Vasculata 2022
TUESDAY POSTERS

ANGIOGENESIS

T01 – Investigating the roles of TMEM16F lipid scramblase in angiogenesis

Ke Shan[1], Trieu Le[1], Pengfei Liang[1], Yang Zhang[1], Huanghe Yang[1]
[1]Duke University, Durham, USA

- TMEM16F lipid scramblase is functionally expressed in endothelial cells (ECs).
- TMEM16F controls in vitro tube formation and in vivo angiogenesis.
- Mechanosensitive calcium-permeable channel PIEZO1 and TMEM16F are functionally coupled in ECs.

T02 – The role of CD44 variants in coronary vascular rarefaction and the development of left ventricular diastolic dysfunction in HFpEF.

Katie Anne Fopiano[1], Yanna Tian[1], Vadym Buncha[1], Liwei Lang[1], Zsolt Bagi[1]
[1]Medical College of Georgia at Augusta University, Augusta, Georgia, USA

- CD44 variants may be linked to an abnormal angiogenic response in HFpEF.
- Coronary microvascular rarefaction may lead to a perfusion deficit in HFpEF.
- CD44 could play a role in microvascular endothelial dysfunction in HFpEF.

T03 – Macrophage IL-1β Drives Angiogenesis During Wound Healing

Sheila Sharma[1], Chris Mantsounga[1], Jade Neverson[1], Cadence Lee[1], Julia Pierce[1], Elizabeth Amelotte[1], Rachel Carley[1], Celia Butler[1], Gaurav Choudhary[2], Alan Morrison[2]
[1]OSRI/Brown University, Providence, Rhode Island, USA; [2]OSRI/Brown University/Alpert Medical School, Providence, Rhode Island, USA

- Early infiltrating inflammatory macrophages may help prime angiogenesis during wound healing.
- Macrophage VEGF-A expression is dependent on IL-1β expression in the early inflammatory state.
- Downstream effectors of IL-1β signaling may be required to restore macrophage VEGF-A expression.

ATHEROSCLEROSIS

T04 – Cholesterol activated SREBP1/LGALS3 dyad that is epigenetically blockable.

Jing Li[1]
[1]Department of Surgery, University of Virginia, Charlottesville, Virginia, USA

- Cholesterol-stimulated SREBP1 positively regulates LGALS3 expression in the smooth muscle cells.
- JQ1, as a pan inhibition of BETs, abolishes cholesterol-stimulated SREBP1/LGALS3 protein production.
- BRD2 co-immunoprecipitates with SREBP1's transcription active domain and the Lgals3 promoter DNA.

T05 – Small Molecule PCSK9 Inhibitors Attenuate Inflammatory Response and Decrease Foam Cell Formation in iPSC-derived Vascular Smooth Muscle Cells

Kevin Shores[1], Benny Evison[2], Alexandra Suchowerska[2], James Bonnar[2], Charles Gersbach[1], George Truskey[1]
[1]Duke University, Durham, North Carolina, USA; [2]Nyrada Inc., Gordon, Australia

- Overexpressing PCSK9 in iPSC-derived ECs and SMCs using dCas9-VP64 CRISPR activation
- Treating iPSC-derived SMCs with eLDL and TNFα to simulate atherosclerosis in vitro
- Treating PCSK9 overexpressing iPSC-derived SMCs with novel small molecule PCSK9 inhibitors

T06 – The effects of disturbed flow on endothelial dysfunction and endocytosis of LDL

Jason Irei[1], Javier Lozano-Gerona[1], Kai Hirayama[1], William Boisvert[1]
[1]University of Hawaii John A. Burns School of Medicine, Honolulu, Hawaii, USA

- Disturbed flow significantly alters endothelial gene expression.
- Endothelial cells uptake greater amounts of LDL under disturbed flow compared to healthy flow.
- Disturbed flow and LDL treatment reduced the abundance of acidic vesicles in endothelial cells.

T07 – Endothelial Nck adaptors controls the atherosclerosis progression

Cyrine Ben Dhaou[1], Mabruka Alfaidi[1], Wayne Orr[1]
[1]LSUHS, Shreveport, Louisiana, USA

- Global Nck1 knockout mice have less atherosclerosis compared to endothelial Nck2 knockouts
- Endothelial specific-Nck1 knockout mice exhibited reduced atherosclerosis
- Mass spectrometry data analysis have identified SLC1A2 to directly bind to NCK1 after shear stress

T08 – Impaired Putrescine Synthesis in vSMCs Causes Phenotypic Modulation and Drives ECM Degradation

Chad Stroope[1], Kyle McGee[1], Dhananjay Kumar[1], Arif Yurdagul[1]
[1]LSU Health Shreveport, Shreveport, Louisiana, USA

- Impairments in putrescine synthesis are observed as atherosclerosis progresses.
- Loss of putrescine in vSMCs leads to phenotypic modulation.
- Vascular smooth muscle cells deficient in putrescine synthesis show elevation in ECM degradation.

