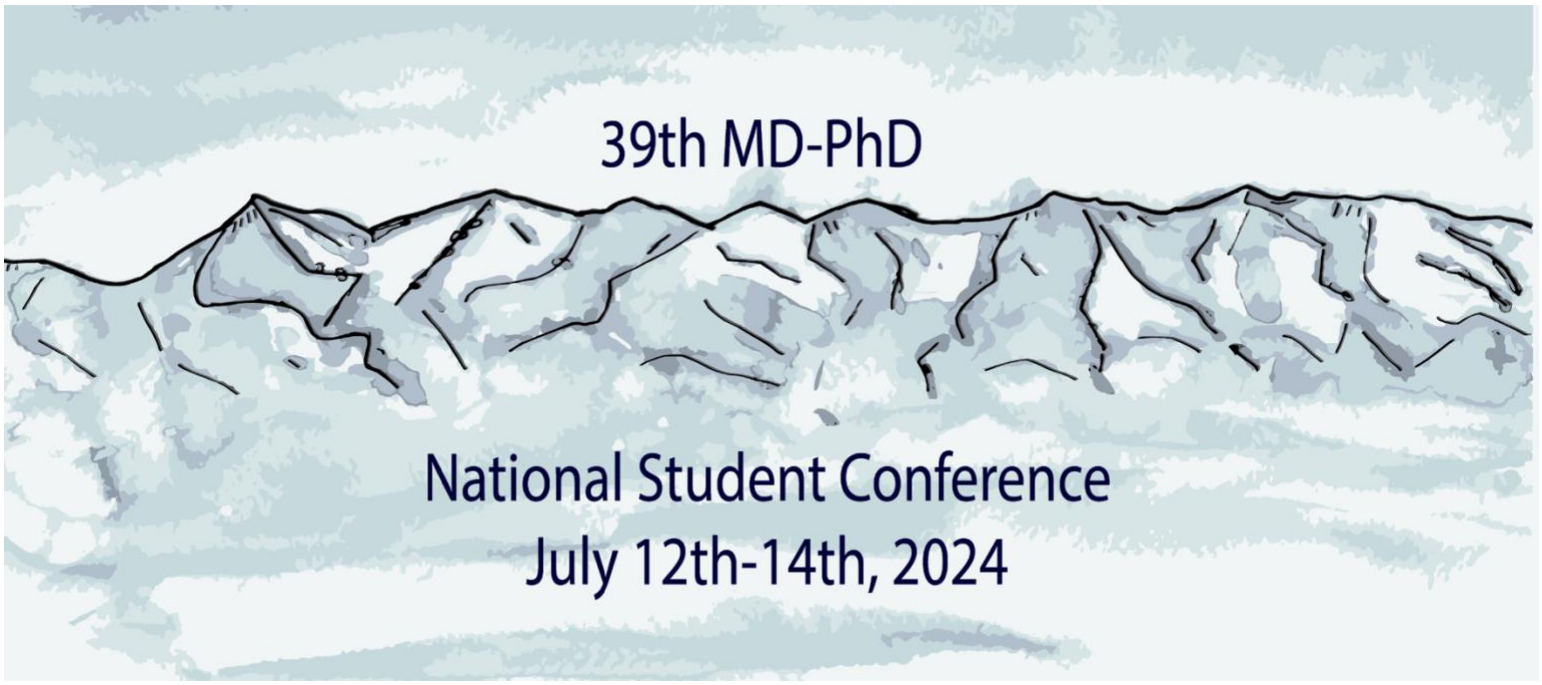

Conference Program

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Welcome

Welcome to the 39th Annual National MD/PhD Student Conference! The National MD-PhD Student Conference brings together students, faculty, and alumni from over 60 institutions across the country to interact with and learn from each other in beautiful Breckenridge, Colorado. At the conference, attendees engage in discussions with current and aspiring physician scientists about current scientific developments and explore career issues specific to the MD-PhD program. ***This event is almost entirely student run and organized***; the planning and organizing committee each year is made up of a small group of current MD-PhD students at the University of Colorado.

We would like to thank our Keynote speakers – Frances Collins, MD/PhD, W. Kimryn Rathmell, MD/PhD, Michael Brenner, MD, and James DiCarlo, MD/PhD – for making the journey to share their work with us. We are also grateful to the discussion leaders and PSTP directors for taking the time to lead discussions and share their insights with us.

We hope that you gain insights into your own research and career as you participate in the scientific and career-oriented portions of the conference, as well as make certain to enjoy the surroundings of the Rocky Mountains. Please note that the most up to date information on the conference can be found on the Webex app, with additional information found on the website (https://medschool.cuanschutz.edu/mstp/md-phd-conference). If you need anything during your time here, please do not hesitate to ask one of the conference organizers.

Sincerely,

The 2024 Organizing Committee

Meet Your Student Planning Committee

The planning committee each year is largely run by a small group of volunteer MD-PhD students at the University of Colorado! All the members participated in activities related to conference planning, with each member spearheading specific tasks.

Rachel Cohen, GS2: Co-Chair, Keynote Speaker Coordinator, Communication Liaison, Breckenridge Resort Liaison

Studies the role of HIF and intraepithelial lymphocytes in the colonic epithelium.

Jackson Stocking, GS2: Co-Chair, Breckenridge Resort Liaison, Keynote Speaker Coordinator

Studies the cerebellar nuclei as a potential target of neuromodulation to treat movement disorders.

Erin Fish, GS2: PSTP Showcase Liaison, Poster Session Coordinator

Studies mechanisms dictating dengue virus clearance from blood circulation.

Ira Fleming, GS2: Website and Registration Manager, Webex Platform Manager

Studies vaccine persistence in the lymph node using novel molecular tools.

Brandon Hilliard, GS2: PSTP Showcase Liaison, DEI and Ethics Organizer

Studies B cell peripheral tolerance and anergy in the context of autoimmunity.

Jessica Beynor, GS2, Fundraising Co-Chair, Keynote Speaker Coordinator, Webex Facilitator

Studies the role of estrogen in the HNSCC tumor microenvironment in response to radiation and immunotherapy treatment.

Daniel Moskop, GS2, Communication Liaison; Poster Session Coordinator

Studies the impact of inflammation on therapy response in MDS.

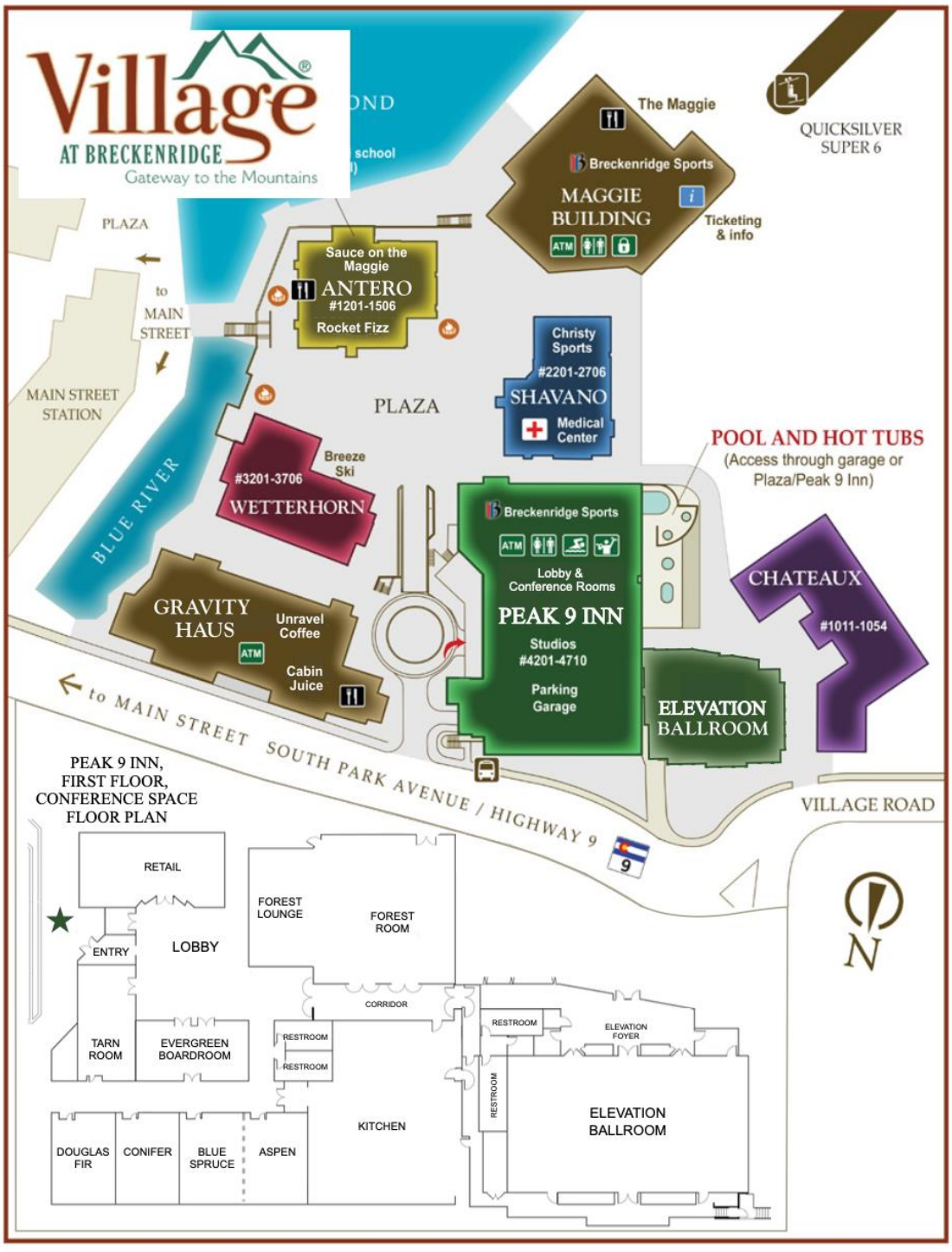
Stefano Ginocchio, GS2, DEI Event Coordinator

Studies impact of tissue stiffness in impaired angiogenesis in severe, early-onset fetal growth restriction.

Ashlyn Stahly, GS2, Fundraising Co-Chair

Studies the dynamics and determinants of tRNA repair.

