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 is centered around understanding how oxidized lipids, caused by oxidative stress, contribute to airway inflammation and remodeling in asthma. Our goal is to show that oxidized lipids contribute to the pathogenesis of asthma and can be targeted by novel therapeutics for the treatment of asthma and other chronic lung diseases.

Where do you see yourself in 5 years?
In 5 years I see myself in academia leading a lab researching the role of oxidized compounds in various chronic conditions.

What do you find is the major benefit of RSF Assembly Membership?
The main advantage of being an RSF member for me is the ability to network and collaborate directly with many influential physiologists and exceptional researchers.

Chris D. Pascoe, PhD
Postdoctoral Fellow
Univ. Of Manitoba/Children’s Research Inst. Of Manitoba

More info on Chris’s research

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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)
Oxidized phospholipids in Asthma

**Background:** Increased oxidative stress and decreased anti-oxidant capacities have been associated with severe exacerbations in asthma. This oxidative burden can damage the lipid lining of the lung by producing oxidized lipids including oxidized phosphatidylcholine (OxPC). We have identified an abundance of OxPC in the lungs of mice challenged with house dust mite and in the lungs of asthmatics challenged with allergen. Now our research aims to understand the role of these compounds in lung disease.

**Hypothesis:** OxPC induce an inflammatory phenotype in human airway smooth muscle (ASM).

**Results:** Incubating primary human ASM with OxPC causes a dose dependent increase in cyclooxygenase-2 (COX-2) mRNA abundance and secretion of pro-inflammatory cytokines IL-6, IL-8, and GM-CSF. This increase is significantly greater than that produced by the negative control (PSPC) at OxPC concentrations over 40 ug/mL (Figure). This increase in COX-2 expression and pro-inflammatory cytokine secretion appears to be mediated by TLR4 signaling (as measured by IkB degradation).

**Conclusion:** OxPC is a bioactive compound produced during oxidative stress that can induce a pro-inflammatory phenotype in ASM, possibly through TLR4 signaling. Targeting their generation or action may provide a novel way to treat severe asthma.