



P A R K E R   B .   F R A N C I S  
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P R O G R A M

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## Class of 2007

The Parker B. Francis Fellowship provides funding to outstanding young lung researchers in the US, Canada and Mexico. Since its inception in 1976, the PBF Fellowship program has supported the work of more than 700 fellows in the fields of lung biology and respiratory disease. These fellows have made remarkable discoveries working in laboratories and clinics in North America and throughout the world. They have published their work in the best-known scientific and medical journals, and their research has improved patient care and identified preventive strategies to decrease the incidence of pulmonary disease, disability and death. Many fill leadership positions in prominent professional organizations in throughout the world.

We are proud to present the class of 2007.

## **Dr. Carolyn J. Baglole**



### **Role of Lung Fibroblasts in Smoke-induced Lung Injury**

Lung inflammation and diseases such as chronic obstructive pulmonary disease (COPD) and cancer caused by cigarette smoke are serious problems, affecting millions of people. Lung structural cells called fibroblasts are stimulated by cigarette smoke to produce mediators that cause inflammation. Chronic production of inflammatory mediators is highly associated with COPD and induction of certain cancers. My studies are aimed at understanding how lung fibroblasts regulate inflammation and their role in smoke-induced lung injury.

**Mentor:** Professor Richard P. Phipps

**Institution:** University of Rochester, Rochester, NY

## **Dr. James P. Bridges**



### **Regulation of Pulmonary Surfactant Production by LPCAT, a Novel Enzyme**

The internal surfaces of the lung are coated with a substance known as surfactant, which is absolutely required for breathing. Surfactant deficiency is the major factor in respiratory distress syndrome (RDS) in premature infants and remains a significant public health problem.

Our lab recently isolated a novel enzyme, LPCAT, that we believe plays a critical role in surfactant production. The goal of my research project is to further characterize the function and regulation of this enzyme with the hope of pioneering new strategies to treat and prevent RDS and other lung pathologies resulting from surfactant deficiency.

**Mentor:** Professor John M. Shannon

**Institution:** Cincinnati Children's Hospital Medical Ctr., Cincinnati, OH

## **Dr. Kecia N. Carroll**



### **Respiratory Syncytial Virus Infection and Asthma**

Approximately 1/3 of children with a history of severe respiratory syncytial virus (RSV) bronchiolitis during infancy will develop asthma during early childhood. I will investigate whether bronchiolitis that is less severe or caused by non-RSV viruses is also associated with an increased risk of asthma. In addition, I will study whether having a familial predisposition to develop asthma or exposure to environmental tobacco smoke modifies the risk of asthma following bronchiolitis during infancy.

**Mentor:** Professor Tina V. Hartert

**Institution:** Vanderbilt University School of Medicine, Nashville, TN

## **Dr. Navneet K. Dhillon**



### **Nanoparticle Delivery of Gene Therapy for HIV to the Lungs**

My research is focused on developing a novel type of gene therapy that is aimed at inhibiting HIV replication in the target organ, the lungs. I will use nanoparticles to deliver antisense DNA against a host factor, interleukin-4 (IL-4) in an attempt to curtail replication of the virus, thereby preventing the damage to the lungs from the infection. This kind of therapy using particles in the nanometer range is safe, does not generate immune responses and can effectively deliver therapeutic genes over a long period of time.

**Mentor:** Professor Shilpa Buch

**Institution:** University of Kansas Medical Center, Kansas City, KS

## **Dr. Anke Di**



### **Activation of CFTR Chloride Channel in Bacterial Killing in the Lungs**

**Lungs** We have shown that intracellular CFTR, a chloride channel defective in cystic fibrosis (CF) patients, regulates the acidity of lysosomal/phagosomal compartments of certain lung cell types such as alveolar macrophages. These cells are crucial for defending lungs against infection. We believe that acidification of macrophages plays a fundamental role in regulating bacterial killing function of macrophages. Our studies will identify the signaling pathways that lead to intracellular CFTR activation and bacterial killing. Thus, the objective is to develop approaches that might be useful for combating infection in CF patients.

**Mentor:** Professor Asrar Malik

**Institution:** University of Illinois College of Medicine, Chicago, IL

## **Dr. Laura E. Fredenburgh**



### **COX-2 and Vascular Remodeling Due to Pulmonary Hypertension**

Pulmonary hypertension is a disease of the blood vessels in the lung in which the pressure in the pulmonary arteries rises, leading to progressive right-sided heart failure as the blood vessels in the lung are remodeled. New therapies that target the vascular remodeling process are needed to halt progression of this incurable disease. The goal of this research is to determine how the COX-2 enzyme affects the development of pulmonary hypertension, the inflammation that occurs with hypoxia-induced pulmonary hypertension, and the progressive vascular remodeling that leads to worsening symptoms and death. We hope that this research will lead to new insights and eventually new therapies for this disease.

