

Department of Anesthesiology

Research Faculty Profiles



2022 Edition Baltimore, Maryland

Our Mission

To deliver state-of-the-art anesthesia services in perioperative care, pain management and critical care; educate students, residents, and fellows; be recognized for its contributions to the specialty of anesthesiology through education, research, and scholarly activities; and contribute to the success of the Medical School and Medical System.

Anesthesiology Research

Anesthesiology Research consists of more than twenty principal investigators and four main areas of NIH-funded research: 1) mechanisms and treatment of traumatic and ischemic brain and spinal cord injury, 2) sepsis and myocardial injury, 3) acute lung injury, and 4) critical-care outcomes research. Our Department of Defense-funded investigators lead programs in 1) aeromedical transport safety, 2) traumatic injury and resuscitation, 3) developing predictive clinical algorithms for life-saving interventions, and 4) education research.

Department of Anesthesiology School of Medicine

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James P. Barrett, PhD

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Research Interests

The primary focus of my research is identifying molecular pathways and systemic factors that drive the chronic inflammatory responses observed in the brain during aging, neurodegenerative disease, and following Traumatic Brain Injury (TBI). My work involves the use of pharmacological agents and transgenic models that aim to modulate the inflammatory response to improve neurological function. My current work focuses on the role of Type I IFNs and Reactive Oxygen Species in the development of chronic microglial activation in aging and TBI.

- Barrett JP, Knoblach SM, Bhattacharya S, Gordish-Dressman H, Stoica BA, Loane DJ. Traumatic Brain Injury Induces cGAS Activation and Type I Interferon Signaling in Aged Mice. *Front Immunol*. 2021 Aug 24;12:710608. eCollection 2021. <u>PMID: 34504493</u>.
- Barrett J, Henry R, Shirey K A, Makarevich O, Doran S. J., Meadows V. A., Ritzel R.R., Vogel S. N., Faden A.I., Stoica B. A. and Loane D. J. Interferon-β plays a detrimental role in experimental traumatic brain injury by enhancing neuroinflammation that drives chronic neurodegeneration. *The Journal of neuroscience: the official journal of the Society for Neuroscience.* 2020. 40(11), 2357–2370. PMID: 32029532.
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- 4. **Barrett J,** Henry R, Villapol S, Stoica B, Kumar A, Burns M, Faden A, and Loane D. NOX2 deficiency alters macrophage phenotype through an IL-10/STAT3 dependent mechanism: implications for traumatic brain injury. *J Neuroinflammation*. 2017. 14: 65. PMID: 28340575.
- Kumar A, Barrett J, Alvarez-Croda D, Stoica B, Faden A, and Loane D. NOX2 drives M1-like microglial/macrophage activation and neurodegeneration following experimental traumatic brain injury. Brain Behav Immun. 2016. <u>PMID: 27477920.</u>



Konstantin G. Birukov, MD, PhD

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Research Interests

Dr. Birukov's laboratory is a part of the Lung Biology Program of which he is the director. This growing program currently includes collaborative studies between researchers from the Departments of Anesthesiology and Medicine but also develops programmatic links with Departments of Radiology/Oncology, Surgery, and with the Center for Advanced Sensor Technology at UMBC.

Dr. Birukov's group works to better understand the pathologic mechanisms of development and resolution of vascular endothelial dysfunction and lung injury, the two key features of many life-threatening conditions including ARDS, shock/trauma, sepsis, and others.

Topics of Focus:

- New roles of oxidized phospholipids in modulation of septic inflammation, coagulopathy and traumatic injury and age-related exacerbation of lung injury
- Mechanochemical regulation of vascular permeability and inflammation; the role of pathologic mechanical stretch and substrate stiffness in endothelial pathobiology.
- Discovery of novel signaling pathways and compounds contributing to resolution and recovery of lung injury.
- Cell interactions in lung injury, sepsis, and trauma

- Kim J, Nguyen T, Li Y, Zhang C, Cha B, Ke Y, Mazzeffi M, Tanaka K, Birukova A, Birukov KG. Contrasting effects of stored allogeneic red blood cells and their supernatants on permeability and inflammatory responses in human pulmonary endothelial cells. *American Journal Of Physiology. Lung Cellular And Molecular Physiology*. 2020. 318(3), L533–L548. <u>PMID: 31913681</u>.
- 2. Fang Y, Wu D, & **Birukov KG**. Mechanosensing and Mechanoregulation of Endothelial Cell Functions. *Comprehensive Physiology*. 2019. 9(2), 873–904. <u>PMID: 30873580</u>.
- 3. Wu D, **Birukov KG**. Endothelial Cell Mechano-Metabolomic Coupling to Disease States in the Lung Microvasculature. *Frontiers in Bioengineering and Biotechnology*. 2019. 7, 172. <u>PMID: 31380363</u>.
- 4. Karki P, **Birukov KG**. Lipid mediators in the regulation of endothelial barriers. *Tissue Barriers*. 2018; 6(1): e1385573. <u>PMID: 29083949</u>.
- 5. **Birukov KG**, Karki P. Injured lung endothelium: mechanisms of self-repair and agonist-assisted recovery (2017 Grover Conference Series). *Pulm Circ*. 2018 Jan-Mar;8(1):2045893217752660. <u>PMID: 29261029</u>.



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Research Interests

<u>My laboratory</u> is a part of the Anesthesiology Translational Research Program. We are interested in the molecular and cellular mechanisms of sepsis, traumatic injury, and ischemic myocardial injury. We have studied the role of innate immune signaling in the pathogenesis of these critical illnesses. For these studies, we use a combination of mouse genetics (transgenics and knockouts), physiology, biochemistry, and immunology.

