ATS 2023 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

Dr. Emmanuel T. Osei, MSc. Ph.D.

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Is your research clinical, basic science or translational?

Basic Science

Tell us about your research?

My research focuses on understanding the role of defective cell-cell & cell-ECM crosstalk in lung diseases such as asthma and COPD to uncover possible therapeutic targets. To do this, I establish a variety of bioartificial or biomimetic models ranging from systems such as 3D transwell co-culture models & cell-embedded hydrogels to more recently, lung organoids, lung-on-chips & 3D bioprinted systems.

Where do you see yourself in 5 years?

As a 2nd year Early Career Researcher & Principal Investigator with my independent research group & program, I have strong ambitions to bring in more competitive funds to strengthen my research & further establish a truly intentionally collaborative program in Lung biomimetic models & multicellular-ECM crosstalk studies. I envision this will aid me to obtain tenure in the next 5-yrs to secure the academic freedom I need to do even more cutting edge research.

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

The immense benefit from the RSF has been the exposure to ATS programming and connections to my peers from all over the world. Through our regular meetings, my network has further developed and I foresee that these extraordinary connections can only improve my career for many years to come.

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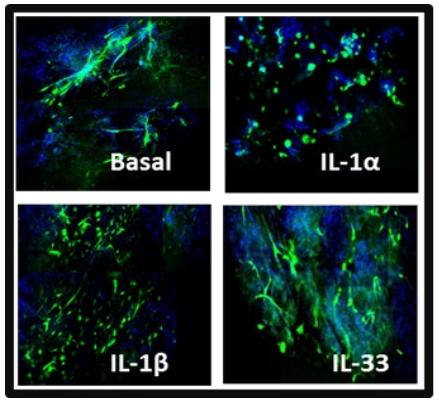

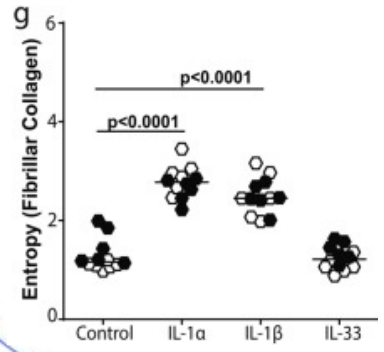

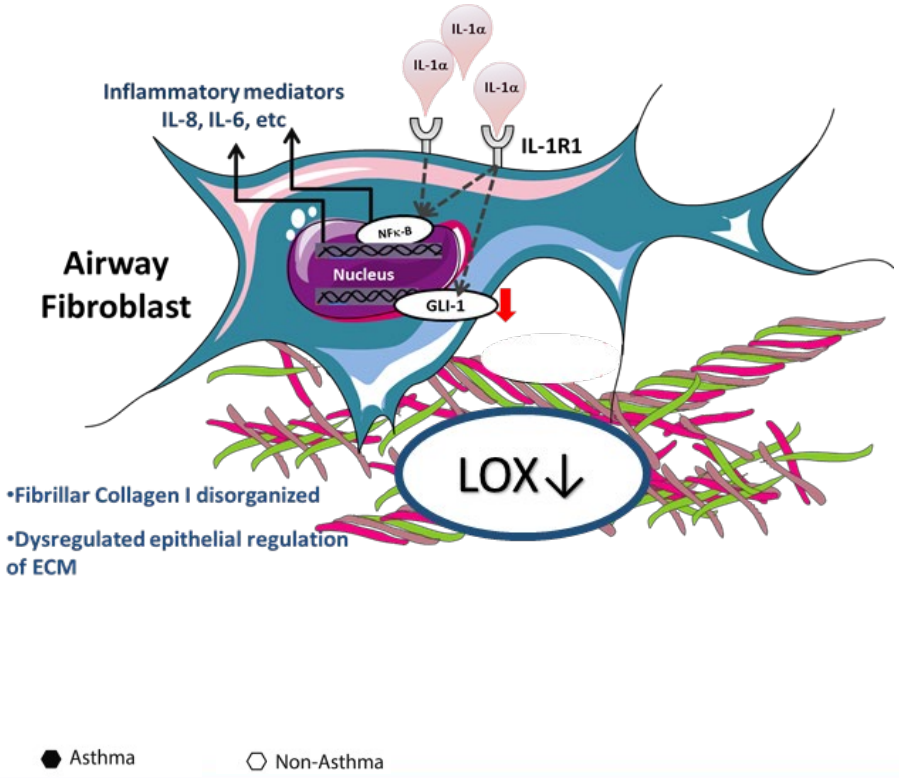

Abstract Title: Epithelial-interleukin-1 inhibits collagen formation by airway fibroblasts: Implications for asthma (PMID: 32457454)

Objective: In asthma, the airway epithelium has an impaired capacity to differentiate and plays a key role in the development of airway inflammation and remodeling through mediator release. The study objective was to investigate the release of (IL)-1 family members from primary airway epithelial-cells during differentiation, and how they affect primary airway fibroblast (PAF)-induced inflammation, extracellular matrix (ECM) production, and collagen I remodeling.
Methods: The release of IL-1α/β and IL-33 during airway epithelial differentiation was assessed over 20-days using air-liquid interface cultures. The effect of IL-1 family cytokines on airway fibroblasts grown on collagen-coated well-plates and 3-dimensional collagen gels was assessed by measurement of inflammatory mediators and ECM proteins by ELISA and western blot, as well as collagen fiber formation using non-linear optical microscopy after 24-hours.
Results: The production of IL-1α is elevated in undifferentiated asthmatic-PAECs compared to controls. IL-1α/β induced fibroblast pro-inflammatory responses (CXCL8/IL-8, IL-6, TSLP, GM-CSF) and suppressed ECM-production (collagen, fibronectin, periostin) and the cell's ability to repair and remodel fibrillar collagen I via LOX, LOXL1 and LOXL2 activity, as confirmed by inhibition with β-aminopropionitrile.
Conclusion: These data support a role for epithelial-derived-IL-1 in the dysregulated repair of the asthmatic-EMTU and provides new insights into the contribution of airway fibroblasts in inflammation and airway remodeling in asthma.

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