Breckenridge Resort Map



Conference Schedule

All times listed in **Mountain Standard Time**

Friday July 12th

Time	Event	Location
9:30 AM – 12:30 PM	Registration	Atrium
12:30 PM – 2:00 PM	Lunch Keynote Speaker: Dr. Rathmell	Elevation Ballroom
2:00 PM – 3:00 PM	Oral Presentations: Immunology & Microbiology General Medicine & Physiology	 Elevation Ballroom Forest
3:00 PM – 3:30 PM	Coffee Break	Atrium
3:30 PM – 4:30 PM	DEI / Ethics (Session A)	DEI: Elevation Ballroom Ethics: Blue Spruce/Aspen
4:45 PM – 5:45 PM	Career Panel	Elevation Ballroom
5:45 PM – 7:15 PM	Dinner Keynote Speaker: Dr. DiCarlo	Elevation Ballroom
7:15 PM – 7:30 PM	Poster set up	Forest/Atrium
7:30 PM – 9:00 PM	Poster Session I & Happy Hour	Forest/Atrium

Saturday July 13th

Time	Event	Location
7:30 AM – 9:00 AM	Breakfast	Elevation Ballroom
8:30 AM – 12:00 PM	Funday Activities	Various
11:30 AM – 2:00 PM	Lunch Career Panel & Networking Session	Elevation Ballroom
2:00 PM – 3:00 PM	Keynote: Dr. Brenner	Elevation Ballroom
3:00 PM – 3:30 PM	ASPA Presentation	Elevation Ballroom
3:30 PM – 4:00 PM	Coffee Break	Atrium
4:00 PM – 5:00 PM	DEI / Ethics (Session B)	DEI: Blue Spruce/Aspen Ethics: Elevation Ballroom
5:15 PM – 6:30 PM	Poster Session II	Forest
6:30 PM – 8:30 PM	Dinner Keynote Speaker: Dr. Collins	Elevation Ballroom

Sunday July 14th

Time	Event	Location
8:30 AM – 9:00 AM	Breakfast	Elevation Ballroom
9:30 AM – 10:30 AM	Oral Presentations: Bioengineering & Medical Imaging Cancer Biology	 Blue Spruce/Aspen Elevation Ballroom
10:45 AM – 11:45 AM	Oral Presentations: Neuroscience Molecular Biology & Pharmacology	 Blue Spruce/Aspen Elevation Ballroom
12:00 PM – 1:00 PM	Lunch Closing Remarks	Elevation Ballroom

Keynote Speakers

Dr. Francis S. Collins, MD-PhD

NIH Distinguished Investigator in the intramural program of the National Human Genome Research Institute

Francis S. Collins, M.D., Ph.D., currently serves as an NIH Distinguished Investigator in the intramural program of the National Human Genome Research Institute, pursuing genomics research on type 2 diabetes and a rare disorder of premature aging called progeria. Dr. Collins is a physician-geneticist noted for his landmark discoveries of disease genes and his previous leadership of the international Human Genome Project, which culminated in April 2003 with the completion of a finished sequence of the human DNA instruction book. He served as director of the National Human Genome Research Institute at NIH from 1993-2008.
Dr. Collins then served as the 16th Director of the National Institutes of Health (NIH), appointed by President Barack Obama and confirmed by the Senate in 2009. In 2017, President Donald Trump asked Dr. Collins to continue to serve as the NIH Director. President Joe Biden did the same in 2021. For those 12 years, serving an unprecedented three administrations, Dr. Collins oversaw the work of the largest supporter of biomedical research in the world, spanning the spectrum from basic to clinical research. Dr. Collins stepped down as NIH Director on December 19, 2021.

From February 2022 to October 2022, Dr. Collins served as Acting Science Advisor to President Biden. From November 2022 to May 2023 he continued his White House service as a Special

Advisor to the President for Special Projects. He continues to serve as the White House lead for a bold program to eliminate hepatitis C in the United States.

Dr. Collins is an elected member of both the National Academy of Medicine and the National Academy of Sciences, was awarded the Presidential Medal of Freedom in November 2007, and received the National Medal of Science in 2009. In 2020, he was elected as a Foreign Member of the Royal Society (UK) and was also named the 50th winner of the Templeton Prize, which celebrates scientific and spiritual curiosity.

Dr. W. Kimryn Rathmell, MD-PhD

Director of the National Cancer Institute

W. Kimryn Rathmell, M.D., Ph.D., M.M.H.C., was sworn in as the 17th NCI director on December 18, 2023. She previously led the Vanderbilt University Medical Center as physician-in-chief and chair of the Department of Medicine.

Dr. Rathmell is a recipient of the 2019 Louisa Nelson Award for Women of Achievement, Vision, and Inspiration the 2019 Eugene P. Schonfeld Award from the Kidney Cancer Association, and the Paragon Award for Research Excellence from the Doris Duke Foundation. She was a leader of The Cancer Genome Atlas's (TCGA) kidney cancer projects and served as a TCGA analysis working group member across the spectrum of cancers, winning the 2020 American Association for Cancer Research Team Science Award. She has served on the NCI Board of Scientific Advisors, and the Forbeck Foundation Scientific Advisory Board.

Dr. Rathmell has held leadership positions with the American Society of Clinical Oncology and the American Society for Clinical Investigation, serving as secretary–treasurer and president. As a result of her efforts, Dr. Rathmell has been elected to the Association of American Physicians, the American Academy of Arts and Sciences, and the National Academy of Medicine.

Dr. Rathmell's specialty is the research and treatment of complex and hereditary kidney cancers. She also focuses on underlying drivers of kidney cancers using genetic, molecular, and cell biology to develop interventions to improve patients' lives. Dr. Rathmell's research has resulted in more than 250 articles in leading peer-reviewed journals, including The New England Journal of Medicine, Nature, Proceedings of the National Academy of Sciences, and the Journal of Clinical Investigation.

Dr. Rathmell earned undergraduate degrees in biology and chemistry from the University of Northern Iowa and her Ph.D. in biophysics and M.D. from Stanford University. She completed an internal medicine internship at the University of Chicago and an internal

medicine residency, medical oncology fellowship, and postdoctoral studies at the University of Pennsylvania. In 2022, she completed her Master of Management in Health Care from the Vanderbilt University Owen Graduate School of Management.

Dr. Michael B. Brenner, MD

E. F. Brigham Professor of Medicine at Harvard Medical School, Director of the Human Immunology Center, Director of the Single Cell Genomics Core, and Director of Cell and Molecular Immunology at Brigham and Women's Hospital

Michael B Brenner MD is the E. F. Brigham Professor of Medicine, at Harvard Medical School and Director of the Human Immunology Center, Director of the Single Cell Genomics Core, and Director of Cell and Molecular Immunology at Brigham and Women's Hospital. Selected honors include: Election to the National Academy of Sciences and in 2023 named as Highly Cited Researcher by Clarivate (top 1% of scientists by citations).

Brenner's laboratory designs and implements high dimensional immunophenotyping, single cell transcriptomic analyses and functional studies to deconstruct human autoimmune disorders. Discovery-based single cell and spatial analyses carried out in humans are used to define pathologic cell populations which are then interrogated in vitro or in animal models. In recent studies in the T cell field, his laboratory defined a new T helper cell population called T peripheral helper T (Tph) cells that are pathologically expanded in rheumatoid arthritis, lupus and many other autoimmune diseases and drive B cell responses and antibody production. He recognized the major CD8 T cell population expanded in inflamed human tissues was not the well-known granzyme B+ cytotoxic T cells, but rather CD8 T cells that express granzyme K. Exciting new studies now reveal that granzyme K drives a new pathway of complement activation. He identified a super-activated macrophage state that produces the highest levels of inflammatory factors in inflamed synovium as well as in tissues across several diseases. His laboratory has defined the central role of fibroblasts in inflammation and tissue damage, including novel subsets of highly inflammatory fibroblasts expanded in RA, and the role of Notch3 signalling in driving fibroblast differentiation and pathologic function. Current studies are defining the transcriptional regulation of pathologic fibroblasts. Together, these studies are "reconstructing" the immunopathology of RA and other autoimmune diseases.

Some of Professor Brenner's other research accomplishments in basic biology include the discovery of gd T cells and their functions. In contrast to peptide MHC presentation, he identified a new system of antigen presentation by which immune system T cell receptors recognize lipids as cognate antigens presented by CD1 antigen presenting molecules. He defined the trafficking, loading and function of CD1a, b, c and d, and the functions of CD1-restricted T cells and iNKT cells in host defense and immunoregulation. He identified the integrin molecule aEb7, its counter-receptor (E-cadherin), and its role in mucosal leukocyte

homing. He defined the role of calnexin in protein assembly and quality control, and the role of small GTPase Arl proteins in endocytic trafficking.

Dr. James J. DiCarlo, MD-PhD

Peter de Florez Professor of Neuroscience Director, MIT Quest for Intelligence Co-Director, and Center for Brains, Minds, and Machines Investigator at McGovern Institute for Brain Research at Massachusetts Institute of Technology

Jim DiCarlo is a Professor of Systems and Computational Neuroscience at the Massachusetts Institute of Technology. His research team's primary goal is to discover and artificially emulate the brain mechanisms that underlie human visual intelligence. Over the past 20 years, using the non-human primate animal model organism, DiCarlo and his collaborators have helped develop our contemporary, engineering-level understanding of the neural mechanisms that underlie visual information processing in the ventral visual stream — a complex series of interconnected brains areas — and how that processing supports core cognitive abilities such as object and face recognition. He and his collaborators aim to use this newly emerging scientific understanding to guide the development of more robust artificial vision systems ("AI"), to reveal new ways to beneficially modulate brain activity via modulations of images striking our eyes, to expose new methods of accelerating visual learning, to provide a basis for new neural prosthetics (brain-machine interfaces) to restore lost senses, and to provide a scientific foundation to understand how sensory processing is altered in conditions such as agnosia, autism and dyslexia.

DiCarlo trained in biomedical engineering, medicine, systems neurophysiology and computing at Northwestern (BSE), Johns Hopkins (MD/PhD), and Baylor College of Medicine (Postdoc). He served as Head of MIT's Department of Brain and Cognitive Sciences from 2012 to 2021, and he is currently the Director of the MIT Quest for Intelligence (2021-present) where he and his leadership team are working to advance interdisciplinary research at the interface of natural and artificial intelligence. DiCarlo is an Alfred P. Sloan Research Fellow, a Pew Scholar in Biomedical Sciences, a McKnight Scholar in Neuroscience, and an elected member of the American Academy of Arts & Sciences.

Panelists:

Diversity, Equity, and Inclusion: Dr. Cherise Hamblin, MD

Founder and President of Patients R Waiting, Assistant Professor in OBGYN at UMass Chan Medical School, Medical Director of the UMass Memorial Health Doula Program, Director of the Underrepresented in Medicine Workforce Development and Capacity Building for the UMass Chan Collaborative in Health Equity

Dr. Cherise Hamblin was born and raised in the Bronx, NY where she attended Bronx High School of Science. After graduation, Dr. Hamblin attended Franklin & Marshall College

where she received a double major in biology and Spanish in 2003. During her time at Franklin & Marshall College, she served as the Black Student Union Vice President and the Admission Office intern for Minority Recruitment. She also played rugby and studied abroad in Guadalajara, Mexico. Dr. Hamblin subsequently attended medical school at Northwestern University and completed her training in Obstetrics & Gynecology in Phoenix, AZ. Dr. Hamblin is passionate about working with the next generation of physicians both in her clinical roles and nonprofit work. For over a decade she has worked with advisors, students, and community organizers on various mentoring and health career exposure programs.

She is the founder and president of Patients R Waiting, a non-profit organization dedicated to eliminating health disparities by increasing diversity in medicine. She is a board certified OBGYN with a career focus on health care workforce development and antiracism in medicine. She is an Assistant Professor in OBGYN at UMass Chan Medical School and the Medical Director of the UMass Memorial Health Doula Program. Additionally, she serves as the Director of Underrepresented in Medicine Workforce Development and Capacity Building for the UMass Chan Collaborative in Health Equity.