Our current work focuses on four major projects. First, we have imaged myocardial extracellular (ex) nucleic acids released following ischemia-reperfusion (I/R) injury and demonstrated the importance of ex-RNA in myocardial inflammation and infarct following I/R insult. Second, in collaboration with multiple investigators within the STAR and the SOM, we study the role of ex-miRNAs→TLR7 signaling in the pathogenesis of polymicrobial sepsis, such as innate immune activation, acute lung injury, myocardial bioenergetics, coagulopathy, brain inflammation, and long-term neurocognitive dysfunction. We characterized the pro-inflammatory properties of a group of U-rich miRNAs and investigated the role of ex-mRNAs and their plasma extracellular vesicle (EV) carriers in murine and human sepsis. Third, in a mouse model of polytraumatic injury, we investigate the role of innate immune signaling in the secondary inflammatory damage following trauma and the impact of hypobaric condition on the innate immune response and organ injury. Finally, in collaboration with investigators at Mass General Hospital, Boston, and the STAR Clinical Informatics Group, we launched an interdisciplinary project two years ago designed to use machine learning and computer algorithms to study animal behaviors following neuropathic pain. We are supported by grants from the NIH (R01s, R35s, K08), Air Force, International Anesthesia Research Society, Shock Society, and National Science Foundation.

- 1. Chen F, Zou L, Williams B, **Chao W**. Targeting Toll-Like Receptors in Sepsis From Bench to Clinical Trials (Review). *Antioxid Redox Signal*. 2021; 35(15):1324-1339 PMID: 33588628.
- Wang S, Yang Y, Suen A, Zhu J, Williams B, Hu J, Chen F, Kozar R, Shen S, Li Z, Jeyaram A, Jay SM, Zou L, Chao W. Role of extracellular microRNA-146a-5p in host innate immunity and bacterial sepsis. *iScience*. 2021;24(12):103441. <u>PMID: 34877498</u>.
- 3. Zou L, He J, Gu L, Shahror RA, Li Y, Cao T, Wang S, Zhu J, Huang H, Chen F, Fan X, Wu J, **Chao W**. Brain innate immune response via miRNA-TLR7 sensing in polymicrobial sepsis. *Brain Behav Immun*. 2021; 100:10-24. <u>PMID: 34808293</u>.
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- Shimada BK, Yang Y, Zhu J, Wang S, Suen A, Kronstadt SM, Jeyaram A, Jay SM, Zou L, Chao W. Extracellular miR-146a-5p Induces Cardiac Innate Immune Response and Cardiomyocyte Dysfunction. *Immunohorizons*. 2020; 4(9):561-572. <u>PMID: 32958516</u>.



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Research Interests

My research focuses on signal transduction of innate and adaptive immune cells in lung inflammation and development. My major research project examines the role of IL-4 signal transduction in macrophage function in neonates who develop Bronchopulmonary dysplasia (BPD). The main objective of this project is to identify targets of macrophage modulators to mitigate BPD. Additionally, I am interested in the changes within the adaptive immune system following respiratory viral infection, such as respiratory syncytial virus and human rhinovirus that may increase the risk of allergic lung inflammation years later.

- 1. Deshpande SP, Sankova S, **Dorsey NJ**, Dawood MY, Tanaka K. Transcatheter tricuspid valve-in valve replacement-hope for the forgotten valve!. *Korean J Anesthesiol*. 2020;73(3):264-266. <u>PMID: 32160737</u>.
- Yang S, Menne A, Hu P, Dorsey NJ, Chiu W, Shackelford S, Mackenzie C. Acoustic sensor versus electrocardiographically derived respiratory rate in unstable trauma patients. *J Clin Monit Comput*. 2017;31(4):765-772. <u>PMID: 27270963</u>.
- 3. Dasgupta P, **Dorsey NJ**, Li J, et al. The adaptor protein insulin receptor substrate 2 inhibits alternative macrophage activation and allergic lung inflammation. *Sci Signal*. 2016;9(433):ra63. <u>PMID: 27330190</u>.
- Dorsey NJ, Chapoval SP, Smith EP, Skupsky J, Scott DW, Keegan AD. STAT6 controls the number of regulatory T cells in vivo, thereby regulating allergic lung inflammation. *J Immunol*. 2013;191(4):1517-1528. <u>PMID: 23825312</u>.
- Chapoval S, Dasgupta P, Dorsey NJ, Keegan AD. (2010). Regulation of the T helper cell type 2 (Th2)/T regulatory cell (Treg) balance by IL-4 and STAT6. *Journal of Leukocyte Biology*. 2010. 87(6), 1011–1018. PMID: 20335310.



Alan I. Faden, MD

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Research Interests

Alan I. Faden, M.D. is the David S. Brown Professor in Trauma in the Department of Anesthesiology. Dr. Faden's laboratory uses multi-disciplinary approaches- including molecular and cellular biology, animal modeling, behavior, imaging, and drug discovery- to examine the pathobiology of experimental brain and spinal cord injury and their treatment. Specific research focuses include neuroinflammation, cell cycle regulation, cell death pathways, metabotropic glutamate receptors, brain-systemic interactions, micro RNAs, and extracellular vesicles, as well as multifunctional drug treatment strategies for neurotrauma. Our lab uses several in vitro and in vivo models, along with diverse outcome measures to address experimental questions.

Publications

- Li Y, Ritzel RM, Lei Z, Cao T, He J, Faden AI, Wu J. Sexual dimorphism in neurological function after SCI is associated with disrupted neuroinflammation in both injured spinal cord and brain. *Brain Behav Immun*. 2021 Dec 23; 101:1-22. <u>PMID: 34954073</u>.
- Li Y, Ritzel RM, Khan N, Cao T, He J, Lei Z, Matyas JJ, Sabirzhanov B, Liu S, Li H, Stoica BA, Loane DJ, Faden AI, Wu J. Delayed microglial depletion after spinal cord injury reduces chronic inflammation and neurodegeneration in the brain and improves neurological recovery in male mice. *Theranostics*. 2020 Sep 14;10(25):11376-11403. <u>PMID: 33052221</u>.
- Makarevich O, Sabirzhanov B, Aubrecht TG, Glaser EP, Polster BM, Henry RJ, Faden AI, Stoica BA. Mithramycin selectively attenuates DNA-damage-induced neuronal cell death. *Cell Death Dis*. 2020 Jul 27;11(7):587. <u>PMID: 32719328.</u>
- Sabirzhanov B, Makarevich O, Barrett JP, Jackson IL, Glaser EP, Faden AI, Stoica BA. Irradiation-Induced Upregulation of miR-711 Inhibits DNA Repair and Promotes Neurodegeneration Pathways. *Int J Mol Sci.* 2020 Jul 23;21(15):5239. <u>PMID: 327180901.</u>
- 5. Li Y, Cao T, Ritzel RM, He J, **Faden AI**, Wu J. Dementia, Depression, and Associated Brain Inflammatory Mechanisms after Spinal Cord Injury. *Cells*. 2020 Jun 8;9(6):1420. <u>PMID: 32521597</u>.



