Commentary
Volume 16:1, 2022

Journal of Molecular and Genetic Medicine

ISSN: 1747-0862 Open Access

Role of Stem Cell Therapy in Lung Diseases

Veena Priyadarshini*

Department of Life Sciences, BSAR University, Chennai, Tamil Nadu, India

Commentary

Although new medications have been demonstrated in clinical trials to be beneficial in slowing disease development and increasing quality of life in individuals with pulmonary fibrosis, the damaged lung tissue does not regenerate. The establishment of regenerative therapy, such as stem cell therapy or tissue engineering, is critical. Furthermore, therapeutic application of mesenchymal stem cell (MSC) therapy in individuals with idiopathic pulmonary fibrosis has been found to be safe (IPF). A combination of MSC transplantation and pharmacological therapy appears to provide additional benefits; nevertheless, an experimental design to test its efficacy is still needed. Idiopathic pulmonary fibrosis (IPF) is a type of fibrosing interstitial pneumonia that develops over time for no apparent reason. It affects primarily older persons (median age at diagnosis: 66 years, range: 55–75 years), is restricted to the lungs, and is characterized by fibroblast proliferation and extracellular matrix remodeling, which leads to gradually worsening dyspnea and pulmonary function, with a dismal prognosis. On high-resolution computed tomography, the diagnosis of IPF needs the histopathologic and/or radiologic pattern of typical interstitial pneumonitis.

Other known causes of ILD must be ruled out first, such as connective tissue diseases, medication toxicity, and other environmental exposures. IPF is a deadly lung disease with a three- to five-year median survival time. The majority of patients experience a slow and steady deterioration over several years, with some people remaining stable and others experiencing a rapid decrease. A small percentage of IPF patients (about 5–10 percent) may experience an acute exacerbation each year. The global incidence of IPF is estimated to be between 4.6 and 16.3 cases per 100,000. An estimated prevalence of between 14.0 and 42.7 per 100,000 people was reported based on healthcare claims data from the United States. Several studies have found that the antifibrotic medicines nintedanib and pirfenidone dramatically reduce the decline in forced vital capacity (FVC), decrease acute exacerbation, and improve mortality rates in individuals with IPF. They have recently been conditionally recommended for the treatment of IPF in numerous guidelines; nonetheless, present therapy can only halt the advancement of IPF and cannot reverse the fibrosis process.

Although numerous potential targets from animal studies have proven their potential, there is currently a shortage of effective choices for treating IPF. Even though some off-label medications, such as inhaled NAC, may lessen the time it takes to complete a meta-analysis, the response is limited when compared to targeted target therapy. Previous research has yielded benefits for both stem cell and pharmaceutical treatments, but there is currently a dearth of data on the combination of the two [1-2].

Regulatory mechanism

IPF progression is a complicated process including epithelial-dependent fibroblast-activated mechanisms and a poor response to anti-inflammatories, at the very least. The downstream pathways of epithelial mesenchymal transition (EMT) and an immune response are involved in these consequences. Through the Wnt and BMP pathways, several EMT regulators, including as WISP-1 and BMP4, have been identified to have a role in fibroblast differentiation and collagen synthesis, respectively. In myofibroblasts and fibroblasts, PGE2 pathways can inhibit Fas ligand-induced apoptosis; however, alveolar type II cells are still susceptible to apoptosis due to their reduced production of PGE2. Apoptosis in alveolar epithelial cells but not in myofibroblasts or fibroblasts could be explained by variations in expression [3-4] .

Cell cycle-related proteins have also been suggested as a possible treatment for IPF. Skp2 (S-phase kinase-associated protein 2) is an effector protein that promotes p27 degradation and is required for IPF progression. BLM-induced lung fibrosis could be prevented by increasing p27 expression with an antagonist for Skp2, SZL-P1-41. Furthermore, BLM therapy promotes ER stress-related proteins such as GRP78, CHOP, and ATF4 in fibroblasts, as well as enhancing fibroblast proliferation via the PI3K/Akt pathway. Another upstream regulator of EMT, the PI3K/Akt pathway, is activated, making the response more unexpected and irreversible. Despite the fact that each signalling pathway has few connections, evidence has indicated that using inhibitors like LY294002 and Tauroursodeoxycholic acid (TUDCA) to treat pulmonary fibrosis is effective. MSCs generated from bone marrow (BM) are used more frequently in mechanism investigations than in clinical studies, possibly because the source is easier to come by.

The immunomodulatory effects of the injected cells are expected to help heal lung tissue. In terms of immunology, BM-derived MSCs had lower levels of expression of the immunosuppressive markers PDL-1 and CD1a than placenta-derived MSCs, and IFN-induction had a lesser ability to activate T cell proliferation in BM-derived MSCs than placenta-derived MSCs. Placenta-derived MSCs, on the other hand, suppressed cytokine-stimulated natural killer lymphocytes, though the cell type is still unknown. In addition, IPF is frequently accompanied by inflammation and profibrotic cytokinesis. MSCs reduce IL-1, IL-6, TNF-, TGF-, and vascular endothelial growth factor (VEGF) in BLM-induced lung injury to produce an anti-inflammatory effect. Although no leftover stem cells were found in injured lungs in these studies, the secreted circulating mediators were considered to improve the therapeutic effects for IPF.

Because of their multi-lineage differentiation potential, migratory ability, and self-renewal properties, mesenchymal stem cells (MSCs) have received increased attention in the field of regenerative applications in recent years. MSCs can be found in a variety of organs and tissues, including bone marrow and adipose tissues, and can travel to injury sites. MSCs have been shown to have therapeutic value in ischemic heart failure, pulmonary arterial hypertension, stroke, chronic kidney disease, and sepsis, among other diseases [5].

***Address for Correspondence:** Veena Priyadarshini, Department of Life Sciences, BSAR University, Chennai, Tamil Nadu, India; E-mail: veenapriya31@gmail.com*

Received: 05-Jan-2022, Manuscript No: jmgm-22- 52700; **Editor assigned:** 07- Jan-2022, Pre QC-No. P- 52700; **Reviewed:** 12- Jan-2022, QC No. Q- 52700; **Revised:** 17- Jan -2022, Manuscript No. R- 52700; **Published:** 22- Jan-2022, DOI: 10.37421/1747-0862.22.16.525

References

1. Agostini, Carlo. "Stem cell therapy for chronic lung diseases: hope and reality." *Resp Med* 104 (2010): S86-S91.
2. Weiss, Daniel J., Ivan Bertoncello, Zea Borok, Carla Kim, and et.al . "Stem cells and cell therapies in lung biology and lung diseases." *Proceedings of the American thoracic society* 8(2011): 223-272.

3. Geiger, Sabine, Daniela Hirsch, and Felix G. Hermann." Cell therapy for lung disease." *Euro respi review* 26(2017).
4. Tzouvelekis, Argyris, Paschalis Ntolios, and Demosthenes Bouros. "Stem cell treatment for chronic lung diseases." *Respiration* 85 (2013): 179-192.
5. Chrzanowski, Wojciech, Sally Yunsun Kim, and Lana McClements. "Can stem cells beat COVID-19: advancing stem cells and extracellular vesicles toward mainstream medicine for lung injuries associated with SARS-CoV-2 infections." *Front Bioeng Biotechnol* 8 (2020): 554.

How to cite this article: Priyadarshini, Veena. "Role of Stem Cell Therapy in Lung Diseases." *J Mol Genet Med* 16(2022): 525.