Ethics Discussion: Dr. Insoo Hyun, PhD

Director of the Center for Life Sciences and Public Learning at the Museum of Science in Boston

Insoo Hyun, PhD, is a prominent figure in bioethics, with significant contributions to research ethics and policy in advanced biotechnologies. He is the former director of research ethics at Harvard Medical School's Center for Bioethics and taught at HMS as a senior lecturer in Global Health and Social Medicine. Currently, Dr. Hyun is the director of the Center for Life Sciences and Public Learning at the Museum of Science, Boston. He also works as a bioethics consultant for the Broad Institute of MIT and Harvard.

Dr. Hyun's expertise is widely recognized through his appearances on National Public Radio and contributions to journals like Nature and Science. He authored *Bioethics and the Future of Stem Cell Research* and was honored as one of Cleveland's Most Interesting People of 2019.

Career Panelists

Dr. W. Kimryn Rathmell, MD, PhD, MMMHC	National Cancer Institute Director
Dr. Charles Emala, MD	Colombia University Department of Anesthesiology Vice Chair for Research
Dr. Melanie Cree, MD, PhD	Pediatric Endocrinology at University of Colorado Anschutz and Children's Hospital Colorado
Dr. Taylor Soderborg, MD, PhD	Medical Director at Tiny Health

PSTP Showcase

The Ohio State University	Internal Medicine PSTP	Dr. Robert Baiocchi, MD, PhD
Harvard University	Internal Medicine PSTP	Dr. Jatin Vyas, MD, PhD
University of California San Francisco	UCSF Physician Scientist Scholar Program	Dr. Arun Wiita, MD, PhD
University of North Carolina Chapel Hill	Pediatric PSTP	Dr. Misty Good, MD, MS
Columbia University	Anesthesiology Physician Scientist Research	Dr. Charles Emala, MD
Duke University	Anesthesiology PSTP	Dr. Mara Serbanescu
University of North Carolina Chapel Hill	Internal Medicine PSTP	Dr. Joseph (Alex) Duncan, MD, PhD
Stanford University	Anesthesiology PSTP (FARM Program)	Dr. QiLiang Chen, MD, PhD
Stanford University	Internal Medicine PSTP (TIPS Program)	Dr. Paul Cheng, MD, PhD
University of Colorado	Internal Medicine PSTP	Dr. David Schwartz MD
University of Colorado	Pediatric PSTP	Dr. Christine Vohwinkel MD, PhD
University of Michigan	Pathology PSTP	Dr. Mark Rudolf, MD, PhD

Student Oral Presentation Schedule and Abstracts

Concurrent Session 1A

I. Immunology and Microbiology (Elevation Ballroom)

Host response during urinary tract infection alters mammary extracellular matrix and accelerates Brca1 tumorigenesis, Steven Lewis, Stony Brook University

Environmental factors are known to potentiate the tumorigenic potential of genetically predisposed cells. While breast cancer is among the most common tumors in women and many genetic risk factors are known, there is a paucity of known environmental risk factors. Our recent work uncovered that an environmental factor, urinary tract infection, through systemic inflammatory mediators induced during the host response can remodel the mammary gland at the cellular and tissue level. We found that wildtype mice with an unresolved UTI demonstrate ductal hyperplasia, fibroblast activation and collagen deposition, which depended on systemic TIMP1 induced by UTI. Because ECM changes can induce epithelial proliferation through the YAP pathway, which we found using scRNAseq was elevated in mammary epithelial cells during UTI, we hypothesized that the UTI host-response might impact breast tumorigenesis in a genetically predisposed host. In our autochthonous mouse model of Brca1-deficient breast cancer, we discovered that the systemic inflammatory response triggered by UTI promotes tumorigenesis. Through histological analysis of pre-tumor and tumor tissue, scRNA-seq and transplant experiments, we identified that the systemic response to UTI induced expansion of a Krt6a-expressing basal-luminal (BL) epithelial population with enhanced plasticity and tumor initiating potential. We traced the activation of these Krt6a+ basal-luminal cells through mechano-sensing gene transcription driven by YAP/Tead1 back to ECM deposition of collagen and tenascin-C by fibroblasts activated after UTI. These ECM changes engaged Itgb6 and induced proliferative signaling in BL cells. This collagen deposition and activation of the tumor initiating capacity of BL cells by UTI could be reversed through resolution of the infection using antibiotic treatment or TIMP1 neutralization. This finding provides the first example of how a local infection, commonly experienced by women globally, can impact the expressivity of a common cancer associated mutation in breast cancer and presents an avenue for improved prevention and surveillance efforts in the BRCA1 population.

B cell peripheral tolerance is promoted by cathepsin B protease, Marissa Chou, University of California San Francisco

B cells that bind soluble autoantigens receive chronic signaling via the B cell receptor (signal-1) in the absence of strong costimulatory signals (signal-2), and this leads to their elimination in peripheral tissues. The factors determining the extent of soluble autoantigen-binding B cell elimination are not fully understood. Here we demonstrate that elimination of B cells chronically

exposed to signal-1 is promoted by cathepsin B (Ctsb). Using hen egg lysozyme- (HEL) specific immunoglobulin transgenic (MD4) B cells and mice harboring circulating HEL, we found improved survival and increased proliferation of HEL-binding B cells in Ctsb-deficient mice. Bone marrow chimera experiments established that both hematopoietic and non-hematopoietic sources of Ctsb were sufficient to promote peripheral B cell deletion. Depletion of $CD4^+$ T cells overcame the survival and growth advantage provided by Ctsb deficiency, as did blocking CD40L or removing CD40 from the chronically antigen-engaged B cells. Thus, we suggest that Ctsb acts extracellularly to reduce soluble autoantigen-binding B cell survival and that its actions restrain CD40L-dependent pro-survival effects. These findings identify a role for cell-extrinsic protease activity in establishing a peripheral self-tolerance checkpoint.

Exploring the role of fibroblast Reg3 proteins in response to intestinal inflammation, Sritejasvinthi Karimikonda, Medical College of Wisconsin

Fibroblasts are multifaceted cells involved in extracellular matrix formation, immune regulation, and tissue homeostasis. Fibroblasts proliferate in response to disease and are involved in the pathology of many inflammatory diseases including inflammatory bowel disease (IBD) by contributing to complications like tissue fibrosis and stricture formation. *However, the processes which drive fibroblast response to intestinal inflammation and the role of fibroblasts in host defense are still poorly understood.* To investigate the intestinal fibroblast response, we employ a bacterial enteritis model system. Using spatial transcriptomics, we found increased expression of *regenerating islet-derived protein beta* (*Reg3*β) and *gamma* (*Reg3*γ) in the cecum in response to infection. Only epithelial and gamma-delta T cells have been shown to express *Reg3*β and *Reg3*γ in the gut thus far. Flow cytometry analysis showed that in addition to epithelial cells and immune cells, an Epcam- Cd45- subset of cells, which includes fibroblasts, also produced Reg3γ. *Fibroblast expression of Reg3 proteins has not been previously demonstrated.* Integration of our spatial transcriptomics dataset with a small intestine scRNAseq dataset showed that within the non-epithelial and non-immune cells subset, both fibroblasts and endothelial cells can express *Reg3*β and *Reg3*γ transcripts. We explored this further using the spatial transcriptomics data, and found an increased number of fibroblast marker transcripts, *Pdgfra, Thy1, Pdpn,* and *Vim*, co-localized with *Reg3*β and *Reg3*γ transcripts in response to intestinal inflammation. Moreover, there were more transcript co-localizations associated with fibroblast markers compared to endothelial cell markers, suggesting that fibroblasts are a key cell type involved in *Reg3* expression. Overall, our results suggest a novel fibroblast innate immune response to intestinal inflammation via antimicrobial peptide production.

Concurrent Session 1B

II. General Medicine and Physiology (Blue Spruce/Aspen)

Investigating the role of heme in acute and chronic sickle cell disease pain, Samuel Zorn, Medical College of Wisconsin

Individuals with sickle cell disease (SCD) suffer from complex stimulus-evoked and spontaneous pain that is mediated in part by the hyperexcitability of their peripheral sensory neurons. However, it is unclear whether acute or chronic elevation of cell free heme, a key pathological feature of SCD, contributes to the aberrant activity of sensory neurons. Thus, determining whether heme drives pain behavior and enhanced excitability of dorsal root ganglia (DRG) primary sensory neurons involved in pain signal transduction is critical for the development of targeted pain therapies for SCD. In the current experiments, we determine whether cell free heme accumulates within the DRG and further use evoked and ongoing pain behavior assays and *in vitro* calcium imaging and patch clamp electrophysiology to evaluate the hypothesis that heme causes direct sensitization of nociceptive DRG neurons in wildtype (WT) mice. Our preliminary data reveal that similar to pain behaviors in the mouse model of SCD, hind paw injection of heme into WT mice induces mechanical and cold hypersensitivity, place aversion, and facial grimace. These data suggest that sensory terminals in the skin become hyperexcitable when exposed to elevated heme. We next administered heme to primary cultured DRG neurons *in vitro* and observed calcium flux across the neuronal cell membrane that was dependent on extracellular calcium Together, these data suggest that heme opens calcium channels on sensory neurons to drive acute pain behaviors. As ongoing activity of nociceptive neurons may cause pain signal transduction in the absence of external stimuli, we will next use current clamp electrophysiology to determine whether application of heme to cultured DRG neurons is sufficient to induce action potential firing. Finally, our preliminary data suggests that injection of exogenous haptoglobin, a hemoglobin scavenger that prevents heme accumulation, alleviates steady-state pain behaviors in Townes SCD mice. Completion of these experiments will shed light on the contributions of cell free heme to SCD pain and illuminate downstream effectors that may be targeted to provide analgesic relief for this historically understudied and debilitating disease.

Longitudinal blood pressure trajectories associated with exposure to metals and metal mixtures, Margaret Weiss, University of Illinois at Chicago

Background: Elevated blood pressure is a leading risk factor for cardiovascular disease, the leading cause of mortality worldwide. Evidence suggests that metal exposure may increase blood pressure; however, most previous studies have been cross-sectional or underpowered. Furthermore, underserved racial/ethnic groups are both at higher risk of hypertension and metal exposure, including Mexican Americans. Consequently, we aimed to evaluate associations between longitudinal blood pressure traits in relation to urinary levels of metals and metal mixtures among 431 Mexican American adults. **Methods**: A longitudinal cohort of 431 Mexican

American adults, ages 35 to 69, were followed over 3 years with 6 repeated examinations. Urinary metals were quantified at baseline. Linear mixed effects models were fitted to estimate the per month increase in systolic blood pressure, diastolic blood pressure, pulse pressure, and mean arterial pressure associated with higher urinary metal levels. Metal mixture scoring, principal component analysis, weighted quantile sum regression, and quantile-based g computation were utilized to assess the associations of the metal mixture. **Results**: After adjustment, single metal models identified arsenic, cobalt, molybdenum, and zinc with faster increases in blood pressure. Arsenic was associated with a faster per month increase in systolic blood pressure, pulse pressure, and mean arterial pressure; molybdenum with systolic and pulse pressure; and zinc and cobalt with pulse pressure. The metal mixture was associated with a faster per month increase in pulse pressure. **Conclusion**: This study identifies several metals, both individually and as mixtures, associated with increased blood pressure traits over time. Since elevated blood pressure is a driver of cardiovascular disease, for which incidence continues to disproportionately affect Hispanic/Latino individuals, this study provides insight into the impacts of metals on cardiometabolic health.