T09 – Immune mediated mechanisms of macrophage Rac1-IL-1β signaling leading to atherosclerotic calcification

Rachel Carley[1], Cadence Lee[2], Christopher Mantsounga[2], Shelia Sharma[2], Jade Neverson[3], Julia Pierce[1], Elizabeth Amelotte[3], Celia Butler[3], Gaurav Choudhary[2], Alan Morrison[2]
[1]Brown University/ Providence VA, Providence, Rhode Island, USA; [2]Providence VA/Brown University, Providence, Rhode Island, USA; [3]Providence VA, Providence, Rhode Island, USA

- Atherosclerosis
- Vascular Calcification
- Macrophage

CARDIOVASCULAR DISEASE

T10 – Endothelial lipid droplets link metabolic syndrome to blood pressure elevation

Boa Kim[1], Soon Tang[1], Wencao Zhao[1], Ayon Ibrahim[1], Yifan Yang[1], Emilia Roberts[1], Jian Li[1], Rick Assoian[1], Garret FitzGerald[1], Zoltan Arany[1]
[1]University of Pennsylvania, Philadelphia, USA

- High fat consumption leads to the accumulation of lipid droplets in the endothelium.
- Lipid droplet accumulation in endothelium leads to endothelial dysfunction and hypertension.
- Lipid droplets activates an inflammatory signaling cascade that suppresses eNOS and NO production.

T11 – Autoimmune valvular carditis requires endothelial cell TNFR1 expression.

Jessica Faragher[1], Jennifer Auger[1], Victoria Osinski[1], Bryce Binstadt[1]
[1]Center for Immunology, University of Minnesota Medical School, Minneapolis, Minnesota, USA

- Endothelial specific TNFR1 facilitates mitral valvular carditis in systemic autoimmune disease.
- Endothelial TNFR1 facilitates a variety of pathological processes in the mitral valve.
- Mitral valve disease remodeling is reversible in the absence of endothelial TNFR1.

T12 – Therapeutic development for targeting cadherin-11 to treat heart valve disease.

Oluwalade Ogungbesan[1], W. David Merryman[1]
[1]Vanderbilt University, Nashville, Tennessee, USA

- Cadherin-11 has been shown to be a valuable target for treating heart valve disease
- Preventing expression of cadherin-11 using siRNA therapeutic strategies
- Use of dimethyl celecoxib analogues to target cadherin-11 in heart valve interstitial cells

T13 – Vascular Endothelial Barrier Protection Prevents Atrial Fibrillation by Preserving Cardiac Nanostructure

Louisa Mezache[1], Andrew Soltisz[1], Scott Johnstone[2], Brant Isakson[3], Rengasayee Veeraraghavan[1]
[1]The Ohio State University, Columbus, Ohio, USA; [2]Virginia Tech, Blacksburg, Virginia, USA; [3]University of Virginia, Charlottesville, Virginia, USA;

- Vascular dysfunction promotes cardiomyocyte intercalated disc (ID) remodeling
- ID remodeling promotes conduction slowing and increased arrhythmia inducibility
- Protecting the vascular barrier prevents arrhythmias

T14 – A potential role of arhGEF17 in hemorrhagic intracranial aneurysms.

Zaneta Markowska[1], Jin Li[1], Lisa Post[1], Heather Ferris[1], Avril Somlyo[1]
[1]University of Virginia, Charlottesville, Virginia, USA

- ArhGEF17 may play a role in endothelial cell permeability and smooth muscle cell contractility.
- ArhGEF17 is potentially involved in the regulation of vascular tone.
- Defective arhGEF17 signaling may underlie the development of hemorrhagic intracranial aneurysms.

T15 – Modeling CADASIL, a hereditary form of vascular dementia affecting mural cells in the cerebrovasculature

Juan Cerda III[1], Joshua Wythe[1], Hamed Jafar-Nejad[1]
[1]Baylor College of Medicine, Houston, Texas, USA

- Assessing mice behavior differences in locomotor and cognitive function.
- Notch3 loss behavior data suggest no differences in motor and cognititve function in mice.
- 3D imaging and quantification of the cerebrovasculature can serve as an important tool to study CADASIL phenotypes.

T16 – Fetuin A alters crystallinity and morphology of calcifications to mimic biological sex in a model for calcific aortic valve disease

Raphaela Allgayer[1], Diego Mantovani[2], Marta Cerruti[1]
[1]McGill University, Montreal, Canada; [2]Laval University, Quebec, Canada

- Higher levels of fetuin A in women could cause sex-differences in calcific aortic valve disease.
- We incubated collagen hydrogels in simulated body fluid with different levels of fetuin A.
- Different fetuin A levels replicate sex-differences in mineral amount, crystallinity and morphology.

T17 – Endothelial c-Myc knockout triggers cardiac dysfunction through modulation of contractility mechanisms and increase in inflammation.

