

POSTER PRESENTATIONS AT VASCULAR BIOLOGY 2026

ANGIOGENESIS I

M02

PAK1 epigenetically regulates diabetes-relevant endothelial cell function and wound healing

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- The epigenetic enzyme PAK1 is downregulated in type 2 diabetic patient wound endothelial cells.
- Pharmacologic inhibition of PAK1 decreased TGF- β -induced proangiogenic VEGF gene expression.
- PAK1 activation by FDA-approved fingolimod improved wound healing in normal and diabetic mice.

M03

Prenatal alcohol exposure inhibits cerebrovascular cell sprouting by downregulation of Tensin 1 and Tensin 2 by miR-150-5p

Richard Justice, Amy Gardiner PhD

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- miR-150-5p impairs angiogenesis and BBB, driving vascular dysfunction in PAE.
- Tensin 1 and Tensin 2 are molecular targets of microRNA-150-5p during PAE.
- Tensin 1 and Tensin 2 help rescue endothelial cell sprouting during PAE.

ATHEROSCLEROSIS I

M04

Putrescine biosynthesis couples metabolism to epigenetic control of smooth muscle cell identity and atherosclerotic plaque stability

Louise Frausto¹, Chad Stroope¹, Theresea-Anne Governale¹, Dhananjay Kumar PhD¹, Rajan Pandit¹, Abraham Quaye PhD¹, Tanvir Ahmed¹, Judith Sluimer PhD², Karen Y Stokes PhD¹, Amr R Salem PhD¹, Nirav Dhanesha PhD¹, A. Wayne Orr PhD¹, Oren Rom PhD¹, Arif Yurdagul Jr PhD¹

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- Putrescine biosynthesis preserves vascular smooth muscle cell contractility
- Putrescine sustains Myocardin expression in smooth muscle cells by stabilizing H3K4me2
- Dysregulated putrescine biosynthesis in vascular smooth muscle cells drives plaque instability

M05

Senomorphic intervention demonstrates limited benefits in advanced atherosclerosis in mice

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- Senomorphic agents reduce atherosclerosis
- SMC and EC- lineage tracing mice and advanced atherosclerosis
- Senescence and senomorphics

M06

Downregulation of mitochondrial genes promotes atherosclerosis via activating integrated stress response in endothelial cells

Huiling Hong Ph.D., Xiao Yu Tian Ph.D

The Chinese University of Hong Kong, Hong Kong, Hong Kong

- TFB2M-mediated mitochondrial gene transcription is downregulated in ECs in atherosclerosis.
- TFB2M deficiency impairs mitochondrial respiration and activates mtISR via DELE1 in ECs.
- Dele1-targeted ASO nanoparticles reduce lipid accumulation and enhance plaque stability in mice.

M07

Investigating the effects of a smooth muscle cell-specific DDR1 knockout on atherosclerotic plaque development

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- Understanding atherosclerosis through smooth muscle cell specific knockout
- DDR1 in vascular smooth muscle cells influence atherosclerosis
- Vascular smooth muscle cells stabilize plaque by increasing matrix proteins and decreasing plaque

M08

CXCL5 is atheroprotective and signals through CXCR2 on a NETosis-prone neutrophil population in human atherosclerosis

Rebekah Sanchez-Hodge MPH-VPH¹, Sryia Kongala BS¹, Jaqueline Haitian Wu², Gavin Hatalosky², Antonella Piscoya Castro BS², Eden Hunsader BS², Kendall Cannon², Aliyaa Pathan BS², Allison McKay², Alex Garris BS², Bruno Munsetti PhD², Saranya Ravi PhD MD², Sean Cade Smithey², Edward Bahnson PhD², Robert Wirka MD², Jonathan Schisler PhD^{1,2,3}

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- Circulating CXCL5 falls with CAD severity; CAD risk allele lowers CXCL5
- CXCR2+ neutrophils with NET machinery mark a distinct plaque population
- Cholesterol-efflux macrophages form a cluster distinct from CXCR2+ neutrophils

M09

Extracellular vesicle-associated Nef reprograms macrophage heterogeneity to drive proatherogenic cellular migratory signaling

Sarvesh Chelvanambi PhD, Yuto Nakamura PhD, Luisa Weiss PhD, Ahmad Samiei MS, Prabhash K Jha PhD, Taku Kasai PhD, Mary C Whelan, Hooman Mirzakhani MD, Sasha A Singh PhD, Elena Aikawa MD, PhD, Masanori Aikawa MD, PhD

Brigham and Women's Hospital, Boston, MA, USA

- Nef extracellular vesicles drive pathogenic macrophage migration to promote atherosclerosis
- Multiomics shows Nef drives proatherogenic macrophage states to promote vascular inflammation.
- A novel EV-mediated mechanism linking HIV infection to atherosclerosis via macrophage activation.

CARDIOVASCULAR DISEASES I

M10

Cell-autonomous Wnt activity promotes transient re-programming and cell cycle re-entry of coronary artery endothelial cells

Bhavnes Bishnoi¹, Alfia Saini¹, Vinay Rao², Omkar Golatkar^{1,3}, Ravindra Zirmire PhD¹, Shruthi Viswanath¹, Dhandapany Perundurai², Soumyashree Das¹

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- Cell autonomous Wnt regulates artery cell plasticity during cardiac regeneration
- Exogenous Wnt promotes coronary collateral formation in non-regenerative hearts
- Identification of pathogenic genetic variants of WNT genes in (Asian) Indian population

M11

Intracranial Aneurysm Susceptibility gene hspg2/Perlecan regulates pericyte coverage and vascular stability in zebrafish

Surya Prakash Rao Batta, Suman Gurung, Nicole Restrepo, Vishal Mardhekar, Saulius Sumanas

University of South Florida, Tampa, FL, USA

- Hspg2 regulates Pdgfb-Pdgfr α signaling, pericyte coverage and vascular stability
- Hspg2 loss is correlated with an increase incidence of Intracranial Aneurysm.
- Hspg2 regulates blood-brain-barrier by modulating pericyte dynamics.

M12

The role of Rab11-FIPs in the endosomal-lysosomal pathway and congenital heart disease

Carolyn Lazaroff, Amber Stratman PhD

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- Cardiovascular Development
- Rab adaptor proteins
- Protein trafficking

M13

Targeting Alu RNA attenuates endothelial inflammation induced by DNA Hypomethylating cancer therapy

Gabriela Valentina Aguilar Escajadillo Biomedical Engineering¹, Isabella Hetherington PhD Candidate², Hana S Totary-Jain PI^{1,3}

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- 5-aza induces inflammatory signaling in human coronary artery endothelial cells.
- 5-aza treatment increases Alu RNA and induces expression of inflammatory genes IL1B, IL6, and VCAM1
- Alu-targeting siRNA attenuates inflammatory gene expression and reduces pro-IL-1 β protein

M14

Altered cardiac and circulating miR-208a is associated with cardiac atrophy and tumor progression in hypertension-associated cancer

Luis Felipe Rodrigues, Beatriz Rafaelle Góes-Santos, Bruno R.A. Pelozin, Alex Improta-Caria, Edilamar M. Oliveira, Tiago Fernandes

School of Physical Education and Sport of University of Sao Paulo (EEFE-USP), São Paulo, São Paulo, Brazil

- Hypertension aggravates tumor growth and inflammatory cardiac remodeling
- Cardiac miR-208 downregulation accompanies cardiac atrophy in hypertension associated with cancer
- Circulating miR-208a suggests heart-tumor crosstalk in in hypertension associated with cancer

M15

Induction of YAP/TAZ in monocytes differentiates BMPR2 mutant PAH-Patients from unaffected BMPR2 mutation carriers

Yapeng Liu M.D., Aiqin Cao Ph.D., Lingli Wang M.D., Marlene Rabinovitch M.D.

Sanford University, Palo Alto, CA, USA

- Reduced BMPR2 in monocytes promotes PAH
- YAP/TAZ -related features is initiating PAH in BMPR2 mutation patients
- Unaffected BMPR2 mutation carriers are protected from YAP/TAZ induced PAH

CELL BIOLOGY AND SIGNALING I

M16

Biological sex defines subcellular cAMP landscape in vascular smooth muscle

Eric Aian Pereira da Silva PhD¹, Ildernandes Vieira PhD¹, Hannah Voorhees BS¹, Padmini Sirish PhD¹, Miguel Martín-Aragón Baudel PhD¹, Yang K Xiang PhD², Manuel F Navedo PhD¹, Madeline Nieves-Cintrón PhD¹

¹University of California, Davis, CA, USA. ²University of California, Los Angeles, CA, USA

- Females have an estrogen-dependent SR cAMP pool absent in ovariectomized females and males.
- PDE3/PDE4 localization controls sex-specific SR cAMP signaling.
- SR cAMP compartmentalization shapes vascular relaxation and stiffness.

M17

Acute e-cigarette exposure impairs cAMP signaling in systemic arterial smooth muscle cells

Sophia Salbato M.S., Nuria Daghbouche Rubio Ph.D., Maryann K Ferrara B.S., Eric A Pereira da Silva Ph.D., Miguel Martin Aragon Baudel Ph.D., Manuel F Navedo Ph.D., Madeline Nieves-Cintrón Ph.D.

UC Davis, Davis, California, USA

- 10 days of e-cigarette exposure impairs mesenteric artery cAMP signaling
- Sex differences in e-cigarette-induced changes in vascular cAMP signaling
- E-cigarette-induced changes in vascular cAMP signaling are vascular bed-dependent

M18

SMC released IL-6 promotes macrophage M2 polarization via activation of STAT3/IL-10 autocrine signaling

Mark C Renton PhD¹, Adam C Hoch MS², Rodrigo Suarez Lopez Gallo BS¹, Scott R Johnstone PhD^{1,2}

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- Transwell co-culture shows secreted SMC factors polarize macrophages towards M2-like phenotype.
- HCASMC secrete IL-6 sufficient to drive gp130/STAT3 macrophage polarization.
- A feed-forward SMC-macrophage signalling axis may promote neointima formation.

M19

Lysophosphatidic acid-induced vascular smooth muscle cell proliferation is mediated by the matricellular protein CCN2 via a unique signaling pathway

Mei-Zhen Cui PhD¹, Xuemin Xu PhD¹, Feng Hao MD, PhD²

¹Texas Tech Health Science Center, Paul L. Foster School of Medicine, El Paso, El Paso, TX, USA. ²UTPB, Odessa, TX, USA

- The role of matricellulae proteins in the development of atherosclerosis.
- LPA induction of smooth muscle cell proliferation is mediated by the matricellular protein CCN2.
- The interaction of ERK2 and JNK1 is responsible for LPA-induced CCN2 expression.

M20

STAT1 hyperactivation accelerates vascular smooth muscle cell aging in mice

Tahrima Arman Tusty^{1,2}, Longbiao Yao², Lorin Olson^{2,1}

¹University of Oklahoma Health Sciences Center, Oklahoma City, USA. ²Oklahoma Medical Research Foundation, Oklahoma City, USA

- A mouse model with constitutive PDGFR β signaling exhibits aortic VSMC senescence at 6 weeks of age.
- STAT1 hyperactivation may be a key mediator of vascular smooth muscle cell senescence.
- Socs1 deletion in vascular smooth muscle cells causes hyperactivation of STAT1 signaling.

M21

Impact of smooth muscle actin pathogenic variants on INO80-mediated chromatin remodeling

Jeison Garcia-Serrano PhD, Callie S Kwartler PhD

Department of Internal Medicine. McGovern Medical School at UTHealth Houston, Houston, Texas, USA

- Smooth muscle alpha- (SMA) and beta-actin both interact with INO80 chromatin remodeling complex
- SMA alters nucleosome sliding of INO80 compared to beta-actin; mutations in SMA disrupt activity
- SMA and its mutations also alter H2A.Z/H2A histone variant exchange by INO80

CEREBROVASCULAR HEALTH AND DISEASE I

M22

De novo pathogenic variant in RNF213 disrupts smooth muscle cell differentiation and causes pediatric-onset moyamoya disease

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UTHealth Houston, Houston, TX, USA

- Moyamoya disease results from increased migration and proliferation of immature smooth muscle cells.
- RNF213 pathogenic variants are the major genetic contributor to moyamoya disease worldwide.
- Mutant RNF213 SMCs are immature and lead to occlusive lesions post-carotid artery injury in mice.

