ATS 2016 Highlights

Respiratory Structure and Function Early Career Professionals

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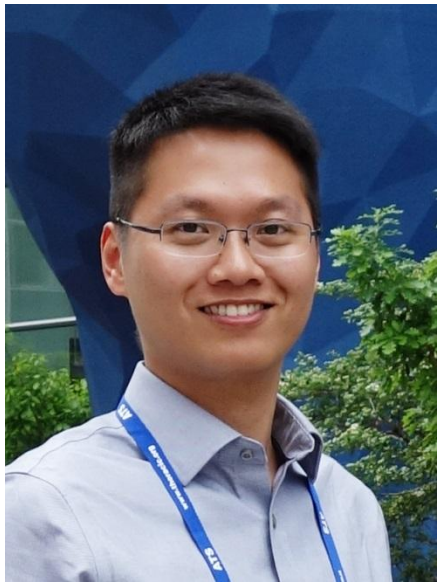

Jun Chen, PhD

Postdoctoral Research Associate
University of Mass. Medical School
More info on Jun's research

Is your research clinical, basic science or translational?

Basic /translational Science

Tell us about your research?

To understand the alterations in airway smooth muscle cell (ASMC) physiology that results in excessive airway contraction and airway hyperresponsiveness associated with asthma by using a precision cut lung slices preparation
to characterize the mechanisms of Ca^{2+} signalling and Ca^{2+} sensitivity that control the contraction and relaxation of ASMC in airways.

Where do you see yourself in 5 years?

Academia and teaching

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

Being an RSF Assembly member helps me to advance my career by networking with excellent researchers in my field and lets more people know about my work, which makes the establishment of collaborations much easier.

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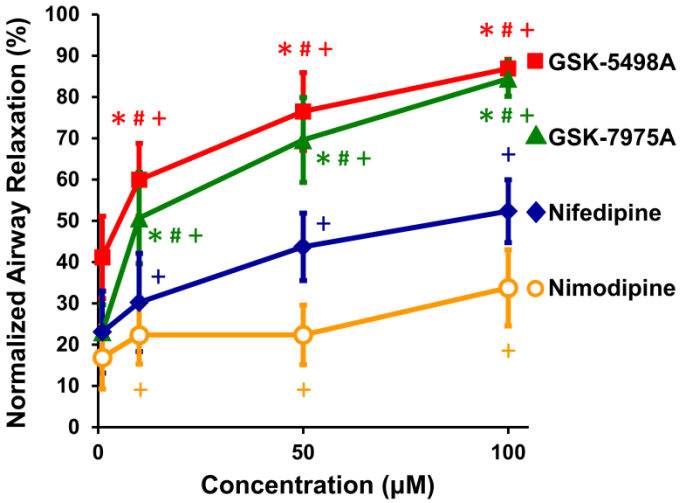

Figure 1. Methacholine-induced airway contraction is relaxed by calcium channel inhibitors

Calcium signaling and airway hyper-responsiveness

Background: Airway hyper-responsiveness in asthma is driven by excessive contraction of airway smooth muscle cells (ASMCs). Agonist-induced Ca^{2+} oscillations underlie this contraction of ASMCs and the magnitude of this contraction is proportional to the Ca^{2+} oscillation frequency. Sustained contraction and Ca^{2+} oscillations require an influx of extracellular Ca^{2+} but the mechanisms and pathway mediating this Ca^{2+} influx during agonist-induced ASMC contraction are not well defined.
Hypothesis: Store-operated calcium entry (SOCE) is the major pathway for the Ca^{2+} influx in agonist-induced airway contraction and Ca^{2+} oscillations within ASMCs.
Results: SOCE inhibitors, GSK7975A and GSK5498A, were able to fully relax methacholine (MCh)-induced airway contraction (Figure 1) by abolishing the Ca^{2+} oscillations, in a manner similar to that observed in zero extracellular Ca^{2+} ($[Ca^{2+}]_e$). In addition, GSK7975A and GSK5498A inhibited increases in intracellular Ca^{2+} ($[Ca^{2+}]_i$) in ASMCs with depleted Ca^{2+}-stores in response to increased $[Ca^{2+}]_e$, a response consistent with an inhibition of SOCE. By contrast, L-type voltage-gated Ca^{2+} channel (VGCC) inhibitors, nifedipine and nimodipine, only partially reduced airway contraction, Ca^{2+} oscillation frequency and SOCE-mediated Ca^{2+} influx.
Conclusion: These data implicate that SOCE is the major Ca^{2+} influx pathway for ASMCs to sustain agonist-induced airway contraction and the underlying Ca^{2+} oscillations. The mechanisms of SOCE may therefore form novel targets for new bronchodilators.

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