Gary Fiskum, PhD

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Research Interests

My research interests include molecular mechanisms of adult and pediatric traumatic and ischemic brain injury, with emphasis on mitochondrial bioenergetic dysfunction, failure of cerebral energy metabolism, oxidative stress, apoptosis, and neuroprotection. My staff and collaborators have demonstrated how hyperoxia can be either neuroprotective or detrimental, depending on the form of brain injury and the time during which the injured brain is exposed to hyperoxia. This NIH-funded research resulted in a change in American Heart Association and International Advanced Cardiac Life Support Guidelines from the long-used indiscriminate use of 100% supplemental oxygen during resuscitation after cardiac arrest to the use of the minimal amount of oxygen necessary to achieve systemic normoxia. Recent work performed in collaboration with engineers at the University of Maryland, College Park has resulted in the first animal model of mild traumatic brain injury caused by the intense acceleration experienced by passengers within vehicles targeted by land mines. Our US Army-sponsored research using this model demonstrated how blast-induced acceleration alone can induce long-term anxiety, associated with acute cerebrovascular and axonal damage, inflammation, and loss of neuronal synapses. Our US Air Force-funded research found that exposure of rats to aeromedical evacuation-relevant hypobaria can exacerbate traumatic brain injury, particularly if the animals are exposed to hyperoxia during hypobaria. We are also determining if alterations in human blood cell oxygen consumption can be used as a biomarker of risk toward the development of sepsis by traumatic brain injury patients at the R Adams Cowley Shock Trauma Center.

- Proctor JL, Medina J, Rangghran P, Tamrakar P, Miller C, Puche A, Quan W, Coksaygan T, Drachenberg CB, Rosenthal RE, Stein DM, Kozar R, Wu F, **Fiskum G**. Air-Evacuation-Relevant Hypobaria Following Traumatic Brain Injury Plus Hemorrhagic Shock in Rats Increases Mortality and Injury to the Gut, Lungs, and Kidneys. *Shock*. 2021 Nov 1;56(5):793-802. <u>PMID: 33625116</u>.
- DeVience SJ, Lu X, Proctor JL, Rangghran P, Medina JA, Melhem ER, Gullapalli RP, Fiskum G, Mayer D. Enhancing Metabolic Imaging of Energy Metabolism in Traumatic Brain Injury Using Hyperpolarized [1-¹³ C] Pyruvate and Dichloroacetate. *Metabolites*. 2021 May 24;11(6):335. <u>PMID: 34073714</u>
- Goodfellow MJ, Borcar A, Proctor JL, Greco T, Rosenthal RE, Fiskum G. Transcriptional activation of antioxidant gene expression by Nrf2 protects against mitochondrial dysfunction and neuronal death associated with acute and chronic neurodegeneration. *Exp Neurol*. 2020 Jun;328:113247. <u>PMID</u>: <u>32061629</u>.
- Lee D, Pearson T, Proctor JL, Rosenthal RE, Fiskum G. Oximetry-Guided normoxic resuscitation following canine cardiac arrest reduces cerebellar Purkinje neuronal damage. *Resuscitation*. 2019 Jul;140:23-28.
 PMID: 31063840.
- Klinedinst NJ, Schuh R, Kittner SJ, Regenold WT, Kehs G, Hoch C, Hackney A, Fiskum G. Post-stroke fatigue as an indicator of underlying bioenergetics alterations. *J Bioenerg Biomembr*. 2019 pr;51(2):165-174. Epub 2019 Jan 7. <u>PMID: 30617735</u>.

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Research Interests

The primary goal of my research is to coordinate, develop, and lead efforts to advance the science of aeromedical critical care, combining regional applications with a global perspective, through the cultivation of a versatile and robust research methodology. I have secondary research interests in critical care regionalization/organization, patient safety, trauma anesthesiology, and advanced monitoring for the critically ill. My clinical work in the areas of emergency medicine, anesthesiology, and critical care medicine has helped me develop several hypotheses. In both civilian and military settings worldwide, aeromedical transport has been understood as an integral component of trauma systems, but the evidence for how to best use this expensive and limited resource is often lacking. In my efforts to improve the care of critically injured patients, I plan to spend the remainder of my career performing studies that will help shape local and global policies for how best to allocate aeromedical resources and how to improve patient care en route from the scene of the injury to the hospital and beyond.

