ATS 2022 Highlights

Respiratory Structure and Function Early Career Professionals

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Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?

Basic Science

Tell us about your research?

My research focuses on the role of macrophages in severe asthma and cystic fibrosis. I study how macrophages contribute to airway inflammation and remodeling in severe asthma. Additionally, I am interested in how bacterial infection affects macrophage inflammatory responses in severe asthma and cystic fibrosis.

Where do you see yourself in 5 years?

I am currently applying for the K99/R00 Pathway to Independence Grant that will allow me to be competitive for tenure-track faculty positions. Over the next five years, I plan to establish my own research program as an independent investigator at a top-tier research institution studying lung immunology.

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

The major benefit of RSF Assembly Membership is the connections with peers and senior researchers that we are able to form throughout the Assembly. Through programs such as the Early Career Professionals Group and Mentor Match Program, I have been able to meet and learn from extraordinary scientists in the field.

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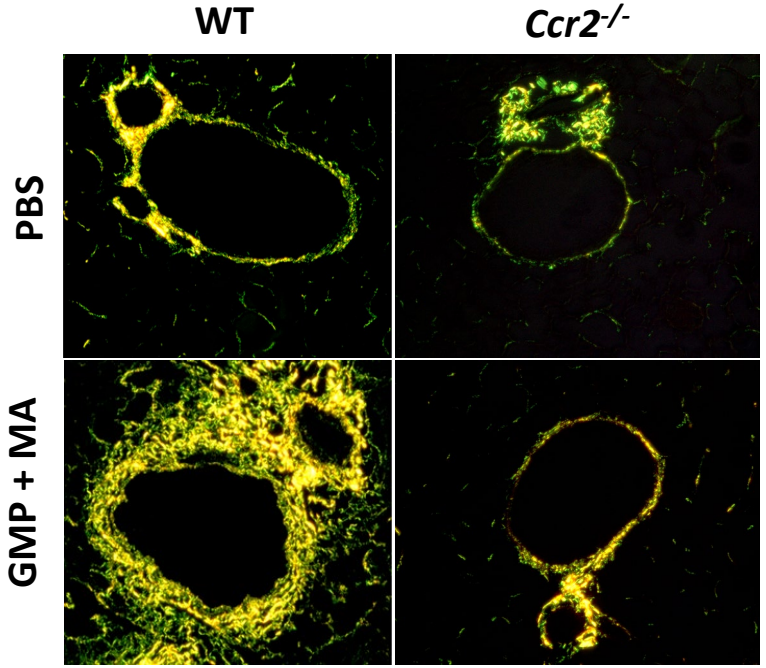

Figure 1. Airway Remodeling. GMP + MA-challenged *Ccr2*$^{-/-}$ mice exhibited decreased collagen deposition around the airways.

Recruited Macrophages Contribute to Immune Cell Infiltration and Remodeling in a Mouse Model of Severe Allergic Airway Inflammation

Objective: Severe asthma is characterized by airway inflammation, remodeling, and impaired lung function that are insensitive to corticosteroids. Studies suggest that macrophages contribute to severe asthma but their role in allergic airway inflammation and remodeling remains unclear.
Methods: Wildtype (WT) and *Ccr2*$^{-/-}$ (impaired monocyte recruitment) newborn mice were sensitized/challenged intranasally with PBS or c-di-GMP + mixed allergen (GMP + MA) 3X a week for 7 weeks. Bronchoalveolar lavage fluid (BAL) was collected 24 hours after last allergen challenge and analyzed for total and differential cell counts and cytokine analyses. Additionally, lung function, histological analyses for peribronchiolar/perivascular airway inflammation, and airway collagen deposition analyses were performed.
Results: Compared to PBS-challenged WT mice, GMP + MA-challenged WT mice exhibited significantly higher CCL2 (CCR2 ligand) expression that was not reduced by FP. Compared to WT mice, *Ccr2*$^{-/-}$ mice challenged with GMP + OVA exhibited lower total cell counts, especially eosinophils. Airway inflammation, collagen deposition, and airway hyperresponsiveness were significantly decreased in GMP + MA-challenged *Ccr2*$^{-/-}$ mice.
Conclusion: Our data suggest that recruited macrophages contribute to immune cell infiltration, airway inflammation, airway remodeling, and airway hyperresponsiveness in severe allergic airway inflammation. These novel studies highlight the need to further understand the role of macrophages in severe asthma.