ATS 2016 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know the newest members of the RSF Assembly

Ling Chen, PhD

Postdoctoral Research Fellow
Univ. of Tasmania, Australia

Contact Ling Chen

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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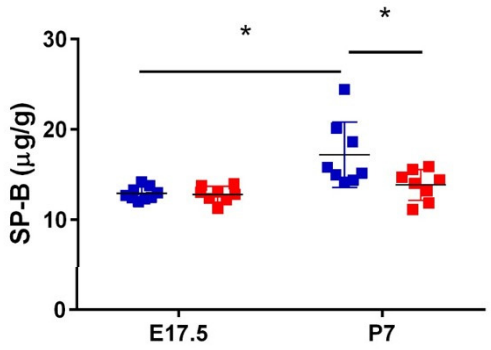

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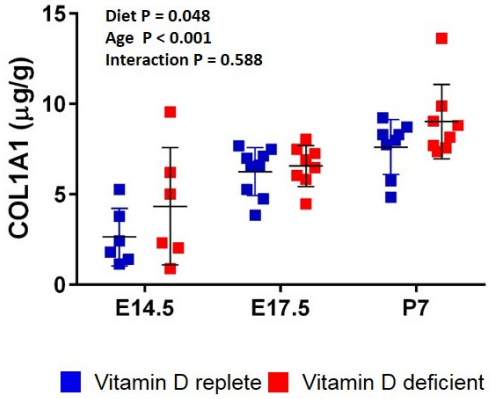

Vitamin D in early lung development

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.

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