BIOGRAPHICAL SKETCH

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NAME: Jennifer E. Van Eyk

eRA COMMONS USER NAME (credential, e.g., agency login): JVANEYK1

POSITION TITLE: Director, Advanced Clinical BioSystems Research Institute; Professor of Medicine, Cedars-Sinai Medical Center

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Waterloo, Waterloo, Canada	B.Sc.	1982	Biology & Chemistry
University of Alberta, Edmonton, Alberta, Canada	Ph.D.	1991	Biochemistry
University of Heidelberg, Heidelberg, Germany	P.D. Fellow	1992	Physiology/Cardiology
University of Alberta, Edmonton, Alberta, Canada	Res. Assoc.	1995	Biochemistry/Muscle
University of Illinois at Chicago, Chicago, IL USA	P.D. Fellow	1996	Biochemistry/Cardiology

A. Personal Statement

The new Advanced Clinical BioSystems Research Institute's motto, "from discovery to patient care," underlies the essence and foundation of our research. Our research focuses i) on understanding the molecular mechanisms underlying acute and chronic disease and the development of precision therapies and ii) in the development of clinically robust circulating biomarkers for personalization of medical care. The central philosophy of our laboratory is that compelling biological and clinical questions drive innovation through development, optimization and adaption of proteomic technologies, functional analysis, and large-scale data handling. We specialize in developing robust technological pipelines and automation systems to precisely quantify proteins and their post-translational modifications (PTM) in disease pathways. This includes understanding interplay between competing PTMs like phosphorylation and multiple oxidative modifications. Our group uses automation in sample preparation to allow for high-throughput and robust MS analysis, which includes discovery and the novel approach, Data independent acquisition-MS (also known as SWATH) that allows complete and reproducible analysis of 1-1000s of proteins in 1000s of samples. As well, absolute quantification of key targets can be quick and cost effective via ultra-sensitive ELISA platforms or multiple reaction monitoring assay, a targeted MS-based quantitative method, for tracking multidimensional signaling pathways in tissue and body fluids. We are now expanding to open an academic MS service lab and CAP/CLIA clinical chemistry laboratories (The Cedars Sinai Precision Biomarker Labs) to facilitate the goals of our institute.

B. Positions and Honors

Positions and Employment

2017-now Co-Director, The Cedars-Sinai Precision Biomarkers Labs, Cedars-Sinai Medical Center, CA
2014–now Professor, Heart Institute & Department of Medicine, Cedars-Sinai Medical Center, CA
2014–now Director, Basic Research for the Barbara Streisand Women's Heart Center, Cedars-Sinai, CA
2014–now Director, Advanced Clinical Biosystems Institute, Cedar Sinai Medical Center, CA
2014–now Adjunct Professor, Medicine, Johns Hopkins University, MD
2013–2017 Scientific Advisor, ImmunArray and Veracis Incorporated, Johns Hopkins University, MD
2007–2014 Professor, Medicine and Biol. Chem. and Biomedical Engineering, Johns Hopkins University, MD
2007–2014 Director, Johns Hopkins CTSA/ITCR Biomarker Development Group, Johns Hopkins University
2003–now Director, NHLBI Innovation Proteomic Group, Johns Hopkins University, MD
2003–2007 Assoc. Prof. Depts. Medicine, Biomed. Eng., and Biol. Chem., Johns Hopkins University, MD
2001–2003 Associate Professor (Tenured) Department Physiology, Queen's University, Kingston, Canada
1996–2001 Assistant Professor, Depart. Physiology (cross-appt. Biochemistry), Queen's University, Canada