- Leung S, Pastores SM, Oropello JM, Lilly CM, Galvagno SM Jr, Badjatia N, Jacobi J, Herr DL, Oliveira JD; Academic Leaders in Critical Care Medicine Task Force of the Society of Critical Care Medicine. Regionalization of Critical Care in the United States: Current State and Proposed Framework From the Academic Leaders in Critical Care Medicine Task Force of the Society of Critical Care Medicine. *Crit Care Med.* 2022 Jan 1;50(1):37-49. <u>PMID: 34259453.</u>
- Galvagno SM Jr, Naumann A, Delbridge TR, Kelly MA, Scalea TM. The Role of a Statewide Critical Care Coordination Center in the Coronavirus Disease 2019 Pandemic-and Beyond. *Crit Care Explor.* 2021 Oct 28;3(11):e0568. <u>PMID: 34729494.</u>
- Zadorozny EV, Weigel T, Stone A, Gruen DS, Galvagno SM Jr, Yazer MH, Brown JB, Guyette FX. Prehospital Lactate is Associated with the Need for Blood in Trauma. *Prehosp Emerg Care*. 2021 Nov 16:1-10. <u>PMID: 34550050.</u>
- 4. **Galvagno SM Jr**, Smith CE, Varon AJ, Hasenboehler EA, Sultan S, Shaefer G, To KB, Fox AD, Alley DE, Detillo M, Joseph BA, Robinson BR, Haut ER. Pain management for blunt thoracic trauma: A joint practice management guideline from the Eastern Association for the Surgery of Trauma and Trauma Anesthesiology Society. *J Trauma Acute Care Surg*. 2016 Nov; 81(5): 936–951. <u>PMID: 27533913</u>.
- Hirshon JM, Galvagno SM Jr, Comer A, Millin MG, Floccare DJ, Alcorta RL, Lawner BJ, Margolis AM, Nable JV, Bass RR. Maryland's helicopter emergency medical services experience from 2001-2011: System improvements and patients' outcomes. *Ann Emerg Med.* 2016 Mar; 67 (3): 332–340.e3. <u>PMID:</u> <u>26433494</u>.



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Research Interests

During my 21-year career in the U.S. Air Force, I had the honor of participating in the development of multiple academic programs as both a fellowship and program director for the Air Force's only anesthesiology residency, as the creator of the first multi-disciplinary training program for the Critical Care Air Transport Teams, and finally as director of the Center for the Sustainment of Training and Readiness Skills in Baltimore. In my current position as a trauma anesthesiologist at the R Adams Cowley Shock Trauma Center, I have been able to continue working with traumatically injured patients with a focus on airway management, resuscitation, and simulation-based training.

- 1. J Richards, R Samet, A Koerner, **TE Grissom**. Tranexamic acid in the perioperative period: yes, no, maybe? *Adv Anesth*. 37:87-110, 2019. <u>PMID: 31677661</u>.
- 2. **TE Grissom**. Walking the tightrope of bleeding control: fibrinolysis in trauma. *Anesth Analg.* 129:644-6, 2019. <u>PMID: 31425200.</u>
- 3. Copeland CC, Conti BM, Fouche-Weber LY, **Grissom TE**. Butterfly Vertebrae. *Anesthesiology*. 2018 August; 129 (3): 582–582. <u>PMID:29762183.</u>
- Gatz JD, Grissom TE, Chauhan R. Emergent needle decompression for tension pneumothorax resulting in life threatening intercostal artery injury. *Glob Anesth Perioper Med*. 2018 May; 3:237-240. DOI: 10.15761/GAPM.1000163.
- 5. **Grissom TE**. Management of the Trauma Patient on Direct Oral Anticoagulants. *Curr Anesthesiol* Rep. 2018 Mar, 8(1):107-118. DOI: 10.1007/s40140-018-0253-x.



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Research Interest

My research interest is mainly focused on the improvement of patient blood management. Clinically, I am a cardiothoracic anesthesiologist where allogeneic transfusion rates are high. With blood conservation techniques such as acute normovolemic hemodilution, viscoelastic guided transfusion, factor concentrate administration, and alternative blood substitutes bloodless cardiac surgery can be achieved. I have utilized coagulation assessments to monitor the effects of novel agents in clinical and pre-clinical studies. I look to further study allogeneic blood transfusion and its effects on endothelial cell function, for which I recently received funding from the Society for the Advancement of Blood Management.

I am also interested in valvular and left/right ventricular assessment by transesophageal echocardiography. With the improvement in 3D technology, we will be able to determine the operative planning and overall outcomes prior to intervention.

Publication

- 1. Tanaka KA, **Henderson RA**, Williams B. Heparin-induced thrombocytopenia and cardiac surgery: can we do it all with cangrelor? *A&A Practice*. 2019;13(9):366. <u>PMID: 31567127.</u>
- 2. Tanaka KA, Bharadwaj S, Hasan S, Judd M, Abuelkasem E, **Henderson RA** et al. Elevated fibrinogen, von Willebrand factor, and Factor VIII confer resistance to dilutional coagulopathy and activated protein C in normal pregnant women. *British Journal of Anaesthesia*. 2019;122(6):751-9. <u>PMID: 30916034</u>.
- 3. **Henderson RA**, Mazzeffi MA, Strauss ER, Williams B, Wipfli C, Dawood M et al. Impact of intraoperative high-volume autologous blood collection on allogeneic transfusion during and after cardiac surgery: a propensity score matched analysis. *Transfusion*. 2019;59(6):2023-9. <u>PMID: 30882929</u>.
- 4. **Henderson RA**, Chow JH, Tanaka KA. A bridge to bloodless surgery: use of hemoglobin-based oxygen carrier for anemia treatment and autologous blood preservation during redo pulmonic valve replacement. *Journal Of Cardiothoracic and Vascular Anesthesia*. 2019;33(7):1973-6. <u>PMID: 30529178</u>.
- Abuelkasem E, Mazzeffi MA, Henderson RA, Wipfli C, Monroe A, Strauss ER et al. Clinical impact of protamine titration-based heparin neutralization in patients undergoing coronary bypass grafting surgery. *Journal Of Cardiothoracic and Vascular Anesthesia*. 2019. <u>PMID: 30737123</u>.



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Research Interests

Bingren Hu, MD, PhD, is a Professor in Trauma in the Department of Anesthesiology. Dr. Hu is the director of the Tissue Ischemia-Reperfusion Research Program within the Department of Anesthesiology. Dr. Hu's laboratory's main research interests include molecular mechanisms, treatments of brain ischemia, traumatic brain injury, and hemorrhagic shock. In brain ischemia research, his laboratory employs both global and focal ischemia, and neonatal hypoxia-ischemia rat and mouse models to study: (i) protein misfolding and aggregation; (ii) membrane trafficking; (iii) protein degradation pathways; (iv) blood-based biomarkers; and (v) novel therapeutics against ischemia-reperfusion brain injury. In traumatic brain injury research, his laboratory employs a rat fluid percussion injury model to investigate aberrant synaptogenesis and post-TBI seizures. In hemorrhagic shock research, his laboratory employs both rat and swine lethal hemorrhagic shock in the brain, spinal cord, and abdominal organs.