Jacqueline Freire Machi[1], Isabella Altilio-Bove[2], Yue Qi[2], Alejo Morales[2], Claudia Rodrigues[1]
[1]Florida Atlantic University, Boca Raton, Florida, USA; [2]University of Miami, Miami, Florida, USA

- Cardiovascular disease is the leading cause of death worldwide.
- Endothelial cells play and essential role in organ function and tissue homeostasis.
- The mechanisms by which endothelial dysfunction contributes to cardiovascular disease remain elusive

T18 – Loss of Transforming Growth Factor Beta2 in Postnatal Aorta Causes Thoracic Aortic Aneurysm and Dissection

Mengistu Gebere[1], Mrinmay Chakrabarti[1], John Johnson[1], Mohamad Azhar[1]
[1]University of South Carolina, Columbia, South Carolina, USA

- Loss of Tgfb2 in postnatal SMC leads to aortic aneurysm and dissection, and aortic rupture
- SMC-specific deletion of TGFβ2 results in increased TGFβ signaling as a compensatory response.
- Increased SMC proliferation and myeloid cells infiltration contributes to aneurysm in Tgfb2CKO mice

T19 – Investigating the effects of diabetic conditions on the progression of calcific aortic valve disease

Maristella Donato[1], Taleb Ahsan[1], Subramanian Dharmarajan[2], Mei Speer[1], Elizabeth Leaf[1], Marta Scatena[1], Cecilia Giachelli[1]
[1]University of Washington, Seattle, Washington, USA; [2]University of California, San Francisco, California, USA

- Diabetes promotes the progression of calcific aortic valve disease (CAVD), a common valvulopathy
- We aim to assess the effect of hyperglycemic conditions on the progression of CAVD
- Several inflammatory, immune and cardiac genes are dysregulated in a mouse model of diabetic CAVD

T20 – Irf8 and securinine in abdominal aortic aneurysm-seeking pharmacological treatment

Yae Hyun Rhee[1], Joshua Spin[1], Alicia Deng[1], Colwyn Headley[1], Philip Tsao[1]
[1]Stanford Cardiovascular Institute, Stanford University School of Medicine, Stanford, USA

- Abdominal aortic aneurysm is a disease of progressive dilatation of the aortic diameter.
- To date, no pharmaceutical treatments are available; surgical repair is offered to large aneurysms.
- Irf8 inhibition by securinine may inhibit aortic tissue inflammation and aneurysm growth.

T21 – Impact of gonadal sex and chromosomal sex at single cell resolution in hyperoxic lung injury

Manuel Cantu Gutierrez[1], Abiud Cantu[1], Krithika Lingappan[1]
[1]Children's Hospital of Philadelphia, Philadelphia, Pennsylvania, USA

- Male sex is a risk factor in lung injury of preterm births and the mechanisms are poorly understood.
- Our study uses scRNA-Seq to distinguish the impact of chromosomal sex versus gonadal sex.
- We observed acap cell expansion with the largest changes in mice with male testis (XXM and XYM).

T22 – Female Mice Are Resistant to Impaired Parenchymal Arteriole TRPV4 Activation and Inward Remodeling During Hypertension

Laura Chambers[1], William Jackson[1], Anne Dorrance[1]
[1]Michigan State University, East Lansing, Michigan, USA

- Cerebral parenchymal arterioles (PAs) are highly dependent on TRPV4 channel activation for dilation.
- Hypertensive male mice have impaired TRPV4 function and inward remodeling in PAs.
- Hypertensive female mice are protected against impaired PA TRPV4 function and inward remodeling.

T23 – Endothelial dysfunction drives aneurysm development in Marfan syndrome

Anna Cantalupo[1], Keiichi Asano[1], Lauriane Sedes[1], Sergey Dikalov[2], Carmen Halabi[3], Robert Mecham[3], Ravi Iyengar[1], Francesco Ramirez[1]
[1]Icahn School of Medicine at Mount Sinai Dept Pharmacol Sciences, New York, New York, USA; [2]Vanderbilt University Medical Center, Nashville, Tennessee, USA; [3]Washington University Dept of Cell Biology and Physiology and Pediatrics, St. Louis, Missouri, USA

- Altered endothelial-derived signaling triggers aneurysm disease in Marfan syndrome
- Endothelial fibrillin-1 has a key role in preserving aortic ultrastructure
- Endothelial fibrillin-1 is required to maintain proper EC-SMC communication.

INFLAMMATION

T24 – The Role of Complement in Vascular Dysfunction Associated with Monocrotaline Induced Pulmonary Hypertension

James Pawlak[1], Alexa Smith[1], Alaeddin Abukabda[1]
[1]Lake Erie College of Osteopathic Medicine, Erie, Pennsylvania, USA

- Complement system and vascular dysfunction
- Pulmonary hypertension may be complement driven
- Monocrotaline and microvascular dysfunction

T25 – The Atypical Angle: Ongoing Explorations into Understanding Atypical MAPK p38 in Pulmonary Inflammatory Disease

Jeremy Burton[1], Neil Grimsey[1]
[1]University of Georgia, Athens, Georgia, USA

- Mitogen-activated protein kinase (MAPK) p38 mediates of vascular edema and inflammatory signaling
- There is a need to explore mechanisms that selectively regulate pathological p38 signaling pathways
- Atypical p38 activation may play a key role in vascular disruption and pulmonary inflammation

T26 – An In-vitro Cardiopulmonary Bypass Model to Study Endothelial Cell Responses to Shear Activated Monocytes

Hao Zhou[1], Lan Tu[2], Vishal Nigam[2], Cecilia Giachelli[1], Marta Scatena[1]
[1]University of Washington, Seattle, Seattle, Washington, USA; [2]Seattle Children's Hospital, Seattle, Washington

- Shear stress in cardiopulmonary bypass affects both monocytes and endothelial cells.
- An in-vitro system is developed to study the interaction of cells after CPB.
- IL-8 downstream pathway may regulating the interaction between the two cells.

