ATS 2025 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

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Is your research clinical, basic science or translational?

Basic science.

Tell us about your research?

My research explores the complex relationship between maternal obesity and offspring respiratory health, shedding light on the intricate physiological mechanisms that contribute to asthma pathogenesis. I focus on why nutritional interventions during pregnancy are critical to preventing the transgenerational programming of obesity and asthma in children. To investigate this, I use mouse models exposed *in utero* to a maternal high-fat diet, examining how this influences the development of airway hyperresponsiveness, nerve hyperinnervation, and insulin resistance across genetically diverse offspring. I assess changes in airway epithelial sensory nerve density, airway function, and metabolic health using innovative, cutting-edge techniques developed in our laboratory. Ultimately, this work aims to deepen our understanding of how maternal obesity impacts asthma and obesity risk in the next generation

Where do you see yourself in 5 years?

Fast forward five years, I see myself deeply immersed in maternal-offspring physiology, working to uncover the mechanisms behind airway dysfunction in children and ultimately breaking the cycle of respiratory disease linked to maternal obesity. To achieve this, I plan to pursue a postdoctoral position where I can further hone my technical and intellectual skills. Most importantly, I am committed to sharing my knowledge and mentoring the next generation of scientists—because there's a place for everyone in science, and I'm here to help make sure that remains true!

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

Access to supportive researchers and clinicians is an unmatched resource and experience provided to trainees by the RSF Assembly. Membership has consistently facilitated trainee networking, access to funding opportunities, and most importantly a shared passion to improve global respiratory health.

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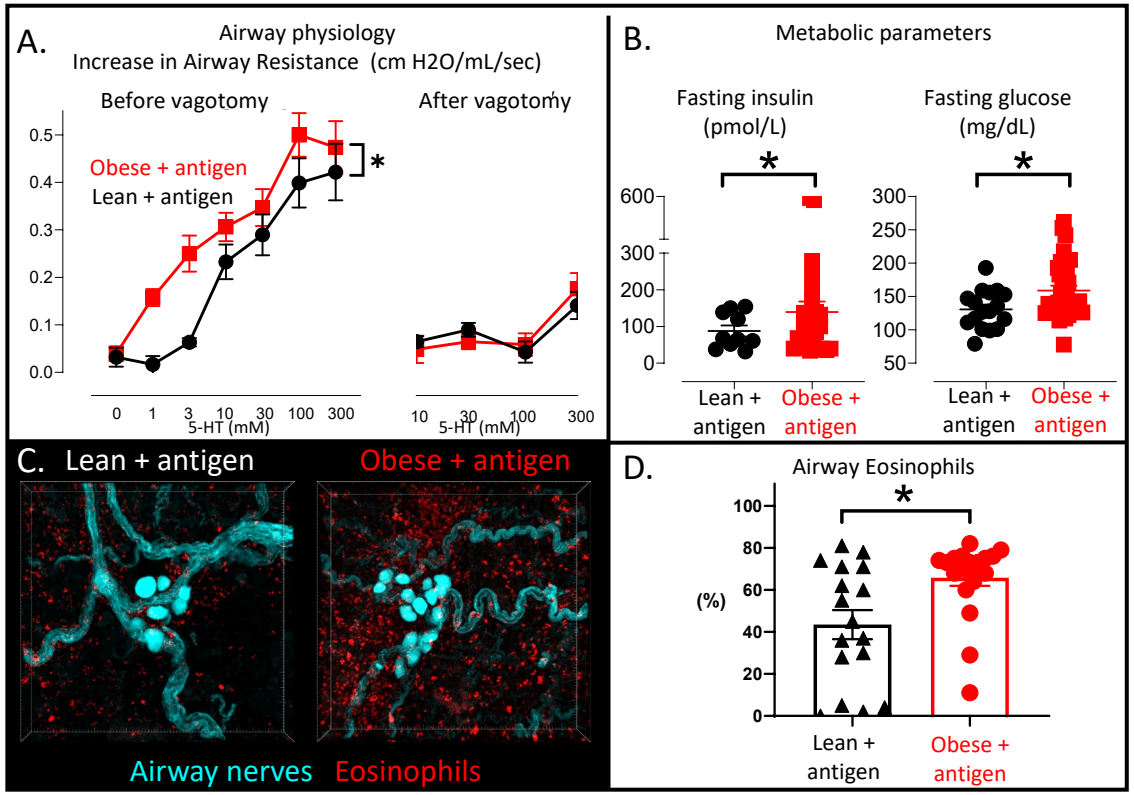

Antigen treatment in offspring of obese dams potentiates nerve mediated bronchoconstriction (A), metabolic parameters (fasting insulin, fasting glucose (B), and airway eosinophils (D). Representative immunostaining images are shown in (C).

Maternal Obesity Potentiates Airway and Inflammatory Responses to Allergen in Offspring

Objective: Children born to obese mothers are more likely to develop asthma, yet the underlying mechanisms remain unclear. Both airway allergen exposure and maternal obesity separately increase airway nerve-mediated reflex bronchoconstriction in mice, and are mediated by different mechanisms, eosinophils (allergen) and insulin (obesity). Here we test whether airway hyperresponsiveness to house dust mite (HDM/antigen) is further increased in adult offspring of obese dams

Methods: Six-week-old female C57BL/6 mice were fed a 45% high-fat diet (HFD) or a 14% regular diet (RD) for 8 weeks before breeding and throughout pregnancy and lactation. After weaning, all offspring were fed a regular diet. At 15 weeks of age, some offspring were sensitized with HDM (25 uL of 2 mg/ml, i.n.) or saline daily on days 0-1 and challenged with HDM (25 uL of 1 mg/ml) or saline daily from days 14-17 HDM or sterile saline intranasally). 24 hours after the last challenge, bronchoconstriction to inhaled 5-HT (1-100mM, 10ul) was measured in anesthetized, paralyzed, and mechanically ventilated offspring before and after vagotomy. Bronchiolar lavage fluid was collected for inflammatory cell counts and differentials and blood and serum were collected for metabolic parameters.

Results: In offspring, HDM treatment increased total inflammatory cells in bronchoalveolar lavage fluid regardless of maternal diet. However, the total number of BAL inflammatory cells, particularly eosinophils, in the offspring of obese dams treated with HDM was significantly higher than that in the lean offspring (who also received HDM treatment). Bronchoconstriction in response to inhaled 5-HT was increased by maternal obesity in offspring treated with saline and further potentiated by HDM treatment. These antigen treated offspring of obese dams also had significantly increased bronchoconstriction when compared to offspring of lean also treated with antigen. All 5-HT induced bronchoconstriction was blocked by vagotomy indicating it was medicated by airway nerves

Conclusion: Maternal obesity amplifies the offspring's susceptibility to airway hyperresponsiveness and inflammation following allergen exposure. Our findings show that obesity during pregnancy primes airway nerve pathways and heightens eosinophilic inflammation in response to antigen challenge. These results reveal that maternal obesity and allergen exposure act synergistically to increase asthma risk.

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