- 1. Kristian T, Hu B. The Protein Modification and Degradation Pathways after Brain Ischemia. *Transl Stroke Res.* 2018 Oct 1. <u>PMID: 28967038</u>.
- 2. Yuan D, Liu C, Wu J, **Hu B**. Nest-building activity as a reproducible and long-term stroke deficit test in a mouse model of stroke. *Brain Behav*. 2018 Jun;8(6): e00993 PMID: 30106254.
- 3. Yuan D, Liu C, Wu J, **Hu B.** Inactivation of NSF ATPase Leads to Cathepsin B Release After Transient Cerebral Ischemia. *Transl Stroke Res.* 2018 Jun;9(3):201-213. <u>PMID: 29039034</u>.
- 4. Yuan D, Liu C, **Hu B**. Dysfunction of Membrane Trafficking Leads to Ischemia-Reperfusion Injury After Transient Cerebral Ischemia. *Transl Stroke Res.* 2018 Jun;9(3):215-222. <u>PMID: 29022237</u>.
- 5. Sun X, Crawford R, Liu C, Luo T, **Hu B**. Development-dependent regulation of molecular chaperones after hypoxia-ischemia. *Neurobiol Dis*. 2015 Jun 9; 82:123-131. <u>PMID: 26070787</u>.



Peter F. Hu, PhD

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Research Interests

Clinical Informatics and Analytical Research Group are composed of research faculty; Professor and Chairman of Anesthesiology, Peter Rock, Professor; Peter Hu, Assistant Professor; Shiming Yang, Professor Emeritus; Colin Mackenzie, 2-4 medical students, and residents; and 4-8 PhD students in computer science and engineering in our lab. Our research is focused on developing machine learning-based predictive algorithms for near and long-term patient outcomes based on the continuous vital signs from the field to in-hospital resuscitation, the intensive care unit bedside. Our research has been continuously funded by DoD (USAF, Naval Research, US Army, and Veterans Administration). In the past, we also were funded by NIH/NLM, NSF, NASA, AHRQ, and industry. Specifically, we have developed and tested a Bleeding Risk Index (BRI) Monitor for a minute-by-minute analysis of continuous photoplethysmograph (PPG)



waveform (shown in the figure to the right). This monitor could be used for predicting future transfusion needs in the field. We also developed an ICU Viewer, which takes real-time patient monitor data and provides an at-aglance view for the units (SICU, NTCC, MTCC) or an individual bed view for up to 7 days (shown in the figure to the right). Currently, we have 6 extramural funded projects with over \$12 million in funding.

- Yang S, Mackenzie CF, Rock P, Lin C, Floccare D, Scalea T, Stumpf F, Winans C, Galvagno S, Miller C, Stein D, Hu PF. Comparison of massive and emergency transfusion prediction scoring systems after trauma with a new Bleeding Risk Index score applied in-flight. *J Trauma Acute Care Surg.* 2021 Feb 1;90(2):268-273. <u>PMID: 33502145</u>.
- Chow JH, Richards JE, Galvagno SM, Coleman PJ, Lankford AS, Hendrix C, Dunitz J, Ibrahim I, Ghneim M, Tanaka KA, Scalea TM, Mazzeffi MA, Hu P. The Algorithm Examining the Risk of Massive Transfusion (ALERT) Score Accurately Predicts Massive Transfusion at the Scene of Injury and on Arrival to the Trauma Bay: A Retrospective Analysis. *Shock*. 2021 Oct 1;56(4):529-536. <u>PMID: 34524267</u>.
- 3. Galvagno SM Jr, Sikorski RA, Floccare DJ, Rock P, Mazzeffi MA, DuBose JJ, Scalea TM, Miller C, Richards JE, O'Connor JV, Mackenzie CF, **Hu P**. Prehospital Point of Care Testing for the Early Detection of Shock and Prediction of Lifesaving Interventions. *Shock*. 2020 May 21. <u>PMID: 32453248</u>.
- 4. Tisherman SA, **Hu FM.** Can we stop patients from "falling off the cliff"? *Resuscitation*. 2019 Jun; 139:363-364. Epub 2019 Apr 24. <u>PMID: 31028825</u>.
- 5. Yang S, Stansbury LG, Rock P, Scalea T, **Hu PF**. Linking Big Data and Prediction Strategies: Tools, Pitfalls, and Lessons Learned. *Crit Care Med*. 2019 Jun;47(6):840-848. <u>PMID: 30920408</u>.



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Research Interests

Endothelial barrier dysfunction is an underlying cause of vascular leak, pulmonary edema, and infiltration of inflammatory cells in the lungs leading to acute lung injury (ALI) and its more severe form, acute respiratory distress syndrome (ARDS). ARDS may develop in response to inadequate mechanical ventilation, blood transfusion, sepsis, and trauma, etc. Normal endothelial cell (EC) barrier function is maintained by a dynamic balance among internal EC cytoskeletal tension, focal adhesions, adhesive junctions, and microtubule network. A variety of signaling molecules including G-protein coupled receptors and protein kinase/phosphatases regulate assembly/disassembly of the cytoskeletal components and modulate vascular permeability in the lungs. Cell signaling mediated by Rac1-Pak1 is among those that antagonize the loss of endothelial barrier integrity induced by endotoxin/inflammatory mediators and enhance barrier function. Specific phospholipids and other extracellular factors such as prostaglandins protect endothelial barriers through activation or suppression of multiple signaling pathways that are coordinated and often converged at the cytoskeletal complexes. My research is focused on understanding the molecular mechanisms and intracellular processes that control endothelial barrier functions. One of my current projects is to illuminate the molecular mechanisms underlying the development of ARDS induced by traumatic brain injury. To address these questions, I employ a broad spectrum of methods at molecular, cellular, and organismic levels with the ultimate goal to develop novel therapeutic strategies for the treatment of acute lung injury and pathologic conditions associated with the increased vascular leak.