Trajectories of mHealth-Tracked Mental Health Symptoms and Their Predictors in Chronic Pelvic Pain Patients, Emily Leventhal, Icahn School of Medicine at Mount Sinai

Background: Female chronic pelvic pain disorders (CPPDs; e.g., endometriosis, adenomyosis, fibroids) are prevalent and debilitating conditions associated with co-morbid clinical anxiety and depressive symptoms. Characterizing variable inter-individual symptom trajectories and lifestyle factors as possible predictors is a starting point to enable patient self-efficacy and symptom management. We herein investigate the association between mental health, pain symptoms, and physical activity (PA) in CPPDs and demonstrate a method for handling multi-modal, non-linear mobile health (mHealth) data for this clinical scenario. **Methods**: Study sample included 3,640 person-level days of data from 62 CPPD patients. Participants completed the McGill Pain Questionnaire (MPQ), PROMIS pain interference (PI), pain self-efficacy (PSEQ), PROMIS global mental health (GMH) for 14 weeks using a research mHealth App, and the pain catastrophizing scale (PCS) and psychiatric history at baseline. We used activity trackers to collect daily moderate-to-vigorous PA (MVPA) data. **Data analysis:** We used generalized additive mixed-effects models (GAMMs) to regress weekly GMH scores on 7-day MVPA and other weekly pain outcomes and adjusted for baseline measures, time in study, and the random intercept of the individual. We converted 7-day MVPA data into a single smooth using spline basis functions with a polynomial term (n=3), to model the potential non-linear relationship. **Results**: MVPA was a significant, curvilinear predictor of GMH scores (B_1=1.60; B_2=-7.07, B_3=14.10, $p < 0.05$), independent of psychiatric diagnoses and weekly pain-related factors. PI and PCS were negatively associated with GMH scores (B=-2.41, B=-3.35, respectively; $p<0.05$). **Conclusion**: Our findings underscore the complex, non-linear relationship between PA and mental health in CPPD and demonstrate the potential of ambulatory mHealth-based data combined with GAMMs for delineating inter-individual and temporal variability.

Concurrent Session 2A

I. Bioengineering and Medical Imaging (Blue Spruce/Aspen)

Rethinking Spinal Fusion Surgery: A new paradigm in the biology of spinal fusion, Duby Okonkwo, Vanderbilt School of Medicine

Introduction: Fusion of the spine is crucial for treating many spinal disorders that cause pain and dysfunction. Forced ossification between vertebra is essential for successful fusion, and failure rates remain high at 35%, even with supplemental bone grafts. The current paradigm posits that spinal fusion is driven by the differentiation of mesenchymal stem cells (MSCs) from the intraosseous bone directly into osteoblasts without a chondrocyte intermediate. This approach neglects the outer bone membrane, periosteum, which is removed to expose trabecular bone. Supported by recent findings in fracture repair, we propose a new paradigm: MSCs originating from the periosteum drive ossification through a chondrocyte intermediate. *Methods:* Advanced lineage tracing animals were used to determine the origin of MSCs and type of bone formation required for spinal fusion in a clinically relevant pre-clinal murine model. We also explored if different surgical techniques in regard to periosteal health affect fusion. Bone formation was assessed through radiographs, microCT analysis, and histology. *Results and Discussion:* Our findings reveal that MSCs originate from the periosteum for bone formation during spinal fusion, undergoing chondrogenesis as an intermediate step. Cauterizing the periosteum significantly reduced MSC recruitment, highlighting the vital role of the periosteum. *Conclusion:* Our study shifts the paradigm of the origin of MSCs required for spinal fusion from the intraosseous trabeculae to the outer membrane of bone, the periosteum. This shift has significant implications for spinal fusion surgery, which previously viewed the periosteum as a layer needed to remove to expose the trabecular bone. Additionally, our findings challenge the focus on promoting intramembranous ossification, suggesting that grafts which promote chondrogenesis, as opposed to osteogenesis, improve clinical results.

Polybromo-1 bromodomain missense variants associated with clear cell renal cell carcinoma exhibit variable biochemical fitness, Karina Bursch, Medical College of Wisconsin

The tumor suppressor Polybromo-1 (PBRM1) modulates chromatin accessibility via six bromodomains that bind acetylated nuclear proteins. PBRM1 mutations exist in ~40% of clear cell renal cell carcinoma (ccRCC) cases, often ablating protein expression and tumor suppressor activity. However, missense mutations cluster in PBRM1 bromodomains and exist in ~15% of ccRCC cases, yielding full-length protein variants with unknown structural and functional characteristics. With >900 PBRM1 missense variants reported in the Catalogue of Missense Mutations in Cancer, mechanistic studies of every missense variant are intractable; *in silico* approaches may instead yield actionable assessments of PBRM1 missense variant impacts in ccRCC and other cancer types. We computationally predicted the impacts of ccRCC-associated missense variants in the second (BD2) and fourth bromodomains (BD4) of PBRM1 on

bromodomain stability and ligand binding at the levels of protein sequence (2D), structure (3D), and molecular dynamics (4D). These predictions were used to assign missense variants a pathogenicity score between 0-1; scores near 1 indicate missense variants predicted to decrease stability and ligand binding. We purified all reported ccRCC-associated PBRM1-BD2 and -BD4 missense variants to evaluate the concordance between predicted pathogenicity and experimental stability (differential scanning fluorimetry, circular dichroism), and ligand binding (AlphaScreen assays, electrophoretic mobility shift assays). 3D and combined 3D/4D scores correlated with measures of acetylated ligand binding, while 2D and total pathogenicity scores correlated with measures of protein thermal stability. Our results demonstrate that computational prediction of PBRM1 missense variant pathogenicity accelerates mechanistic understanding of missense variant impacts on protein stability and ligand binding, supporting future studies correlating specific missense variants with PBRM1 tumor-suppressive functions.

Comprehensive Independent Component Analysis Denoising Assistant (CICADA): A new simple and comprehensive fMRI denoising tool, Keith Dodd, University of Colorado

BACKGROUND: Manual independent component (IC) classification, a gold-standard functional magnetic resonance imaging (fMRI) denoising method, is time and training intensive. Automated methods, meanwhile, may denoise less effectively. To address this, we developed the Comprehensive Independent Component Analysis Denoising Assistant (CICADA) to better target all common noise sources and therefore hypothesized denoising metric improvements over current automated denoising methods. **METHODS:** CICADA was assessed in schizophrenia resting-state fMRI (N = 55) against FIX, ICA-AROMA, eight parameter (8p) regression, and manual IC denoising in a data subset (N = 20). Denoising efficacy was evaluated through gray matter connectivity correlations with motion (median framewise displacement [FD]) and distance dependence. Modularity quality and its correlation to motion evaluated network identifiability. Denoising effectiveness was further evaluated through noise profile correlations and IC classification accuracy. **RESULTS:** CICADA significantly reduced correlations between gray matter connectivity and both motion ($p < 0.001$ for all techniques) and distance dependence ($p < 0.001$ for 8p). 8p's moderate motion correlation ($r = 0.42$) suggests that the increased modularity quality is reflective of motion effects rather than neuronal activity. CICADA further consistently outperformed FIX, AROMA, and 8p in noise profile correlations and improved IC classification accuracy compared to AROMA and FIX ($p < 0.001$). **CONCLUSION:** CICADA appears to offer similar fMRI denoising effectiveness in resting-state fMRI to the manual IC denoising gold standard, with significant improvements compared to current automated methods.

Concurrent Session 2B

II. Cancer Biology (Elevation Ballroom)

*Evidence of microsatellite-instability and high tumor mutational burden in HIV+/ART-experienced diffuse large B-cell lymphoma, **Sophia Roush**, University of North Carolina at Chapel Hill*

Background: Diffuse large B-cell lymphoma (DLBCL) is highly associated with HIV and outcomes remain poor for many patients. With improved global access to antiretroviral therapy (ART), the proportion of DLBCLs arising in HIV+/ART-experienced (HIV+/ART-exp) people is increasing. Mutational profile, tumor mutational burden (TMB) and microsatellite-instability (MSI) are used as biomarkers in a variety of cancers, particularly for immunotherapy response, but have not been well studied in HIV+ DLBCL. Methods: We performed whole exome sequencing of n=24 DLBCL tumors and matched germline of patients enrolled in the Kamuzu Central Hospital Lymphoma Study (n=8 HIV-, n=12 HIV+/ART-exp, n=4 HIV+/ART-naïve). Pairwise Wilcoxon rank-sum test was used to compare TMB by HIV/ART status. "High" vs. "low" TMB was determined by median cut-off and difference in 5-year OS was assessed by log-rank test. For mutation enrichment analysis, pairwise Fisher's exact test was used with FDR correction. MSI was predicted by the Microsatellite Analysis for Normal-Tumor InStability pipeline. Protein expression of MLH1, MSH6, MSH2, PMS2 was determined by immunohistochemistry (IHC). Results: Median TMB was higher in HIV+/ART-exp vs. HIV- DLBCL (1.6-fold, p=0.04), and TMB was positively prognostic among HIV+/ART-exp patients (20 vs. 10 mo. median survival, p=0.016), but not in the overall cohort. HIV+ DLBCLs were enriched for *ARID1B*, *CHD5, POLE*, *PTPRB*, *RELN*, and *RNF213* mutations, while HIV- DLBCLs were enriched for *NCOR2* and *CARD11* mutations. n=3 HIV+/ART-exp and n=1 HIV- DLBCLs were predicted to be MSI-positive, supported by mutations in DNA mismatch repair genes and protein loss by IHC. Conclusion: HIV+/ART-exp DLBCLs demonstrated increased TMB, and high TMB associated with improved survival in this group only. Our data suggest that MSI and defective DNA damage repair mechanisms play a role in HIV+/ART-exp DLBCL, and these patients may especially benefit from immune checkpoint inhibitor therapy.