M23

SETD5 haploinsufficiency disrupts neural crest-derived smooth muscle cell differentiation

Angie D Gonzalez BS, Kiara E Bornes BS, Alexei M Robin BS, Xue-Yan Duan, Aminat O Dosunmu MS, Jose E Esparza Pinelo BS, Alexis Richard PhD, Amelie Pinard PhD, Dianna M Milewicz MD PhD, Callie S Kwartler PhD

UTHealth Houston, Houston, Texas, USA

- De novo heterozygous loss-of-function SETD5 variants are associated with moyamoya disease (MMD)
- SETD5 binds SMC contractile promoters and knockdown reduces SMC contractile protein levels
- Impaired SMC maturation from neural crest precursors is a common pathogenic mechanism of MMD

M24

Adenylyl cyclase 2 negatively regulates tight junctions at the Blood-Brain Barrier in a mouse model of intracerebral hemorrhage

Marsilla Gray, Yao Yao

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- Deletion/inhibition of Adcy2 cause no change to BBB homeostasis, but overexpression causes leakiness
- ICH outcomes are improved in mice lacking Adcy2, and worsened in Adcy2 overexpression
- Proteins of the paracellular, rather than transcellular, pathway are affected by modulation of Adcy2

M25

Context-dependent CaV1.2 regulation: S1928 phosphorylation mediates vascular PKA responses while rad remains dispensable

Hannah Voorhees BS¹, Jade Taylor PhD², Maartje Westhoff PhD¹, Johannes W Hell PhD¹, Rose E Dixon PhD¹, Madeline Nieves-Cintrón PhD¹, Miguel Martín Aragón Baudel PhD¹, Manuel F Navedo PhD¹

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- PKA potentiates vascular CaV1.2 during hyperglycemia independent of Rad.
- S1928 phosphorylation is essential for hyperglycemia-driven vascular CaV1.2 potentiation.
- Tissue-specific PKA targets create opportunities for vascular-selective therapeutics.

M26

A smooth muscle-specific GrabATP biosensor mouse reveals hyperglycemia-induced ATP release in intact arteries

Liuruimin Xiang PhD, Declan Manning PhD, Hannah Voorhees MS, Fernando Santana PhD, Madeline Nieves-Cintrón PhD, Manuel F Navedo PhD

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- We developed the first smooth muscle-specific GrabATP biosensor mouse.
- Isolated arteries from this model show robust, dose-dependent GrabATP fluorescence responses to ATP.
- Hyperglycemia triggers pannexin-dependent ATP release in intact arteries.

M27

Pia-FLOW 2.0 enables hemodynamic mapping of skull, meningeal, and pial vascular networks

Hanna Preuss^{1,2}, Lin Tang^{3,4}, Quanyu Zhou^{3,4}, Chaim Glück^{2,3}, Nala Gockel³, Jeanne Droux^{1,2}, Zhenyue Chen^{3,4}, Lukas Glandorf^{3,4}, Susanne Wegener^{1,2}, Daniel Razansky^{2,3,4}, Bruno Weber^{2,3}, Mohamad El Amki^{1,2}

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- Integrated imaging across skull, meningeal, and pial vascular layers.
- High-speed, single-vessel flow and cell tracking.
- Studying vascular responses to arterial and venous thrombosis.

M28

Structural and mechanical remodeling of the human cerebrovasculature with age and neuropathological disease progression

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¹Boston University, Boston, MA, USA. ²Boston University School of Medicine, Boston, MA, USA. ³US Dept. of Veterans Affairs, Boston, MA, USA

- Cerebrovascular remodeling occurs in aging and neuropathological disease affecting brain health
- Biomechanical characterization of human cerebral arteries showed significant stiffening with age.
- Quantitative structural imaging and compositional analyses correlate with arterial stiffening.

DEVELOPMENT I

M29

Tlx-dependent regulation of vegfa controls angiogenic and hematopoietic development in zebrafish

Pritha Das PhD candidate

MDI Biological Laboratory, Bar Harbor, Maine, USA

- tlx loss disrupts Vegfa signaling, impairing vasculature integrity and radial glial architecture.
- During embryogenesis affects hemangioblasts migration, perturbs angioblast and hematopoietic lineage
- Olig2+ cells co-express gata1a in vessels, acting as marker of blood cell populations.

M30

Identification of KIF11 as a new player in lymphatic development

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Temple University, Philadelphia, Pennsylvania, USA

- KIF11 variants are linked to congenital lymphedema, indicating a role of KIF11 in lymphatics.

- KIF11's role in lymphatic endothelial cell function and mammalian lymphatic development is unknown.
- KIF11 regulates lymphatic processes throughout development, offering insights into lymphedema.

M31

Gli3 regulates vascular development and is implicated in vascular malformations

Zaneta Markowska MS, Nafiisha Genet PhD, Elizabeth A Nelson MS, Shelby R Cain MS, Madeline G Jackson MS, Aleksandra Cwiek PhD, Jordon W Aragon PhD, Valentin Delobel PhD, Brian Ruis MS, Uma D Paila PhD, Karen K Hirschi PhD

University of Virginia School of Medicine, Charlottesville, Virginia, USA

- GLI3 regulates endothelial cell proliferation, migration, and arterial specification.
- Endothelial-specific Gli3 loss impairs retinal vascular development in vivo.
- Patient-derived GLI3 R1537C mutation is implicated in vascular malformation pathogenesis.

M32

Connexin 43 regulates lymphatic valve formation via AKT/FOXO1 signaling

Razieh Dehghan, Luz A Knauer, Richa Banerjee PhD, Joshua Scallan PhD, Ying Yang PhD

University of South Florida, Tampa, FL, USA

- Connexin 43 is required for lymphatic valve formation.
- Cx43 loss-of-function induces nuclear translocation of FOXO1 in lymphatic endothelial cells (LECs).
- Foxo1 deletion rescue the valve loss in Cx43-deficient mice.

M33

A murine model of microcephaly and chorioretinopathy reveals the importance of TUBGCP6 in angiogenesis

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- Defects in the gamma-tubulin ring complex result in retinal vascular abnormalities and microcephaly
- TUBGCP6 is essential for viability, embryogenesis, and angiogenesis in mice
- EC loss of TUBGCP6 in mice recapitulates human microcephaly and chorioretinopathy phenotypes

M34

NOTCH1 at the junction of lymphatic button development

Abigail Price B.S.¹, Diandra Mastrogiacomo Ph.D.¹, Kunyu Li B.A.¹, Razieh Dehghan M.S.², Richa Banerjee Ph.D.³, Ying Yang Ph.D.³, Joshua Scallan Ph.D.¹

¹University of South Florida, Tampa, FL, USA. ²University of South Florida, Wesley Chapel, FL, USA. ³University of South Florida, Tampa, FL, USA

- Notch1 is required to form lymphatic button junctions and prevent their reversion to zippers.
- NOTCH1 knockdown in hdLECs inhibits RhoA activation.
- NICD physically interacts with RhoA in hdLECs.

M35

Pervasive expression and function of microRNA-223 help coordinate vascular and hematopoietic development

Elizabeth A. Jones, Carolyn L. Winston, Dionna M. Kasper Ph.D.

Dartmouth Geisel School of Medicine, Hanover, NH, USA

- miR-223 is expressed in diverse types of hematovascular progenitors when they arise in development.
- Etv2+ hematovascular cells in the lateral plate mesoderm are decreased in miR-223 mutants.
- miR-223 coordinates hematovascular formation via distinct regulatory mechanisms for each cell type.

ENDOTHELIUM IN HEALTH AND DISEASE I

M36

PTPN14 acts via SMAD4 to maintain endothelial cell identity in the mature lung vasculature

Deblina Sain PhD, Ons Mamai PhD, Darren Li BSC, Tanuja Desai BSC, Rosemary Akhurst PhD

UCSF, San Francisco, CA, USA

- PTPN14-ENG co-operate to maintain EC identity
- Arterio-venous duality at cellular level in AVM lesions
- Endothelial Cell identity loss in HHT1

M37

Endothelial soluble guanylyl cyclase transnitrosation drives vascular hyperpermeability during ischemia-reperfusion injury

Pia C Burboa PhD, Mauricio A Lillo PhD

Rutgers New Jersey Medical School, Newark, NJ, USA

- GC1-Dependent Transnitrosation Is a Novel Mechanism Driving Vascular Leak
- Cys610 in GC1 Is Critical for Endothelial Hyperpermeability
- Targeting GC1 Transnitrosation Protects Against I/R-Induced Vascular Injury

M38

Human lung single-cell atlas identifies EPAA4 control of neo-arterialization in PAH

Hanqiu Zhao¹, Amy Tian², Zhiqing Wang¹, Jason Hong³, Mark Nicolls², Zhiyu Dai¹

¹Washington University in St. Louis, St. Louis, Missouri, USA. ²Stanford University School of Medicine, Stanford, California, USA. ³University of California, Los Angeles, Los Angeles, California, USA

- A novel arterial endothelial subtype localizes to plexiform and occlusive lesions in PAH lungs.
- EPAA4 promotes partial endothelial-mesenchymal transition and neo-arterialization.
- EPAA4 knockdown improves survival and inhibits PAH development in monocrotaline-exposed rats.

M39

NR2F2 marks a protective venous endothelial response to endothelial HIF-1 α loss in pulmonary hypertension

Qi Zheng PhD, Hanqiu Zhao MS, Susie Cao MS, PhD, Jeffrey R Koenitzer MD, PhD, Zhiyu Dai PhD

Washington University School of Medicine, St. Louis, Missouri, USA

- HIF-1 α loss amplifies HIF-2 α -dependent venous remodeling in EglN1-deficient PH.
- Transitional venous endothelial cells expand markedly and shift toward later pseudotime.
- NR2F2 rises along the venous trajectory and functionally restrains pulmonary hypertension.

M40

The effects of endothelial cell autophagy inhibition on the vascular barrier

Ryan Bench

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- Autophagy inhibition increases endothelial permeability
- Autophagy loss alters endothelial junction organization
- Autophagy inhibition causes VE-cadherin and β -catenin mislocalization

M41

Flow cessation rapidly remodels infection-relevant endothelial programs established by shear

Jiaqi Hong

Duke University, Durham, NC, USA

- CFD-defined orbital shear generates distinct low- and arterial-shear endothelial phenotypes.

- Stopping flow alters KLF2, inflammatory, and adhesion gene expression within 5 hours.
- Endothelial de-adaptation depends on prior shear history, favoring infection studies under flow.

M42

Endothelial JunB deletion preserves renal function and reprograms the kidney response to acute sepsis

Daniel Gonsales Spindola MS.C, Samantha Clark BS, Nina Martino BS, Alejandro Adam PhD, Ramon Bossardi Ramos PhD

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- JunB shapes endothelial inflammatory and injury programs during sepsis.
- JunB loss preserves vascular homeostasis and renal function after CLP.
- Single-cell multiomics reveals endothelial-driven effects across kidney cell types.

M43

Dapagliflozin modulates doxorubicin-induced senescent phenotype in hPSC-derived endothelial cells

Cory P Johnson PhD¹, Abigail L Stathis², Abigail R McNally BS¹, Harish Krishna Nagarajan¹, Frederic P. Bonnet PhD¹, Hermann Haller MD¹

¹MDI Biological Laboratory, Bar Harbor, ME, USA. ²Colby College, Waterville, ME, USA

- Aging leads to the loss of microvasculature.
- The SGLT2 inhibitor, Dapagliflozin, prevents age-related microvascular loss in killifish.
- Dapagliflozin directly modulates the profile of senescent human endothelial cells in culture.

M44

The role of PolyA Binding Protein Cytoplasmic 1 (PABPC1) in suppressing inflammatory Extracellular Matrix mRNA in endothelial cells to maintain Vascular Homeostasis

Hina Iqbal Ph.D., Jesse Cullison, Emily Clifford, Katherine Hamm, Ziqing Liu

Medical College of Wisconsin, Milwaukee, Wisconsin, USA

- RBPs are understudied in vascular biology, especially in EC activation and vascular homeostasis
- Our results challenge the paradigm that PABPC1 universally binds and stabilizes all poly(A) mRNAs
- A novel role of PABPC1 in vascular homeostasis by suppressing specific ECM mRNAs

M45

Bone marrow resident macrophages are essential component of bone marrow endothelial niche

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- Bone marrow macrophages are required for BM endothelial expansion.
- Macrophages may support sinusoidal networks through paracrine and contact mechanisms.
- MIF, TWEAK, and EDA are among candidate macrophage-to-endothelial pathways.

LYMPHATIC BIOLOGY I

M46

VEGF-D-induced intraosseous lymphangiogenesis drives site-specific heterotopic bone resorption

Michael Dellinger¹, Neda Vishlaghi¹, Joseph Rutkowski², Benjamin Levi¹

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- Heterotopic ossification is abnormal bone formation in soft tissues.
- VEGF-D-induced lymphangiogenesis promotes heterotopic bone resorption.

- Lymphangiogenesis is a promising therapeutic strategy for HO.