Other Experience

2017-now Early Career Committee, chair, International HUPO
2017-now Past-Chair, Disease/Biology Initiative, International HUPO
2015-now Research Council Member, American Heart Association CA
2014-2017 Chair, Disease/Biology Initiative, International HUPO
2010 Expert lecturer for Institute of Medicine (National Academy) rare and orphan diseases research
2009–now International Society of Heart Research (ISHR), North American Section, Secretary
2009–now Consulting Editor, Circulation Research
2009–2012 Research Council Member, American Heart Association Mid-Atlantic
2009–2010 Institute of Medicine (National Academy), committee member on the Quantification of Biomarkers as Surrogate Endpoints
2008-2012 Scientific Advisory Board for Plasma Proteome Inc.
2008–2012 NHLBI grant review panel permanent member (MIM)
2007–now Senior Editorial Board, Proteomics, Clinical Applications
2007–2010 American Heart Association Functional Genomics and Translational Science, chair (2008-10)
2007 Clinical Proteomics: from Diagnosis to Therapy (editors: J.E. Van Eyk and M. Dunn) Wiley-VCH Publishers.
2006 Member of NHLBI Strategic Plan Cardiovascular Working Group on Emerging Technologies
2005 Co-chair NHLBI workshop entitled "The next Step: population studies in the "OMIC" age"
2004–2008 American Heart Association Basic Council Member-At-Large
2003 Genomic and Proteomic Analysis of Cardiovascular Disease: molecular mechanism, therapeutic targets and diagnostics (editor, J. Van Eyk and M. Dunn) Wiley-VCH Publishers.
2003–now ISHR (International Society of Heart Research) North American Section Board Member
2002–now Editorial Board, Proteomics and Associate Editor, Technical Brief sections, Proteomics
2000–2009 Editorial Board member, Circulation Research

Honors

2017 Molecular and Cellular Proteomics Lectureship Award, Mol. Cell. Proteomics.
2017 The Analytic Scientist's Power list: 10 Top 10s (4th ranked international proteomic/metabolomic) https://theanalyticalscientist.com/power-list/2017/
2015 International human proteome organization. HUPO Translational Science Award.
2014 American Heart Association (AHA) Functional Genomics Translational Biology Council Medal of Honor for exceptional science
2014 Inaugural Erika Glazer Endowed Chair in Womens' Heart Health, Cedar Sinai Medical Center, CA
2013 American Heart Association Functional Genomics Translational Biology Council award (1st awarded) for exceptional contribution to the society.
2013 American Heart Association Top 10 research papers in Functional Genomics and Translational Research for Zhang *et. al*., *Circ. Res*. *2012:***126**:1828.
2012 The Johns Hopkins University David Levine Mentorship Award for School of Medicine
2011 American Heart Association Top 10 research papers in Functional Genomics and Translational Research for Cammarato A *et. al., Plos 1*, **6**:e18497.
2011 The David Grimm Lecture, Ottawa Heart, Ottawa, Canada
2010 Annual Dean's lecture, School of Medicine, Johns Hopkins University
2010 Fellow of the International Society of Heart Research (FISHR)
2010 Fellow of the American Heart Association (FAHA) for Functional Genomics and Translational Biology Council
2009 American Heart Association top 10 research papers for Matt *et. al., Circ*. 2009*:***120**:526.
2001 Heart and Stroke of Ontario Career Investigator Award and Basmajian Top Research Award (Queen's University, Faculty of Medicine); Fellow of American Heart Association (FAHA) for the Basic Science Council
2000 American Heart Association Top 10 research papers in 2000 for Murphy *et. al., Science*, 2000; **287**:488.
1998 Canadian Foundation for Innovation Scholar; Chancellor's Research Award, Queen's University, Canada; PREA (Premier's Research Excellence Award), Canada
1996–2001 Heart and Stroke Foundation of Canada Young Principal Investigator Award, Canada

C. Contributions to Science (26 patents or patent applications; 266 articles/reviews+14 editorial+25 chapters+2 books, 10 in 2018)

1. *Dissecting the mechanism of disease-induced modifications of cTnI and cardiac myofilament proteins and diagnostic value. These modified forms impacted the detection and quantification of circulating cTnI is the gold standard diagnostic for myocardial infraction (MI) which has been greatly impacted by the discovery of the disease-induced modifications. This resulted in many generations of cTnI clinical assay development and importantly, resulted in the concept of using disease-induced modifications to increase specificity and personalize diagnostics.*
   a. McDonough J, Arrell K, **Van Eyk JE.** Troponin I degradation and covalent complex accompanies myocardial ischemia/reperfusion injury. *Circ. Res.* 1999; **84:9** PMID: 991577. Comment: Solaro RJ. *Circ Res* 1999;**84**:122-4.This is the first paper to show the cTnI is specifically proteolyzed in ischemia reperfusion in animal and human and has broad implication to contractility and diagnostics.
   b. Murphy AM, Kogler H, Georgakopoulos D, McDonough JL, Kass DA, **Van Eyk JE.** Marban E. Transgenic mouse model of stunned myocardium. *Science* 2000; **287**:488. PMID:10642551. *Top 10 research advances for 2001 by the American Heart Association*. Showed that the selected disease-induced degradation of cTnI was sufficient to reduce heart muscle force by 50% recapitulating stunning.
   c. Fert-Bober J, Giles JT, Holewinski RJ, Kirk JA, Uhrigshardt H, Crowgey EL, Andrade F, Bingham CO 3rd, Park JK, Halushka MK, Kass DA, Bathon JM, **Van Eyk JE**. Citrullination of myofilament proteins in heart failure. *Cardiovasc Res*. 2015;**108**:232-42. PMCID: 4614685 Comment**:** Solaro RJ, *Cardiovasc Res.* 2015:**108**:212-4. First paper to show this modification occurs in human hearts and affects contractility.

