ATS 2025 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 and translational

Tell us about your research?

My research focuses on elucidating the role of pericytes in airway remodeling associated with allergic asthma. This often focuses on the contribution of growth factor and cytokine signaling to the migration and differentiation of pericytes, in particular TGF-β, periostin, IL-13 and CXCL12. Part of my research also concerns the development of potential therapeutics targeting these pathways, with two prospective drugs currently being in the pipeline. I am also currently in the process of commercializing my research and forming a spinout company which is encouraging me to work outside of my comfort zone and develop my business acumen.

Where do you see yourself in 5 years?

In 5 years I hope to be leading my company through Phase I clinical trials and potentially negotiating a buyout. I'd like to then return to more fundamental research and potentially start my own lab to continue to study pericytes in lung conditions. I'd also like to contribute more to scientific communication, whether in a teaching capacity or to external bodies as I love to get people excited about science!

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

The RSF is a great platform to meet influential and supportive researchers and like-minded colleagues who have really encouraged me during my career. It also has an added benefit for me as it has introduced me to the US research space and has given me invaluable international contacts.

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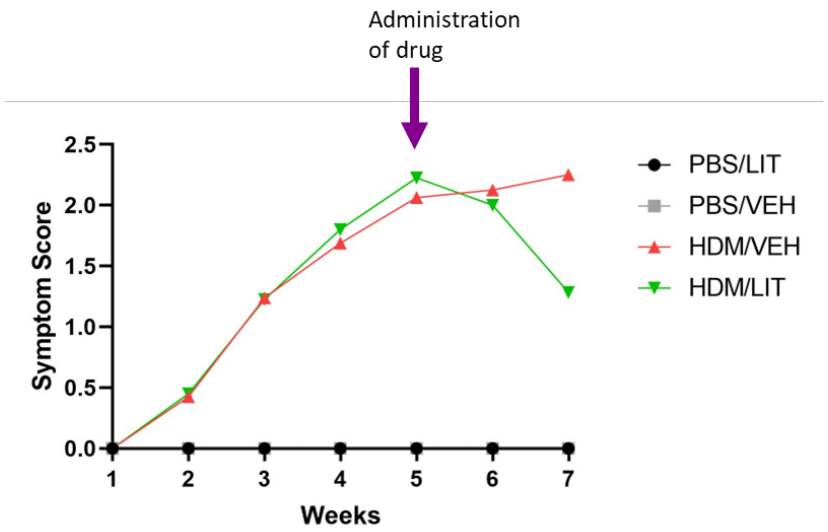

Female C57/Bl6 mice (6–8 weeks old) were subjected to intranasal delivery of sterile PBS (10 µl) or house dust mite extract (HDM; 25 µg in 10 µl) 5 days a week for 7 consecutive weeks. Commencing on week 5, mice were also subjected to intranasal delivery of methyl-β-cyclodextrin (VEH, 10% w/v) or LIT-927 (LIT, 197ng/ml) 5 days a week for the remaining 2 weeks. Symptom scores were monitored throughout the 5-week protocol. Data shown is the average score throughout each week.

Abstract title: Therapeutic Intervention with Novel Drug Abrogates Aspects of Airway Remodeling in a Mouse Model of Severe Allergic Asthma

Objective: It is estimated that around 10-15% of the 260 million people worldwide who suffer from asthma do not respond well to currently available treatments. Gold standard treatments for asthma consist of a long-acting beta agonist for bronchodilation and a corticosteroid for immunomodulation. Neither of these target airway remodeling, the structural changes underlying the pathophysiology of asthma. We have designed a novel first-in-class drug to target remodeling and halt or even reverse these changes. We have previously shown efficacy in a mouse model of mild asthma, but here we aim to explore its effects in a mouse model of severe allergic asthma.

Methods: The house dust mite (HDM) model of allergic asthma was used in which male and female mice were intranasally dosed with HDM 5 times a week for 7 weeks (n= 6). After 5 weeks of HDM exposure, mice were treated with LIT-927 (300 nmol/kg) for 2 weeks. Throughout the experiment symptoms were tracked visually using a 5-point scale. Lung sections and tracheas were obtained and interrogated via immunostaining for markers of fibrosis (α-SMA) and our drug target CXCL12. In addition, bronchoalveolar lavage samples were taken to further enumerate immune cells.

Results: Chronic HDM exposure reliably induced dyspnoea which was seen to reduce following treatment with LIT-927. In results from bronchoalveolar lavage analysis, no effect was shown on the number or proportion of immune cells such as macrophages or eosinophils following treatment with LIT-927. Despite this, structural changes are seen to be abrogated following LIT-927 treatment with fibrotic markers and CXCL12 levels appearing markedly reduced in immunostained images. In addition, the number of migrating pericytes seen in the tracheal images were decreased following treatment with LIT-927.

Conclusion: In conclusion, treatment with our drug has been shown to reduce structural changes observed in a mouse model of severe allergic asthma without modulating the immune response. This both highlights a prospective alternative treatment for those patients who currently do not respond well to corticosteroids and shows a potential for the development of a three-pronged treatment that can target both the immune response and the structural changes in the future.

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