Malignant mast cells are chemoresistant and associated with poor outcome in core binding factor-mutated pediatric acute myeloid leukemia, *Denis Ohlstrom, Emory University/Georgia Institute of Technology*

Background. Pediatric acute myeloid leukemia (pAML) is genetically heterogenous with malignancy driving translocations such as RUNX1-RUNX1T1 and CBFB-MYH11, collectively the core binding factor (CBF) pAMLs. Mutations inform pre-treatment risk, though 5-year relapse rates remain ~30% for standard risk (CBF) patients. Single-cell RNA sequencing (scRNAseq) enabled characterization of cellular diversity in pAML, but it is unclear which cells survive treatment and promote relapse. Methods. We analyzed 649,192 cells from 167 bone marrow (BM) biopsies of 98 patients (37 with sequential samples). PAML cells were identified by occupancy scoring, copy number variants, and immunophenotyping. Differentially expressed genes (DEG) were used for pathway analysis and deconvolution of the TARGET bulk RNAseq dataset. High-risk subtypes were defined by proportional increase at end-of-induction (EOI), enrichment in patients who experienced relapse, and worse overall survival (OS) in TARGET. Results. We identified 33 subtypes of pAML by cell surface gene expression. Surprisingly, one subtype expressed mast genes (e.g. TPSB2 $Log_2FC=4.92$, $p=1.0x10^{-250}$). Malignant mast cells (MMC) were specific to CBF pAML, with >5% MMCs in ~29% CBF patients vs 1% non-CBF. MMCs were chemoresistant (5.74-fold proportional increase at EOI vs diagnosis) and enriched in patients who experienced relapse vs sustained remission (2.38-fold). Patients enriched for MMCs in TARGET had worse OS (HR=2.24, $p=6.7x10^{-4}$). DEGs included markers of immaturity (GATA2

$Log_2FC=2.63$, $p=1.0x10^{-250}$) and immune interaction (CXCL8 $Log_2FC=2.53$, $p=1.0x10^{-250}$). Pathway analysis similarly found enrichment for inflammation and immune cell chemotaxis. Conclusions. We report the first description of MMCs in CBF pAML. MMCs are chemoresistant, relapse-enriched, and associated with worse OS. MMCs express mixed mature and immature genes and immune interacting pathways. Future studies will dissect the spatial interaction of MMCs and immune cells in the BM.

Patient-derived glioblastoma organoids as real-time avatars to assess responses to clinical CAR-T cell therapy, Yusha Sun, University of Pennsylvania

Patient-derived tumor organoids have been increasingly leveraged for disease modeling and preclinical studies, but rarely in real-time to aid with the interpretation of patient treatment responses in the clinical setting. Whether tumor organoids can be applied to mirror clinical activity in patients and even to predict responses for personalized medicine remains uncertain. We recently demonstrated promising early efficacy signals in a first-in-human, phase 1 study of dual-targeting chimeric antigen receptor T cells (EGFR-IL13Rα2 CAR-T cells) in patients with recurrent glioblastoma (clinical trial identifier: NCT05168423; Bagley et al. Nature Medicine 2024). Determination of meaningful clinical efficacy, in particular for CAR-T cell therapy, may take months to manifest. Given this challenge, real-time analysis of patient-derived glioblastoma organoids (GBOs) could help to provide early assessment of clinical efficacy and guide patient management. Here we analyzed six sets of GBOs generated on the same timeline as the production of patients' CAR-T cell products. GBOs demonstrated the presence and retention of both targeted tumor-associated antigens EGFR and IL13Rα2 by immunohistochemistry. These GBOs were then treated with the same autologous CAR-T cell products received by patients in our phase 1 study in parallel with patient treatment. CAR-T cell treatment led to target antigen reduction, and we confirmed cytolysis of tumor cells in GBOs by cleaved caspase-3 immunostaining and by co-culture with the Axion Maestro Cellular Impedance platform. Importantly, the degree of cytolysis *ex vivo* significantly correlated with CAR-T cell engraftment detected in patients' cerebrospinal fluid (CSF) for these patients. Furthermore, cytotoxic activity of CAR-T cells as measured by TNFα, IL-2, and IFN release kinetics in GBOs mirrored cytokine levels in patient CSF samples over time. Our findings highlight a unique trial design and suggest GBOs as a valuable platform for real-time assessment of CAR-T cell bioactivity and for insights into immunotherapy efficacy.

Concurrent Session 3A

I. Neuroscience (Blue Spruce/Aspen)

SUR1-TRPM4 expression due to chronic seizures contributes to epileptogenesis, Mitchell Moyer, University of Maryland

Rationale: One third of epilepsy patients become resistant to current anti-seizure therapies. A promising novel target for reducing seizures in epilepsy is SUR1-TRPM4, a sodium channel minimally expressed in healthy brain that upregulates *de novo* after status epilepticus and other epilepsy-associated pathologies. SUR1-TRPM4 inhibition was shown to be therapeutically beneficial in numerous CNS pathologies in preclinical and clinical studies; however, this has not been explored in epilepsy. Here we investigated whether SUR1-TRPM4 upregulation increases chronic seizure susceptibility in epilepsy. **Methods**: SUR1-TRPM4 expression was assessed by immunohistochemistry in resected brain tissues electrographically sorted as normal or epileptic from epilepsy patients. To investigate the mechanism that SUR1-TRPM4 promotes seizures through neuron hyperexcitability, cortical cultures were recorded with microelectrode array, treated with low Mg^{2+} to induce hyperactivation, and co-treated with TRPM4 inhibitor CBA. To assess effects of SUR1-TRPM4 inhibition on chronic seizure susceptibility, a pentylenetetrazol (PTZ) kindling epilepsy model was used in mice. SUR1-TRPM4 was inhibited during PTZ kindling either pharmacologically by the FDA-approved SUR1 inhibitor glyburide or the TRPM4 inhibitor 9-phenanthrol, or genetically by constitutive or neuron-specific TRPM4 knock-out. **Results**: Compared to controls, SUR1-TRPM4 expression was increased within epileptic tissues resected from patients ($p<0.05$, paired t-test). Inhibition of SUR1-TRPM4 *in vitro* reduced neuronal hyperactivity induced by low Mg^{2+} ($p<0.01$, 2-way ANOVA). In all PTZ experiments, SUR1-TRPM4 inhibition significantly attenuated seizures ($p<0.0001$, logistical regression). **Conclusions**: This study suggests SUR1-TRPM4 expression is upregulated in epilepsy and increases chronic seizure susceptibility through neuronal hyperexcitation. SUR1-TRPM4 may be a clinically relevant biomarker and therapeutic target to reduce seizures.

A tale of two cell types: calcitonin gene related peptide activates neurons and oligodendrocyte precursors cells in a sex- and region-dependent fashion, Rebecca Lorsung, University of Maryland

Calcitonin gene-related peptide (CGRP)-expressing neurons in the parabrachial nucleus (PB) densely project to several brain structures implicated in the emotional processing of stressful stimuli, including the extended amygdala and the insula cortex. We are testing the overarching hypothesis that PB CGRP release in these structures is causally involved in anxiety. Using *in vivo* fiber photometry of a peptide sensor, we found that PB CGRP-expressing neurons release large dense core vesicles, containing neuropeptides including CGRP, in the extended amygdala in response to an acute stressful stimulus – foot shock. Once CGRP is released, it can directly modulate brain activity via CGRP receptors on neurons, or indirectly via receptors on oligodendrocyte precursor cells (OPCs). Using a combination of whole-cell, voltage-clamp electrophysiology in live brain slices, as well as *in vitro* culturing of primary mixed brain culture, we discovered that CGRP exerts a sex- and region-dependent effect on these two cell types. In

the extended amygdala, CGRP preferentially promotes hyperexcitability in female neurons by potentiating neuronal responsiveness to excitation while simultaneously decreasing responsiveness to inhibition (disinhibition). Male extended amygdala neurons are partially protected due to a compensatory, CGRP-dependent increase in inhibitory tone. However, in the insula cortex, CGRP acts exclusively via CGRP receptors on OPCs; neither endogenous CGRP release from PB terminals, nor acute application of exogenous CGRP, alters insula neuronal responsiveness. These findings are supported by single cell RNAseq studies in both mice and humans, and RNAscope in mice, where we confirmed that only OPCs, not neurons, express CGRP receptors in the insula cortex. We are investigating whether CGRP in the extended amygdala plays a causal role in driving sex differences in anxiety and pain behaviors, and determining how CGRP-dependent activation of OPCs alters neuronal activity.

Early life injury alters spinal astrocyte development, Judy Yoo, University of Cincinnati

9% of children born in the United States are admitted to the neonatal intensive care unit where many experience invasive surgical procedures as part of their medical care. Exposure to invasive procedures in early life increases the excitability of the spinal nociceptive network and contributes to 'priming' of the nociceptive pathway, causing increased pain perception in response to noxious stimuli later in life. Astrocytes orchestrate synapse development and function; however, nothing is currently known about how developing spinal astrocytes respond to early life injury. Thus, we sought to elucidate the effects of neonatal surgical injury on the structure and transcriptional profile of astrocytes in the developing dorsal horn. At postnatal day 3 (P3), mice expressing tdTomato in astrocytes received either a unilateral incision of the hindpaw skin and underlying muscle or anesthesia only as controls. We then imaged astrocytes in the ipsilateral dorsal horn at P4, P10, and P24 using confocal microscopy in cleared tissue for accurate 3D reconstruction. Using quantitative morphological analysis, we found that astrocytes in the P3 incision group exhibit increased cell size and complexity at P4, while their size and complexity were reduced at both P10 and P24 when compared to naïve controls. Next, we used mice with green fluorescent protein (GFP)-expressing astrocyte nuclei and performed bulk RNA sequencing on sorted GFP+ nuclei to identify differentially expressed genes (DEGs) in astrocytes between incised mice and naïve controls. Our transcriptomic analysis revealed 76 DEGs between naïve and incision groups at P4, 2 DEGs at P10, and 8 DEGs at P24. Among these DEGs, we found that genes coding for matricellular proteins such as *Thbs1* and *Efemp1* and cytoskeletal genes such as *Acta1* and *Acta2* are upregulated at P4 following incision. Our data show, for the first time, that early life injury evokes morphological and transcriptional changes in developing spinal astrocytes.

Concurrent Session 3B

II. Molecular Biology and Pharmacology (Elevation)

RyR2 mediated calcium dysregulation increases glyoxalase l in the aging brain, Elizabeth Woo, Yale School of Medicine

The greatest risk factor for sporadic Alzheimer's disease is aging. With age, there is loss of regulation of cAMP-PKA-calcium signaling, which is critical for supporting working memory, leading to chronically elevated cytosolic calcium. Specifically, phosphorylation of ryanodine receptor 2 (RyR2) at S2808 causes calcium to "leak" from the smooth endoplasmic reticulum, occurring in Alzheimer's disease, chronic stress models, and long COVID-19. However, the potential compensatory mechanisms utilized by neurons in response to calcium dysregulation are not well understood. Our study utilized a genetic mouse model, S2808D-RyR2 (S2808D), causing a constitutive calcium leak through a knock-in mutation, and the aging rhesus macaque model. We performed mass spectrometry on synaptosomes from the frontal cortex (FC) and hippocampus (HP) of 3-month-old mice, followed by Ingenuity Pathway Analysis. Among the significantly differentially expressed proteins, glyoxalase 1 (Glo1) was 2.17 times higher ($p= 0.0014$) and 2.22 times higher ($p=0.011$) in the S2808D FC and HP synaptosomes, respectively. All findings were validated using immunoblotting techniques. S2808D synaptosomes also had significantly increased Glo1 activity in the FC ($p=0.0015$) and the HP ($p=0.0146$). Glo1 is a critical enzyme responsible for removing glycative stress, and it has been implicated in diabetes, neurodegeneration, and psychiatric disorders. The significant increase in Glo1 expression and activity was found in adult S2808D FC and HP synaptosomes. In aged rhesus macaques, we also found significantly increased Glo1 expression in the prefrontal cortex (PFC) ($p=0.0381$), indicating this restorative mechanism also occurs in primates. Pairing MLIF and immuno-electron microscopy, we quantified and characterized the localization of Glo1 for the first time in macaque PFC. Taken together, these findings suggest that elevated Glo1 expression may provide resilience in response to dysregulated calcium levels in neurons.