2. *Signaling and redox protein modification in the mitochondria response in disease injury. Quantitative analysis of PTMs provides novel insights in druggable pathways for heart disease.*
   a. Arrell DK, Elliott ST, Kane LA, Guo Y, Ko YH, Pedersen PL, Robinson J, Murata M, Murphy AM, Marban E, **Van Eyk JE.** Proteomic Analysis of Pharmacological Preconditioning. Novel Protein Targets Converge to Mitochondrial Metabolism Pathways. *Circ. Res.* 2006; 99:5:706. PMID:16946135. Comment: Cohen RA, McComb ME. *Circ Res.* 2006;**99:**663-5. First to show the cardiac mitochondrial proteome is rapidly phosphorylated. This remarkably includes ATP synthase located in the inner mitochondrial membrane implying mito-localized kinase signaling.
   b. Wang SB, Foster DB, Rucker J, O'Rourke B, Kass DA, **Van Eyk JE**. Redox Regulation of Mitochondrial ATP Synthase: Implications for Cardiac Resynchronization Therapy. *Circ. Res*. 2011;**109**:750-7. PMCID: 3500591. Comment: Zweier JL, Chen CA, Talukder MA *Circ Res.* 2011;**109**:716-9. First to demonstrate that cardiac resynchronization therapy (CRT) increases ATP synthase activity via alterations in oxidative posttranslational modifications, in addition to its phosphorylation status. This is conceptually important and suggest that assessment/control of a patient's mitochondrial antioxidant defense system may be required for personalize CRT.
   c. Lee DI, Zhu G, Sasaki T, Cho GS, Hamdani N, Holewinski R, Jo SH, Danner T, Zhang M, Rainer PP, Bedja D, Kirk JA, Ranek MJ, Dostmann WR, Kwon C, Margulies KB, **Van Eyk JE,** Paulus WJ, Takimoto E, Kass DA. Phosphodiesterase 9A controls nitric-oxide-independent cGMP and hypertrophic heart disease. *Nature.* 2015; **519:472**. PMCID: 4376609. This paper showed that PKG-protection against HF development that unlike PDE5, PDE9A can regulate cGMP signaling independent of the nitric oxide pathway, and its role in stress-induced heart disease suggests potential as a therapeutic target. Comments: Kuhn M. *Nature* 2015; **519:416**-7 and Bray N. *Nat Rev Drug Discov.* 2015;**14**:310.
   d. Chung HS, Kim GE, Holewinski RJ, Venkatraman V, Zhu G, Bedja D, Kass DA, **Van Eyk JE**. Transient receptor potential channel 6 regulates abnormal cardiac S-nitrosylation in Duchenne muscular dystrophy. *Proc Natl Acad Sci U S A*. 2017;**114**: E10763-E10771. PMID:29187535 This work showed SNOylation is dramatically increased in DMD and genetic deletion of Trpc6, calcium channel reversed PTM status and improved cardiac function which maybe a new drug target.

