

ATS 2026 Highlights

Respiratory Structure and Function Early Career Professionals



Vijaya Kumar Gangipangi, PhD

(He/Him)

Postdoctoral Fellow

Department of Pathology

Temple University, Philadelphia

Vijaya.kumar.Gangipangi@temple.edu/www.linkedin.com/in/dr-vijaya-kumar-gangipangi-9a62b111b

Get to know members of the RSF Assembly

Is your research clinical, basic science or translational?

Translational.

Tell us about your research?

My research aims to uncover the molecular mechanisms that drive asthmatic airway remodeling. I study how ubiquitin-mediated protein regulation with a particular focus on the E3 ubiquitin ligase RNF145. By integrating cellular, molecular, and ex vivo lung models, my goal is to identify novel therapeutic targets that can ultimately improve the treatment of chronic lung diseases.

Where do you see yourself in 5 years?

I hope to become an independent investigator leading a research program on the molecular basis of lung diseases and the development of innovative therapeutic strategies. I hope to build a collaborative research team, obtain competitive research funding, mentor trainees, and contribute to discoveries that advance respiratory medicine and improve patient care.

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

The RSF Assembly provides excellent opportunities for networking, and mentorship. As an early-career investigator, it allows me to learn from experts, explore new research ideas through valuable scientific discussions, and build collaborations that strengthen my research and professional growth.



Please follow us on LinkedIn



and Bluesky



If you or someone you know would like to be featured as an ATS RSF ECP please email Carolyn Wang (carolyn.wang@hli.ubc.ca)

ATS 2026 Highlights

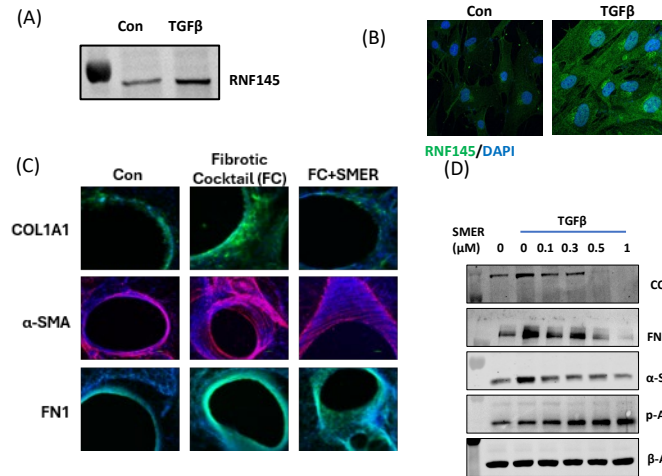
Respiratory Structure and Function Early Career Professionals

Vijaya Kumar Gangipangi, PhD
(He/Him)

Postdoctoral Fellow

Department of Pathology

Temple University, Philadelphia



TGF-β1 induces RNF145 expression and promotes profibrotic response. (A, B) Immunoblotting and immunofluorescence analyses showing increased RNF145 expression following TGF-β1 stimulation in human ASM cells. (C) Immunofluorescence analysis showing that SMER reduces fibrotic cocktail-induced ECM deposition in mouse PCLS. (D) Immunoblotting showing that SMER attenuates TGF-β1-induced ECM protein expression in human ASM cells.

E3 Ligase RNF145 Modulates AMPK-Dependent Fibrotic Signaling and Remodeling in Airway Smooth Muscle Cells

Objective: To determine the role of the E3 ubiquitin ligase RNF145 in regulating AMPK signaling, extracellular matrix production, and airway smooth muscle remodeling during fibrosis.

Methods: RNF145 expression in human airway tissue was determined by immunohistochemical analysis. Primary human ASM cells were subjected to lentiviral RNF145 overexpression, shRNA-mediated knockdown, or pharmacological inhibition using SMER3, followed by TGFβ1 (2.5 ng/mL) stimulation. Cell migration, fibrotic signaling, and ECM expression were assessed by wound-healing assays and immunoblotting, respectively. Mouse precision-cut lung slices (PCLS) were exposed to a fibrotic cocktail (TGFβ1, 5 ng/mL; TNFα, 10 ng/mL; PDGF-AB, 10 ng/mL; and LPA, 5 μM) with or without SMER3 for 72 h, and ECM deposition was assessed.

Results: Immunohistochemistry of airway tissues from asthmatic donors showed significantly increased RNF145 expression in ASM bundles compared to non-asthmatic controls. In human ASM cells, TGFβ1 stimulation induced a robust, time-dependent increase in RNF145, collagen, and fibronectin expression. RNF145 expression positively correlated with ECM protein accumulation. Both RNF145 knockdown and SMER3 treatment suppressed TGFβ1-induced cell migration and reduced collagen, fibronectin, and α-smooth muscle actin expression in a dose-dependent manner. In mouse PCLS, SMER3 co-treatment attenuated fibrotic cocktail-induced ECM accumulation. Mechanistically, RNF145 overexpression and TGFβ1 stimulation reduced phospho-AMPK levels. Conversely, RNF145 knockdown or inhibition increased AMPK phosphorylation, suggesting that RNF145 represses AMPK-dependent antifibrotic signaling.

Conclusion: RNF145 functions as a negative regulator of AMPK-mediated antifibrotic signaling in ASM cells. Pharmacological inhibition of RNF145 with SMER3 restores AMPK activation, suppresses ECM production, and mitigates fibrotic remodelling. These findings identify RNF145 as a novel modulator of ASM fibrosis and a potential therapeutic target for airway remodelling in obstructive airway diseases.