PDGFRa signaling regulates Srsf3 transcript binding to affect PI3K signaling and endosomal trafficking Thomas Forman, University of Colorado

Signaling through the platelet-derived growth factor receptor alpha (PDGFRa) plays a critical role in craniofacial development, as mutations in *PDGFRA* are associated with cleft lip/palate in humans and *Pdgfra* mutant mouse models display varying degrees of facial clefting. Phosphatidylinositol 3-kinase (PI3K)/Akt is the primary effector of PDGFRa signaling during skeletal development in the mouse. We previously demonstrated that Akt phosphorylates the RNA-binding protein serine/arginine-rich splicing factor 3 (Srsf3) downstream of PI3K-mediated PDGFRa signaling in mouse embryonic palatal mesenchyme (MEPM) cells, leading to its nuclear translocation. We further showed that ablation of *Srsf3* in the murine neural crest lineage results in severe midline facial clefting, due to defects in proliferation and survival of cranial neural crest cells, and over a thousand differential alternative RNA splicing events. Here, we demonstrate via enhanced crosslinking and immunoprecipitation (eCLIP)-seq analysis of MEPM cells that PDGF-AA stimulation leads to preferential binding of Srsf3 to exons. Further, an unbiased motif enrichment analysis of Srsf3 binding sites revealed a loss of binding to canonical Srsf3 CA-rich motifs in stimulated samples, which could be due to changes in ribonucleoprotein composition

or loss of RNA-binding due to electrostatic repulsion. Through the analysis of complementary RNA-seq data, we show that the subset of transcripts that are bound by Srsf3 and undergo alternative splicing upon PDGFRa signaling commonly encode regulators of Wnt signaling, a pathway known to be critical for mammalian craniofacial development. Taken together, these findings provide considerable insight into the mechanisms underlying gene expression regulation during mammalian craniofacial development.

The non-coding cause of congenital heart defect: abnormal RNA splicing with multiple novel isoforms as a mechanism for heterotaxy, John Wells, Indiana University

Heterotaxy is a disorder characterized by severe congenital heart defects (CHDs) and abnormal left-right patterning in other thoracic or abdominal organs. Clinical and research-based genetic testing has previously focused on evaluation of coding variants to identify causes of CHDs, leaving non-coding causes of CHDs largely unknown. Variants in the transcription factor Zinc finger of the cerebellum 3 (*ZIC3*) cause X-linked heterotaxy. We identified an X-linked heterotaxy pedigree without a coding variant in *ZIC3*. Whole genome sequencing revealed a novel, deep intronic variant (*ZIC3* c.1224+3286A>G) predicted to alter RNA splicing. An *in vitro* minigene splicing assay confirmed the variant acts as a cryptic splice acceptor. CRISPR/Cas9 served to introduce the *ZIC3* c.1224+3286A>G variant into human embryonic stem cells demonstrating pseudoexon inclusion caused by the variant. Surprisingly, Sanger sequencing of the resulting *ZIC3* c.1224+3286A>G amplicons revealed several novel isoforms, many of which by-pass the normal coding sequence of the third exon of *ZIC3*, causing a disruption of a DNA binding domain and a nuclear localization signal. Short- and long-read mRNA sequencing confirmed these initial results and identified additional novel splicing patterns. Assessment of four novel isoforms determined an abnormal function *in vitro* and *in vivo* while treatment with a splice-blocking morpholino partially rescued ZIC3. These results are the first reported instance of pseudoexon inclusion in heterotaxy and provide functional validation of non-coding disease causation. Our results suggest the importance of non-coding variants in heterotaxy and the need for improved methods to identify and classify non-coding variation that may contribute to CHDs.

Poster Sessions

Poster Session I

Sponsored by FAER

1. **Maya Anand**
   University of Rochester
   Discussion Prioritization Tool Eliciting Preferences of Older Adults with Breast Cancer Considering Adjuvant Chemotherapy: A Pilot Usability Study
2. **Adriana Baker**
   South Texas MSTP at University of Texas Health San Antonio
   ERX-315: A Potential Treatment Option for Hepatocellular Carcinoma (HCC)
3. **Emily Balczewski**
   University of Michigan MSTP
   Computable phenotyping of antiphospholipid syndrome with electronic health record data
4. **Carl Bannerman**
   Perelman School of Medicine
   The Role of Interferon Epsilon in Human Neurons
5. **Riley Behan-Bush**
   University of Iowa
   Polychlorinated biphenyls induce macrophage immmunometabolic plasticity
6. **John Bennett**
   University of Rochester
   Chemoenzymatic Synthesis and Anticancer Activity of a New Class of Skeletally Diverse Natural Product-like Compounds
7. **Caylin Billingsley**
   Indiana University School of Medicine
   Cellular Growth Mindset: HPV 16E6 and NFX1-123 Partnership Amplifies Proliferation of Long-Term Keratinocyte Cultures
8. **Stephanie Busch**
   Zucker School of Medicine at Hofstra/Northwell
   Isolation, in-vitro expansion, and characterization of menstrual effluent-derived uterine natural killer cells
9. **Justin Campbell**
   University of Utah School of Medicine
   DIRECT ELECTRICAL STIMULATION OF THE BASOLATERAL AMYGDALA MODULATES OSCILLATORY DYNAMICS IN THE HIPPOCAMPUS
10. **Nicholas Chargo**
    Michigan State University College of Osteopathic Medicine DO/PhD Program
    Dynamic effects of dietary prune on the gut-bone axis in healthy female mice
11. **Steve Cho**
    University of Utah MSTP

Directed evolution of a nascent immune function

12. **Aidan Crowley**
    University of Pennsylvania Perelman School of Medicine
    Performance of Physician Groups and Hospitals in Bundled Payments for Care Improvement Advanced for Lower Extremity Joint Replacement
13. **Paarth Dodhiawala**
    University of Minnesota Medical School
    KRAS inhibition enhances NK cell-mediated tumor killing via potential ULBP an IL-15 mediated mechanism in pancreatic cancer
14. **Nada Elsayed**
    Vanderbilt University
    THE CELLULAR LANDSCAPE OF CORTICAL DEVELOPMENT IN TUBEROUS SCLEROSIS COMPLEX
15. **Brianna Evans**
    Pennsylvania State College of Medicine
    Acute treatment with the glucagon-like peptide-1 receptor agonist, liraglutide, reduces cue-induced fentanyl seeking and associated c-Fos and tyrosine hydroxylase activation patterns in brain in rats
16. **Ilaria Ferrari**
    University of Louisville
    Exercise Prior to Myocardial Infarction has Minimal Impact on Ventricular Remodeling
17. **Sarah Florig**
    University of Nebraska Medical Center
    The Impact of Telehealth on Clinician Ordering Behaviors and Test Completion Rates
18. **Arlene Garcia**
    Vanderbilt University
    Elucidating mechanisms of mitochondrial DNA copy number decline in aging
19. **Elsie Gonzalez-Hurtado**
    Yale University School of Medicine
    Nerve-associated macrophages control adipose homeostasis across lifespan and restrain age-induced inflammation
20. **Freedom Green**
    South Texas MSTP at University of Texas Health San Antonio
    Leading the Pack: Strategies for Supporting Service Dog Handlers and Individuals with Disabilities and Chronic Illnesses in STEM
21. **Candace Grisham**
    Vanderbilt University
    Role of Fibroblast Activation Protein in Cancer Therapeutic Efficacy
22. **Cristhian Gutierrez Huerta**
    Medical College of Wisconsin MSTP
    Mitochondrial fission in the microvascular endothelium leads to microvascular dysfunction in humans

23. **Alexandra Hommer**
    Cornell University Combined DVM/PhD
    The role of subcellular protein methionine oxidation and methionine sulfoxide reductase (MSR) activities in aging
24. **Fengling Hu**
    University of Pennsylvania School of Medicine
    Automated Segmentation of Multiple Sclerosis Lesions, Paramagnetic Rims, and Central Vein Sign on MRI
25. **Miranda Jankovic**
    UT Southwestern Medical Center
    The Foxp2 Influence on the Developing Auditory Pathway
26. **Claudia Rose Keating**
    Loyola University Chicago
    Characterizing the role of connective tissue growth factor in hepatocellular carcinoma
27. **Thomas Kelly**
    Medical College of Wisconsin
    Selective deletion of mTOR in midbrain dopamine neurons impairs dopamine transmission and induces attention deficiency, hyperactivity and impulsivity
28. **Ruchita Kothari**
    Johns Hopkins MSTP
    Investigating the Role of a Mast Cell-Specific Receptor in Mediating Stroke Associated Inflammation
29. **Curtis Kuo**
    University of Michigan MSTP
    Loss of Myonuclear Type IIb and IIx Specificity in Spinal and Bulbar Muscular Atrophy
30. **Jessica Lam**
    University of Pennsylvania School of Medicine
    YY1 controlled regulatory connectivity and transcription ?are influenced by the cell cycle
31. **Kaylin Langer**
    University of Colorado School of Medicine
    Oligodendrocyte response and myelin composition following neonatal hypoxic ischemic brain injury
32. **Catherine Li**
    University of North Carolina at Chapel Hill MD-PhD Program
    Per-protocol effects of dolutegravir-based therapy compared to standard of care in children living with HIV in the ODYSSEY trial
33. **Mark Libowitz**
    University of Utah MSTP
    Underlying Effects of Intracranial Stimulation on Insula Functional Connectivity
34. **Samuel Liburd**
    Yale University

Vascular phenotype as predictive markers of CAR-T cell therapy response in patients with diffuse large B-cell lymphoma

35. **Ryan Mack**
    Loyola University Chicago
    RESOLUTION OF COMMON MYELOID PROGENITOR HETEROGENEITY AND IDENTIFICATION OF A NOVEL DIFFERENTIATION PATHWAY
36. **Charles Marcucci IV**
    University of Virginia MSTP
    Deciphering the role of tenascin-n in peripheral spinal motor nerve development
37. **Michael Meadow**
    MSTP at University of Rochester School of Medicine & Dentistry
    Proteome birthdating reveals age-selectivity of protein ubiquitination
38. **Bani Medegan Fagla**
    University of Illinois College of Medicine MSTP Program
    APOE4 genotype increases susceptibility to fetal growth restriction in the context of preeclampsia in a human cohort and in a mouse model of preeclampsia
39. **Amanda Paskavitz**
    Wayne State University School of Medicine
    Second-generation sperm epigenetic clock predicts male fertility age and time-to-pregnancy in general population
40. **Gavin Piester**
    University of Rochester School of Medicine & Dentistry
    THE SIGMA-1 RECEPTOR DRIVES ASTROCYTE PATHOGENIC ACTIVITIES IN PARKINSON'S DISEASE
41. **John Quinlan**
    University of Maryland, College Park
    Photodynamic Therapy in Glioblastoma Multiforme using Self-Assembled Verteporfin Nanoparticles
42. **Sarah Reed**
    Vanderbilt University MSTP
    Clonal hematopoiesis of indeterminate potential alters tumor microenvironment and clinical outcomes in solid tumors
43. **Kishan Sangani**
    University of Chicago
    Gut Check: Tet2 and Th2 Responses in the Intestine
44. **Luke Schroeder**
    University of Louisville
    Advancement of a 3D human U87 glioblastoma spheroid suspension model to augment traditional 2D tumor-derived small extracellular vesicle investigations
45. **Kallen Schwark**
    University of Michigan
    Optimizing PDGFRA Inhibition Therapy for Pediatric High-Grade Glioma
46. **Karishma Shah**
    University of Utah MSTP

