Chrishan S Samuel, J Cell Sci Ther 2017, 8:1 (Suppl)
http://dx.doi.org/10.4172/2157-7013.C1.038

Enhancing the protective effects of stem cell-based therapies with an anti-fibrotic drug in experimental chronic allergic airways disease

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While stem cell-based therapies have demonstrated immunomodulatory, anti-inflammatory and tissue-reparative functions in acute disease settings, they are less effective when administered to chronically damaged organs. This is likely attributed to tissue fibrosis which can impair stem cell survival, proliferation, migration and integration with resident tissue cells. Hence, the therapeutic efficacy of human bone marrow-derived mesenchymal stem cells (MSCs) or human amnion epithelial stem cells (AECs) were evaluated in the setting of chronic allergic airways disease (AAD), in the absence or presence of an anti-fibrotic drug (serelaxin; RLX). Female Balb/c mice subjected to the 9-week model of ovalbumin (OVA)-induced chronic AAD, were either vehicle-treated (OVA alone) or treated with MSCs or AECs alone (intranasally (i.n)-administered with 1x106 cells once weekly), RLX alone (i.n-administered daily) or a combination of MSCs or AECs and RLX from weeks 9-11 (n=6/group). Measures of airway inflammation (AI), airway remodeling (AWR) and airway hyper-responsiveness (AHR) were then assessed. OVA-injured mice exhibited exacerbated AI, epithelial damage/thickness, sub-epithelial and total collagen deposition (fibrosis) and AHR compared to their saline-treated counterparts (all $p<0.01$ vs. saline-treated controls). RLX or AECs (but not MSCs) alone normalized epithelial thickness and partially diminished the OVA-induced fibrosis and AHR by ~40-50% (all $p<0.05$ vs. OVA alone). Furthermore, the combination treatments normalized airway epithelial thickness, fibrosis and AHR to that in normal mice and significantly decreased AI. These findings showed that the presence of an anti-fibrotic enhanced MSC- or AEC-induced reversal of the three central components of chronic AAD/asthma, to a greater extent than stem cell-treatment alone.

Biography

Chrishan S Samuel is an Associate Professor and has completed his PhD from University of Melbourne, Victoria, Australia. He has completed his Post-doctoral studies at the Stanford University School of Medicine and Molecular Medicine Research Institute. He is currently a Senior Research Fellow of the National Health & Medical Research Council of Australia and Head of the Fibrosis Laboratory at the Department of Pharmacology, Monash University (Melbourne, Victoria, Australia). He has over 125 career publications, which have been cited over 5100 times and his research interests are focused on establishing novel therapeutic strategies for organ fibrosis.

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