T27 – Overcoming the Challenges of Preclinical Murine Hemapheresis with State-of- the-Art Microfluidics

Kristina Chapman[1]
[1]Boise State University, Meridian, Idaho, USA

- MSM pump is a safe and highly selective blood filtering device to use in murine hemapheresis.
- the MSM pump and antibody combination is effective in removing circulating cytokines.
- Enable a "bench-to-bedside" screening tool for testing new hemapheresis modalities.

T28 – TNIK is a novel activator of Interferon signaling in endothelial cells

Abishai Dominic[1], Guangyu Wang[2], Jun-ichi Abe[3], Nhat-Tu Le[2]
[1]Texas A&M Health Science Center, Houston, Texas, USA; [2]Houston Methodist Research Institute, Houston, Texas, USA; [3]The University of Texas MD Anderson Cancer Center, Houston, Texas, USA

- Role of TNIK in interferon signaling and activation

- Role of TNIK in control of the expression of STAT1&2
- TNIK regulates interferon stimulated genes and chemokine secretion in endothelial cells

DEVELOPMENTAL VASCULAR BIOLOGY

T29 – The endothelial chromatin remodeling enzymes BRG1 and CHD4 transcriptionally regulate extracellular matrix production to promote lung development.

Meng Ling (Melinda) Wu[1], Courtney Griffin[1]
[1]Oklahoma Medical Research Foundation, Oklahoma, Oklahoma, USA

- Deletion of endothelial chromatin remodeling enzymes BRG1 and CHD4 causes developmental lung defects
- The double mutant lungs were compact and small because of lacking extracellular matrix.
- Endothelial cells promote lung development via producing collagen IV and elastin in distal lungs.

T30 – microRNA-223 limits hemogenic endothelial cell specification and myelopoiesis

Yinyu Wu[1], Umadevi Paila[1], Gael Genet[1], Karen Hirschi[1]
[1]University of Virginia, CHARLOTTESVILLE, USA

- miR-223 limits murine hemogenic endothelial cell and hematopoietic stem/progenitor cell generation.
- miR-223 negatively regulates RA signaling, which is known to promote definitive hematopoiesis.
- Loss of miR-223 promotes the generation of myeloid-biased hemogenic endothelial cells and HSPCs

T31 – Role of the Retinoic Acid in Placental Endothelial Cell Specification during Vascularization

Aleksandra Cwiek[1]
[1]University of Virginia, Charlottesville, Virginia, USA

- Abnormal vascularization of the placenta is its most prevalent pathology during the pregnancy
- Retinoic acid plays a critical role in EC specification during placental development
- Retinoic acid plays a role in proper placental vascular development

T32 – Novel ETV2 isoforms are differentially expressed and may dynamically regulate endothelial cell differentiation.

Jordon Aragon[1], Madison Mehlferber[1], Leon Sheynkman[1], Jingyao Qiu[2], Gloria Sheynkman[1], Karen Hirschi[1]
[1]University of Virginia, Charlottesville, Virginia, USA; [2]Yale University, New Haven, Connecticut, USA

- Using long-read RNA sequencing we have identified multiple, novel ETV2 mRNA isoforms.
- These novel Etv2 isoforms are dynamically regulated during vascular development in the embryo.
- Fluorescent in situ hybridization revealed these ETV2 isoforms may be spatially regulated as well.

T33 – Mechanisms of Blood Retinal Barrier Development

Jessica Furtado[1], Kevin Boyé[2], Anne Eichmann[1]
[1]Yale University, New Haven, Connecticut, USA; [2]Paris Centre de Recherche Cardiovasculaire – PARCC, Paris, France

- Unc5B regulates angiogenesis and blood retina barrier permeability.
- Netrin1 binding to Unc5B regulates Norrin/β-catenin signaling via pLRP5.

VASCULAR BIOLOGY I

T34 – Evidence for P53 protection of Car4+ lung endothelial cells in neonatal hyperoxia.

Jonathan Bywaters[1], Lisandra Vila Ellis[1], Jichao Chen[1], Jichao Chen[2]
[1]Pulmonary Medicine, The University of Texas M. D. Anderson Cancer Center, Houston, Texas, USA; [2]M. D. Anderson UTHealth Graduate School of Biomedical Sciences, Houston, Texas, USA

- In a hyperoxia model of BPD, pulmonary capillary (Cap) 1 ECs stall in their fate transition to Cap2.
- Stalled Cap1 ECs activate numerous p53 target genes in hyperoxia.
- Cap2 cells are preferentially reduced in p53-deleted mice undergoing hyperoxia treatment.

T35 – MMP19-Deficient Mice Are Susceptible to Chronic Allergen-Induced Pulmonary Vascular Remodeling

Mark Ihrie[1], Victoria McQuade[1], Jack Womble[1], Sudarshan Rajagopal[2], Jeffrey Everitt[3], Jennifer Ingram[1]
[1]Division of Pulmonary, Allergy and Critical Care Medicine, Duke University, Durham, North Carolina, USA; [2]Cardiology Division, Duke University, Durham, North Carolina, USA; [3]Department of Pathology, Duke University, Durham, North Carolina, USA

- A better understanding of the mechanisms of pulmonary hypertension is needed to facilitate treatment
- MMP-19 deficient mice exposed to HDM exhibit increased vascular remodeling and CX3CL1 levels.
- MMP-19 may protect against pulmonary arterial hypertension through CX3CL1 signaling.