3. *Elucidation of novel disease pathways opens up possibly of new mechanistic therapeutic models*.
   a. Agnetti G, Halperin VL, Kirk J, Chakir K, Guo Y, Lund L, Nicolini F, Gherli T, Guarnieri C, Caldarera CM, Tomaselli GF, Kass DA, **Van Eyk JE.** Desmin Modifications Associate with Amyloid Oligomers Deposition in Heart Failure. *Cardiovasc. Res.* 2014; **102:24**. PMCID: 3958618. This is first paper to report on a new mechanism underlying cardiac toxicity that is based on GSK 3beta, disease-induced PTMs of

desmin. The phosphorylation of desmin drives accumulation of amyloid-like oligomers in heart failure. CRT decreases the amyloid-like oligomers as did a targeted small molecule.

b. Kirk JA, Holewinski R, Kooij V, Tunin RA, Witayavanitkul N, de Tombe PP, Gao W, **Van Eyk JE**, Kass DA. Cardiac resynchronization sensitizes the sarcomere to calcium by reactivating GSK3 beta. *J. Clinical Investigations,* 2014;**124**:129. PMCID: 3470471 Proteomics identified the underlying signaling pathway that explains the improved contractile function induced by CRT is due, in part, to activation of GSK3b and its unexpected phosphorylation of Z and M line proteins – indicating that these structural/organizational proteins have an important role in contraction.

c. Kirk JA, Chakir K, Lee KH, Edward Karst E, Holewinski RJ, Piront G,. Tunin RS, Pozios I, Abraham TP, de Tombe, Rockman HA, **Van Eyk JE,** Craig R, Farazi TG, Kass DA, Pacemaker Induced Transient Asynchrony Suppresses Heart Failure Progression, *Science Translation,* 2015*,*7:319ra207. PMCID: 4858435. This paper shows that there is a dramatic improvement in ejection fraction of patients with heart failure using a novel heart pacing protocol. Using proteomics, we show that it is due to a realignment and re-establishment of the correct molar ratio (stoichiometry) of the myofilament proteins.

d. Wang S, Venkatraman V, Crowgey EL, Liu T, Fu Z, Holewinski RJ, Ranek MJJ, Kass DA, O'Rourke B, **Van Eyk JE.** Protein S-Nitrosylation Controls Glycogen Synthase Kinase 3β Function Independent of its Phosphorylation State. *Circ Res*. 2018, *In press*. PMID:29563102. GSK 3beta is regulated by phosphorylation which modulates metabolism can also be independently regulated by SNOylation. SNOylation of GSK 3beta drives this kinase to the nucleus where it activates the DNA repair response.

4. *Tool development for protein quantification and their post-translational modification. There is a need to develop quantitative workflows and tools for the broader scientific community. Here are some examples of tools our group has provided*.

a. Zhang P, Ji W, dos Remedios CG, Kirk JA, Kass DA, **Van Eyk JE***, Murphy AM* (*equal contribution), Multiple reaction monitoring to identify site-specific troponin I phosphorylated residues in the failing human heart. *Circ.* 2012**; 126:182***.* PMCID:3733556. Comment: Cordwell SJ, White MY, *Circ.* 2012;126:1803-7. This is the first targeted MS-based assay that simultaneously provide absolute quantification of cTnI and each of its many phosphorylation sites – showed the PKA sites were increased in heart failure but only accounted for less than 5% of all TnI being modified. Tool is available to the community. Patented.

b. Chung HS, Murray CI, Venkatraman V, Crowgey EL, Rainer PP, Cole RN, Bomgarden RD, Rogers JC, Balkan W, Hare JM, Kass DA, **Van Eyk JE.** Dual labeling biotin switch assay to reduce bias derived from different cysteine subpopulations: A method to maximize S-nitrosylation detection *Circ. Res.* 2015; **117:846**-57. PMCID: PMC4979977. Comments: Seth D, Stamler JS *Circ Res.* 2015;**117**:826-9.This current paper used two different MS-based labels to uncover multiple unique subpopulations of SNO-cysteine residues. The novel concept of the presence of unique and distinctive SNO susceptible proteins within a cell is paradigm changing.

c. Zhang S, Raedschelders K, Santos M, **Van Eyk JE.** Profiling B-type Natriuretic Peptide cleavage peptidoforms in human plasma by capillary electrophoresis with electrospray ionization mass spectrometry *J Proteome Res*. 2017; **16:4515**-4522. PMID:28861997. BNP, is a circulating hormone, a heart failure biomarker and can be used for treatment but, plasma enzymes rapidly cleave circulating BNP rapidly into inactive fragments making it challenging to monitor. We developed a MS method that provides a quantitative multipoint BNP proteolytic profile within an hour allowing for assess the potential relation between plasma-based BNP proteolysis. 2 Patents. Journal cover.

d. Fu Q, Kowalski MP, Mastali M, Parker SJ, Sobhani K, van den Broek I, Hunter CL, **Van Eyk JE**. Highly Reproducible Automated Proteomics Sample Preparation Workflow for Quantitative Mass Spectrometry. *J Proteome Res*. 2018;1**7**:420-42 PMID:29083196. We developed and commercialized a complete hands-free, reproducible and quantification workstation for MS peptide sample preparation.