M47

Computational modeling links lymphatic endothelial signaling dynamics to junction state and predicts lymphatic responses to obesity

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- A quantitative model and "zippering propensity" metric link LEC signaling dynamics to junction state
- RAC1–RhoA antagonism and dephosphorylation rates control junction state across heterogeneous cells
- Obesity engages RAC1–RhoA in mesenteric, but not adipose, lymphatics

M48

Lymphatic mechanisms in tertiary lymphoid structure formation

Guilherme Pedron Formigari PhD¹, Marcella Neves Datilo PhD¹, Magdalena Karolak¹, Hayoung Cho MSc¹, Harini Bagavant PhD², Illaria Del Gaudio PhD³, Ravi Keshari PhD¹, Lijuan Chen¹, Zheila Azartash-Namin MSc¹, Nathan Pezant MSc⁴, Jennie Criley PhD⁵, Courtney Griffin PhD¹, Florea Lupu PhD¹, Gwendalyn Randolph PhD⁶, Chunliang Li PhD⁷, Umesh Deshmukh PhD², Eric Camerer PhD³, Xin Geng PhD¹, Sathish Srinivasan PhD¹

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- S1PR1 limits lymphatic overgrowth and intestinal TLS formation.
- Lymphatic vessel overgrowth, microbiota, macrophages, and TNF- α drive TLS formation.
- S1PR1–TNF- α crosstalk regulates intestinal TLS development.

M49

Comparative analysis of cardiac lymphatic vessels across regenerative and non-regenerative heart injury models

Yuhan Sun PhD, Jiang Qian MD. PHD., Xidi Feng PhD, Ching-Ling Lien PhD

Children's hospital Los Angeles, Los Angeles, CA, USA

- ADMA and ACKR3 regulate cardiac lymphatic-mediated immune clearance during heart regeneration
- Cross-species comparison reveals pathways underlying cardiac regeneration.
- snRNA-seq identifies lymphatic and immune signaling during cardiac repair.

M50

Interrogating the effects of intraflagellar transport protein depletion on lymphatic endothelial gene expression in vitro

Evan Feeldy¹, Nile Laake¹, Prafulla Shrestha¹, Jose L. Gonzalez Hernandez², Darci M. Fink¹

¹South Dakota State University, Department of Chemistry, Biochemistry & Physics, Brookings, SD, USA. ²South Dakota State University, Department of Agronomy, Horticulture & Plant Science, Brookings, SD, USA

- Primary cilia sense shear stress in other cell types and are present on LECs
- Depletion of primary cilia by KO/KD of IFT proteins changes LEC gene expression in vitro
- Primary cilia may contribute to LEC mechanosensation

M51

Intraflagellar transport proteins in lymphatic endothelial cells promote lymphatic valve formation

Prafulla Shrestha, Caden Johnson, Noah Johnson, Alex Schemek, Evan Feeldy, Nile Laake, Darci M Fink
South Dakota State University, Brookings, SD, USA

- Primary cilia sense shear stress in other cell types, are present on LECs, and function via IFT
- Lymphatic valves form during development and inflammation and can be tracked by live imaging
- LEC-specific IFT KO impairs lymphatic valve formation in various tissues

M52

Investigating the molecular mechanisms underlying diffuse pulmonary lymphangiomatosis

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- The pathogenic mechanisms underlying diffuse pulmonary lymphangiomatosis (DPL) remain unclear.
- Genetic mouse models recapitulate key features of DPL.
- Spatial transcriptomics reveals molecular mechanisms underlying DPL.

MECHANOBIOLOGY AND MECHANOTRANSDUCTION I

M53

Calcium-dependent AC1-cAMP signaling mediates mechanotransduction in vascular smooth muscle and cAMP responses are reduced in hypertension

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- Mechanical stretch triggers cAMP generation in vascular smooth muscle.
- Calcium-dependent AC1 mediates mechanosensitive cAMP signaling in vascular smooth muscle cells.
- Mechanosensitive cAMP responses are reduced in hypertension.

M54

Flow-dependent regulation of spatiotemporal calcium signaling in endothelial cells

Soon-Gook Hong Ph.D., Amy Nguyen B.S., Julia Jeannine Mack Ph.D.

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- Blood flow patterns generate distinct spatiotemporal Ca²⁺ signaling in endothelial cells.
- ER remodeling orchestrates flow-dependent Ca²⁺ signaling to control endothelial gene expression.
- The NFAT2–CXCR4 axis limits endothelial inflammation under disturbed flow.

MICROCIRCULATION I

M55

Pannexin 1 ablation compromises vascular smooth muscle cell differentiation

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- Panx1 knockout vascular smooth muscle cells have reduced differentiation capacity and signaling.
- A novel AlphaPulldown pipeline was used to model direct protein-protein interactions.
- PANX1 binds the actin cytoskeleton, suggesting its role in stabilizing contractile machinery.

M56

Targeting oxidative stress via NAD⁺ modulation to mitigate diabetes-associated coronary microvascular disease and heart failure with preserved ejection fraction

Anita Kaw M.D., Ph.D.¹, Tao Yu Ph.D.¹, Eshant Bhatia Ph.D.², Kanami Irie², Laxminarayanan Krishnan MBBS, Ph.D., M.S.², Ankur Singh Ph.D.², Laura Hansen Ph.D.¹

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- NAD⁺ reduces diabetes-induced endothelial inflammation and mitochondrial oxidative stress.
- Supplemental NAD⁺ attenuates adverse myocardial remodeling in diabetic mice.
- NAD⁺ augmentation may preserve cardiac function despite persistent diabetes.

M57

Relationship between vascular reactivity and medial aortic diameter in patients with POTS and PSWT

Arisa Khan, Heer Shah BA, Tayyab Tanvir MBBS, MPhil, Amna Butt MBBS, MPH, Amer Suleman MD

The Heartbeat Clinic, Dallas, Texas, USA

- Vascular reactivity differences in POTS and PSWT
- Aortic structure changes in autonomic dysfunction
- Structural and functional vascular changes in POTS and PSWT

M58

Lysophosphatidylcholine and microvascular dysfunction in knee osteoarthritis

Hanyu Jiang MSc, Tom Appleton MD, PhD

Western University, London, Ontario, Canada

- Synovial microvascular dysfunction is a key feature of knee osteoarthritis
- Abnormal LPA-LPAR6 signaling disrupts synovial endothelial function in the knee joint organ
- Targeting the LPA signaling axis may improve synovial microvascular outcomes in knee osteoarthritis

M59

Oxidative stress-mediated activation of tissue transglutaminase is a key driver of vascular stiffening in aging and obesity

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- Arterial stiffening is an independent predictor of hypertension in obese and older individuals
- Aortic stiffness, oxidative stress markers, and TG2 expression increase with age and obesity
- TG2 knockdown or inhibition prevents oxidative stress-induced F-actin formation and VSMC stiffening

VASCULAR ANOMALIES AND MALFORMATIONS I

M60

Activating variants of RIT1 in central conducting lymphatic anomaly

Lola Zerbib PharmD, PhD

Unit on Vascular Malformations, Division of Intramural Research, Eunice Kennedy Shriver National Institute of Child Health and Human Development, Bethesda, Maryland, USA

- RIT1 variants identified as a genetic cause of CCLA in participant cohort
- Zebrafish model recapitulates lymphatic and vascular defects of RIT1-CCLA
- Inhibitor screening and scRNA-seq could guide precision therapies for RIT1-CCLA

M61

Identification of novel therapeutic compounds for capillary malformations based on phosphoproteomics mapping of calcineurin-NFAT-DSCR1.4 signaling

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³Swammerdam Institute for Life Sciences, Amsterdam, Netherlands

- Quantitative phosphoproteomics identifies calcineurin–NFAT signaling as a key pathogenic pathway
- Patient samples confirm endothelial NFAT dysregulation and increased DSCR1 expression.
- FK506 and phenotypic drug screening support targeted therapy development for Gαq-R183Q driven CMs.

M62

PIP2 steal hypothesis: overactive Gαq-PLCβ3 reduces PIP2 available for the PI3Kα/AKT1 pathway in GNAQ p.R183Q capillary malformations

Joyce Bischoff PhD, Annegret Holm MD, Jill Wylie-Sears MS, Patrick Smits PhD, Arin K Greene MD, Sana Nasim PhD
Boston Children's Hospital and Harvard Medical School, Boston, Massachusetts, USA

- GNAQ p.R183Q driven constitutive activation of PLCβ3 reduces PIP2 available for PI3K/AKT signaling.
- Restoring pAKT in GNAQ p.R183Q endothelium normalizes angiopoietin-2 and endothelial permeability.
- Pharmacologic activation of PI3Kα/AKT signaling in CMs represents a novel therapeutic concept.

M63

The role of second heart field progenitors in venous development and malformation

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¹Rutgers Biomedical Health Sciences, Newark, NJ, USA. ²New Jersey Medical School, Newark, NJ, USA

- VEGFR2 is not strictly required for venous ECs recruitment from SHF-derived progenitors
- The loss of Etv2 significantly impairs the contribution of SHF-derived progenitors to venous ECs
- Tbx1 is required for SHF contribution to the cardinal vein and venous valves.

M64

Role of PTPN14 in regulating BMP9–Hippo signaling in HHT-Associated arteriovenous malformations

Anirudh Gaur Ph.D., Chenyang Xu B.S., Krystal Aqua B.S., Zahra Sajid B.S., Daniah Thompson Belefond MD/PhD
University of California, Davis, Davis, CA, USA

- PTPN14 as genetic modifier in HHT
- Integration of TGF-β/BMP9 and Hippo–TAZ signaling
- PTPN14 as a novel regulator of endothelial signaling and a potential therapeutic target for HHT

VASCULAR FUNCTION IN HEALTH AND DISEASE I

M65

Endothelial loss of KLF4 drives a PE-like phenotype

Emily Meredith PhD, Andrew Meredith BA, Martin Schwartz PhD

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- PE is manageable in the short-term, but long-term impacts on CV health remain unaddressed.

- Understanding the mechanism of PE impact on ECs can help mitigate long-term negative outcomes.
- KLF2/4 are implicated in PE via eNOS KO and L-NAME studies. We confirm its involvement in this model

M66

CRL5 as a novel modulator of vascular function in health and disease

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- CRL5 is a novel regulator of vascular smooth muscle and blood pressure control.
- CRL5 shows sex-dependent effects in hypertension, protective in males and detrimental in females.
- These findings have translational potential for new antihypertensive treatments.

M67

Pulmonary arterial hypertension exacerbated by SOX17 deficiency and high shear stress is reversed by TMEM100 via dual regenerative and anti-inflammatory programs

Chongyang Zhang, Lingli Wang, Mauro Lago-Docampo, Olga Pushkarev, Yannick Lee-yow, Tsutomu Shinohara, Jesse Engreitz, Marlene Rabinovitch
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- SOX17 loss with high shear stress drives endothelial dysfunction in pulmonary hypertension.
- AAV-mediated TMEM100 delivery reverses PAH in SOX17-deficient mice exposed to PA high shear stress.
- TMEM100 links dual programs of endothelial regeneration and anti-inflammation.

M68

Circulating mitochondrial DNA induces endothelial dysfunction in pregnancy and a pro-contraction shift postpartum

Nataliia Hula PhD, Renee Oliveira da Silva PhD, Desirae Escalera BS, Styliani Gouloupoulou PhD
Loma Linda University, Loma Linda, California, USA

- Exposure to cell-free mitochondrial DNA in pregnancy induces maternal aortic endothelial dysfunction
- Mitochondrial DNA insult in pregnancy impacts coronary and uterine artery function postpartum
- ccf-mtDNA challenge has short-term and long-term impact on maternal vascular adaptation

M69

Multiphoton-Confocal correlative imaging reveals novel molecular architecture and cellular dynamics in living blood vessels

Olena Kamenyeva PhD¹, Juraj Kabat PhD¹, Steven Brooks PhD¹, Md Abdul Hakim PhD¹, Sundar Ganesan PhD¹, Paola C Valenzuela Leon PhD¹, Erin McCaffrey PhD¹, Jeremy Davis PhD², Xuming Tang PhD³, Eric Calvo PhD¹, Manfred Boehm PhD³, Hans Ackerman PhD¹, Owen M Schwartz PhD¹

¹NIH/NIAID, Bethesda, Maryland, USA. ²NIH/NCI, Bethesda, Maryland, USA. ³NIH/NHLBI, Bethesda, Maryland, USA

- Combined MP/1P imaging reveals alpha- and beta-globin binding eNOS in vascular endothelium
- Imaging Mtb infected vascular organoids enables new model for studying TB immunity
- Light-induced thrombus formation shows how mosquito salivary proteins inhibit coagulation

M70

Ensheathing pericyte dysfunction and cerebral blood flow impairments in the 5xFAD mouse model of Alzheimer's disease

Catherine D Bland, Jessica Resillas Msc, John D Rosario, Dmytro Koval, Jillian Stobart PhD
University of Manitoba, Winnipeg, MANITOBA, Canada

- Early calcium and blood flow dysregulation are both time- and branch-specific in prodromal AD.
- Ensheathing pericyte calcium changes are associated with impaired vasomotion in AD.
- Ensheathing pericyte dysfunction contribute to early vascular changes in AD.

