BIO

Jason H.T. Bates is a respiratory biomedical engineer and physiologist working predominately in the field of lung mechanics with a focus on the biophysical mechanisms of airways responsiveness and the pathophysiology of ventilator-induced lung injury. Dr. Bates spent the earlier part of his career until 1999 in the Meakins-Christie Laboratories of McGill University. He is currently a Professor of Medicine, Molecular Physiology & Biophysics, and Electrical & Biomedical Engineering at the University of Vermont. From 2010-14 he also served as Interim Director of the University of Vermont School of Engineering. Dr. Bates has co-authored more than 290 peer-reviewed scientific papers as well as numerous other articles. In 2009 he authored a book published by Cambridge University Press entitled “Lung Mechanics. An Inverse Modeling Approach”. Dr. Bates is Deputy Editor for the Journal of Applied Physiology, a Fellow of the American Institute for Medical & Biological Engineering, a Fellow of the Biomedical Engineering Society, a Fellow of the American Thoracic Society, and a Senior member of the Institute of Electrical & Electronic Engineers.

ABSTRACT

Asthma is a syndrome that is diagnosed on the basis of symptoms that represent the common clinical phenotype of a number of different disease processes. Key among these symptoms is airways hyperresponsiveness whereby the mechanical behavior of the lungs become excessively deranged following inhalation of a standard dose of smooth muscle agonist (usually methacholine). The pathogenesis of airways hyperresponsiveness is still controversial because it can potentially be explained by a number of disparate mechanisms including excessive force generation and shortening of the airway smooth muscle, inflammatory thickening of the airway wall, excessive secretions that occlude the airway lumen, and altered transport of the inhaled agonist to the smooth muscle. All these factors involve physical processes that can be described mathematically. Computational models fit to measurements of lung function in both animal models of asthma and human asthmatic patients will be discussed. These models manifest over a range of length scale from isolated smooth muscle tissue up to the entire airway tree.