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 PhD project explores the mechanisms underlying the association between asthma and obesity, following the novel discovery of adipose tissue in the airway wall. Elucidating the structural and functional implications of airway-associated adipose tissue may help us understand its role in asthma development.

Where do you see yourself in 5 years?
I hope to pursue research in respiratory physiology, whether it involves continued exploration of the mechanisms underpinning comorbid asthma-obesity by bringing knowledge developed from basic science into a translational and clinical context, or a side-step into another field within respiratory research. I am hoping to take on an international postdoc position where I can expand my skillset, formulate collaborations and establish myself within a dynamic research team.

What do you find is the major benefit of RSF Assembly Membership?
I am eager to connect with other members of the RSF Assembly, learn about upcoming research within and beyond this niche, and share the latest findings from our research group.
Objective: To explore the genotypic effects of Kiss1/Kiss1r signalling on airway structure-function and determine whether these effects occur in a sex-dependent manner.

Methods: Male and female Kiss1r knockout (KO) and wildtype (WT) mice were studied at 6-weeks of age. Mice underwent glucose tolerance testing to assess for glucose intolerance. These mice were later euthanised for bronchoalveolar lavage to determine inflammatory cell counts and collection of lung and white adipose tissue weights. A separate group of mice were tracheostomised to assess lung mechanics in vivo following methacholine challenge, with lungs subsequently inflation-fixed for airway morphometry.

Results: Male WT were larger in body size than other groups but despite this, mice had comparable body adiposity. Mice did not exhibit glucose intolerance and showed no differences in airway inflammatory cell counts between genotypes or sexes. Airway responsiveness to methacholine was comparable between WT and KO mice. Greater accumulation of airway-associated adipose tissue but no differences in airway smooth muscle layer thickness or total wall area was observed in KO mice when compared with WT mice.

Conclusion: Blockade of Kiss1/Kiss1r signalling leads to airway-associated adipose tissue accumulation irrespective of body adiposity. Airway histology from A) WT and B) KO mice, where black arrows denote airway-associated adipose tissue. C) KO mice had more airway-associated adipose tissue than WT mice. D) White adipose tissue weights were comparable between groups. KO, knockout; WT, wildtype.

Genetic susceptibility to fatty airway remodeling through the Kiss1/Kiss1r signaling pathway

Authors: Carolyn J. Wang, David Lu, Jeremy T. Smith, Peter B. Noble, Kimberley C. W. Wang

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Conclusion: Blockade of Kiss1/Kiss1r signaling in male and female mice leads to the accumulation of airway-associated adipose tissue in the absence of inflammation. Interestingly, this can occur independently of body adiposity. This genetic susceptibility to fatty airway remodeling may contribute to asthma development whereby greater deposition of airway-associated adipose tissue may impact lung function later in life.