ATS 2025 Highlights

Respiratory Structure and Function Early Career Professionals

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Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?

Translational.

Tell us about your research?

I am interested in lung injury that affects the neonate and their long-term outcomes. I am studying molecular mechanisms that play critical roles in acute lung injury and investigating their potential utility to improve healthcare outcomes.

Where do you see yourself in 5 years?

Develop into an independent scientist with expertise in acute lung injury.

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

Networking and learning from all the amazing scientists!

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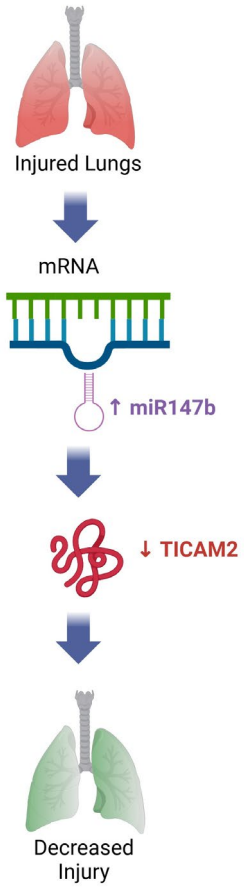

miR-147b critical to downregulate TICAM2 gene expression to alleviate lung injury

Alveolar Epithelial Derived miR-147b Provides Lung Protection During Lung Injury Through Repression of TICAM2

Objective: Investigate functionally critical microRNA (miRNA) induction during ventilator induced lung injury (VILI) can modulate acute lung injury (ALI) by repressing target genes and explore their potential as therapeutic agents.

Methods: Wild type C57BL/6 mouse (WT) and transgenic mouse lines including constitutive knockout (KO) of miR147b (miR147loxp/loxp), inducible alveolar epithelial type II cells (ATII)-specific CRE expressor (SPCCreER), inducible ATII-specific KO of miR147b (miR147loxp/loxp SPCCreER), and mutated 3' UTR of TICAM2 to prevent binding of miR-147b (TICAM2/miR147 loxp/loxp) were ventilated for 3 hours with high pressures. To study miR-147b role, WT mice were injected with miR-147b mimic 72 hours prior to VILI to increase miR-147b levels. Markers for lung injury were then analyzed.

Results: We identified miR-147b to have a selective role during pulmonary insult. MiR147loxp/loxp mice had significantly increased BALF albumin, CXCL1 mRNA, MPO and lung injury score compared to WT. miR147loxp/loxp SPCCreER had significantly elevated albumin, IL-6, CXCL1 and lung injury score as compared to SPCCreER controls while WT mice treated with miR-147b mimics had significantly decreased inflammatory cytokines and lung injury score. Overexpression of miR-147b in Calu 3 cells identified TICAM2 gene as the sole binding target for miR-147b. TICAM2/miR147 loxp/loxp mice under VILI had significantly increased BALF albumin and IL1β compared to WT.

Conclusion: miR-147b is critical for mitigating alveolar inflammation in mice exposed to VILI through downregulation of TICAM2 gene expression. Deletion of miR-147b led to significantly increased pulmonary edema and inflammation while increased levels of miR-147b significantly reduced lung injury. Loss of TICAM2 regulation by miR-147b significantly exacerbated lung injury. Our results suggest that targeting this pathway could provide protective effects against lung injury..

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