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Research Interest

My research interests include capnography, physiology of pregnancy, airway changes during labor, laparoscopy during pregnancy, coagulation and blood transfusion, hemodynamic changes during cesarean delivery, and operating room efficiency. My work showed, for the first time, that arterial to end-tidal CO2 difference in pregnancy is almost zero. Furthermore, end-tidal CO2 reflects arterial CO2 during laparoscopic surgery in pregnancy. Airway change during labor was one of the highlighted publications in *Anesthesiology*, accompanied by an editorial. I was the first to demonstrate that pulmonary ventilation decreases during labor following neuraxial analgesia and may be the cause of epidural-related fever.

Continuing the work on coagulation and respiratory physiology in pregnancy, I spearheaded the evaluation of a novel SEER technology that uses state-of-the-art ultrasound technology in pregnant women to assess coagulation expeditiously. The study showed SEER technology to be reliable in detecting low levels of fibrinogen. The pandemic of COVID offered an unfortunate opportunity to study respiratory physiology in COVID Parturients requiring mechanical ventilation and/or ECMO. Our division published lead articles on this subject. The division is also collaborating on transitional research with Dr. Wei Chao's Lab on mRNA in preeclampsia.

I also maintain a very comprehensive website <u>www.capnography.com</u>. *Anesthesiology* noted this website to be a comprehensive dynamic textbook on capnography.

Publications

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 PMID: 33684077.
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Research Interests

Research activity in my lab can be divided into two major projects: 1) the role of cell-type-specific mitochondrial dynamics in acute brain injury; (2) disturbed NAD⁺ metabolism and its contribution to the cell death mechanism in neurodegenerative disease. Our recent studies, which utilize transgenic animals expressing neuronal or astrocytic mitochondria-targeted fluorescent markers in the brain, show that mitochondria in neurons and astrocytes differentially respond to stress conditions. We first reported that the mitochondria in cells destined to die are not able to re-fuse and regain their pre-insult morphology and functions (Owens et al. 2015) and that both neuronal and astrocytic mitochondria are damaged by excitotoxic insult during ischemic conditions.

It is well established that massive degradation of NAD⁺ can significantly compromise cell survival. Recently, we reported that administering nicotinamide mononucleotide (NMN), a precursor for NAD⁺ synthesis, inhibits NAD⁺ degradation and leads to dramatic protection against ischemic brain injury (Park et al. 2016). We recently revealed that NMN affects several downstream targets that promote the survival of brain cells following pathologic stress (Klimova et al. 2019). We are now characterizing the mechanism of NMN neuroprotection by determining the post-translational modifications of proteins controlling mitochondrial dynamics (Klimova et al. 2020).

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- 4. Long A, Park JH, Klimova N, Fowler CB, Loane DJ, **Kristian T**. CD38 knockout mice show significant protection against ischemic brain damage despite high level poly-ADP-ribosylation. *Neurochem Res*. 2017; 42(1):283-293. <u>PMID: 27518087</u>.
- Park JH, Long A, Owens K, Kristian T. Nicotinamide mononucleotide inhibits post-ischemic NAD+ degradation and dramatically ameliorates brain damage following global cerebral ischemia. *Neurobiol Dis*. 2016; 95:102-110. <u>PMID: 27425894</u>.



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Research Interests

Autophagy is a catabolic process mediating the turnover of bulk cytoplasmic constituents including organelles and protein aggregates in a lysosome-dependent manner. It is necessary for cellular homeostasis and protects organisms from a variety of diseases, including neurodegeneration and aging. Accumulation of autophagosomes has been observed following traumatic brain injury (TBI) and spinal cord injury (SCI), but its mechanisms and function in those contexts remain unknown. We use in vivo and in vitro models to examine the role of autophagy after TBI and SCI, and to delineate the molecular mechanisms involved. Our data demonstrate that although autophagosomes accumulate in the brain and spinal cord after TBI and SCI, respectively, autophagic degradation cannot proceed to completion. In neurons, this block of autophagy is caused by phospholipase-mediated lysosomal membrane damage and contributes to neuronal cell death. Inhibition of autophagy is also observed in activated microglia and infiltrating macrophages and may contribute to neuroinflammation. We are currently investigating the effects of TBI-induced perturbation in brain lipid homeostasis on microglial and macrophage autophagy and assessing the contribution of the autophagy-lysosomal pathway to delayed development of neurodegeneration and dementia after TBI. Additionally, we are using in vitro models, including human induced pluripotent stem (iPS) cells, to examine the function and mechanisms of USP24, a novel gene associated with Parkinson's disease (PD). Our long-term goal is to define novel target molecules and pathways for safe and effective modulation of autophagy as a treatment against neurodegeneration induced by both acute (trauma) and chronic (neurodegenerative diseases) causes.

- Hegdekar N, Lipinski MM and Sarkar C. N-acetyl-L-leucine treatment attenuates neuronal cell death and suppresses neuroinflammation after traumatic brain injury in mice. *Sci Reports*. 2021; 11(1):9249. <u>PMID</u>: <u>33927281</u>.
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- Li Y, Jones JW, Choi HMC, Sarkar C, Kane MA, Koh EY, Lipinski MM[#] and Wu J[#]. cPLA2 activation contributes to lysosomal defects leading to impairment of autophagy after spinal cord injury. *Cell Death* & Disease. 2019; Jul 11:10(7):531. <u>PMID: 31296844</u> [#]co-senior authors.
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Patrick N. Odonkor, MB, ChB

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Research Interests

The primary focus of my research is improvement in clinical outcomes in patients having cardiac surgery. I have worked in collaboration with other anesthesiologists and cardiac surgical colleagues on perioperative clinical management of patients during cardiac surgery. Areas of interest include blood coagulation management, anesthetic management for high-risk procedures and prevention of cardiac surgery-associated acute kidney injury.

