ATS 2024 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?

Basic science and translational.

Tell us about your research?

My research mainly deals with investigating the therapeutic efficacy of biological therapy and chemical agents in developmental pulmonary disorders. I mainly focus on novel compounds that can activate the transcription factors involved in the normal lung development and/or tissue repair after injury. In addition, I also deal with biological therapy which is one of the novel approaches to cure the rare developmental disorders. Validating and/or improving the efficiency of the cell therapy approaches will be a significant contribution in the field. This research is a significant contribution towards unravelling the pharmacological importance of drugs and biological agents which could lead to development of new therapies in rare developmental disorders.

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Where do you see yourself in 5 years?

In the next five years, I aspire to continue my research journey towards in-depth analysis of lung disorders and finding novel compounds from natural origins that can be used to treat the pulmonary disorders. In addition, I am also eager to apply my knowledge and expertise by participating in university level teaching and research to help new talent to secure their future goals in the field and to make a significant contribution of my research in academia.

What do you find is the major benefit of RSF Assembly Membership?

Networking, friends.

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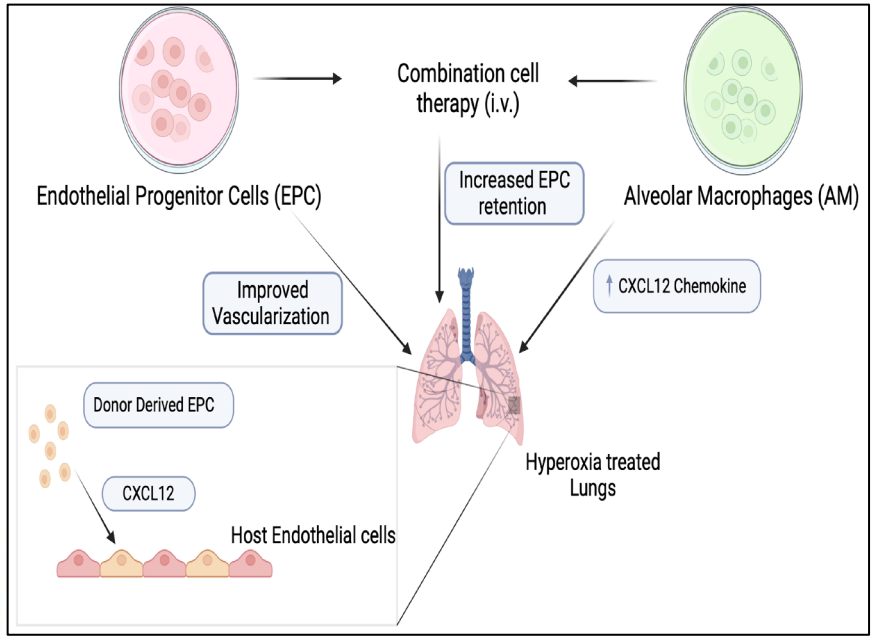

Co-Transplantation of Alveolar Macrophages Improves the Efficacy of Endothelial Progenitor Cell Therapy in Mouse Model of Bronchopulmonary Dysplasia

Objective: To improve the efficacy of endothelial cell therapy in mouse model of bronchopulmonary dysplasia (BPD).

Methods: Fluorescence-activated cell sorting (FACS) was used to purify rAM (CD45+CD64+SiglecF+) from neonatal mice lungs expressing green fluorescent protein gene Tg(CAG-EGFP), or cKIT+ EPCs (CD31+cKIT+CD45-) from neonatal mice lungs expressing red fluorescent protein gene Tg(DsRed.MST), on C57BL/6 background. Recipient mice were exposed to 85% hyperoxia from P0-P3. Following hyperoxia, recipient mice were transplanted with FACS sorted cKIT+ EPCs, rAM or combination of both through retro-orbital injection. In another set of mice, rAM were depleted using chlodronate- containing liposomes prior to transplantation of cKIT+ EPCs. Recipient mice were harvested at P18 to analyze the retention of donor-derived endothelial cells in lungs using flow cytometry. Structural changes in lungs were analyzed by quantifying airspace diameter and capillary density.

Results: Transplantation of rAM alone did not result in engraftment of donor rAM into the lung tissue, but increased the protein concentration of proangiogenic CXCL12 chemokine in recipient mouse lungs. Depletion of rAM by chlodronate-liposomes decreased the retention of donor EPCs after their transplantation into hyperoxia-injured lungs. Adoptive transfer of rAM in combination with EPCs enhanced the therapeutic efficacy of EPCs as evidenced by increased retention of EPCs in the lung tissue, increased capillary density and improved alveolarization in hyperoxia-injured mouse lungs.

Conclusion: Current study demonstrated that rAM mediate the efficacy of EPC therapy in neonatal hyperoxia injury by facilitating the retention of transplanted EPCs and augmenting the vascularization (Figure 1). This property of rAM may lead to the development of new cell therapies directed on enhancing the engraftment and efficacy of EPCs therapy in BPD.

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