List of Selected Published Work:
https://www.ncbi.nlm.nih.gov/sites/myncbi/1VsYqQYH8535l/bibliography/48183272/public/?sort=date&direction=ascending

D. Additional Information: Research Support and/or Scholastic Performance

Ongoing Research Support

P01HL112730 (Gottlieb)**	07/01/2014-12/31/2018	1.2 months

NIH-NHLBI
Mitochondrial Quality in Cardioprotection: Overcoming Co-Morbidities
The major goals of this project are to define biochemical signatures of autophagy and cardioprotection.
Role: Co-Investigator
Overlap: none

1U01 DK103260-01 (Anger/**Van Eyk**)** 07/01/2014-06/30/2019 0.24 months
NIH-NIDDK
Microbiome and Proteome as Predictive Biomarkers of UCPPS
The major goals of this project are to identify the prostatitis/chronic pelvic pain syndrome (UCPPS)-associated proteome in plasma.
Role: Co-Investigator – To carry out core lab protein quantification on 600 plasma samples.
Overlap: none

1U54NS091046-01 (Svendsen/**Van Eyk**) 06/01/2014-05/30/2019 0.3 months
NIH-NINDS
Neuron and Glial Cellular Signatures from Normal and Diseased iPS Cells
The major goals of subproject are to assess proteome changes in iPSC and iPSC-derived motor neurons derived from control and patients with SLA and SMA.
Role: Co-PI – To carry out multi-omics on patient derived IPSC motor neurons to understand disease pathways in ALS and SMA.
Overlap: none

AHA Grand Challenge (Rader/**Van Eyk**) 07/01/2015-06/30/2019 0.12 months
American Heart Association
Interrogation of the Plasma Proteome for the Development of New Biomarkers and Therapeutic Targets for Cardiovascular Disease
The major goals of this project are to identify and validate cardiovascular biomarkers across gender and ethnicity.
Role: Co-PI – To develop new plasma markers for risk assessment.
Overlap: none

R01 AT001576 (Lu/**Van Eyk**) 07/01/2015-06/30/2019 0.24 months
NIH
DHHSP-S-Adenosylmethionine in Fatty Liver Disease
The major goals of this project are to identify the role of sumoylation and methylation on SAME treatment in liver disease.
Role: Co-Investigator – To determine mechanisms underlying progression to liver damage and interplay of PTMs.
Overlap: none

16W81XWH-16-1-0592 (Marban/**Van Eyk**) 06/01/2016-05/30/2019 1.2 months
DOD
Heart Failure With Preserved Ejection Fraction: Mechanisms and Novel Therapeutics
The major goals of this project are to identify the underling mechanisms in stem cell based therapy.
Role: Co-PI – To determine the post-transcriptional regulation induced by cardiac derived stem cells and their exosomes.
Overlap: none

W81XWH-15-PRCRP-TTSA (Kanodia/**Van Eyk**)*** 09/01/2016-08/31/2019 1.2 months
DOD
HMGB1 and Its Isoforms as Biomarkers for Mineral Fiber Exposure and MM Detection
Goals: In this project, we are developing a quantitiave and specific mass spectrometry based assay for HMGB1, a secreted nuclear protein linked to cancer. As well, we will expand the assay to include disease-induced modifications which will increase specificity.
Role: Co-PI

Overlap: None

Proteomic assays for TOPMED program of NHLBI (Van Eyk)*** 09/01/2016-08/30/21
NIH-NHLBI
TOPMED (Trans-Omics for Precision Medicine Program)
Goals: To carry out large scale protein precise quantification for population and epidemiological studies.
Overlap: none