T36 – moved to W03

T37 – Vertebral Body Adherent Allogeneic Mesenchymal Stromal Cells Increases Perfusion and Muscle Function in Diabetic Mouse Models of Critical Limb Threatening Ischemia

Humraaz Samra[1], Michael Ingram[1], Justin King[1], Kara Allen[1], Anush Motaganahalli[2], Theresa Doiron[1], Leni Moldovan[1], Chang-Hyung Gil[1], Greg Westin[1], Michael Murphy[1], Steven Miller[1]
[1]Indiana University School of Medicine, Indianapolis, Indiana, USA; [2]Indiana University, Bloomington, Indiana, USA

- Critical Limb Threatening Ischemia is a priority, autologous bone marrow therapy is ineffective.
- Allogeneic Mesenchymal Stromal cells via healthy vertebral body donors can provide benefit
- Intramuscular injection with positive results, further avenue for stem cell therapy and exploration

WEDNESDAY POSTERS

ENDOTHELIAL CELL BIOLOGY

W01 – Interrogating the endothelial barrier-strengthening proteome upon Sphingosine 1-Phosphate (S1P) signaling

Avishek Ghosh[1], Timothy Hla[1]
[1]Boston Children's Hospital, Harvard Medical School, Boston, Massachusetts, USA

- Proximity labeling coupled to Mass spectrometry
- Finding novel targets to control vascular leakage in Sepsis
- adherens junction proteomics

W02 – Role of Thrombospondin-1 in Disturbed Flow-Mediated Arterial Stiffening in Human Aortic Endothelial Cells

Gloriani Sanchez Marrero[1], Feifei Li[2], Luke Brewster[1]
[1]Georgia Institute of Technology, Emory University, Atlanta, Georgia, USA

- Understand modifiable molecular pathways that contribute to arterial stiffening in PAD.
- ECs lining PAD arteries experience disturbed blood flow and a stiff matrix environment.
- TSP-1 and CTGF are upregulated in disturbed blood flow locations in PAD arteries.

W03 – The role of mitochondrial respiratory complex I in vascular smooth muscle cell proliferation

Alishba Maira[1], Nicholas Sibinga[1], Dario Riascosbernal[1]
[1]Albert Einstein College of Medicine, Bronx, New York, USA

- Vascular Biology - Smooth muscle cell
- Mitochondrial respiration Complex 1
- AtherosIcerosis

W04 – Endothelial Cell Cycle Responses to Laminar Shear Stress

Natalie Tanke[1], Ziqing Liu[1], Bryan Kistner[1], Jean Cook[1], Vicki Bautch[1]
[1]University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA

- Endothelial cells under laminar shear stress enter a quiescent cell cycle state.
- Flow mediated quiescence has unique profiles compared to serum starvation or contact inhibition.
- Understanding endothelial flow mediated quiescence has implications for wound healing and disease.

W05 – Can Myogenic Tone Protect Endothelial Function? Integrating Myogenic Activation and Dilator Reactivity for Cerebral Resistance Arteries in Metabolic Disease

Brayden Halvorson[1], John McGuire[1], Krishna Singh[1], Joshua Butcher[2], Julian Lombard[3], Paul Chantler[4], Jefferson Frisbee[1]
[1]Western University, London, Canada; [2]Oklahoma State University, Stillwater, Oklahoma, USA; [3]Medical College of Wisconsin, Milwaukee, Wisconsin, USA; [4]West Virginia University Health Sciences Center, Morgantown, West Virginia, USA

- The contributions of the pro-oxidant environment and an increased myogenic tone is variable.
- Under high myogenic activation endothelial function receives some degree of protection.
- Results highlight the importance of considering data in aggregate and within the individual animals.

W06 – Ultrasound contrast imaging via gas-filled microbubbles: characterization of flow parameters and targeted imaging of endothelial biomarkers.

Pingyu Zhang[1], Sean Wood[1], Sunil Unnikrishnan[1], Galina Diakova[1], Alexander Klibanov[1]
[1]University of Virginia, Charlottesville, Virginia, USA

- In vivo imaging of vascular biomarkers
- In vivo imaging of blood flow
- Antibody-mediated targeted imaging

W07 – The Effects of Preeclamptic Milieu on Cord Blood Derived Endothelial Colony-Forming Cells

Eva Hall[1], Erin Neu[1], Azizah Ziauddin[1], Laura Alderfer[1], Laura Haneline[2], Donny Hanjaya-Putra[1]
[1]University of Notre Dame, Notre Dame, Indiana, USA; [2]Indiana University School of Medicine, Indianapolis, Indiana

- Preeclampsia, despite being a prolific disease, is not fully understood and has limited treatments.
- Preeclampsia has a notable effect on the function and genetics of endothelial progenitor cells.
- Cells may compensate for increased senescence with rapid proliferation which affects tube formation.

