ATS 2025 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Jazmin Calyeca, PhD

(She/Her/Hers)
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Is your research clinical, basic science or translational?

Translational.

Tell us about your research?

My research focuses on understanding *how aberrant repair alters the phenotype and function of epithelial stem cells and mesenchymal cells, particularly in pediatric airway surgery and lung diseases*. By leveraging advanced bioinformatics and preclinical models, I aim to uncover the mechanisms underlying these complications and translate this knowledge into therapeutic interventions.

Where do you see yourself in 5 years?

In five years, I envision myself as a leading independent investigator dedicated to understanding the molecular and cellular mechanisms participating in tissue regeneration and repair. My goal is to contribute to the development of innovative therapeutic approaches for children with airway defects by bridging basic research with translational applications, particularly in the context of airway/lung regeneration and fibrosis.

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

Being a member of the RSF Assembly has deepened my involvement in clinical and translational research, providing valuable opportunities to engage with cutting-edge advancements in pulmonary science. It has also allowed me to expand my professional network, enriching my perspective, and providing invaluable opportunities for knowledge exchange and interdisciplinary partnerships.

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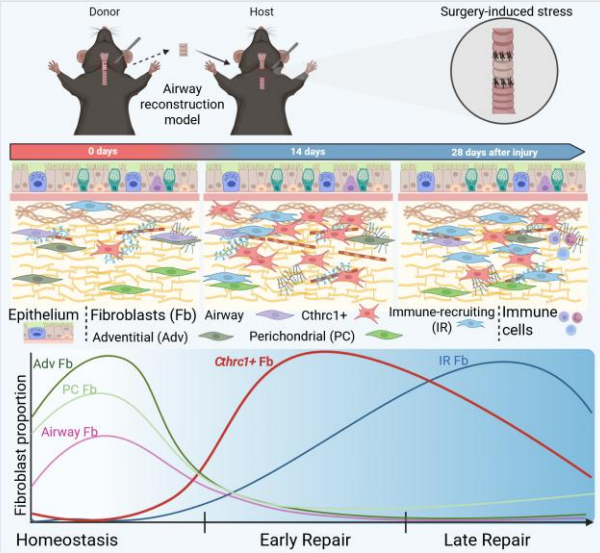

Schematic of surgical resection model and emergence of fibroblast subpopulations during airway repair.

Orchestrated response from heterogenous fibroblast subsets contributes to repair from surgery-induced stress after airway reconstruction.

JCI Insight. 2025;10(5):e186263. https://doi.org/10.1172/jci.insight.186263.

Objective: In this study, our aim is to delineate cellular hubs and interactions involved in anastomotic repair in response to surgery-induced stress (SIS) after airway reconstruction and elucidate how disruptions in the activated pathways can lead to pathological conditions such as fibrosis.

Methods: We used a long-term survival preclinical model of segmental airway reconstruction, multiplex immunofluorescence microscopy, and single-cell RNAseq to determine how the cellular composition of the graft responds to surgical stress and characterize changes in their specific gene expression profiles in the absence of chronic airway disease.

Results: We found that the surgical trauma triggers a stress response (surgery-induced stress) that acts on homeostatic fibroblasts and influences them to differentiate through transitional states. Evidence of fibroblast contribution to airway repair following reconstruction involves differentiation to chondrocytes, orchestration of immune response, and regulation of collagen production/remodeling. Specifically, we identified the presence of a Cthrc1+ fibroblast population as the major contributor to the remodeled ECM, affecting Fibroblast-basal cell crosstalk.

Conclusion: We conclude that response to SIS involves a coordinated emergence of distinct fibroblast subsets participating in airway repair remodeling and a possible determinant of post-surgical stenosis and/or fibrosis.

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