M71

Senescent pulmonary microvascular endothelial cells impair pericyte function via senescence-associated secretory phenotype signaling in PAH

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¹Stanford University, Palo Alto, CA, USA. ²Indiana University, Indianapolis, IN, USA

- Endothelial cells exposed to H₂O₂ demonstrate increased senescence and dysfunction
- Conditioned medium from senescent EC causes PC senescence and dysfunction
- EC-PC interaction is disrupted through SASP signaling and disturbs vessel integrity

ANGIOGENESIS II

T01

Rg2 improves angiogenic function in aged endothelial cells

Shijie Jin PhD

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- Endothelial aging reduces angiogenic capacity.
- Rg2 restores angiogenesis and proliferation in aged endothelial cells.
- Rg2 shows efficacy comparable to direct mitochondrial transfer.

T02

Membrane remodeling at force-dependent endothelial junctions guides collective migration during vascular development

Stephan Huveneers

Amsterdam UMC - University of Amsterdam, Amsterdam, Netherlands

- Nanoscale membrane curvature appear at endothelial junctions during migration.
- Curved junctional membranes recruit BAR proteins to coordinate junction remodeling.
- BAR protein-mediated remodeling enables collective endothelial migration and vascular development

T03

Loss of endothelial nucleoporin93 impairs angiogenesis through RhoA/ROCK hyperactivity

Miles D Eckrich B.S., Tung D Nguyen PhD, Yumna Z Khan, Michael A Winek, Monica Y Lee PhD

University of Illinois Chicago, Chicago, IL, USA

- Nup93 regulates endothelial migration in vitro via Sun1 expression.
- Endothelial-specific Nup93 loss leads to impaired retinal angiogenic outgrowth.
- Nup93 acts a key regulator of angiogenesis, and may contribute to age-related angiogenic defects.

ATHEROSCLEROSIS II

T04

Smooth muscle cell TNF-TNF Receptor 1 signaling in atherosclerosis decreases markers of plaque stability in male but not female mice

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- TNF drives smooth muscle cell lineage expansion in atherosclerotic plaques
- TNFR1 contributes to atherosclerotic plaque progression in male, but not female, mice
- SMCs express TNFR1 in plaques, which induces structural changes for plaque instability in male mice

T05

Matrix regulation of endothelial priming during atherosclerosis

Gerardo A Cruz-Marquez BS¹, Cyrene Ben Dhaou PhD¹, Anthony W Orr PhD²

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- Endothelial cells' inflammatory response is regulated by intimal extracellular matrix proteins (ECM)
- Inhibition of ECM receptors, integrin $\alpha 5$ & αv , decreases endothelial response to cytokines.
- ECM receptors, integrin $\alpha 5$ & αv , prime endothelial cells through NF- κ B expression.

T06

Role of endothelial Nck1 in atherosclerosis and Metabolic-Associated Steatotic Liver Disease

Cyrene Ben Dhaou PhD, Brenna Pearson-Gallion PhD, Elizabeth D Irwin Ms, Ali Gerardo Cruz Markez Ms, Arif Yurdagul PhD, Oren Rom PhD, A. Wayne Orr PhD

LSU Health Shreveport, Shreveport, LA, USA

- Endothelial Nck1 deletion protects against both atherosclerosis and MASLD
- Nck1 promotes endothelial inflammation through the PERK-eIF2 α -ATF3 stress pathway
- Targeting Nck1 may treat cardiometabolic disease without impairing ischemic angiogenesis

T07

Exploring functional divergence in human and chimpanzee endothelial cells identifies a novel regulator of LDL uptake

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¹Department of Biology, Stanford University, Stanford, CA, USA. ²Howard Hughes Medical Institute, Chevy Chase, MD, USA

- studying human and chimpanzee endothelial cells differences provide insights into atherosclerosis
- Human endothelial cells take up more LDL than their chimpanzee counterparts
- mTORC2 inhibitions lowers LDL uptake by endothelial cells

T08

Chronic exposure to HIV proteins accelerates the development of atherosclerosis via T cell and IL-1 α dependent mechanisms

Paula Rodrigues de Barros PhD¹, Rahul Shivaji Patil PhD¹, Shengzhou Wen PhD¹, Beryl N. Khakina MS¹, Ishara M.G. Menik BS¹, Jing Ma¹, Rafal Pacholczyk PhD², Simone Kennard PhD¹, Alexander D. Verin PhD¹, Eric J. Belin de Chantemele PhD¹

¹Vascular Biology Center, Medical College of Georgia, Augusta, GA, USA. ²Department of Biochemistry and Molecular Biology, Medical College of Georgia, Augusta, GA, USA

- Chronic HIV proteins exposure accelerates plaque formation
- IL-1 α and CD3+ T cells are causal targetable mediators of HIV-associated atherosclerosis
- Mechanisms converge on an IL-1 α -NOX1 signaling cascade that mediates endothelial barrier disruption

BIOENGINEERING I

T09

Investigating vascular remodeling in pulmonary arterial hypertension using a 3D bioengineered platform

Kaveeta Kaw MD/PhD¹, Vaahini Badre Narayanan¹, Michael Clark¹, Elaine Do², Izzie Thompson¹, Ethan Peng¹, Ian Lin¹, Wyatt DeBord BS², Maher Saadeh PhD³, Vahid Serpooshan PhD³, Holly Bauser-Heaton MD/PhD³, Hananeh Fonoudi PhD³

¹Emory, Atlanta, GA, USA. ²Georgia Tech, Atlanta, GA, USA. ³UTHealth, Houston, TX, USA

- Modeling pulmonary vascular remodeling
- Pressure-controlled human pulmonary artery platform

- Mechanistic studies of pulmonary arterial hypertension

T10

Engineering a vascularized human skin equivalent for the study of inflammatory skin conditions

Hana Kubo¹, Casey Miller¹, Hanadi Almazroue¹, Andrew JH Sedlack², Daniel Rivas¹, Suji Choi¹, Hope Burks¹, Quinn Roth-Carter¹

¹Biohub, Chicago, Illinois, USA. ²Department of Biomedical Engineering, Northwestern University, Evanston, Illinois, USA

- Built a vascularized dermis equivalent to model immune cell recruitment during inflammation.
- Loaded microfluidics chip with human primary fibroblasts and endothelial cells.
- Endothelial cells self-organized in the human dermis equivalent with interstitial flow.

T11

Adapting a micro blood-brain barrier model to study endothelial progenitor cell functional and molecular mechanisms underlying cognitive resilience

Ulia Lopatin¹, Lien M. Reolizo MSc, PhD¹, Daniel Nation PhD², Christopher C.W. Hughes PhD¹

¹University of California, Irvine, Irvine, CA, USA. ²University of Southern California, Los Angeles, CA, USA

- Adapted 3D in vitro culture system of blood-brain barrier on a chip.
- Enabling high throughput study of patient endothelial progenitor cells barrier properties.
- Platform for investigating endothelial mechanisms of cognitive resilience in Alzheimer's disease.

CARDIOVASCULAR DISEASES II

T12

DNA damage response-mediated suppression of CD73 in medial arterial calcification

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¹University of Pittsburgh, Pittsburgh, PA, USA. ²UPMC, Pittsburgh, PA, USA

- Medial arterial calcification is a key pathology in patients with peripheral artery disease.
- ACDC, a genetic disease caused by mutations in CD73, results in medial arterial calcification.
- DNA damage response may decrease CD73 expression and lead to vascular calcification

T13

Loss of UTY recapitulates LOY-associated macrophage aging signatures in murine heart

Anny Thuy Duong T Tran BS¹, Nicholas Chavkin PhD^{1,2}

¹Seattle Children's Research Institute, Seattle, WA, USA. ²University of Washington School of Medicine, Seattle, WA, USA

- Single-cell profiling revealed shared macrophage programs in UTY-deficient and LOY aging hearts.
- UTY loss reproduced LOY-like pro-fibrotic signaling, ECM remodeling, and cell crosstalk.
- UTY deficiency may underlie LOY-associated cardiac remodeling and fibrosis during aging.

T14

Cardiometabolic disease drives the emergence of a macrophage-like basophil population in the spleen

Wyatt Schug, Skylar A Loeb, Luke S Dunaway, Zuzanna Juskiewicz, Tajbir Raihan, Brant Isakson

University of Virginia, Charlottesville, VA, USA

- Oxidative stress promotes basophil proliferation and inflammatory gene programs.
- Cardiometabolic disease drives tissue-specific changes in basophil phenotype.
- Splenic basophils acquire macrophage-like transcriptional signatures during disease.

T15

Inflammatory regulation of FN3K links vascular inflammation to mitochondrial dysfunction

Nazia Aga PhD, Mohmad Gyth Zouabi PhD, Kinza Khan Postdoctoral Researcher, Paraskevi Goggolidou Dr, Paul Kirkham Professor

University of Wolverhampton, Wolverhampton, United Kingdom

- Inflammatory signalling suppresses FN3K in vascular cells.
- FN3K loss disrupts mitochondrial and transcriptional homeostasis.
- Metformin induces FN3K, but inflammation limits this response.

T16

PROX1-Dependent regulation of connexin 37 preserves cardiac valve integrity

Marcella Neves Datilo PhD¹, Yen-Chun Ho PhD¹, Zheila J Azartash-Namin BS¹, Guilherme Pedron Formigari PhD¹, Kumar Subramani PhD¹, Natalia Lopez-Andres PhD², Harold M. Burkhardt MD³, Jasimuddin Ahamed PhD¹, Xin Geng PhD¹, R Sathish Srinivasan PhD¹

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³University of Oklahoma College of Medicine, Oklahoma City, OK, USA

- PROX1 regulates Cx37 expression in cardiac valve endothelial cells.
- Loss of CX37 causes structural and functional changes in heart valves.
- PROX1-CX37-PDGF-B signaling axis contributes to the progression of heart valve disease.

T17

Hepatocyte-derived Galectin-1 couples atherogenic dyslipidemia to defective macrophage efferocytosis

Heena Agarwal, Lale Ozcan

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- Metabolic stress induces Galectin-1, uncovering a novel liver-to-vascular signaling axis
- Hepatic Galectin-1 disrupts systemic lipid clearance to drive atherogenic dyslipidemia
- Galectin-1 triggers CD45-dependent MerTK/LRP1 shedding and defective efferocytosis

CELL BIOLOGY AND SIGNALING II

T18

HDAC11 epigenetically regulates endothelial-to-mesenchymal transition and is dysregulated in diabetic wound repair

James Shadiow PhD, Tyler M. Bauer MD, He Zhang MS, Kevin D. Mangum MD, PhD, Amber Estor BS, Lindsey D. Hughes PhD, Matthew White Jr. BS, Amrita D. Joshi PhD, Amy Stark PhD, Yana Astter-Sompel BS, Gabriela Saldana de Jimenez MS, Sabrina Rocco MS, Samuel Buckley BS, Jadie Y. Moon PhD, Jorge J. Reyes-Arbutas BS, Lam C. Tsoi PhD, Johann E. Gudjonsson MD, PhD, Andrea T. Obi MD, Frank M. Davis MD, Katherine A. Gallagher MD

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- Diabetes promotes maladaptive EndMT during wound repair.
- HDAC11 mediates chromatin control of EndMT transition.
- HDAC11 fine-tunes endothelial plasticity and angiogenic wound healing.