1R01HL132075-01A (Gottlieb/**Van Eyk**) 12/15/2016-11/30/2020 1.2 months
NIH-NHLBI
Mitochondrial Turnover in the Human Heart
The major goals of this project are to elucidate the dynamic regulation of mitochondria turnover and cardiac failure
Role: Co-Investigator – To determine regulation of mitochondria protein synthesis and its ties to nuclear and mitochondrial transcriptome.
Overlap: none

2 R01 AR050026-12A1 (Bathon/**Van Eyk**) 06/01/2017-05/31/2022 0.12 months
NIH/NIAMS
Inflammation and Cardiovascular Disease in Rheumatoid Arthritis
The overall goal of this project is to understand the causes of the increased risk of myocardial dysfunction in rheumatoid arthritis. Our aims are: 1) to determine whether baseline myocardial inflammation and/or microvascular dysfunction in patients with RA are risk factors for myocardial remodeling and dysfunction; 2) if RA therapies reduce myocardial inflammation; and 3) if antibodies to citrullinated myocardial antigens are associated with myocardial inflammation, microvascular dysfunction and myocardial remodeling in patients with RA.
Role: Co-Investigator
Overlap: none

Neoteryx Grant (**Van Eyk**/McGovern) 09/01/2017-08/31/2019 0.12 months
Neoteryx
Remote Volumetric Absorptive Microsampling to Facilitate Mass Spectrometry Based Proteomics in Patients with Inflammatory Bowel Disease to Identify Biomarkers of Inflammation (REVAMP IBD)
The goals of this study are to evaluate the utility of high frequency, low-burden, remote proteomic based disease monitoring amongst patients with IBD starting new therapy to identify serum biomarkers of mucosal inflammation and predictors of response to therapy
Role: PI
Overlap: None

1R21HL140274-01 (Korley/**Van Eyk**) 09/15/2017-08/31/2019 0.12 months
NIH
Predicting Incident Stroke Using Blood Biomarkers of Brain Injury
The goals of this study are to carry out protein quantification using the ultra-high sensitivity ELISA platform, Quanterix, for neurofilament L (NF-L) and brain derived neuron factor (BDNF). The hypothesis is that these proteins will be altered in the blood with incident stroke. We will run ~700 plasma samples and provide the concentration and %CV for each sample.
Role: PI
Overlap: None

R01HL111362 (Herrington/**Van Eyk**) 04/01/2018-01/31/2022 1.20 months
NIH-NHLBI
Genomic and Proteomic Architecture of Atherosclerosis
The overall goals of the study are to carry out using mass spectrometry of I) total protein quantification on aortas obtained from 100 individuals with early atherosclerosis, ii) determine the post-translational modification (e.g. Citrullination) and protein isoforms in targeted and full proteome including the secreted proteins and iii)

develop targeted assays of key analytes for use as circulating biomarker for risk of development of athersoclerosis in human populations.
Role: Co-PI
Overlap: none

1R01AA026759-01 (Lu) 05/15/2018-01/31/2023 0.24 months
NIH-NHLBI
Methionine Adenosyltransferase Alpha 1 Alcoholic Liver Disease
The overall goals of the study are to address the novel role of methionine adenosyltransferase α1 (MATα1) in regulating cytochrome P450 2E1 (CYP2E1) expression and mitochondrial function, two highly relevant targets in ALD, that will advance our understanding of the pathogenesis of ALD and may offer new therapeutic strategies.
Role: Co-Investigator
Overlap: none

Smidt Heart Institute Grant (**Van Eyk**) 09/01/2018-07/31/2019 0.00 months
Smidt Heart Institute
Takotsubo Proteomic Registry: mechanistic signatures of disease and disease status
The overall goals of this study are to provide preliminary data for extra-mural funding solicitation for ongoing follow-up and repeated Mitra sampling with proteomic analysis.
Role: PI
Overlap: none

Smidt Heart Institute Grant (Chung/Emerson/**Van Eyk**)09/01/2018-07/31/2019 0.00 months
Smidt Heart Institute
The Role of Inflammatory Cytokines in Vasoplegic Shock within the Cardiac Device Population
The overall goals of this study are to better determine the relationship between BK, NO, the inflammatory state, and observed physiologic derangements combined under the general term of vasoplegic shock.
Role: Co-PI
Overlap: none