

# ATS 2019 Highlights

## Respiratory Structure and Function Early Career Professionals



### *Get to know members of the RSF Assembly*

*Is your research clinical, basic science or translational?*

Basic science.

*Tell us about your research?*

My current area of research is identifying fetal origins of asthma, specifically the impact of intrauterine growth restriction on asthma development. Rather than the conventional viewpoint that asthma is triggered by the exposure to environmental triggers throughout postnatal life, asthmatic patients may have altered airway structure and function since birth.

*Where do you see yourself in 5 years?*

Postdoc in academia.

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

It would be to be able to personally meet some respiratory scientists whom papers that I've been reading. It is good to be able to put a face to a name and to introduce my work to them!

### **Kimberley Wang, PhD**

*NHMRC Early Career Research Fellow*

*School of Human Sciences*

*University of Western Australia*

<https://research-repository.uwa.edu.au/en/persons/kimberley-wang>

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*If you or someone you know would like to be featured as an ATS RSF ECP please email Katrina Tonga ([katrina.tonga@sydney.edu.au](mailto:katrina.tonga@sydney.edu.au))*



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## Respiratory Structure and Function Early Career Professionals

### Kimberley Wang, PhD

NHMRC Early Career Research Fellow

School of Human Sciences

University of Western Australia

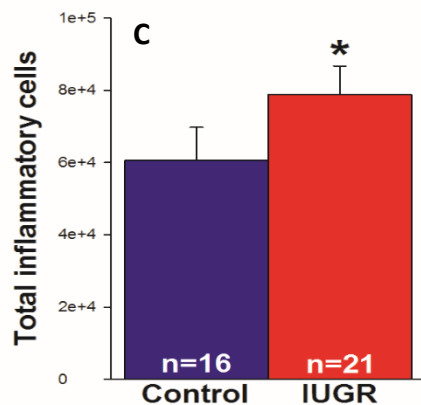
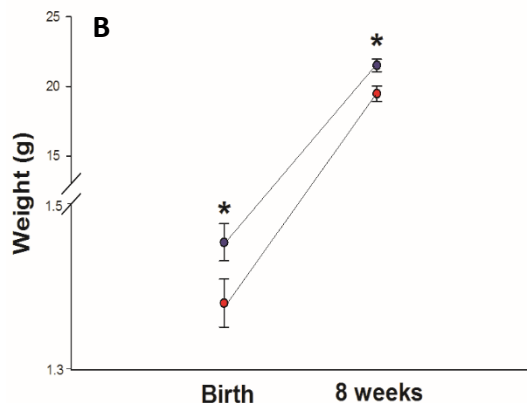
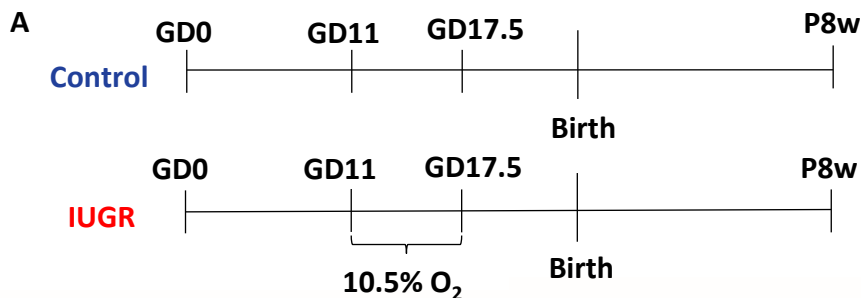
### Intrauterine growth restriction mediates an inflammatory phenotype in offspring but does not affect airway epithelial tight junction expression

**Objective:** Intrauterine growth restriction (IUGR) is associated with an increased risk of developing asthma in later life. Susceptibility to disease could be explained by abnormal airway epithelial barrier properties and/or a shift to an inflammatory phenotype. This study used a maternal hypoxia-induced mouse model of IUGR to assess sexual dimorphism in the response to IUGR on bronchoalveolarlavage fluid (BALF) inflammatory cell counts and epithelial tight junctions (TJs) protein expression in male and female mice offspring.

**Methods:** Pregnant female BALB/c mice were housed under hypoxic conditions (10.5% O<sub>2</sub>) from gestational day (GD) 11 – GD17.5 (IUGR group; term, GD21; Figure A). Following hypoxic exposure, mice were returned to a normoxic environment (21% O<sub>2</sub>; Figure A). A second group of pregnant mice were housed under normoxic conditions throughout pregnancy (Control). Weights of offspring were recorded until 8 weeks of age at which point they were euthanized, BALF collected and total and differential cell counts performed. From a separate set of mice, right lungs were snap frozen for Western blotting (claudin-1, claudin-18, occludin and zonula occludens (ZO)-1).

**Results:** The IUGR offspring were lighter at birth (Control, n=57; IUGR, n=49;  $P=0.047$ ; Figure B) and remained lighter at 8 weeks of age compared with Controls (Control male, n=11; IUGR male, n=12, Control female, n=12, IUGR female, n=12;  $P<0.001$ ; Figure B). The IUGR offspring had higher total inflammatory cells in the BALF ( $P=0.004$ ; Figure C), but there were no differences in the differential counts of macrophages, neutrophils, lymphocytes or eosinophils. The protein expression for claudin-1 ( $P=0.344$ ), claudin-18 ( $P=0.898$ ), occludin ( $P=0.640$ ) and ZO-1 ( $P=0.760$ ) were not altered by IUGR. There were also no differences between sexes in the BALF inflammatory cells or TJs protein expression.

**Conclusion:** Maternal hypoxia-induced IUGR increases inflammatory cell counts and some sex-specific changes in the BALF of IUGR offspring but does not affect epithelial TJs protein expression. Increased inflammation in adulthood indicates a phenotypic change that has a fetal origin and warrants further investigation.



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