T19

FAT1-Dependent extracellular vesicle signals regulate smooth muscle cell function and vascular remodeling

Amado A Quintar MD PhD, Dario F Riascos-Bernal MD PhD, Nicholas ES Sibinga MD

Albert Einstein College of Medicine, Bronx, NY, USA

- Extracellular vesicles (EVs) express the atypical cadherin FAT1
- Loss of FAT1 converts smooth muscle cells-derived EVs into pro-migratory, pro-remodeling signals
- FAT1-containing EVs restrain vascular SMC proliferation and metabolism

T20

Suppression of STAT3 activity in acute lung injury reduces vascular permeability and macrophage infiltration

George Moussa¹, Li Wang PhD¹, David A Frank², Luke H Hoepfner PhD^{1,3}

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- Pharmacologic STAT3 inhibition reduces vascular permeability and inflammation in lung injury models
- Vascular permeability is decreased in endothelium-specific STAT3 KO mice challenged with lung injury
- Suppression of STAT3 activity reduces pulmonary macrophage infiltration in acute lung injury models

T21

A Wnt mimetic restores pericyte coverage and vascular integrity by increasing PDGFB signaling

Miranda Howe¹, Jacklyn Levey^{1,2}, Kaia Douglas^{1,3}, Emmanuel Odame¹, Neal Rajvansh^{1,4}, Ha-Neul Jo^{1,5}, Lingling

Zhang^{1,6}, Christina Chung^{1,7}, Heidi Roehrich¹, Somasekar Seshagiri⁸, Stephane Angers⁹, Zhe Chen¹, Harald Junge¹

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⁶University of St. Thomas, St Paul, MN, USA. ⁷Chicago College of Osteopathic Medicine, Chicago, IL, USA. ⁸AntlerA Therapeutics, Foster City, CA, USA. ⁹University of Toronto, Toronto, ON, Canada

- Wnt mimetic promotes pericyte proliferation and coverage in the retina
- Wnt mimetic restores Pdgfb mRNA expression levels
- Preliminary data suggests Wnt mimetic alleviates hemorrhages in the retina and brain

T22

The role of HDAC7 in ERG-mediated transcription

Kyla Vazquez¹, Cecilia Careaga MS¹, Anna Golebiewski PhD¹, Alhan Mehrabi Yazdi MS¹, Paul Langlais PhD², Casey Romanoski PhD¹

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- ERG/HDAC7 transcriptional regulatory axis
- Endothelial cell homeostasis
- Transcriptional co-factors

CEREBROVASCULAR HEALTH AND DISEASE II

T23

Oxidative stress drives vascular damage in cerebral small vessel disease CADASIL

Kevin Emmerich Ph.D., Manfred Boehm M.D.

National Heart, Lung, and Blood Institute, Bethesda, MD, USA

- Oxidative stress in cerebral small vessel disease
- Vascular organoids as disease model
- Toxic protein aggregation induces mitochondrial stress

T24

Endothelial Pannexin1 regulates cerebral vascular function in a mouse model of Alzheimer's disease

Amanda K Mauro PhD¹, Mauricio Ruiz Soler MS¹, Maria Tomás-Gracia MS¹, Colleen K Duffy¹, Miranda E Good PhD^{1,2}

¹Tufts Medical Center, Boston, MA, USA. ²Tufts University, Boston, MA, USA

- Vascular dysfunction is causally linked to Alzheimer's disease.
- We previously demonstrated that endothelial Pannexin1 regulates cerebral vascular function.
- We aim to examine the role of endothelial Pannexin1 in models of Alzheimer's disease.

T25

Capsaicin in obesity and aging: exploring cerebrovascular changes with nutraceutical intervention

Sharon Negri PhD, Madison Milan PhD, Jennifer Ihuoma, Rakesh Rudraboina, Aleksandra Kosmider, Stefano Tarantini

OUHC, Oklahoma City, OK, USA

- Obesity accelerates age-related neurovascular dysfunction.
- Capsaicin preserves cerebrovascular function during aging and obesity.
- Dietary capsaicin targets vascular dysfunction through TRPV1.

T26

Longitudinal assessment of cerebrovascular recovery reveals endothelial mitochondrial remodeling after ischemic stroke

Sharon Negri, Roland Patai, Madison Milan, Rakesh Rudraboina, Aleksandra Kosmider, Jennifer Ihuoma, Stefano Tarantini

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- In vivo imaging reveals age-dependent vascular remodeling after ischemia.
- Endothelial mitochondrial dysfunction contributes to poor stroke recovery with aging.
- Mitochondrial targeting enhances cerebrovascular repair in aged mice.

T27

Epigenetic targeting of EZH2 rescues SMC differentiation in Smooth Muscle Dysfunction syndrome

Jose E Esparza Pinelo B.S., Shivi Garg M.S., Alexei Robin B.S., Mario Carrera B.S., Kennedy Zinn M.S., Bree M Lege PhD, Dianna M Milewicz MD, PhD, Callie S Kwartler PhD

University of Texas Health Science Center at Houston, Houston, TX, USA

- ACTA2 p.R179 disrupts SMC differentiation and causes SMC-filled occlusive cerebrovascular lesions
- Acta2R179C/+ SMCs have elevated global levels of H3K27me3, which are reduced by EZH2 inhibitor
- An EZH2 inhibitor rescues SMC differentiation in vitro and SMC occlusive lesions in the brain

T28

Penetrance of ALS-Like features following endothelial deletion of TDP-43 is determined by genetic background in an animal model

Samantha DeRosa¹, Alyssa Sirianni¹, Ashok Cheemala PhD¹, Amy Kimble¹, Emily Burrage PhD¹, Evan Jellison PhD¹, Bo Reese PhD², Jenna Bartley PhD¹, Riqiang Yan PhD¹, Patrick Murphy PhD¹

¹University of Connecticut, Farmington, CT, USA. ²University of Connecticut, Storrs, CT, USA

- Endothelial contributions to ALS
- Genetic modifiers of vascular dysfunction
- Combination of SNP based genetic analysis and single nuclei sequencing to dissect disease modifiers

T29

PROX1-driven endothelial reprogramming disrupts the blood-brain barrier in neurovascular diseases

Sara González-Hernández PhD, Yoh-suke Mukouyama PhD

National Heart, Lung, and Blood Institute, Bethesda, MD, USA

- PROX1 drives endothelial reprogramming and BBB dysfunction
- Ectopic PROX1 disrupts BBB integrity and increases vascular permeability
- BBB integrity requires active repression of lymphatic endothelial identity

DEVELOPMENT II

T30

Characterization of soluble platelet-derived growth factor receptor beta during gestation

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- Soluble PDGFR β is present in multiple isoforms in human serum.
- A correlation between early gestational age and low relative soluble PDGFR β appears to exist.
- The relative quantities of soluble PDGFR β appear to change rapidly after birth.

T31

Adams-Oliver syndrome CdGAP variant Q683X drives defective placental development in a mouse model

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- CdGAP loss disrupts placental labyrinth morphogenesis and maternal-fetal exchange.
- CdGAP mutant embryos develop hypovascularization, edema, and atrioventricular cushion defects.
- ARHGAP31 is integrated into trophoblast differentiation and extravillous trophoblast regulation.

T32

Spatiotemporal dynamics of smooth muscle cell phenotypic switching during ductus arteriosus remodeling

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- Spatial transcriptomics reveals prenatal MYOCD upregulation and contractile SMC accumulation.
- After birth, SMCs switch from a contractile to a synthetic phenotype as MYOCD expression declines.
- Spatial transcriptomics also reveals a similar synthetic SMC population in the postnatal human DA.

T33

CCR2+ cells are critical for normal aortic development

Rira Choi M.S/B.S.E., Jay D Humphrey Ph.D.

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- DTAs in CCR2 null mice exhibits intramural delamination in physiologic loading ex vivo.
- DTAs in CCR2 null mice reach mechanical homeostasis at different rates than in control mice.
- Fibroblast-macrophage crosstalk appear to be important not just in disease, but also in homeostasis.

ENDOTHELIUM IN HEALTH AND DISEASE II

T34

FAT1 regulation of endothelial repair

Oscar Bermudes MS, Nicholas Sibinga MD

Albert Einstein College of Medicine, Bronx, New York, USA

- FAT1 regulates endothelial metabolism and proliferation during arterial repair

- FAT1 fragments have distinct mitochondrial and nuclear functions
- Endothelial FAT1 loss may accelerate reendothelialization after vascular injury

T35

ERG as a therapeutic to reverse EndMT and restore endothelial cell identity

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- E-26 transformation specific (ETS) family transcription factor (ERG)
- Endothelial cell identity
- Endothelial-to-mesenchymal transition (EndMT)

T36

Coronary microvascular expansion and maturation across somatic cardiac growth revealed by single cell RNA sequencing

Kaitlyn Ford¹, Nicholas W Chavkin^{2,3,4}

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- 4- and 8-week-old mouse hearts correspond to 7- and 14-year-old human hearts, respectively.
- Murine somatic cardiac growth curves do not differ between male and female mice.
- Coronary endothelial cell proportion increases relative to other cell types during somatic growth.

T37

Combined uremic and hemodynamic stress drives endothelial dysfunction in hemodialysis patients

Dylan Cook, Gianna Gordon, Marc Wahab, Yun Fang PhD, Sara Gaines MD, Mary Hammes DO, Anindita Basu PhD
University of Chicago, Chicago, IL, USA

- Cellular response to multimodal stresses.
- In vitro systems of end-stage renal disease.
- Weibel-Palade bodies' role in thrombosis.

T38

Dapagliflozin and Empagliflozin inhibits iNOS participation in endothelial cells exposed to Doxorubicin

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- SGLT2 inhibitors prevent DOXO-induced iNOS-mediated NO overproduction.
- SGLT2 inhibitors restore endothelial redox balance after doxorubicin exposure.
- SGLT2 inhibitors modulate endothelial dysfunction independent of cell viability.

T39

Endothelial iron accumulation mediates coronary rarefaction in chronic kidney disease

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- Chronic kidney disease may promote endothelial iron accumulation.

- Endothelial iron dysregulation promotes microvascular rarefaction in chronic kidney disease.
- Targeting endothelial iron uptake rescues rarefaction and improves myocardial perfusion in CKD.

T40

Defining the cellular dynamics and molecular programs governing vascular regeneration during cutaneous wound healing in zebrafish

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¹University of Pittsburgh, Pittsburgh, PA, USA. ²National Institutes of Health, Bethesda, MD, USA

- Impaired vascular regeneration contributes to defective wound healing in aging and disease.
- Longitudinal live imaging reveals immune-vascular dynamics and age-dependent repair defects.
- Endothelial translomics identifies dynamic regenerative states during vascular repair.

T41

Isoform-specific regulation of VE-PTP/PTPRB controls endothelial function

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¹University of Chicago, Chicago, IL, USA. ²National University of Singapore, Singapore, Singapore

- Vascular endothelial function
- Alternative gene expression
- Endothelial junction integrity

T42

Doxorubicin impairs arterial specification of coronary microvascular endothelial cells

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- Doxorubicin promotes coronary endothelial senescence and disrupts cell cycle regulation
- Coronary microvascular endothelial cells show impaired arterialization during doxorubicin treatment
- Human doxorubicin-induced cardiomyopathy samples exhibit coronary disruptions in vascular remodeling

T43

Capillary endothelial cell dysfunction and resilience in lung tissue repair

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¹Yale University, New Haven, Connecticut, USA. ²National Cancer Institute, Frederick, Maryland, USA

- Endothelial cell response to acute inflammation in the lung can cause long-term tissue damage.
- Injury-induced transcriptional changes to endothelial cells cause changes in cell behavior.
- Disease-associated lung endothelial cell states are induced by localized tissue hypoxia.

INFLAMMATION I

T44

Inter-Alpha inhibitor protein restores retinal vascular homeostasis by promoting reparative angiogenesis and limiting microglia-driven inflammation in oxygen-induced retinopathy

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²Department of Pathology and Laboratory Medicine, Brown University, Providence, USA. ³Laboratoire d'Oncologie Moléculaire, Département de Chimie, Université du Québec à Montréal, Montreal, Quebec, Canada

- IAIP restores retinal vascular homeostasis by limiting microglia-driven inflammation.
- IAIP promotes reparative angiogenesis and preserves endothelial function.
- IAIP reduces retinal vaso-obliteration in ischemic retinopathy rat model.

T45

Epigenetic mechanisms of endothelial inflammatory hyporesponsiveness in diabetic wound repair

Amber Estor, James Shadiow, Yana Astter-Sompel, Jorge Reyes-Arbutas, Amrita Joshi, Gabriela Saldana de Jimenez, Sabrina Rocco, Amy Stark, Sam Buckley, Matthew White Jr., Jadie Moon, Lindsey Hughes, He Zhang, Lam Tsoi, Johann Gudjonsson, Andrea Obi, Frank Davis, Katherine Gallagher
Michigan Medicine, Ann Arbor, USA

- Cell-intrinsic mechanism of endothelial inflammatory hyporesponsiveness in diabetic wound healing.
- Cytokine-treated DMVECs enhanced macrophage migration, supporting EndoT communication.
- ESCO2 as a potential epigenetic target to restore endothelial-to-immune communication.

T46

Oxidatively stressed red blood cells as drivers of lymphocyte activation

Megan E Butler, Sushma Bharrhan PhD, Tashawna Esmond BS, Matthew D Woolard PhD, Karen Y Stokes PhD
LSU Health Shreveport, Shreveport, LA, USA

- Sick red blood cells exhibit oxidative dysfunction and altered immune signaling.
- Sick red blood cells interact directly with lymphocytes.
- Sick red blood cells induce subset-specific phenotypic changes in healthy lymphocytes.