When We Become Our Pain

47. **William Sheeran**
    University of Colorado
    Ventral hippocampal contributions to lasting social memory in prairie voles.
48. **Samantha Stone**
    University of Utah MSTP
    Mechanistic Interactions Between Depression, Selective Serotonin Reuptake Inhibitors (SSRIs), And Cancer Patient Survival
49. **Rhea Sullivan**
    Penn State College of Medicine
    Opioid-responsive microRNAs in brain stem cell fates: implications for neonatal opioid withdrawal syndrome infants
50. **Maya Talukdar**
    Harvard/MIT MD-PhD Program
    Genes of the fatty acid oxidation pathway are upregulated in the female as compared to male human heart
51. **Kaylee Tutrow**
    Indiana University School of Medicine
    A human induced pluripotent stem cell model of Alzheimer's Disease-associated fractalkine receptor polymorphism to assess AD-related microglial dysfunction
52. **Mayuri Viswanathan**
    University of Chicago MSTP
    Gamma constant usage tunes γδ T cell receptor sensitivity, thymic programming, and peripheral function
53. **Qi Wang**
    University of Iowa Carver College of Medicine
    Dynamic Mechanical Loading of Photopolymerized Hydrogels for Investigating the Interplay of Mechanical and Biochemical Signals in Pulmonary Fibrosis
54. **Nathaniel Wright**
    University of Chicago
    BRWD1 establishes epigenetic states for germinal center initiation, maintenance, and function
55. **Crystal Young**
    University of Michigan MSTP
    Hypothyroidism with Defective Thyroglobulin Co-opts the Machinery of Thyroid Hormone Synthesis to Drive Thyroid Cell Death
56. **Ashley Zachery-Savella**
    University of Utah MSTP
    Using Seizure Onset and Offset Patterns to Predict Drug Response in an Animal Model of Temporal Lobe Epilepsy
57. **Walter Zhao**
    Case Western Reserve University
    Automatic Risk Priors for Prediction of Glioblastoma Peritumoral Infiltration Using MR Fingerprinting and Multiparametric MRI

Poster Session II

Sponsored by Department of Pathology University of Colorado Anschutz

1. **Joshua Baker**
   Michigan State University College of Osteopathic Medicine
   Insights of Contemporary Diffusion MRI Modeling Techniques in Mild Traumatic Brain Injury: A TRACK-TBI Study
2. **Eric Barrientos**
   University of Colorado
   Shear-Dependent Volume Analysis of Platelet Aggregates under Pharmacological Inhibition in Microfluidic Devices
3. **Ben Battistone**
   University of Utah
   T cell miR-155 Regulates mRNA Vaccine Immunotherapy Against Acute Myeloid Leukemia
4. **Riley Bergman**
   Vanderbilt University School of Medicine
   The role and tolerability of SF3B1 hotspot mutations in breast cancer
5. **Daniel Caballero**
   Perot Family Scholars MSTP at UT Southwestern
   The yeast Mkt1/Pbp1 complex promotes adaptive responses to respiratory growth
6. **Hannah Carter**
   University of Michigan
   Dendritic Cell - Fibroblast Crosstalk Via Toll-like Receptor 9 and Aryl Hydrocarbon Receptor Signaling Drives Lung Fibrogenesis
7. **Thomas Delgado**
   University of Rochester
   Depletion of Astrocytic TG2 Enhances the Ability of Astrocytes to Metabolically Support Neurons through Lipid Metabolism
8. **Julie Disharoon**
   Medical University of South Carolina MSTP
   The role and regulation of BRD4 in the DNA Damage Response
9. **Caroline Doherty**
   Mayo Clinic
   In Vivo Selection of anti-GBM DNA aptamers in an orthotopic patient derived xenograft model
10. **Jacob Edmondson**
    University of Arkansas for Medical Sciences
    ATF6 activation in melanoma promotes anti-tumor immunity and improves ICB therapy response
11. **Eric Engelbrecht**
    University of Louisville School of Medicine MD/PhD Program

Impact of Human Immunoglobulin Genetic Variants on the Expressed Antibody Repertoire

12. **Alan Finkelstein**
    University of Rochester
    Integrated Microvascular and Microstructural Characterization through Intravoxel Incoherent Motion Tensor Imaging
13. **Kevin Fundora**
    Penn State College of Medicine
    Targeting VPS4 as a novel anticancer therapy
14. **Kerrie Greene**
    Yale University
    Intranasal Delivery of CD4 T Cell Peptides for Enhanced Therapeutic Access to the CNS
15. **Julian Grosskopf**
    Medical College of Wisconsin
    Modeling protein conformational ensembles using experimental DEER spectroscopy data
16. **Samson Hennessy-Strahs**
    Texas A&M School of Medicine
    Conditional Deletion of mTOR/TRAF6 Reduces Alveolar Macrophages and Leads to Alveolar Proteinosis
17. **Laurel Hiatt**
    University of Utah
    STRategies for STRs' STRangeness with STRchive, a dynamic resource for short tandem repeat disease loci in humans
18. **Reuben Hogan**
    University of California, San Francisco
    Comparâtes Analysis of Glycoproteomic Software Tools Using a Tailored Glycan Database
19. **Anna Hudson**
    University of Miami Miller School of Medicine
    Overcoming the Immunosuppressive Glioblastoma Microenvironment by targeting HERV-K
20. **Katherine Irwin**
    Johns Hopkins MSTP
    A fluid biomarker reveals loss of TDP-43 splicing repression in presymptomatic ALS–FTD
21. **Amanda Jiang**
    University of Utah
    Unraveling MC1R Gene Variants in Lentigo Maligna Melanoma in the Utah Population
22. **Austin Jolly**
    University of Colorado
    Remodeling of the vascular wall is regulated by the chromatin remodeler Brg1

23. **Jakub Kaczmarzyk**
    Stony Brook University
    Towards transparent and trustworthy AI in precision oncology and computational pathology
24. **Shanawaj (Roy) Khair**
    University of Colorado School of Medicine
    ROLE OF ANTIMICROBIAL PEPTIDES IN POST-BURN PULMONARY INFLAMMATION
25. **Katie Kruk**
    University of Maryland, Baltimore
    A Mechanism by Which Estradiol (E2) Regulates Adenosinergic Signaling in the Median Preoptic Nucleus (MnPO)
26. **Michael Lee**
    University of Texas Southwestern
    LINE-1 retrotransposon transcription mediates oncogenic chromatin interaction hubs
27. **Siying Lin**
    Medical College of Wisconsin
    Satb1 regulates the stemness of antigen-specific CD8 T+ cells
28. **Matthew Loberg**
    Vanderbilt University
    Multi-omic analysis identifies fibroblast subpopulations associated with aggressive thyroid cancer behavior
29. **Alexander Lu**
    Texas A&M College of Medicine
    O-GlcNAcylation Regulates Angiogenic Transdifferentiation Through HIRA Dependent H3.3 Deposition
30. **Adarsha Malla**
    University of Maryland, Baltimore
    Adjuvant transcranial microbubble-enhanced focused ultrasound treatment of infiltrating gliomas in humans and rodents
31. **Anna Maximova**
    University of Maryland Baltimore
    Investigating the impact of a unique peri-hippocampal mast cell population in postnatal rat neurodevelopment
32. **Jennifer Messina**
    SUNY Upstate Medical University
    Battling Hyperinflammation and Pyroptosis in Sepsis Using Bioactive Nanoparticles
33. **Raymond Ng**
    University of Pennsylvania School of Medicine
    The role of tumor transcriptional variability in evading acute CD8+ T cell exposure in melanoma
34. **Fae Nova**
    Pennsylvania State University College of Medicine

A multimodal 3D developmental mouse brain common coordinate framework for cell census mapping

35. **Ian Outhwaite**
    SUNY Stony Brook University
    Kinase Inhibitor Combinations Maximize Selectivity and Enable Rational Multitargeting
36. **Julia Phan**
    University of Texas Southwestern
    Retrotransposons are Co-opted by Hematopoietic Stem Cells During Pregnancy to Increase Erythropoiesis
37. **Randall Rainwater**
    University of Arkansas School for Medical Sciences
    The Novel Role of DNA-PKcs in LAT-mediated T cell Signaling
38. **Roshan Ravishankar**
    University of Texas Southwestern Medical Center
    Automated Detection of Rho GTPase Signaling Microdomains
39. **Melissa Requist**
    University of Utah MSTP
    Proposed Mechanisms of Osseous Morphology Differences in Charcot Marie Tooth Disease
40. **Juan Santiago Moreno**
    University of Colorado Anschutz Medical Program
    THE DISTRIBUTION OF CHROMATIC TUNING IN THE MOUSE EARLY VISUAL SYSTEM
41. **Vivien Sauer**
    University of Cincinnati College of Medicine MSTP
    Investigating the role of SMAD6 in the development of tracheoesophageal birth defects
42. **Ankita Saxena**
    SUNY UPSTATE MEDICAL UNIVERSITY
    Application of Graph Neural Networks in Disease Risk Predictions: Augmenting GWAS with Novel Insights into Biological Networks
43. **Justin Schumacher**
    University of Maryland School of Medicine
    Clinical multimodal elastography for measuring spatial-varying mechanical properties of the crystalline lens.
44. **Kayla Schwartz**
    University of Miami MSTP
    Pancreatic cancers suppress fibroinflammatory signals to support metastatic outgrowth
45. **Manlin Shao**
    University of Arizona College of Medicine-Tucson
    Orthotopic and Immunocompetent Mouse Models for Oral Squamous Cell Carcinoma
46. **Kelli Sommers**

Medical College of Wisconsin
Immune-mediated microtubule destabilization induces axonal transport defects in a mouse model of optic neuritis

47. **Jonathan Sussman**
University of Pennsylvania School of Medicine
A longitudinal single-cell and spatial multiomic atlas of pediatric high-grade glioma
48. **Josephine Trichka**
Case Western Reserve University MSTP
CHMP5 restrains the tissue-intrinsic interferon response in skeletal muscle
49. **Jude Tunyi**
The Ohio State MSTP
Computational and Biochemical Characterization of Amino Acid Transporter Protein-protein Interactions
50. **Amanda Velez**
University of North Carolina at Chapel Hill
Staphylococcus aureus pathogenesis within the polymicrobial diabetic chronic wound
51. **Madison Wahlen**
University of Iowa
Identifying Adaptations to Preserve Core Functions of an Evidence-Based Intervention to Improve Cancer Care Quality in Rural Hospitals
52. **Jenna Weber**
University of Utah
Paternal Age Effect Mutations: Selfish Drivers of Germline Stem Cell Clonality?
53. **Clare Wieland**
University of Michigan
Neuron specific regulation of repeat associated non-AUG translation in age related dementias
54. **John Yap**
Loyola University Chicago, Stritch School of Medicine
Unraveling the role of PDK4 in sex dependent outcomes during sepsis induced cardiomyopathy
55. **Grace Yu**
Mayo Clinic MSTP
Effect of Senescent Melanocytes on Epidermal Skin Aging
56. **Deanne Yugawa**
Yale MD-PhD Program
ERK potentiates its own activity in pancreatic cancer via alterations in RNA splicing
57. **Victor Zhang**
University of Rochester
Type III collagen recreates the fibrotic myofibroblast microenvironment for studying peritendinous scarring in a human Tendon-on-a-Chip (hToC)
58. **Sherry Zhou**
Mayo Clinic M.D.-Ph.D. Program

Investigating post-translational protein acylation in SDH-loss pheochromocytoma and paraganglioma

2024 Travel Awardees

Travel Awardees	
Brianna Evans, Pennsylvania State University	**Marissa Chou**, University of California San Francisco
Claudia Rose Keating, Loyola University Chicago	**Caylin Billingsley**, Indiana University
Kaylee Tutrow, Indiana University	**Ashley Zachery-Savella**, University of Utah
Siying Lin, Medical College of Wisconsin	**Jacob Edmonson**, University of Arkansas
Cristhian Gutierrez Huerta, Medical College of Wisconsin	**Jonathan Sussman**, University of Pennsylvania

Saturday Morning Activities

Breckenridge is an ideal location for exploring the Rocky Mountains! Several activities will be planned and organized by University of Colorado MSTP students. While these activities are subject to change due to inclement weather, they have usually happened as planned in years past.