Over the last 5 years, I have been actively involved in the development of a long-term survival model in orthotopic cardiac xenotransplantation in primates. These efforts have led to the achievement of reliable medium-term survival in baboons that have undergone cardiac xenotransplantation using a genetically modified pig heart. Our experience in the lab led to a recent successful genetically modified pig to human orthotopic cardiac xenotransplant at the University of Maryland School of Medicine.

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Research Interests

Limiting damage to mitochondria, the primary energy-generating organelles of the cell is crucial for neuroprotection. My laboratory studies basic subcellular mechanisms that govern neuroinflammation and cell death in neurodegenerative disorders, with a focus on mitochondrial bioenergetics. One of our current projects focuses on how inflammatory microglial activation influences injury to neurons and astrocytes through nitric oxide production and how mechanisms differ depending on oxygen availability. We are exploring pharmacological ways to bypass deficits in mitochondrial electron transport. In another project, we are investigating the role of mitochondrial structural and functional remodeling in microglial activation following traumatic brain injury. We are examining the necessity of mitochondrial fragmentation, respiratory changes, and reactive oxygen species production to the pro-inflammatory phenotype. We have pioneered the development and implementation of two novel applications of Seahorse Bioscience Extracellular Flux Technology, a real-time assessment of mitochondrial respiration within permeabilized brain cells and from whole brain tissue slices, expanding the ways in which mitochondrial function can be studied in cells of the central nervous system.

- Jaber SM, Ge SX, Milstein JL, VanRyzin JW, Waddell J, **Polster BM**. Idebenone Has Distinct Effects on Mitochondrial Respiration in Cortical Astrocytes Compared to Cortical Neurons Due to Differential NQO1 Activity. *J Neurosci*. Jun 2020; 40(23), 4609-4619. <u>PMID: 32350039</u>.
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Justin E. Richards, MD

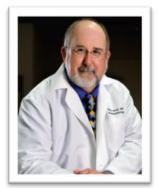
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Research Interests

My research work focuses on the early management of severely injured and critically ill trauma patients. Previous projects have included early markers of depth of shock and multiple organ failure in severely injured patients. We are currently working on further projects utilizing prehospital data to characterize organ failure risk, in addition to describing other admission laboratory markers of organ failure and specific organ dysfunction, such as acute kidney injury and blood failure (i.e. traumatic coagulopathy). We currently also have an ongoing multi-center project with colleagues in France that is evaluating early resuscitation management in trauma patients and the differences between our trauma centers in different countries.

In addition to the acutely injured trauma population, we are studying the specific population of orthopedic trauma patients with severe musculoskeletal injuries. The areas on which we are focusing with this group include perioperative optimization in patients with multiple injuries and significant medical co-morbidities, such as cardiovascular risk factors

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- Richards JE, Mazzeffi MA, Massey MS, Rock P, Galvagno SM, Scalea TM. The Bitter and the Sweet: Relationship of Lactate, Glucose, and Mortality After Severe Blunt Trauma. *Anesth Analg.* 2021;133(2):455-461. <u>PMID: 33475264</u>.
- 3. **Richards JE**, Fedeles BT, Chow JH, Fedeles BT, Chow JH, Morrison JJ, Renner C, Trinh AT, Schlee CS, Koerner K, Grissom TE, Betzold RD, Scalea TM, Kozar RA. Is Tranexamic Acid Associated With Mortality or Multiple Organ Failure Following Severe Injury? *Shock*. 2021 Jan 1;55(1):55-60. PMID: 33337787.
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Research Interests

Peter Rock, MD, MBA, FCCM

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My research focuses on 1) mechanisms resulting in and treatment of acute lung injury; 2) weakness in patients with critical illnesses; 3) identification of genetic determinants of infectious and vascular occlusive complications in patients who undergo surgical procedures, and the use of genetic information to develop tools to identify patients at risk of infectious or thrombotic complications and that allow perioperative physicians to tailor therapy to potentially treat or prevent these complications; 4) prevention of delirium after surgery and prevention of delirium in Intensive care unit patients; 5) medical informatics and "big data" in anesthesiology and critical-care; and 6) machine learning and use of vital signs to predict changes in patient status or for life-saving interventions.

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Research Interests

My research interest is focused on understanding the role and function of lipids in neurodegeneration and neuroinflammation in traumatic brain injury (TBI) and other neurodegenerative diseases. My recent study indicated that peroxisomal etherphospholipid biogenesis is dysregulated in the mouse cortices after TBI. Etherphospholipids are major components of cellular membrane and play an important role in cellular signaling via their structural impact on the formation and function of lipid rafts. They also serve as cellular antioxidants by scavenging reactive oxygen species. Currently, I am assessing how etherphospholipids and peroxisomal functions are dysregulated after TBI.

We previously demonstrated that autophagy, a lysosome dependent cellular degradative process, is disrupted after TBI. We showed that it is associated with neuronal loss after brain injury. In my other projects, I am assessing different treatment strategies to restrict neuronal loss and neuroinflammation in the injured brain by upregulating autophagy using different pharmacological agents. I am also working on improving the effectiveness of stem cell transplantation in the injured brain by enhancing autophagy after TBI.

- Morel Y, Hegdekar N, Sarkar C, Lipinski MM, Kane MA, Jones JW. Structure-specific, accurate quantitation of plasmalogen glycerophosphoethanolamine. *Anal. Chim. Acta*. 2021 Nov. 22;1186:339088. Epub 2021 Sep 22. PMID: 34756256.
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- 3. Klionsky DJ, Abdel-Aziz AK, Abdelfatah S, **Sarkar C** et al. Guidelines for the use and interpretation of assays for monitoring autophagy. *Autophagy*, 2021. 1-382. <u>PMID: 22966490</u>.
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Research Interests

The main focus of my research is to understand the molecular mechanisms of neuronal cell death and neuroinflammation after central nervous system trauma. My studies are based on the hypothesis that brain trauma initiates multiple maladaptive mechanisms (secondary injury) that lead to improper activation of neuronal cell death pathways and/or prevent efficient activation of neuronal repair mechanisms. Thus, neurons that receive a survivable injury are unnecessarily removed and/or fail to undergo effective repair/regeneration. An important driver of these changes is injury-induced activation of microglia toward specific persistent pro-inflammatory phenotypes resulting in secondary neurotoxicity.