T47

Atherosclerosis destabilizes regulatory T cells (Tregs) resulting in multiple families of exTregs

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- Treg destabilization proceeds through a staged cTreg–eTreg–exTreg differentiation pathway.
- Distinct exTreg subsets arise through branching differentiation trajectories.
- Treg-intrinsic IL-6R signaling promotes exTreg differentiation in atherosclerosis.

T48

Loss of CCM2 confers resistance to endothelial senescence and inflammation selectively in arterial endothelium

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- CCM2 LOF: Implications for Arterial Disease Protection and Venous Endothelial Vulnerability
- Vascular Bed-Specific Effects of CCM2 Loss on Endothelial Senescence
- CCM2 Loss Protects Arteries, Sensitizes Veins, to Senescence

LYMPHATIC BIOLOGY II

T49

Lymphatic endothelial FcRn maintains antibody pools for lymph node antiviral defense

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- LEC FcRn recycles antibody into lymph node sinuses, maintaining a pool for viral neutralization.
- LEC FcRn loss paradoxically traps antibody inside cells by preventing endosomal recycling.
- LEC FcRn-maintained sinus antibody limits viral spread beyond the draining lymph node.

T50

GLP-1 receptors are enriched in the lymphatic endothelium and their pharmacological activation with semaglutide improves the pumping capacity of lymphatic vessels

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- GLP-1Rs are expressed in the lymphatic endothelium.
- Activation of GLP-1Rs causes robust vasodilation and activation of lymphatic muscle cells.
- Activation of GLP-1Rs significantly increased the pumping capacity of lymphatic vessels.

T51

From paralysis to promise: Targeting STIM-Orai signaling in lymphedema

Soumiya Pal Ph.D., Adi Patro B.S., Grace A. Pea B.S., Sarah Broyhill B.S., Karen Bromert M.S., Yi-Xiao Liu Ph.D., Scott D. Zawieja Ph.D.

University of Missouri, Columbia, Missouri, USA

- Lymphatic muscle cell-mediated lymphatic pacemaking.
- Store-operated calcium entry driven lymphatic contractile rigor.
- Store-operated calcium entry: A plausible therapeutic target for lymphatic contractile dysfunction.

T52

CXCR4 controls lymphatic junctional integrity to alleviate secondary lymphedema

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Lemole Center for Integrated Lymphatics and Vascular Research, Department of Cardiovascular Sciences, Lewis Katz School of Medicine, Temple University, Philadelphia, Pennsylvania, USA

- Lymphedema
- Notch signaling
- Nanoparticles

T53

Lymphatic capillary Piezo2 regulates adiposity

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- Lymphatic capillary Piezo2 deficiency in adipose leads to changes in body composition
- Lymphatic capillary Piezo2 regulates adiposity
- Piezo2 in contact with adipocytes regulates production of TGFβ

MICROCIRCULATION II

T54

Disrupted endothelial cell – pericyte communication in cystic fibrosis revealed in an engineered model of human lung tissue

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Columbia University, New York, NY, USA

- Endothelial cell – pericyte coupling is critical for lung function, but is understudied in CF.
- Cells from CF iPSCs have increased inflammatory cytokine secretion and impaired vessel stability.
- Disruption of microvascular coupling in CF primes vasculature for further disruption.

T55

Pericyte and endothelial cell responses within murine cerebral capillaries after blood flow cessation

John C Chappell PhD, Hanaa Abdelazim PhD

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- Loss of blood flow triggers inflammation and ECM remodeling in brain capillaries.
- Pericytes may drive capillary constriction through Endothelin-1 signaling.
- ET-1 regulates Claudin5 remodeling, preserving but reorganizing endothelial junctions.

T56

Diabetes progression impairs Kv7-mediated mesenteric arterial tone in male UC Davis type-2 diabetes mellitus (UCD-T2DM) rats

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³Midwestern University, Glendale, Arizona, USA

- ACh-induced vasorelaxation worsens with diabetes and further deteriorates with NOS/COX inhibition.
- PE-induced vasoconstriction is elevated in diabetes, while Retigabine-induced relaxation is blunted.
- Kv7.1 mRNA expression is upregulated significantly in prediabetic and diabetic arteries.

T57

Claudin-11 depletion causes progressive, time-dependent loss of endothelial barrier resistance

Tram Q Le, Jerome Breslin

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- Our earlier negative result at 48 h reflected the assay endpoint rather than claudin redundancy.
- By 140 hours, knockdown monolayers retained 34% of baseline resistance, compared with 79% in control
- A parallel rise in capacitance indicates that junctional loosening is not the sole mechanism.

VASCULAR ANOMALIES AND MALFORMATIONS II

T58

Endothelial GNA11 hyperactivation induces vascular remodeling and coagulopathy in a novel inducible mouse model

Sandra Schrenk PhD, Kara Soroko, Chhiring Sherpa, Lydia Knutson, Elisa Boscolo PhD

Cincinnati Children's Hospital, Cincinnati, Ohio, USA

- Novel inducible endothelial GNA11 mouse model for vascular anomalies
- Endothelial GNA11 hyperactivation drives vascular remodeling and lesion formation
- Endothelial GNA11 activation induces consumptive coagulopathy

T60

Whole genome sequencing reveals a candidate NOTCH1 variant that segregates with disease in an ENG- and ACVRL1-negative family with Hereditary Hemorrhagic Telangiectasia

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- NOTCH1 variant segregates with HHT severity
- Whole-genome sequencing identifies non-canonical vascular genes
- Endothelial signaling disruption in panel-negative HHT

T61

MKK6/p38 signaling in vascular smooth muscle cells protects against aortic rupture in a mouse model of Vascular Ehlers-Danlos Syndrome

Rhiannon M Edwards B.S., Hal C Dietz MD, Elena Gallo MacFarlane PhD
Johns Hopkins University, Baltimore, MD, USA

- vEDS causes life-threatening aortic dissection without adequate pharmacologic therapy
- Map2k6 modifies aortic rupture risk in a mouse model of vEDS
- VSMC MKK6/p38 signaling influences ECM integrity and mechanotransduction

T62

Metabolic mechanisms underlying lymphatic malformation

Irma Gryniuk MS, Jie Zhu PhD, Pengchun Yu PhD
Oklahoma Medical Research Foundation, Oklahoma City, USA

- Cellular metabolism is essential for normal lymphatic vascular development.
- The role of cellular metabolism in lymphatic malformation (LM) pathogenesis remains unclear.
- Transcriptomic and metabolic analyses identify metabolic dysregulation during LM development.

VASCULAR FUNCTION IN HEALTH AND DISEASE II

T63

Differential impact of Sigma-1 receptor agonist PRE-084 on arterial tone with adrenergic or thromboxane receptor-mediated precontraction

Trang Nguyen, Laura Hurtado Osorio BS, Jerome W. Breslin PhD
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- PRE-084 relaxes human arteries precontracted with phenylephrine.
- PRE-084 does not cause relaxation when the arteries are precontracted with U46619.
- Blockade of NO synthesis or EDHF did not consistently block PRE-084-induced relaxation.

T64

Preserved BMP-responsive KDR regulation in unaffected BMPR2 mutation carriers suggests protective endothelial modifiers

Kamakshi Dattatray Bichu MS
Stanford University, Palo Alto, CA, USA

- Preserved endothelial KDR signaling in unaffected BMPR2 carriers
- Unaffected carriers show stronger BMP and KDR responses
- Searching for the protective modifiers of PAH

T65

Vascular bed-specific contributions of mitochondrial fission to contractile function in nulliparous and parous rats

Desirae Escalera, Nataliai Hula, Styliani Goulopoulou

Loma Linda University, Loma Linda, CA, USA

- Parity, the act of childbearing, is associated with long-term cardiovascular adaptations and risk.
- Uterine arteries, specialized reproductive vessels, undergo functional adaptations during pregnancy
- Mitochondrial fission contributes to vascular function, in a parity and vascular bed-specific manner

T66

Impacts of microvascular leakage and NGF signaling on neuropathic pain

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- Obesity induces fenestrations and hyperpermeability in skin capillary vessels.
- Inhibition of capillary endothelial leakage alleviates neuropathic pain in obesity.
- Targeting microvascular permeability provides a new therapeutic approach for obesity-induced pain.

T67

Doxorubicin induces mesenteric artery proteomic signatures of mitochondrial dysfunction, NRF2 suppression, and anemia-associated heme stress

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- DOX suppresses mitochondrial energy pathways in mesenteric arteries via PPARGC1A inhibition.
- NRF2 inhibition and GPX4/ACSL4 dysregulation prime mesenteric arteries for ferroptosis.
- DOX-induced hemolytic anemia amplifies vascular oxidative stress via free hemoglobin exposure

T68

Adipose inflammation induces sympathetic denervation and a loss of vascular reactivity during collateral arteriogenesis

Nathan V Tran BS, Sydney Luong BS, Mallary Greenlee-Wacker PhD, Trevor R Cardinal PhD

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- The natural history of collateral arteriogenesis from the perspective of vascular reactivity.
- The impact of adipose inflammation on systemic responses.
- The relationship between sympathetic innervation and vascular reactivity.

T69

Blood flow perception mitigates retinal endothelial cell death and capillary regression associated with diabetes

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- Retinal capillary loss is an early-stage contributor to diabetic retinopathy.
- Endothelial blood flow-sensing pathways protect diabetic retinal capillaries in mice.
- Retinal endothelial cell transcriptomics highlight diabetic pathogenic pathways rescued by flow.

T70

TTN-AS1 isoforms regulate Titin RNA processing and mechanical function in vascular smooth muscle cells

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- Distinct antisense lncRNA isoforms track with contractile versus modulated VSMC states.
- Isoform-specific depletion alters TTN exon abundance and lowers CNN1 expression.
- Selected isoform loss impairs contraction and migration, indicating reduced mechanical competence.

ANGIOGENESIS III

W01

An Ovol1-Slug signaling axis in endothelial cells regulates tumor angiogenesis

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- Endothelial cells might undergo partial-EndoMT during angiogenesis.
- Slug regulates angiogenesis potentially by regulating pEndoMT.
- Ovol1 is an upstream regulator of Slug during angiogenesis.

W02

In vivo characterization of RHOA in regulating blood vessel endothelial tip cells using zebrafish

Ekaterina R. Monakhova, Laura M. Pillay

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- Zebrafish embryos with altered RHOA activity exhibit angiogenesis defects.
- RHOA modulates actin-based cytoskeletal structures in blood vessel endothelial cells.
- Altering endothelial cell RHOA activity impacts blood vessel tip cell morphology and filopodia.

W03

Transcriptionally distinct populations of T cells regulate vascular and neurodevelopmental programs in the human brain

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⁷Neonatology, Sutter West Bay Medical Group, Alta Bates Summit Medical Center, Oakland, CA, USA

- T cells occupy distinct niches in the developing human brain
- CD4⁺ T cells in the prenatal brain promote vascular and neural development
- Prenatal T cells support homeostasis and growth of the developing brain

W04

Collagen type VIII modulates endothelial cytoskeleton and promotes proangiogenic activation

Qian Li MD, PhD, Qiong Yi MD, PhD, Catherine Wang, Lifang Yi MD, PhD, Yin Tintut PhD, Jeffrey J. Hsu MD, PhD

UCLA, Los Angeles, CA, USA

- COL8A1 marks activated, angiogenic endothelial cells in atherosclerosis
- COL8A1 impacts endothelial cytoskeleton, stiffness, and migration
- COL8A1 matrix cues drive a proangiogenic, tip cell-like endothelial state

ATHEROSCLEROSIS III

W05

Dichotomous roles of inflammation in atherosclerosis

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⁴University of Virginia, Charlottesville, VA, USA

- Dichotomous roles of SMC inflammation in atherosclerosis
- Inflammaging
- smooth muscle lineage tracing MICE AND ATHEROSCLEROSIS

W06

Gut microbial phenylalanine metabolism in cardiometabolic diseases

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- Gut microbial metabolites may improve cardiovascular risk prediction
- Microbiome therapies should target specific pathways.
- Microbial metabolites may guide personalized prevention.

W07

Immune checkpoint inhibition induces atherosclerosis-associated phenotypic modulation of vascular smooth muscle cells

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- PD-1 blockade promotes atherosclerosis-associated smooth muscle cell (SMC) phenotypic modulation.
- The anti-PD-1 antibody nivolumab activates AKT/mTOR-HSF1-HMGR-PERK signaling in SMCs
- Statins and SMC-specific HSF1/PERK deletion attenuate nivolumab-induced vascular toxicity.

