ATS Highlights 2025:
Critical Care Assembly Early Career Professionals

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Tell us about yourself. I am a PCCM physician scientist at the University of Pittsburgh. I completed my residency and fellowship training at the University of Colorado and additional research training at Massachusetts General Hospital before joining the faculty at Pitt in 2024 to establish my own lab!

Tell us about your research. I do both basic and translational research on the impact of glycocalyx degradation on the pathogenesis of ARDS and sepsis. My work includes mouse models of glycocalyx degradation to define underlying mechanisms and observational human subjects research to study the impact of glycocalyx degradation on the physiology of critical illness and patient outcomes.

Where do you see yourself in 5 years? I am excited to begin to build my research team over the coming years. My laboratory's primary goal is to develop glycan-targeted precision medicine strategies for critical illnesses such as ARDS and sepsis. If you are interested in joining the lab or collaborating with us reach out!!

How has the Critical Care Assembly contributed to your career? I have benefitted greatly from the CCA mentoring program, which helps trainees and early career faculty to establish connections with senior CCM researchers from across the country. I would recommend all to apply!

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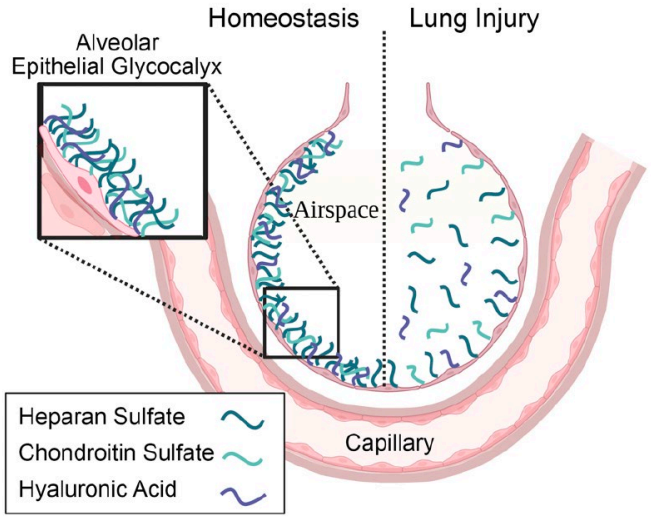

Alveolar epithelial glycocalyx degradation mediates surfactant dysfunction and contributes to acute respiratory distress syndrome

Rationale: The alveolar epithelial glycocalyx is a layer of glycosaminoglycans (GAGs) that lines the airways and is degraded in murine models of lung injury. We sought to determine whether glycocalyx degradation contributes to ARDS and study the physiologic function of this structure in mice.

Methods: We performed mass spectrometry on airspace fluid noninvasively collected from mechanically ventilated patients. We also utilized mouse model of epithelial glycocalyx degradation with intratracheal injection of heparanases I/III.

Results: We found that airspace GAG shedding (an index of glycocalyx degradation) occurred predominantly in patients with direct lung injury and was associated with duration of mechanical ventilation. Male patients had increased shedding, which correlated with airspace concentrations of matrix metalloproteinases. Selective epithelial glycocalyx degradation in mice was sufficient to induce surfactant dysfunction, a key characteristic of ARDS, leading to microatelectasis and decreased lung compliance.

Conclusions: Epithelial glycocalyx degradation occurs in a subset of patients with ARDS and is associated with worse outcomes. These effects may be mediated by surfactant dysfunction. Future work is needed to develop pharmacologic strategies to attenuate glycocalyx degradation as well as define the subgroup of patients who may benefit from these therapies.

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