An area of special interest is the modulation of secondary injury mechanisms by microRNAs, a group of regulatory non-coding small RNA molecules following experimental traumatic brain injury (TBI). Our recent data suggest that injury-induced changes in specific microRNAs are key to the activation of neuronal cell death pathways and ultimately to neuronal cell loss after TBI.

By identifying the injury-induced molecular dysfunctions we can design optimal therapeutic approaches that will shift microglia activation toward neurorestorative phenotypes to increase neuronal survival and recovery after brain trauma, thus improving neurological deficits.

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Research Interests

My research focuses on investigating the effects of stress and stress-inducing genes on traumatic brain injury pathophysiology and functional outcomes. We use *in vivo* models of physiological, psychological, and environmental stress to determine the detrimental effects of stress on traumatic brain injury associated markers of oxidative stress, blood-brain barrier dysfunction, inflammation, and neuronal cell death and their consequences on behavioral functions including learning/memory, anxiety, and motor performance. We use targeted experimental therapeutic approaches to evaluate the therapeutic properties of promising test compounds on stress-induced pathological alterations and functional deficits following traumatic brain injury. Our goal is to conduct successful translational research and take our findings from the laboratory benchtop to the patient's bedside.

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Research Interests

My research interests have focused on studying the link between systemic inflammation and altered coagulation. Coagulopathy is commonly described as impaired endogenous clotting ability with loss of localization and risk of intravascular thrombosis and bleeding, and greatly affects mortality in critically ill patients. My research has mainly focused on characterizing and describing intravascular coagulation dysfunction in the setting of systemic inflammation and the study of the role of innate immune signaling in platelet activation. My future goal is to continue to make effort in this field to gain further understanding into abnormal coagulation responses in systemic proinflammatory states.

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Research Interests

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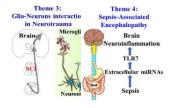
Dr. Wu's laboratory studies secondary injury processes following traumatic spinal cord and brain injury (SCI/TBI) and pharmacological/gene therapeutic interventions for CNS trauma. Specifically, we focus on: (1) Elucidating molecular mechanisms responsible for SCI/TBI-induced CNS neuroinflammation and neurological dysfunction. (2) Addressing the function and the mechanisms of autophagy-lysosomal pathway and specific microRNAs in neuronal injury after SCI

Theme 1: Mechanisms of Motor





which could open a potential novel treatment avenue against SCI as well as identify candidate molecular targets for these manipulations; (3) Identifying the genetic and genomic factors that impact SCI-PAIN as well as identifying new therapeutic targets to reduce or eliminate SCI-PAIN, including a truncated isoform of the BDNF receptor tropomyosin related kinase B (trkB), trkB.T1 and NOX2. (4) Examining the function and mechanisms of voltage-gated proton channels Hv1 on neuroinflammation and neuropathic pain after SCI and TBI. (5) Identifying how plasma exosomes-associated microRNAs drive remote brain neuroinflammation after SCI in order to allow future development of novel therapies. The research tools used in my lab include rodent models of SCI and TBI, animal



behavior testing (for motor function, pain, cognition, and depression), characterization of extracellular vesicles (EVs) using Nanoparticle Tracking Analysis and ExoViewTM, advanced flow cytometry technology, quantitative image analysis, stereological cellular assessments, in vivo administration of therapeutics, as well as primary neuronal and glial culture. My ultimate goal is to understand the cellular and molecular mechanism of functional recovery after CNS trauma and also to develop potential therapeutic strategies.

- Ritzel RM, He J, Li Y, Cao T, Khan N, Shim B, Sabirzhanov B, Aubrecht T, Stoica BA, Faden AI, Wu L-J, Wu J. Proton extrusion during oxidative burst in microglia exacerbates pathological acidosis following traumatic brain injury. *Glia*, 2021 Mar;69(3):746-764. <u>PMID: 33090575.</u>
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- Ritzel RM, Li Y, He J, Khan N, Doran S, Faden AI, and Wu J. Sustained neuronal and microglial alterations are associated with diverse neurobehavioral dysfunction long after experimental brain injury. *Neurobiology of Disease*, 2020; 136:104713. <u>PMID: 31843705.</u>



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Research Interests

My research interest is focused on large scale data analysis and medical sensor signal processing with a goal of developing an efficient machine learning algorithm, to predict lifesaving interventions and long-term outcomes for trauma patients.

- Zeineddin A, Hu P, Yang S, Floccare D, Lin CY, Scalea TM, Kozar RA. Prehospital continuous vital signs predict need for resuscitative endovascular balloon occlusion of the aorta and resuscitative thoracotomy prehospital continuous vital signs predict resuscitative endovascular balloon occlusion of the aorta. *Journal of Trauma and Acute Care Surgery*. 2021 Jun 8. <u>PMID: 33797486</u>.
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- 5. Wasicek PJ, **Yang S**, Teeter WA, Hu P, Stein DM, Scalea TM, Brenner ML. Traumatic cardiac arrest and resuscitative endovascular balloon occlusion of the aorta (REBOA): a preliminary analysis utilizing high fidelity invasive blood pressure recording and videography. *European Journal of Trauma and Emergency Surgery*. 2019 Dec;45(6):1097-105. <u>PMID: 30032348</u>.



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Research Interests

Sepsis is a clinical syndrome with life-threatening organ dysfunction that is caused by a dysregulated host response to infection. The lung is one of the first organs to fail during sepsis and contributes to high mortality. Lung endothelial dysfunction is a hallmark of sepsis-induced acute lung injury (ALI), where inflammation plays an important role. We have recently found a rise in the plasma cell-free RNA including miRNAs in sepsis. These extracellular (ex) RNAs are released from host cells as well as invading bacteria and closely correlated with sepsis severity. My research interest is to test the hypothesis that ex-miRNA plays an important role in acute lung injury during polymicrobial sepsis. The research is supported by NIH R35GM124775.

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