genetically predisposed to MS. Similar to progressive MS, oligodendrocyte progenitor cell (OPC) maturation abnormalities and OL degeneration in ALS lead to myelin degradation and eventual neuronal death [3,4]. It has been suggested that ALS-causing genes that interfere with the efficiency of myelin-related mRNA processing are the ones that cause OL disintegration in ALS patients. Moreover, these genes produce protein mutants that agglomerate and become toxic. Routes that directly control OL differentiation are negatively impacted, which could result in OPCs failing to mature as is the case with ALS. In ALS, the normal metabolic and trophic support provided by OLs to neurons is down-regulated, which significantly impairs axon shape and function [5, 6]. All neuronal degeneration cases include intricate mechanisms with numerous cascading pathways. In laboratory models, researchers have had some success targeting one molecule at a time and blocking one route at a time. There hasn't yet been any human success using these techniques. The specific cause of neuronal cell death and the aetiology of the majority of these disorders are unknown at this time. Even though the pathogenic processes are poorly understood, earlier research indicates that significant function recovery may be possible by demyelination of undamaged axons. Functional recovery by demyelination may show to be a practical replacement and regeneration strategy when used as a treatment technique [7].

A desirable cell source for cell therapy and regeneration is stem cell therapy. The potential use of neuronal replacement therapy has significantly improved because to the use of stem cells and strictly regulated cellular programming techniques. The length of the motor axons and the mounting evidence that the neurodegeneration in ALS may be mediated by neuronal and glial effects make stem cell therapy more difficult in ALS than in other neurodegenerative illnesses. Recent research demonstrated that stem cells can multiply, migrate, and develop into neurons and glial cells within injured brain and spinal cord tissue [8].

Conclusion

Without the efforts, mistakes, and corrections made with ESCs and iPSCs, the current procedures for oligodendroglia lineage commitment would not exist. 3-D cultures have emerged as an innovative example of differentiation induction techniques that can imitate in vivo conditions [9, 10].

***Corresponding author:** Darcy Armour, Department of Microbiology, College of Medicine, Ethiopia,E-mail: Darcy23@gmail.com

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Conflict of Interest

None.

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