Dr. Atamas's research focuses on the molecular and cellular mechanisms of pulmonary fibrosis and asthma, specifically the interplay between immune inflammation and fibrosis. The Atamas lab utilizes advanced methods of gene delivery in experimental animals in vivo (adenovirus- and lentivirus-mediated delivery of mouse and human genes), various genetically altered mouse models, cell culture-based molecular research that focuses on intracellular signaling pathways that regulate fibroblast differentiation, proliferation and production of extracellular matrix, and advanced methods of gene and protein expression. Prior work has focused on the cytokines an alternatively spliced variant of IL-4, CCL18/PARC, oncostatin M, CCL2/MCP-1, and IL-33, and the cell surface molecules CD40–CD40L and T cell-associated integrins. More recently, the focus of the Atamas lab has expanded to include intracellular/intranuclear regulators of inflammation and fibrosis, including IL-33 precursor, NEU1 sialidase, and sirtuins.

Highlighted Publications:
3. Luzina IG, Pickering EM, Kopach P, Kang PH, Lockatell V, Todd NW, Papadimitriou JC, McKenzie AN, Atamas SP*. Full-length IL-33 promotes inflammation but not Th2 response in


Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Atamas-Sergei/
Google Scholar https://scholar.google.com/citations?user=C2LOTocAAAAJ

Konstantin Birukov (kbirukov@anes.umm.edu):

Dr. Birukov’s research interests cover several areas including: a) signal transduction; b) cytoskeletal mechanisms of endothelial permeability and inflammation; c) control of endothelial function by mechanical forces; d) role of oxidized phospholipids in lung pathobiology. His laboratory uses advanced biophysical and imaging methods, endothelial cell culture models of mechanical stress, and animal models of lung injury to understand the autoregulatory cascades
providing recovery and resolution of acute lung injury. He developed a new area of research addressing novel, barrier-protective and anti-inflammatory properties of oxidized phospholipids and proposed a new group of synthetic phospholipase resistant lipid mediators for future treatment of lung injury, inflammation and vascular barrier dysfunction.

Highlighted Publications:


Links:

Anna Birukova (abirukova@som.umaryland.edu):

Dr. Birukova’s research addresses mechanisms of cytoskeletal regulation of lung inflammation and resolution of acute lung injury with focus on microtubule (MT)-associated signaling and endothelial barrier function. Her prior studies showed a link between MT disassembly, activation of small GTPase Rho, and leak in human lung endothelial cells (EC). Rho activation is triggered by the MT-bound Rho-specific guanine nucleotide exchange factor, GEF-H1. Cyclic-AMP suppresses Rho signaling and EC permeability by stabilizing MT. This MT-dependent pathway is activated by pathologic (high amplitude) cyclic stretch as well as vasoactive and pro-inflammatory agonists such as TGFβ and TNFα. Importantly for recovery from acute lung injury/ARDS, Dr. Birukova has discovered that stimulation of MT growth promotes restoration of peripheral cytoskeleton, cell junctions, and EC barrier integrity by facilitating activation of Rac GTPase. Dr. Birukova made the interesting and clinically relevant observation that unlike pathologic stress, physiologic cyclic stretch accelerates restoration of endothelial barrier function through Rap1 GTPase- and cingulin-dependent downregulation of RhoA signaling.

Highlighted Publications:


Links:

**Simeon Goldblum** (sgoldblu@som.umaryland.edu):

Research in the Goldblum lab has focused on mechanisms through which septic and proinflammatory processes lead to pulmonary leukostasis and acute pulmonary microvascular endothelial injury and more recently glycobiology. Mechanisms studied include (1) protein tyrosine kinases and phosphatases that regulate the cell-cell adherens junctions or zonula adherens in response to exogenous and endogenous mediators; (2) serine/threonine phosphorylation events that provoke reversible disassembly of the tight junction in response to *Vibrio cholera*-derived zonula occludens toxin (ZOT), and its human homologue, zonulin; and (3) the role of protein sialylation and the human sialidases in regulating the human airway epithelial
cell response to environmental cues and danger signals, including the role of the sialidase NEU1 in desialylating surface receptors, including MUC1.

Highlighted Publications:


Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Goldblum-Simeon/

**Bart Griffith** (BGriffith@som.umaryland.edu):

Dr. Griffith’s research focuses on development of an artificial lung and mechanical blood pumps, lung transplantation, and advanced treatment of ARDS with anti-inflammatory treatment, including stem cells and ECMO.

Highlighted Publications:
2. **Griffith BP.** Children are not necessarily “small” adults: The growing field of miniaturized mechanical circulatory support. *Editorial Commentary* 2010 [http://www.jhltonline.org](http://www.jhltonline.org)