Hiking

For all hikes, please bring the following: a pair of supportive shoes or boots, sunscreen, water, snack food, loose-fitting clothes, a warm layer, and a rain/wind-resistant layer.

***Easy Hike* – Minne Mine (2.9 mi 300 ft elev. gain.)**: Just a five-minute drive from Breckenridge, you and your friends will be positioned to explore the easy trails and explore old mining ruins. The hike from B&B to Reiling Dredge to Minnie Mine near Breckenridge offers a scenic and historical experience. Starting at the B&B Trailhead, the trail takes you through lush forests and past the historic Reiling Dredge, an old mining dredge from the early 1900s. The hike continues to Minnie Mine, where you can explore remnants of mining activity while enjoying picturesque views of the surrounding mountains. Breckenridge offers a shuttle service to trail head leaving every 30 min.

Meet at the Registration Table at 9:00am.

***Intermediate Hike* - Mayflower Lake (5.5 mi 1000 ft elev. gain.)** The hike to Mayflower Lake near Breckenridge offers stunning alpine scenery, leading you past cascading waterfalls, historic mining ruins, and lush wildflower meadows, culminating in breathtaking views of the pristine high-altitude lakes.

Meet at the Registration Table at 8:00am.

Volleyball

There's nothing like a crazy game of volleyball to have a good time! We will set up a volleyball net at Carter Park and Pavilion about a 15-minute walk from the Conference Center. If you want to partake in some fun rallies, come join the game!

Meet at the Registration Desk at 9:15am.

Day Trip to Breckenridge

Downtown Breckenridge offers a charming blend of historic and modern attractions, with a vibrant Main Street lined with unique shops, art galleries, and a variety of food options. Visitors can explore cultural sites like the Barney Ford House Museum, which showcase the town's rich mining history. The downtown area features an array of specialty shops, including boutiques, souvenir stores, and artisan craft shops, providing a delightful shopping experience for all visitors.

Meet at the Registration Desk at 9:30am

Breakfast and Board Games

In case of inclement weather, or for those who prefer a more leisurely Sunday morning indoors, we will have a room reserved where students can enjoy a variety of traditional games such as Chess and GO as well as modern classics like Catan and Carcassonne. Players of all experience levels are welcome!

Meet in the Evergreen Ballroom at 9:30 am

** All activities are weather dependent, non-refundable, non-transferrable, and we do not offer any refunds regardless of activity closures. Additionally, Activity Day Passes are day specific and we do not offer refunds on partially used day passes.

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Student Participants

First Name	Last Name	University Program
Mauricio	Alvarez	University of Colorado MSTP
Maya	Anand	University of Rochester
Adriana	Baker	South Texas MSTP at University of Texas Health San Antonio
Joshua	Baker	Michigan State University
Emily	Balczewski	University of Michigan MSTP
Amandip	Bangar	University of Colorado MSTP
Carl	Bannerman	Perelman School of Medicine
Eric	Barrientos	University of Colorado MSTP
Ben	Battistone	University of Utah
Hannah	Beatty	University of Colorado MSTP
Riley	Behan-Bush	University of Iowa
David	Beltran-Cardona	University of Colorado MSTP
John	Bennett	University of Rochester
Riley	Bergman	Vanderbilt University School of Medicine
Jessica	Beynor	University of Colorado MSTP
Caylin	Billingsley	Indiana University School of Medicine
Karina	Bursch	Medical College of Wisconsin
Stephanie	Busch	Zucker School of Medicine at Hofstra/Northwell
Daniel	Caballero	Perot Family Scholars MSTP at UT Southwestern
Alex	Camai	University of Colorado MSTP
Justin	Campbell	University of Utah School of Medicine
Hannah	Carter	University of Michigan
Sofia	Celli	University of Colorado MSTP
Nicholas	Chargo	Michigan State University College of Osteopathic Medicine
Michelle	Cho	University of Colorado MSTP Denver MSTP
Steve	Cho	University of Utah MSTP
Marissa	Chou	University of California, San Francisco
Rachel	Cohen	University of Colorado MSTP
Jacob	Connolly	University of South Florida
Aidan	Crowley	University of Pennsylvania Perelman School of Medicine
Thomas	Delgado	University of Rochester
Julie	Disharoon	Medical University of South Carolina MSTP
Keith	Dodd	University of Colorado MSTP
Paarth	Dodhiawala	University of Minnesota Medical School
Caroline	Doherty	Mayo Clinic
Jacob	Edmondson	University of Arkansas for Medical Sciences
Selin	Ekici	University of Colorado MSTP

Nada	Elsayed	Vanderbilt University
Eric	Engelbrecht	University of Louisville School of Medicine MD/PhD Program
Elena	Esch	University of Colorado MSTP
Sofia	Esquibies	University of Colorado MSTP
Brianna	Evans	Pennsylvania State College of Medicine
Ilaria	Ferrari	University of Louisville
Alan	Finkelstein	University of Rochester
Erin	Fish	University of Colorado MSTP
Ira	Fleming	University of Colorado MSTP
Ian	Fleming	University of Colorado MSTP
Sarah	Florig	University of Nebraska Medical Center
Thomas	Forman	University of Colorado MSTP
North	Foulon	University of Colorado MSTP
Kevin	Fundora	Penn State College of Medicine
Allison	Galante	Renaissance School of Medicine at Stony Brook University
Arlene	Garcia	Vanderbilt University
Stefano	Ginocchio	University of Colorado MSTP
Volha	Golubeva	Ohio State University School of Medicine
Elsie	Gonzalez-Hurtado	Yale University School of Medicine
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Kerrie	Greene	Yale University
Candace	Grisham	Vanderbilt University
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Cristhian	Gutierrez Huerta	Medical College of Wisconsin MSTP
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Laurel	Hiatt	University of Utah
Brandon	Hilliard	University of Colorado MSTP
Reuben	Hogan	University of California, San Francisco
Alexandra	Hommer	Cornell University Combined DVM/PhD
Shannon	Hu	Renaissance School of Medicine at Stony Brook University
Fengling	Hu	University of Pennsylvania School of Medicine
Isabelle	Hua	University of Colorado MSTP
Anna	Hudson	University of Miami Miller School of Medicine
Katherine	Irwin	Johns Hopkins MSTP
Miranda	Jankovic	UT Southwestern Medical Center
Amanda	Jiang	University of Utah
Austin	Jolly	University of Colorado MSTP
Jakub	Kaczmarzyk	Stony Brook University
Raisa	Karim	Renaissance School of Medicine at Stony Brook University
Sritejasvinithi	Karimikonda	Medical College of Wisconsin

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Thomas	Kelly	Medical College of Wisconsin
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Ward	Kirschbaum	University of Colorado MSTP
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Haley	Klimaszewski	The Ohio State University MSTP
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Katie	Kruk	University of Maryland, Baltimore
Curtis	Kuo	University of Michigan MSTP
Jessica	Lam	University of Pennsylvania School of Medicine
Kaylin	Langer	University of Colorado MSTP
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Emily	Leventhal	Icahn School of Medicine at Mount Sinai
Steven	Lewis	Stony Brook University
Catherine	Li	University of North Carolina at Chapel Hill MD-PhD Program
Mark	Libowitz	University of Utah MSTP
Samuel	Liburd	Yale University
Shujian	Lin	University of Colorado MSTP
Siying	Lin	Medical College of Wisconsin
Matthew	Loberg	Vanderbilt University
Vincent	Lok	University of South Florida Morsani College of Medicine
Rebecca	Lorsung	University of Maryland School of Medicine
Alexander	Lu	Texas A&M College of Medicine
Ryan	Mack	Loyola University Chicago
Adarsha	Malla	University of Maryland, Baltimore
Charles	Marcucci IV	University of Virginia MSTP
Matthew	Mardo	University of Colorado MSTP
Anna	Maximova	University of Maryland Baltimore
Nicholas	McQuillan	University of Colorado MSTP
Michael	Meadow	MSTP at University of Rochester School of Medicine & Dentistry
Bani	Medegan Fagla	University of Illinois College of Medicine MSTP
Jennifer	Messina	SUNY Upstate Medical University
Daniel	Moskop	University of Colorado MSTP
Mitchell	Moyer	University of Maryland
Rahul	Nachnani	Pennsylvania State University College of Medicine
Raymond	Ng	University of Pennsylvania School of Medicine
Fae	Nova	Pennsylvania State University College of Medicine
Younhye	Ock	University of Colorado MSTP
Denis	Ohlstrom	Emory MSTP
Duby	Okonkwo	Vanderbilt School of Medicine MSTP

Margo	Orlen	University of Pennsylvania School of Medicine
Ian	Outhwaite	SUNY Stony Brook University
Amanda	Paskavitz	Wayne State University School of Medicine
Julia	Phan	University of Texas Southwestern
Gavin	Piester	University of Rochester School of Medicine & Dentistry
Megan	Pino	The Ohio State University College of Medicine
Charlotte	Pollack	University of Colorado MSTP School of Medicine
John	Quinlan	University of Maryland, College Park
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Roshan	Ravishankar	University of Texas Southwestern Medical Center
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Courtney	Vetter	University of Colorado MSTP
Mayuri	Viswanathan	University of Chicago MSTP
Madison	Wahlen	University of Iowa
Madison	Walker	University of Colorado MSTP
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Margaret	Weiss	University of Illinois at Chicago
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Clare	Wieland	University of Michigan
Ben	Wong	University of Colorado MSTP
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Deanne	Yugawa	Yale MD-PhD Program
Ashley	Zachery-Savella	University of Utah MSTP
Victor	Zhang	University of Rochester
Walter	Zhao	Case Western Reserve University
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Samuel	Zorn	Medical College of Wisconsin