W08 – Age-associated early immune response of lung endothelial cells from humanized K18-ACE2 mice infected with SARS-CoV-2

Saravanan Subramaniam[1], Devin Kenney[2], Archana Jayaraman[1], Aoife Kateri O'Connell[2], Sarah Walachowski[1], Paige Montanaro[2], Nicholas Crossland[2], Florian Douam[2], Markus Bosmann[1]
[1]Pulmonary Center, Boston University, MA, Boston, Massachusetts, USA; [2]NEIDL, Boston University, MA, Boston, Massachusetts, USA

- SARS-CoV-2 infected aged mice showed less survival when compared to young mice.

- Endothelial cells from aged mice infected with SAR-CoV-2 have less immune response than young mice.
- Endothelial cells from infected mice showed enhanced prothrombotic and leukocytes adhesion markers.

W09 – Hutchinson-Gilford Progeria Syndrome impairs the endothelium's genetic response to flow.

Crystal Kennedy[1], George Truskey[1]
[1]Duke University, Durham, North Carolina, USA

- HGPS deaths are primarily caused by atherosclerosis. Little is known about how ECs may contribute.
- RNAseq shows a diminished genetic response to physiologically relevant flow in HGPS ECs.
- Gene sets altered by flow in HGPS ECs differ from those in healthy ECs and indicate dysfunction.

W10 – Left Ventricle Diastolic and Endothelial Dysfunction Develops in Mice Lacking the Endothelial Cell-Selective Adhesion Molecule

Vadym Buncha[1], Katie Anne Fopiano[1], Yanna Tian[1], Liwei Lang[1], Zsolt Bagi[1]
[1]Medical College of Georgia, Augusta University, Augusta, Georgia, USA

- ESAM plays a mechanistic role in the development of both microvascular dysfunction and LVDD
- Echocardiography shows impaired diastolic parameters in mice with genetically deleted ESAM.
- Lack of ESAM is related to decreased myocardial vascularization and vascular endothelial dysfunction

LYMPHATICS

W11 – FGF-2 and PDGF-BB are non-canonical drivers of recurrent corneal lymphangiogenesis

Ahana Majumder[1], Zachary Budden[1], Jacob Paulson[1], Mason Crow[1], Darci Fink[1]
[1]South Dakota State University, Brookings, South Dakota, USA

- Recurrent Lymphangiogenesis is driven by non-canonical lymphangiogenic factors
- FGF-2 and PDGF-BB are sufficient to drive recurrent corneal lymphangiogenesis
- Macrophages release lymphangiogenic factors driving recurrent corneal lymphangiogenesis

W12 – Podoplanin regulates angiogenesis and lymphangiogenesis through physical recognition

Donghyun Jeong[1], Eva Hall[1], Erin Neu[1], Donny Hanjaya-Putra[1]
[1]University of Notre Dame, Notre Dame, Indiana, USA

- Lymphatic and blood endothelial cells form distinct vascular networks.
- Podoplanin is responsible for the separation of the two vascular networks.
- Expression of folliculin in blood endothelial cells maintain blood EC identity.

W13 – Investigating the role of S1PR1 signaling in the maintenance of adult meningeal vasculature structure and function

Anjali Gupta[1], Timothy Hla[1]
[1]Boston Children's Hospital/Harvard Medical School, Boston, Massachusetts, USA

- Meningeal lymphatics are newly discovered vessels crucial for brain waste clearance.
- Meningeal endothelial cell biology in CNS health and immunity.
- Meningeal lymphatic vessels are important for CNS health.

W14 – Loss of primary cilia protein IFT20 disrupts LEC proliferation and migration playing a critical role in lymphatic vessel development and inflammation-induced remodeling

Delayna Paulson[1], Rebecca Harms[1], Cody Ward[1], Mackenzie Latterell[1], Zachary Lehmann[1], Luke Knutson[1], Gregory Pazour[2], Darci Fink[1]
[1]South Dakota State University, Brookings, South Dakota, USA; [2]University of Massachusetts Medical School, Worcester, South Dakota, USA

- Primary ciliary and IFT20 dependant signaling on lymphatic endothelial cells
- Inflammation-induced lymphatic vessel remodeling
- Lymphatic vessel development

W15 – Using intravital microscopy to identify features of corneal lymphatic remodeling during inflammation and healing

Heather Collazo[1], Rebecca Harms[1], Darci Fink[1]
[1]South Dakota State University, Brookings, South Dakota, USA

- Lymphatic Remodeling
- Inflammation
- Wound healing

MECHANOSTRANSDUCTION

W16 – Pericyte-Endothelial Cell Interaction Following Cessation of Blood Flow

Hanaa Abdelazim[1], John Chappell[1]
[1]Fralin Biomedical research institute- Virginia Tech, Roanoke, Virginia, USA

- The impact of blood flow changes on the vascular component of the blood-brain barrier
- Creating an ex-vivo model simulating the absence of flow (static conditions) in mature brain vessels
- The presence of a two-phase response to the loss of flow

W17 – Microfluidic approach for quantifying vascular permeability in the presence of transmural flow

Stephanie Huang[1], William Polacheck[1]
[1]University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA

- Microfluidic vessel to quantify vascular permeability
- Endothelial barrier function in the presence of transmural flow
- Decoupling convective and diffusive contributions towards vascular extravasation

VASCULAR CROSSTALK

W18 – Effects of Endothelial-Derived Extracellular Vesicles from Obese/Hypertensive Adults on Cardiomyocytes

Hannah Fandl[1], Vinicius Garcia[1], Lillian Brewster[1], John Treuth[1], Jared Greiner[1], Kevin Davy[2], Brian Stauffer[3], Christopher DeSouza[1]
[1]University of Colorado Boulder, Boulder, Colorado, USA; [2]Virginia Tech, Blacksburg, Virginia, USA; [3]Anschutz Medical Center, Denver, Colorado, USA

- Obesity and hypertension are associated with increased risk of heart failure.
- Circulating EMVs are involved in the develop and progression of vascular disease and cardiomyopathy
- Potential role of EMVs in obese/hypertensive-related heart failure.

W19 – The role of adipose tissue identity in blood pressure regulation

Mascha Koenen[1], Tobias Becher[1], Sarah Halix[1], Ilaria Del Gaudio[2], Scott Buttler[3], Annarita DiLorenzo[4], Paul Cohen[1]
[1]Rockefeller University, NEW YORK, USA; [2]Université Paris Cité, Inserm, PARCC, Paris, France; [3]Cornell University, Ithaca, New York, USA; [4]Weill Cornell Medicine, NEW YORK, New York, USA

- Thermogenic adipose tissue affects blood pressure regulation
- Crosstalk between thermogenic adipocytes and the vasculature
- Thermogenic adipose tissue affects angiotensin response

W20 – The lung microvasculature promotes alveolar type 2 cell differentiation before birth

Paolo Panza[1], Hyun-Taek Kim[2], Till Lautenschläger[1], Didier Stainier[1]
[1]Max Planck Institute for Heart and Lung Research, Bad Nauheim, Germany; [2]Soonchunhyang Institute of Medi-bio Science (SIMS), Cheonan, Korea (Republic of)

- Lung vascularization is important in alveolar morphogenesis.
- Capillary endothelial cells promote AT2 cell differentiation.
- The angiocrine factor SPARCL1 promotes AT2 cell differentiation in vitro.

VASCULAR SIGNALING

W21 – Akt3 activation by R-Ras stabilizes endothelium via intercellular crosstalk mediated by Jagged1-Notch

Jose Herrera[1], Masanobu Komatsu[1]
[1]Johns Hopkins University-All Children's Hospital, St. Petersburg, Florida, USA

- The endothelial Small GTPase R-Ras is involved in vascular stabilization
- Jagged1-Notch signaling between endothelial cells is important for vascular stabilization
- Akt3 is important to control endothelial cell migration, proliferation, and vessel stabilization.

W22 – Investigating Pluridimensional Signaling of Vasoactive G-Protein Coupled Receptors

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- Vascular Signaling
- BRET biosensors
- RhoA signaling, Ca2+ signaling, MAPK/ERK signaling

W23 – Hyperactive GNAQ mutation in endothelial cells drive aberrant vascular morphology and signaling

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[1]Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, USA

- HyperactiveGNAQ mutation in endothelial cells cause vascular anomaly in mice
- Hyperactive GNAQ stimulates MAPK/ERK signaling in endothelial cells
- Mutant GNAQ may stimulate NF-kB and endothelial cell pro-inflammatory signaling

W24 – Renal vascular defects in mouse models of Von Hippel Lindau disease

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[1]Virginia Tech, Fralin Biomedical Research Institute, Roanoke, Virginia, USA

- Early adult mouse models of Vhl mutation develop polycythemia and erythema.
- Gene expression of various vascular-associated proteins is altered in the kidneys of the mutant mice
- Notch3 expression is significantly increased in the kidneys of the Vhl null and 2B mutant mice.

VASCULAR BIOLOGY II

W25 – KRAS pathway-targeted therapy with MEK inhibition for a severe facial arteriovenous malformation in an adult patient

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- MEK inhibitor Trametinib for a severe facial AVM was well tolerated with no serious adverse events.
- Daily Trametinib resulted in significant reduction in AVM size and symptoms within 6 months.
- We embark on a pilot safety trial of Trametinib as a genotype-guided therapy for patients with AVMs.

W26 – Generation of Lymphatic endothelial cell from human iPSC

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[2]Department of Chemical and Biomolecular Engineering, University of Notre Dame, IN

- Xenofree differentiation from human induced pluripotent cells
- Effect of transcription factor (ETV2) on the differentiation towards LEC
- Monolayer based, step wise differentiation process

W28 – Characterization of protein isoform diversity in human umbilical vein endothelial cells (HUVECs) via long-read proteogenomics

Madison Mehlferber[1], Ben Jordan[1], Erin Jeffery[1], Leon Sheynkman[1], Jamie Saquing[1], Bipul Acharya[2], Karen Hirschi[1], Gloria Sheynkman[1]
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- Long-read RNA-sequencing and mass-spectrometry-based proteomics, long-read proteogenomics
- Alternative splicing in endothelial cells and characterization of expressed isoforms
- Nextflow developed analysis pipeline

W29 – Endothelial Deletion of Rbpj Leads to Perivascular Abnormalities in Mouse Model of Brain Arteriovenous Malformation

Sera Nakisli[1], Samantha Selhorst[1], Shruthi Kandalai[1], Corinne Nielsen[1]
[1]Ohio University, Athens, Ohio, USA

- Brain pericyte area was expanded without increased proliferation with endothelial Rbpj deletion.
- Increased brain pericyte area was spatially and temporally regulated.
- Communication between brain pericytes and endothelial cells was disrupted.