**Mentor:** Professor Mark A. Perrella

**Institution:** Brigham and Women's Hospital, Boston, MA

## **Dr. Eliot B. Friedman**



### **Genetic Risk Factors for Obstructive Sleep Apnea**

Obstructive Sleep Apnea (OSA) affects 2-4% of the U.S. adult population and is independently associated with increased risk of developing strokes, heart disease and diabetes. The main focus of the current project is to harness the genetic tools available in *Drosophila* (fruit fly) to obtain a better understanding of the genes that lead to increased risks of developing complications associated with OSA.

**Mentor:** Professor Amita Sehgal

**Institution:** University of Pennsylvania, Philadelphia, PA

## **Dr. Jennifer L. Ingram**



### **Airway Structural Changes Due to Asthma**

My research focuses on an investigation of the molecular mechanisms that direct structural changes within the airways over time in asthma. These alterations in asthmatic airways result in gradually diminished lung function. The aims of my study are to evaluate the specific effects of an abundantly expressed protein in asthma on structural cells within the airway and to investigate interactions that this protein may have with corticosteroids, the current primary treatment strategy for asthma. My goals in this study are to define specific pathways within the airway structural cells that result in cell growth and contribute to airway fibrosis and obstruction.

**Mentor:** Professor Monica Kraft

**Institution:** Duke University, Durham, NC

## **Dr. Matthew R. Jones**



### **Innate Immune Lung Defenses Against Infection: Role of Zcchc11**

The innate immune response is the initial defense against bacterial infection of the lungs. When bacteria are detected by the host, a series of innate proteins are released to help guide inflammatory cells in the blood, which are called neutrophils, to the site of infection within the air spaces. The expression of these innate proteins, cytokines and chemokines, and neutrophil trafficking are essential to a healthy host response. Our laboratory research is focused on how an uncharacterized protein called Zcchc11 regulates the expression of cytokines and chemokines during pneumonia.

**Mentor:** Professor Joseph P. Mizgerd

**Institution:** Harvard School of Public Health, Boston, MA

## **Dr. Kevin K. Kim**



### **Lung Cell Injury and Idiopathic Pulmonary Fibrosis**

Idiopathic pulmonary fibrosis is a devastating disease which still lacks effective treatment. The underlying cellular mechanisms leading to pulmonary fibrosis remain unclear. This project explores a novel mechanism of pulmonary fibrosis in which injury causes normal lung cells to undergo dramatic changes which allows them to directly contribute to the fibrotic process. The goals of this project are to better understand this mechanism with the hope of designing new targets for therapy.

**Mentor:** Professor Harold A. Chapman

**Institution:** University of California, San Francisco, CA

## **Dr. Anice C. Lowen**



### **Factors Influencing the Spread of Influenza**

The severity of annual influenza epidemics and rare influenza pandemics is dependent in part on the efficiency with which the circulating strain can pass from person to person. To gain more insight into what factors determine the rate of influenza virus spread, we will investigate aspects of the host, the virus, and the environment. For example, we will assess whether the susceptibility of the host is the limiting factor in transmission and whether cold temperatures or low humidities - as seen in winter - increase transmission.

**Mentor:** Professor Peter Palese

**Institution:** Mount Sinai School of Medicine, New York, NY.

## **Dr. Timothy M. Moore**



### **Lung Microvascular Epithelial Cells and Acute Lung Injury**

Morbidity and mortality associated with acute lung injury (ALI) the adult respiratory distress syndrome (ARDS) remain high despite forty years of investigation. We are studying a novel mechanism by which the lung's microvascular endothelial cells may participate in the lung injury and possibly repair process. Our studies will characterize the molecular signaling between soluble CD40 ligand and endothelial cell CD40 receptors which lead to changes in microvascular endothelial barrier function, with the goal of our studies being translation of findings into better early diagnosis and treatment of ALI and ARDS in the critically ill patient.

