ATS Highlights 2024:
Critical Care Assembly Early Career Professionals

Colin E. Evans, PhD

Assistant Professor

Cardiovascular Translational Research Center, Department of Cell Biology and Anatomy, University of South Carolina School of Medicine, USA

Tell us about yourself. I am a Vascular Biologist and Early-Stage Principal Investigator interested in inflammatory vascular diseases. Our research is currently funded by the University of South Carolina School of Medicine, the American Heart Association, and the National Institute of General Medical Sciences. I have obtained grant and travel support from >15 sources and published >60 papers.

Is your research clinical, basic science, or translational? Basic and Translational. We perform studies of fundamental disease processes that can be targeted using novel treatment strategies.

Tell us about your research. We study the vascular response to thrombosis with a view to improving treatments for inflammatory vascular diseases. Using clinically relevant disease models and targeted gene editing and drug delivery techniques, we focus on two discrete but related disease areas: (i) thrombus formation and resolution; and (ii) inflammatory lung injury and repair.

Where do you see yourself in 5 years? As a tenured Associate Professor leading an established and productive biomedical research team with a growing reputation in inflammatory vascular research.

How has the Critical Care Assembly contributed to your career? Through the Critical Care Assembly, I have improved my network of near-peers and participated in career development sessions.

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Profile: sc.edu/study/colleges_schools/medicine/about_the_school/faculty-staff/colin_evans.php

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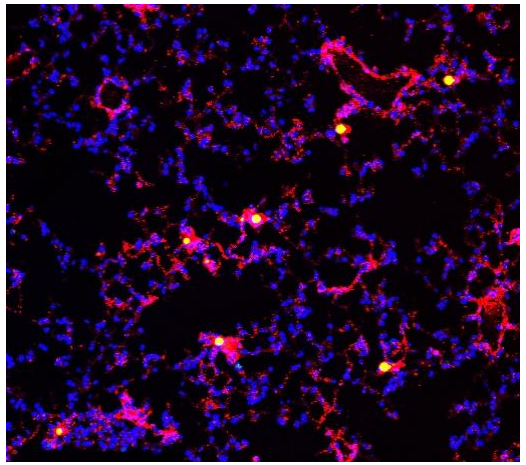

Inflammatory lung showing blood vessels (red) with thrombotic capillary micro-occlusions (yellow)

Developmental Research Project funded by SC INBRE / NIGMS (2024-2026):
Endothelial Survival and Proliferation in Inflammatory Lung Injury and Repair

Rationale: There is a lack of effective treatments for sepsis-induced acute lung injury (ALI) and acute respiratory distress syndrome (ARDS). As a result, mortality rates remain as high as 40% in ARDS patients. Novel and effective treatments for ALI/ARDS could arise from a better understanding of the mechanisms that control ALI and subsequent vascular repair. Lung endothelial cell (EC) survival is reduced in ALI, while the vascular repair process following ALI relies upon lung EC proliferation.

Results: Our data show that an innocuous level of capillary occlusion reduces ALI via EC expression of phospholipase A2 group IID (Pla2g2d). Our supporting data also show that this innocuous level of lung capillary occlusion increases vascular repair following ALI and enhances the pulmonary expression of the key mitochondrial complex III subunit, ubiquinol-cytochrome C reductase complex III subunit VII (Uqcrq), while post-ALI vascular repair is impaired by EC-specific Uqcrq deletion.

Hypothesis and Methods: We hypothesize that EC Pla2g2d promotes lung EC survival to inhibit ALI and that EC Uqcrq promotes lung EC proliferation to enhance vascular repair after ALI. In this work, we will define and target EC Pla2g2d- and Uqcrq-dependent signaling to promote EC survival and proliferation, thereby inhibiting ALI and accelerating vascular repair. We will test the efficacy of a dual therapy that simultaneously promotes lung EC survival and proliferation. We will verify the mechanisms of PLA2G2D- and UQCRQ-dependent signaling in human lung ECs.

Conclusions: Our studies will reveal and promote novel EC survival and proliferation pathways that inhibit inflammatory lung injury. These studies could reveal a therapeutic strategy for ALI/ARDS.

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