Get to know the newest members of the RSF Assembly

Is your research clinical, basic science or translational?
Basic & Translational Science.

Tell us about your research?
My research interests involve the early origin of lung disease and airway remodelling. Using an in utero vitamin D deficiency mouse model, I’m trying to identify the mechanisms linking vitamin D with lung development.

Where do you see yourself in 5 years?
In academia and also teaching.

What do you find is the major benefit of RSF Assembly Membership?
RSF assembly organises a variety of programs to support the development of postgraduates and early career fellows. Being an ATS RSF Member, I have enjoyed excellent networking with the research leaders in our field and collaborations with peers/colleagues.

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If you or someone you know would like to be featured as an ATS RSF ECP please email Jade Jaffar (jade.jaffar@monash.edu)
Vitamin D in early lung development

Introduction: Many studies have shown cross-sectional associations between vitamin D deficiency and chronic lung diseases. However, these associations are confounded by the effect of chronic disease on physical activity levels, which are highly correlated with sun exposure, and hence, vitamin D synthesis. We have strong longitudinal evidence to suggest that vitamin D deficiency has a detrimental impact on lung development. In this study, we aimed to identify the potential mechanisms linking vitamin D with lung development.

Methods: We used an established vitamin D deficient mouse model involving dietary manipulation. Female offspring were euthanized at key developmental timepoints and lung tissue was collected. Lung protein extracts were analysed by LTQOrbitrap tandem mass spectrometry. Label-free quantitation was used to identify the differentially expressed proteins.

Results: 52 differentially expressed proteins were identified in P7 lungs. There was significant interaction between diet and age on the production of pulmonary surfactant associated protein B (SPB) in the lungs, such that SPB was reduced in lungs of P7 vitamin D deficient mice compared to P7 vitamin D replete mice but not in E17.5 mice (A). When mice at E14.5 mice were included, the expression of collagen type 1 alpha 1 (COL1A1) was higher in lungs of vitamin D deficient mice compared to replete mice across these developmental timepoints (B).

Conclusion: The lack of difference in protein expression in the early developmental timepoints suggests that vitamin D deficiency induced alterations in lung structure and function occur during alveolarization and are driven by altered surfactant and collagen synthesis. These data provided a plausible mechanism linking maternal vitamin D deficiency with altered postnatal lung function.