W30 – Engineered Microphysiological systems for testing effectiveness of cell-based cancer immunotherapies

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[1]Dana-Farber Cancer Institute, Boston, Massachusetts, USA; [2]Belfer Center for Applied Cancer Science - Dana-Farber Cancer Institute, Boston, Massachusetts, USA

- Testing cell therapies using microfluidic models of the vascularized tumor microenvironment
- Leveraging STING agonism to activate vascular endothelium to prone immune cell infiltration
- Model of small-cell lung cancer microenvironment and testing of NK cell therapies

W31 – Preclinical development and signaling actions of a novel quinone-nitroalkene hybrid molecule for sickle cell anemia

Fabliha Chowdhury[1], Megan Miller[2], Katherine Wood[2], Shuai Yuan[2], Stefanie Taiclet[2], Derek Sinchar[2], Elizabeth Rochon[2], Francisco Schopfer[1], Bruce Freeman[1], Adam Straub[1]
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- Reduce oxidative stress in sickle cell anemia by activating Nrf2 and CYB5R3.
- Induce hematopoiesis and inhibit hemolysis in sickle cell anemia.
- Alleviate cardiovascular complications associated with sickle cell anemia.

W32 – Toll-like receptor 4 prevents cellular senescence by inhibiting SOX9/miR-223 signaling in emphysema

So-Jin Kim[1], Hyojin Kim[2], Emma Lofgren[3], Youwei Chen[1], Elias Coutavas[1], Cheol Hwangbo[2], Patty Lee[1]
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- COPD afflicts millions of people every year by leading to respiratory failure.
- TLR4 on lung structure cells is required to maintain lung integrity.
- miR-223 is one of the most expressed miRNAs in COPD patients.

W33 – Evaluating the Potential of Collateral Flow for Microvessel Access and Stabilization during Vascular Blockage

Caroline Willi[1], John Chappell[1]
[1]Fralin Biomedical Research Institute at Virginia Tech-Carilion, Roanoke, Virginia, USA

- The potential utility of collateral flow in sustaining capillary access and stability post-ischemia.
- Development of an ex vivo tissue-based microvascular fluidics model.
- Evaluation of the microvascular response to a loss-of-flow event.

W34 – Atherosclerotic risk of branched chain amino acids in a tissue-engineered blood vessel model

Ellery Jones[1], Joanna Peng[1], Jamie King[1], De Shanna Johnson[1], George Truskey[1]
[1]Duke University, Durham, North Carolina, USA

- We use a tissue-engineered blood vessel model to study the effects of BCAA on vascular health.
- In our model, high BCAA and oxLDL interact to impair vasodilation and induce monocyte adhesion.
- In 2D endothelial cell studies, high BCAA increase mitochondrial oxidative stress.

W35 – Stacking thick perfusable human microvascular networks promotes host integration and rapid vascularization

Ariana Frey[1], Nicole Zeinstra[1], Zhiying Xie[1], Ruikang Wang[1], Charles Murry[1], Ying Zheng[1]
[1]University of Washington, Seattle, Washington, USA

- Heart attacks lead to permanent loss of cardiac muscle, requiring cardiac regenerative therapies.
- We developed perfusable thick multilayer microvessels which support vascular remodeling *in vitro.*
- These microvessels support early vascular remodeling and host vascular integration *in vivo*.

W36 – Microphysiological Model for Rheumatoid Arthritis and Atherosclerosis

Mingzhi Xu[1], George Truskey[1]
[1]Duke University, Durham, North Carolina, USA

- Rheumatoid arthritis increase the risk of cardiovascular disease.

- Using microphysiological systems to uncover the influence of RA muscle on engineered blood vessels.
- RA muscle influenced engineered blood vessels presented a more atherosclerotic phenotype.

W37 – Development of a Biomimetic Endometrial Decidua Organ-a-chip Model: A Proof of Concept

Sebastian Naranjo[1], Somin Lee[2], Noo Jeon[2], Catherine Klapperich[1], Joyce Wong[1]
[1]Boston University, Boston, Massachusetts, USA; [2]Seoul National University, Seoul, Korea (Republic of)

- Endometrium organ-on-a-chip proof of concept
- Co-culture driven neovascularization

W38 – Rescuing Aging-Associated Cardiovascular Dysfunction Through Mitochondrial Transfer

Colwyn Headley[1], Yae Rhee[1], Alicia Deng[1], Joshua Spin[1], Philip Tsao[1]
[1]Stanford University, Palo Alto, California, USA

- mitochondrial dysfunction and oxidative stress are central to aging-associated vascular diseasesCo-culture driven neovascularization
- developing mitochondria-centric therapies would significantly impact elder health
- transferring mitochondria into aged vascular cells may be a novel therapy