ATS 2016 Highlights
Respiratory Structure and Function Early Career Professionals

Get to know the newest members of the RSF Assembly

Bo Lan, PhD

Postdoctoral Research Fellow
Harvard T.H. Chan School of Public Health

Is your research clinical, basic science or translational?

Basic and translational science.

Tell us about your research?

I am interested in airway smooth muscle and airway narrowing in asthma. Specifically, my research focuses on airway smooth muscle mechanics, structure and the interaction between airway smooth muscle and airway epithelial cells. These studies may provide novel targets for asthma therapy.

Where do you see yourself in 5 years?

I could see myself staying in academia and maybe get involved with teaching.

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

Networking within the RSF Assembly helps me stay connected with all the leading researchers in my field. I have enjoyed wonderful friendships and research collaborations.

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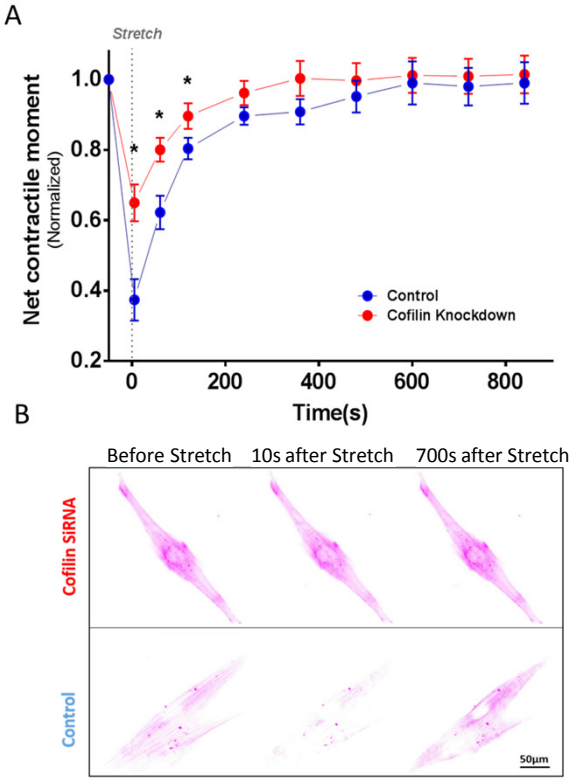

Deep inspiration, fluidization and actin binding protein cofilin

Background: Healthy subjects can reverse bronchoconstriction with a simple Deep Inspiration (DI) whereas this beneficial response is impaired in asthmatics. Bronchodilation response to a DI can be partially explained by stretch-induced cytoskeletal fluidization, in which the airway smooth muscle cytoskeleton undergoes a rapid transition from a solid-like contracted phase to a fluid-like relaxed phase. However, the molecular mechanism underlying this process, and the loss of this beneficial response to a DI in asthma, remain unclear.

Hypothesis: Actin filaments disassembly regulated by cofilin, is the key to fluidization response and DI induced bronchodilation.

Results: In control human airway smooth muscle (HASM) cells studied in isolation in culture , transient stretch (8%) simulating a DI caused a prompt ablation of contractile forces by 64±6% as assessed by traction microscopy. When cofilin was knocked down using SiRNA, by contrast, the resulting ablation of contractile force was far smaller ($p < 0.01$), only 29±4% (Fig A). Actin filaments disassembly induced by transient stretch is significantly less in cofilin knockdown group comparing to control (Fig B).

Conclusion: These findings identify a molecular mechanism by which a DI dilates the constricted airway, and suggests that cofilin may provide a novel target for asthma therapy.

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