W08

PRDM16-DT, a coronary artery disease risk lncRNA, regulates smooth muscle cell phenotype and atherosclerosis

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⁵Lewis Katz School of Medicine, Temple University, Philadelphia, PA, USA

- PRDM16-DT is a causal CAD risk gene with reduced expression linked to higher disease risk.
- Global Prdm16os deficiency accelerates atherosclerosis development in mice.
- PRDM16-DT is enriched in VSMCs and helps maintaining VSMC homeostasis.

W09

PRDM16 alters coronary artery disease risk by regulating phenotypic modulation of smooth muscle cells through two novel mechanisms

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- PRDM16 is a coronary artery disease associated gene
- Smooth muscle deletion of Prdm16 leads to protective plaque phenotypes
- Prdm16 alters smooth muscle cell function through distinct epigenetic and cofactor pathways

W10

Methamphetamine worsens atherosclerosis through enhanced inflammation and vascular calcification

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- Methamphetamine abuse is an increasing problem throughout the USA
- Cardiovascular disease risk is significantly exacerbated by methamphetamine abuse
- Increased inflammation and vascular SMC-mediated calcification are key contributors to this problem

BIOENGINEERING II

W11

Codon-optimized gene therapy via innovative lipid nanoparticles restores elastin production and establishes a blueprint for translationally challenged ECM targets

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- Gene therapy for elastin related vascular diseases
- LNP-mRNA rescues elastin fiber assembly in vivo
- Codon-optimization overcomes elastin translation barriers

W12

Modular assembly of arterial organoids enable rapid blood perfusion to ameliorate ischemic diseases

Kai Wang, Zihang Pan PhD

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- Homogenous generation of functional arterial organoids (AOs) in a dynamic bioreactor
- Reconstruction of transcriptional dynamics along with arterial differentiation
- Modular assembly enables the rapid construction and perfusion of complex vascular networks

W14

From blood vessels to target organs: Engineering AAV delivery

Tatjana Schlechtweg PhD, Skye Storrie PhD, Constance A Rich PhD, Silk Shi, Lili Zhou, Arianna Liu, Jack Hong, Amy Chen, Grace S Yu PhD, Bruce T Lahn PhD

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- AAV capsid engineering
- cardiac muscle delivery
- cardiovascular disease

W15

From transwell to Organ-on-Chip: TEER as a scalable, non-destructive readout for vascular barrier integrity and therapeutic toxicity

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- TEER enables non-invasive, functional readouts of endothelial barriers, measuring vascular health.
- Automated TEER measurements allow high throughput screening of drugs for vascular toxicity,
- Integrating TEER for Organ on Chip platforms enables non-invasive biosensing for QC and screening.

CARDIOVASCULAR DISEASES III

W16

Empagliflozin prevents pathological vascular CaV1.2 remodeling in experimental HFpEF

Miguel Martin Aragon Baudel PhD¹, Junyoung Hong PhD², Yumna Moustafa PhD¹, Hannah Voorhees¹, Bence Hegyi Hegyi PhD¹, Don Bers PhD¹, Madeline Nieves-Cintrón PhD¹, Manuel Navedo PhD¹

¹University of California, Department of Pharmacology, Davis, CA, USA. ²Kyonggi University, Gyeonggi-do, Korea, Republic of

- Vascular CaV1.2 remodeling drives arterial dysfunction in HFpEF
- Empagliflozin prevents pathological vascular CaV1.2 remodeling
- Novel vascular mechanism of SGLT2 inhibition

W17

Hypoxia-induced collagen V autoantigen secretion by pulmonary artery smooth muscle cells: implications for pulmonary hypertension progression.

Omar J Rodriguez Moncivais PhD, Obed J Blanco, Nikki L Jernigan PhD, Laura V Gonzalez Bosc PhD
University of New Mexico, Albuquerque, New Mexico, USA

- Chronic hypoxia drives collagen V deposition in pulmonary vascular remodeling.
- Integrin $\alpha 2\beta 1$ mediates autocrine collagen V signaling in PSMCs.
- Targeting integrin $\alpha 2\beta 1$ may reduce remodeling in pulmonary hypertension.

W18

Sex-Specific TLR4 signaling shapes smooth muscle cell responses in human and experimental cardiovascular disease

Olga A. Cherepanova PhD, Junyoung Hong PhD, Nischay Pothineni, Irene Krukovets BS
Cleveland Clinic, Cleveland, OH, USA

- TLR4 unexpectedly protects smooth muscle cells from osteogenic transition
- Female vascular protection persists despite increased osteogenic SMC remodeling
- Sex-dependent innate immune programs shape SMC responses across disease

W19

Functional genomics identifies an ANXA9 regulatory axis linking genetic susceptibility and female hormones in SCAD

Wei Sun MD

University of California, San Diego, La Jolla, CA, USA

- Causal non-coding variant regulates ANXA9 to drive coronary smooth muscle cell dysfunction in SCAD
- Female hormones regulate ANXA9 to promote arterial wall vulnerability
- Post-GWAS functional genomics identifies disease mechanisms and novel pathogenic targets

W20

Role of leiomodin 1 in hypoxic neovessel maturation

John Lee Franklin, Hongxia Wang, Sara Wennersten, Vivek Nanda

University of Alabama at Birmingham, Birmingham, AL, USA

- Peripheral artery disease (PAD) is attributed to atherosclerosis and impaired angiogenesis
- Reduced LMOD1 impairs formation of mature neovessels, promoting PAD pathogenesis
- LMOD1 regulates PAD independent of atherosclerosis

W21

ALDH2*2 genetic susceptibility to PM2.5-induced endothelial dysfunction

Hongchao Guo PhD, Christian Eaton PhD, J David Symons PhD, Cassandra Rice PhD, Christopher Reilly PhD

University of Utah, Salt Lake, UT, USA

- ALDH2*2 increases susceptibility to PM2.5-induced endothelial dysfunction
- PM2.5 activates a ROS–KLF5–IL-18/IL-1 β inflammatory signaling pathway.
- KLF5 may be a therapeutic target for genetically susceptible populations.

W22

Endothelial PTBP1 deletion in transplanted cardiac tissue limits cardiac allograft vasculopathy in an animal model

Chris Pathoulas¹, Koki Hayashi MD², Ivy Rosales MD², Amy Kimble¹, Krish Dewan MD³, Ryan Gross MD³, Jenna Lancey PhD², Lifang Ye PhD⁴, Qian Li⁴, Yunfeng Li¹, Bing Hao PhD¹, Bo Reese PhD⁵, Evan Jellison PhD¹, Antoine Menoret PhD¹, Greg Fishbein MD⁴, Anthony Vella PhD¹, Dawn Bowles PhD³, Nicole Valenzuela PhD⁶, Jeff Hsu MD⁴, Alessandro Alessandrini PhD², Patrick Murphy PhD¹

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- Endothelial splicing regulation in immune cross talk
- Prevention of allograft injury by endothelial modulation
- Metabolic regulation by alternative splicing

CELL BIOLOGY AND SIGNALING III

W23

Sustained ANP exposure desensitizes NPR1 through depletion of the mature surface receptor pool: implications for chronic NPR1 agonist therapy

HaEun Kim, Laura Axford, Joo Young Jeong, Loren Lindsley, Julian Kwan, Vanitha Subramanian, Jinghua Yu, Jonathan D. Horvath, Hilary Clay, Andrei Voznesensky, Jennifer R. Allport

Novartis BioMedical Research, Cambridge, MA, USA

- Acute and sustained ANP trigger distinct NPR1 signaling outcomes.
- Chronic ANP reduces mature surface NPR1 and limits cGMP signaling.
- Reversible NPR1 desensitization may constrain long-term agonist efficacy.

W24

A Src/Panx1 Y198 signaling axis couples hyperglycemia to ATP release and enhanced vascular reactivity

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- Src phosphorylates Panx1 at Y198 to open the channel and drive ATP release in arterial myocytes
- Blocking Src or Panx1 Y198 phosphorylation prevents glucose-induced CaV1.2 potentiation

- Panx1 Y198F mice resist hyperglycemia-driven vasoconstriction and diabetic myogenic tone increase

W25

Immune–vascular crosstalk regulates aortic remodeling in elastin deficiency

Yanina Tkachenko PhD¹, Russell Knutsen¹, Sanzida Afrin¹, Rohit Colora¹, Meysam Ganjibaksh¹, Jessica E. Wagenseil², Beth Koziel¹

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- Immune–vascular crosstalk drives aortic remodeling in elastin deficiency.
- Adaptive immunity regulates vascular remodeling and cell maturation.
- ELN transcriptional networks control vascular maturation and elastogenesis.

W26

Apoptotic cell releasate stabilizes macrophage SLC36A4 through a CD83–MARCH1 resolution pathway

Santosh Reddy Sukka Ph.D., Xiaobo Wang Ph.D., Ira Tabas M.D., Ph.D.

Columbia University Irving Medical Center, New York, NY, USA

- Bystander macrophages receive pro-resolving signals from nearby apoptotic cells.
- Site-directed mutagenesis reveals lysine-dependent control of transporter stability.
- Macrophage CD83 is required for efficient apoptotic-cell clearance in vivo.

W27

Role of Connexin 43 in vascular malformations of Hereditary Hemorrhagic Telangiectasia

Edward K Looker BA, Michael Flynn, Sophia Harris BS, Jennifer S Fang PhD

Tulane University, New Orleans, LA, USA

- Connexin43, a protein that enables signaling between neighboring cells, is increased in HHT lesions.
- Cx43 is required for EC migration, for angiogenesis, and for blood vessel network organization.
- By altering Cx43 expression or function, we are able to rescue HHT-associated dysangiogenesis.

W28

A potential role for the trafficking regulator Rab11a in the active restoration of endothelial barrier function after agonist-induced hyperpermeability

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- During hyperpermeability inactivation, eNOS relocalizes to the membrane driven by cAMP-Epac1.
- The recycling regulator Rab11a colocalizes with eNOS and may regulate its relocalization.
- Endothelial monolayers depleted of Rab11a fail to inactivate hyperpermeability.

W29

Identification of novel epigenetic mechanisms and regulatory proteins involved in vascular regeneration in a zebrafish caudal fin amputation model

Miranda Marvel, Isabella Cisneros, Jian Ming Khor, Sudul Kadawedduwa, Daniel Castranova, Aniket V. Gore, Kiyohito Taimatsu, Brant M. Weinstein

NIH, Bethesda, USA

- A novel vascular-specific epigenetic reporter was generated in zebrafish
- Multiomic profiling will identify epigenetic and transcriptional changes in vascular regeneration
- AngioTag identified several epigenetic regulators enriched in regenerating vessels

CEREBROVASCULAR HEALTH AND DISEASE III

W31

VT-109 restores Blood–Brain Barrier integrity and reduces neuropathology in a primate model of COVID

Janette Garcia Campos PhD candidate, Wan Chin Chan PhD, Man Long Kwok PhD, Raman Singh BA, Justin Richner PhD, Yulia Komarova

University of Illinois Chicago, Chicago, IL, USA

- SARS-CoV-2 disrupts BBB integrity and promotes neurovascular inflammation
- EB3-mediated Ca²⁺ dysregulation drives endothelial junction instability
- Targeting EB3 offers a new strategy to repair the injured blood-brain barrier

W32

Utilization of intravital two-photon microscopy to assess cerebral microvascular physiology and pathology

Adam Nyul-Toth PhD

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- Brain microvascular dysfunction in neurodegenerative disease
- Real-time assessment using intravital two-photon microscopy
- Translational testing of neurovascular therapies

W33

Loss of myogenic tone in cerebral small vessel disease affects perfusion

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¹University of Vermont, Burlington, VT, USA. ²Florida International University, Miami, FL, USA

- Loss of myogenic tone impairs CBF autoregulation
- Upregulated Kv channels drive vascular dysfunction and flow redistribution.
- Restoring myogenic tone reverses abnormal perfusion in CADASIL.

DEVELOPMENT III

W34

From centriole to cytoskeleton: unexpected roles for WDR90 in congenital heart disease

Sarah Colijn¹, DeHaven McCrary¹, Nahyun Kong¹, Ku-Chi Tsao², Mayssa Mokalled¹, Alex Francette¹, Rita Brookheart¹, Michael Harrison², Jessica Henty-Ridilla³, Sheng Chih Jin¹, Silvia Jansen¹, Amber Stratman¹

¹Washington University School of Medicine in St. Louis, St. Louis, MO, USA. ²Weill Cornell Medicine, New York City, NY, USA. ³SUNY Upstate Medical University, Syracuse, NY, USA

- Genetic variants in centriolar gene WDR90 have been identified as drivers of CHD
- Zebrafish wdr90 mutants display myocardial development delay, partial lethality, and fitness defects
- WDR90 interacts with actin and tubulin, particularly at adherens junctions

W35

Jam2 signaling functions downstream of Hand2 to initiate the formation of organ-specific vascular progenitors in zebrafish

Martyna Griciunaite, Julius Martinkus, Sanjeeva Metikala, Saulius Sumanas

University of South Florida, Tampa, FL, USA

- Late forming Etv2+ progenitors contribute to the intestinal and venous vasculature.
- Jam2a/jam2b is required for the late vascular progenitor and intestinal vessel formation.
- Hand2 functions upstream of jam2b and is required for etv2 expression in late vascular progenitors.