**Links:**

**Jeffrey Hasday** ([jhasday@som.umaryland.edu](mailto:jhasday@som.umaryland.edu)):

The Hasday lab has focused on how febrile-range hyperthermia and hypothermia modify biological processes relevant to disease pathogenesis with emphasis on acute lung injury/ARDS and fibrosis. Using approaches that span structural biology, gene and protein expression, cell culture, animal models and human trials, the Hasday laboratory has shown that hyperthermia worsens and hypothermia improves lung injury by modifying endothelial permeability, neutrophil recruitment, epithelial injury, and cytokine and heat shock protein expression. Dr. Hasday is expanding on his open-label trial of therapeutic hypothermia in ARDS by currently conducting a randomized clinical trial of hypothermia vs. standard temperature management in patients with ARDS. The p38 MAP kinase pathway appears to be a major contributor to the temperature-dependence of endothelial barrier function and expression of pro-inflammatory cytokines. The Hasday laboratory is following up on exciting data showing that the structure and function of
p38alpha, the proinflammatory p38 family member, but not p38beta is temperature-dependent in the 33° to 39°C range. Finally, the Hasday laboratory in collaboration with Dr. Paul Shapiro in the School of Pharmacy is designing novel p38alpha inhibitors that target the substrate binding domain rather than the catalytic domain of p38alpha. These novel compounds modify rather than inactivate its downstream signaling, are superior to conventional catalytic p38 inhibitors in preclinical testing, and are being developed into potential new drugs to treat ARDS and other inflammatory diseases. Dr. Hasday also directs the University of Maryland Cytokine Core Laboratory (www.cytokines.com).

Highlighted Publications:


Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Hasday-Jeffrey/
PubMed publications:
Erik Lillehoj (elillehoj@peds.umaryland.edu):

The Lillehoj laboratory focuses on the host response to lung infections caused by Pseudomonas aeruginosa, an opportunistic pathogen that impacts the morbidity and mortality of patients with a variety of respiratory diseases, including cystic fibrosis, ventilator-associated pneumonia, and chronic obstructive pulmonary disease. We have established that P. aeruginosa binds to airway epithelial cells through interaction of its flagellin with MUC1, a membrane-tethered mucin. More recently, using in vitro and in vivo model systems, we demonstrated that the P. aeruginosa flagellin-MUC1 interaction is regulated by neuraminidase-1 (NEU1), a host cell enzyme that desialylates MUC1. NEU1-mediated MUC1 desialylation not only renders it hyperadhesive for flagellin, but also increases its proteolytic shedding as a soluble decoy receptor that competitively inhibits P. aeruginosa adhesion to cell-associated MUC1. Our future goal is to develop the MUC1 decoy receptor as a novel therapeutic intervention for P. aeruginosa, and potentially other flagellated microbial pathogens, infecting the lungs.

Highlighted Publications:


Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Lillehoj-Erik/
PubMed publications:

Nirav Shah (nshah@som.umaryland.edu):

Dr. Shah’s research interests are in clinical studies of therapeutic hypothermia on lung injury and systemic inflammation and developing and evaluating medical education, including curriculum design and incorporation of new teaching methods such as flipped classroom, small group learning, and simulation into fellowship training.

Highlighted Publications:


Links:

**Carl Shanholtz** (Cshanhol@som.umaryland.edu):

Dr. Shanholtz has had a longstanding interest in improving management of critically ill patients and has a long and successful record of clinical research in critical illness in general, and acute respiratory distress syndrome specifically. Together Dr. Roy Brower, Dr. Shanholtz conducted the phase II clinical trial of low tidal volume ventilation in ARDS on which the ARDSNet ALVEOLI study was based and he has been site director for ARDSnet-I and II. Dr. Shanholtz also studies pain management/sedation pathways and pain assessment in critically ill patients, methods to reduce fluid administration to critically ill patients, and is a co-investigator on Dr. Hasday’s clinical trial of therapeutic hypothermia in ARDS patients.

Highlighted Publications:


Links:
Stefanie Vogel (svogel@som.umaryland.edu):

Dr. Vogel's focuses on the innate immune response to infection, the mechanisms by which inflammatory responses are regulated, macrophage differentiation and disease outcome, and targeting TLR signaling pathways to blunt pathogen-mediated acute lung injury. Dr. Vogel's most recent work has identified novel strategies for treating influenza therapeutically by blocking Toll-like receptor 4 signaling, the role of metabolism in the differentiation of macrophages, the cross-talk between innate immune signaling pathways, and other related topics. Innate immune responses to respiratory infections

Highlighted Publications:


Links:
PubMed publications:

**Zeljko Vujaskovic** (zvujaskovic@som.umaryland.edu):

Dr. Vujaskovic's research has focused on identifying the underlying mechanisms associated with radiation-induced normal tissue injury, including lung, and development of radioprotectants/radiomitigators to prevent and/or mitigate the acute and chronic side effects of radiation exposure, including radiation pneumonitis. Dr. Vujaskovic has extensive experience developing small and large animal models of acute radiation sickness and testing mitigators and/or treatment options to improve survival and reduce the long-term consequences associated with acute radiation exposure.
Highlighted Publications:

Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Vujaskovic-Zeljko/
PubMed publications:

Lin Zou (lzou@som.umaryland.edu):

My research interest has been inflammation and organ injury in sepsis, specifically the role of innate immune signaling, such as Toll-like receptors (TLRs) and complement, in the
development of cardiac and renal injury during sepsis. More recently, we demonstrated that extracellular RNAs/miRNAs are released during tissue ischemia or polymicrobial sepsis and are capable of activating TLR7-dependent cytokine/complement production in cardiac cells and macrophages. We are currently extending this work by studying the role of extracellular miRNA in sepsis induced acute lung injury.

Highlighted Publications:


Links:
Med School faculty page: http://www.medschool.umaryland.edu/profiles/Zou-Lin/