David Rowlands, PhD

Co-Mentor: Ellie Growcott, PhD

Respiratory Diseases

Cambridge, Massachusetts, United States

Current therapies for chronic respiratory diseases such as chronic obstructive pulmonary disease (COPD) and idiopathic pulmonary fibrosis (IPF) are largely palliative, providing short-term relief of symptoms and improvement in quality of life. With an aging population, increased chronic tobacco use, and a decline in air quality, COPD is projected to become the third leading cause of death by 2030.

Within the Respiratory Diseases group at NIBR, our goals are to develop transformative therapies that halt and reverse the underlying disease mechanisms of 1) increased mucus burden resulting in air flow obstruction, and 2) impaired mucociliary clearance leading to increased risk of respiratory infection, and to develop therapies aimed at regenerating the alveolus in emphysema and IPF. In chronic lung diseases, disease mechanisms including those listed above may present barriers to the ability of inhaled therapeutics to reach their intended site of action within the lung. A goal of the group is to characterize physical barriers that may limit target engagement of inhaled therapeutics and leverage these data in devising novel approaches to overcome such barriers and maximize clinical efficacy of new inhaled therapeutics for chronic lung diseases. Central to these goals, our group uses novel in vitro, in vivo, and ex vivo lung preparations and molecular profiling of patient material to identify and validate central pathways responsible for airway epithelial cell dysfunction, in addition to developing new approaches for inhaled drug delivery.

Selected Publications

MicroRNA-140-5p and SMURF1 regulate pulmonary arterial hypertension.
Rothman AM, Arnold ND, Pickworth JA, Iremonger J, Ciuclan L, Allen RM, Guth-Gundel S, Southwood M, Morrell NW, Thomas M, Francis SE, Rowlands DJ, Lawrie A.
J Clin Invest. 2016 Jul 1;126(7):2495-508.

Mitochondrial transfer from bone-marrow derived stromal cells to pulmonary alveoli protects against acute lung injury.
Islam NM, Das SR, Emin MT, Wei M, Sun L, Westphalen K, Rowlands DJ, Quadri SK, Bhattacharya S, Bhattacharya, J.
Nat Med. 2012 Apr 15; 18(5):759-65.

Activation of TNFR1 ectodomain shedding by mitochondrial Ca2+ determines the severity of lung inflammation in mouse lung microvessels.
Rowlands DJ, Islam MN, Das S, Huertas A, Quadri SK, Horiuchi K, Inamdar N, Emin MT, Lindert J, Ten VS, Bhattacharya S, Bhattacharya J.
J Clin Invest. 2011 May; 121(5):1986-99.

Click here for additional publications.

Source URL: *https://prod1.novartis.com/careers/career-programs/postdoc-program/postdoc-research-themes/pathways-biology-postdoc-mentors/david-rowlands-phd*

List of links present in page

1. https://prod1.novartis.com/careers/career-programs/postdoc-program/postdoc-research-themes/pathways-biology-postdoc-mentors/david-rowlands-phd
2. https://pubmed.ncbi.nlm.nih.gov/27214554/
3. https://pubmed.ncbi.nlm.nih.gov/22504485/
4. https://pubmed.ncbi.nlm.nih.gov/21519143/
5. https://pubmed.ncbi.nlm.nih.gov/?term=%28Rowlands%20DJ%5BAuthor%5D%29%20%28vascular%20OR%20lung%29