W36

Organization of lamin-associated heterochromatin domains in Acta2R179C/+ cells identifies a candidate regulator of SMC phenotype

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- ACTA2 p.R179 variants prevent nuclear localization of alpha-actin and smooth muscle differentiation.
- Immunostaining and H3K9me2 Cut&Tag show disordered lamin associated domains in Acta2R179C/+ SMCs
- Integration of RNA-seq and H2K9me2 Cut&Tag finds Prune2, a candidate regulator of SMC phenotype

W37

A comprehensive atlas of murine mesenteric lymph node blood endothelial cells

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- MLN heterogeneity
- Endothelial cell development
- Organ-specific diversity

ENDOTHELIUM IN HEALTH AND DISEASE III

W38

Endothelial NADPH oxidase 4 promotes retinal fibrosis through ECM signaling in diabetic microvascular complications in the eye

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- Nox4-induced oxidative stress in endothelial cells promotes endothelial senescence
- Pericyte injury through ECM-mediated signaling contributes to retinal fibrosis.
- Targeting endothelial senescence may help preventing vascular destabilization and retinal fibrosis

W39

STING-STAT3-SOX18 mediated endothelial-mesenchymal transition promotes pulmonary fibrosis in SAVI patients

Dan Yang MD/PhD¹, Guibin Chen², Adriana de Jesus², Colton McNinch², Adam Miranda², Atul Mehta², Noemi Kedei², Maria Hernandez², Sachin Gaurav², Clifton Dalgard³, Manfred Boehm¹, Raphaela Goldbach-Mansky²

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- STING activation induced EndMT contributes to lung fibrosis in SAVI patients.
- cGAS/STING induced EndMT is partially mediated through activation of STAT3/SLUG pathway.
- STING-driven endothelial stability networks associated with SOX18 repression

W40

Endothelial RIPK3 drives inflammatory and fibrotic remodeling in a mouse model of systemic sclerosis-associated interstitial lung disease

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- Endothelial dysfunction is an early, poorly understood driver of SSc-ILD pathogenesis.
- RIPK3 is elevated in SSc-ILD and may mediate endothelial injury in a bleomycin mouse model.
- Endothelial RIPK3 loss is linked to reduced lung inflammation and fibrotic remodeling.

W41

Tumor microenvironment modulates nanoparticle–endothelial cell interactions at the vascular barrier

Mobina Mohammadnejad

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- Tumor microenvironment affects endothelial cells
- Nanoparticles must cross the vascular barrier to reach tumor cells
- Exploring nanoparticle-vessel interactions

W42

Isolation and transcriptomic analysis of circulating endothelial cells in pulmonary hypertension

Robin Brice¹, Sakshi Vanjani¹, Anita Kovacs-Kasa¹, Avery Arons², Paul Kirkpatrick², Shaheen Islam², Laszlo Kovacs^{1,2}

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- Comprehensive molecular characterization of CECs across broader PH classifications.
- CECs reveal a remodeling-dominant molecular signature in patients with pulmonary hypertension.
- Classification-specific CEC signatures may point to new therapeutic targets and biomarkers for PH.

W43

Nucleoporin93 phosphorylation is required to maintain endothelial function and vascular barrier integrity

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- Endothelial Nup93 (T47 & T49) is a direct target substrate of Akt kinase.
- Loss of Nup93 phosphorylation results in enhanced TNF α -induced inflammatory responses.
- Nup93 phosphorylation loss increases RhoA/ROCK and MMP activity for EC permeability defects.

W44

Thioredoxin regulates pathological neovascularization in oxygen-induced retinopathy

Henry Song PhD¹, Sabiha Khatoon PhD¹, Brent Biddy PhD¹, Lily Wu PhD¹, Daniyal Khan BS², Jun Li PhD¹, Trent Tipple MD¹, Lynette Rogers PhD¹, Faizah Bhatti MD, MS¹

¹University of Oklahoma Health Sciences Campus, Oklahoma City, OK, USA. ²Georgia Institute of Technology, Atlanta, GA, USA

- Thioredoxin 1 (TXN1) is expressed in retinal endothelial cells, as well as other retinal cell types.
- Loss of EC TXN1 accentuates both retinal vasoobliteration and neovascular disease in neonatal mice
- Pathway analysis revealed anti-oxidant activity, cytokine mediated signaling and leukocyte migration

W45

Cerebrovascular endothelial cell derived extracellular vesicles contain protein cargo reflective of dysfunction in the brain

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- EV proteins for a specific vascular bed were identified across 6 EC lines
- EV proteins indicative of dysfunctional brain ECs were identified
- EC-EV profiles for the brain can act as biomarkers of a healthy or diseased state

INFLAMMATION II

W46

Cx43 S-Nitrosylation directs eNOS trafficking and spatial nitric oxide signaling to regulate inflammatory endothelial hyperpermeability

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- Cx43 S-nitrosylation regulates eNOS trafficking during inflammatory signaling.
- Cx43 dysfunction redistributes NO signaling by retaining eNOS within the ER.
- Spatial, rather than total, NO signaling governs endothelial hyperpermeability.

W47

Front-back polarization regulates endothelial inflammatory response through Wnt pathway modulation

Sarah Root¹, Shailaja Seetharaman PhD², Aaron Dinner PhD¹, Margaret Gardel PhD¹

¹The University of Chicago, Chicago, IL, USA. ²Mechanobiology Institute, National University of Singapore, Singapore, Singapore

- Endothelial cells exhibit inflammatory response heterogeneity.
- Endothelial heterogeneity is modulated by front-back polarity in both flow and wound healing.
- Polarity modulates inflammation through regulation of the Wnt-NfκB axis

W48

A novel myeloid-specific Dhcr24 Knock-In mouse reveals a role for DHCR24 in macrophage polarization

Gustavo Falero-Diaz PhD, Scott Kemp PhD, Ana Valentin Cabrera, Roberta M Lassance-Soares PhD

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- Lipid reprogramming of monocytes may influence recovery from limb ischemia.
- Dhcr24 catalyzes the final step of cholesterol biosynthesis.
- Myeloid Dhcr24 overexpression may impair recovery from limb ischemia.

W49

Endothelial ACKR1 regulation of lung inflammation and remodeling across acute and chronic timelines

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- Acute LPS exposure induces ACKR1 expression in pulmonary endothelial cells.
- Repeated intranasal LPS challenge generates organized iBALT in the lung.
- Endothelial-specific Ackr1 deletion will test immune trafficking and vascular remodeling.

LYMPHATIC BIOLOGY III

W50

HSPA5-Dependent cardiac lymphangiogenesis mediates exercise cardioprotection in diabetes

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¹Massachusetts General Hospital, Boston, MA, USA. ²Harvard Medical School, Boston, MA, USA. ³Medical College of Wisconsin, Milwaukee, WI, USA

- Cardiac lymphangiogenesis is impaired in diabetic cardiomyopathy.
- Exercise improves cardiac lymphangiogenesis in diabetic cardiomyopathy.
- Exercise regulates diabetic cardiomyopathy through HSPA5-mediated unfolded protein response.

W51

Aged-induced regression and dysfunction of valvular structures in mouse mesenteric lymphatic networks

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- Servo-null & confocal imaging enables comprehensive assessment of valve function & structure
- Pressure back-leak were found in mesenteric lymphatic valves in aged group vs. young
- Structure defects in valve leaflets correlates with functional deficiencies

W53

Activation of Nav1.3 restores collecting lymphatic pump function impaired by chronic MRSA infection

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- TF2 restored lymphatic contraction frequency and ejection fraction in MRSA-infected wild-type mice.
- Chronic MRSA infection was associated with 33% lower Scn3a (Nav1.3) expression in lymphatics
- Nav1.3-deficient (San^{-/-}) mice show partial resistance to MRSA-induced lymphatic pump dysfunction.

MECHANOBIOLOGY AND MECHANOTRANSDUCTION II

W55

Heart of Glass 1 (HEG1) controls eNOS activity to prevent endothelial dysfunction, hypertension, and atherosclerosis

Michael D Clark¹, Yerin Kim¹, Cesar A Romero¹, Dong Won Kang¹, Kyung I Baek¹, Eun Ju Song¹, Cailin E Kellum², Jay A Bowman-Kirigin¹, Christian Park¹, Rwei-Chun Hung³, Leandro Choi¹, Vir Kapoor³, Shoutaro Tsuji⁴, Jennifer S Pollock², Hanjoong Jo¹

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³Georgia Institute of Technology, Atlanta, Georgia, USA. ⁴Gunma University, Maebashi, Gunma, Japan

- Heart of Glass 1 (HEG1) controls flow-mediated eNOS activation, preventing endothelial dysfunction
- HEG1 interacts with eNOS in a flow-dependent manner via KRIT1
- HEG1 endothelial KO causes spontaneous hypertension, treatment of which ameliorates atherosclerosis

W57

Investigating the role of Discoidin Domain Receptor-1 in the alveolar epithelium

Ayla Shahid HBSc, Amanda Farrington HBSc, Roger Hsu MD, FRCPC, Michelle Bendeck PhD

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- DDR1 knockout mice demonstrate impaired alveolar development.
- DDR1 plays a role in the pathogenesis of atherosclerosis, lung, and kidney fibrosis.
- DDR1 regulates alveolar epithelial cell function.

W59

Nucleoporin93 coordinates endothelial response to laminar shear stress

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- Nup93 coordinates shear stress-induced cytoskeletal responses via Sun1.
- Nup93 loss impairs nucleocytoplasmic localization of KLF and Yap independently of Sun1 expression.

- Endothelial Nup93 maintains vessel homeostasis in disturbed flow-induced atherogenesis.

VASCULAR FUNCTION IN HEALTH AND DISEASE III

W63

Unveiling the role of CD201+ adventitial progenitor cells in atherosclerotic vascular calcification

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¹Johns Hopkins University School of Medicine, Baltimore, MD, USA. ²Johns Hopkins University, Baltimore, MD, USA. ³University of Maryland, Baltimore, MD, USA. ⁴University of California Los Angeles, LA, California, USA

- Transcriptomic mapping of adventitia of human and mouse calcified blood vessels
- Contribution of CD201 expressing adventitial cells in progression of vessel calcification
- CD201 expression as potential therapeutic target to treat calcification

W65

Haemodynamic control of zonated liver function via instructive vascular Wnt signalling

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- Blood flow is a crucial milieu factor to induce vascular Wnt signaling in the liver.
- Vascular Wnt response machinery is spatially coordinated and biased towards the central vein.
- Vascular Wnt modulates vascular functions in an autocrine manner.

W67

Metabolic basis of adventitial fibroblasts activation in aortic aneurysm

Cameron DA Mackay PhD Candidate, Anshul S Jadli PhD, Megan B Meechem PhD Candidate, Vaibhav B Patel PhD
University of Calgary, Calgary, AB, Canada

- Explore the role of aortic adventitial fibroblasts in aortic aneurysm development and progression.
- Explore how mitochondrial dynamics influences fibroblast phenotype and function.
- Explore therapeutic potential of targeting fibroblast mitochondrial dynamics in aortic aneurysm.

W69

Reduced endothelial glycolysis recapitulates Type 1 diabetes-induced vascular dysfunction

Adam Saloň PhD, Ishara Menik, Simone Kennard, Jing Ma, David Stepp, Rudolf Lucas, Tohru Fukai, Masuko Ushio-Fukai, David Fulton, Eric Belin de Chantemèle

Vascular Biology Center, Medical College of Georgia at Augusta University, Augusta, USA, Augusta, Georgia, USA

- Reduced endothelial glycolysis is sufficient to impair vascular function.
- Glucose metabolism and oxidative stress contribute to T1D vascular dysfunction.
- Androgen signaling influences vascular function in male T1D.

W71

Prophylactic siRNA-mediated fibrinogen attenuation reduces chronic venous thrombus burden without clinically significant bleeding in mice and swine models of deep vein thrombosis

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- Fibrinogen-targeted siRNA reduces clot burden, the substrate that drives PTS after DVT.
- Dose-dependent thrombus reduction shown in novel swine partial iliac ligation DVT model.
- Blood loss stayed under 10% TBV; EXTEM shows compensation preserves global clot strength