ATS 2020 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Gregoire Ruffenach, PhD

Post-doctoral fellow
UCLA, David Geffen School of Medicine,
Department of Anesthesiology,
Division of Molecular Medicine

[Greg's Linkedin]

Is your research clinical, basic science or translational?

Translational

Tell us about your research?

My research focus on the molecular mechanism underlying the development of pulmonary hypertension as a primary disease or secondary to interstitial lung disease. These investigations combining patient's data and pre-clinical models aim to further our understanding of these life-threatening diseases and to lead to the identification of new suitable therapeutic target.

Where do you see yourself in 5 years?

I hope to continue my research in academia by leading my own team and training the next generation of scientist.

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

The RSF assembly is an incredible gathering of the world leader and bright young investigator that creates intellectual stimulation and great carrier opportunities.

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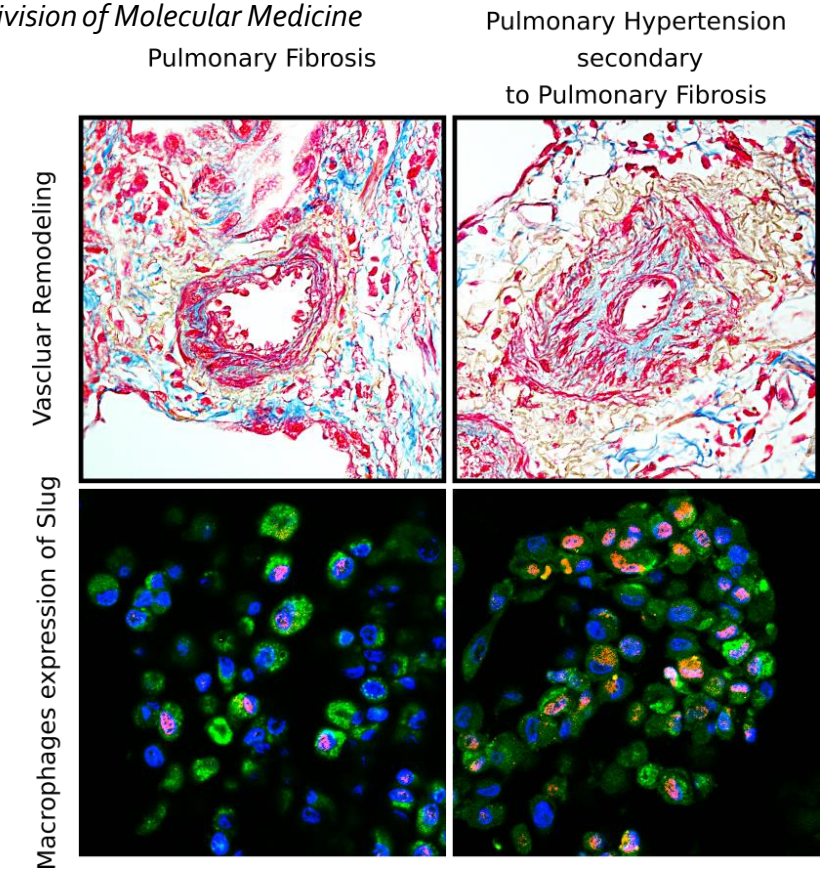

Histological hallmarks and role of Slug/PIP axis in pulmonary hypertension secondary to pulmonary fibrosis.

Objective:
Pulmonary hypertension secondary to pulmonary fibrosis (PF-PH) is one of the most common causes of PH, and there is no approved therapy. PH arise from thickening of distal pulmonary vascular walls as a result of uncontrolled proliferation of vascular wall cells. The molecular signature of PF-PH and underlying mechanism of why PH develops in PF patients remains understudied and poorly understood.

Methods:
Using human and a pre-clinical model of PF-PH, our team investigated vascular wall thickness in fibrotic and non-fibrotic area in PF and PF-PH. We also measured the expression of the transcription factor Slug and its target the prolactin-induced protein.

Results:
We observed significantly increased vascular wall thickness in both fibrotic and non-fibrotic areas of PF-PH patient lungs compared to PF patients. The increased vascular wall thickness in PF-PH patients is concomitant with a significantly increased expression of the transcription factor Slug within the macrophages and its target prolactin-induced protein, an extracellular matrix protein that induces pulmonary arterial smooth muscle cell proliferation. Furthermore, Slug inhibition, by i intra-tracheal nebulization of an Si-RNA, decreases PH severity in a pre-clinical model of PF-PH.

Conclusion:
Our study provides new insight on the histo-pathological and molecular characteristics of PF-PH and identify Slug has a promising therapeutic target. [PMID:31468711]

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