**Mentor:** Professor Mary I. Townsley

**Institution:** Univ. of South Alabama College of Medicine, Mobile, AL.

## Dr. Christoph O. Randak



**AMP Binding Site and Regulation of CFTR Chloride Channel in Airway Cells** The cystic fibrosis transmembrane conductance regulator (CFTR) is a regulated chloride channel that is defective in cystic fibrosis. We previously discovered a mechanism that controls the opening and closing of CFTR that involves a signaling molecule called AMP. The goals of this research project are to study how agents that bind the AMP-site interact with CFTR to alter the movement of chloride across the membrane and to test the hypothesis that the AMP-site can be pharmacologically targeted to stimulate CFTR in cultured human airway epithelial cells. This work may provide new insights into how CFTR is regulated as well as new strategies to stimulate defective CFTR activity in cystic fibrosis.

**Mentor:** Professor Michael J. Welsh

**Institution:** University of Iowa College of Medicine, Iowa City, IA

## Dr. Emma L. Rawlins



### Genetic Characterization of Adult Stem Cells in the Lungs

Adult tissue-specific stem cells are thought to play crucial roles in the maintenance and repair of many organs, and changes in adult stem cell behavior can contribute to various diseases. Unfortunately little is known about adult stem cells in the lung, making their roles in different lung diseases very difficult to investigate. My research aims to address this problem by using genetic techniques to identify and characterize stem cells in the adult lung. Stem cells may have a critical role in repair and regeneration of injured lungs.

**Mentor:** Professor Brigid L. Hogan

**Institution:** Duke University, Durham, NC

## Dr. David Stoltz



### Defenses Against Pseudomonas Lung Infection in CF Patients

The lungs of cystic fibrosis patients are frequently infected by the bacteria *Pseudomonas aeruginosa*. This infection causes progressive illness and a decline in lung function. My proposed research will investigate how a common genetic variant in the enzyme paraoxonase-2 alters its protective effects against *P. aeruginosa* infection. The results of these studies will provide a better understanding of the control of *P. aeruginosa* infection and potentially new therapeutic options for patients with cystic fibrosis.

**Mentor:** Professor Joseph Zabner

**Institution:** University of Iowa College of Medicine, Iowa City, IA

## **Dr. Omar Tliba**



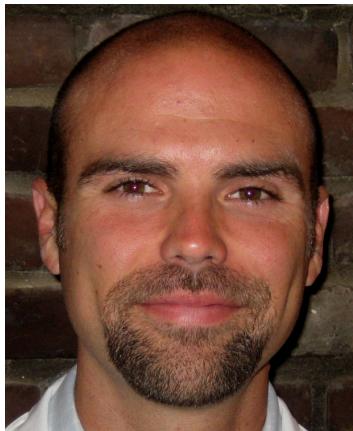
### **Role of Inflammatory Mediators in Steroid Resistant Asthma**

Although steroids are highly effective in the control of asthma, some patients with asthma fail to respond even to high doses and experience persistent airway obstruction and inflammation. I will investigate how inflammatory mediators modulate steroid responsiveness of airway structural cells. Finding the “bad” molecules that impaired steroid actions will offer new insight in the design of new therapeutic interventions to treat steroid-resistant asthmatics.

**Mentor:** Professor Reynold A. Panettieri

**Institution:** University of Pennsylvania, Philadelphia, PA

## **Dr. D. Walter Wray**



### **Antioxidants and Statins and Cardiovascular Function in COPD**

Chronic obstructive pulmonary disease (COPD) is a disorder that affects not just the lungs, but also the cardiovascular system. Cardiovascular problems in COPD patients are so severe that they are often unable to exercise, increasing the risk of cardiovascular disease. We plan to test the effectiveness of antioxidant vitamins and cholesterol-lowering ("statin") medications to improve muscle and blood vessel function in COPD patients. If proven effective in COPD patients, these drugs may directly improve muscle and vascular function, and also increase the patients' ability to exercise, further improving cardiovascular health.

**Mentor:** Professor Russell S. Richardson

**Institution:** University of California, San Diego, CA

## **Dr. Lisa R. Young**



### **Genetic Factors Influencing Interstitial Lung Disease**

Interstitial lung diseases (ILD) are disorders which cause progressive shortness of breath and respiratory failure due to scarring (fibrosis) in the lung. The cause of ILD is largely unknown, and no effective treatments exist. Hermansky-Pudlak Syndrome (HPS) is a rare inherited disorder in which almost all affected adults develop pulmonary fibrosis. This proposal will use a powerful genetic mouse model to understand how scarring occurs in the lung, and how scarring in the lung might be prevented.

**Mentor:** Professor Francis X. McCormack

**Institution:** Cincinnati Children's Hospital Medical Ctr., Cincinnati, OH